THE INTERSECTION OF GENES, THE ENVIRONMENT, AND CRIME AND DELINQUENCY: A LONGITUDINAL STUDY OF OFFENDING

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ABSTRACT

The discipline of criminology has been dominated by social and environmental explanations to crime, criminality, and delinquency. At the same time, biogenic theories of antisocial behavior have historically been marginalized, ridiculed, and ignored by criminologists. This is somewhat surprising given the large and ever-expanding body of empirical research revealing strong genetic underpinnings to most behaviors and most personality traits. However, recent behavioral genetic research has shown that the most accurate explanations to human development incorporate both biological/genetic factors and social influences. The current dissertation builds off this line of literature and uses a genetically-sensitive subsample of the National Longitudinal Study of Adolescent Health (Add Health) to examine whether genetic forces combine with the social environment to create antisocial behaviors. Specifically, five different genetic polymorphisms (DAT1, DRD2, DRD4, 5HTT, and MAOA) are used to test for gene X environment correlations and gene X environment interactions in the etiology of crime and delinquency. The results of the multivariate models revealed genetic influences are important contributors to the field of criminology. The most consistent effects, however, were found when examining gene X environment correlations and gene X environment interactions. The implications for criminology and criminologists are discussed.

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I dedicate this project to my wife, Shonna, who unselfishly sacrificed so much throughout the duration of my graduate studies. She silently endured the hardships of living in less-than-ideal conditions, of having husbandless nights and weekends, and of sharing me with the computer so I could pursue my degree. For all of those reasons (and many more) I am eternally grateful and forever indebted to her. To my beautiful baby girl, Brooke, who along with my wife, has taught me more about life than she will ever know. Thank you. We finally can begin our "real life" together as a family.

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CHAPTER 1

INTRODUCTION

Criminology has been dominated by sociological explanations of crime and criminals. For example, the leading criminological theories—social disorganization theory, social bonding theory, social learning theory, and strain theory—emphasize the role of social forces, such as the influence of neighborhoods (Sampson and Groves, 1989; Sampson, Raudenbush, and Earls, 1997; Wilson, 1987), families (Loeber and Stouthamer-Loeber, 1986; Patterson, 1982), and subcultures (Anderson, 1999) on the development of offending behaviors. The hegemony of these sociological theories has, however, come at a price: biological and genetic explanations of antisocial behavior have historically been cut out of criminology.

Part of the reason that biological/genetic theories of crime have been marginalized is because they are viewed as deterministic, dangerous, and ideologically incorrect (Kaplan, 2000). Perhaps the most worrisome reservation, however, is that genetic forces will outperform environmental influences in the scientific study of offending. Caspi, Roberts, and Shiner (2005:464) respond to this concern when they argue that "the responsible way to tackle the genetic challenge to socialization research is head on, by using genetically sensitive designs that can provide leverage in identifying environmental risks."

There is mounting and undeniable evidence revealing that most behaviors and personality traits are at least partly influenced by genetic factors. Additionally, a wealth of research investigating the causes of crime has shown empirically that certain dimensions of the social environment are particularly salient sources of variation in antisocial conduct. Depending on the specific trait or behavior of interest, the relative effects of both genetic and environmental

influences vary; sometimes genes are the dominant force and in other circumstances the environment is more potent. In general, however, both appear to be implicated, at least to varying degrees, in the development of most behaviors and traits. To take these two disparate lines of research into account, there has been a growing interest in the melding together of biological and social explanations of crime—an emerging perspective referred to as biosocial criminology (Walsh, 2002).

Statement of the Problem

Despite the growing interest in biosocial explanations to crime, much remains unknown about how genes impact the development of criminal behaviors and personalities. The mapping of the human genome and the spate of research attempting to uncover the functionality of certain genetic polymorphisms, however, has set the stage for more accurate and more detailed explanations of how genetics may influence crime and delinquency. Perhaps the most promising genes, at least in the etiology of deviancy, are those that aid in the production, transportation, and breakdown of certain neurotransmitters. Two of the most widely studied neurotransmitters—dopamine and serotonin—are functionally related to the regulation of behavior that may affect crime and offending. This dissertation will examine the direct effects that a dopamine transporter gene (DAT1), two dopamine receptor genes (DRD2 and DRD4), a serotonin transporter gene (5HTT), and monoamine oxidase A (MAOA) have on a range of antisocial outcomes.

Research also reveals that the relationship among genes, the environment, and crime and delinquency may be more complex than simple linear statistical models are able to detect. Indeed, recent findings suggest that genes may not have a direct effect on crime but rather may

interact with, or modify, certain environments to increase the odds of offending behavior (Caspi et al., 2002a; Haberstick et al., 2005). The process of the environment interacting with specific genes is referred to as a gene X environment interaction (GxE). In addition, genetic influences may exert their effects indirectly through the environment. This type of gene-environment interplay is referred to as a gene X environment correlation (rGE). The analysis for this dissertation will examine a series of GxEs and rGEs to determine if they are implicated in the production of crime, delinquency, and drug/alcohol use.

To examine these hypotheses, the current dissertation will use a biosocial approach to determine in what way genetic polymorphisms and the environment may affect the development of violent crime, aggressive behavior, and drug/alcohol abuse. Data come from a restricted-use data file of the National Longitudinal Study of Adolescent Health (Add Health). The Add Health data contain detailed information about adolescent delinquent behavior, drug and alcohol use, and adult criminality, including official arrest measures. Also available in the Add Health data are measures pertaining to neighborhood conditions, family life, economic circumstances, social relationships, and peer networks.

One of the unique features of the Add Health data is that unlike most nationally representative data sets, DNA information was also collected. A subsample of Add Health participants agreed to submit their DNA to be genotyped for genes that regulate the production and transportation of two neurotransmitters: dopamine and serotonin. In addition, monoamine oxidase A (MAOA), a gene that codes for an enzyme that synthesizes neurotransmitters, was also genotyped. The inclusion of both genetic variables and social measures provides an excellent opportunity to examine the biosocial influences on a wide range of criminal and deviant behaviors in adolescent and early adulthood.

Conclusion

Theoretical and empirical work seeking to understand offending behavior has tended to take a fragmentary and intra-disciplinary approach. Dominant environmental theories have narrowly focused on predicting crime in terms of social factors. Conversely, genetic explanations have sought to explain crime primarily through hereditary influences. Each of these perspectives, when examined separately, has left us with an incomplete and somewhat impoverished view into the etiology of criminality. The rigid boundaries between these two perspectives, however, are beginning to blur, and recent work suggests that one of the most promising approaches in criminological research is the blending together of environmental and genetic explanations (Caspi et al., 2002a). This dissertation adds to the biosocial literature and examines the direct, indirect, and interactive effects of five different genetic polymorphisms on antisocial behavior.

CHAPTER 2

THE GENETIC BASIS OF BEHAVIOR

One of the greatest accomplishments in the history of science was the mapping of the human genome, which was formally called the Human Genome Project (HGP). The HGP officially began in 1990 with financial-backing from the U.S. Department of Energy and the National Institutes of Health. The primary goals for the HGP were to identify all of the genes in humans (i.e., the human genome) and to determine the sequential arrangement of all the nucleotides found in DNA. Upon completion, the HGP would provide researchers with a wealth of information that would allow them to study the potential genetic origins of behavioral disorders, mental illnesses, various forms of psychopathology, and terminal diseases, among others. In 2003, after thirteen years of research, and the concerted effort of an international cast of scientists, the human genome—with its 3 billion base pairs—was mapped.

At the beginning of the HGP scientists estimated the human genome was composed of 100,000 genes. By the project's completion this number had shrunk to around 25,000. Even with the identification of the 25,000 genes that make up the human genome, there is still much to be learned about the functionality of these genes. Molecular biologists and molecular geneticists, for example, are actively engaged in research designed to reveal the role that certain genes play in healthy human development and in normal life functioning. This line of inquiry also holds particular promise for identifying the genes that are responsible for phenotypic differences in behavior. In a relatively short period of time, molecular genetic research has discovered specific genes linked to a wide range of disorders, including ADHD, alcoholism, delinquency, and even anorexia and bulimia. With an ever-expanding line of research examining

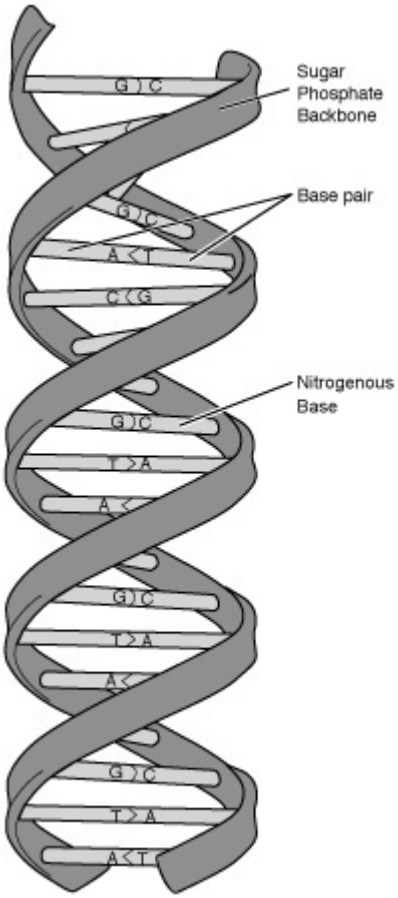
the link between human genetic variation and different outcomes, the list of genes implicated in the etiology of behavioral development and the formation of personality most certainly will grow. In order to understand how genes affect behavior, however, it is first necessary to present an introduction to genetics.

Introduction to Genetics

Deoxyribonucleic acid (DNA) is a chemical code that contains the genetic programming and information needed for an organism to form, develop, and live. Essentially, DNA can be thought of as a genetic blueprint that orchestrates the development and functioning of the human body. DNA is stored in the nucleus of every cell except red blood cells and, as will be discussed in detail below, is primarily responsible for two main functions: transcription and translation. The information encoded in DNA determines eye color, hair color, skin pigment, and practically every other imaginable physical feature. Human variation, in short, simply reflects each person's unique genetic code transcribed into their DNA.

The structure of DNA consists of two genetic fibers—each referred to as a polynucleotide—twisted around each other to form what is known as the double helix. The backbones (one backbone for each polynucleotide) of the double helix are formed from sugar phosphates. Along the backbone of each polynucleotide is a sequence of nucleotides (also called bases), which are carbon-nitrogen molecules. There are four nucleotides present in DNA: adenine (A), thymine (T), cytosine (C), and guanine (G). As shown in Figure 2.1, the bases protrude from the backbone of each DNA strand. The two strands of DNA are held together by base pairs. The formation of a base pair requires that two nucleotides—one from each strand of DNA—combine to join the two polynucleotides. Nucleotides, however, do not pair randomly

Figure 2.1. The Double-Helix Structure of DNA



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with one another; A can only bond with T, T can only bond with A, G can only bond with C, and C can only bond with G. The A-T and T-A base pairs are held together by two hydrogen bonds and the C-G and G-C base pairs are held together by three hydrogen bonds.

Figure 2.2 presents a hypothetical example of the formation of base pairs. In this example, the polynucleotide in the top portion of the diagram contains a series of nucleotides arranged as ACTGACTCCA. Given that A can only pair with T (and vice versa) and that C can only combine with G (and vice versa), the nucleotide sequence of the complementary strand of DNA, by default, is TGACTGAGGT. Of course, this example using only ten base pairs is oversimplified. This process is at work for the approximately 3 billion base pairs found in human DNA.

The sequential ordering of nucleotides and base pairs is just as important as the quantity of base pairs. Along with differences in the number of base pairs, the unique arrangement of nucleotides is what separates humans from all other forms of life. Very small divergences in the ordering of nucleotides translate into observable differences both within- and between-species. For example, humans and chimpanzees (*Pan troglodytes*) share 96 percent of their DNA (The Chimpanzee Sequencing and Analysis Consortium, 2005).[1] Only a 4 percent difference in DNA distinguishes humans from chimpanzees and accounts for qualities that make humans unique, such as the ability to talk, the ability to form complex thoughts, and the ability to process abstract information. In humans, even smaller variations in DNA create measurable and substantive changes. All humans share approximately 99.9 percent of their DNA (monozygotic twins, however, share ~100 percent of their DNA). Remarkably, each individual's unique sequential

[1] "Genetically speaking," claims Ghiglieri (1999:70) "humans are not just one more ape; they are a 'sibling species' so closely related to chimps that if anthropologists followed the same criteria of relatedness that mammalogists and ornithologists do when classifying genera, chimps and humans would be classified in the same genus: *Homo*."

Figure 2.2. A Hypothetical Example of a Base-Pair Sequence of DNA

A	**C**	**T**	**G**	**A**	**C**	**T**	**C**	**C**	**A**
..	…	..	…	..	…	..	…	…	..
T	**G**	**A**	**C**	**T**	**G**	**A**	**G**	**G**	**T**

Note:
Adapted from Rowe (2002)

arrangement of nucleotides—what geneticists call a genotype—differs by only .1 percent.[2] The variation that exists in the remaining .1 percent of human DNA is of particular interest to molecular geneticists because this difference accounts partially for human variation in personality, in behaviors, in physical characteristics, and in other personal attributes.

At various segments along the strands of DNA, in seemingly random places, contiguous base pairs work together to perform specialized functions. These groups of base pairs, operating in collaboration, are called genes. For instance, the following is a hypothetical example of a sequence of nucleotides: TAC**TGGGAT**TAG. Within this string of DNA, the bold-typed nucleotides could act in unison and thus conceivably make up part of a gene. In reality, however, genes are frequently comprised of 1,000 or more base pairs.

The main function of genes, and the base pairs within a gene, is to code for the production and regulation of proteins. Each gene is responsible for manufacturing one protein; multiple genes, however, may code for the synthesis of the same protein. Proteins are essential to human life. They form the shape and structure of cells, enable bodily movement, account for eye and skin color, provide the body with energy, form antibodies to ward off infections, and perform many other functions. Proteins are divided into two main categories: structural proteins and functional proteins. Structural proteins make up most of the solid material in the human body. Keratin and collagen are two of the most frequently occurring structural proteins. These two proteins are the main compounds found in hair, muscle tissue, tendons, fingernails, ligaments, and skin. Another structural protein, elastin, is a component of arteries, including the

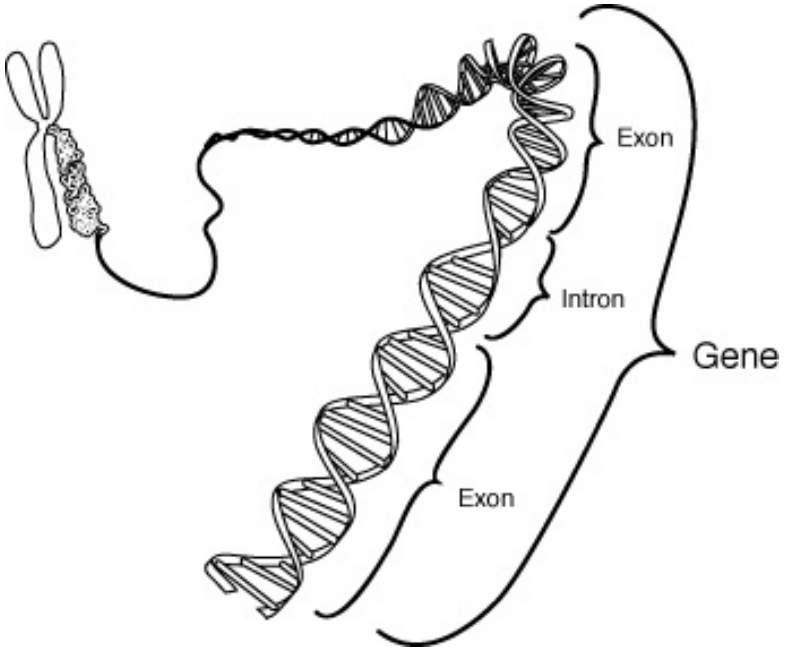
[2] It is important to point out that the human genome is comprised of approximately 3 billion base pairs. Even though human DNA differs, on average, by only .1 percent (of 3 billion base pairs), this small divergence translates into a difference of 3,000,000 base pairs—a difference large enough to explain, at least partially, phenotypic variation. As Wilson (1998:129) notes "…if even a mere thousand genes out of fifty thousand to a hundred thousand in the human genome were to exist in two forms in the population, the number of genetic combinations conceivable is 10^{500}, more than all the atoms in the visible universe."

aorta. In short, the structure of the human body, including organs and tissues, is contingent on structural proteins.

In contrast to structural proteins, functional proteins are responsible for coordinating the operations and activities of the human body. One functional protein—hemoglobin—is found in red blood cells and transports oxygen throughout the body. Insulin, another functional protein, regulates the storage and metabolism of glucose. Functional proteins, such as myosin, are also found in certain human tissues and aid in the contraction of muscles. Enzymes make up a special subcategory of functional proteins and are involved in most of the metabolic and physiological functions of the human body. Through sequences of chemical reactions, enzymes regulate breathing, repair damaged muscle tissue, digest and breakdown food, and perform a host of other duties necessary to sustain life.

Proteins are complex molecules produced by linked chains of amino acids—the basic building blocks of human life. Genes code for the production of twenty different amino acids through sequential arrangements of three adjacent DNA nucleotides. For example, the nucleotide sequence of TGG synthesizes the amino acid tryptophan which, among other functions, is a precursor to the neurotransmitter, serotonin. The three adjacent nucleotides that code for the production of amino acids (in this example, TGG) are referred to as codons. Each of the twenty amino acids is produced by a unique three letter combination of the four bases (A, T, C, and G). Although some amino acids are produced by more than one codon (isoleucine, for example, is coded for by any one of three codons: ATT, ATC, and ATA), single codons do not code for more than one amino acid (e.g., TGG only codes for tryptophan). The unique sequence of DNA bases of each person results in the coding of different proteins.

Figure 2.3. The Exons and Introns of a Gene



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Recall that the main purpose of a gene is to code for the production of proteins. Only a small percentage of the entire human genome (approximately 10 percent), however, actually regulates the synthesis of proteins. The nucleotide sequences of a gene that code for protein production are known as exons. Regions of the gene that are not implicated in the formation of proteins are known as introns. Figure 2.3 depicts the intermittent assemblage of introns and exons on a single gene. Interestingly, the average gene contains nearly 3,000 base pairs, but only about 1,200 actually code for protein production.

As discussed above, genes code for the synthesis of proteins. Genes do not, however, manufacture proteins; they only provide the *instructions* necessary for the creation of a protein. The process by which genes ultimately create proteins has become known as the "central dogma" of molecular biology. The central dogma of biology is made up of two steps—transcription and translation—that are ultimately responsible for converting the genetic code (i.e., DNA) into proteins. In transcription, a segment of DNA (i.e., a gene) duplicates[3] itself onto a new molecule called nuclear ribonucleic acid (nRNA). The new molecule—RNA—contains only the DNA base sequences that correspond to one gene; it does not contain the entire nucleotide arrangement found on a polynucleotide. The bases found on RNA code for the production of amino acids that will synthesize the protein specified by a given gene.

RNA differs from DNA in three important ways. First, immediately after nRNA is created, the noncoding regions of DNA (introns) are deleted, leaving only the important protein-coding sequences of base pairs (exons). This "pruning" of introns is referred to as splicing, and splicing transforms nRNA into messenger RNA (mRNA).[4] Second, as shown in Figure 2.4,

[3] Because of splicing (see footnote 4), the RNA code does not correspond exactly to the DNA code.
[4] Ridley (2003) presents evidence showing one gene may actually code for more than one protein because of complex splicing schemes that are not wholly understood. Until recently most researchers thought splicing was a relatively simple occurrence (as described above). However, during the past thirty years or so, some researchers

RNA is not in the shape of a double helix, but rather is only a single strand of nucleotides. Third, RNA uses the nucleotide uracil (U) instead of thymine (T) in its genetic alphabet. RNA translates the DNA code (A, C, T, and G) into a new corresponding sequence of bases using A, C, U, and G. After DNA has been replicated on the RNA molecule, RNA then travels outside of the cell nucleus and into the cytoplasm where it will eventually transport the instructions needed for a ribosome to synthesize the appropriate protein.

The second step in the "central dogma" of biology is referred to as translation. Translation occurs on ribosomes which are protein-manufacturing machines. Remember that codons (3 adjacent nucleotides) found on DNA code for amino acids, which are the subunits of proteins. DNA codons, however, do not directly communicate with ribosomes. Instead, mRNA and tRNA are used as intermediary messengers. During the process of transcription, DNA codons were translated into the new "mRNA language." These mRNA codons, each reflecting one of the twenty different amino acids, are then transported to the appropriate ribosome via transfer RNA (tRNA). tRNA binds to a ribosome and the ribosome then links together chains of amino acids (polypeptides) to produce the specified protein coded for by the gene. The average protein is comprised of 1,200 chains of amino acids. Once created, the protein migrates away from the ribosome and performs its specialized function for the cell.

In summary, DNA is a four-letter alphabet code (A, C, G, and T) containing nearly 25,000 genes that perform very specific duties for the body. Each person has their own unique genetic code, and this unique genetic code brings about observable human differences, such as

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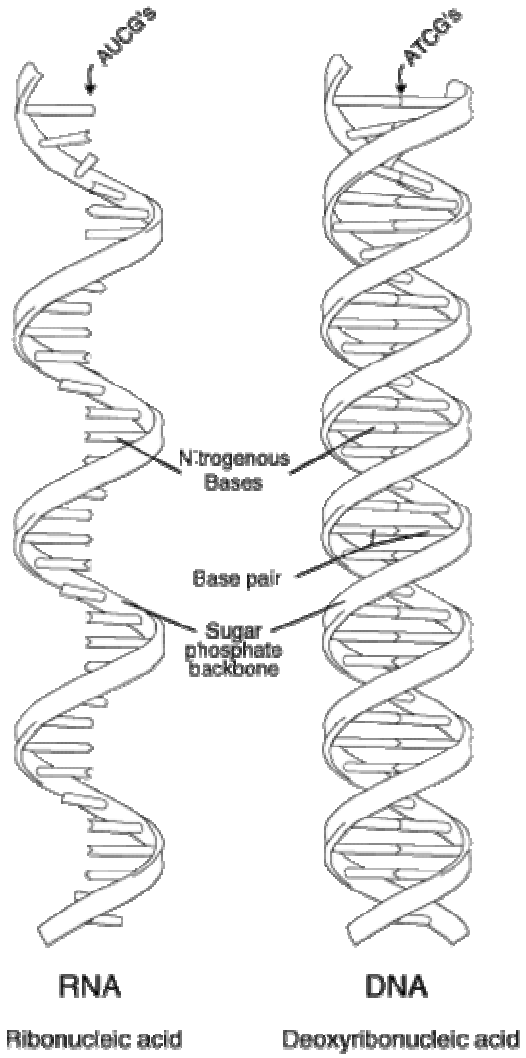
have contended that "there was more to splicing than merely cutting out the nonsense. In some genes, there are several alternative versions of each exon, lying nose to tail, and only one is chosen; the others are left out. Depending on which one is chosen, slightly different proteins can be produced from the same gene. Only in recent years, however, has the full significance of this discovery become apparent. Alternative splicing is not a rare or occasional event. It seems to occur in approximately half of all human genes; it can even involve the splicing in of exons from other genes; and in some cases it produces not just one or two variants from the same gene but hundreds or even thousands" (Ridley, 2003:141-142).

Figure 2.4. Comparison Figures of RNA and DNA



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different eye colors and various skin pigments. Genes are also responsible for coordinating the activities and functions of the human body. Genes, however, do not actually perform all of these duties—that job is accomplished by proteins. Proteins, which are manufactured through a process known as the "central dogma" of biology, are the workhorses of the human body. They give the body its structural characteristics and its form, they provide the body with energy, and they execute almost every other function necessary to sustain life. Different genes code for the production of different proteins, and these different proteins may give rise to heterogeneity in human characteristics. This variation in human traits is largely a reflection of the different DNA sequences for each person. Thus, it is critically important to understand how and why genes vary from person-to-person. The proceeding section will provide a detailed description of genetic variation.

Genetic Variation

Genes are organized on threadlike configurations called chromosomes. The human body contains twenty-three pairs of chromosomes, with one set inherited maternally and the other set inherited paternally. One pair of chromosomes—referred to as the sex chromosomes—determines whether a person is a male or a female. In general, females have two X chromosomes (one X inherited from both parents), whereas males have an X chromosome and a Y chromosome (the X is always inherited maternally and the Y paternally). The remaining twenty-two pairs of non-sex chromosomes are referred to as autosomes. The nucleus of almost every cell in the human body contains the two sex-determining chromosomes and the forty-four autosomes. Each gene is located on a specific position of a specific chromosome. Together, the

twenty-three pairs of chromosomes contain the necessary information to produce an individual's genotype.

Every person has two copies of each gene, one copy located on one of the twenty-three maternal chromosomes and one copy located on one of the twenty-three paternal chromosomes. The two copies make up the entire structure of the gene and each copy of the gene is what geneticists call an allele (i.e., 2 alleles = 1 gene). There can be any number of different alleles for each gene, with each one representing a variant found in the human population. Eye color, for example, has many different variants, including an allele for brown eyes, an allele for blue eyes, an allele for hazel eyes, and an allele for green eyes.

For the vast majority of all genes, only one known allele exists—that is, the entire human population has the same allelic combinations for these genes. This single allele makes up both copies of the gene and obviously these genes can not vary across the population. But for a small fraction of all genes, there are at least two alternative alleles that can be inherited. When there is more than one allele available in the population for a gene, the gene is called a genetic polymorphism, or polymorphism for short. More specifically, "a gene is said to be polymorphic (poly = many, morphic = forms) when the rarer allele has a frequency of 1 percent or higher, and the more common allele has a frequency of 99 percent or lower" (Rowe, 2002:94). Just because two alleles may be available for a particular gene does not necessarily mean that the gene will be formed from two different alleles. As will be outlined below, a polymorphic gene can be comprised of two similar alleles or two different alleles.

The inheritance of alleles in a polymorphic gene can be exemplified by using a simple example of a person's height. Suppose there are two different alleles for a hypothetical "height gene": a "tall" allele (T) and a "short" allele (S). Suppose further that an individual receives a T

allele from their mother and a T allele from their father. In this example, the "height gene" would be comprised of identical alleles: two T alleles. Polymorphic genes that are formed from the same alleles are referred to as homozygous genes. In this case, the height gene is homozygous because it is created by two T alleles.

However, not all genes are formed by two identical alleles. Instead, heterozygous genes are created when two different alleles are possessed by one person. The formation of heterozygous genes can be demonstrated by using the previous example of height. Suppose this time that a person inherited a T allele from their mother and an S allele from their father. The polymorphic gene would be considered heterozygous because it is comprised of two different alleles: a T allele and an S allele. Overall, then, variations in the "height gene" simply reflect the unique combination of alleles that are possible.

Genetic polymorphisms have the potential to account for variation in detectable human characteristics. For example, referring back to the previous example, the "height gene" may be one of several different genes that determine variation in human height. Everyone falls somewhere along a continuum for height, ranging from very short to very tall. People who inherit two T alleles are more likely to be closer to the very tall end of the continuum. People who inherit two S alleles are more likely to be closer to the very short end of the continuum. And people who inherit an S allele and a T allele will fall somewhere in the middle of the continuum. In this example, variation in measured height partially reflects variation in the "height gene." Importantly, genetic polymorphisms because of the different possible combinations of alleles, are the source of genetic variation and genetic variation has the capacity to account for behavioral variation.

Protein production is related to the allelic combinations found in a polymorphic gene. Some variants of a polymorphic gene may code for the synthesis of a particular protein, whereas another variant may code for the production of a different protein, and yet another variant of the gene may render the protein ineffective. The production of distinctive proteins may cause substantially divergent effects. One sequence of alleles in a certain polymorphism may maintain healthy functioning of the human body. Yet, a different allelic arrangement in this same polymorphic gene may have potentially deleterious ramifications. Certain allelic combinations may cause mental retardation, disease, and even death. Huntington's disease, for example, is caused by the inheritance of certain alleles in a single gene. Some alleles, moreover, may increase the likelihood of certain maladaptive behaviors and socially-taxing personality traits. The important point to remember, however, is that the unique combination of alleles found in polymorphic genes may code for different proteins. And "protein differences," notes Plomin (1990:17), "…can contribute to behavioral differences among individuals."

Before proceeding, it is important to make the distinction between a phenotype and a genotype. A genotype is an individual's unique combination of genes (or the sequences of nucleotides that make up genes). Genotypes differ from person-to-person because of the almost infinite number of different possible allelic combinations that are found among polymorphisms. Each person has an exclusive arrangement of allelic sequences found in polymorphisms, thus making each person's genotype different. Phenotypes are variations in observable human characteristics that are expressions of the individual's genotype. In the preceding example, the allelic sequence (e.g., S/T, T/T, S/S) of the "height gene" would be considered a part of an individual's genotype and their measured height would be the phenotype. Eye color, too, is a

phenotype because it is an observable physical characteristic that is determined by each person's unique combination of alleles that code for eye color.

The inheritance of alleles in polymorphic genes is a little more complex than the simple tall/short dichotomy in the "height" example captures. Genetic variation, for example, is often the result of a varied number of alleles available for one polymorphism. Using the height example again, perhaps the alleles available for the "height gene" are more nuanced and include the following alleles: very short (VS), short (S), average (A), tall (T), and very tall (VT). Now the potential allelic combinations for the polymorphic "height gene" have increased substantially, resulting in potentially much more genetic variation in the "height gene." Even this example is somewhat oversimplified and could be broken-down into more specific alleles (e.g., very, very short), but it does provide some insight into the creation of human genetic variation.

Three Types of Genetic Polymorphisms

Recall that genes are formed by segments of nucleotides (A, C, G, and T) working collaboratively to produce a specified protein. Each gene, moreover, is comprised of two copies of alleles, one inherited maternally and one paternally. So, for example, part of a hypothetical gene may take the following form:

Maternal allele ACTTTACTAGGAGAGTTA

Paternal allele ACTTTACTAGGAGAGTTA

As can be seen, the maternal allele and the paternal allele have the exact same sequential arrangement of nucleotides. In this example, the gene would be homozygous because the two alleles are duplicates of each other. In the population, however, there may be other variants of

the allele (i.e., a polymorphic gene) that differ by only one nucleotide. The following string of nucleotides demonstrates this slight change.

Maternal allele ACTTTACTA**G**GAGAGTTA

Paternal allele ACTTTACTA**A**GAGAGTTA

Note that in the above example seventeen of the eighteen nucleotides are identical. As shown by the underlined letters, the only difference in alleles occurs at the location of the tenth nucleotide. This alteration in a single nucleotide, while small, can result in the production of different amino acids. For instance, the GGA nucleotide sequence in the maternal allele produces the amino acid glycine. The corresponding three letter string of nucleotides on the paternal allele spells AGA instead of GGA and manufactures the amino acid arginine instead of glycine. Given that the nucleotides of the two alleles vary, this gene would be considered heterozygous. Genes comprised of alleles that differ by one nucleotide are the first type of genetic polymorphisms and are called single nucleotide polymorphisms (SNPs; pronounced "snips").

SNPs are the most common source of genetic variation, occurring in approximately 1 out of every 100 to 300 bases and accounting for nearly 90 percent of all polymorphisms. Most SNPs are relatively inconsequential and have no affect on cellular functioning (Human Genome Project Information, no date). Some SNPs, however, have strong effects on the activities of the human body. SNPs, for example, may increase susceptibility to certain diseases (e.g., Alzheimer's disease), may manufacture a nonfunctioning protein, and may impact the development of aggressive personality traits (Rujesco et al., 2003). Thus very slight changes in the arrangement of nucleotides can have sweeping consequences on the operations of the human body.

In SNPs, a single nucleotide difference in alleles (i.e., one nucleotide being replaced with another nucleotide) is responsible for the genetic polymorphism. In addition to genetic variation being caused by nucleotide differences, genes can also vary in their end-to-end length. Along various segments of genes, a small number of adjoined base pairs may be repeated a number of times. For instance, the three letter nucleotide sequence TAG_n can be repeated *n* number of times. The number of repeats varies considerably among different alleles, but the segment of DNA being repeated usually contains two (e.g., TG), three (e.g., TGA), or four base pairs (e.g., TGAG). The number of times a base pair can be repeated also depends upon the specific gene of interest. The following example illustrates the repetition of three base pairs (TTA):

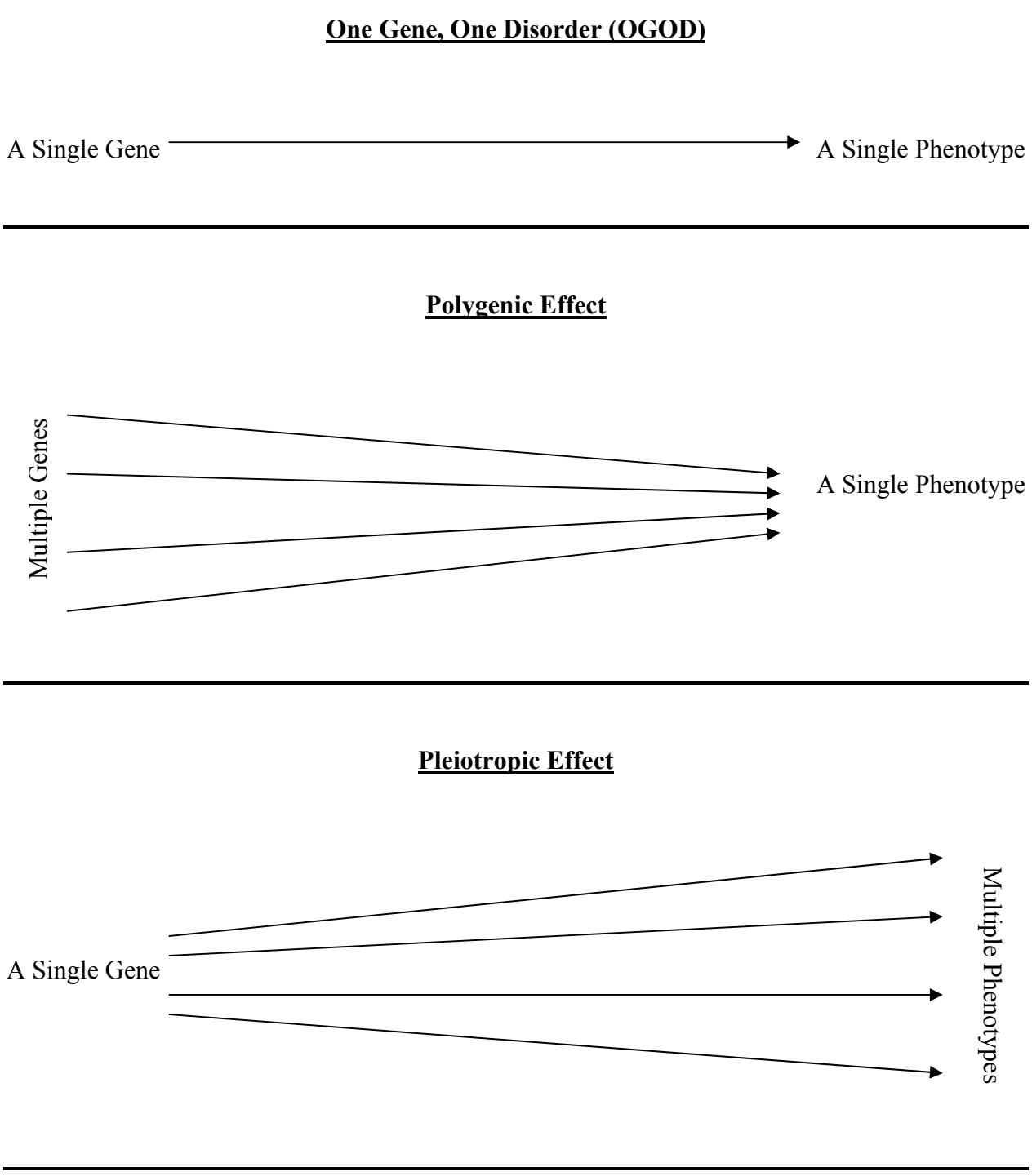
Maternal allele TAGGAA**TTATTATTATTATTA**

Paternal allele TAGGAA**TTATTATTA**

In the above example, the three base pair sequence, TTA, is repeated five times in the maternal allele and three times in the paternal allele. Genes that are comprised of alleles that differ in the repetition of a small number of base pairs are the second type of genetic polymorphisms and are known as short tandem repeats (STRs).

As noted, the number of base pairs repeated in STRs is variable, but ranges between two base pairs and ten base pairs. Sometimes, however, the base pairs involved in the repeat sequence are much longer than the ten base pair limit observed in STRs. For example, one dopamine receptor gene, DRD4, is comprised of a string of forty-eight base pairs that can be repeated over eight times. Long strings of base pairs repeated consecutively are the third and final category of genetic polymorphisms and are called variable number of tandem repeats (VNTRs) instead of STRs. The key distinguishing feature between STRs and VNTRs is the number of base pairs involved in the sequence of DNA repeated. STRs are repeat regions that

Figure 2.5. Visual Depiction of Three Ways Genes Can Directly Impact Phenotypes



contain less than ten base pairs, whereas a much larger number of base pair repeats are involved in VNTRs. Also of importance is that VNTRs are much more prevalent in the human genome than are STRs.

A Note on How Genes Influence Phenotypes

There are three main ways that genes can directly affect a phenotype. First, and as depicted on the top panel of Figure 2.5, one gene can be responsible for the development of a single disease, a single personality trait, or some other observable characteristic. Cystic fibrosis, sickle-cell anemia, Huntington's disease, and fragile-X syndrome are four of the more than 1,200 diseases that are caused by a single gene (Wilson, 1998). For single-gene diseases, people who possess a particular gene will inevitably manifest signs of the disorder. A one-to-one correspondence between a specific gene and a phenotype is referred to by the acronym OGOD (one gene, one disorder) and OGODs can be the result of either recessive (e.g., fragile-X syndrome) or dominant (e.g., achondroplasia) patterns of inheritance (Plomin, Owen, and McGuffin, 1994).

Most behavioral geneticists recognize that complex traits are unlikely to be caused by a single gene. Instead, variation in traits and behaviors is probably due, in part, to the confluence of many genes acting together.[5] When more than one gene affects the development of a trait, the

[5] It is also instructive to state that arguments and allegations of genetic determinism and eugenics are often invoked to warn of the danger of examining the genetic basis of traits and behaviors (Kaplan, 2000). Traits that are under the influence of many genes (i.e., polygenic effects), however, are somewhat insulated from such attacks. There is good reason to believe, however, that environmental correlates of offending behavior may be just as deterministic and just as immutable as genetically-based explanations of crime (Ridley, 2003). For example, as Ridley (2003) accurately points out, some of the most potent environmental influences, especially prenatal exposure to alcohol, drugs, and other neurotoxins, are irreversible. Likewise, Niehoff (1999:258) notes that "the belief that social factors, divorced from their biological impact, are the 'cause' of violence is just as misguided as the belief that violent behavior is written in the genes. Despite its good intentions, it has unwittingly hurt the people it set out to help, saddling those who already bear the brunt of economic dislocation, urban deterioration, and educational decline with the greatest responsibility for the violent behavior of an entire society. As long as violence remains a social problem rather than

trait is said to be polygenic, and polygenic effects are the second way that genes can impact a phenotype. The middle panel of Figure 5 depicts a polygenic effect. Attention deficit hyperactivity disorder (ADHD), for example, has been linked with numerous genes, such as DAT1 and DRD4, which suggests that ADHD is polygenic (Barr et al., 2000; Gill et al., 1997).

The third and final way in which genes can directly affect phenotypes is called a pleiotropic effect and is shown in the bottom panel of Figure 2.5. In this example, a single gene can have multiple effects that cut across a broad range of phenotypes. An example of a pleiotropic effect can be found with the gene that causes the potentially lethal disease, Phenylketonuria (PKU). PKU is a single-gene disorder, but the variant of the gene that causes PKU also causes a deficiency of tyronise, an increase in the amino acid phenylalanine, mental retardation, and lightening of the hair, among other visible physiological changes (Wilson, 1998).

Gene-Environment Interplay

Many diseases, certain personality traits, some behavioral patterns, and various forms of psychopathology are influenced by genetic forces. However, most phenotypes are not the result of just one gene; instead, there is good reason to believe that phenotypic variation is due to a complex and multifarious arrangement of environmental influences and genetic effects acting independently and interactively (Licinio, 2002; Plomin, Owen, and McGuffin, 1994). Indeed, most cutting-edge scientific research has moved away from the nature/nurture distinction to more detailed research designs that are able to probe the interplay between genes and the environment (Moffitt, 2005; Ridley, 2003). By gene-environment interplay, behavioral geneticists mean the

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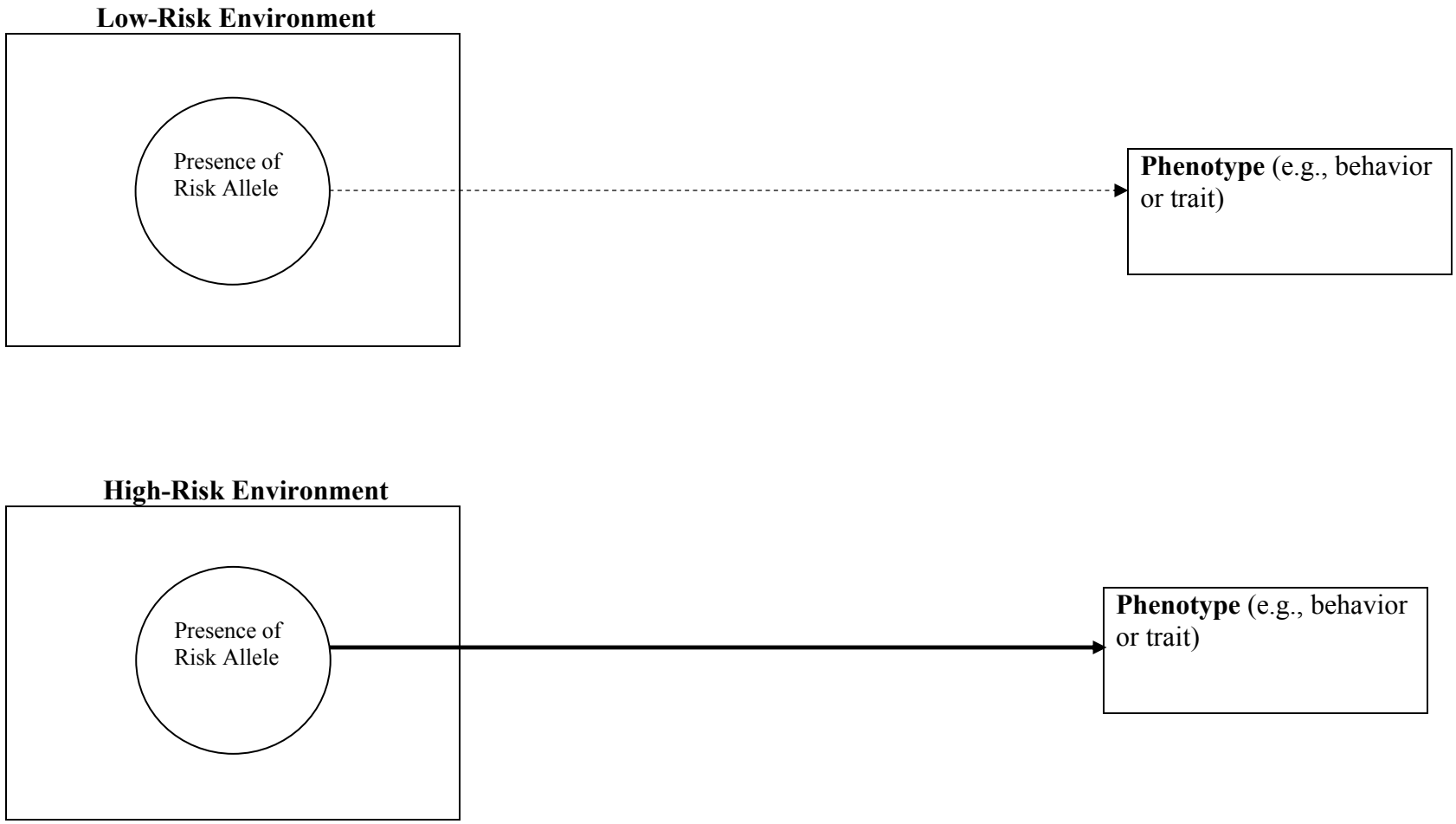
a human problem, the unscrupulous and the unjust won't need pedigrees or genetic screening to discriminate against groups of people on their 'violence potential.' All they need is an address."

ways in which genetic influences interlock with environmental forces to bring about measurable phenotypic differences. There are two overarching types of gene-environment interplay—gene X environment interactions and gene X environment correlations—both of which will be reviewed in detail below (Caspi and Moffitt, 1995; Moffitt, 2005; Rutter et al., 1997; Rutter and Silberg, 2002; Scarr and McCartney, 1983; Walsh, 2002).

Gene X Environment Interactions (GxE)

A gene X environment interaction (hereafter, GxE) can be defined as a genetic polymorphism that causes the development of a phenotype only when the person possessing the genetic polymorphism encounters, or is otherwise presented with, a certain environmental condition (Moffitt, 2005; Rutter et al., 1997; Rutter and Silberg, 2002; Walsh, 2002). In other words, the effect of the risk allele is contingent on a specific environmental influence (or vice versa); without the environmental stimulus, the effect of the genetic polymorphism would remain muted. Figure 2.6 helps flush out the conceptualization of a GxE. The rectangular boxes on the left hand side of the figure represent different environmental risk levels. The circles inside each box indicate the presence of a certain risk allele. And the rectangular boxes on the right hand side depict an undefined phenotype. In the top part of the figure, people possessing a hypothetical risk allele are embedded within a low-risk environment. The dotted line running from the risk allele in the low-risk environment to the phenotype shows that there is a nonsignificant effect of the risk allele on the phenotype. A different finding is depicted in the bottom panel, where people with the same hypothetical risk allele are this time embedded within a high-risk environment. In this case, there is a thick black line running from the risk allele in the high-risk environment to the phenotype. The thick black line indicates a significant

Figure 2.6. Hypothetical Example of a Gene X Environment Interaction



Notes: The dashed arrow indicates a non-significant relationship
The thick black line indicates a statistically significant relationship

relationship between the risk allele and the phenotype. Taken together, this example illustrates that the effect of the risk allele on the phenotype is conditioned by the type of environment in which the person lives. This example underscores the importance of examining the effect of risk alleles in different environmental conditions.

To understand more clearly the underlying logic of a gene X environment interaction, it is necessary to review the statistical difference between an additive model and a multiplicative interactive model. The following mathematical equation captures the additive effect of the predictor variables on an outcome measure:

(Equation 1) $\mathbf{Y = \alpha_0 + b_1x_1 + b_2x_2 + b_3x_3 \ldots b_nx_n + \varepsilon,}$

where Y is the phenotype of interest, α_0 is the intercept, $b_1 \ldots b_n$ are the parameter coefficients for the corresponding values of x ($x_1 \ldots x_n$), and ε is the error term. Suppose Y is a delinquency scale, b_1 is the regression coefficient for a parenting measure, b_2 is the parameter estimate for a neighborhood measure, and b_3 is the coefficient for a genetic measure. Looking at the equation, it is easy to see that each of the parameter coefficients exerts an independent (unconditional) effect on Y. Y, in other words, is a function of the linear additive effects of b_1x_1, b_2x_2, and b_3x_3. The example given with respect to equation 1 illustrates a simple additive model (also called a main effects model) usually estimated with a standard ordinary least squares (OLS) regression equation.

Additive models, while useful for some research scenarios, are unable to test the conditional effect of the environment on a certain genetic polymorphism (or vice versa). To estimate a conditional effect, a purely additive model must be abandoned in favor of an interaction model. Mathematically, the interactive model takes the following form:

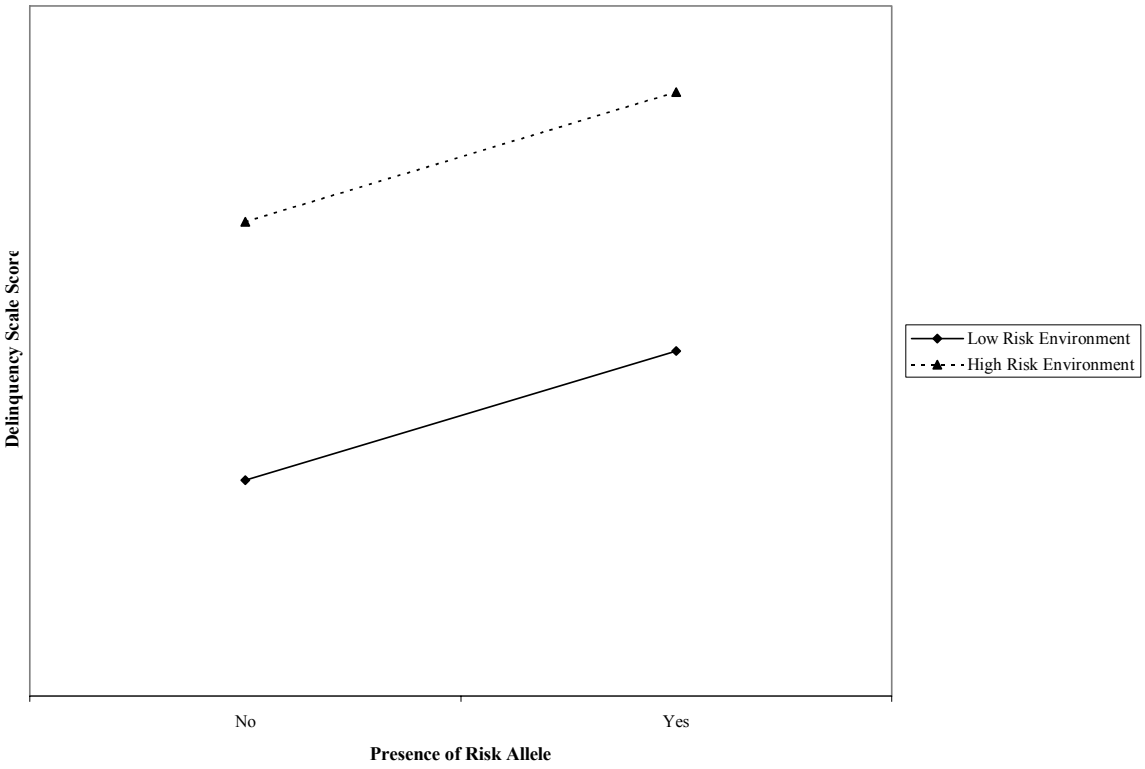
(Equation 2) $\mathbf{Y = \alpha_0 + b_1x_1 + b_2x_2 + b_3x_3 \ldots b_nx_n + (b_1x_1 * b_3x_3) + \varepsilon,}$

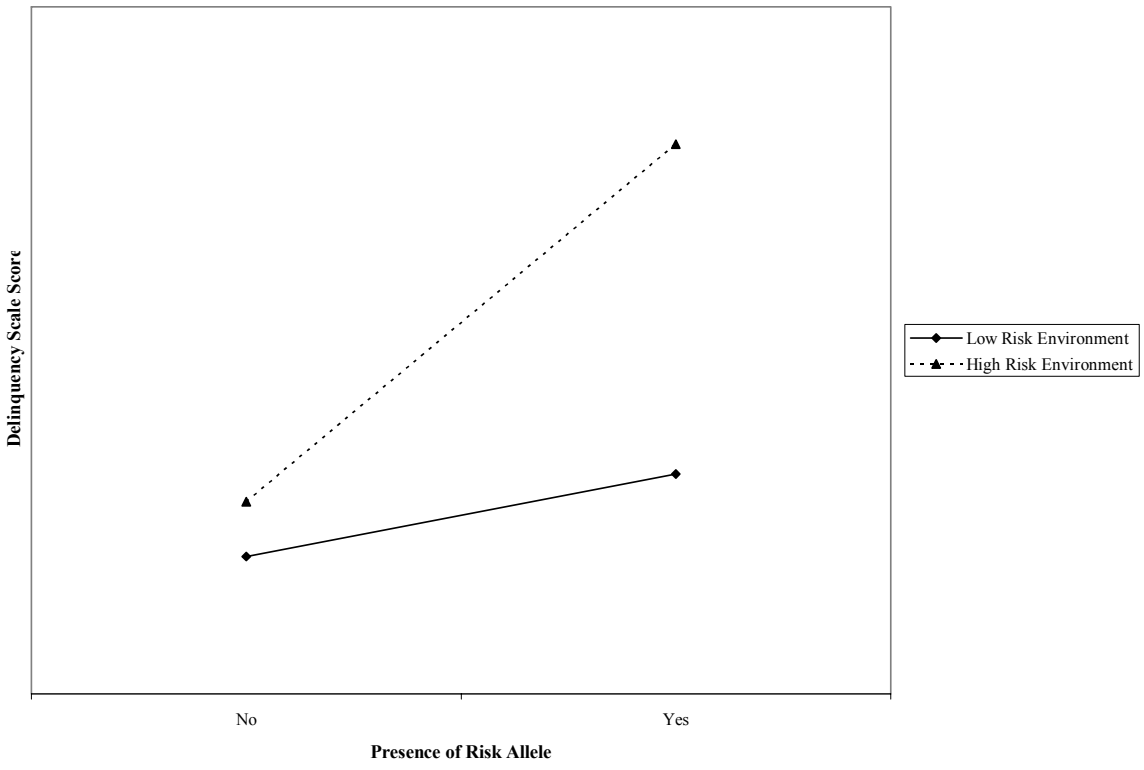
where Y is the phenotype of interest, $b_1 \ldots b_n$ are once again the parameter coefficients for the corresponding values of x ($x_1 \ldots x_n$), and ε is the residual term. Note, however, that in comparison with Equation 1, Equation 2 also includes an additional term, ($b_1x_1 * b_3x_3$), that represents the conditional effect of b_1x_1 on b_3x_3. If the previous example is used, where Y is a delinquency scale, b_1 is the regression coefficient for a parenting measure, b_2 is the parameter estimate for a neighborhood measure, and b_3 is the coefficient for a genetic measure, then $b_1 * b_3$ represents the joint effect of the parenting measure and the genetic measure. In other words, $b_1 * b_3$ is the coefficient of interest when testing for a GxE. Importantly, the interaction model also estimates the additive effects of each of the main terms prior to estimating the interaction term.

Figure 2.7 presents a graphical depiction of a hypothetical additive statistical model and a hypothetical interactive statistical model. For both models, the delinquency scale is the dependent variable and the scores for this scale are plotted along the y-axis. The delinquency scale scores are a function of the number of risk alleles (plotted on the x-axis) and two different risk environment groups: a low-risk group (depicted by a solid line) and the high-risk group (depicted as a dashed line). The top panel of Figure 2.7 contains the additive model. For both the low-risk group and the high-risk group, the delinquency scale score increases linearly and at the same rate as one moves from having zero risk alleles to having at least one risk allele. Stated differently, the risk allele measure has the same (positive) effect on both risk groups.

The bottom panel of Figure 2.7 provides a graphical representation of a GxE. As can be seen, the effect of possessing the risk allele is much stronger (as evidenced by the steep slope for the dashed line) for individuals characterized as residing in high-risk environments. The effect of the risk allele on low-risk people, however, is much weaker (as evidenced by the

Figure 2.7. Graphical Depiction of the Difference between an Additive and an Interactive Effect





comparatively flat slope for the solid line). The effect of the risk allele, in short, is contingent on risk level. Since the risk allele measure exerts a more powerful effect on the high-risk group when compared to the low-risk group, this finding would be considered evidence of a GxE.

Although GxEs can be estimated and measured using a number of different statistical techniques, the most common method is by using a multiplicative interaction term in some type of multivariate analysis (Moffitt, Caspi, and Rutter, 2005; Rutter, 1983; van den Oord and Snieder, 2002). However, some behavioral geneticists are hesitant to equate GxEs with statistical interactions (Rutter, 1983, 2006; Rutter and Pickles, 1991; Rutter and Silberg, 2002). Part of the reason for the objection to using interaction terms to measure GxEs is that statistical interactions are inherently difficult to detect (McClelland and Judd, 1993). For example, as was shown in Equation 2, the main effects of each variable are allowed to absorb or predict variation in Y prior to estimating the interactive effective, thereby restricting the amount of variation that is left to explain (Rutter and Silberg, 2002). The problem of estimating interactions is compounded by the fact that the main effect terms are often transformed or otherwise subjected to scaling variations (e.g., mean centering) prior to creating the interaction term to reduce problems with collinearity. Statistical interactions are very sensitive to such data transformations, making it difficult to observe a GxE (Rutter and Silberg, 2002). Rutter and Silberg (2002:466) also note that "the statistical power for detecting GxE is much less than that for detecting main effects." As a result, much larger sample sizes are needed to observe an interaction than are needed to observe a significant main effect (Rutter and Silberg, 2002). Despite these reservations, the extant literature has overwhelmingly used interaction terms when probing the close interplay between genes and the environment (Beaver and Wright, 2005; Caspi et al., 2002a; Foley et al., 2004; Haberstick et al. 2005).

GxEs are grounded in empirical research revealing that personality traits, temperament, and other individual differences affect the way in which people filter information, process social cues, and respond to environmental stimuli (Caspi and Moffitt, 1995; Dodge, 1986; Dodge and Coie, 1987). Two people embedded in the exact same environment may experience it and react to it in very divergent ways because of their different genotypes. Take, for example, two teenage boys walking toward each other on a street. One youth is relatively docile, passive, and level-headed. The other teenager is aggressive and has an explosive temper. As they pass each other, they barely rub shoulders; a relatively innocuous and quite frequent occurrence. The docile teenager thinks nothing of the event and continues walking down the street. The other adolescent—the one with the aggressive personality—immediately approaches the other youth, pushes him down, and begins to kick him violently. These very disparate reactions to the same event underscore GxEs.

GxEs can also potentially explain why shared environmental influences (e.g., the effects of the family) have relatively little effect on the development of personality and later life outcomes (Wright and Beaver, 2005). As Turkheimer and Waldon (2000) point out, shared environments and shared events may be experienced quite differently depending on the person's age, the person's genetic make up, and other qualities that vary between people. For instance, divorce may impact siblings differently. One child may become withdrawn, while the other child remains relatively resilient and manifests no signs of being affected by the divorce. These divergent outcomes, once again, may simply reflect the fact that siblings have different genotypes—genotypes that differentially impact reactions to the same environment or event.

Empirical Evidence of Gene X Environment Interactions

A rapidly growing body of empirical evidence has demonstrated the importance of GxEs in the development of mental illnesses, alcoholism, and other pathological diseases (Caspi et al., 2003; Caspi et al., 2005; Eley et al., 2004; Heath and Nelson, 2002; Kaufman et al., 2004). Only a handful of studies, however, have examined GxEs as they relate to antisocial behavior. Of these studies, only two have examined directly GxEs by including a *measured* genotype and a measured environmental condition (Caspi et al., 2002a; Haberstick et al., 2005). The overarching reason for the paucity of GxE research investigating the origins of crime is the lack of available data that includes measures of DNA markers. Researchers have thus been forced to search for innovative ways to test for GxEs indirectly. From this work, research has provided circumstantial evidence of GxEs by using proxy indicators for genetic risk. The following sections will review the studies that indirectly test for GxEs and the studies that directly test for GxEs in the etiology of crime, aggression, and delinquency.

Indirect Evidence of GxEs. The earliest studies that (indirectly) examined whether GxEs were related to antisocial behavior employed adoption-based research designs. Adoption samples allow for researchers to examine whether the adoptee more closely resembles their biological parent(s) or their adoptive parent(s) in terms of offending behaviors. If the adoptee is more similar to their biological parents than to their adoptive parents, then genetic factors are thought to be the dominant force. If the reverse is true, and the adoptee resembles their adoptive parents more than their biological parents, then environmental forces would be considered the prominent influence. By comparing patterns of resemblance between the adoptee and their biological parents and their adoptive parents, indirect evidence of GxEs can also be garnered (Raine, 2002b).

Table 2.1. The Proportion of Adoptees Who Have Been Convicted of a Felony by the Criminal Status of Their Adoptive Parents and Their Biological Parents

		Do either of the biological parents have a criminal record?	
		Yes	No
Do either of the adoptive parents have a criminal record?	Yes	24%	8%
	No	13%	2%

Note: Hypothetical scenario

An example of how a GxE can be inferred from adoption-based research designs is shown in Table 2.1. This table presents the results of a hypothetical distribution of the proportion of adoptees who have been convicted of a felony. The columns indicate whether one of the adoptee's biological parents have a criminal record and the rows indicate whether one of the adoptee's adoptive parents have a criminal record. The percentages inside of the quadrants reveal the proportion of adoptees who have been convicted of a felony for each possible combination of columns and rows. As revealed in Table 2.1, adoptees that neither have a criminal biological parent nor a criminal adoptive parent have the lowest odds of being convicted of a felony (lower right quadrant). Moreover, only 8 percent of adoptees with a criminal adoptive parent but a noncriminal biological parent have been convicted of a felony. Because it is assumed that the adoptee does not have a genetic risk factor (because their parents are crime free) this quadrant (biological parent is not a criminal; adoptive parent is a criminal) is of particular interest when examining the environmental effect on offending behaviors. Table 2.1 also shows that 13 percent of adoptees with a criminal biological parent but without a criminal adoptive parent have been convicted of a felony. This quadrant of the table is of interest when examining the genetic basis to criminal activity. In this case, the adoptee presumably does not live in a criminogenic environment, but does have a genetic risk factor. When comparing the quadrants thus far, the hypothetical data reveal that genetic factors are slightly more important than environmental influences in the etiology of criminal behavior.

Most importantly, however, is the upper left quadrant in Table 2.1 showing that adoptees with both a criminal biological parent and a criminal adoptive parent have the greatest likelihood of being convicted of a felony. In the adoption research designs, having a biological criminal parent is equated with a genetic risk; having an adoptive criminal parents is interpreted as an

environmental risk. So, in the example presented in Table 2.1, those adoptees with a combination of both a genetic risk and an environmental risk are at the greatest risk for becoming criminal, which researchers have interpreted as indirect evidence of a GxE.

Crowe (1974) conducted the first study revealing support in favor of the role GxEs play in the development of antisocial personalities. The sample consisted of fifty-two adopted offspring (n=27 males, n=25 females) born to forty-one incarcerated female offenders. A control group of adopted children, matched on age, sex, race, and age, were also included in the sample for comparison purposes. When they were adults, forty-six probands and forty-six controls were re-interviewed and subjected to a battery of tests assessing their mental health status, criminal history, and antisocial personality. The central outcome measure, at least as it applied to GxEs, was antisocial personality. Antisocial personality was measured by allowing three judges to interview and screen each study participant for symptoms of antisocial personality (details about the symptoms were not provided). Each judge then made a recommendation as to whether the study member suffered from having antisocial personality. The results revealed that none of the control group members were diagnosed with antisocial personality, but thirteen of the probands were judged to have antisocial personality. Crowe concluded that this pattern of results revealed that genetic factors were implicated in the etiology of antisocial personality.

To determine how, and in what way, genetic factors interact with environmental influences, Crowe also measured the length of time spent in temporary custody (e.g., orphanages and foster homes) prior to their final adoption. The amount of time in temporary custody was considered to be an indicator of adverse environmental conditions. Crowe's data revealed a marginally significant GxE where probands who had lived in temporary custody for a longer period of time were more likely to be diagnosed with antisocial personality. This early adoption

study set the stage for future research to use similar research designs to investigate the relation between antisocial behavior and GxEs.

Perhaps the most well-known adoption-based research design that examined the genetic and environmental bases to criminal convictions was conducted by Mednick, Gabrielli, and Hutchings (1984). They used a very large sample (N=14,427) of Denmark children who were adopted between 1927 and 1947. To measure criminal involvement, court conviction information was obtained for the biological parents, adoptive parents, and the adoptee. If either of the biological parents had a criminal record, then Mednick et al. considered this a genetic risk factor. If either of the adoptive parents had a criminal record, then Mednick et al. considered the adoptee to have an environmental risk to criminal behavior. A statistical procedure similar to the one detailed in Table 2.1 (see above discussion) was used to examine the genetic and environmental contributors to criminal behavior. The results revealed that only 13.5 percent of adoptive sons were convicted of a criminal offense if they had neither a biological parent nor an adoptive parent who was convicted of a crime. If one of the adoptive parents had been convicted but none of the biological parents, then 14.7 percent of adoptive sons were convicted—a slight increase due to the environment. If one of the biological parents had been convicted of a criminal offense (but the adoptive parents were crime-free,) then 20 percent of sons had a criminal history—an increase due to genetic factors. Finally, if the adoptive parents and the biological parents had criminal convictions, then 24.5 percent of adoptive sons were convicted. Clearly, then, adoptees who had both an environmental risk (adoptive parent convicted) and a genetic risk (biological parent convicted) had the greatest chance of being convicted of a crime—evidence supporting GxEs in the etiology of crime (see also Hutchings and Mednick, 1975).

Researchers have moved away from relying solely on the adoption-based research design and have developed new ways of indirectly examining whether there is a link between GxEs and crime/delinquency. Cadoret, Cain, and Crowe (1983), for example, used ordinary least squares regression to estimate the independent and interactive effects of environmental and genetic measures on misconduct. Three different samples were included in their study. The first sample consisted of N=367 adoptees from Des Moines, Iowa (referred to as Iowa 1980). The biological parents of these adoptees had histories of alcoholism, mental retardation, and antisocial behavior. All adopted children were separated at birth and did not have any future contact with their biological parents. The second sample, Iowa 1974 study, included a sample of 75 adoptive children whose biological mothers were incarcerated offenders (a control group was also embedded in this sample; details about sample size were not provided). The final sample, the Missouri sample, consisted of 108 adoptees born to parents with a variety of psychopathological symptoms (details were not provided). A control group was also included in this sample (details were not provided).

The dependent variable for all three data sets was an adolescent antisocial behaviors scale that included questions pertaining to truancy, trouble with the law, and lying. The reporting source for this scale was the adoptive parents for the Iowa 1980 sample, the adult adoptee for the Iowa 1974 sample (retrospective account), and the adoptive parents for the Missouri sample. Although the reporting source varied across the three data sets, the items comprising the scales were the same.

Two different groups of independent variables were included in the analysis: environmental measures and genetic variables. For Iowa 1980, age adopted and an adverse adoptive-home environment were included as independent variables in the analysis. For Iowa

1974 and for Missouri, an adverse adoptive-home environment was the main environmental independent variable. The genetic variables were created by obtaining information about the adoptee's biological parents. Since the Iowa 1974 data set was constructed by interviewing adoptees' whose mothers were incarcerated, they were all considered as having a genetic predisposition to engage in crime; the control group members were not coded as being genetically at-risk for antisocial behaviors. In Iowa 1980 and Missouri samples, information about the biological parents' antisocial behaviors and alcoholism were used as proxies for genetic risk.

OLS models were calculated separately for each of the three samples. The main effects of the environmental measures and genetic variables were included as well as a multiplicative interaction term created by multiplying the environmental measures by the genetic variable (i.e., a GxE). The results revealed three broad findings. First, the main effect of the genetic measure was statistically significant only for the Iowa 1980 sample. Second, the environmental measures reached statistical significance for all three of the samples. Finally, and of most importance, the GxE interaction coefficient was significant for the Iowa 1980 sample (b=2.20, $P<.0001$), the Iowa 1974 sample (b=2.51, $P<.05$), and the Missouri sample (b=.10, $P<.05$). These results revealed a strong and robust GxE effect for antisocial behaviors in three samples of adopted children.

Similar results revealing the importance of GxEs in the study of crime and misconduct were gleaned in another adoption-based study conducted by Cadoret and his colleagues (1995). The sample consisted of adoptees whose biological parents had a history of alcohol abuse/dependence or an antisocial personality. This group was considered to have a genetic or biological predisposition to engage in antisocial acts. A control group of adoptees whose parents

were relatively crime-free were also included for comparison purposes. This group was viewed as not having a genetic/biological vulnerability to criminal conduct. Information about the adoptee's home environment was obtained from parental interviews and adoptee interviews. According to Cadoret et al. (2005:918), the

> "adverse adoptive home environment factor was equivalent to the total number of the following conditions that were met: presence of marital problems in adoptive parents; divorce or separation of adoptive parents; alcohol or other drug abuse and/or dependence in a parent; depression in a parent; anxiety condition in a parent (e.g., panic disorder and generalized anxiety disorder); other psychopathologic condition in a parent (e.g., conduct disorder and somatization); and legal problems in a parent."

GxE interaction terms were created by multiplying the adverse home environment scale by whether the biological parent was an alcoholic (yes/no) and by whether the biological parent had been diagnosed with antisocial personality disorder (yes/no). These interaction terms were considered proxy indicators of GxEs.

Four different outcome measures were used to determine the role of GxEs in the development of aggression. First, a childhood aggression scale was constructed by summing 16 items (reported on retrospectively by the parent) about the adoptee's aggressive behaviors in preschool and grade school. Second, adolescent aggressivity was a retrospective scale indexing the aggressiveness of the adoptee during adolescence. Third, conduct disorder was measured retrospectively with responses to a set of questions adopted from DSM criteria. Fourth, items pertaining to an adult diagnosis of antisocial personality disorder were used to construct an adult antisocial behavior scale.

OLS regression models were then calculated to examine the effects of the independent variables (including GxEs) on the four dependent variables. The results of the multivariate analyses revealed that the biological predisposition measures and the adverse home environment scale had significant main effects on all four outcome measures. The GxE measures also exerted

comparatively strong and consistent effects on childhood aggressivity, on adolescent aggressivity, and on conduct disorder. In this study, GxEs were shown to influence early childhood and adolescent risk of antisocial conduct.

Jaffee and her colleagues (2005) also employed an innovative research design to examine the interaction between genetic vulnerabilities and physical maltreatment on conduct problems. Unlike the early studies that used samples of adoptive children to test for GxEs, Jaffee et al. used the Environmental Risk (E-Risk) Longitudinal Twin Study. The E-Risk Study is a longitudinal sample of 1,116 families with twin children born in England and Wales in 1994 and 1995 (two consecutive birth cohorts). The families were interviewed when the twin children were five years old and two years later when the children were seven years old. To assess physical maltreatment, mothers completed an in interview protocol from the Multisite Child Development project. Children's conduct problems were measured with maternal and teacher responses to items from the Achenbach instrument and with additional items designed to approximate conduct and oppositional defiant disorder as defined by DSM-IV criteria. From these items, a dichotomous measure of conduct disorder was created. Children were categorized as conduct disordered if their mothers or their teachers indicated that the child displayed three more symptoms of conduct disorder; children scoring below three on the checklist were considered not to have conduct disorder.

The unique aspect of their research, however, was the way in which they measured genetic risk. One twin from each twin pair was selected as the target twin and their sibling was included as the co-twin. Then, each co-twin's score on the dichotomous measure of conduct disorder was determined. A continuum of genetic risk for the target twin was then created by examining the co-twin's conduct disorder status in combination with their twin status (i.e.,

monozygotic or dizygotic). If conduct disorder is genetically influenced, then MZ twins, whose co-twin has been diagnosed with conduct disorder, will have the greatest genetic risk for also developing conduct disorder. DZ twins whose co-twin has been categorized as having conduct disorder will have a lower genetic risk for also being characterized as having conduct disorder. DZ twins whose co-twin does not have conduct disorder will have an even lower genetic risk score. And, finally, MZ twins whose co-twin has not been designated as having conduct disorder will have the lowest genetic risk for conduct disorder. In Jaffee et al.'s analysis, genetic risk scores ranged from a low of 0 (MZ co-twins without conduct disorder) to a high of 3 (MZ co-twins with conduct disorder).

Jaffee and her associates calculated ordinary least squares regression equations with the continuous measure of conduct disorder as the dependent variable. The measure of genetic risk and the measure of physical maltreatment were included as predictor variables in the models. An interaction term was also created by multiplying the genetic risk score by the physical maltreatment variable. The results of these models revealed a significant main effect for genetic risk (β=.27) and a significant main effect for physical maltreatment (β=.15), and a significant interaction effect between these two measures (β=.11). The significant interaction term was interpreted as empirical documentation of a GxE in the etiology of conduct disorder.

Beaver and Wright (2005) also examined the effect of GxEs on adolescent delinquency. Specifically, their OLS regression models included measures of pubertal development, different temperaments, and delinquent peers as the independent variables and a delinquency scale as the dependent variable. Importantly, Beaver and Wright conceptualized the pubertal development scale as a genetic measure and the delinquent peers scale as the environmental measure. In addition to the main effects of the independent variables, they also included an interaction term

created by multiplying the pubertal development scale by the delinquent peers scale (GxE). Data for their study came from the publicly available version of the Add Health sample (N=6,504). Analyses were conducted separately for males (n=2,474) and for females (n=2,680). The results for males revealed that the pubertal development scale (β=.12) and the delinquent peers measure (β=.36) had significant independent additive effects on delinquency. However, there was also a significant effect for their proxy GxE measure: the interaction term for pubertal development X delinquent peers exerted a statistically significant effect on delinquency (β=.07). Although the main effects for pubertal development (β=.05) and for delinquent peers (β=.30) were statistically significant for females, the GxE interaction term failed to reach significance.

Lastly, Button and her colleagues (2005) examined whether family dysfunction interacted with genes in the creation of antisocial conduct. They measured conduct problems by using five items extracted from the Strengths and Difficulties Questionnaire. Family dysfunction was indexed by using twelve questions from the General Functioning subscale of the McMasters Family Assessment Device. These questions tapped two dimensions of the home life: family pathology and family health. Button et al. (2005) examined whether the measure of family dysfunction interacted with (unmeasured) genetic forces to predict conduct problems. The results of their statistical analysis supported "both a heritable component to conduct problem and a small but significant association between family dysfunction and childhood and adolescent conduct problems" (Button et al., 2005).

Direct Evidence of GxEs. In general, the results generated from studies indirectly testing for GxEs have revealed the importance of examining the interactive effects of genetic and environmental factors in the development of antisocial behaviors. While useful, these studies have been unable to identify the precise genes that may be implicated in GxEs. In the past

number of years, however, two studies have emerged that directly assess GxEs by including a measured gene and a measured environment to determine how they combine together to promote misconduct.

The first study, published in 2002 by Caspi and his associates, examined the independent and additive effects of childhood maltreatment and of monoamine oxidase A (MAOA) on violence (Caspi et al., 2002a). Most importantly, they also investigated the potential interaction between the MAOA gene and childhood maltreatment. To test for a GxE, they employed the Dunedin Longitudinal Study, a prospective study of 1,037 children born in New Zealand between April of 1972 and March of 1973. Thus far, data have been collected from the participants when they were ages 3, 5, 7, 9, 11, 13, 15, 18, 21, and 26. Remarkably, 96 percent of the original sample was contacted and re-interviewed in the latest wave of data collection. The final analytic sample used by Caspi et al. was N=442 Caucasian males.

Four different dependent variables indexing antisocial behaviors were used in their analysis. The first measure, conduct disorder, was measured by using the criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). The DSM-IV defines conduct disordered individuals as consistently engaging in behavior that violates others and that may escalate to full-blown physical aggression. Dunedin participants were assessed for conduct disorder at ages 11, 13, 15, and 18. A "lifetime diagnosis" dichotomous measure was created by summing across all of the assessment waves and placing those participants who had been categorized as conduct disordered at any of the waves into one group and placing those participants who had never been classified as conduct disordered into another group.

Secondly, court records were searched for violent convictions (e.g., domestic violence, violent assault, manslaughter, etc.) for 97 percent of the male interviewees. Overall, 11 percent

of the male sample had been convicted of a violent crime. The third dependent variable used in the analysis was a disposition towards violence scale. At age 26, study members were asked to complete the Multidimensional Personality Questionnaire (MPQ) that includes an aggression subscale. Questions such as, when I get angry, I am ready to hit someone, were included in the scale (alpha=.71).

The fourth outcome measure used was an antisocial personality disorder scale. For this scale, male study members nominated one person who knew them very well (e.g., a friend, spouse, or family member). These nominated individuals were then contacted and asked a series of questions pertaining to antisocial personality symptoms exhibited by the Dunedin participants. For example, informants were asked about the study member's anger, impulsiveness, and empathy. The responses to these items were then summed together to form an additive scale of antisocial personality symptoms (alpha=.84).

The interrelationships among these four different scales were then analyzed and results demonstrated moderate inter-scale correlations. Additional model-fitting techniques revealed that a common factor accounted for the four antisocial behavior measures. As a result a composite index was created by summing together scores for these four scales. As Caspi et al. (2002b:3) note, "this summary index counts whether they (a) met diagnostic criteria for adolescent conduct disorder, (b) were convicted for a violent crime, (c) scored in the top quartile of the distribution on a self-reported disposition toward violence, and (d) scored in the top quartile of the distribution on informant-reported antisocial personality disorder symptoms." All four of the antisocial behavior scales and the composite index were used as outcome measures in the statistical analyses.

The greatest contribution of Caspi et al.'s work was that they included a measured polymorphic gene, MAOA, in their analysis. MAOA, which has a VNTRs, was coded as a dichotomous measure, where study members were either classified as having the low functioning version (2 and 3 repeats) or as having the high functioning version (3.5 and 4 repeats) of the MAOA gene. Based on animal knock-out studies and family linkage studies, the authors hypothesized that the low functioning version of the MAOA gene was the risk polymorphism in the etiology of violence.

Physical maltreatment was measured by using behavioral observations, parental reports, and retrospective reports reported on by Dunedin participants. At the age 3 assessment, independent observers watched the mother and the child interact. The observer then rated the mother on eight different categories, such as harshness toward child and indifferent to child's performance. Observers who indicated that the mother engaged in two or more of these negative actions were characterized as rejecting their child. Second, at the age 7 and age 9 interviews, parents were presented with a checklist of disciplinary behaviors, including items that tapped physical punishment. Parents who were in the top ten percent of on this scale were coded as unusually harsh disciplinarians. Third, wave-to-wave changes in the primary caregiver were tracked. Those children who had more than two primary caregivers were classified as having suffered disruptive caregiver changes. Fourth, at the age 26 assessment, study members completed a retrospective questionnaire asking about incidents of physical abuse occurring before the age of 11. Finally, during the age 26 interviews, Dunedin participants were asked about unwanted sexual abuse. Based on this information, study members were grouped as either having been sexually abused or not having been sexually abused. A cumulative physical maltreatment scale was created by adding the number of maltreatment experiences together.

To examine the relationship between MAOA, maltreatment, and delinquency/crime, Caspi et al. (2002) first used the composite measure of antisocial behavior in a moderated regression analysis. The results revealed that MAOA did not have a significant main effect on the antisocial composite measure (b=.01, *P*=.89) but that physical maltreatment exerted a significant positive effect on antisocial behavior (b=.35, *P*=.001). Most importantly, however, was the finding that the interaction between MAOA and maltreatment was significantly predictive of the composite measure of antisocial behaviors (b=-.36, *P*=01). These findings suggested that the high-functioning MAOA allele buffered the effects of physical maltreatment on antisocial behaviors, whereas the low-functioning MAOA allele intensified the effects of maltreatment on antisocial behaviors.

GxEs were also examined for each of the separate antisocial scales. The results revealed marginally significant or significant effects of MAOA X maltreatment for the conduct disorder measure (b=-.63, *P*=.06), for violent crime conviction (b=-.83, *P*=.05), for the disposition toward violence (b=-.24, *P*=.10), and for the antisocial personality symptom scale (b=-.31, *P*=.04). The Caspi et al. study provided the first documented evidence of a measured gene interacting with an environmental condition to heighten the risk of involvement in crime, delinquency, and antisocial behaviors.

Caspi et al. (2002) concluded their *Science* article by calling for future research to replicate the MAOA X maltreatment interaction by using samples other than the Dunedin Longitudinal Study. Since that time, two studies have been published that examine whether the MAOA gene interacts with different environmental measures in the causation of antisocial problems. Foley et al. (2004) provided the first study attempting to replicate the results reported

by Caspi and his associates. Their sample consisted of 514 white males who were participants in the Virginia Study for Adolescent Behavioral Development.

Conduct disorder was measured by using diagnostic criteria outlined in the Diagnostic and Statistical Manual (DSM) manual. The child, their mother, and their father were presented with a series of symptoms that indexed antisocial behavior. If any of the respondents responded affirmatively to these items, then the child was rated as having this symptom.[6] Instead of using a measure of childhood maltreatment (Caspi et al., 2002a), Foley et al. (2004) included a scale that tapped childhood adversity. This scale indexed three different dimensions of the family environment: parental neglect, exposure to parental violence, and inconsistent parental discipline. Finally, the MAOA variable was coded as a dichotomous variable: participants with the low-activity version of the MAOA gene (2-, 3-, and 5-repeat alleles) were grouped together and participants with the high-activity allele (3.5- and 4-repeat alleles) were placed into the other category.

The results of their multivariate equations revealed that the childhood adversity measure exerted a significant main effect on conduct problems. At the same time, the MAOA variable did not maintain a statistically significant association with conduct problems. However, there was a significant interaction between childhood adversity and MAOA that was a significant predictor of childhood and adolescent conduct disorder. The findings reported by Foley et al. (2004) thus provided additional evidence supporting the role of GxEs in the etiology of antisocial behaviors.

Haberstick and his colleagues (2005) also examined possible interaction between MAOA and childhood maltreatment by using a restricted version of the Add Health data. To provide a

[6] Detailed information about the items used, or the number of symptoms needed in order to be considered conduct disordered, were not provided.

sample comparable to the one used by Caspi et al., only Caucasian males were used in the analysis; females and racial minorities were removed from the final analytic sample. A total of N=774 males were included in the statistical models.

Three different scales tapping antisocial behaviors were included as dependent variables in the analysis. First, they used four different measures to serve as a proxy for DSM-IV criteria for a diagnosis of conduct disorder. The conduct problems scale included items indexing frequency of fighting, violence, and delinquency. The conduct problems scale were calculated for each of the three waves of data and averaged together. Second, a binary measure of violent offending was also used as an outcome measure (details about the scale were not presented). They also created a composite antisocial index by including a scaled version of the conduct problems scale and items related to violent convictions.

At wave III, buccal cells were collected from a subsample of Add Health study members and genotyped for a number of different genes, including the MAOA gene. The MAOA gene was coded as a dichotomous measure: those with the 2-repeat or the 3-repeat were classified as having the low activity MAOA gene; those with the 3.5-repeat, the 4-repeat, and the 5-repeat were classified as having the high activity MAOA gene.

Childhood maltreatment was measured by using retrospective accounts, collected at wave III, of maltreatment occurring before entrance into sixth grade. Study members were presented with a list of items that indexed abuse and neglect (e.g., hit, kicked, or slapped by parents) and were asked to indicate which, if any, of the forms of abuse they had experienced. Responses to these items were then added together to form a global measure of childhood maltreatment.

Haberstick et al. (2005) calculated regression analyses to examine the additive and interactive effects of MAOA and childhood maltreatment on the conduct problems scale, on the

violent convictions index, and on the composite measure of antisocial behaviors. The results revealed that the childhood maltreatment scale had a statistically significant effect on the conduct problems scale (b=.179, *P*<.0001) and on the composite measure of antisocial behaviors (b=.195, *P*<.0001); however, it was not significantly related to the violent convictions index (b=-1.48, *P*=.979). In addition, no significant main effects for the MAOA measure were found on any of the three outcome measures. Of particular importance were the findings for the GxE term. The results of the regression models revealed that the MAOA X maltreatment coefficients were insignificant for conduct problems (.04, *P*=.333), for violent convictions (b=-1.93, *P*=.975), and for the composite measure (b=.042, *P*=.109). Although there was an insignificant trend in the predicted direction, the results of the study conducted by Haberstick et al. (2005) using the Add Health data did not confirm the GxE found by Caspi et al. (2002).

Gene X Environment Correlations (rGE)

GxEs are useful in explaining why two people, when presented with the same environment, may ultimately turn out quite differently. GxEs, however, do not provide any insight into how genotypes may be partially responsible for nudging a person into a particular environment. To understand and explain why certain genotypes are closely related to, and reinforced by, given environments another type of gene-environment interplay—gene X environment correlations—will be discussed. Gene X environment correlations (hereafter, rGE) refer to the consistent finding that a person's environment is not necessarily knifed off from their genetic makeup—that is, a person's genotype and their environment are often correlated. Genetic factors are, in other words, thought to impact the way in which people select, modify, and create their own environments (Scarr and McCartney, 1983). For example, an alcoholic may

frequent bars and other liquor establishments—environments conducive to maintaining inebriation. The question becomes, then, why do alcoholics find themselves in environments that reinforce their preference for consuming alcohol? According to the logic of rGEs, the alcoholic's genotype plays a role in "pushing" the person towards environments (e.g., bars) that allow optimal gene expression. Statistically speaking, rGEs can be detected by determining whether there is a correlation between a genetic polymorphism and a particular environment.

Thus far it may seem as if all rGEs are the same. In one sense they are: all rGEs are due to the role that genes play in structuring environments. Yet to pretend that all rGEs are the same would be a mistake. The *processes* that lead to rGEs are what makes rGEs different and are what distinguishes one type of rGE from another. In general, there are three different types of rGEs—passive rGEs, evocative rGEs, and active rGEs—each reflecting a unique way in which genes shape the environment (Rutter et al., 1997; Rutter and Silberg, 2002). The following discussion will provide details about these three rGEs.

Passive Gene X Environment Correlations. Passive rGEs build upon the fact that parents usually pass along two different elements to their children: genes and an environment. Although the environment is often assumed to be orthogonal with the child's genetically-influenced behavioral and temperamental propensities, this assumption is wrong (Harris, 1995, 1998). Instead it is probably more accurate to assume that the child's familial environment is largely a reflection of their parents' genotypes. Thus, the child receives not only half of their genes from each parent but also is born into a home environment that is largely created from their parents' genetic makeup. This type of rGE is referred to as a passive rGE because the child does not have an active voice in choosing their genotype or their familial environment—they are passively passed on from parent to offspring.

For example, intelligence is one of the most highly heritable individual characteristics (Herrnstein and Murray, 1994). Children who are born to intellectually savvy parents are likely to have high cognitive capabilities. At the same time, intelligent parents are also likely to provide an environment that stimulates their child's brain development. The child thus has a genetic predisposition to be "smart" and also lives in a home environment that promotes intelligence. Without considering the possibility that the parent's genes are partially responsible for their child's intelligence it would appear on the surface that the environment is the main cause of their child's IQ. In reality, however, the familial environment is so closely intertwined with genetic influences, that only genetically-sensitive research designs are able to parcel out the relative effects of genes and the environment.

rGEs can also be applied to antisocial behavior. Some research suggests, for example, that parental management practices are important correlates to delinquency (Loeber and Stouthamer-Loeber, 1986). Parents who abuse their children, who fail to supervise their children, and who otherwise take a lackadaisical approach to raising their children are more likely to have antisocial children. Parents who raise their children this way, however, are probably more likely to be antisocial themselves. As a result, the child not only passively receives a genetic predisposition to delinquent behavior but also is born into an environment that promotes wayward behavior. In this case, the criminogenic family environment is created by the fact that the parents are antisocial.

Evocative Gene X Environment Correlations. Evocative rGEs are the second type of rGEs and reflect the fact that people elicit certain responses from the environment based, in part, on their genotype (Caspi and Moffitt, 1995). A person with one genotype may evoke one type of response from the environment, whereas another person, with their own unique genotype, may

evoke a completely different response. For example, when an attractive woman enters a room or other social setting, she is likely to draw the attention of many people, especially men. Based on her appearance, she may even receive preferential treatment. Another woman, who is of average attractiveness, may enter the same room without much notice. The preferential treatment conferred for the attractive women may not be extended to the less striking woman. This simple example using attractiveness (a genetically-influenced feature) demonstrates how different genotypes are implicated in chiseling out very distinct environments—environments that simply reflect the person's genotype.

Moreover, family researchers have long recognized that parents treat their children very differently depending upon how their children behave (Lytton, 1990). A difficult and taxing child, for example, will likely be reprimanded, punished, and disciplined regularly by their parents. Their sibling, however, who has an easy-going personality and who is relatively obedient, will be much more enjoyable for their parents to raise and punishment will be less frequent. In this case, children, depending on their unique genotypes, evoke differential responses from their parents. These different familial environments are largely correlated with the child's genetically-influenced temperaments. Evocative rGEs can be best summarized by stating that certain genetic polymorphisms elicit particular responses from the environment, and these responses are correlated with the person's genotype.

Active Gene X Environment Correlations. Active rGEs are the third and last type of rGEs. Active rGEs are often described as "niche-picking" and refer to the observation that individuals play an active role in seeking out environments that are conducive with their genotypes (DiLalla, 2002; Scarr, 1992; Scarr and McCartney, 1983). Adolescents who are exceptionally good singers are likely to search for choirs or other vocal groups that allows them

to practice and refine their talent of singing. Even the well-known similarities that exist between spouses (i.e., assortive mating) and the similarities that exist between friends (i.e., social homophily) can also be explained as examples of an active rGE (Alvarez and Jaffe, 2004; Rushton and Bons, 2005).

In a similar vein, low self-control is a largely genetically-influenced trait that is a robust predictor of delinquent behavior (Pratt and Cullen, 2000; Wright and Beaver, 2005). Youths with low levels of self-control are predisposed to engage in delinquent activities. The same genetic and biological factors that are related to the development of self-control may also pressure wayward youths to search for other peers who lack self-control to befriend (Beaver, Wright, and DeLisi, 2006). This example highlights active rGEs and shows how a genetic or biological susceptibility to antisocial behavior (e.g., low self-control) may propel an individual to actively seek out criminogenic social factors, such as delinquent friendship networks (Beaver and Wright, 2005).

Empirical Evidence of Gene X Environment Correlations

As revealed above, rGEs capture the genetic underpinnings to environments and/or how genetic polymorphisms can influence environments. Although relatively little empirical research has explored how rGEs may be related to misconduct, the importance of rGEs should not be casually glossed over. "These correlations [gene-environment correlations]," contends DiLalla (2002:598) "probably occur with most of the behaviors that we study, but they are extremely difficult to measure." The difficulty in measurement and the lack of available data probably are the overriding reasons for the scant research examining rGEs. No studies, for example, exist that directly examine the way in which a *measured* genetic polymorphism may correlate with the

environment. There are, however, a handful of studies that use somewhat disparate research designs to examine genetic components to different environments. Each of these studies will be discussed momentarily.

Before proceeding, it is important to first describe biometric model-fitting techniques used in behavioral genetic research. Some of the research that examines rGEs does so indirectly by estimating the proportion of variance in an environmental measure that is due to genetic factors. If an environmental measure is found to be genetically-influenced, then this finding is often offered as evidence of rGEs.[7] Most of this work uses twin samples that include both MZ twins and DZ twins to isolate the effects of genetic factors and environmental factors on a given phenotype. In particular, biometric model-fitting's major assumption is that most phenotypes are created from environmental and genetic factors. By comparing the resemblance of twins within MZ twin pairs to the resemblance of twins within DZ twin pairs, a fairly accurate estimate of heritability can be calculated. Specifically, if the environment is comparatively more important in explaining a given phenotype, then twins within each DZ twin pair should resemble each other as much as twins within each MZ twin pair. Extending this logic a step further, if genetic factors are important in explaining a given phenotype, then twins within each MZ twin pair should be more alike than twins within each DZ twin pair. Thus the estimation of different variance components to a phenotype entails comparing MZ twin pairs to DZ twin pairs.

Behavioral genetic research partitions the variance in a given trait or a given behavior into three distinct components: 1) a heritability component, 2) a shared environmental

[7] While useful, traditional model-fitting techniques do not tell researchers what specific genetic factors are implicated in the rGE. Take a hypothetical scenario where h^2=.50 and e^2=.50 (for simplicity, I have assumed that c^2=.00) are estimated for a measure of family dynamics. This information clearly shows that both environmental and genetic factors are important contributors to the dynamics of the family. Yet, unanswered questions still remain such as, what genetic factors are important? To answer this question we must move inside of the "black box" of heritability estimates and use a different analytical strategy—one that includes a measured genetic polymorphism as a predictor/correlate of an environmental measure.

component, and 3) a nonshared environmental component. Heritability (h^2) refers to the proportion of variance in a behavior or trait that is due to genetic factors. The shared environment (c^2) captures all of the environmental influences that are the same between siblings. For example, neighborhood structural characteristics are identical for all siblings residing in one household. Finally, the nonshared environment (e^2) is comprised of all the social factors that vary between siblings (plus measurement error). Different peer groups for two siblings exemplify the nature of nonshared environmental influences.

To explain more clearly how estimates for h^2, c^2, and e^2 are formulated, a brief description of twin correlations will be presented. The correlation between twins within each twin pairs is thought to reflect both environmental and genetic influences. Mathematically,

(Equation 3) $\mathbf{r_{MZ} = h^2 + c^2,}$

where r_{MZ} designates the cross-twin correlations of a specified phenotype for MZ twins, h^2 represents the genetic influences, and c^2 symbolizes the environmental factors. The correlation for DZ twins is slightly different and takes the following form:

(Equation 4) $\mathbf{r_{DZ} = (1/2)h^2 + c^2,}$

where r_{DZ} is the cross-twin correlations of a specified phenotype for DZ twins, h^2 once again represents the genetic influences, and c^2 symbolizes the environmental factors. Note, however, that the only difference between Equation 3 and Equation 4 is the (1/2) before h^2 in Equation 4. The reason h^2 is multiplied by 1/2 for DZ twins (Equation 4) and not for MZ twins (Equation 3) is because DZ twins share approximately 1/2 of their genes, whereas identical twins share all of their genes.

Now that the equations describing MZ twins and DZ twins have been presented, it is possible to employ some basic algebraic properties to gain an estimate of h^2. An approximate

estimate of heritability can be garnered by first subtracting Equation 4 from Equation 3, as shown in the following equation:

(Equation 5) $\mathbf{r_{MZ} - r_{DZ}}$

However, Equation 5 only provides information regarding the absolute difference between MZ twins and DZ twins. As shown above in Equations 3 and 4, MZ twins share 100 percent of their DNA, while DZ twins share only 50 percent of their genes. To take this difference in genetic similarity into account, the difference found in Equation 5 must be doubled, yielding the final equation needed to gain an estimate of heritability.

(Equation 6) $\mathbf{h^2 = 2(r_{MZ} - r_{DZ})}$

These equations can easily be extended to calculate the proportion of variance in a phenotype that is accounted for by the shared environment (c^2).

(Equation 7) $\mathbf{c^2 = 2r_{DZ} - r_{MZ}}$

Equation 7 presents the formula needed to calculate an estimate of c^2. The correlation for DZ twins (r_{DZ}) in Equation 7 is doubled to standardize for the proportion of variance accounted for be genetic factors. Finally, to calculate an estimate of the proportion of variance that is due to the nonshared environment, e^2 needs to be solved for. This can be accomplished as shown in the following equation:

(Equation 8) $\mathbf{e^2 = 1 - (h^2 + c^2)}$

Remember, variation in any phenotype is usually assumed to be the additive result of h^2, c^2, and e^2. Once h^2 and c^2 have been estimated, these two values can be added together and the resulting product then subtracted from 1. The remaining value is the estimate for e^2.

These same variance decomposition equations can be used to estimate the heritability of environmental measures. Some of the research examining rGEs (see below) uses traditional

biometric model-fitting techniques (e.g., Iervolino et al., 2002). When a significant proportion of the variance in a given environmental measure is influenced by genetic factors (i.e., h^2) then evidence of an rGE is usually inferred (see, for example, Cleveland, Wiebe, and Rowe, 2005).

Indirect Evidence of Gene X Environment Correlations. Given the strong connection between delinquency and antisocial peer groups, behavioral genetic researchers have been interested in determining whether the formation of delinquent peer groups is due to genetic forces (i.e., rGE). Iervolino and colleagues (2002), for example, used two genetically-sensitive data sets to examine the environmental and genetic influences on adolescent peer group socialization. The first sample, the Nonshared Environment in Adolescent Development (NEAD) study, was comprised of siblings from 395 families. The second sample consisted of participants in the Colorado Adoption Project (CAP). The CAP included 81 adoptive sibling pairs and 99 nonadoptive sibling pairs. Iervolino et al. (2002) measured a number of dimensions of peer-group preference (e.g., peer college orientation, peer popularity), but only one—peer delinquency—will be the focus of the current review. For both samples, peer delinquency was measured with self-reported questionnaires that indexed the friends' rebelliousness, drug-taking behavior, and unconformity.

Traditional model-fitting statistical techniques were used to decompose the proportion of variance in peer delinquency that was accounted for by genetic factors, by the nonshared environment, and by the shared environment. The models were calculated separately for both samples. For the NEAD sample, the results revealed that genetic factors accounted for virtually none of the variance in peer delinquency (3 percent). The shared environment and nonshared environment, however, accounted for 20 percent and 77 percent of the variance in peer delinquency, respectively. Very different results were gleaned for the CAP sample. Genetic

factors accounted for 65 percent of the variance in peer delinquency, whereas the nonshared environment explained 35 percent of the variance; the shared environment had no effect on peer delinquency. The results from these two samples produced divergent results and thus point to the need for additional research to determine the importance of genetic factors in the formation of antisocial peer groups.

A similar research question was posed by Cleveland, Wiebe, and Rowe (2005) when they sought to uncover the genetic and environmental sources to substance-abusing friends. Their study used a restricted data file of the Add Health study that consisted of sibling-pairs of different genetic relatedness (analytic sample N=1,036 sibling pairs). The substance-abusing peers scale was comprised of two different items. First, respondents were asked how often, within the past 12 months, they smoked cigarettes. Second, study members were asked how often, within the past 12 months did they drink beer wine, or liquor. The responses to these two questions were then added together to form a composite measure of licit substance use.

During wave I interviews, Add Health participants were asked to provide the names of their male and female friends (who were also included in the Add Health study). Based on these peer nominations Cleveland et al. linked each of the Add Health participants with their friend's data file and merged the cases together. The friends' scores on the licit substance use scales were then summed and an average peer licit substance use scale was created. The peer drug use scale was then used as the main variable of interest in the study.

To determine the genetic and environmental influences on peer drug use, biometric model-fitting techniques were employed. Based on these models, Cleveland and his colleagues (2005:164) "found strong support for genetic influences on adolescents' exposure to friends' substance use, but no support for the social influence of families when we used a behavioral-

genetic design. In the best fitting model, we estimated the parameter for shared environment to be zero. We divided the total variance between the genetic (64%) and the noshared environmental (36%) factors." The fact that genetic factors strongly influenced peer group formation reveals evidence supporting the role of rGEs in the etiology of antisocial behaviors.

The genetic influences on peer group selection can be viewed as an active rGE (see discussion above). Evocative rGEs have, on the other hand, primarily been investigated by looking at children's behaviors, determining whether these behaviors are genetically influenced, and then examining and how they elicit certain responses from their parents. In the first study to assess empirically evocative rGEs, Ge et al. (1996) used an adoption-based research design. The sample consisted of 25 male and 20 female adoptees between the ages of 12 and 18. Genetic risk was determined by tracking down the hospital and prison records of the adoptees' biological mother and biological father. Adoptees were assigned a genetic risk score based on whether their biological parents had a history of antisocial personality disorder, substance abuse, substance dependency, or both abuse and dependency. Measures were also included that indexed the adoptee's antisocial/hostile behaviors, the adoptive fathers' and mothers' nurturant/involved parenting, the adoptive fathers' and mothers' harsh/inconsistent parenting, the adoptive fathers' and mothers' hostility, and the adoptive fathers' and mothers' warmth.

The study by Ge et al. found that adoptee's who had a biological parent with a history of antisocial behaviors scored significantly higher on the aggressive/hostile scales—thus, acting aggressively was transmitted genetically from biological parent to adopted offspring. Moreover, and of particular importance, was the finding that the adoptees' biological parents' psychiatric status was a robust predictor of the adoptive parent's behaviors. Having a biological parent with a history of substance abuse/dependency or with a history of antisocial behavior generated more

harsh/inconsistent parenting, less parental nurturance, and less parental warmth. These differential parental reactions, however, were largely mediated by the adoptee's own antisocial/hostile conduct—initial evidence of an evocative rGE. In this case, children elicited certain responses from their parents based in large part on how aggressive/hostile they acted (which was shown to be at least partially genetically transmitted).

In another study exploring evocative rGEs, O'Connor et al. (1998) also used an adoption-based research design to examine the nexus between childhood and adolescent misconduct and measures of parenting behaviors. The sample consisted of adopted children and adolescent study members of the Colorado Adoption Project (CAP). The CAP included 88 adolescents who were classified as either having a genetic risk or not having a genetic risk for antisocial behavior based on their biological mother's self-reported history of antisocial misconduct. Parental warmth, negative control, and inconsistency in discipline were included as the three parenting scales in the analysis. Childhood behavioral problems were indexed by using the Child Behavior Checklist.

Similar to the findings from the Ge et al. (1996) study, results generated from the CAP revealed that adopted children who were classified as having a genetic predisposition to misbehave were significantly more likely to score high on the parental measure of negative control; the findings for parental warmth and inconsistency in discipline failed to reach statistical significance. The findings thus suggested that children and adolescents who have a genetic risk for antisocial behavior are significantly more likely to receive more negative control from their adoptive parents. O'Connor et al. (1998:977) concluded that "the findings indicate that the correlation between the biological mother's characteristics and the adoptive parents' style of interaction with the children does indeed represent a genotype-environment correlation." Taken

together, different studies, using different samples, and different research designs, have consistently shown that rGEs are an important source of variation in childhood and adolescent behavioral problems.

Conclusion

The study of antisocial behavior and of personality development has been dominated by socialization theories—theories that ignore the potential effect of genetic and biological forces (Harris, 1998, 2006; Massey, 2002; Robinson, 2004; Rowe, 2002; Udry, 1995; Walsh, 2002). Indeed, most criminologists and criminological theories downplay the influence of genetics or are openly hostile towards genetic explanations of crime causation (see, for example, Gottfredson and Hirschi, 1990). The reasons for this disdain with genetics include ideological alliances, political views, a misunderstanding of the subject, and a lack of exposure to the literature (Degler, 1991; Udry, 1995; Walsh, 2002; Walsh and Ellis, 2004). However, with the mapping of the human genome and with the recent surge in studies documenting links between different genetic polymorphisms and certain phenotypes, criminologists are being confronted with a new reality of human behavior—a reality that shows convincingly that most behaviors and traits are heavily influenced by biological and genetic influences (Walsh and Ellis, 2003; Wright and Beaver, 2005).

Only recently has mainstream criminological research begun to recognize the importance of genetics in the etiology of criminal behavior. Of course this does not mean that genetics are the only explanation of antisocial behavior and that sociologically-based explanations should be abandoned; instead the most promising theories are those that integrate both environmental and genetic factors into a unified perspective accounting for why some people engage in crime and

deviancy (Raine, 2002a; Robinson, 2004; Walsh, 2000, 2005; Walsh and Ellis, 2003). The melding together of social and genetic/biological explanations—that is, a biosocial approach—is perhaps the most useful way to study criminals and their offending behaviors (Raine, 2002a; Walsh, 2002; Walsh and Ellis, 2003). For example, two of the most powerful methods of examining the close nexus between genes and the environment are gene X environment interactions (GxEs) and gene X environment correlations (rGEs). GxEs and rGEs are just two of the many ways that biosocial criminology can contribute substantially to the understanding of crime, criminality, and human behavior in general (Beaver and Wright, 2005; Raine, 2002; Rutter, 2006; Walsh, 2002; Walsh and Ellis, 2004).

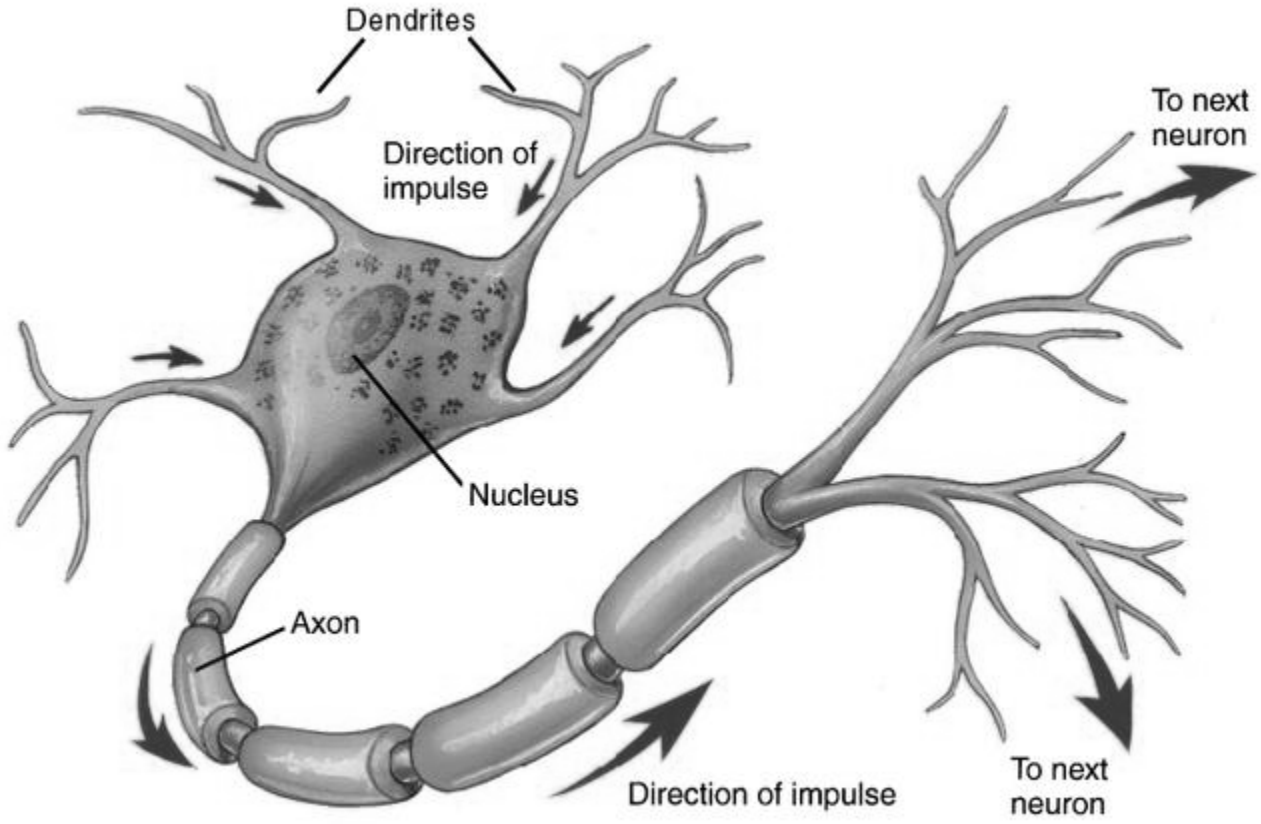
CHAPTER 3

DOPAMINE, SEROTONIN, AND MONOAMINE OXIDASE A

The human brain is comprised of billions of nerve cells called neurons (Dowling, 1998). The main function of each neuron is to receive, process, and transmit information from one brain cell to another (Dowling, 1998; Kotulak, 1997; Thompson, 1985). All neurons consist of two different types of neuronal branches, which are referred to as axons and dendrites. As shown in Figure 1, dendrites are connected to the cell nucleus, are made up of numerous branches, and receive input or incoming messages from other neurons. Figure 3.1 also depicts a neuronal axon. The axon is a comparatively long and spindly band of fibers but, unlike the dendrite that receives information, the axon sends or relays messages to other neurons (Dowling, 1998). In sum, information travels from neuron to neuron by moving from the cell nucleus down the axon until it eventually relays the information to the dendrite of another neuron. The process of information traveling from axon to dendrite to axon is repeated across neurons until the message reaches its final destination (Dowling, 1998; LeDoux, 2002).

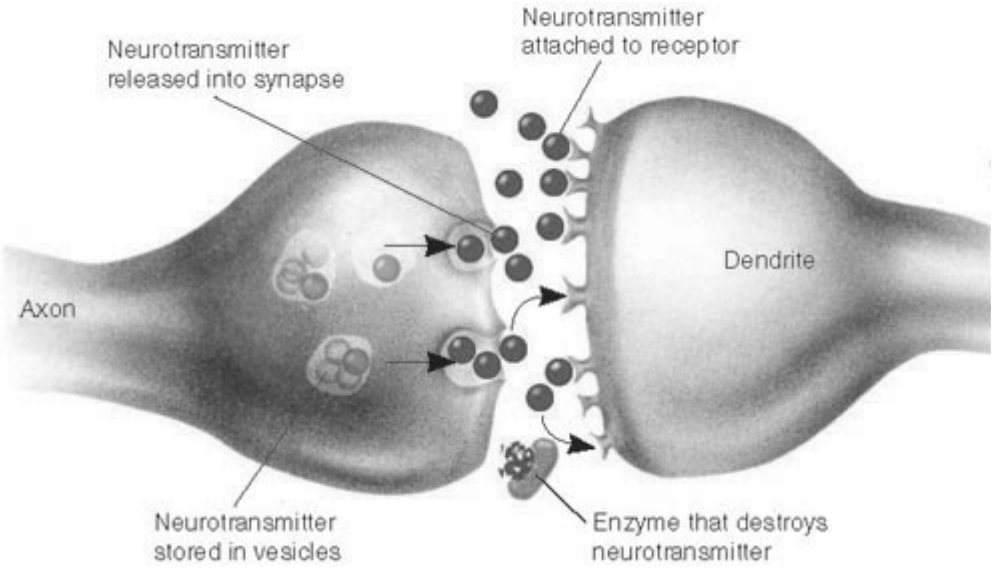
Although it may seem like the axon of one neuron is physically connected to the dendrite of another neuron, there is, in fact, a small gap that exists between axons and dendrites. This gap, portrayed in Figure 3.2, is referred to as the synapse or synaptic cleft (LeDoux, 2002). At first glance, this gap may seem to impede the transference of messages in the brain because in order for a message to move from neuron to neuron, the synaptic cleft must be bridged. Actually, however, moving messages across the synapse is accomplished easily by the release of neurotransmitters. Neurotransmitters are chemical messengers that are stored in the vesicles of the axon. When an axon needs to relay a message to an adjacent dendrite, neurotransmitters are

Figure 3.1. The Different Components to a Neuron



Notes: Available online at: http://www.mhhe.com/socscience/intro/ibank/ibank/0002.jpg

Figure 3.2. The Synaptic Cleft of a Neuron



Notes: Available online at: http://www.txtwriter.com/Backgrounders/Drugaddiction/synapse.jpg

released from the vesicles.[8] These neurotransmitters then cross the synaptic gap where they lock into the neurotransmitter receptor on the dendrite. The dendrite receives the message from the neurotransmitter where the information is then processed and transmitted to another neuron (Dowling, 1998). By using neurotransmitters for communication, a chemical reaction occurs, which allows messages to be transferred from one neuron to another quickly.

After the message has been delivered to the dendrite, neurotransmitters are removed from the synaptic cleft in one of two ways. First, the axon that released the neurotransmitters from its vesicle may reabsorb the neurotransmitters by manufacturing a membrane protein called a transporter (Dowling, 1998). Transporters eliminate the neurotransmitter in the synapse by capturing it and returning it to the vesicle. This process is referred to as reuptake and reuptake is very important in maintaining an appropriate level of neurotransmitters. If, for some reason, too many neurotransmitters or too few neurotransmitters are left in the synaptic cleft, then the human body and brain may experience adverse effects, such as depression.

The second way that neurotransmitters can be eliminated is through enzymes that target and remove neurotransmitters in the synapse. Enzymes break down neurotransmitters into inactive products (Dowling, 1998). Similar to the process of reuptake, enzymes are particularly vital to achieving normal levels of neurotransmitters. The key point to remember is that both the processes of reuptake and enzymatic degradation are essential in order to keep the body and the brain properly functioning.

[8] The release of neurotransmitters into the synapse is the end result of a sequential process that includes a series of electrical and chemical reactions. According to Dowling (1998:35) "When an action potential travels down an axon and reaches a synaptic terminal, the membrane surrounding the terminal becomes more positive because of the Na+ channels. In the terminal membrane are other channels that respond to this voltage change; they open and admit an ion that has two extra positive charges, calcium (Ca2+). Calcium ions, in ways still not well understood, promote the docking of vesicles to the membrane. This results in the fusion of the vesicle to the membrane and the opening of the vesicle to the outside. Neurotransmitter is then released and flows to channels on the postsynaptic membrane, thus activating them."

The human body contains a multitude of different neurotransmitters, each with their own unique functional properties; some excite, some inhibit, and still others' precise roles are unknown (Dowling, 1998; LeDoux, 2002). Depending on the particular neurotransmitter that is released, the body may respond in several ways. Some neurotransmitters may cause fear, delight, or aggression, while the release of other neurotransmitters, such as norepinephrine, is responsible for the body's "fight or flight" instincts. However, two of the most studied and perhaps most important neurotransmitters are dopamine and serotonin. Differential levels of these neurotransmitters have been linked to more than 25,000 psychological, behavioral, and mental problems, including depression, ADHD, and conduct disorders (Collins, 2004; Dowling, 1998; Hamer and Copeland, 1998; Niehoff, 1999; Raine, 1993).

Part of the reason for why levels of dopamine and serotonin vary from person to person is because many of the genes that code for the production, transportation, and breakdown of these neurotransmitters are polymorphic—and different variant of these genes can affect the level of neurotransmitters dispensed throughout the body and brain. For example, the dopaminergic system contains at least three different genetic polymorphisms—a dopamine transporter and two dopamine receptors—that are responsible for regulating dopamine levels while the serotoninergic system contains a highly polymorphic gene that is responsible for the reuptake of serotonin from the synapse. In addition, monoamine oxidase A (MAOA)—a gene that codes for an enzyme that breaks down dopamine and serotonin—is also a polymorphic gene that has been found to be important to maintaining healthy levels of neurotransmitters in the human body. Indeed, researchers have identified these specific genetic polymorphisms that regulate and control dopamine and serotonin levels as among the most promising candidate genes in

understanding the genetic sources of psychopathology (Clark and Grunstein, 2000; Ellis, 1991; Hamer and Copeland, 1998; Morley and Hall, 2003; Rowe, 2002).

The remainder of the current chapter draws from molecular genetic research and describes the functionality of dopamine, serotonin, and the five genetic polymorphisms that are implicated in the manufacturing and breaking down of these two neurotransmitters. Attention will also be devoted to the empirical research examining the linkages between these polymorphisms and various maladaptive outcomes. This chapter is divided into three sections. The first section focuses on the dopaminergic system, the second section focuses on the serotoninegic system, and the last section focuses on monoamine oxidase A (MAOA), a genetic polymorphism that eliminates neurotransmitters from the synapse.

Limitations of Genetic Research

Before discussing the findings garnered from the extant genetic research, it is important to point out the following four limitations that cut across many of these studies.

- The use of clinical (nonrepresentative) samples
- The use of small sample sizes that do no support multivariate analysis
- Univariate analyses that lack adequate statistical controls
- Restricted variation in the independent and dependent variables

The most common methodological problem in the genetic literature is the use of clinical and nonrepresentative samples. For example, many of the samples are comprised of subjects who have been diagnosed with serious mental disorder or some other type of psychological problem. Findings based on studies using clinical samples may not be generalizable to other groups of people. Second, and relatedly, the samples employed in genetic research also tend to

contain a small number of subjects, resulting in unstable parameter estimates. Also, the small sample size makes it difficult or impossible to conduct multivariate analyses. Third, most of the genetic research calculates univariate analyses that lack adequate statistical controls. Without controlling for other factors, it is not possible to determine whether the effects of the polymorphisms operate indirectly through other variables. The fourth and final limitation to genetic research is that the independent and dependent variables often have restricted variation. This problem stems from the heavy reliance on using clinical samples. The use of clinical samples results in less variation in the variables, making it difficult to detect significant relationships. Taken together, these four limitations hamper the ability to draw any firm conclusions about the relationship between certain genetic polymorphisms and antisocial outcomes.

Quantitative genetic research that examines whether *measured* polymorphisms are related to phenotypes is still in its infancy. Therefore it is probably not too surprising that this line of research is host to numerous shortcomings. Most promising, however, is that these limitations are dynamic—that is, now that they have been identified, the methodological and statistical problems can be systematically targeted and eventually nullified in future research. As genetic research becomes more and more refined by addressing these major concerns, the association between certain polymorphisms and different phenotypes will become much clearer and much more firmly established.

The Dopaminergic System

The dopaminergic system has long been thought to play an integral role in the development of maladaptive personality traits, in the creation of diseases and addictions, and in

the emergence of a wide array of aggressive and impulsive behaviors (Cloninger, 1987; Coccaro and Kavoussi, 1996; Depue et al., 1994; Ebstein et al., 1996; Hamer and Copeland, 1998; Niehoff, 1999; Raine, 1993). The overriding reason for why researchers have suspected the dopaminergic system is a potential cause to such diverse phenotypes is because dopamine is part of the reward or pleasure system of the human body (Blum, Cull, Braverman, and Comings, 1996; Wise and Rompre, 1989). Sudden surges in levels of dopamine provide immediate gratification to the human body leading to the repetition of the actions and behaviors that brought about this rise in dopamine. Subsequent declines in dopamine erase the pleasurable side effects experienced with the rise of dopamine. Molecular genetics researchers have identified the polymorphisms responsible for manufacturing and breaking down dopamine as potentially important genes in the etiology of delinquent and criminal behaviors. Before proceeding to a discussion of these genes, it is first necessary to provide some background information about the functional role of the neurotransmitter, dopamine, and the physiological responses that occur when dopamine is released in the human body.

Dopamine and Its Effects on the Body and Brain

Dopamine is a monoamine neurotransmitter manufactured by the body and is a member of the catecholamine family. Dopamine is produced in the brain by the arcuate nucleus of the hypothalamus, but is also synthesized by adrenal glands in the central and peripheral nervous systems (Velasco and Luchsinger, 1998). Although less than .5 percent of all neurons in the brain are responsible for synthesizing dopamine, the importance of these dopamine-producing cells should not be overlooked. Normal levels of dopamine are essential to keeping the body working in a normal and healthy capacity. The brain's potential to control movements, to

coordinate motor skills, and to perform other physical tasks is contingent on the release of dopamine into the body and brain in appropriate amounts. Of equal importance is that normal concentrations of dopamine are needed in order to allow the brain to operate at an optimal level; the ability to stay focused and problem-solving skills are contingent on the production of dopamine in proper quantities (Joel and Weiner, 2000; Rinne et al., 2000; Shatner, Havazelet-Heimer, Raz, and Bergman, 2003).

In addition to these functional roles, dopamine is also an instrumental part of the pleasure/reward system of the human body and brain (Blum et al., 1996; Dowling, 1989; Reif and Lesch, 2003; Wise and Rompre, 1989). When high concentrations of dopamine are released, the body experiences a naturally-occurring "high" that is accompanied by intense sensations of euphoria and joy. The behaviors and actions that brought about this increase in dopamine are thus reinforced making subsequent repetition of these acts more likely to occur.

Activities required for survival, such as sleeping and eating, are encouraged by triggering the release of dopamine. Engaging in sexual intercourse and using certain addictive substances (e.g., cocaine and amphetamines), moreover, are also associated with increases in dopamine levels—a powerful enticement to repeat these actions in the future (Clark and Grunstein, 2000; Hamer and Copeland, 1998). Human motivation, including the incentive to engage in certain behaviors, is due, in part, to the naturally-reinforcing pharmacological properties of the neurotransmitter, dopamine (Wise and Rompre, 1989).

When the production of dopamine is disrupted, or when the dopaminergic system malfunctions, the body may experience adverse and debilitating effects. Too much dopamine or too little dopamine, for example, can wreak havoc with the body and brain and lead to serious disorders, including psychosis, schizophrenia, Parkinson's Disease, anorexia, bulimia, mania,

and depression (Clark and Grunstein, 2000; Cooper, Blum, and Roth, 1986; Dowling, 1998; Hamer and Copeland, 1998; Niehoff, 1999; Raine, 1993). Symptoms of these diseases, however, can be ameliorated through the use of medication (Keefe, Silva, Perkins, and Lieberman, 1999; Wahlbeck, Cheine, Essali, and Adams, 1999). Many antipsychotic drugs are effective because they target the dopaminergic system and work to return aberrant levels of dopamine back into the normal range of variation (Farde, Wiesel, Halldin, and Sedvall, 1988; Moghaddam and Bunney, 1990; Velasco, 1989). The effectiveness of these drugs is testimony to the salience of dopamine to the human body.

Of primary concern, however, is the research that has examined whether different concentrations of dopamine are related to aggressive, violent, and impulsive behaviors (Coccaro and Kavoussi, 1996; Niehoff, 1999; Raine, 1993). Given that dopamine is an excitatory neurotransmitter, high levels of dopamine should be related etiologically to increases in antisocial conduct (Niehoff, 1999; Raine, 1993). Studies using human and nonhuman subjects have found mixed evidence bearing on this hypothesis; some research has supported the connection between dopamine and aggression (Baier, Wittek, and Brembs, 2002; Niehoff, 1999), whereas other studies have failed to replicate this finding (Raine, 1993; Scarpa and Raine, 2000).

Although more research needs to be conducted before a definitive conclusion can be reached on the relationship between levels of dopamine and aggression, one important qualification to the literature needs to be advanced at this time. While it may be tempting to infer that certain concentrations of dopamine cause an individual to act in a particular fashion, this would be a serious mistake. The findings from these studies are cross-sectional and thus are only able to imply a correlational relationship, not a causal one. Indeed, it is quite possible, and perhaps more realistic, to assume that dopamine levels wax and wane in response to certain

behaviors and environmental stimuli (Pincus, 2001; see also Sapolsky, 1997). If this is the case, then the temporal ordering between dopamine levels and aggression/violence may actually be reversed, wherein misconduct causes dopamine levels to rise and fall and not vice versa.

Dopaminergic Genetic Polymorphisms

In order to examine the connection between levels of dopamine and violent and delinquent behaviors, researchers have begun to study the genetic polymorphisms that are part of the dopaminergic system. Recall from Chapter 1 that genetic polymorphisms have the capacity to account for phenotypic variation. A dopamine transporter gene (DAT1) and two dopamine receptor genes (DRD2 and DRD4) have been of particular interest to geneticists studying aggression because these three polymorphisms are responsible for regulating and controlling levels of dopamine (Chen et al., 2005). Different variants of these genes may explain why quantities of dopamine vary from person to person—a potentially important discovery that could shed light on the genetic origins of crime and aggression. To examine the role of these genes in the functioning of the human body and to explore their effects on violence, a detailed description of DAT1, DRD2, and DRD4 will be provided. At the same time, empirical research examining how each genetic polymorphism relates to a range of phenotypes will also be presented.

Dopamine Transporter Gene (DAT1). The dopamine transporter gene (DAT1) has been mapped to chromosome 5 at location 5p15.3 (Vandenbergh et al., 1992). DAT1 has a 40 base pair variable number of tandem repeats (VNTRs). The number of repeats that an individual can posses ranges between 3 and 11 copies. The 40 base pair polymorphism is located in the 3' untranslated area of the gene (SLC6A3)—the same region of the gene responsible for the translation of the striatal dopamine transporter (DAT) protein (Heinz et al., 2000). The

frequency with which different allelic combinations occur, however, vary depending on an individual's ancestry and ethnic/racial background (Allele Frequency Database, 2006; Kang, Palmatier, and Kidd, 1999). The most common polymorphisms accounting for over 90 percent of all alleles in Caucasians and the African Americans are the 9-repeat and the 10-repeat alleles (Doucette-Stamm et al., 1995). In humans, "the 10-R allele of the DAT1 gene may be associated with a dopamine transporter that is abnormally efficient at the re-uptake process" (Swanson et al., 2000:24; see also Michelhaugh et al., 2001).

The DAT1 gene codes for the production of the dopamine transporter protein, which aids in reuptake (Vandenbergh et al., 1992). Recall that after a neurotransmitter—in this case, dopamine—has been released from the vesicle of the presynaptic neuron and has transmitted the message to the postsynapatic cell, the excess dopamine needs to be removed from the synapse. One way of eliminating the remaining dopamine is by removing it from the synapse and returning it to the vesicle—a process called reuptake. The dopamine transporter protein is one of the main agents in the reuptake of dopamine. Manipulations of the DAT1 gene have been found to influence the transcription of the dopamine transporter protein (Loder and Melikian, 2003). Suppressing or stimulating the activity of the DAT1 gene can alter levels of dopamine found in the body, thereby making it a potentially important gene in the etiology of psychopathology (Loder and Melikian, 2003; Morley and Hall, 2003; Niehoff, 1999; Rowe, 2002).

Two additional lines of research also point to the possibility that the DAT1 gene may play a role in the development of antisocial behaviors. First, many of the prescription drugs that have gained widespread success for muting the symptoms associated with ADHD and other psychopathological problems target specifically the DAT1 gene (Loo et al., 2002). These medications work by interfering with the reuptake process. Reuptake is depressed because the

medications block the dopamine transporter protein from clearing dopamine in the synapse (Seeman and Madras, 1998). As a result, more dopamine is available in the brain, alleviating the symptoms of ADHD. There is even evidence to suggest that the effectiveness of these drugs is contingent on the variant of the DAT1 polymorphism possessed by an individual (Kirley et al., 2003; McGough, 2005; Winsberg and Comings, 1999). Just as important is that illegal drugs, such as cocaine and amphetamines, get their psychotropic effects by rendering the dopamine transporter ineffective (Kang, Palmatier, and Kidd, 1999; Ritz, Lamb, Goldberg, and Kuhar, 1987).

Several animal knockout studies have been used to help pinpoint genes that may be related to aggression. In knockout studies, animals, usually rats or mice, have specific genes surgically removed or rendered inoperative (Plomin and Crabbe, 2000; Zeiss, 2005). Additionally, knockout studies can be conducted with strains of rats or mice that have been bred with a nonfunctioning gene. These animals are then monitored and subjected to a variety of conditions to see how they react. If they behave differently compared to animals who still have their genotype intact, then researchers infer that the gene that has been removed (i.e., knocked-out) is in some capacity related to the behavior that is being studied. Animal knockout studies have been used frequently to examine the role of dopaminergic system genes, including the DAT1 gene (Neihoff, 1999; Rodriguiz, Chu, Caron, and Wetsel, 2004; Russell, Sagvolden, and Johansen, 2005; Zeiss, 2005).

Research that has examined animals that have had their DAT1 gene knocked-out have, in general, found this gene to be particularly important to behavioral inhibition. Mice engineered to have a nonfunctioning DAT1 gene, for example, have been found to be relatively violent and aggressive (Gainetdinov and Caron, 2000; Gainetdinov and Caron, 2001; Niehoff, 1999;

Rodriguiz et al., 2004; Trinh et al., 2003). In a recent study, Rodriguiz et al. (2004) compared DAT1 knockout mice to mice with a normal functioning DAT1 gene and found that the knockout mice exhibited more signs of aggression and abnormal behaviors when they were presented with a social stimulus. ADHD-like symptoms, various manifestations of hyperactivity, and novelty-seeking behaviors are also observed more frequently in mice that are missing the DAT1 gene (Gainetdinov and Caron, 2000; Gainetdinov and Caron, 2001; Pogorelov et al., 2005; Trinh et al., 2003). DAT1 knockout mice even demonstrate deficiencies with memory recall and have a relatively limited ability to learn new tasks (Gainetdinov and Caron, 2001; Trinh et al., 2003). Taken together, the evidence gleaned from the knockout studies reveals that the DAT1 gene has functional importance for animals and possibly for humans.

Ethical considerations obviously prohibit researchers from manipulating human DNA and knocking out the DAT1 gene to determine its effects on behavioral patterns. Instead, geneticists have examined whether different variants of the DAT1 gene have diverse effects on a number of outcome measures. The most conventional way of isolating the effect of DAT1 is by comparing people with the 9R allele to people with the 10R allele. Subjects who possess uncommon or atypical alleles, such as the 3R allele, are either removed from the study or are placed into the 9R or the 10R group. Usually, people who possess alleles that range between 3 repeats and 8 repeats are placed into the 9R allele group, whereas people with 11 repeats are placed into the 10R allele group. Individuals are then assigned a score ranging between 0 and 2, indicating the number of risk alleles that they have. A score of 2, for example, would signify the presence of two risk alleles—one passed on from the mother and one passed on from the father. In the case of DAT1, the 10R allele is usually considered the risk allele, but some studies have revealed that the 9R allele confers a genetic risk to certain behaviors (Young et al., 2002).

Molecular geneticists have examined the effects of DAT1 on a number of different phenotypes, including ADHD, alcoholism, novelty-seeking, and gambling, among others. Table 3.1 contains the results of studies analyzing the linkages between the DAT1 gene and various measures related to antisociality. In order to facilitate the presentation of findings from these studies, Table 3.1 includes information about the sample, the outcome measures, the research design, the effect size, and conclusions about the relationship between DAT1 and the outcome measure. The DAT1 research can be grouped into three categories: studies examining ADHD, studies examining alcoholism, and studies examining other psychopathologies. The following discussion will highlight the broad findings that cut across Table 3.1.

The brunt of research presented in Table 3.1 has investigated the effect of the DAT1 polymorphism on ADHD. This should not be too surprising because after the mapping of the human genome, DAT1 was the first dopaminergic gene to be studied in relationship to the etiology of ADHD (Kirley et al., 2002). These initial studies set the stage for a wave of empirical research examining whether different variants of DAT1 affected the odds of being diagnosed with ADHD (Swanson et al., 2000). Fifteen studies in Table 3.1 examined the link between DAT1 and ADHD, and six (40 percent) found a statistically significant relationship—that is, these studies found that having the 10R allele increased the chances of developing ADHD. Of the studies that found a significant effect, two used a family-linkage research design, one used a genetic association research design, and the remaining three used both a family-linkage and a case control research design. The relationship between DAT1 and ADHD was observed in samples that were heterogeneous in respect to the respondents' age, ethnicity/race, and gender.

Table 3.1. The Effect of the Dopamine Transporter Gene (DAT1) on Various Outcome Measures

Study	Sample Characteristics	Outcome Measure(s)	Research Design	Effect Size	Findings
Bakker et al. (2005)	236 Dutch children and their parents were included in the analysis	ADHD	Family-linkage study	TDT=ns (values not reported)	No effect of 10R on ADHD
Barr et al. (2001)	333 persons (including both parents and an ADHD child) from 102 families	ADHD	Family-linkage study	χ^2=9.14	DAT1 related to ADHD
Blum et al. (1997a)	129 white respondents (58 males and 71 females; average age=40.9); 142 controls were also included	Schizoid/avoidant behaviors (SAB)	Case-control study	χ^2=6.3	Small effect of 10R on SAB
Comings et al. (2000a)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=.006 for ADHD; r^2=.009 for conduct disorder; r^2=.00 for ODD; All r^2s ns	No effect of 10R on ADHD, CD, or ODD
Comings et al. (2000b)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=.007 for ADHD; r^2=.000 for conduct disorder; r^2=.000 for ODD; All r^2s ns	No effect of 10R on ADHD, CD, or ODD
Comings et al. (2001)	139 pathological gamblers and 139 age, race, and sex-matched controls	Pathological gambling	Case-control study	r^2=.018	DAT1 related to pathological gambling
Franke et al. (1999)	87 alcohol-dependent probands and their parents (N=261 individuals)	Alcoholism	Family-linkage study	χ^2=2.13 (ns)	No effect of DAT1 on alcoholism
Gill et al. (1997)	40 children and 68 parents were included in the sample (no other demographics were reported)	ADHD	Family-linkage study	χ^2=6.07	10R related to ADHD

Holmes et al. (2000)	137 white children from Britain (126 males and 11 females) between 6 and 12 years old; 132 mothers and 107 fathers were included in the analysis; A total of 133 families participated; A control group consisting of 442 people were also included	ADHD	Family-linkage and case-control study	odds ratio=.96 for case control (ns); χ^2=.56 for family-linkage analysis (ns)	No effect of 10R on ADHD
Hopfer et al. (2005)	Data for this study came from the Add Health data set (n=233 at wave 1; n=258 at wave 2; and n=381 at wave 3)	Alcohol consumption	Genetic association study	β=-.29 (wave 1); β=.01 (wave 2) ns; β=-.01 (wave 3) ns	10R related to alcohol consumption (negative relationship)
Mill et al. (2005)	329 dizygotic twin pairs (N=658 twins)	ADHD	Genetic association study	*P*<.01	DAT1 related to ADHD
Muglia et al. (2002)	152 adults (53 females and 99 males) and 72 of their parents	ADHD	Genetic association and family-linkage study	Z=.16 (ns)	10R not related to ADHD
Parsian and Zhang (1997)	162 alcoholics (117 males and 45 females) and 89 control group subjects; additionally, 29 parents of the alcoholic group were also genotyped	Alcoholism	Case-control and family linkage study	χ^2=1.39 (ns)	DAT1 not related alcoholism
Payton et al. (2001b)	92 pairs of twins (50 scoring high on ADHD and 42 scoring low on ADHD) were included in the analysis	ADHD	Family-linkage study	Odds ratio=1.34 (ns)	DAT1 not related to ADHD
Qiujin et al. (2004)	202 families were included in the family-based analysis; 340 ADHD subjects and 226 controls were included in the case-control analysis	ADHD	Family-linkage and case-control study	χ^2=4.578 for the case-control analysis; χ2=.676 for family-based study (ns)	Some evidence that longer alleles related to ADHD

Roman et al. (2001)	81 Brazilian children and adolescents (average age=10.1; 86% males), 130 parents from 77 families and an ethnically-matched control sample	ADHD	Family-linkage and case-control study	χ^2=1.90 for case-control (ns); χ^2=.36 for family-linkage analysis (ns)	No effect of 10R on ADHD
Rowe et al. (1998a)	169 children from 125 families and 71 children in a control sample from 53 families (83% male; average age=9.8)	Generalized anxiety; major depression	Family-linkage and case-control study	r=.16 for generalized anxiety; r=.13 for major depression; QTDT=.23 for generalized anxiety; QTDT=.08 for major depression (ns)	10R related to generalized anxiety; small effect of 10R on major depression
Rowe et al. (2001)	The parents of ADHD children (80 fathers and 107 mothers) and a control sample of parents of non-ADHD children (42 fathers and 51 mothers)	ADHD; Conduct disorder	Family-linkage and case-control study	F=.32 for ADHD for fathers (ns); F=.44 for conduct disorder for fathers (ns); F=6.05 for ADHD for mothers; F=1.26 for conduct disorder for mothers (ns)	Small effect of 10R on ADHD mothers; no effect of 10R on conduct disorder for mothers; Results null for fathers
Simsek et al. (2005)	92 Omani children (62 boys and 30 girls) and 110 controls (60 males and 50 females)	ADHD	Case-control study	Hardy-Weinberg test ns (values not reported)	10R not related to ADHD
Sullivan et al. (1997)	Two groups of individuals were included in the analysis. The first group consisted of 86 subjects with a depressive illness (mean age=32 years old; 39.5 percent male). The second group consisted of 181 individuals who were members of alcoholic families (mean age=39.7 years old; 49.7 percent male)	Novelty seeking	Genetic association study	All p values <.05	No effect of 10R on novelty seeking

Todd et al. (2001)	Twins residing in 523 different families (ages ranged from 7 to 19 years old) were included in the sample; parents were also included in the analysis	ADHD	Family-linkage study	χ^2=1.4 (ns)	No effect of 10R on ADHD
Waldman et al. (1998)	117 children referred for ADHD (74% males; 68% white; average age=9.26), 41 of their siblings, and their parents; A control sample of 756 twins was also employed (49% male; 82% white; average age=8.5)	ADHD	Family-linkage and case-control study	*t*=1.87 for hyperactive-impulsive symptom; *t*=1.10 for inattentive symptoms (ns); Z=2.43 for hyperactive-impulsive symptom; Z=2.27 for inattentive symptoms; (*t*-tests for between-family analysis; Z-tests for within-family analysis)	DAT1 related to ADHD
Young et al. (2002)	790 children extracted from the Colorado Longitudinal Twin Study and the Colorado Adoption Project	Externalizing behavior problems	Genetic association study	*P*=.001 for behavior problems at age 4; *P*=.02 for behavior problems at age 7; *P*=.92 for behavior problems at age 9 (ns)	9R related to behavior problems

Notes:
ns=non-significant
TDT=transmission disequilibrium test (Speilmen et al., 1993)
QTDT=quantitative transmission disequilibrium test (Allison, 1997)

But the question remains whether the findings from the studies in Table 3.1 lend support in favor of, or against, the role DAT1 plays in the emergence of ADHD. While it is not possible to draw firm conclusions based solely on inspecting Table 3.1, a recent meta-analysis helps to shed light on this question. In 2002, Maher, Marazita, Ferrell, and Vanyukov conducted a meta-analysis based on eleven studies that examined the effects of DAT1 on ADHD. The results of this meta-analysis revealed that the DAT1 genetic polymorphism did not have a significant impact on ADHD. However, caution should be exercised before dismissing DAT1 as a potentially important genetic risk factor to ADHD. First, the null findings of the meta-analysis were primarily attributable to one large study that failed to find a significant effect of DAT1 on ADHD. Also, Maher et al.'s (2002) meta-analysis was conducted in 2002 and, since that time, a number of additional studies, including those reported in Table 3.1, have been published demonstrating that the 10R allele of DAT1 is related to ADHD. These studies may have shifted the weight of the evidence, and a more current meta-analysis might detect an overall effect of DAT1. Taken together, it is likely that DAT1 is one risk factor—but not the only risk factor—that is related to the development of ADHD (Kirley, 2002; Swanson et al., 2000).

The dopaminergic system is activated by the ingestion of certain drugs and by the consumption of alcohol. As a result, researchers have hypothesized that variants of the DAT1 gene may contribute to the risk for alcoholism. Table 3.1 displays the results of three studies examining the effect of DAT1 on alcoholism. Only one of these three studies found a significant relationship between DAT1 and alcohol consumption (Hopfer et al., 2005). Interestingly, this finding was somewhat anomalous to those of other studies: it was not the 10R allele that increased alcohol consumption, but rather the 9R allele that was related to higher rates of alcohol ingestion (Hopfer et al., 2005; but see Franke et al., 1999). The small number of studies that

have focused on DAT1 as a potential genetic risk-factor to alcoholism precludes a definitive conclusion about whether different variants of DAT1 may be related to alcoholism. Future research would benefit by continuing to assess the possible relationship between DAT1 and alcohol consumption (Hopfer et al., 2005).

Lastly, Table 3.1 includes studies that examine the effect of DAT1 on a hodgepodge of other outcome measures. Across these studies there is evidence to suggest that the 10R allele of the DAT1 gene is related to schizoid/avoidant behaviors (Blum et al., 1997a), to pathological gambling (Comings et al., 2001), and to generalized anxiety and major depression (Rowe et al., 1998a). The 9R allele has also been found to contribute to the development of externalizing behavioral problems (Young et al., 2002). Only one study in this last category (Sullivan et al., 1997) did not detect a significant relationship; the effect of the 10R allele DAT1 on novelty seeking failed to reach significance. Overall, the studies presented in Table 3.1 reveal that the 10R allele of the DAT1 gene (but see Hopfer et al., 2005; Young et al., 2002) has effects on a wide range of psychopathologies making it a particularly promising candidate gene that may also contribute to the development of more serious antisocial personalities and behaviors.

Dopamine Receptor Gene (DRD2). The dopamine receptor gene (DRD2) is a member of the D2 receptor family and has been mapped to chromosome 11 at location 11q23 (Itokawa et al., 1993; Grandy et al., 1989). The DRD2 gene codes for the D2 receptor and is found throughout the body, but especially in the striatum, the pituitary gland, the amygdala, the caudatus, the putamen, and other regions of the brain as well (Marino et al., 2004). Control of bodily movements and regulation of brain activity are two of the more important functions of the DRD2 gene. DRD2 has genetic variations that arise from single nucleotide polymorphisms (SNPs), restriction endonuclease sites, and di-nucleotide repeats. In addition, DRD2 has a

polymorphic TaqI restriction endonuclease site approximately 2,500 base pairs downstream (3' untranslated region) from the coding section of the gene (Grandy et al., 1989). The site of the TaqI restriction endonuclease is referred to as the TaqIA site to keep it distinct from the TaqIB restriction site also found on the DRD2 gene. The minor TaqIA (A1) allele has a point mutation C → T (TCGA to TTGA) that erases the TaqI site, whereas the A2 allele has the TaqI site intact. The TaqI allele is the most frequently studied polymorphism of the DRD2 gene (Gelernter and Kranzler, 1999).

The distributions of the A1 allele and the A2 allele of DRD2 vary considerably across different races and ethnic groups of the human population (Allele Frequency Database, 2006; Gelernter et al., 1998). For example, a recent analysis by Gelernter and associates (1998) revealed that 35.7 percent of African Americans possessed the A1 allele of DRD2, whereas only 19 percent of European Americans carried this allele ($p<.0055$). Additionally, 35.4 percent of the Japanese sample was identified as carrying the A1 allele, which was also statistically different from the frequency of A1 alleles in the European American sample. Significant differences in the occurrence of the allelic frequencies of other polymorphisms of the DRD2 gene, including TaqID and -141C*Ins*/*Del*, were also detected for African Americans, European Americans, and Japanese.

The A1 allele of the DRD2 gene is considered the risk allele and is compared with the A2 allele to determine whether these two genetic variants have differential effects on the human body and brain. Research investigating the functional role of the A1 allele has found that carriers of this allele, in contrast to carriers of the A2 allele, have less brain D2 dopamine receptors (Berman and Noble, 1995; Noble et al., 1991), have diminished glucose metabolism in the brain (Noble et al., 1997), are more attuned and responsive to stress (Berman and Noble,

1997), and exhibit reduced dopaminergic activity in the central nervous system (Berman and Noble, 1995). As a result of the findings from these studies, the A1 allele of DRD2 has been tagged as a major contributor to the "reward deficiency syndrome" of the human body (Blum et al., 1996; Blum et al., 1997b).

Reward deficiency syndrome describes people who need high levels of excitement and stimulation in order to activate their reward system in the same capacity as people with normal functioning reward systems. Addictive substances that act on the dopaminergic system, such as certain drugs, alcohol, and nicotine, are hypothesized to be used more frequently by people with reward deficiency syndrome. Consequently, carriers of the A1 allele of DRD2 (i.e., a genetic marker for the reward deficiency syndrome) use addictive substances to counteract their high threshold for rewards. The ingestion of certain drugs and the consumption of alcohol increases dopamine in the body and brain and allows the individual to experience the rewards associated with the release of high concentrations of dopamine. The A1 allele is thus considered a particularly important candidate gene in the etiology of addiction and alcohol abuse.

Similar to DAT1, DRD2 is also one of the target areas for anti-hyperactivity medications (Seeman and Madras, 1998). These pharmacological drugs blunt the activation of postsynaptic D1 and D2 dopamine receptors. Since these dopamine receptors are partially responsible for psychomotor regulation, changes to their activity levels are hypothesized to decrease or otherwise control excessive movement that so often characterizes children with ADHD (Seeman and Madras, 1998). The fact that anti-hyperactivity medicines change the activity levels of dopamine receptor genes points strongly to the possibility that DRD2 may be related to the development of attention deficiencies (Kirley et al., 2002; but see Todd and Lobos, 2002).

In addition, molecular geneticists have also searched for how carriers of the A1 allele of the DRD2 gene may be differentially predisposed to develop neuropsychiatric disorders and debilitating diseases (Itokawa et al., 1993; Noble, 2003, 2000; Sweet et al., 1998). Studies have found that DRD2 genetic polymorphism is related to Parkinson's disease (Plante-Bordeneuve et al., 1997), to obesity (Comings et al., 1993), to schizophrenia (Dubertret et al., 2004), and to obsessive-compulsive disorder (Denys, Van Nieuwerburgh, Deforce, and Westenberg, 2006). Different variants of the DRD2 gene have even been linked to certain personality styles (Munafò et al., 2003), age of first sexual intercourse (Miller et al., 1999), and visuospatial performance (Berman and Noble, 1995; but see Petrill et al., 1997; Moises et al., 2001). These findings suggest that the DRD2 gene may also contribute to the development of aberrant conduct, such as addiction and impulsive behaviors (Chen et al., 2005).

Table 3.2 is constructed in the same format as Table 3.1 and includes detailed information about the studies that examine the relationship between DRD2 and various maladaptive outcome measures. The overwhelming majority of research presented in Table 3.2 investigates whether the A1 allele of DRD2 contributes to an increased risk of becoming an alcoholic. Part of the reason for this heightened focus on alcoholism is because research using both human and nonhuman subjects has implicated the A1 allele of DRD2 as an important genetic pathway to addiction (Noble, 1996, 1998, 2000). Of the nineteen studies that examined the nexus between DRD2 and alcoholism in Table 3.2, ten (53 percent) found a statistically significant association. The results from these studies were garnered by using diverse research designs, by employing a wide range of samples, and by measuring alcoholism in a number of different ways. Based on the findings from Table 3.2, there is some evidence to suggest that the

Table 3.2. The Effect of the Dopamine D2 Receptor Gene (DRD2) on Various Outcome Measures

Study	Sample Characteristics	Outcome Measure(s)	Research Design	Effect Size	Findings
Amadéo et al. (1993)	49 French alcoholic patients and 43 controls were included in the sample	Alcoholism	Case-control study	χ^2=5.746	DRD2 associated with alcoholism
Amadéo et al. (2000)	71 Polynesians (61 males and 10 females), with an average age=40.8 and a control sample of 59 subjects (26 males and 33 females; average age=29.5)	Alcoholism	Case-control study	χ^2=ns (values not reported)	No effect of DRD2 (the A1 allele) on alcoholism
Arinami et al. (1993)	78 Japanese alcoholics (74 males and 4 females), with an average age=51 and a control group of 100 Japanese, with an average age=44	Severity of alcoholism	Case-control study	F=3.44	DRD2 associated with alcoholism (the A1allele)
Bau, Almeida, and Hutz (2000)	115 Brazilian male alcoholics and 114 controls	Alcoholism; Antisocial personality symptoms	Case-control study	Interaction between stress and DRD2 on alcohol dependence (B=.25); Interaction between harm avoidance and DRD2 on alcohol dependence (B=.23); Interaction between DRD2 and harm avoidance on antisocial personality symptoms (B=.26)	DRD2 interacted with certain items to predict alcohol dependence and antisocial personality symptoms
Berman et al. (2002)	110 sons of alcoholic fathers and 93 sons of nonalcoholic social drinkers, with an average age=12.4	Harm avoidance; Novelty seeking; Reward dependence	Genetic association study	F=4.82 for novelty seeking; F=ns for harm avoidance (values not reported); F=ns for reward dependence (values not reported)	Small effect of DRD2 on novelty seeking; DRD2 not associated with harm avoidance or with reward dependence

Blum et al. (1997a)	129 white respondents (58 males and 71 females; average age=40.9); 142 controls were also included	Schizoid/avoidant behaviors (SAB)	Case-control study	χ^2=9.2	DRD2 associated with SAB
Burt et al. (2002)	137 families (n=348 twins, their mothers and their fathers); the twins were seventeen years old	Harm avoidance; Traditionalism; Control; Constraint	Family-linkage study	Effect sizes not reported (all ns)	No effect of A1 on harm avoidance, traditionalism, control, or constraint
Chen et al. (1997)	A total of 203 alcoholics (168 males and 35 females) and 213 ethnically-matched controls (178 males and 35 females) from five different groups were included. The five groups were: Atayal, Ami, Bunun, Paiwan, and Han	Alcoholism	Case-control study	*P* for FET=.39 for Atayal (ns); *P* for FET=.08 for Ami (ns); *P* for FET=.78 for Bunun (ns); *P* for FET=1.00 for Paiwan (ns); *P* for FET=.18 for Han (ns)	No effect of A1 on alcoholism
Comings et al. (2000a)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=ns for ADHD; r^2=ns for conduct disorder; r^2=ns for ODD	No effect of A1 on ODD, CD or ADHD
Comings et al. (2000b)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=.004 for ADHD; r^2=.013 for conduct disorder; r^2=.010 for ODD; All r^2s ns	No effect of A1 on ADHD, CD, or ODD
Comings et al. (2001)	139 pathological gamblers and 139 age, race, and sex-matched controls	Pathological gambling	Case-control study	r^2=.022	DRD2 related to pathological gambling

Connor et al. (2002)	106 Australian Caucasian subjects recruited from an alcohol detoxification program (75 males and 31 females), with an average age=41.4 years	Alcohol consumption	Genetic association study	F=8.88 for alcohol quantity; F=2.28 for drinking frequency (ns); F=9.03 for alcohol consumption F=4.02 for alcohol dependence scale	Effect of A1 allele on alcohol use
Gelernter and Kranzler (1999)	160 European American alcoholics and 136 European American control group subjects	Alcoholism	Case-control study	χ^2=.001 (ns)	No effect of DRD2 on alcoholism
Gelernter, Kranzler, and Satel (1999)	96 European-American cocaine addicts, 77 African-American cocaine addicts, 87 European-American controls, and 45 African-American controls	Cocaine use	Case-control study	χ^2=.64 for European Americans (ns); χ^2=.15 for African Americans (ns)	No effect of DRD2 on cocaine use
Goldman et al. (1997)	459 Southwestern American Indians	Alcoholism; Substance abuse; Schizophrenia	Genetic association and family-linkage study	Effect sizes not reported (all ns)	No effect of DRD2 on alcoholism, substance abuse, or schizophrenia
Gorwood et al. (2000a)	113 alcoholic patients (average age= 43.6) and a control sample of 49 subjects (average age=32.4)	Alcoholism	Case-control study	χ^2=.05 (ns)	No effect of A1 on alcoholism
Gorwood et al. (2000b)	122 French subjects were included; 21 were bipolar patients with co-morbid alcohol dependence; 31 bipolar patients without co-morbid alcohol dependence; 35 patients with alcohol dependence but without bipolar disorder; 35 healthy controls	Alcoholism	Case-control study	χ^2=.15 (ns)	No effect of A1 on alcoholism
Hallikainen et al. (2003)	A representative sample of 884 Finnish Caucasian males, with an average age=56.1 years	Alcohol consumption	Genetic association study	F=3.2 in a multivariate model	Effect of DRD2 on alcoholism

Hopfer et al. (2005)	The Add Health genetic subsample was included in the analysis	Alcoholism	Genetic association study	β=-.12 for wave one (ns); β=-.12 for wave two (ns); β=-.22 for wave three (sig.)	Effect of DRD2 on alcoholism in one of the three waves examined
Katsuragi et al. (2001)	105 Japanese volunteers (57 males and 48 females), with an average age=24.6 were included in the sample	Novelty seeking; Harm avoidance; Reward dependence	Genetic association study	F=.88 for novelty seeking (ns); F=1.13 for harm avoidance (ns); F=1.22 for reward dependence (ns)	No effect of DRD2 on novelty seeking, harm avoidance, or reward dependence
Kono et al. (1997)	100 alcoholics (78 males and 22 females) and 93 controls (70 males and 23 females) were included in the sample (all Japanese)	Early-onset alcoholism	Case-control study	P<.05 for A1/A1 genotype frequency (values not reported)	Effect of DRD2 on early-onset alcoholism
Lee et al. (2003)	243 Koreans (70 males and 173 females; average age=13.87)	Novelty seeking; Harm avoidance; Reward dependence; Persistence	Genetic association study	F=.07 for novelty seeking (ns); F=.55 for harm avoidance (ns); F=.04 for reward dependence (ns); F=.96 for persistence (ns)	DRD2 not related to novelty seeking, harm avoidance, reward dependence, or persistence
Li et al. (2002)	121 Chinese heroin addicts (average age=25.72) and 194 control subjects (average age=28.60)	Heroin use	Case-control study	χ^2=1.56 (ns)	No effect of DRD2 on heroin use
Lu et al. (1996)	46 Chinese Hans, 42 Atayal, and 40 Ami were included in the sample	Alcoholism	Genetic association study	χ^2=.41 for Chinese Han sample (ns); χ^2=.19 for Atayal sample (ns); χ^2=.23 for Ami sample (ns)	No effect of DRD2 (the A1 allele) on alcoholism

Marino et al. (2004)	120 children (97 males and 23 females; average age=10.9) and their parents	Withdrawn; Somatic; Anxious-depressed; Social problems; Attention problems; Delinquent behavior; Aggressive behavior	Genetic association and family-linkage study	r^2=.01 for withdrawn; r^2=.01 for somatic; r^2=.01 for anxious-depressed; r^2=.04 for social problems; r^2=.00 for attention problems; r^2=.00 for delinquent behavior; r^2=.00 for aggressive behavior; All r^2s are ns	No effect of DRD2 on any of the scales
Noble et al. (1998a)	119 white males (mean age=12.1) from the Las Angeles area	Novelty seeking; Reward dependence; Harm avoidance	Genetic association study	F=4.71 for novelty seeking; F=1.35 for reward dependence (ns); F=1.11 for harm avoidance (ns)	Boys with A1 had higher novelty seeking scores; A1 not related to the other two personality scales
Noble et al. (1998b)	57 severe alcoholic Caucasians, 114 less severe alcoholic Caucasians, and 45 nonalcoholic controls were included in the analysis	Alcoholism	Case-control study	χ^2=23.2	Effect of DRD2 on alcoholism
Noble et al. (2000)	92 alcoholic Caucasians (average age=48.9) and 85 nonalcoholic controls (average age=52.3) were included in the analysis	Alcoholism	Case-control study	χ^2=24.2	Effect of DRD2 on alcoholism
Parsian, Cloninger, and Zhang (2000)	173 alcoholics (163 whites and 10 blacks); A control group of 88 participants were also included in the analysis	Alcoholism	Case-control study	χ2=ns for different variants of the DRD2 gene	No effect of DRD2 on alcoholism

Ponce et al. (2003)	103 male alcoholic Spanish subjects (mean age=41) predominantly from middle- to low- socioeconomic status	Alcoholism; Antisocial personality disorder (APD)	Genetic association study	χ^2=4.13 for alcoholism family history; χ^2=21.5 for APD	Effect of DRD2 on family history of alcoholism and on APD
Suarez et al. (1994)	88 white alcoholics and 89 matched controls were included in the sample	Alcoholism	Case-control study	No significant differences in RFLP frequencies (values not reported)	No effect of DRD2 on alcoholism

Notes:
ns = non-significant
FET=Fisher's exact test
RFLP = restriction fragment length polymorphisms

DRD2 gene is related to the risk alcoholism. This interpretation has been confirmed by a number of meta-analyses that find a statistically significant association between the A1 allele of DRD2 and alcoholism (Blum et al., 1995; Cloninger, 1991; Cook and Gurling, 1994; Gorwood, Ades, and Feingold, 1994; Pato et al., 1993) and by large reviews of the literature by the leading expert on the DRD2-alcoholism link (Noble, 1996, 1998).

The remainder of the studies in Table 3.2 examine whether DRD2 is associated with a scattering of different psychopathologies. Findings from these studies reveal that DRD2 is not associated with ADHD (Comings et al., 2000a, 2000b), cocaine and heroin use (Gelernter, Kranzler, and Satel, 1999; Li et al., 2002), and novelty seeking (Katsuragi et al., 2001; Lee et al., 2003). Other research, however, has found that DRD2 is related to novelty seeking (Berman et al., 2002; Noble et al., 1998), schizoid/avoidant behaviors (Blum et al., 1997), pathological gambling (Comings et al., 2001), and antisocial personality disorder (Bau, Almeida, and Hutz, 2000; Ponce et al., 2003). Taken together, the research in Table 3.2 demonstrates that the A1 allele is related to some antisocial behaviors, but may not be related to others.

There is good reason to believe, however, that when methodological, theoretical, and statistical shortcomings are addressed, the relationship between DRD2 and various disorders would be much more consistent and robust across studies (Comings, 1998). For example, Comings (1998) points out that most, if not all, disorders are due to the confluence of multiple genes, not one gene in isolation. If this is the case, then each gene would have relatively small effects. These small effects are often difficult to detect because of the lack of statistical power (Comings, 1998). Mathematical simulations have shown convincingly that studies attempting to replicate a genetic effect found by one study need to have increasingly larger samples to be able to detect the same effect using a different sample (Suarez, Hampe, and Van Eerdewegh, 1994).

The small sample sizes used in many of the studies presented in Table 3.2 probably contribute to the lack of consistent findings. Therefore, even studies that fail to show a significant effect of the A1 allele do not rule out DRD2 as a candidate gene for a number of psychopathologies (Comings, 1998; Suarez, Hampe, and Van Eerdewegh, 1994). Additional research needs to be conducted before firm conclusions can be drawn about the role of the A1 allele of DRD2 in the etiology of behavioral disorders.

Dopamine Receptor Gene (DRD4). The dopamine receptor gene (DRD4) has been mapped to chromosome 11 at location 11p15.5 and is found on the third exon (Gelernter et al., 1992). Similar to DRD2, DRD4 also belongs to the D2 dopamine family but manufactures the D4 dopamine receptor protein instead of the D2 dopamine receptor protein. The D4 dopamine receptor protein is found in areas of the brain that are responsible for the expression of emotions and for the stimulation of cognitive faculties (Schmidt et al., 2001). Moreover, the DRD4 gene, like other genes in the dopaminergic system, regulates attention processes, promotes motivation, and has been linked to exploratory behaviors (Schmidt et al., 2001). Molecular genetic research reveals that different DRD4 polymorphisms may actually demonstrate unique pharmacological properties that may affect a wide range of phenotypes (Van Tol et al., 1992).

DRD4 is a highly polymorphic gene that consists of a 48 base pair VNTR that can be repeated 2 to 11 times (Add Health Biomarkers Team, no date; Chan et al., 1996; Lichter et al., 1993). The 7-repeat allele has been shown to mediate a blunted intracellular response to dopamine and may also encode a receptor that is subsensitive to dopamine (Asghari et al., 1995). Importantly, Becker and his colleagues (2005:848) point out "…that the number of repeats in DRD4 can affect the binding of ligands to the receptor and that dopamine mediates the exploratory behaviour in experimental animals" (Ebstein et al., 1996). Based off this

information, molecular geneticists have singled out the 7R allele of DRD4 as one of the most promising candidate genes to many behavioral, psychiatric, and neuropsychological disorders (Faraone, Doyle, Mick, and Biederman, 2001; Keltikangas-Järvinen, Räikkönen, Ekelund, and Peltonen, 2004).

The 2-repeat (2R), 4-repeat (4R), and 7-repeat (7R) alleles are the most commonly occurring variants of the DRD4 gene; however, there is a great deal of variability in the distribution of DRD4 alleles across different continents and across different racial/ethnic categories (Add Health Biomarkers Team, no date; Allele Frequency Database, 2006; Chang et al., 1996; Chen, Burton, Greenberger, and Dmitrieva, 1999; Ding et al. 2000; Ding et al., 2002; Harpending and Cochran, 2002; Wang et al., 2004). For example, the 7R allele is the most frequently observed allele for people residing in North America and South America, but is a relatively scarce allele for inhabitants of Africa, and is almost nonexistent for Asians (Allele Frequency Database, 2006; Chang et al., 1996; Wang et al., 2004). After analyzing the DRD4 genetic polymorphism for 1,327 individuals from thirty-six different populations, Chang and associates (1996, p. 99) concluded that "the DRD4 locus shows an extraordinary level of expressed polymorphism with significant variation in allele frequencies in different populations."

Since DRD4 exhibits a functional polymorphism, molecular and behavioral geneticists have devoted a considerable amount of time to studying this gene. Animal knockout studies have proven to be particularly useful in isolating the effects of this gene. This line of research uses mice with their DRD4 gene knocked out to identify how different alleles of DRD4 contribute to phenotypic variation (Dulawa, 1999; Rubinstein et al., 1997). For example, Dulawa and her colleagues (1999:9550) found that mice lacking the DRD4 gene were less responsive to novel stimuli, suggesting that DRD4 may be implicated in "novelty-related

exploration." In another study, Rubinstein et al. (1997) found that mice with their DRD4 gene knocked out were supersensitive to doses of ethanol, cocaine, and methamphetamines when compared to those mice that possessed the DRD4 gene. These findings showing that DRD4 is important to the functioning of the body, however, are not limited to animal models. Studies using human subjects have found, for instance, that DRD4 may be implicated in the etiology of mood disorders (León et al., 2005), major psychosis (Serretti et al., 1999) and even sexual behaviors (Hamer and Copeland, 1998).

Of particular importance, however, is whether different variants of the DRD4 gene contribute to antisocial outcomes. Table 3.3 contains the results from studies that examined the relationship between DRD4 and a number of measures tapping misbehavior. Similar to DAT1, the majority of the studies investigated whether the DRD4 gene was linked with a clinical diagnosis of ADHD. For example, twenty-seven of the forty-four studies (61 percent) presented in Table 3 employed ADHD as the dependent variable. Of these studies, fourteen (52 percent) detected a statistically significant relationship between DRD4 and ADHD. In most cases the 7R allele was identified as the risk allele that increased the risk of developing ADHD.[9] As revealed in Table 3.3, the studies that did observe an association between DRD4 and ADHD used a variety of different research designs including case-controls (e.g., LaHoste et al., 1996), genetic association designs (e.g., El-Faddagh et al., 2004), and family-linkage techniques (e.g., Arcos-Burgos et al., 2004). In addition, the DRD4-ADHD link was observed in males and females (Rowe et al., 1998b; Rowe et al., 2001; Smalley et al., 1998), in children and adults (LaHoste et

[9] Only two studies (Leung et al., 2005; Qiujin et al. 2004) found that another allele was associated with an increased risk of ADHD and both of these used samples that consisted of Chinese subjects. It is important to note that for both samples, because of the variation in allelic distributions across different ethnicities, none of the subjects were carriers of the 7R allele. As a result, different alleles were analyzed to determine whether the DRD4 gene was related to ADHD—in both studies, a relationship between DRD4 and ADHD was detected. For the Leung et al. (2005) study, the 2R conferred a greater risk to ADHD, whereas the Qiujin et al. (2004) study found evidence suggesting that longer alleles (i.e., more repeats) increased the risk of being diagnosed with ADHD.

Table 3.3. The Effect of the Dopamine D4 Receptor Gene (DRD4) on Various Outcome Measures

Study	Sample Characteristics	Outcome Measure(s)	Research Design	Effect Size	Findings
Arcos-Burgos et al. (2004)	14 extended, multigenerational families from Colombia	ADHD	Family-linkage study	z=1.897 for PDT	Effect of 7R on ADHD
Auerbach et al. (2004)	64 one-year old infants	Attention; Information Processing	Genetic association analysis	F=8.06 for attention; F=4.65 for information processing	Effect of 7R on attention and information processing
Bakker et al. (2005)	236 Dutch children and their parents were included in the analysis	ADHD	Family-linkage study	TDT=ns (values not reported)	No effect of 7R on ADHD
Barr et al. (2000)	82 families were included in the analysis	ADHD	Family-linkage study	χ^2=2.882 for TDT; χ^2=4.9 for TDT	DRD4 related to ADHD
Becker et al. (2005)	303 adolescents (144 males and 159 females)	Novelty seeking; Harm avoidance; Reward dependence; Persistence	Genetic association study	t=3.21 for novelty seeking for males; t=2.03 for harm avoidance for males;	Effect of 7R on novelty seeking and harm avoidance for males; Results for females were null
Burt et al. (2002)	137 families (n=348 twins, their mothers and their fathers); the twins were seventeen years old	Harm avoidance; Traditionalism; Control; Constraint	Family-linkage study	Effect sizes not reported (all ns)	No effect of 7R on harm avoidance, traditionalism, control, or constraint
Castellanos et al. (1998)	41 children with severe ADHD (average age=9.7); 56 control subjects (average age=17.6)	ADHD	Case-control study	χ^2=.06 (ns)	No relationship between 7R and ADHD
Comings et al. (2000a)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=ns for ADHD; r^2=ns for conduct disorder; r^2=ns for ODD	No effect of 7R on ADHD, ODD, or conduct disorder

Comings et al. (2000b)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=.000 for ADHD; r^2=.000 for conduct disorder; r^2=.000 for ODD; All r^2s ns	No effect of 7R on ADHD, CD, or ODD
Comings et al. (2001)	139 pathological gamblers and 139 age, race, and sex-matched controls	Pathological gambling	Case-control study	r^2=.035	DRD4 related to pathological gambling
De Luca et al. (2003)	95 3-year old children (41 males and 54 females)	Activity level; Attention span; Distractibility	Genetic association study	*t*=-.78 for activity level (ns); *t*=.06 for attention span (ns); *t*=-.42 for distractibility (ns)	No association between 7R and activity level, attention span, and distractibility
Eisenberg et al. (2000)	46 families were included (no other information was reported)	ADHD	Family-linkage study	χ^2=.14 (ns)	7R not related to ADHD
El-Faddagh et al. (2004)	265 children (126 males and 129 females)	ADHD	Genetic association study	Fisher's exact test significant for males but not for females (values not reported)	7R related to ADHD in boys but not for girls
Faraone et al. (1999)	27 ADHD parents, their spouses, and their ADHD children (no demographics reported)	ADHD	Family-linkage study	χ^2=7.4 *z*=2.5 for TDT	Small effect of 7R on ADHD
Frank et al. (2004)	81 children and adolescents with ADHD and a control group of 24 children	ADHD; Novelty seeking; Harm avoidance; Reward dependence	Case-control study	F=ns; Correlation coefficients=ns; (values not reported)	No effect of 7R on ADHD, novelty seeking, harm avoidance, or reward dependence

Hawi et al. (2000)	21 families consisting of an ADHD child and their mother and 78 families consisting of an ADHD child, their mother, and their father (all from Ireland)	ADHD	Family-linkage study	χ^2=0.0	7R not related to ADHD
Holmes et al. (2000)	137 white children from Britain (126 males and 11 females) between 6 and 12 years old; 132 mothers and 107 fathers were included in the analysis; A total of 133 families participated; A control group consisting of 442 people were also included	ADHD	Family-linkage and case-control study	Odds ratio=1.9 for case-control; χ^2=5.97 for family-linkage analysis (ns)	7R related to ADHD in case-control analysis; 7R not related to ADHD for family-linkage analysis
Hopfer et al. (2005)	Data for this study came from the Add Health data set (n=241 at wave 1; n=286 at wave 2; n=435 at wave 3)	Alcohol consumption	Genetic association study	β=.03 (wave 1) ns; β=-.13 (wave 2) ns; β=.06 (wave 3) ns	No effect of DRD4 on alcohol consumption
Keltikangas-Järvinen et al. (2004)	92 respondents (45 females and 47 males) ranging in age from 6-15 in the first wave of data and 20-29 years of age in the last wave of data; All respondents were from Finland	Novelty seeking	Genetic association study	Odds ratio=2.44; Logit=.70 for interaction between DRD4 and emotional distance; Logit=.74 for interaction between DRD4 and disciplinary style	Interaction of 2R or 5R and environment on novelty seeking
Kirley et al. (2004)	178 Irish families with a clinically diagnosed ADHD child	ADHD and ODD	Genetic association study	χ^2=1.94 for ADHD (ns); χ^2=6.7 for ADHD with comorbid ODD	7R not related to ADHD; Effect of 7R on ADHD with comorbid ODD
Kotler et al. (2000)	49 children (42 males and 7 females; average age=9.89) from Israel and a control sample were included in the study	ADHD	Case-control study	Likelihood ratio=7.94 (ns); Likelihood ratio=5.50 for short vs. long allele (controls more likely to have long allele)	7R not related to ADHD; Control group had an excess of the long allele

LaHoste et al. (1996)	39 children aged 7-12 years old and 39 control children	ADHD	Case-control study	χ^2=10.03	7R related to ADHD
Laucht et al. (2005)	303 15-year olds (144 males and 159 females) from Germany	Novelty seeking; Smoking	Genetic association study	*t*=3.21 for novelty seeking for males only; χ^2=9.11 for smoking for males only	Boys with 7R had higher novelty seeking scores and had higher rates of smoking; Results for girls were null
Lee et al. (2003)	243 Koreans (70 males and 173 females; average age=13.87)	Novelty seeking; Harm avoidance; Reward dependence; Persistence	Genetic association study	F=4.6 for novelty seeking; F=2.32 for harm avoidance (ns); F=.16 for reward dependence (ns); F=3.27 for persistence (ns)	DRD4 related to novelty seeking; No effect of DRD4 on harm avoidance, reward dependence, or persistence
Leung et al. (2005)	32 Hans Chinese boys, ranging in age from 6-15 years old (average age =9.1)	ADHD	Genetic association study	χ^2=5.9 for the 2R	2R related to ADHD; No subjects had the 7R
Marino et al. (2004)	120 children (97 males and 23 females; average age=10.9) and their parents	Withdrawn; Somatic; Anxious-depressed; Social problems; Attention problems; Delinquent behavior; Aggressive behavior	Genetic association and family-linkage study	r^2=.06 for withdrawn; r^2=.02 for somatic (ns); r^2=.02 for anxious-depressed (ns); r^2=.05 for social problems (ns); r^2=.01 for attention problems (ns); r^2=.00 for delinquent behavior (ns); r^2=.01 for aggressive behavior (ns)	Small effect of 7R on withdrawn; 7R not related to other scales

Mill et al. (2005)	329 dizygotic twin pairs (N=658 twins)	ADHD	Genetic association study	*P*>.01 (ns)	No effect of 7R on ADHD
Muglia et al. (2000)	66 adult ADHD subjects (37 males and 29 females; average age=34.3) and a control sample of 66 subjects (37 males and 29 females; average age=34.9); a second sample of 44 families were also included	Adult ADHD	Case-control and family-linkage study	χ^2=5.65 for the case-control study; χ^2=2.0 for the family-based study (ns)	7R related to adult ADHD for case-control study; Results null for family-based study
Noble et al. (1998a)	119 white males (mean age=12.1) from the Las Angeles area	Novelty seeking; Reward dependence; Harm avoidance	Genetic association study	F=3.96 for novelty seeking; F=2.95 for reward dependence (ns); F=.38 for harm avoidance (ns)	Boys with 7R had higher novelty seeking scores; 7R not related to the other two personality scales
Payton et al. (2001a)	150 ADHD children between 6 and 13 years old (average age=9.1) from 145 families of U.K origin; 144 mothers and 115 fathers were also included	ADHD	Family-linkage study	TDT (ns) (values not reported)	No effect of 7R on ADHD
Payton et al. (2001b)	92 pairs of twins (50 scoring high on ADHD and 42 scoring low on ADHD) were included in the analysis	ADHD	Family-linkage study	Odds ratio=1.37 (ns)	DRD4 not related to ADHD
Qiujin et al. (2004)	202 families were included in the family-based analysis; 340 ADHD subjects and 226 controls were included in the case-control analysis	ADHD	Family-linkage and case-control study	χ^2=8.08 for males in the case-control analysis; χ^2=.498 for females in the case-control analysis (ns); χ^2=0.00 for the family-based analysis	No subjects had the 7R; For males there is evidence that longer alleles are related to ADHD; Results for females were null

Rogers et al. (2004)	267 bipolar subjects (107 males and 160 females; average age=43.2); 172 alcoholics (84 males and 88 females; average age=39); 143 depressed subjects (60 males and 83 females; average age=31.6); 148 people suffering from a major depressive disorder (42 males and 106 females; average age=35)	Novelty seeking	Genetic association on four groups	*t*=2.556 for bipolar group; *t*=3.24 for alcoholic group; *t*=1.04 for depressed group (ns); *t*=.84 for major depressed group (ns); F=13.33 for within-family analysis of the bipolar group	120 bp repeat in the DRD4 gene is linked to novelty seeking
Roman et al. (2001)	81 Brazilian children and adolescents (average age=10.1; 86% males), 130 parents from 77 families and an ethnically-matched control sample	ADHD	Family-linkage and case-control study	χ^2=11.55 for case-control; χ^2=.37 for family-linkage analysis (ns)	Small effect of 7R on ADHD
Rowe et al. (1998b)	168 children from 123 different Families and a control sample of 71 children from 53 unique families; Of the 239 total children, 191 were males and 48 were females (average age=10); 155 mothers and 122 fathers were also included in the analyses	ADHD	Family-linkage and case-control study	χ^2=5.9 for combined type of ADHD; χ^2=4.6 for inattentive type of ADHD	7R related to ADHD
Rowe et al. (2001)	The parents of ADHD children (80 fathers and 107 mothers) and a control sample of parents of non-ADHD children (42 fathers and 51 mothers)	ADHD; Conduct disorder	Family-linkage and case-control study	F=4.30 for ADHD for fathers; F=4.89 for conduct for fathers; F=.98 for ADHD for mothers (ns); F=.33 for conduct disorder for mothers (ns)	Small effect of 7R on ADHD and conduct disorder for fathers; Results null for mothers

Sander et al. (1997)	252 German alcoholics (all males; mean age=41.9) and 197 German controls	Alcoholism; Novelty seeking; Harm avoidance; Reward dependence	Case-control study	χ^2=2.9 for alcoholism (ns); U-test ns for novelty seeking, harm avoidance, and reward dependence	DRD4 not related to alcoholism; DRD4 not related to novelty seeking, harm avoidance, and reward dependence
Schmidt et al. (2001)	174 primarily white children (81 boys and 93 girls)	Attention problems	Genetic association study	F=5.16	7R related to attention problems in children
Smalley et al. (1998)	220 children (163 boys and 57 girls) with ADHD from 133 families (average age=10.9) and 250 of their parents were included in the analysis	ADHD	Family-linkage study	χ^2=4.85	The presence of the 7R allele increases the risk of ADHD by 1.5
Smith et al. (2003)	105 Caucasians with ADHD and 68 age- and ethnically-matched control subjects	ADHD	Case-control study	χ^2=ns (values not reported)	DRD4 not related to ADHD
Sullivan et al. (1998)	A group of 86 depressed subjects (34 males and 52 females; average age=32) and a group of 181 subjects from alcoholic pedigrees (90 males and 91 females; average age=39.7); both groups contained subjects from New Zealand	Novelty seeking	Genetic association and sibling-pair analysis study	F=.08 for the depressed sample (ns); F=.79 for the alcoholic sample (ns)	7R not related to novelty seeking in either sample
Swanson et al. (1998)	52 children and both of their parents (no demographics reported)	ADHD	Family-linkage study	χ^2=4.65 for HRR	7R is related to ADHD

Szekely et al. (2004)	68 males (average age=22.7) and 89 females (average age=21.8) from Hungary	Novelty seeking; Harm avoidance; Reward dependence; Persistence	Genetic association study	F=2.16 for males in the multivariate genetic analysis (ns); F=6.39 for persistence for males; F=.87 for females in the multivariate genetic analysis (ns);	7R related to persistence for males (persistence decreases in males with 7R); Results were insignificant for females
Tsai et al. (2004)	120 Chinese females, ranging in age from 19-21 years old	Novelty seeking; Reward dependence; Harm avoidance	Genetic association study	ANOVA and *t*-test ns for all outcomes (values not reported)	DRD4 not related to novelty seeking, reward dependence or harm avoidance

Notes:
ns = non-significant
PDT = pedigree disequilibrium test
TDT = transmission disequilibrium test (Speilmen et al., 1993)
HRR = haplotype relative risk

al. 1996; Muglia et al., 2000; Rowe et al., 1998b), and in samples gathered from different areas of the world (Arcos-Burgos et al., 2004; Roman et al., 2001). Findings generated from two recent meta-analyses support the results presented in Table 3.3 by showing statistically that the 7R allele contributes to the development of ADHD across studies using diverse samples, using a wide range of statistical procedures, and using different analytical strategies (Faraone et al., 2001; Maher et al., 2002).

Sparked by early reports of a statistically significant relationship between DRD4 and the personality trait of novelty seeking (Benjamin et al., 1996; Ebstein et al., 1996), a wave of research has examined whether the 7R allele is related to measures of novelty seeking.[10] Six out of the ten studies (60 percent) included in Table 3.3 found that the DRD4 gene was significantly related to novelty seeking. The association between 7R and novelty seeking, however, was not upheld in a meta-analysis conducted by Kluger, Siegfried, and Ebstein (2002). In this meta-analysis, the authors analyzed twenty studies that examined the effect of DRD4 on novelty seeking. The results of their analysis revealed that the average effect size across the studies was d=.06, indicating a nonsignificant relationship between DRD4 and novelty seeking ($p<.05$).

In addition to the studies that examined the effects of DRD4 on ADHD and on novelty seeking, a large pool of research has investigated whether other psychopathologies are related to different variants of the DRD4 gene. The results of these studies are also contained in Table 3.3. A close inspection of this table shows that DRD4 exerts a statistically significant impact on information processing (Auerbach et al., 2004), pathological gambling (Comings et al., 2001), smoking (Laucht et al., 2005), and attention problems (Schmidt et al., 2001). On the other hand, additional studies contained in Table 3.3 failed to find an effect of DRD4 on the personality trait

[10] According to Cloninger (1987) people who score high on measures of novelty seeking are impulsive, excitable, exploratory, disorderly, and distractible.

of harm avoidance (Burt et al., 2002), on oppositional defiant disorder and conduct disorder (Comings et al., 2000a, 2000b), on activity levels (De Luca et al., 2003), and on alcohol consumption (Hopfer et al., 2005). The DRD4 gene, in short, has been found to be predictive of a range of psychosocial problems, but other studies have not been able to replicate these findings.

Summary of the Dopaminergic System

The dopaminergic system is vital to the healthy, normal functioning of the human body. Changes in levels of dopamine can severely alter an individual's personality, their social relationships, their behavioral patterns, and even their mental faculties (Hamer and Copeland, 1998; Niehoff, 1999). Genes that are responsible for regulating dopamine, therefore, have been identified as potentially important in the etiology of numerous disorders and diseases, including symptoms associated with various types of psychopathology (Rowe, 2002). As the above review demonstrated, there is clear and convincing evidence linking three genetic polymorphisms—DAT1, DRD2, and DRD4—of the dopaminergic system to a host of different maladaptive outcomes. There is some molecular genetic literature suggesting that different allelic combinations of these genes correspond to different levels of dopamine found throughout the human body and brain. Perhaps as a result, variants of these genes also explain variation in numerous phenotypes indexing antisociality. However, according to molecular and behavioral geneticists, future research needs to use comparatively larger, nationally representative samples in order to draw firm conclusions about the effect that dopaminergic genetic polymorphisms have on measures of psychopathology (Gordon, Finch, Nothnagel, and Ott, 2002; Kalinowski, 2005; Suarez, Hampe, Van Eerdewegh; Zou and Zou, 2006).

The Serotonergic System

The serotonergic system is instrumental to healthy brain development, to synaptic plasticity, and to keeping the body and brain operating efficiently (Dowling, 1998; Kotulak, 1997; Reif and Lesch, 2003; Thompson, 1985). The serotonergic system is comprised of all the genes that are responsible for manufacturing and breaking down the neurotransmitter, serotonin. Serotonin is the body's natural "brake system" and it regulates and controls impulses—in other words, serotonin allows humans to navigate through life without being distracted by every thought, emotion, and impulse. Equally important is that behavioral inhibition, modulation of eating, circadian rhythmic patterns, suppression of impulses, and aggressive drives are all under the control of the serotonergic system. When the serotonergic system is functioning properly, human emotions and primitive instincts are kept in check; however, when something interferes with the serotonergic system, impulses and innate drives take over, leading to explosive conduct, uncontrollable acts, and various other behavioral and psychological disorders (Dowling, 1998; Kotulak, 1997; Niehoff, 1999; Raine, 1993; Reif and Lesch, 2003; Thompson, 1985).

Given that serotonin controls behaviors and impulses, researchers have identified polymorphisms responsible for regulating the production and transportation of serotonin as candidate genes in the etiology of violence, crime, delinquency, and drug use. One genetic polymorphism in particular—the serotonin transporter gene (5HTT)—has been studied extensively by molecular geneticists. This line of literature has examined whether different alleles of the 5HTT gene are differentially related to the risk of developing disorders of the body and brain. The results of these studies have been promising and suggest that the 5HTT gene may be causally related to various phenotypes, but especially psychopathology. Before presenting a

description of the 5HTT gene and the research bearing on how it is related to crime and aggression, first it is necessary to provide a discussion of the neurotransmitter, serotonin.

Serotonin and Its Effects on the Body and Brain

Serotonin is a monoamine neurotransmitter that is manufactured by serotonergic neurons in the central nervous system and by enterochromaffin cells in the gastrointestinal system (Dowling, 1998; Kotulak, 1997; Thompson, 1985). Serotonin-producing cells in the brain are found primarily in the raphe nuclei, which is a cluster of cells situated along the brain stem that run from the medulla to the midbrain (Cooper, Bloom, and Roth, 1986; Thompson, 1985). The axons of neurons that manufacture serotonin are connected to various areas of the brain, some of which are responsible for higher-order functions. For example, serotonin nerve fibers extend outward from the raphe nuclei to the hypothalamus, to the hippocampus, to the cerebral cortex, to the basal ganglia, and to the amygdala (Bradley, 1991; Thompson, 1985). These areas of the brain are crucial for survival and for normal-life functioning—when functioning properly they decode emotions, control impulses, and process information from the environment. Not surprisingly, however, stimulating or depressing these brain regions—either through the release or the removal of serotonin—can bring about quite drastic swings in behavior and personality (Dowling, 1998; Kotulak, 1997; Moore, Scarpa, and Raine, 2002; Niehoff, 1999; Raine, 1993; Thompson, 1985). Keep in mind that both too much serotonin and/or too little serotonin can impact the body and brain in multiple ways.

Paradoxically, elevated levels of serotonin excite motor neurons leading to an increase in physical activity, but, at the same time, the release of serotonin in large quantities also works to suppress primitive impulses, such as sexual drives, aggression, and overeating (Dowling, 1998;

Thompson, 1985). As a result, researchers have expended a considerable amount of time and energy investigating whether high levels of serotonin are associated with an array of problems, including physical impairments, mental deficiencies, and behavioral disorders (Clark and Grunstein, 2000; Hamer and Copeland, 1998; Lucki, 1998). Findings generated from these studies have revealed that high serotonin levels confer a greater risk of developing psychosis, mood disorders, autism, Alzheimer's disease, anorexia, and schizophrenia (Clark and Grunstein, 2000; Hamer and Copeland, 1998; Lucki, 1998). However, it would be an overgeneralization to assume that high levels of serotonin always lead to deleterious outcomes. Indeed, in a series of experiments, Raleigh and his associates (Raleigh, McGuire, Brammer, and Yuwiler, 1984; Raleigh et al., 1980, 1991) examined whether levels of serotonin covaried with the social standing of vervet monkeys. These classic studies revealed that monkeys occupying the highest positions of social dominance also had the highest levels of serotonin—levels almost twice as high as the lowest-ranking monkeys.

One of the more interesting findings from the experiments with vervet monkeys was that levels of serotonin were the result of, not the cause of, a monkey's place in the social hierarchy. If a monkey dropped from a high social status to a lower social status, their serotonin levels would also drop. On the other hand, if a monkey moved up the social hierarchy, their serotonin levels would also increase. These findings revealing that levels of serotonin correspond to social dominance, however, are not isolated to experiments using nonhuman subjects. Past research indicates that males in the highest positions of power within fraternities also had significantly higher levels of serotonin when compared with lower-ranking fraternity members (cited in McGuire and Raleigh, 1986). Taken together, findings from both human and animal studies

suggest that levels of serotonin are deeply intertwined not only with personalities and behaviors, but also with the social environment.

Low levels of serotonin have also been tied to numerous psychological problems and conduct disorders (Dowling, 1998; Hamer and Copeland, 1998; Moore, Scarpa, and Raine, 2002; Raine, 1993). This should not be too surprising since serotonin is an inhibitory neurotransmitter that controls and regulates conduct. When there is a shortage of serotonin in the human body and brain, behavior is likely to be under-regulated, with the end result being the emergence of uncontrollable and erratic behavior (Kotulak, 1997; Lidberg et al., 1985; Raine, 1993; Thompson, 1985) For example, attempted and completed suicides, alcohol abuse, impulsiveness, depression, and bulimia have all been found to be more common for people with low levels of serotonin or with low levels of serotonin metabolites (Asberg, 1997; Clark and Grunstein, 2000; Dowling, 1998; Hamer and Copeland, 1998; Lucki, 1998; Lidberg et al., 1985; Mann et al., 1989; Niehoff, 1999; Thompson, 1985). Together, findings from these studies suggest that diminished concentrations of serotonin have widespread and debilitating effects on the human body and brain.

Researchers have also examined whether levels of serotonin are linked to violence, aggression, crime, delinquency, and various other forms of disrepute (Coccaro and Kavoussi, 1996; Goldman, 1996; Kotulak, 1997; Lidberg et al., 1985; Moore, Scarpa, and Raine, 2002; Niehoff, 1999; Pincus, 2001; Raine, 1993). Some of the earliest studies reporting a connection between serotonin and aggression used nonhuman subjects (Brammer et al., 1991; Cases et al., 1995; Chamberlain, Ervin, Pihl, and Young, 1987; Spoont, 1992). These experiments revealed that altered levels of serotonin were able to induce assaultive and combative behaviors for primates and for rodents. More recent studies using human participants have both confirmed

and negated the findings generated from the animal studies. Some studies find a positive relationship between serotonin levels and aggression (Moffitt et al., 1998), some studies find a negative relationship between serotonin levels and aggression (Clarke, Murphy, and Constantino, 1999; Kotulak, 1997; Lidberg et al., 1985; Moore, Scarpa, and Raine, 2002; Raine, 1993), and still other studies fail to detect a relationship between serotonin levels and aggression (Lappalainen et al., 1999).[11] Clearly, research examining the effects of serotonin levels on violence is mixed and, in some cases, contradictory, pointing to the likelihood that the serotonin-aggression relationship is complex and not wholly understood (Olivier, 2004). However, according to Raine (1993:85) "in general…research on the effects of serotonergic manipulation on aggression has shown *decreases* in aggression when serotonin is elevated (Brizer, 1988)."

Two meta-analyses have been conducted that generally support Raine's (1993) claim that low levels of serotonin increase violent and aggressive acts (Moore, Scarpa, and Raine, 2002; Raine, 1993). The first meta-analysis, conducted by Raine (1993), examined the effects of dopamine, serotonin, and norepinephrine and included studies that were published between 1974 and 1990. The results of this meta-analysis revealed that levels of serotonin distinguished antisocial individuals from healthy control subjects, wherein low serotonin concentrations were associated with antisocial behavior ($M=-.47$, $p<.05$). When compared with the effects of dopamine and the effects of norepinephrine, serotonin emerged as the strongest and most

[11] Serotonin cannot be measured directly in human subjects; instead, two indirect methods have been developed. The first procedure measures the amount of a serotonin metabolite found in spinal fluid (5-HIAA) and the second procedure measures the amount of serotonin found in blood platelet cells (Rowe, 2002). These different measurement strategies have resulted in what at first glance may appear to be irreconcilable results on the relationship between serotonin levels and antisocial conduct. Yet, Rowe (2002:77) provides a plausible explanation for these anomalous findings when he argues that "the two measures of serotonin levels, (in spinal fluid versus in platelets) have opposite relationships to behavior disorders…" because "…the studies of spinal fluid measure the amount of metabolite after serotonin has been released into the synapse between nerve cells and then used…The platelet serotonin studies measure the amount of serotonin still stored inside the platelet—the amount that has not yet been released for communication. Thus, if the communication between cells is poor, this effect would theoretically result in high concentrations of serotonin stored (in neurones or platelet cells) and low concentrations released to be converted into a serotonin metabolite (by synapse or muscle), conceptually resolving the opposite direction of the associations found with the two assays."

consistent predictor of criminal and deviant conduct, of borderline personality disorder, of violent and assaultive behaviors. Remarkably similar results were garnered in a meta-analysis that examined the relationship between a serotonin metabolite (5-HIAA) and antisocial behavior (Moore, Scarpa, and Raine, 2002). Twenty different reports were identified and ultimately chosen for inclusion in the meta-analysis. These studies were then analyzed and the results demonstrated that low levels of 5-HIAA were significantly associated with increased antisocial involvement (effect size=-.45, $p<.05$). These two meta-analyses reveal that low levels of serotonin are related to an increased risk of antisocial and violent behavior (Moore Scarpa, and Raine, 2002; Raine, 1993). However, similar to the dopamine studies, it is not possible to determine whether low serotonin levels cause explosive conduct, or whether levels of serotonin recede after the completion of violence—that is, cause and effect cannot be established based on the available evidence (Pincus, 2001).

Serotonergic Genetic Polymorphisms

With the growing interest in examining the effects of genes on behavior, geneticists have moved away from simply analyzing the association between levels of serotonin and aggression to more advanced analytic techniques that uncover how polymorphisms of the serotonergic system may be related to the etiology of violence. The serotonergic system is comprised of at least seven different serotonin receptors and a number of different serotonin transporter polymorphisms (Murphy, Lerner, Rudnick, Lesch, 2004; Nichols and Sanders-Bush, 2001). Of all the serotonergic polymorphisms, the serotonin transporter gene (5HTT) has been studied the most extensively. Different allelic combinations of this gene have been found to alter the risk of developing a range of diverse psychopathological phenotypes, including antisocial behaviors.

The proceeding discussion will highlight the functional role of the 5HTT gene and will also explore the literature examining the link between 5HTT and different phenotypes.

The Serotonin Transporter Gene (5HTT). The serotonin transporter gene (5HTT) is located at 17q11.1-17q2 on chromosome 17 (Gelernter, Pakstis, and Kidd, 1995; Heils et al., 1996; Ramamoorthy et al., 1993). The 5HTT gene displays a 44 base pair variable number of tandem repeats in the 5' regulatory segment of the gene (Add Health Biomarkers Team, no date; Heils et al., 1996). The two most prominent alleles for Caucasians and African Americans are the 484 base pair allele—referred to as the short variant (i.e., the "s" allele)—and the 528 base pair allele—referred to as the long variant (i.e., the "l" allele). However, the distributions of these alleles show remarkable global and ethnic/racial variation (Allele Frequency Database, 2006; Gelernter et al., 1999; Gelernter, Kranzler, and Cubells, 1997). For instance, Gelernter, Kranzler, and Cubells (1997) analyzed the distribution of the short and long alleles of the 5HTT gene and found that there was significant variation in these alleles across European Americans, African Americans, and Japanese. Just as important is that although geneticists originally thought that only two alleles existed for the 5HTT gene, recent population genetic studies indicate that different alleles—besides the short and long variants—exist in the African American and Japanese populations, but not in the European American population (Gelernter, Kranzler, and Cubells, 1997). These findings point to the need to conduct analyses separately for each racial/ethnic group.

The 5HTT gene has captured the attention of geneticists, in part, because the short and long alleles have different functional properties. The main function of 5HTT is to synthesize the serotonin transporter protein which, in turn, is responsible for terminating serotonergic activity by removing excess serotonin from the synaptic cleft and returning it to the vesicle of the

presynaptic neuron. Remember that this process is referred to as reuptake and reuptake is important for maintaining appropriate levels of serotonin in the synapse—even slight fluctuations in serotonergic concentrations may alter the functioning of the body and the brain. Thus, there is great interest in determining whether different alleles of the 5HTT gene alter the activity of the serotonin transporter protein. For example, there is mounting evidence suggesting that the long allele, in comparison with the short allele, has significantly higher transcriptional activity (Heils et al., 1996), whereas the short allele has been linked with reduced serotonin receptor binding in the brain (David et al., 2005). Levels of the serotonin transporter protein and basal activity have even been found to vary depending on the exact combination of alleles a person possesses (Heils et al., 1996; Lesch et al., 1996). The different alleles of 5HTT, in short, affect genetic expression and have wide-ranging effects on the human body, on the human brain, and on biochemistry—all of which have the potential to explain phenotypic variation (David et al., 2005; Hamer and Copeland, 1998; Heils, et al., 1996; Lesch et al., 1996; Murphy, Lerner, Rudnick, and Lesch, 2004).

In addition, two somewhat disparate bodies of research have pointed to the 5HTT polymorphism as a potential candidate gene for behavioral and mood disorders, psychiatric problems, and various forms of psychopathology (Morley and Hall, 2003; Murphy, Lerner, Rudnick, and Lesch, 2004; Niehoff, 1999; Rowe, 2002). First, given that low serotonin levels contribute to the risk of many disorders, pharmacological drugs have been developed that aim to increase levels of serotonin by targeting the 5HTT gene. This class of medications—selective serotonin reuptake inhibitors (SSRIs)—are often prescribed for patients who are suffering from depression, severe anxiety, and even some personality disorders. SSRIs, such as Prozac, Zoloft, and Paxil, work by altering the reuptake process by blocking the serotonin transporter protein

from removing serotonin from the synaptic cleft (Edwards and Anderson, 1999; Niehoff, 1999). Since the serotonin transporter protein is rendered ineffective by SSRIs, the level of serotonin in the synapse is increased and brought back into the normal range of variation. As a result of the larger quantities of serotonin made available by SSRIs, the symptoms associated with depression and other neuropsychiatric diseases and psychopathological disorders are often alleviated or erased altogether. These medications implicate the 5HTT gene in a range of disorders and hint at the possibility that 5HTT may also be etiologically related to criminal and aggressive behaviors (Niehoff, 1999; Patkar et al., 2002; Rowe, 2002).

Second, and in a similar vein to some of the dopamine literature, animal knockout studies have proven to be especially important in isolating the effects of 5HTT (Holmes, Murphy, and Crawley, 2002, 2003; Kelaï et al., 2003; Mössner et al., 2004). Animal knockout experiments have shown that the 5HTT gene is related to aggression, certain personality traits, and alcohol consumption (Holmes, Murphy, and Crawley, 2002; Kelaï et al., 2003; Mössner et al., 2004). For example, Holmes and associates (2002) compared male 5HTT knockout mice with healthy controls to see if they reacted differently to two different scenarios where they were presented with a stranger mouse. Mice with their 5HTT gene knocked out were slower to attack the stranger mouse and were significantly less likely to initiate an attack than were mice with the 5HTT gene. Similarly, Kelaï et al. (2003) compared alcohol consumption rates between healthy field mice and mice that had had their 5HTT gene knocked out. The results revealed that 5HTT knockout mice consumed significantly less alcohol than the control mice. Taken together, the animal knockout studies provide circumstantial evidence suggesting that the 5HTT gene plays a critical role in behavioral disinhibition (Holmes, Murphy, and Crawley, 2002, 2003; Kelaï et al., 2003; Morley and Hall, 2003)

The findings garnered from animal knockout studies, and the recognition that antidepressant medications target the 5HTT gene, have led to a wealth of research using human subject to examine whether different allelic combinations of 5HTT contribute to the risk of numerous phenotypes. This line of literature has revealed that carriers of the short allele have an increased risk of committing violent suicidal behavior (Courtet et al., 2001), of developing anxiety-related personality traits (Lesch et al., 1996; Melke et al., 2001), of being highly neurotic (Munafò et al., 2003), and even of being diagnosed with autism (Cook et al., 1997). Based off these findings, the short allele of the 5HTT genetic polymorphism has been identified as the risk allele.[12] Even so, some research has failed to detect any effect of the short allele on neuroticism (Willis-Owen et al., 2005), depression (Willis-Owen et al., 2005), obsessive-compulsive disorder (Kim, Lee, and Kim, 2005), and other neurological and psychiatric conditions (Ebstein et al., 1997; Esterling et al., 1998; Kotani et al., 2002).

Most germane to the current research, however, are those studies that have examined whether variants of the 5HTT polymorphism are associated with the creation of antisocial behaviors and personalities. Table 4 includes information about the sample, about the dependent variable, about the research design, and about the effect size for studies that examined the effects of the 5HTT gene on various outcome measures. Six of the fourteen studies (43 percent) in Table 3.4 sought to determine the effect of different variants of 5HTT on ADHD or hyperkinetic disorder. Three of these studies (50 percent) found a significant relationship between 5HTT and ADHD (Beitchman et al., 2003; Manor et al., 2001; Seeger, Schloss, and Schmidt, 2001). These three studies used both case-control and family-linkage research designs, employed samples that

[12] One exception to this general rule is that the long allele has been found to confer a greater risk to sudden infant death syndrome (SIDS). In a recent study, SIDS victims were examined post-mortem and compared with a group of healthy controls (Weese-Mayer et al., 2003). The results revealed that the long allele was significantly more likely to be possessed by SIDS babies. It appears, however, that the long allele is only the risk allele for a small number of rare disorders.

Table 3.4. The Effect of the Serotonin Transporter Gene (5HTT) on Various Outcome Measures

Study	Sample Characteristics	Outcome Measure(s)	Research Design	Effect Size	Findings
Beitchman et al. (2003)	41 aggressive Caucasian children (33 males and 8 females) and 41 nonaggressive controls	Aggression and ADHD	Case-control study	*P*>.05 for aggression; *P*<.05 for aggressive subjects with ADHD	No effect of HTT on aggression; Subjects with one or two copies of the long allele were significantly more likely to be aggressive and have ADHD
Comings et al. (2000a)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=ns for ADHD; r^2=ns for conduct disorder; r^2=ns for ODD	No effect of 5HTT on ODD, CD or ADHD
Comings et al. (2000b)	326 Caucasian subjects were included in the sample	ADHD, ODD, and conduct disorder	Genetic association study	r^2=.010 for ADHD; r^2=.000 for conduct disorder; r^2=.010 for ODD; All r^2s ns	No effect of 5HTT on ADHD, CD, or ODD
Herman et al. (2003)	204 Caucasian college students (57 males and 147 females)	Alcohol consumption	Genetic association study	*P*<.05 for binge-drinking; *P*<.05 for number of drinks consumed; *P*<.05 for drinking to get drunk	Effect of 5HTT on three measures of consumption of alcohol; subjects homozygous for the S allele were significantly more likely to score higher on all three measures

Hopfer et al. (2005)	The Add Health genetic subsample is included in the analysis	Alcoholism	Genetic association study	β=-.04 for wave one (ns); β=.01 for wave two (ns); β=.06 for wave three (ns)	No effect of 5HTT on alcoholism in any of the three waves
Ishikawa et al. (1999)	387 Japanese males were included in the analysis	Cigarette smoking	Genetic association study	OR=1.7	The long allele was found more often in smokers; Subjects that were homozygous for the short allele were less inclined to smoke and more easily able to quit smoking
Kent et al. (2002)	113 ADHD probands	ADHD	Family-linkage study	χ^2=2.25 (*P*>.05)	No effect of 5HTT on ADHD
Manor et al. (2001)	98 families from Jerusalem (n=45 families) and from Tel Aviv (n=53 families) were included in the analysis; information about both parents and their child was collected	ADHD	Family-linkage study	likelihood ratio=9.62	Effect of 5HTT on ADHD; Subjects with long alleles were more likely to be diagnosed with ADHD
Matsushita et al. (2001)	697 male Japanese alcoholics (average age=50.5) and 270 male Japanese nonalcoholic control subjects (mean age=50.0)	Alcohol consumption	Case-control study	χ^2=.004 (ns)	No effect of 5HTT on alcoholism
Munafò et al. (2005a)	511 individuals between the ages of 33 and 73 (237 males and 274 females from the United Kingdom)	Alcohol consumption	Genetic association study	F=3.63	Effect of 5HTT on consumption of alcohol; Subjects with short alleles consumed more alcohol

Munafò et al. (2005b)	141 heavy smokers from Oxfordshire, UK (average age=54.5) were included in the analysis	Nicotine dependence	Genetic association study	F=3.11	Effect of 5HTT on nicotine use; The short allele was more frequent for people who scored high on nicotine dependence
Patkar et al. (2002)	105 cocaine-addicted African-American subjects and 44 African-American controls	Aggression; Impulsivity; Sensation seeking	Case-control study	r=.01 for aggression (ns); r=.14 for impulsivity (ns); r=-.13 for sensation seeking (ns)	No effect of 5HTT on aggression, impulsivity, or sensation seeking
Seeger, Schloss, and Schmidt (2001)	101 inpatients (mean age=12.3) and 163 control group subjects (83 males and 80 females; mean age=11); all of the subjects were Caucasian	Hyperkinetic disorder	Case-control study	χ^2=7.603 for HD with CD; χ^2=9.127 for HD without CD	Effect of 5HTT on HD; subjects homozygous for the long allele had an increased risk of HD
Türker et al. (1998)	713 young adults were included in the analysis	Alcohol consumption	Case-control study	χ^2=7.58	Effect of 5HTT on consumption of alcohol; Subjects homozygous for the short allele had a higher tolerance for alcohol

Notes:
ns = non-significant
OR= odds ratio

consisted of very different demographic characteristics, and operationalized ADHD in a variety of different ways. Interestingly, for all three studies, it was not the short allele that was associated with ADHD, but rather the long allele that conferred a greater chance of developing ADHD. The rather limited number of studies examining the link between 5HTT and ADHD preclude any definitive conclusions about this association.

Table 4 also includes information about five studies that probed the relationship between 5HTT and alcohol consumption. Of these studies, three (60 percent) revealed that carriers of the short allele were more susceptible to consuming large quantities of alcohol and to becoming an alcoholic (Herman et al., 2003; Munafò et al., 2005a; Türker et al., 1998). This alcohol-5HTT association was fairly consistent across different research designs, for males and females, for different age groups, and for samples gathered in different countries (Herman et al., 2003; Munafò et al., 2005a; Türker et al., 1998). The results of these three studies have been recently confirmed in a recent meta-analysis. Feinn, Nellissery, and Kranzler (2005) used meta-analytic techniques to determine empirically whether the short allele of 5HTT was related to alcohol dependence. The results provided additional support upholding the link between 5HTT and alcohol consumption. They found that the short variant of the 5HTT gene had a consistent and robust effect on alcohol dependence across a spread of heterogeneous studies (odds ratio=1.18, $p<.05$).

The remaining studies in Table 4 examined whether variants of the 5HTT gene were linked to nicotine use and to impulsive or aggressive behaviors and personalities. Two of these studies found an effect of 5HTT on nicotine use; however, the results were contradictory. One study found that the short allele was linked to nicotine dependence (Munafò et al., 2005b), whereas the other study found that the long allele was associated with cigarette smoking

(Ishikawa et al., 1999). More research needs to be undertaken before any definitive conclusions can be drawn about how the 5HTT gene may relate to nicotine dependence (Ishikawa et al., 1999; Munafò et al., 2005b). Finally, a number of studies in Table 4 failed to find that the 5HTT gene was related to aggression, impulsivity, oppositional defiance disorder, and conduct disorder (Beitchman et al., 2003; Comings et al., 2000a, 2000b; Patkar et al., 2002). Again, however, the limited number of studies prevents any solid conclusions about whether the 5HTT gene is etiologically related to these types of antisocial behaviors.

Summary of the Serotonergic System

Serotonin is an important neurotransmitter that modulates behaviors and controls impulses (Dowling, 1998; LeDoux, 2002; Kotulak, 1997; Thompson, 1985). When levels of serotonin are out of equilibrium, human judgment becomes impaired, behavior becomes unpredictable and dangerous, and antisocial and psychotic personalities often begin to emerge (Clark and Grunstein, 2000; Coccaro and Kovoussi, 1996; Hamer and Copeland, 1998; Stoff and Vitiello, 1996). Therefore, genetic polymorphisms that are responsible for maintaining a healthy balance of serotonin in the body and brain have been identified as some of the most important genes in the etiology of antisociality (Holmes, Murphy, and Crawley, 2002; Patkar et al., 2002). The 5HTT polymorphism is crucial to regulating serotonergic activity (Murphy, Lerner, Rudnick, and Lesch, 2004). And as was discussed above, research has revealed that different allelic combinations of 5HTT have different impacts on numerous phenotypes, ranging from mild depression to full-blown aggression. However, the relationship between 5HTT and psychopathology is not ubiquitous across all of the studies; some research detects a strong relationship, whereas other studies fail to observe a significant association. Similar to the

dopaminergic literature, future research examining the effects of 5HTT need to use large, prospective, and nationally-representative samples in order to ascertain the effect, if any, that 5HTT has on crime, delinquency, aggression, and drug/alcohol abuse.

Monoamine Oxidase A

Neurotransmitters, such as dopamine and serotonin, are needed in order for neurons to exchange information and communicate with other brain cells (Dowling, 1998; Kotulak, 1997). When neurotransmitters are released in appropriate amounts, and when excessive neurotransmitters are eliminated from the synapse in a timely fashion, the body and brain are able to operate at peak capacity. However, when concentrations of neurotransmitters deviate from normalcy, behavioral disorders may begin to surface, temperaments may change, and other neuropsychological conditions may become evident (Moore, Scarpa, and Raine, 2002; Niehoff, 1999; Raine, 1993; Rowe, 2002). Aberrant levels of neurotransmitters are quite frequently due to some type of problem or interference with the removal of neurotransmitters from the synapse. Recall that after a neuron has released the appropriate group of neurotransmitters, and after these neurotransmitters have crossed the synapse and relayed the message to a neighboring neuron, the neurotransmitters need to be removed from the synaptic cleft. Bear in mind that there are two ways to "mop-up" surplus neurotransmitters. The discussion thus far has focused on the first process, called reuptake. In reuptake, transporter genes (e.g., DAT1 and 5HTT) code for the production of transporter proteins. These transporter proteins then blunt the activity of neurotransmitters by removing the appropriate neurotransmitter (e.g., dopamine or serotonin) from the synapse and returning it to the vesicle of the presynaptic neuron (Dowling, 1989;

Kotulak, 1997; LeDoux, 2002). Reuptake is a critically important process for maintaining proper concentrations of neurotransmitters in the human body and brain.

In addition to the reuptake process, neurotransmitters can also be eliminated from the synaptic cleft through a process referred to as enzymatic degradation (Dowling, 1998; Kotulak, 1997; LeDoux, 2002; Shih and Thompson, 1999; Thompson, 1985). For this method of degradation, enzymes are released into the synaptic gap where they breakdown neurotransmitters into inactive particles. These enzymes essentially destroy neurotransmitters and terminate their activity, thereby regulating the levels of neurotransmitters found in the body. However, if something interferes with the production of these enzymes, the body loses its ability to eliminate neurotransmitters from the synapse quickly, potentially leading to abnormal neurotransmitter levels (Dowling, 1998; Kotulak, 1997; Thompson, 1985). And as was revealed above, when neurotransmitter levels are too high or too low, numerous psychopathologies abound. Thus, the possibility exists that metabolic enzymes—because they are crucial to regulating the activity of neurotransmitters—may play a role in the etiology of certain behavioral and personality disorders (Caspi et al., 2002a; Haberstick et al., 2005; Morley and Hall, 2003).

Equally important, however, are the genes that code for the production of enzymes that catalyze neurotransmitters. Just as different variants of dopaminergic or serotonergic polymorphisms may have unique effects on the production and regulation of neurotransmitters, different variants of enzymatic degradation genes may also have altered effects on breaking down neurotransmitters. As a result, research has begun to explore the way in which neurotransmitter-metabolizing polymorphisms may impact the human body and how these polymorphisms may be related to aggression and violence (Caspi et al., 2002a; Foley et al., 2004; Haberstick et al., 2005; Shih and Thompson, 1999).

One of the most studied and perhaps most important enzymes in the human body is monoamine oxidase A (MAOA). MAOA is responsible for breaking down a number of different neurotransmitters, including dopamine, serotonin, and norepinephrine—all of which have been tied to antisocial behaviors (Niehoff, 1999; Raine, 1993; Rowe, 2002). Levels of these neurotransmitters depend on the activity level of MAOA: if MAOA is overactive, then levels of neurotransmitters may plummet appreciably; on the other hand if MAOA is under-active, then levels of neurotransmitters may rise quickly. Given the central importance of MAOA in the regulation of neurotransmitters, one polymorphism—the monoamine oxidase A promoter gene—that controls the production of MAOA has been identified as a candidate gene in the etiology of antisocial behaviors (Caspi et al., 2002a; Foley et al., 2004; Haberstick et al., 2005; Morley and Hall, 2003; Shih and Thompson, 1999). The proceeding section will provide an overview of the MAOA enzyme and of the MAOA polymorphism and how they both relate to different phenotypes. Particular attention will be paid to the research that has examined the link between different variants of the MAOA gene and antisocial outcomes.

Monoamine Oxidase A Genetic Polymorphisms

Monoamine oxidase A (MAOA) is one of the two major neurotransmitter-metabolizing enzymes found in the human body (Jahng et al., 1997).[13] Although MAOA's main function is to break down and discard neurotransmitters from the synaptic gap, MAOA also plays an integral role in regulating brain activity (Roth, Breakefield, and Castiglione, 1976), in the etiology of addictive substances (Fowler et al., 1996; Simpson et al., 1999), and in a wealth of other functions of the human body (Ellis, 1991; Shih, Chen, and Ridd, 1999; Shih and Thompson,

[13] The second major metabolic enzyme found in humans and other mammalian animals is monoamine oxidase B (MAOB). However, since there has been comparatively very little research that examines the relationship between MAOB and behavioral disorders, this discussion will only review the literature bearing on MAOA.

1999). Even one class of antidepressant medications—monoamine oxidase A inhibitors (MAOA inhibitors)—are frequently prescribed for depression and other mood disorders. These drugs work by blunting the activity level of MAOA, causing an increase in levels of neurotransmitters that often reduces symptoms of depression (Catalano, 1999). Given that MAOA controls neurotransmitter levels, and that certain antidepressants target the activity level of MAOA, geneticists have long suspected that MAOA may also be implicated in the development of a range of other phenotypes, including aggressive and violent behaviors (Shih, Chen, and Ridd, 1999).

However, the driving force behind why the MAOA gene has been singled out as a potentially important polymorphism in the study of crime is because of the results of a study published in *Science* by Brunner and his associates (1993a). Prior to the *Science* article, Brunner et al. (1993b) described the abnormal conduct of mentally-disabled males from a Dutch kindred. These males engaged in a constellation of antisocial behaviors, such as impulsive aggression, attempted rape, arson, and exhibitionism (Brunner et al., 1993a, 1993b). Females from this kindred, however, appeared to be relatively immune to these behavioral disturbances and to any cognitive deficits. Initially, Brunner and colleagues linked these behaviors to a genetic defect on the X chromosome near the genes that code for MAOA and MAOB (Brunner et al., 1993b). Further analyses suggested that MAOB activity was normal in these males, but the MAOA activity was deficient. Follow-up genetic tests on five males inflicted with these behavioral conditions and mental deficiencies revealed that a point mutation in the eighth exon of the MAOA gene was responsible for producing a useless MAOA enzyme (Brunner et al., 1993a). Essentially, males inflicted with this deficiency were unable to produce functioning MAOA enzymes, which accounted for the aberrant conduct of males from this kindred This was one of

the first studies to document a link between genetic variation and phenotypic variation. Subsequent research has followed Brunner et al.'s (1993a, 1993b) lead and examined whether different MAOA polymorphisms are related to criminality (Caspi et al., 2002a; Foley et al., 2004; Haberstick et al., 2005). One of the most promising of these polymorphisms is found in the promoter region of the MAOA gene. To examine more closely the effects of this polymorphism, a detailed discussion of the MAOA gene will be presented, followed by a review of the literature that examines whether different variants of this gene contribute to the development of problem behaviors.

Monoamine Oxidase A Promoter Gene (MAOA-uVNTR). The monoamine oxidase A gene (MAOA) has been mapped to the X chromosome at location Xp11.23-11.4 (Add Health Biomarkers Team, no date; Levy et al., 1989). MAOA 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 is a functional polymorphism that has been shown to affect the transcriptional efficiency of the gene with different alleles corresponding to differences in the activity level of the MAOA enzyme (Balciuniene et al., 2002; Coron et al., 1996; Hotamisligil and Breakefield, 1991; Ito et al., 2003; Sabol, Hu, and Hamer, 1998). The 3.5-, 4-, and 5-repeat alleles have been shown to be the most efficient at transcription, whereas the 3-repeat allele has been shown to be the least efficient (Deckert et al., 1999; Syagailo et al., 2001). Against this backdrop, alleles with 3-repeats or less are usually placed into one group (i.e., the low-activity or "short" group), whereas alleles with more than 3-repeats are generally grouped together (i.e., the high-activity or "long" group) (Caspi et al., 2002a; Haberstick et al., 2005; Lawson et al., 2003). The low-activity alleles are typically considered the risk alleles for antisocial behavior (Caspi et al.,

2002a; Haberstick et al., 2005), but the high-activity alleles periodically have been found to increase the risk of developing conduct problems (Manor et al., 2002).

The 3- and 4-repeat alleles have been found to be the two most common MAOA variants in the human population (Deckert et al., 1999; Koller et al., 2003; Sabol, Hu, and Hamer, 1998). Unlike the dopaminergic and serotonergic polymorphisms, which have been shown to vary greatly across different racial/ethnic groups, relatively little is known about the allelic distributions of the MAOA promoter polymorphism for different geographical regions and for different ancestral lines (Balciuniene et al., 2001). However, there is some circumstantial evidence suggesting that the frequency of MAOA alleles may vary slightly for different races and ethnicities (Balciuniene et al., 2001; Gilad et al., 2002; Sarich and Miele, 2004). As for now, though, the lack of available data precludes any definitive conclusions about whether or not the distribution of MAOA alleles fluctuates for different racial/ethnic categories.

Since the MAOA gene is located on the X chromosome, males have only one copy of the gene (i.e., one allele) and are thus considered hemizygous for the MAOA polymorphism. Females, on the other hand, have two X chromosomes and therefore have two copies of the gene (i.e., two alleles). The X-linked MAOA polymorphism usually forces researchers to examine either only males or only females or to examine them separately (Caspi et al., 2002a; Haberstick et al., 2005; Samochowiec et al., 1999). And most studies have focused solely on how the MAOA gene impacts males (Beitchman et al., 2004; Caspi et al., 2002a; Haberstick et al., 2005; Lu et al., 2003; Manuck et al., 2000). Nonetheless, studies have found the MAOA polymorphism to affect a range of behaviors both for males and for females (Ito et al., 2003; Manor et al., 2002).

The MAOA gene also impacts males and females quite differently. If, for example, a male has a deficient MAOA allele, then they are unable to manufacture a functional MAOA enzyme; however, females, since they have two copies of the gene, are able to manufacture a functioning MAOA enzyme as long as just one of their alleles is not defective. This is precisely why Brunner et al. (1993a, 1993b) found that males—but not females—from the Dutch kindred exhibited signs of behavioral problems and had very low mental capabilities.

In addition to the Brunner et al. studies (1993a, 1993b), experiments using nonhuman subjects were also very important in pinpointing the polymorphisms on the MAOA gene that were related to enzymatic activity and to behavioral disturbances (Cases et al., 1995; Shih, Chen, and Ridd, 1999; Newman et al., 2005; Shih and Thompson, 1999). For example, Shih, Chen, and Ridd (1999) conducted a large review of the literature that examined the effect of knocking out the MAOA gene in mice. These researchers concluded that mice with their MAOA gene knocked out had higher levels of dopamine, serotonin, and norepinephrine and also resorted to aggression much more frequently than mice with a functioning MAOA gene (see also Cases et al., 1999). These aggressive behavioral patterns closely parallel those observed for humans who have an ineffective MAOA gene and suggest that MAOA alleles may have differential effects on human phenotypes, especially violence and aggression (Caspi et al., 2002a; Foley et al., 2004; Haberstick et al., 2005; Morley and Hall, 2003; Shih, Chen, and Ridd, 1999; Shih and Thompson, 1999).

Indeed, a rapidly growing line of literature has investigated whether the MAOA gene has effects on various antisocial outcome measures, including antisocial conduct. Table 3.5 includes the results of these studies and also contains information about the sample characteristics, about the dependent variable, about the research design, about the effect size, and about the findings.

Table 3.5. The Effect of the Monoamine Oxidase A Promoter Gene (MAOA) on Various Outcome Measures

Study	Sample Characteristics	Outcome Measure(s)	Research Design	Effect Size	Findings
Beitchman et al. (2004)	50 male subjects (average age=9.5) and a control group of ethnically-matched male adults were included in the sample	Aggression	Case-control study	χ^2=6.000	Effect of MAOA on aggression; Longer alleles were positively related to aggression
Ito et al. (2003)	504 Japanese outpatients (217 males and 287 females) were included in the sample	Cigarette smoking	Genetic association study	aOR=ns for males; aOR=.49 for females	No effect of MAOA on smoking for males, but MAOA related to smoking for females
Koller et al. (2003)	169 male German alcoholics (average age=41.8) and 72 control subjects were included in the sample	Aggression; Impulsiveness	Case-control study	OR=1.273 for aggression (ns); OR=.983 for impulsiveness (ns)	No effect of MAOA on aggression or on impulsiveness
Lawson et al. (2003)	171 Caucasian children (153 males and 18 females; average age=9.1 years old) with ADHD, their mothers, and their fathers; An additional 173 control subjects were also included in the sample	ADHD	Family-linkage and case-control study	χ^2=.58 for TDT (ns)	No effect of MAOA on ADHD
Lu et al. (2002)	214 Chinese Han male alcoholics and 77 control subjects were included in the sample	Alcoholism	Case-control study	*P*=ns (values not reported)	No effect of MAOA on alcoholism
Lu et al. (2003)	129 Chinese Han males and 77 control subjects were included in the sample	Antisocial personality disorder	Case-control study	χ^2=2.94 (ns)	No effect of MAOA on antisocial personality disorder
Manor et al. (2002)	133 families from Tel-Aviv were included in the sample	ADHD	Family-linkage study	χ^2=4.37	Longer alleles related to ADHD

Manuck et al. (2000)	110 white males were included in the sample (mean age=45.2)	Aggression; Impulsivity	Genetic association study	*P*<.015 for aggressiveness and for impulsivity	Effect of MAOA on aggression and impulsivity
Parsian et al. (2003)	134 Caucasian alcoholics (104 males and 30 females) and 89 control subjects (47 males and 42 females) were included in the sample	Alcoholism	Case-control study	χ^2=5.87 (ns)	No effect of MAOA on alcoholism
Samochowiec et al. (1999)	303 alcoholic males, 59 alcoholic males with antisocial personality disorder, and 185 healthy male control subjects	Antisocial personality	Case-control study	χ^2=4.645	Effect of MAOA on antisociality for alcohol-dependent males; The low-activity allele increased antisocial behaviors
Schmidt et al. (2000)	298 male alcoholics and 66 female alcoholics along with 182 control males and 180 control females were included in the sample	Antisocial personality	Case-control study	χ^2=3.2 for males; *P*>.05 for females (values not reported)	Shorter alleles related to antisocial traits for males, but not for females
Zammit et al. (2004)	346 schizophrenics were included in the sample (details not provided)	Aggression	Genetic association study	χ^2=.9 (ns)	No effect of MAOA on aggression

Notes:
ns = non-significant
aOR = adjusted odds ratio
OR = odds ratio
TDT = transmission disequilibrium test (Speilmen et al., 1993)

Four of the twelve studies in Table 5 examined how the MAOA polymorphism was related to measures of aggression. Of these studies, two (50 percent) found a statistically significant relationship between MAOA and aggression.[14] These significant findings were garnered using different analytical strategies and two very different samples: one sample consisted of young boys (Beitchman et al., 2004) and the other sample included middle-aged men (Manuck et al., 2000). Similar to the dopaminergic and serotonergic research, the small number of studies hampers the ability to draw any conclusions about the association between MAOA and aggression.

Table 5 also shows that two of the three studies that used antisocial personality disorder as the dependent variable found a significant relationship with MAOA. In contrast to the findings for aggression, these two studies revealed that shorter alleles were associated with increases in antisocial personalities for males and for females (Samochowiec et al., 1999; Schmidt et al., 2000). Both studies used a case-control research design and both studies used samples that consisted of alcoholic subjects. Future research is needed to determine whether these findings are robust enough to be observed in other samples, using different research designs.

The remaining five studies presented in Table 5 examined the effect of MAOA on alcoholism, ADHD, and cigarette smoking. Neither of the two alcoholism studies found a significant MAOA effect (Lu et al., 2002; Parsian et al., 2003), while only one (Manor et al., 2002) of the two (50 percent) ADHD studies detected a significant association between MAOA

[14] The two studies that found a significant effect of MAOA were conducted by Beitchman et al. (2004) and by Manuck et al. (2000). Beitchman et al. (2004) found that longer alleles increased the risk of aggression. The findings for the Manuck et al. (2000) article, however, were not so straightforward. They categorized the MAOA alleles into two groups. The first group consisted of the 2- and 3-repeat alleles and the second group was made-up of the 1- and 4-repeat alleles. The results suggested that males who had intermediate allele lengths (i.e., the 2- and 3-repeat alleles) had lower aggression scores than males with the 1- or 4-repeat alleles.

and attention problems. The lone study that employed cigarette smoking as the outcome measure found that the MAOA gene was related to nicotine addiction for females, but not for males (Ito et al., 2003). Taken together, the research in Table 5 reveals that the MAOA gene has effects on a range of deviant outcomes, but these effects are not necessarily persistent across all of the studies. Replication studies need to be conducted in order to provide more empirical evidence about the relationship between MAOA and antisocial behavior (Caspi et al., 2002a; Foley et al., 2004; Haberstick et al., 2005).

Summary of Monoamine Oxidase A

The MAOA enzyme is crucial to normal life functioning because it works to modulate levels of dopamine, serotonin, and norepinephrine. When the MAOA enzyme is under-active, or when the MAOA enzyme is overactive, levels of these neurotransmitters may wax and wane in ways that are not considered normal. And irregular neurotransmitters may affect an array of phenotypes such as behavioral disorders and numerous neuropsychiatric conditions (Kotulak, 1997; Moore, Scarpa, and Raine, 2002; Raine, 1993; Rowe, 2002). As a result, the MAOA enzyme has been implicated in the etiology of violence (Caspi et al., 2002a; Foley et al., 2004; Haberstick et al., 2005; Morley and Hall, 2003; Shih and Thompson, 1999). At the same time, different polymorphisms of the MAOA promoter gene have been found to produce MAOA enzymes that differ in their activity levels (Balciuniene et al., 2002; Coron et al., 1996; Hotamisligil and Breakefield, 1991; Ito et al., 2003; Sabol, Hu, and Hamer, 1998). Research has thus moved towards examining whether the MAOA polymorphism is related to delinquent and criminal conduct (see, for example, Manuck et al., 2000). The preceding section discussed the results of these studies and showed that they are somewhat ambiguous. Some of the research

revealed that the MAOA gene does contribute to the development of problem behaviors and antisocial personality traits; other research, however, failed to observe a significant association (see Table 5). Future research exploring the nexus between MAOA polymorphisms and phenotypic variation is needed to shed light on whether or not MAOA confers a genetic susceptibility to antisocial behavior (Shih and Thompson, 1999).

Research Questions

The preceding discussion highlighted the research examining the effects that different genetic polymorphisms had on a number of antisocial behaviors. The results of these studies suggested that dopaminergic, serotonergic, and MAOA genetic polymorphisms may contribute to the development of crime and delinquency. Recall, however, that these studies are host to a number of different methodological and statistical shortcomings. These limitations make it difficult to determine whether the genetic polymorphisms are implicated in the etiology of antisocial behaviors. This dissertation addresses some of these limitations and is built around examining three different research questions.

***Research Question One*: Do the dopaminergic, serotonergic, and MAOA genetic polymorphisms exert a direct effect on a range of antisocial outcomes?**

Findings from molecular genetic research reveal that some genetic polymorphisms contribute directly to the development of certain forms of psychopathology (see discussion above). However, the effect sizes of these genetic polymorphisms tend to be small in magnitude, typically explaining no more than 6 percent of the variation in an outcome measure (Rutter, 2006). Part of the reason for why the effect sizes are small is because most behaviors and personalities are created by a number of different genes—that is, they are polygenic (Comings, 1998). Based off this work, it is hypothesized that the dopaminergic, serotonergic, and MAOA

genetic polymorphisms will exert relatively small direct effects on antisocial behaviors and, in most cases, the direct effects will not be statistically significant.

***Research Question Two*: Do the dopaminergic, serotonergic, and MAOA genetic polymorphisms have indirect effects on a range of antisocial outcomes?**

There is a growing consensus among behavioral geneticists that genes and the environment are inextricably tied together. According to this perspective, genetic forces work to place individuals in environments that reinforce their genetic tendencies—that is, a gene X environment correlation (rGE). There is also evidence suggesting that personality development is largely guided by genetic influences. Taken together, there is good reason to believe that the dopaminergic, serotonergic, and MAOA genetic polymorphisms will operate indirectly through the environment and certain personality characteristics. A vast amount of theoretical work underscores the importance of passive, evocative, and active rGEs (Rutter, 2006; Walsh, 2002). Drawing from this work, it is hypothesized that the genetic polymorphisms will be related to measures of the family functioning, delinquent peer group association, and measures of individual differences.

***Research Question Three*: Do the dopaminergic, serotonergic, and MAOA genetic polymorphisms interact with the social environment to predict involvement in antisocial activities?**

One of the more fascinating ways that genes can contribute to phenotypic variation is by interacting with environmental influences—that is, a gene X environment interaction (GxE). Keep in mind that for GxEs, genetic effects are only detected when a particular allele is paired with a particular environmental condition. The genetic effect, therefore, is contingent on, or conditioned by, the environment (or vice versa). Studies examining GxEs have been particularly promising, suggesting that GxEs are important in the etiology of crime and delinquency (Beaver and Wright, 2005; Caspi et al., 2002a; Rutter, 2006). Indeed, one of the most important studies

published in *Science* in 2002 revealed a GxE in the creation of antisocial behavior (Caspi et al., 2002a). Accordingly, it is hypothesized that the dopaminergic, serotonergic, and MAOA genetic polymorphisms will interact with the social environment to predict involvement in antisocial behaviors. Based off previous research (e.g., Caspi et al., 2002a), it is further hypothesized that the genetic effects will be much stronger in the GxE statistical models when compared with the direct effects models.

CHAPTER 4

METHODS

The previous two chapters outlined the research bearing on the genetic origins of human behaviors and personality development. The preceding chapter also set forth a number of hypotheses that will be tested. Recall that that the main purpose of this dissertation is to examine the development of criminal and delinquent behavior from a biosocial perspective. The major obstacle for biosocial criminologists, however, is the lack of available datasets that include measures of biological/genetic factors and measures of environmental influences. One exception to this general rule is The National Longitudinal Study of Adolescent Health (Add Health). This chapter will provide an in-depth description of the Add Health data and the measures that will be used. In addition, a plan of analysis that details the precise statistical approach to analyzing the data will also be laid out.

The National Longitudinal Study of Adolescent Health

Data come from the National Longitudinal Study of Adolescent Health (Add Health), the largest prospective, nationally representative, and longitudinal study of American adolescents in grades seven through twelve (Udry, 2003). The Add Health study has been conducted over three different waves and spans a total of seven years (Harris et al., 2003). The first wave of data was collected between September 1994 and December 1995 when respondents were between the ages of 11 and 19 years old. Approximately one to two years later, the second wave of data was collected. The third wave of questionnaires were administered between 2001 and 2002 when most of the Add Health participants were between the ages of 18 and 26 years old. As detailed

below, information about the respondents was gathered through an in-school survey and an in-home survey. Before discussing the different waves of data and the different components (i.e., in-school and in-home surveys) of the Add Health study, a description of the research design and sampling techniques will be presented.

Research and Sampling Design

Participants for Add Health were selected through the use of a multistage stratified random sampling procedure (Chantala, 2003; Harris et al., 2003). The initial sampling frame consisted of 26,666 public and private high schools with an eleventh grade and with an enrollment of at least thirty students. These high schools were then stratified into clusters based on enrollment size (<125, 126-350, 351-775, or >775), school type (public, private, or parochial), geographic region (Northeast, Midwest, South, or West), urbanicity (urban, suburban, or rural), and percentage of white students (0, 1-66, 67-93, or 94-100) (Tourangeau and Shin, 1999). Schools were then sorted by these clusters and systematic sampling techniques were used to select a final sample of 80 high schools. Of the original 80 high schools that were asked to participate, 52 agreed to take part in the study, while 28 refused to participate. Twenty-eight additional schools were then selected to replace the schools that declined to take part in the study (Tourangeau and Shin, 1999). In total, 80 high schools were chosen for inclusion in the Add Health study.

Administrators from each high school were then asked to supply a list of junior high or middle schools which usually send at least five incoming students to their high school (Harris et al., 2003). These schools were referred to as "feeder" schools and one feeder school for each of the 80 high schools without a seventh or eighth grade was recruited to participate in the study.

The feeder school's probability of being selected into the sample was directly proportionate to the school's percentage contribution of incoming freshmen. For example, if 75 percent of the incoming students for a particular high school were contributed by Feeder School A, then Feeder School A would have a .75 chance of being selected. Fifty-six feeder schools were recruited to participate and four declined the invitation (Tourangeau and Shin, 1999). Overall, 80 high schools and 52 middle and junior high schools were included in the Add Health study (N=132 schools).

The Three Waves of Data

Wave I In-School Interview. Students attending the selected 132 high schools and feeder schools were eligible to complete the wave I in-school survey of the Add Health data. Between September 1994 and April 1995, an Add Health team of researchers administered questionnaires during a selected class period to all students in attendance that had permission of their parents to participate in the study. The survey included questions requesting information to an array of topics pertaining to the student's home life, relationship patterns, sexual behaviors, peer groups, and individual demographic information. The self-report instrument was designed to be completed in a 45 to 60 minute time frame. Overall, 90,118 students submitted completed in-school questionnaires.

Wave I In-Home Interview. A subsample of the original 90,118 students was also selected to be included in the wave I in-home component of the Add Health study. This subsample was designed to be a nationally representative cross-section of seventh- through twelfth-grade students. Only those adolescents attending 1 of the 132 high schools/feeder schools and who were listed on school enrollment rosters for the 1994-1995 academic year were

eligible for the in-home sample. The school rosters were then stratified by gender and by current grade level. Nearly 17 percent of all students in each stratum were contacted and asked to participate. Overall, 20,745 youths completed questionnaires between April 1995 and December 1995 and were included in the wave I in-home Add Health sample (Harris et al., 2003). The wave I in-home sample contained detailed questions indexing the youth's delinquent activities, friendship networks, risky behaviors, relationships, and school activities.

Most of the wave I in-home interviews typically lasted between one and two hours and were conducted in the confines of the adolescent's home. However, instead of using the traditional paper-and-pencil based self-report survey format, Computer Assisted Personal Interviewing (CAPI) was employed to expedite the data collection process. With CAPI, interviewers key the respondent's answers to questions directly onto a laptop/portable computer. CAPI was not appropriate for all of the topics covered in the interview. In particular, the sensitive nature of some questions, especially those asking about sexual behaviors and delinquent conduct, necessitated the use of a different data-collection strategy. For these personal questions, Audio Computer Assisted Self-Interviewing (ACASI)—a computer-based self-guiding interview tool—was used. Unlike the CAPI, where questions are read aloud and responses to the questions are verbally articulated from interviewee to interviewer, ACASI uses headphones that are connected to a computer to gain responses to sensitive questions. Specifically, the respondent listens to a series of prerecorded questions on headphones and then enters directly their answers onto the computer. The use of ACASI is supposed to encourage the interviewee to respond truthfully to sensitive questions (Udry, 1998).

The wave I in-home data also contained supplementary information about the adolescent as reported on by one of the respondent's parents (usually the mother). The paper-and-pencil

based parent questionnaire was shorter than the adolescent in-home interview and was typically completed in 40 minutes or less. A range of topics were covered in the parent interview including items relating to neighborhood characteristics, household dynamics, economic conditions, and the parent-adolescent relationship. In total, 17,700 parents completed the wave I parent questionnaire (Harris et al., 2003).

Genetically-related siblings were oversampled for inclusion in the wave I in-home component (Harris et al., 2003; Tourangeau and Shin, 1999). The genetic subsample contains monozygotic twins (MZ), dizygotic twins (DZ), full siblings (FS), half siblings (HS), and non-related youths (NR) (e.g., stepsiblings). During the wave I in-school and in-home interviews, respondents were asked to indicate whether they had an MZ twin or a DZ twin. If they responded affirmatively, then their cotwin was added to the genetic subsample (n=2,658 twins; n=1,329 twin pairs). In addition, for the wave I in-school survey, participants were asked to list any household members who were in grades 7 through 12. For each person listed, information about the person's sex and biological mother and biological father was also requested. Based off this information, additional genetically-related siblings were added to the sample. Specifically, 208 non-twin siblings of twins, 1,611 full siblings, 1,177 half siblings, and 491 adolescents living in the same household but who did not share a biological mother or a biological father were included in the genetic subsample of the Add Health data (Harris et al., 2003; Tourangeau and Shin, 1999).

Wave II In-Home Interview. Wave II in-home data collection efforts were undertaken between April and August 1996 when follow-up questionnaires were administered to approximately 71 percent of the original in-home sample (Harris et al., 2003). All of the siblings comprising the genetic subsample were re-interviewed for the wave II in-home survey. An

additional 65 siblings, who were not part of the original wave I genetic sample, were added to the data and interviewed at wave II. Respondents who were enrolled in twelfth grade during wave I were not included in the wave II sample. Similarly, during wave I, disabled individuals were oversampled, but were subsequently dropped from the wave II in-home sample. The structure and format of the wave II in-home surveys were very similar to the wave I interviews. CAPIs were used for most of the questions, but items that were sensitive were administered by ACASI. Overall, 14,738 adolescents participated in the wave II in-home survey and provided information about relationships, delinquency, alcohol and drug use, sexual behaviors, and peer groups.

Wave III In-Home Interview. The wave III in-home interviews were administered between July 2001 and April 2002. All wave I in-home respondents were eligible to participate in the wave III in-home interview except for those who were currently outside of the United States. Importantly, great efforts were taken to re-interview Add Health respondents who were residing in correctional facilities. Given that most of the original Add Heaths respondents were 18 to 26 years old at wave III, the questions used at waves I and II would no longer be valid ways to gain important information about the interviewees. The wave III questionnaires were thus amended to include topics germane to young adults. For example, interviewees were asked questions pertaining to marriage, employment, criminal history, and pregnancy/childrearing. In addition, a number of retrospective questions measuring different domains of childhood were included to gain insight into the interviewee's upbringing. Over 15,000 wave I participants were located and agreed to participate in the wave III in-home interview. The average interview lasted 134 minutes and was administered by CAPI or by ACASI (Harris et al., 2003).

Wave III DNA Subsample

One of the unique aspects of the Add Health data is that DNA information was collected from a subsample of participants at wave III. Wave I respondents who also had a participating sibling or cotwin in the study (i.e., they comprised the genetic subsample; see above description) were eligible for inclusion in the DNA subsample. At wave III, 3,787 siblings were identified, contacted, and asked if they would be willing to provide samples of their saliva for genotyping. The DNA analysis targeted for screening the following six different candidate polymorphisms: a dopamine transporter gene (DAT1), two dopamine receptor genes (DRD2 and DRD4), a serotonin transporter gene (5-HTT), cytochrome P450, and monoamine oxidase A (MAOA). Participants of the DNA sample were required to read and eventually sign an informed consent sheet. They were also told that they would not be provided with any incentives for providing DNA samples. Altogether, 2,574 individuals agreed to participate and submitted buccal cells for genetic typing and analysis (Add Health Biomarker Team, no date).

DNA Extraction Procedures

To collect buccal cells for DNA analysis, participants were asked to rub the inside of their cheeks and their gums with a cytology brush for 20 seconds. The end of the cytology brush was then inserted into a 2 ml screw cap tube holding 200 μl of lysis buffer (1 percent isopropyl alcohol [v/v] in 50 mM Tris-HCl, 1 mM EDTA and 1 percent sodium dodecyl sulfate, pH 8.0). Participants then rinsed 10 ml of 4 percent sucrose in their mouths. After 30 seconds, they next emptied the sucrose rinse into a 50 ml conical test tube. The tube was then sealed with parafilm and the contents of the tube were referred to as "wash 1." A second mouth rinse was conducted following the exact same process and was referred to as "wash 2." The tubes containing the two

washes and the cytology brush tip were tagged and prepared for shipment to the University of Arizona laboratory in ice to sustain a temperature of 4˚C (Add Health Biomarker Team, no date).

DNA samples were prepared for analysis under the direction of David C. Rowe at the University of Arizona. The first step was to separate genomic DNA from buccal cells. The brush and washes (wash 1 and wash 2) were analyzed individually, but later were combined for subsequent genetic analyses. The first day of preparing the DNA samples entailed adding 1 ml of lysis buffer (6 M guanidine-HCl, 100 mM Tris-HCl and 10 mM EDTA, pH 7.5) and 25 µl of proteinase K (10mg/ml) to each tube containing the cytology brush head. The tubes were then placed in a 55˚C rotator for the duration of one night (Add Health Biomarker Team, no date).

On the same day the swabs were being prepared, the two washes were combined together and centrifuged for 10 minutes at a rate of 1,800 revolutions per minute at room temperature. Upon completion the supernatant was removed and 1 ml of lysis buffer was added to the remaining pellet. Pellet DNA was then placed into a new 2 ml tube and 25 µl of proteinase K (10mg/ml) was poured into the container. The samples were transferred into a 55˚C rotating incubator and remained there overnight (Add Health Biomarker Team, no date).

The next day the cytology brush heads were extracted from the test tubes. Then 200 µl of binding matrix (10 mM sodium acetate and .1g/ml diatomaceous earth [Sigma] in lysis buffer) was added to the brush heads and to the tubes containing the combined washes. The wash tubes were transferred to a rotator where they were left for 15 minutes at room temperature. The tubes were then centrifuged at maximum speed for 2 minutes and the supernatant fluid was removed. 1 ml of wash buffer (50 percent ethanol [v/v] in 400 mM sodium chloride, 20 mM Tris-HCl and 2 mM EDTA, pH 7.5) was combined with the pellet DNA remaining in the bottom of the tube. The tubes were again placed on a rotator for 15 minutes at room temperature and then

immediately centrifuged for 2 minutes at maximum speed. The supernatant fluid was disposed and the resulting pellet DNA was vacuum dried overnight (Add Health Biomarker Team, no date).

On the third day, 200 µl of elution buffer (10 mM Tris-HCl, .1 mM EDTA, pH 8.8) was added to each dried pellet DNA. The tubes containing the pellet DNA and elution buffer and the tubes containing the wash solution were then transferred into a rotating incubator for 30 minutes and centrifuged at maximum velocity for 2 minutes. The supernatant fluids from each person's cytology brush tube and their wash tube were collected, combined together, and stored in a .5 ml tube. On average, 58 ± 1 µg of DNA was extracted for each person. Finally, DNA samples were sent to the Institute for Behavioral Genetics at the University of Colorado for genotyping (Add Health Biomarker Team, no date).

Genotyping the Dopaminergic, Serotonergic, and MAOA Polymorphisms

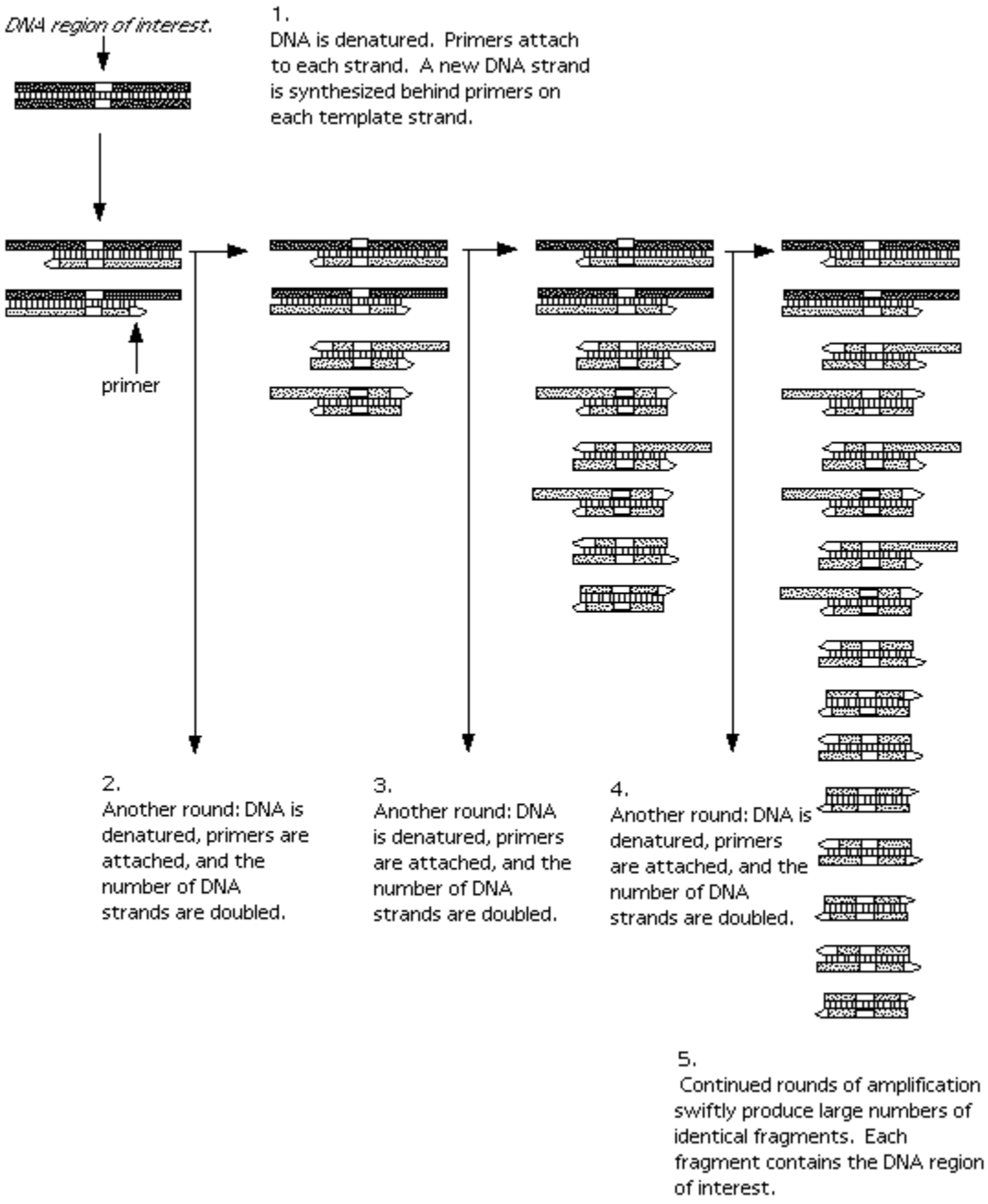
The University of Colorado laboratory used polymerase chain reaction (PCR) techniques to genotype the DNA samples for DAT1, DRD2, DRD4, 5HTT, and MAOA. The purpose of PCR is to increase the quantity of a fragment of DNA from one template copy to millions or billions of duplicate copies. The manufacturing of identical copies of a specific section of DNA is necessary to decode a sequence of nucleotides. And more copies of a small piece of DNA result in more accurate and more reliable coding of the allelic combination of genetic polymorphisms. Before providing an explanation of PCR, a brief discussion of how organisms copy their DNA is in order.

Most living organisms duplicate their DNA in a similar manner. When a cell begins to divide, the two coils of DNA detach from each other. After they detach, polymerase—an

enzyme that duplicates genetic material—makes replica of each DNA strand. However, DNA polymerases must be provided with a short sequence of nucleotides to "prime," or begin the duplication process. Thus each cell contains another enzyme, primase (i.e., the primer), that replicates the first few nucleotides of a DNA segment and sets the duplication process into motion. Once the primer is in place, polymerase is able to replicate the remaining DNA sequence.

PCR works by paralleling closely the duplication process that occurs naturally in living organisms. Before the PCR process can begin, a sequence of target DNA (that will be replicated), an unlimited number of the four nucleotides (A, C, T, and G), a predetermined primer sequence, and Taq polymerase (the polymerase) must be placed into a single test tube. Once the test tube is prepared with the necessary components, PCR proceeds in three interrelated steps. As shown in Figure 4.1, first the two tightly coiled strands of DNA must somehow be separated into two single strands. When DNA is heated to 90˚C-96˚C, the two strands of DNA detach from each other. The first step in PCR entails heating the genetic material and thereby "unzipping" the DNA. This process is known as *denaturation*. However, primers are unable to bind to the separated chains of DNA at such high temperatures and therefore the contents of the vial must cool to 55˚C. Once the vial reaches this temperature, the primers anneal (bind) to each of the strands of DNA. The cooling of the vial and the binding of the primers to the DNA strands is the second step of PCR, and is called *hybridization* or *annealing*. The third and final step of PCR is *extension* and consists of reheating the vial. The Taq polymerase works best when the contents of the vial reach 75˚C. Once the target temperature of 75˚C is achieved, the Taq polymerase starts with the primer and begins to decode the remaining segment of DNA by adding the appropriate complementary nucleotide to make an exact replica copy of each DNA

Figure 4.1. Visual Depiction of the Polymerase Chain Reaction Process



Note:
Available online at http://web.mit.edu/esgbio/www/rdna/graphics/pcr.gif

strand. For example, if a particular sequence of one separated strand of DNA is ACTGA, then the Taq polymerase working in unison with the nucleotides and the primer would display the complementary nucleotide arrangement, TGACT. Nucleotides continue to be added for the remainder of the entire DNA sequence. Once the DNA strand has been copied completely, the first PCR cycle is completed.

At the end of the first cycle, two exact duplicate copies of DNA are available. Each of the two copies consists of one original strand of DNA and a manufactured complementary strand. Usually, numerous PCR cycles are conducted, and with each successive cycle the number of DNA strands decoded increases exponentially. For example, after the second cycle four DNA strands are duplicated; yet after 30 cycles a billion copies will be made! The average time to finish one PCR cycle is between 1 to 3 minutes.

After all of the PCR cycles are complete, electrophoresis is used to examine allelic length differences. Electrophoresis works by taking advantage of the fact that DNA is a negatively-charged molecule. When the copies of DNA are placed in agarose gel (a chain of sugar molecules from seaweed), and electrodes are activated, the longer DNA sequences take more time to navigate through the agarose gel. By examining the arrangement of DNA in the agarose gel, short alleles can be distinguished from long alleles. As a result, electrophoresis provides specific information about the allele length of different segments of DNA and also allows researchers the opportunity to decipher the nucleotide sequence for genetic polymorphisms, including DAT1, DRD2, DRD4, 5HTT, and MAOA.

Dopamine Transporter Gene (DAT1). The main function of the dopamine transporter gene (DAT1) is to blunt dopaminergic activity in the synapse by facilitating the reuptake of dopamine back into the presynaptic terminals. The DAT1 polymorphism has a 40 base pair

variable number of tandem repeats (VNTRs). DAT1 was amplified by using the following primer sequences: forward, 5'-TGTGGTGTAGGGAACGGCCTGAG-3' (fluorescently labeled), and reverse, 5'-CTTCCTGGAGGTCACGGCTCAAGG-3' (Add Health Biomarkers Team, no date). This method resulted in PCR products of 320 (6-repeat allele), 360 (7-repeat allele), 400 (8-repeat allele), 440 (9-repeat allele), 480 (10-repeat allele), and 520 (11-repeat allele) base pairs.

Dopamine Receptor Gene (DRD2). The dopamine D2 receptor gene (DRD2) encodes for the production of the D2 receptor. DRD2 has a polymorphic TaqI restriction endonuclease site approximately 2,500 base pairs downstream (3' untranslated region) from the coding section of the gene. The A1 allele has a point mutation C → T (TCGA to TTGA) that erases the TaqI site, but is considered a nonfunctioning polymorphism. Geneticists working at the Institute for Behavioral Genetics at the University of Colorado originated an SNP assay by employing the Applied Biosystem's "Taqman© Assays by Design™ for SNP Genotyping Service" (Add Health Biomarkers Team, no date; Haberstick and Smolen, 2004). To genotype the DRD2 TaqI polymorphism, the following primers and probes were used: forward primer, 5'-GTGCAGCTCACTCCATCCT-3', reverse primer, 5'-GCAACACAGCCATCCTCAAAG-3', probe 1, 5'VIC-CCTGCCT***T***GACCAGC-NFQMGB-3' and probe 2, 5'-FAM-CTGCCT***C***GACCAGC-NFQMCB-3' (Add Health Biomarkers Team, no date). The DRD2 polymorphisms were scored by two independent observers.

Dopamine Receptor Gene (DRD4). The dopamine D4 receptor gene (DRD4) is a highly polymorphic gene that consists of a 48 base pair VNTR that can be repeated 2 to 11 times, although the 2, 4, and 7 are the most common alleles (Add Health Biomarkers Team, no date). The DRD4 gene was amplified by using the two proceeding primer sequences: forward, 5'-

AGGACCCTCATGGCCTTG-3' (fluorescently labeled), and reverse, 5'-GCGACTACGTGGTCTACTCG-3' (Add Health Biomarkers Team, no date). This assay resulted in PCR products of 379, 427, 475 (4-repeat allele), 523, 571, 619 (7-repeat allele), 667, 715, 763, and 811 base pairs (Add Health Biomarkers Team, no date).

Serotonin Transporter Gene (5HTT). The serotonin transporter gene (5HTT) has a 44 base pair variable number of tandem repeats in the 5' section of the gene (Add Health Biomarkers Team, no date; Heils et al., 1996). The assay used to genotype the 5HTT polymorphism was a variant of the method developed by Lesch et al. (1996). The 5HTT gene was amplified by using the following primer sequences: forward, 5'-GGCGTTGCCGCTCTGAATGC-3' (fluorescently labeled), and reverse, 5'-GAGGGACTGAGCTGGACAACCAC-3' (Add Health Biomarkers Team, no date). This procedure resulted in PCR products of 484 (short) or 528 (long) base pairs.

Monoamine Oxidase A (MAOA-uVNTR). The monoamine oxidase A (MAOA) polymorphism contains a 30 base pair variable number of tandem repeats in the 5' regulatory section of the gene (Samochowiec et al., 1999). The assay used to genotype the MAOA polymorphisms was a variant (Haberstick et al., 2005) of the method developed and used by Sabol, Hu, and Hamer (1998). Primer sequences were: forward, 5'ACAGCCTGACCG-TGGAGAAG-3' (fluorescently labeled), and reverse, 5'-GAACGTGACGCTCCATTCGGA-3' (Add Health Biomarkers Team, no date). Genotypes were scored by two independent raters. This assay resulted in PCR products of 291 (2-repeat allele), 321 (3-repeat allele), 336 (3.5-repeat allele), 351 (4-repeat allele), and 381 (5-repeat allele) base pairs (Add Health Biomarkers Team, no date).

Analytical Sample

Most empirical studies that use the Add Health sample employ the public-use date file (see, for example, Beaver and Wright, 2005; Bellair, Roscigno, and McNulty, 2003). Because of the potential problem of deductive disclosure of the respondents' identities, the widely available public-use version of the Add Health data contain information for only a subset of respondents (Harris et al., 2003). Approximately one-half of the core sample was randomly selected for inclusion in the public Add Health data file. The public-use version contains information about respondents collected from in-home interviews with the adolescent and with one of their parents. A total of 4,882 participants were followed in the public Add Health sample from wave I through wave III. Unfortunately, the public-use data file does not include any measures pertaining to the genetic subsample, such as genetic relatedness between siblings (e.g., MZ, DZ, full sibling, or half sibling) or DNA markers (Harris et al., 2003).

Given that the thrust of the current study centers on the effects of specific genetic polymorphisms in the etiology of antisocial behavior, the public-use version of the Add Health would not be an appropriate sample to use. Fortunately, Add Health does allow certified researchers to obtain restricted-use data files that contain highly sensitive information, such as the results of DNA tests. To gain access to the restricted-use Add Health sample, however, requires a contractual agreement with the Carolina Population Center/University of North Carolina at Chapel Hill. The contract provides details about how to store the data, how to destroy statistical output, and who is allowed to access the data. Moreover, the contract also was reviewed and approved by The Institutional Review Board—Social and Behavioral Sciences at the University of Cincinnati and by The Add Health Team.

The Carolina Population Center granted me access to a restricted-use Add Health data file that contains genetically-sensitive information for a subset of participants. The genetic Add Health subsample includes 2,574 respondents followed longitudinally at wave I, wave II, and wave III. Detailed information about the respondents was garnered through self-report questionnaires at all three waves and parental interviews at wave I (see discussion above). All of the variables that are available in the public-use Add Health sample are also contained in the genetic subsample. In addition, the restricted Add Health sample contains variables tapping the participant's genetic relatedness with their sibling (e.g., MZ twin, DZ twin, full sibling, half sibling, and unrelated siblings). Most importantly for the current study, however, is that the results of the DNA tests revealing each individual's allelic combinations for five different genetic polymorphisms are included in the restricted sample. This dissertation uses data from the restricted-use Add Health genetic subsample.

Population genetics research indicates that the allelic combinations of dopaminergic and serotonergic genetic polymorphisms vary considerably across different racial and ethnic groups (Allele Frequency Database, 2006; Chang et al., 1996; Chen, Burton, Greenberger, and Dmitrieva, 1999; Ding et al. 2000; Ding et al., 2002; Gelernter et al., 1998; Gelernter et al., 1999; Gelernter, Kranzler, and Cubells, 1997; Harpending and Cochran, 2002; Kang, Palmatier, and Kidd, 1999; Sarich and Miele, 2004). In statistical analyses the effects of the genetic polymorphisms may be masked if different racial minority groups, such as Asians, Hispanics, and African Americans, are combined into a single "nonwhite" category (Cardon and Palmer, 2003). The analytical data set thus includes only those respondents who self-reported they were either white (n=1,592) or African American (n=431); all other racial categories were removed

from the final data set. The inclusion of only whites and African Americans resulted in two relatively homogenous groups for data analysis.

In addition, DNA analyses were, in some cases, performed for both twins of MZ twin pairs. The problem with including both twins from the same MZ twin pair in genetic analyses is that they share 100 percent of their DNA, essentially double-counting each twin. In line with pervious research analyzing the genetic Add Health subsample (Haberstick et al., 2005), one MZ twin from every MZ twin pair was removed from the sample. With this selection criteria in place, and after deleting missing cases, the final analytic sample is N=2,023.

Measures

Genetic Polymorphisms

The central goal of this dissertation is to examine the effects of different genetic polymorphisms on various measures of antisocial behavior. The most conventional way of coding genetic polymorphisms for statistical analyses is by determining the number of risk alleles that a person possesses (see, for example, Hopfer et al., 2005). Keep in mind that most genes are created from two alleles: one inherited maternally and one inherited paternally. Every person, therefore, has the potential to have zero risk alleles, one risk allele, or two risk alleles. In the Add Health data, two variables—one corresponding to each of the two alleles that makeup a gene—are available for most of the genetic polymorphisms. The variables provide information about the number of base pairs and the repeat sequence of each allele. Based off this information, the variables can then be used to determine whether the person is a carrier of the specified risk allele. If the respondent possessed the risk allele, then the variable was assigned a score of "1"; if they do not possess the risk allele, then the variable was scored "0." This same

coding scheme was used for both of the variables (i.e., one for each allele) that correspond to one polymorphism. These variables were then added together to form an overall "risk allele" measure. Scores on the genetic polymorphism measures range between zero and two, with the value indicating the number of risk alleles for the polymorphism.

It is noteworthy to point out that statistical models may not always detect a significant association between a genetic polymorphism and a phenotype even though they may be etiologically related (i.e., a type II error). There are at least three potential reasons for this problem. First, many genetic polymorphisms are controlled by promoter genes. Promoter genes control and regulate the expression of other genes by "turning them on" or "turning them off." So, even if two people have the exact same polymorphism, the effects of the polymorphism may differ quite drastically depending on the promoter genes inherited by each person (Ridley, 2003). For example, in one person, the promoter gene may have triggered the activation of the polymorphism, whereas the promoter gene in the other person may have kept the polymorphism from exerting its effects. Without measuring promoter genes, it is difficult or nearly impossible to determine whether the polymorphism is active or dormant.

Second, most phenotypes are polygenic—that is, they are created from numerous genes acting together. As a result, most genes account for only a small percentage of variance in any given phenotype (Rutter, 2006). Sometimes the percentage of variance accounted for by a polymorphism may be too small to be detected in statistical analyses, especially in studies using small sample sizes. Of course, it would be a mistake to assume that just because a gene is not statistically related to a phenotype that that gene does not have an effect on the phenotype. Instead, great caution needs to be exercised when examining the impact of genes on

behaviors/personalities because sometimes the genetic effect may be too small to be captured by statistical models.

Third, because of different splicing schemes, the same gene may produce different proteins in two different people. Recall that proteins are the means by which genes ultimately affect behaviors and other phenotypes. A certain polymorphism may code for the production of one protein for one person, but that exact same polymorphism may code for the production of a different protein in another person (Ridley, 2003). Essentially, two identical genes may produce two different proteins that impact behaviors in quite different ways. If so, then the measurement of the genetic polymorphisms contains a large amount of error—error that artificially deflates the coefficients of the genetic measures. Taken together, the effects of genes may be suppressed when using standard analytical techniques, such as OLS.

Dopamine Transporter Gene (DAT1). Prior research has established that the 10-repeat allele (10R) of the dopamine transporter gene (DAT1) is the risk allele because it heightens the susceptibility to a number of different antisocial outcomes (Barr et al., 2001; Comings et al., 2001; Gill et al., 1997; Mill et al., 2005). Typically, genetic researchers compare whether there are differences between carriers of the 10R allele and carriers of the 9-repeat allele (9R). The assay used by the Add Health Biomarkers Team to genotype the DAT1 polymorphism resulted in six different PCR products, with two of them corresponding to the 9R and 10R alleles. The two alleles were then inspected to determine whether the 10R allele was present. If it was, then the variable was assigned a score of "1"; if not it was scored "0." The two variables (each corresponding to one allele) were then added together to create a scale that indexed the number of 10R alleles (i.e., risk alleles) that each person possesses. In line with previous literature using

the Add Health sample, participants who had an allele other than a 9R or a 10R were removed from the analytical sample (Hopfer et al., 2005).

Dopamine Receptor Gene (DRD2). There are two different alleles—the A1 allele and the A2 allele—that can makeup the dopamine D2 receptor polymorphism (DRD2). In general, the A1 allele is considered the risk allele to a number of behavioral and psychiatric disorders (Arinami et al., 1993; Berman et al., 2002; Blum et al., 1997a; Comings et al., 2001; Connor et al., 2002; Hopfer et al., 2005). The Applied Biosystem's "Taqman© Assays by Design™ for SNP Genotyping Service" was used to genotype the TaqI site on the DRD2 polymorphism (Add Health Biomarkers Team, no date; Haberstick and Smolen, 2004). The variables were recoded so the A2 allele corresponded to a score of "0" and the A1 allele corresponded to a score of "1." The DRD2 genetic polymorphism measure indicates how many risk alleles were present for each respondent.

Dopamine Receptor Gene (DRD4). The dopamine D4 receptor gene (DRD4) is one of the most examined polymorphisms in genetic research (Faraone et al., 1999; Faraone, Doyle, Mick, and Biederman, 2001). Findings from these studies indicate that the 7-repeat allele is the risk allele for a variety of psychopathologies (Faraone et al., 1999; Faraone, Doyle, Mick, and Biederman, 2001). The Add Health Biomarkers Team used an assay that created ten different PCR products. Following prior research using the Add Health data (Hopfer et al., 2005), alleles that had repeat sequences less than 7 were assigned a value of "0"; alleles that had repeat sequences greater than or equal to 7 were assigned a value of "1." Higher scores on the DRD4 scale represent more risk alleles.

Serotonin Transporter Gene (5HTT). The short allele (484 base pairs) of the serotonin transporter gene (5HTT) is usually considered the risk allele (Munafò et al., 2005b; Türker et al.,

1998); however, some research has found the long allele (528 base pairs) also to increase the risk of developing certain disorders (Beitchman et al., 2003; Seeger, Schloss, and Schmidt, 2001). The Add Health Biomarkers Team used an assay similar to the one developed by Lesch et al. (1996) to genotype the 5HTT polymorphism. The long allele was assigned a value of "0" and the short allele was assigned a value of "1." Similar to the other polymorphism measures, the scores for the two alleles were summed to create a risk allele index for 5HTT.

Monoamine Oxidase A Promoter Gene (MAOA). The low-activity allele, when compared with the high-activity allele, is usually identified as the risk allele (Caspi et al., 2001; Foley et al., 2004). Five different PCR fragment lengths were available in the assay process used by the Add Health Biomarkers Team. Consistent with prior research using the Add Health data (Haberstick et al., 2005), the 2- and 3-repeat alleles were categorized as the low-activity alleles and assigned a value of "1" and the remaining three fragment lengths (3.5-, 4-, and 5-repeat alleles) were assigned a score of "0."

The monoamine oxidase A gene (MAOA) is located on the X-chromosome. Recall that males have one X-chromosome and one Y-chromosome, whereas females have two X-chromosomes. The coding strategy for females is exactly the same as the one used for the other polymorphisms and the value for the MAOA scale indicates the number of low-activity alleles that the female inherited. However, the MAOA coding scheme for males is slightly different than the one used for females. Since MAOA is located on the X-chromosome, and since males have only one X-chromosome, they also only have one MAOA allele. Therefore, for males, the MAOA scale simply reflects whether they have the low-activity allele or the high-activity allele.

Environmental Measures

To examine the close interplay between the environment and an individual's genotype, measures of delinquent peers and family adversity were developed from the Add Health data. By including these environmental variables along with the genetic polymorphism scales in the same statistical models, it is possible to examine whether gene X environment interactions (GxEs) and gene X environment correlations (rGEs) are implicated in the development of offending behaviors. From a biosocial criminology standpoint, GxEs and rGEs are two of the most important and most promising ways of examining how the environment and genes work together to produce crime and criminality (Raine, 1993, 2002; Walsh, 2002).

Delinquent Peers. One of the strongest and most robust correlates to crime and delinquency is associating with delinquent friends (Warr, 2002). Measures of delinquent peers have been found to be predictive of a wide range of antisocial behaviors across different samples, across different time periods, and using different analytical strategies (Akers, 1998; Haynie, 2001; Haynie, 2002; Matsueda and Anderson, 1998; Warr, 1996, 2002). Just as revealing is that contact with criminal peers has the potential to explain not only why some people engage in crime, but also why some people, even after fairly lengthy periods of delinquent involvement, begin to desist from crime (Giordano, Cernkovich, and Holland, 2003). For example, changes in the amount of time spent with antisocial associates have been able to account, at least partially, for why people desist from delinquency and drug use (Maume, Ousey, and Beaver, 2005; Warr, 1998). The link between antisocial peers and misconduct is so well-established and so consistently replicated that Warr (2002:40) has boldly contended that "few, if any, empirical regularities in criminology have been documented as often or over as long a period of time as the association between delinquency and delinquent friends."

Research has thus established a strong association between antisocial friends and criminal involvement. At the same time, some research suggests that peer groups may largely be the reflection of an individual's genetic makeup (Scarr, 1992; Scarr and McCartney, 1983). To take these findings into account, a measure of delinquent peers was created. Past research using the Add Health data has employed a three-item scale that indexes an adolescent's delinquent peer network (Beaver and Wright, 2005; Bellair, Roscigno, and McNulty, 2003). At wave I, respondents were asked how many of their three closest friends smoked at least one cigarette a day, drank alcohol at least once a month, and smoked pot at least once a month. Responses to these three questions were then summed together to form a measure of delinquent peers (alpha=.76).[15]

The delinquent peers measure only includes questions pertaining to those friends who engage in relatively minor acts of misconduct. An optimal measure would have included items that measure the spectrum of peers' involvement in delinquent activities. However, caution should be exercised before dismissing the delinquent peers measure as invalid. Prior research using this scale has established its predictive validity; the pattern of correlations observed with the delinquent peers measure are similar to those using alternative measures of antisocial peers (Beaver and Wright, 2005; Bellair, Roscigno, and McNulty, 2003). For example, in a recent analysis of the Add Health data, the three-item delinquent peers measure was the strongest predictor of delinquent involvement in an OLS equation that controlled for a number of biological, psychological, and sociological variables (Beaver and Wright, 2005).

Family Risk. Family-level explanations of crime and delinquency—especially those that focus on parents—have dominated mainstream criminological thought (Gottfredson and Hirschi, 1990; Loeber and Stouthamer-Loeber, 1986; Patterson, 1982). A wealth of empirical evidence

[15] Lists of the items used in each of the scales are available in Appendix A.

suggests that parents play at least some role in the creation of antisocial behaviors and personalities (Laub and Sampson, 1988; Loeber and Stouthamer-Loeber, 1986). There is, however, a small but growing line of literature revealing that once genetic influences are controlled, parents have little effect on their children's personality development and on their children's behavioral patterns (Cohen, 1999; Harris, 1995, 1998; Pinker, 2002; Rowe, 1994; Wright and Beaver, 2005).

To examine the effect that parents may have on their children, three different measures of family risk were created that index various dimensions of the mother-offspring relationship. The first scale, maternal attachment, assesses the emotional closeness of the mother and her adolescent. In line with prior research using the Add Health data (Haynie, 2001; Schreck, Fisher, and Miller, 2004), two items reported on by the adolescent during wave 1 were included in the maternal attachment scale (alpha=.64). Specifically, the adolescent was asked how close they felt to their mother and how much they thought their mother cares about them. Responses to these two items were then summed together, with higher scores indicating more maternal attachment.

A maternal involvement scale was also developed to determine the extent to which the mother engaged in a variety of activities with their child. During wave I interviews, the adolescent was presented with a list of different activities, such as shopping, playing a sport, going to a movie, play, or sporting event, talking about a personal problem, and working on a project for school. They were then asked to indicate which activities they had completed with their mother in the past four weeks. Those activities that the adolescent responded to affirmatively were assigned a value of 1; otherwise they were coded 0. Similar to the scale used

by Crosnoe and Elder (2004), the maternal involvement scale was created from ten different activities reported on by the adolescent (alpha=.55).[16]

Finally, five different questions, reported on by the adolescent at wave 1, were used to create the maternal disengagement scale (alpha=.84). This scale tapped whether the adolescent's mother was cold and withdrawn. Adolescents, for example, were asked whether they are satisfied with the way their mother communicates with them.

All of the scales were then recoded such that higher scores represented less maternal attachment, less maternal involvement, and more maternal disengagement (i.e., higher scores indicated more family risk). To create a composite family risk measure, the items composing the three scales were then factor analyzed. The analysis and inspection of the scree plot indicated that the three family measures could be accounted for by a single factor. The regression factor scores were then calculated from the factor analysis to create an interval level scale measuring global family risk. This scale was scored such that high values indicated elevated levels of family risk.

Control Variables

Age. Given the strong relationship between age and delinquent involvement (Farrington, 1986; Gottfredson and Hirschi, 1990; Hirschi and Gottfredson, 1983), the respondent's age is included as a control variable. Age is continuous variable measured in years.

[16] The maternal attachment scale is identical to the scales used by Haynie (2001) and by Schreck, Fisher, and Miller (2004). The maternal involvement scale is similar to the scale used by Crosnoe and Elder (2004), except we only included items pertaining to the mother's involvement in their adolescent's life; however, because of the large number of missing cases, we were unable to include items that asked about the adolescent's father. Similar to prior research using the Add Health data, the maternal attachment and the maternal involvement scales have moderate alpha values. Nonetheless, we calculated bivariate correlations for the maternal attachment scale, the maternal involvement scale, and the delinquent peers measure. The matrixes revealed significant correlations ($p<.05$) between both of the maternal scales and the delinquent peers scale. Thus the maternal attachment and maternal involvement scales used in the current analysis not only have face validity, but also have predictive validity.

Gender. Research has consistently found gender to be a significant predictor of crime and delinquency (Gottfredson and Hirschi, 1990). Specifically, males are much more likely than females to engage in most types of law-breaking behaviors, but especially serious violent acts. To take this gender gap in offending behaviors into account, a variable tapping the respondent's gender was coded as a dichotomous dummy variable (0=female; 1=male).

Race. Add Health participants were asked to self-identify their racial background. Only those respondents who indicated they were either white or black were included in the final analytic sample. Race is included as a dichotomous dummy variable (0=white; 1=black).

Cognitive Complexity. Low intellectual capacity is a relatively consistent predictor of crime and delinquency (Herrnstein and Murray, 1994; Hirschi and Hindelang, 1977; McGloin and Pratt, 2003; McGloin, Pratt, and Maahs, 2004; Wilson and Herrnstein, 1985). To control partially for intelligence, a cognitive complexity measure is included in the analyses. At wave III, respondents completed the Peabody Picture Vocabulary Test (PPVT), which is a norm-referenced and standardized assessment of verbal ability. The cognitive complexity measure is a continuous variable with low scores representing low levels of verbal intelligence.

Dependent Variables

Seven different dependent variables will be employed to determine if the effects of the genetic polymorphisms are ubiquitous across different types of law-breaking behaviors. Three of the outcome measures are delinquency scales, two of the outcome measures tap the respondent's contact with the criminal justice system, and two of the outcome measures index drug and alcohol abuse. Together, these dependent variables index some of the most common and most serious behaviors that antisocial individuals display.

Delinquency at Wave I. During wave I interviews, respondents were asked to indicate how many times in the past year they had engaged in fifteen different delinquent activities. These items tapped into a variety of different antisocial behaviors, including lying, fighting, and stealing. The response set for the questions about delinquent involvement was as follows: 0=never, 1=one or two times, 2=three or four times, 3=five or more times. Responses to these questions were then summed together to form the wave I delinquency scale (alpha=.78). Past research using the Add Health data has used similar delinquency scales (Beaver and Wright, 2005).

Delinquency at Wave II. Information pertaining to the respondent's involvement in delinquency was also garnered at wave II. The items making up the wave II delinquency scale are almost exactly identical to those used to create the wave I delinquency scale.[17] The wave II delinquency scale was created by adding fourteen different items that indexed the adolescent's misconduct in the past year (alpha=.79). Similar to the wave I delinquency scale items, answers to each question were coded as 0=never, 1=one or two times, 2=three or four times, 3=five or more times. Higher scores on the wave II delinquency scale represent a greater involvement in law-breaking behaviors.

Delinquency at Wave III. The Add Health data also included measures of delinquent and criminal behavior at the wave III interviews. Twelve different items were summed together to create the delinquency scale for this wave of data (alpha=.71). Recall, however, that at wave III the respondents ranged in age from 18-26 years old. As a result, some of the questions that were asked to the respondents at wave I and wave II were no longer valid or appropriate ways to index criminal behavior later in life. Thus, some of the questions that were used previously were

[17] The one exception is that the wave I delinquency scale included a question asking how many times in the past 12 months had the respondent hurt someone badly enough to need bandages or care from a doctor or nurse? This item was not available in the wave II data.

replaced with items that were more age-appropriate. For example, instead of asking the respondent how many times they ran away from home in the past year (which were asked at waves I and II), they were asked if they had deliberately written a bad check in the past year. The wave III delinquency scale provides the opportunity to examine the correlates to offending in young adulthood.

Number of Police Contacts. The three delinquency scales are a useful way to examine the frequency with which the respondents engage in a wide array of delinquent acts. However, delinquency is a relatively common and quite normal experience for adolescents (Moffitt, 1993). Therefore, using a variable that measures the number of police contacts an individual has had in their lifetime helps to delineate chronic offending from transitory delinquency that is confined to adolescence. At wave III, Add Health participants were asked to divulge how many times they had been stopped or detained by the police in their life. The number of police contacts measure is a continuous variable with higher scores representing more encounters with the police.

Ever Arrested. A measure tapping whether the respondent had ever been arrested was also included as a dependent variable in the analysis. This variable builds off of the number of police contacts measure, and helps to identify those offenders who have been caught for serious offenses. This one-item measure was constructed from answers at the wave III interview. Interviewees were first screened by asking how many times they had been detained by the police. If they responded in the affirmative, then they were asked a follow-up question about whether they were arrested or taken into custody by the police. If they indicated that they had been arrested, they were assigned a value of "1" for the ever arrested variable; if not, this variable was coded with a value of "0."

Marijuana Use. Many studies have examined whether genetic polymorphisms are related to drug abuse and drug addiction. To explore this possibility, and to examine whether GxEs may also be able to explain why some people become addicted to illegal substances, a one-item marijuana use variable was developed. Although some may view marijuana use as a relatively innocuous activity, it is important to point out that measures of marijuana use have been used quite extensively in the criminological literature (see, for example, Maume, Ousey, and Beaver, 2005; Warr, 1998). At wave I, adolescents were asked to indicate how many times in the past 30 days they had used marijuana. The marijuana use variable is a continuous measure that indexes the frequency of marijuana use in the past month.

Alcohol Abuse. A recent report by the United States Federal Government estimates that nearly 36 percent of offenders had consumed alcohol prior to committing their criminal offense (Greenfeld, 1998). Given the close correspondence between alcohol consumption and acts of serious violence, an alcohol abuse scale was developed from wave III of the Add Health data. Eight different items tapping into problems that resulted from drinking alcohol were summed together to form the alcohol abuse scale (alpha=.77). For example, respondents were asked how often in the past year they had problems with their friends because they had been drinking. The response set for the alcohol abuse items were as follows: 0=never, 1=once, 2=twice, 3=three or four times, and 4=five or more times. Higher scores on this scale indicate more problems associated with consuming alcohol.

Plan of Analysis

The analysis for this dissertation will proceed in a series of incremental steps. Three different models will be calculated for each of the seven dependent variables: 1) a direct effects

model, 2) an indirect effects model, and 3) an interactive effects model. For the direct effects models, ordinary least squares (OLS) regression techniques will be employed for the wave I, wave II, and wave III delinquency scales. In addition, OLS will also be used for the alcohol abuse scale. Negative binomial regression will be used for the frequency of marijuana use scale and for the number of police contacts scale because these two measures are severely skewed (skewness statistic=32.32 for marijuana use; skewness statistic=2.89 for number of police contacts). Finally, binary logistic regression will be used for the ever arrested measure because it is a dichotomous variable. Moreover, all of the models will be estimated separately for the dopaminergic genes, the serotonergic gene, and for the MAOA gene.

The statistical models will first be calculated for the dopaminergic polymorphisms. Two different direct effects models will be estimated for each dependent variable. In order to preserve degrees of freedom and in order to isolate the effects of the socialization variables, the first direct effects model will include the three dopaminergic measures, all of the control variables (age, gender, race, and cognitive complexity), and the delinquent peers measure. These models will provide estimates of the direct and independent effects that the dopaminergic polymorphisms have on the different outcome measures, net of the effects of delinquent peers and of the key control variables. Since distributions of the dopaminergic polymorphisms vary significantly across people of different racial and ethnic groups (Allele Frequency Database, 2006; Chang et al., 1996; Chen, Burton, Greenberger, and Dmitrieva, 1999; Ding et al. 2000; Ding et al., 2002; Gelernter et al., 1998; Harpending and Cochran, 2002; Kang, Palmatier, and Kidd, 1999; Wang et al., 2004), statistical analyses must be calculated separately for each racial category to avoid population stratification (Cardon and Palmer, 2003). Thus, all of the models

will be calculated for the full sample, for black males, for black females, for white males, and for white females.

The direct effects models will provide important information about how the genetic polymorphisms directly influence different antisocial behaviors. However, there is good reason to believe that genes may also exert their effects indirectly through environments—a process that is referred to as gene X environment correlations (rGEs) (Moffitt, 2005; Rutter, 2006; Scarr, 1992; Scarr and McCartney, 1983). To explore the potential effects of rGEs, the delinquent peers measure and the cognitive complexity measure will be used as dependent variables in two separate OLS regression equations. The dopaminergic genes will be entered into the OLS models as predictor variables to determine whether they are intertwined with the formation of peer groups and with cognitive capabilities. The results of these models will reveal whether dopaminergic polymorphisms are related to the formation of antisocial peers. Given that offenders tend to have lower cognitive abilities than nonoffenders (Herrnstein and Murray, 1994; Hirschi and Hindelang, 1977; McGloin and Pratt, 2003; McGloin, Pratt, and Maahs, 2004; Wilson and Herrnstein, 1985), the indirect models will also examine the effects that DAT1, DRD2, and DRD4 have on cognitive complexity. Similar to the direct effects models, all of the equations for the indirect effects models will be broken-down by gender and race (Cardon and Palmer, 2003).

The final model—the interactive effects models—will be used to determine whether gene X environment interactions (GxEs) are predictive of the seven different dependent variables. To test for GxEs, the measure of delinquent peers was transformed into a dichotomous variable. Those respondents who indicated that they had either zero or one delinquent friends (i.e., they had a score of "0" or "1" on the delinquent peers measure) were placed into one group. The

remaining Add Health participants—those who had more than one delinquent friends—were placed into the other group. The analyses then will follow the some format used in the direct effects model, except the delinquent peers measure will no longer a predictor variable. Instead, the analyses will first be calculated only for those respondents who had 0-1 delinquent friends. Then the analyses will be conducted for the group who indicated they had 2 or more delinquent friends. The patterns of results will then be examined for GxEs. Specifically, if the regression coefficients for a particular dopaminergic gene differ significantly between the two groups, then an interaction between delinquent peers and the dopamanergic gene will have been detected. In essence, when estimating regression models by delinquent peer status, the results will reveal whether or not the effect of the dopaminergic genes depend upon, or are conditioned by, the number of antisocial friends the respondent associates with. If there are not any significant differences in the coefficients between the two groups, then statistically speaking, there is not an interaction between the dopaminergic polymorphisms and delinquent peers in the creation of antisocial behaviors. Again, all of the models will be calculated separately by race and gender.

A second set of models will also be calculated using the family risk scale instead of the delinquent peers measure. Besides this change in predictor variables, the analyses will be identical. The direct effects models, for example, will again be calculated for all of the seven dependent variables, and will be predicted with the dopaminergic polymorphisms and all of the control variables; however, the family risk scale will be used in place of the delinquent peers scale. These models will be calculated for the full sample, black males, black females, white males, and white females.

A series of indirect effects models will also be estimated by using the family risk scale as the dependent variable in an OLS regression model. The dopaminergic genes will be used as

predictor variables to examine if the level of family risk is partially determined by the allelic combinations of the dopaminergic genes. Similar to the models with the delinquent peers measures, these models will provide an empirical test for rGEs.

The final models will examine whether the dopaminergic polymorphisms interact with the family risk scale in the etiology of criminal and delinquent acts. In so doing, the family risk scale will be transformed into a dichotomous variable by dividing it at the mean; values below or equal to the mean were coded 0 and values above the mean were coded as 1. Respondents with a score of 0 were classified as living in low-risk families, whereas those with a score of 1 were categorized as living in high-risk families. One equation will be calculated for those individuals who are classified as residing in high-risk families and another equation will be calculated for individuals in low-risk families. Essentially, these models assess whether the dopaminergic polymorphisms interact with family risk to predict antisocial outcomes (i.e., a gene X environment interaction). All analyses will be conducted for the full sample and by the race and gender subsamples.

The exact same analytical strategy will be used for the serotonin transporter gene (5HTT) and for the monoamine oxidase A gene (MAOA). However, because MAOA is located on the X-chromosome, and because the MAOA measure is calculated slightly different for males and for females, the analyses will not be conducted for the full sample, but will only be calculated by race and gender. Taken together, these models will provide a fairly exhaustive examination of the potential ways that genetic polymorphisms can directly, indirectly, and interactively affect a variety of different antisocial outcomes across a long swath of the life course.

CHAPTER 5

FINDINGS

The previous chapter outlined the methodological and statistical strategies that will be employed to examine the biosocial contributors to antisocial behavior. In the current chapter, the statistical analyses will be conducted, and the results of the multivariate equations will be introduced. To facilitate the presentation of results, this chapter will be divided into three different sections. The first section will describe the direct, indirect, and interactive effects that the dopaminergic polymorphisms had on the seven dependent measures. The second section will discuss the results of the direct, indirect, and interactive statistical models garnered for the serotonin transporter gene (5HTT). Finally, the last section will reveal the direct, indirect, and interactive findings for monoamine oxidase A (MAOA).

The Dopaminergic Polymorphisms

To provide a comprehensive examination of the effects of the dopaminergic polymorphisms (DAT1, DRD2, and DRD4) in the etiology of misconduct, the following seven measures of antisocial behavior will be used as dependent variables in the analyses: wave I delinquency scale, wave II delinquency scale, wave III delinquency scale, number of police contacts, arrest status, frequency of marijuana use, and alcohol abuse. For each dependent variable, a direct effects model and a series of interactive models will be calculated. The interactive models will examine whether the effects of the genetic polymorphisms are conditioned by the social environment (peers and family)—that is, a gene X environment interaction (GxE). Lastly, gene X environment correlations (rGE) will be estimated by

examining the effects that the dopamine genes have on measures of the environment. As discussed in chapter 4, all of the models will be calculated twice: the first set of models will incorporate the measure of delinquent peers and the second set of models will introduce the measure of family diversity. All of the analyses will be conducted for the full sample of respondents and separately for gender and race subcategories.

Wave I Delinquency Scale

The analysis begins by examining the direct effects of the dopaminergic genes and the measure of delinquent peers on the wave I delinquency scale. Table 5.1 contains the results of for these models estimated by employing ordinary least squares (OLS) regression equations for the full sample, for white males, for white females, for black males, and for black females. Across all of the models, the measure of delinquent peers is the strongest and most consistent predictor of the wave I delinquency scale. However, some important findings surface for the dopamine genes, too. For white females and black males, the DRD4 gene maintains a significant and positive association with wave I delinquency. In addition, DRD2 is positively related to the dependent variable for black males, but DRD2 is negatively related to the wave I delinquency scale for black females.

Next, to determine whether the effects of the dopamine genes are conditioned by delinquent peers status, the OLS equations will be calculated separately for the low delinquent peers group and the high delinquent peers group. Table 5.2 depicts the findings for the low delinquent peers group. Across all of the race and gender subcategories, none of the dopamine genes are significantly related to the wave I delinquency scale. As shown in Table 5.3, a

Table 5.1. The Direct Effects of Dopamine Genes and Delinquent Peers on Delinquency at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.17	-.02	-.14	-.02	-.30	-.04	-.22	-.02	.35	.04
	(.18)		(.31)		(.25)		(.70)		(.56)	
DRD2	-.06	-.01	-.04	-.00	-.07	-.01	1.24	.14*	-1.04	-.16**
	(.17)		(.30)		(.25)		(.64)		(.42)	
DRD4	.15	.02	-.45	-.05	.52	.07**	1.15	.12*	.03	.00
	(.18)		(.31)		(.25)		(.67)		(.46)	
Socialization Variable										
Delinquent peers	.89	.46**	.97	.48**	.82	.47**	.96	.43**	.71	.36**
	(.04)		(.07)		(.06)		(.17)		(.13)	
Control Variables										
Age	-.47	-.16**	-.46	-.14**	-.48	-.17**	-.19	-.05	-.66	-.25**
	(.07)		(.11)		(.09)		(.26)		(.18)	
Cognitive complexity	.01	.03	-.00	-.01	.01	.03	.10	.18**	.01	.02
	(.01)		(.02)		(.02)		(.04)		(.02)	
Race	.70	.06**								
	(.28)									
Gender	1.17	.12**								
	(.21)									
R-squared	.21		.21		.21		.22		.18	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.2. The Effects of Dopamine Genes on Delinquency at Wave I for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.07	-.01	.04	.01	-.38	-.08	.22	.03	.39	.07
	(.18)		(.29)		(.23)		(.87)		(.57)	
DRD2	-.13	-.03	-.20	-.04	.06	.01	-.29	-.04	-.66	-.14
	(.18)		(.32)		(.23)		(.88)		(.44)	
DRD4	.15	.03	-.19	-.03	.34	.07	-.51	-.06	.71	.15
	(.18)		(.32)		(.24)		(.99)		(.45)	
Control Variables										
Age	-.12	-.06*	-.17	-.09	-.08	-.05	.11	.04	-.20	-.11
	(.06)		(.11)		(.08)		(.31)		(.18)	
Cognitive complexity	-.01	-.04	-.03	-.08	-.02	-.07	-.01	-.03	.03	.10
	(.01)		(.02)		(.02)		(.05)		(.03)	
Race	.59	.08**								
	(.28)									
Gender	.82	.13**								
	(.22)									
R-squared	.03		.02		.02		.01		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.3. The Effects of Dopamine Genes on Delinquency at Wave I for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.07	-.01	.10	.01	-.16	-.02	.18	.02	-.05	-.01
	(.33)		(.57)		(.47)		(1.2)		(1.1)	
DRD2	.02	.00	.07	.01	-.02	-.00	2.21	.23**	-1.14	-.20**
	(.31)		(.52)		(.47)		(1.0)		(.77)	
DRD4	.29	.03	-.37	-.03	.53	.06	2.28	.24**	-.33	-.04
	(.32)		(.55)		(.47)		(1.0)		(.92)	
Control Variables										
Age	-.61	-.16**	-.48	-.12**	-.72	-.19**	.53	.12	-1.06	-.32**
	(.13)		(.21)		(.18)		(.51)		(.34)	
Cognitive complexity	.02	.03	.01	.02	.01	.02	.22	.31**	-.03	-.08
	(.02)		(.04)		(.03)		(.08)		(.04)	
Race	.09	.01								
	(.54)									
Gender	1.58	.13**								
	(.38)									
R-squared	.04		.02		.04		.18		.16	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

different pattern of results emerges for the analysis conducted for the high delinquent peers group. For black males, DRD2 and DRD4 are significantly and positively related to the wave I delinquency scale. These results suggest that there is a GxE between DRD2 and delinquent peers and between DRD4 and delinquent peers in the creation of delinquency for black males. At the same time, DRD2 is significantly and negatively related to wave I delinquency for black females. Again, this finding suggests that the effects of DRD2 only surface for black females in the high delinquent peers group.

The next set of analyses are identical to the first three tables, except they include the family adversity scale instead of the delinquent peers measure. As shown in Table 5.4, none of the dopamine genes have a significant direct effect on the wave I delinquency scale when controlling for family risk. In Table 5.5 the equations are an exact duplicate as those in Table 5.4, except they are estimated for respondents residing in low-risk families. Again, none of the dopamine genes are significant. Table 5.6 presents the results for the high-risk family group. One significant finding emerges in these models: DRD2 maintains a positive and statistically significant effect on the dependent variable for black males, revealing a GxE between family risk and DRD2 in the etiology of delinquency.

Summary of the Effects of the Dopamine Genes on the Wave I Delinquency Scale

Tables 5.1-5.6 contained the results of the direct and interactive effects that the dopamine polymorphisms had on the wave I delinquency scale. These tables revealed two broad findings. First, some of the dopamine genes, for some of the gender/race subcategories had significant effects on delinquency when controlling for delinquent peers. However, the dopamine genes did not exert a significant direct effect on delinquency when controlling for family risk. Second, and

Table 5.4. The Direct Effects of Dopamine Genes and Family Risk on Delinquency at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.05	-.01	.13	.02	-.31	-.04	.16	.02	.41	.05
	(.20)		(.34)		(.28)		(.78)		(.57)	
DRD2	.03	.00	-.01	-.00	.03	.00	1.15	.13	-.68	-.11
	(.19)		(.34)		(.28)		(.71)		(.43)	
DRD4	.13	.02	-.34	-.04	.34	.04	1.08	.11	.02	.00
	(.20)		(.35)		(.28)		(.74)		(.48)	
Socialization Variable										
Family risk	1.14	.22**	.88	.15**	1.18	.28**	1.34	.19**	1.26	.31**
	(.12)		(.23)		(.15)		(.55)		(.28)	
Control Variables										
Age	-.15	-.05**	-.03	-.01	-.20	-.07*	.06	.02	-.45	-.17**
	(.07)		(.10)		(.09)		(.27)		(.18)	
Cognitive complexity	-.02	-.04	-.03	-.06	-.02	-.04	.06	.11	-.02	-.06
	(.01)		(.02)		(.02)		(.05)		(.02)	
Race	.23	.02								
	(.28)									
Gender	1.17	.12**								
	(.31)									
R-squared	.07		.03		.09		.08		.14	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.5. The Effects of Dopamine Genes on Delinquency at Wave I for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.06	.01	.17	.02	-.15	-.02	.24	.03	.42	.05
	(.23)		(.42)		(.31)		(.86)		(.71)	
DRD2	.03	.00	.51	.06	-.12	-.02	-.05	-.01	-.78	-.14
	(.22)		(.44)		(.31)		(.77)		(.51)	
DRD4	.13	.02	-.20	-.02	.46	.07	.31	.03	.13	.02
	(.24)		(.44)		(.32)		(.91)		(.63)	
Control Variables										
Age	-.45	-.05	.12	.01	-.59	-.08	-.49	-.05	-1.81	-.21**
	(.29)		(.54)		(.37)		(1.1)		(.79)	
Cognitive complexity	-.01	-.03	.01	.01	-.02	- .05	-.02	-.04	-.02	-.07
	(.01)		(.03)		(.02)		(.05)		(.03)	
Race	.30	.03								
	(.36)									
Gender	1.53	.17**								
	(.28)									
R-squared	.03		.01		.02		.01		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.6. The Effects of Dopamine Genes on Delinquency at Wave I for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.14	-.02	.16	.02	-.52	-.06	.55	.04	.75	.08
	(.35)		(.55)		(.50)		(1.6)		(1.1)	
DRD2	.11	.01	-.51	-.06	.37	.04	3.65	.32**	-1.06	-.14
	(.34)		(.53)		(.50)		(1.5)		(.93)	
DRD4	.20	.02	-.35	-.03	.22	.03	2.05	.20	.03	.00
	(.34)		(.58)		(.51)		(1.3)		(.85)	
Control Variables										
Age	-.80	-.07*	.01	.00	-1.42	-.13	1.01	.07	-1.21	-.12
	(.42)		(.65)		(.63)		(1.9)		(1.2)	
Cognitive complexity	-.03	-.05	-.10	-.15**	-.02	-.04	.21	.34**	-.03	-.07
	(.02)		(.04)		(.03)		(.08)		(.05)	
Race	-.01	-.00								
	(.57)									
Gender	.94	.08**								
	(.41)									
R-squared	.02		.03		.03		.20		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

perhaps more importantly, were the findings for the interactive models. Three GxEs were detected between the dopamine genes and delinquent peers and one GxE was observed between DRD2 and family risk. In summary, the dopamine genes had significant direct effects and significant interactive effects in the prediction of delinquency at wave I.

Wave II Delinquency Scale

The results of the direct effects models predicting the wave II delinquency scale with the dopamine genes and the measure of delinquent peers are contained in Table 5.7. Similar to the results for the wave I delinquency scale, the delinquent peers scale is the strongest predictor of delinquent involvement at wave II in all of the OLS equations. In regards to the dopamine genes, DAT1 maintains a significant inverse relationship with the wave II delinquency scale for the full sample and for the black male subsample. The remaining dopamine polymorphisms fail to reach statistical significance.

Next, multivariate models are estimated separately for the low delinquent peers group and the high delinquent peers group to determine whether the genetic effects are contingent upon the social environment. Table 5.8 shows the results for the low delinquent peers group. Across all of the models in Table 5.8, none of the dopamine genes are significant. The same models are also calculated for the high delinquent peers group with the results of these equations shown in Table 5.9. For the full sample, DAT1 and DRD2 exert statistically significant and negative effects on the wave II delinquency scale. These significant coefficients reveal GxEs between DAT1 and delinquent peers and between DRD2 and delinquent peers, whereby the genetic effects are only visible for respondents in the high delinquent peers group.

Table 5.7. The Direct Effects of Dopamine Genes and Delinquent Peers on Delinquency at Wave II

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.26	-.04*	-.35	-.05	-.09	-.02	-1.09	-.16**	.28	.05
	(.14)		(.25)		(.20)		(.54)		(.43)	
DRD2	-.17	-.03	-.15	-.02	-.27	-.05	.35	.06	-.47	-.11
	(.14)		(.25)		(.20)		(.50)		(.31)	
DRD4	.19	.03	.10	.01	.29	.05	.48	.07	-.11	-.02
	(.14)		(.26)		(.21)		(.53)		(.33)	
Socialization Variable										
Delinquent peers	.43	.30**	.45	.30**	.41	.31**	.57	.35**	.34	.25**
	(.03)		(.06)		(.05)		(.14)		(.10)	
Control Variables										
Age	-.43	-.20**	-.37	-.15**	-.57	-.27**	-.12	-.05	-.46	-.26**
	(.05)		(.09)		(.08)		(.21)		(.13)	
Cognitive complexity	.01	.03	-.00	-.00	.02	.06*	.00	.01	.01	.04
	(.01)		(.02)		(.01)		(.03)		(.02)	
Race	.21	.02								
	(.28)									
Gender	.45	.06**								
	(.17)									
R-squared	.10		.09		.12		.14		.12	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.8. The Effects of Dopamine Genes on Delinquency at Wave II for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.05	-.01	.02	.00	-.17	-.04	-.31	-.06	.60	.13
	(.16)		(.30)		(.23)		(.59)		(.44)	
DRD2	.08	.02	.46	.08	.03	.01	-.21	-.04	-.48	-.14
	(.17)		(.33)		(.23)		(.61)		(.34)	
DRD4	.19	.04	.08	.01	.35	.08	-.11	-.02	.25	.07
	(.17)		(.33)		(.25)		(.70)		(.34)	
Control Variables										
Age	-.76	-.12**	-.97	-.13**	-.90	-.15**	1.06	.15	-1.01	-.20**
	(.06)		(.31)		(.08)		(.80)		(.50)	
Cognitive complexity	-.09	-.03	-.04	-.10**	.01	.02	-.02	-.07	.01	.04
	(.01)		(.02)		(.02)		(.03)		(.02)	
Race	-.18	-.03								
	(.26)									
Gender	.34	.06								
	(.20)									
R-squared	.02		.04		.03		.03		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.9. The Effects of Dopamine Genes on Delinquency at Wave II for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.40	-.06*	-.50	-.07	.03	.00	-1.59	-.19	-.48	-.07
	(.24)		(.40)		(.34)		(.97)		(.88)	
DRD2	-.45	-.07*	-.60	-.09	-.55	-.08	.53	.07	-.59	-.12
	(.22)		(.37)		(.33)		(.85)		(.58)	
DRD4	.21	.03	.24	.03	.06	.01	1.17	.16	-.50	-.09
	(.24)		(.40)		(.34)		(.84)		(.68)	
Control Variables										
Age	-1.68	-.20**	-1.39	-.16**	-2.24	-.28**	-.46	-.05	-1.75	-.24**
	(.28)		(.45)		(.40)		(1.2)		(.87)	
Cognitive complexity	.01	.04	.02	.04	.02	.05	-.00	-.01	-.00	-.00
	(.02)		(.03)		(.02)		(.06)		(.03)	
Race	.29	.03								
	(.39)									
Gender	.54	.06*								
	(.28)									
R-squared	.05		.04		.08		.07		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.10 illustrates the findings for the OLS equations predicting the wave II delinquency scale with the dopamine genes and the family risk scale. The measure of family risk exerts a statistically significant and positive effect on the wave II delinquency scale in all of the models. In contrast, none of the dopamine genes have a significant direct effect on the dependent variable.

Tables 5.11 and 5.12 examine whether the dopamine genes interact with family risk in the creation of delinquency. In Table 5.11, the OLS models are calculated for respondents characterized as residing in low-risk families. The results reveal that the DRD2 polymorphism maintains a significant and negative relationship with the wave II delinquency scale for white females and black females in the low-risk family group. The same models were estimated for the high-risk family group. The results of these equations, depicted in Table 5.12, show that DRD2 is significant for white males, whereas DAT1 and DRD4 are significant for black males in the high-risk family group. These results suggest a GxE between the dopamine genes and family risk.

Summary of the Effects of the Dopamine Genes on the Wave II Delinquency Scale

Taken together, the models for the wave II delinquency scale reveal two broad findings. First, only two of the dopaminergic polymorphisms (DAT1 and DRD2) had a direct effect on the wave II delinquency scale. Second, and most interesting, were the results of the interactive effects models. Altogether, seven different GxEs were detected between the dopaminergic genes and the two measures of the social environment (i.e., delinquent peer and family risk). In summary, variation in the wave II delinquency scale was explained more consistently by GxEs than through the direct effects models.

Table 5.10. The Direct Effects of Dopamine Genes and Family Risk on Delinquency at Wave II

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.20	-.03	-.21	-.03	-.09	-.02	-.84	-.12	.31	.05
	(.15)		(.25)		(.21)		(.56)		(.43)	
DRD2	-.15	-.03	-.13	-.02	-.24	-.04	.24	.04	-.39	-.09
	(.14)		(.25)		(.21)		(.52)		(.31)	
DRD4	.14	.02	.10	.01	.15	.03	.87	.13	-.11	-.03
	(.15)		(.27)		(.22)		(.56)		(.34)	
Socialization Variable										
Family risk	.55	.15**	.55	.13**	.63	.19**	-.15	-.03	.50	.18**
	(.09)		(.18)		(.12)		(.48)		(.20)	
Control Variables										
Age	-.25	-.11**	-.18	-.08	-.37	-.18*	.14	.06	-.38	-.22**
	(.05)		(.09)		(.08)		(.20)		(.13)	
Cognitive complexity	-.00	-.00	-.01	-.02	.01	.03	-.01	-.02	-.01	-.04
	(.01)		(.02)		(.01)		(.03)		(.02)	
Race	.00	.00								
	(.24)									
Gender	.47	.06**								
	(.18)									
R-squared	.04		.02		.06		.04		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.11. The Effects of Dopamine Genes on Delinquency at Wave II for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.12	-.02	-.11	-.02	-.26	-.05	-.16	-.02	.59	.11
	(.18)		(.32)		(.24)		(.66)		(.50)	
DRD2	-.12	-.02	.45	.07	-.49	-.10**	-.14	-.02	-.59	-.16*
	(.17)		(.34)		(.24)		(.60)		(.35)	
DRD4	.07	.01	-.03	-.00	.24	.05	.11	.02	.13	.03
	(.18)		(.35)		(.25)		(.70)		(.42)	
Control Variables										
Age	-.75	-.11**	-.72	-.09*	-1.02	-.17**	.61	.07	-1.39	-.25**
	(.22)		(.42)		(.29)		(.83)		(.53)	
Cognitive complexity	-.02	-.07**	-.02	-.06	-.00	- .01	-.01	-.14	-.03	-.15
	(.01)		(.02)		(.02)		(.04)		(.02)	
Race	.02	.00								
	(.28)									
Gender	.53	.08**								
	(.21)									
R-squared	.02		.02		.04		.03		.11	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.12. The Effects of Dopamine Genes on Delinquency at Wave II for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.32	-.05	-.40	-.06	.14	.02	-2.31	-.29**	-.30	-.05
	(.25)		(.39)		(.39)		(1.1)		(.83)	
DRD2	-.14	-.02	-.77	-.12**	.15	.02	1.33	.18	.06	.01
	(.24)		(.37)		(.39)		(1.0)		(.69)	
DRD4	.21	.03	.23	.03	.04	.01	1.49	.22*	-.45	-.09
	(.25)		(.41)		(.40)		(.89)		(.60)	
Control Variables										
Age	-1.06	-.13**	-.81	-.11*	-1.66	-.20**	.36	.04	-.81	-.12
	(.31)		(.46)		(.49)		(1.3)		(.86)	
Cognitive complexity	.03	.07*	.01	.03	.03	.07	.06	.15	.04	.13
	(.02)		(.03)		(.03)		(.06)		(.04)	
Race	-.12	-.01								
	(.42)									
Gender	.30	.04								
	(.30)									
R-squared	.03		.03		.03		.18		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Wave III Delinquency Scale

Table 5.13 contains the results of the OLS equations employing the wave III delinquency scale as the dependent variable, and the dopamine genes and measure of delinquent peers as independent variables. The delinquent peers scale has a significant and positive effect on the wave III delinquency scale for the full sample, for white males, and for white females; however, the delinquent peers scale is statistically insignificant for black males and black females. Two significant findings are observed for the dopamine genes. First, DAT1 has a significant and negative impact on delinquent involvement at wave III for white females. Second, DRD4 exerts a negative and statistically significant effect on wave III delinquency for black females.

The results of the models examining the potential interactive effects between delinquent peers and the dopamine polymorphisms are found in Tables 5.l4 and 5.15. Table 5.14 contains the findings for the analyses garnered with the low delinquent peers group and Table 5.15 contains the findings for the analyses garnered with the high delinquent peers group. As shown in Table 5.14, DAT1 maintains a significant and positive association with the wave III delinquency scale for the full sample and for white males. For white females, DAT1 has a negative effect on delinquency. In addition, DRD2 has a significant positive effect on wave III delinquency for white females. Table 5.15 shows that none of the dopamine genes are statistically related to wave III delinquency for the high delinquent peers group, suggesting that the effects for the dopamine genes are only observed for respondents with few delinquent peers.

Next, the models examining the effects of family risk and the dopamine genes on wave III delinquency are reported. Table 5.16 reveals that family risk is positively related to the dependent variable for the full sample, for white females, and for black females. Moreover, DAT1 has a significant and positive direct effect on wave III delinquency for white males.

Table 5.13. The Direct Effects of Dopamine Genes and Delinquent Peers on Delinquency at Wave III

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.05	.02	.20	.05	-.11	-.07*	.29	.10	-.05	-.02
	(.06)		(.14)		(.06)		(.23)		(.16)	
DRD2	-.00	-.00	-.07	-.02	.09	.05	.08	.03	-.07	-.04
	(.06)		(.13)		(.06)		(.21)		(.12)	
DRD4	.07	.03	-.14	-.04	.03	.02	-.02	-.01	-.23	-.12*
	(.06)		(.14)		(.06)		(.22)		(.13)	
Socialization Variable										
Delinquent peers	.07	.11**	.13	.16**	.04	.11**	-.06	-.10	.00	.00
	(.02)		(.03)		(.01)		(.06)		(.04)	
Control Variables										
Age	-.13	-.14**	-.26	-.20**	-.05	-.09**	.02	.03	-.09	-.13*
	(.02)		(.05)		(.02)		(.09)		(.05)	
Cognitive complexity	.01	.06**	.02	.09	.00	.03	.01	.05	.01	.06
	(.00)		(.01)		(.00)		(.01)		(.01)	
Race	.08	.02								
	(.10)									
Gender	.71	.22**								
	(.07)									
R-squared	.07		.06		.02		.02		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.14. The Effects of Dopamine Genes on Delinquency at Wave III for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.18	.07**	.47	.13**	-.11	-.10*	.57	.17	-.15	-.08
	(.09)		(.19)		(.06)		(.38)		(.18)	
DRD2	.03	.01	.09	.03	.11	.10*	.04	.01	-.14	-.10
	(.09)		(.21)		(.06)		(.38)		(.14)	
DRD4	-.03	-.01	.02	.01	-.02	-.02	.07	.02	-.21	-.14
	(.09)		(.21)		(.06)		(.43)		(.14)	
Control Variables										
Age	-.10	-.11**	-.30	-.23**	.00	.01	.07	.06	-.02	-.03
	(.03)		(.07)		(.02)		(.13)		(.05)	
Cognitive complexity	.01	.04	.02	.07	.00	.03	-.00	-.01	.00	.01
	(.01)		(.01)		(.00)		(.02)		(.01)	
Race	.16	.04								
	(.14)									
Gender	.75	.23**								
	(.11)									
R-squared	.07		.07		.02		.03		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.15. The Effects of Dopamine Genes on Delinquency at Wave III for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.06	-.02	-.02	-.01	-.10	-.05	-.04	-.02	.01	.00
	(.09)		(.19)		(.10)		(.24)		(.30)	
DRD2	-.03	-.01	-.16	-.05	.07	.04	.07	.04	.00	.00
	(.09)		(.18)		(.10)		(.21)		(.21)	
DRD4	-.10	-.04	-.23	-.06	.06	.03	-.14	-.08	-.20	-.08
	(.09)		(.19)		(.10)		(.21)		(.25)	
Control Variables										
Age	-.14	-.13**	-.16	-.12**	-.11	-.14**	-.04	-.05	-.20	-.23**
	(.04)		(.07)		(.04)		(.10)		(.10)	
Cognitive complexity	.01	.07	.02	.10*	.00	.02	.02	.14	.01	.09
	(.01)		(.01)		(.01)		(.02)		(.01)	
Race	-.06	-.01								
	(.15)									
Gender	.70	.21**								
	(.11)									
R-squared	.07		.03		.03		.03		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.16 shows the results of the direct effects models predicting the wave III delinquency scale with the dopamine genes and with the family risk scale. Across these models, family risk is significant for the full sample, for white females, and for black females. Only one dopamine gene—DAT1—is significantly and positively related to the dependent variable and the effect for this polymorphism is confined only to white males.

The next set of models are the same as those reported in Table 5.16, but are estimated for respondents characterized as residing in low-risk families. As shown in Table 5.17, DRD2 is a statistically significant and positive predictor of wave III delinquency for white females. For black females DRD2 and DAT1 are negatively related to the dependent variable. In Table 5.18, the same models are calculated for the high-risk family group. In these models, DRD2 exerts a significant negative effect on the wave III delinquency scale. Taken together, and supportive of the role of GxEs in the etiology of delinquency, the results in Tables 5.17 and 5.18 reveal that the effects of the dopamine genes are contingent on the risk level of the family.

Summary of the Effects of the Dopamine Genes on the Wave III Delinquency Scale

The relationship between the dopamine genes and the wave III delinquency scale are quite similar to the findings for the wave II delinquency scale. Across all of the direct effects models, three equations detected a significant relationship between the dopamine genes and the wave III delinquency scale. In the interactive models, however, the dopamine genes tended to have much stronger and more consistent effects on the dependent variable. For example, eight significant GxEs were detected between the dopamine genes and the two measures of the social environment. In summary, the results of the models employing the wave III delinquency scale as the outcome variable suggest that GxEs are powerful predictors of delinquency in young adults.

Table 5.16. The Direct Effects of Dopamine Genes and Family Risk on Delinquency at Wave III

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.07	.03	.23	.06*	-.08	-.05	.27	.09	-.11	-.05
	(.07)		(.14)		(.06)		(.24)		(.16)	
DRD2	-.03	-.01	-.13	-.04	.09	.06	-.01	-.00	-.05	-.03
	(.06)		(.14)		(.06)		(.22)		(.12)	
DRD4	-.10	-.03	-.18	-.05	.01	.01	-.07	-.03	-.25	-.13
	(.07)		(.14)		(.06)		(.23)		(.13)	
Socialization Variable										
Family risk	.14	.08**	.14	.06	.12	.14**	.04	.02	.23	.22**
	(.04)		(.09)		(.03)		(.18)		(.08)	
Control Variables										
Age	-.12	-.12**	-.23	-.17**	-.04	-.07*	-.04	-.04	-.10	-.14*
	(.02)		(.05)		(.02)		(.08)		(.05)	
Cognitive complexity	.01	.05*	.01	.07*	.00	.01	.01	.03	.01	.06
	(.00)		(.01)		(.00)		(.01)		(.01)	
Race	.09	.02								
	(.10)									
Gender	.76	.23**								
	(.08)									
R-squared	.07		.04		.03		.01		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.17. The Effects of Dopamine Genes on Delinquency at Wave III for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.03	.01	.16	.05	-.02	-.02	.24	.09	-.40	-.26**
	(.08)		(.17)		(.06)		(.27)		(.13)	
DRD2	.03	.01	.10	.03	.11	.09*	-.02	-.01	-.23	-.21**
	(.07)		(.18)		(.06)		(.25)		(.09)	
DRD4	-.13	-.05	-.21	-.06	-.01	-.01	-.26	-.09	-.07	-.05
	(.08)		(.18)		(.06)		(.29)		(.12)	
Control Variables										
Age	-.11	-.12**	-.21	-.17**	-.04	-.10**	.03	.03	-.11	-.25**
	(.03)		(.06)		(.02)		(.09)		(.05)	
Cognitive complexity	.01	.08**	.03	.11**	.00	.06	.01	.08	.01	.10
	(.01)		(.01)		(.00)		(.01)		(.01)	
Race	.12	.03								
	(.12)									
Gender	.81	.26**								
	(.09)									
R-squared	.09		.05		.02		.03		.19	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.18. The Effects of Dopamine Genes on Delinquency at Wave III for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.13	.04	.36	.09	-.17	-.09	.22	.06	.36	.12
	(.11)		(.23)		(.11)		(.49)		(.36)	
DRD2	-.10	-.04	-.39	-.10*	.09	.05	-.04	-.01	.22	.09
	(.11)		(.22)		(.11)		(.47)		(.31)	
DRD4	-.04	-.02	-.12	-.03	.04	.02	.19	.07	-.40	-.17
	(.11)		(.24)		(.11)		(.41)		(.28)	
Control Variables										
Age	-.12	-.10**	-.22	-.15**	-.01	-.02	-.19	-.15	-.07	-.06
	(.04)		(.08)		(.04)		(.18)		(.12)	
Cognitive complexity	.00	.01	.01	.03	-.00	-.03	-.01	-.07	.01	.06
	(.01)		(.02)		(.01)		(.03)		(.02)	
Race	-.01	-.00								
	(.19)									
Gender	.68	.18**								
	(.13)									
R-squared	.04		.04		.01		.04		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Number of Police Contacts

Table 5.19 shows the results of the negative binomial regression models predicting number of police contacts with the dopamine polymorphisms and the measure of delinquent peers. As shown in the top three rows of Table 5.19, none of the dopamine genes are significantly predictive of police contacts. The delinquent peers scale does, however, increase the number of police contacts for the full sample, for white males, and for white females.

Negative binomial equations were also calculated for the low delinquent peers group and for the high delinquent peers group. Table 5.20 contains the results for the low delinquent peers group. For these models, DAT1 maintains a significant positive relationship with police contacts for the full sample and for white males. In addition, DRD2 also has a significant positive effect on the dependent variable for white males. The remaining dopamine genes do not reach statistical significance. Table 5.21 portrays the findings for the analyses conducted with respondents in the high delinquent peers group. None of the dopamine polymorphisms are statistically related to police contacts.

Table 5.22 depicts the findings of the negative binomial equations predicting police contacts with the family risk measure and the dopamine polymorphisms. The family risk scale has a positive and statistically significant effect on police contacts for all of the samples except for black males. In addition, DRD4 has a significant negative direct effect on police contacts for white females and DRD2 has a significant positive effect on police contacts for black females.

The findings for the models estimated for the low-risk family group and the high-risk family group are presented in Tables 5.23 and 5.24, respectively. In the low-risk family analysis (Table 5.23), DRD2 has a significant positive effect on police contacts for white males. As shown in Table 5.24, DRD4 has a positve impact on police contacts for black females in the

Table 5.19. The Direct Effects of Dopamine Genes and Delinquent Peers on Number of Police Contacts

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.08	.11	.22	.14	-.11	.22	.13	.29	-.53	.61
DRD2	-.05	.10	.04	.14	-.26	.23	-.05	.28	.80	.63
DRD4	.03	.10	.05	.14	-.28	.24	.18	.25	.77	.58
Socialization Variable										
Delinquent peers	.14**	.02	.13**	.03	.21**	.05	.07	.06	.17	.18
Control Variables										
Age	-.20**	.04	-.17**	.04	-.42**	.09	-.07	.10	.05	.25
Cognitive complexity	.00	.01	-.00	.01	.01	.02	.01	.02	-.02	.04
Race	-.06	.17								
Gender	1.61**	.14								
Pseudo R-squared	.07		.02		.06		.01		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.20. The Effects of Dopamine Genes on Number of Police Contacts for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.39**	.17	.65**	.23	-.15	.33	.48	.38	---	---
DRD2	.16	.15	.39*	.23	.08	.33	-.04	.36	---	---
DRD4	.19	.16	.26	.22	-.18	.37	.26	.36	---	---
Control Variables										
Age	-.18**	.06	-.20**	.07	-.39**	.15	-.07	.12	---	---
Cognitive complexity	-.01	.01	-.03	.01	-.01	.02	.01	.02	---	---
Race	-.10	.24								
Gender	1.79**	.21								
Pseudo R-squared	.08		.04		.04		.02		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
†The negative binomial model for black females in the low delinquent peers group failed to converge. Further inspection of the data revealed that only four black females ever had contact with the police. The lack of variability in the dependent variable precluded the model from converging.

Table 5.21. The Effects of Dopamine Genes on Number of Police Contacts for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.13	.14	.01	.18	-.22	.32	-.20	.47	-1.17	.68
DRD2	-.17	.14	-.09	.17	-.50	.33	-.04	.45	.40	.59
DRD4	-.07	.14	-.04	.18	-.41	.33	.21	.39	.11	.75
Control Variables										
Age	-.17**	.05	-.11*	.06	-.34**	.12	.10	.22	-.37	.31
Cognitive complexity	.01	.01	.01	.01	.02	.02	.03	.03	.00	.33
Race	-.07	.24								
Gender	1.49**	.18								
Pseudo R-squared	.06		.00		.04		.01		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.22. The Direct Effects of Dopamine Genes and Family Risk on Number of Police Contacts

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.08	.11	.18	.14	.01	.24	.05	.29	-.73	.55
DRD2	-.07	.11	.00	.14	-.26	.24	-.11	.28	.95*	.56
DRD4	-.06	.11	.01	.14	-.44*	.26	.03	.27	.30	.57
Socialization Variable										
Family Risk	.26**	.06	.18**	.09	.29**	.13	.25	.20	.73**	.26
Control Variables										
Age	-.13**	.04	-.10**	.05	-.28**	.09	-.01	.10	-.07	.22
Cognitive complexity	.00	.01	-.00	.01	.00	.02	.01	.02	-.02	.03
Race	-.09	.17								
Gender	1.67**	.14								
Pseudo R-squared	.07		.01		.03		.01		.12	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.23. The Effects of Dopamine Genes on Number of Police Contacts for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.20	.15	.28	.18	.03	.38	.07	.40	---	---
DRD2	.03	.14	.32*	.18	-.55	.40	-.40	.38	---	---
DRD4	-.12	.15	.12	.18	-.48	.43	-.29	.41	---	---
Control Variables										
Age	-.17**	.05	-.19**	.07	-.30**	.13	-.06	.14	---	---
Cognitive complexity	.01	.01	.01	.01	-.01	.02	.01	.03	---	---
Race	-.10	.22								
Gender	1.93**	.20								
Pseudo R-squared	.08		.03		.03		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
†The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable precluded the model from converging.

Table 5.24. The Effects of Dopamine Genes on Number of Police Contacts for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.02	.16	.14	.21	-.04	.31	.22	.43	-.83	.70
DRD2	-.13	.16	-.32	.21	-.02	.30	.35	.42	.45	.80
DRD4	.06	.16	-.07	.22	-.44	.34	.51	.32	1.36*	.79
Control Variables										
Age	-.06	.05	.00	.07	-.29**	.12	.06	.15	-.01	.31
Cognitive complexity	-.00	.01	-.02	.01	.02	.02	.02	.02	-.01	.04
Race	-.12	.26								
Gender	1.36**	.19								
Pseudo R-squared	.04		.01		.03		.04		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

high-risk family category.

Summary of the Effects of the Dopamine Genes on Number of Police Contacts

The results of the negative binomial equations using number of police contacts as the dependent variable revealed that the dopamine genes did not consistently have direct effects on the dependent variable. Instead, and in line with previous research (Caspi et al., 2002a; Rutter, 2006), the genetic effects were only visible when paired with certain social environments. These results, along with those reported in the preceding tables, begin to reveal the importance of GxEs in the etiology of antisocial behavior.

Ever Arrested

Table 5.25 presents the results of the binary logistic regression equations predicting arrest status (yes/no) with the dopamine genes and with the delinquent peers scale. The delinquent peers measure is positively related to arrest status across all of the models. In comparison, none of the dopamine genes had a significant direct effect on arrest status.

Next, the logistic regression models are estimated for the low delinquent peers group. As shown in Table 5.26, DAT1 has a significant positive effect on arrest status for the full sample and for the sample of white males. The results for the analysis conducted with the high delinquent peers group, however, reveals that none of the dopamine polymorphisms are significantly related to arrest status. Taken together, Tables 5.26 and 5.27 indicate that there is a GxE between DAT1 and delinquent peers in the prediction of arrest status. More specifically, the effects of DAT1 are only visible for Add Health respondents with low levels of delinquent friends.

Table 5.25. The Direct Effects of Dopamine Genes and Delinquent Peers on Arrest Status

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.01	.14	.18	.19	-.26	.32	-.23	.33	-.26	.32
DRD2	-.02	.14	-.12	.18	-.26	.35	.23	.30	-.26	.36
DRD4	-.06	.14	-.04	.18	-.19	.35	.05	.32	-.19	.35
Socialization Variable										
Delinquent peers	.23**	.03	.23**	.04	.27**	.07	.14*	.08	.27**	.07
Control Variables										
Age	-.22**	.06	-.22**	.07	-.27**	.13	-.12	.13	-.27**	.13
Cognitive complexity	.01	.01	.00	.01	.02	.02	.01	.02	.02	.02
Race	.14	.22								
Gender	1.86**	.21								
Cox & Snell R-squared	.08		.06		.02		.03		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.26. The Effects of Dopamine Genes on Arrest Status for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.49*	.25	1.12**	.40	-.26	.52	-.08	.44	---	---
DRD2	.11	.23	-.03	.33	.37	.52	.15	.46	---	---
DRD4	.07	.23	.12	.31	-.03	.59	.22	.51	---	---
Control Variables										
Age	-.22**	.09	-.27**	.12	-.31	.23	-.06	.16	---	---
Cognitive complexity	-.01	.01	-.04*	.02	-.00	.04	.01	.03	---	---
Race	.33	.33								
Gender	2.07**	.36								
Cox & Snell R-squared	.06		.06		.01		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.27. The Effects of Dopamine Genes on Arrest Status for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.15	.18	-.13	.22	-.26	.39	-.17	.47	---	---
DRD2	-.06	.17	-.11	.21	-.65	.49	.37	.42	---	---
DRD4	-.04	.18	-.04	.22	-.33	.44	.15	.42	---	---
Control Variables										
Age	-.12*	.07	-.12	.09	-.14	.15	.03	.20	---	---
Cognitive complexity	.01	.01	.01	.02	.02	.03	.00	.03	---	---
Race	-.15	.30								
Gender	-1.62	1.37								
Cox & Snell R-squared	.07		.01		.01		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

In Table 5.28, the logistic regression equations are predicting arrest status with the family risk scale and with the three dopamine genes. For all of the models, the family risk measure is significantly predictive of arrest status. In line with results found in Table 5.27, none of the dopamine genes have a significant direct effect on arrest status.

To explore the possibility that the effects of the dopamine genes are confined to certain environmental conditions (i.e., a GxE), Tables 5.29 and 5.30 estimate the logistic regression equations for the low-risk family group and the high-risk family group, respectively. In Table 5.29, none of the dopamine genes are significantly related to arrest status for those respondents in the low-risk family category. Only one dopamine gene—DRD2—is significant for the logistic regression equations conducted for the high-risk family group. Specifically, DRD2 is negatively related to arrest status for white males. Once again, these findings reveal a significant GxE between DRD2 and the family environment in the creation of arrests for white males.

Summary of the Effects of the Dopamine Genes on Arrest Status

Tables 5.25 through 5.30 revealed the results for the logistic regression models using arrest status as the outcome variable. The models found no evidence that the dopamine genes directly affect the likelihood of being arrested. A different pattern of results were found for the interactive models. In total, three different GxEs were detected in the multivariate equations, suggesting that the genetic effects for the dopaminergic polymorphisms are conditioned by the social environment (Rutter, 2006).

Marijuana Use

The next six tables use frequency of using marijuana in the past month as the dependent

Table 5.28. The Direct Effects of Dopamine Genes and Family Risk on Arrest Status

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.02	.14	.07	.18	-.17	.30	-.24	.32	---	---
DRD2	-.06	.14	-.16	.18	-.26	.35	.10	.30	---	---
DRD4	-.09	.14	-.09	.18	-.26	.35	-.00	.31	---	---
Socialization Variable										
Family risk	.30**	.08	.31**	.11	.26*	.15	.40*	.21	---	---
Control Variables										
Age	-.10*	.05	-.09	.06	-.11	.12	-.05	.12	---	---
Cognitive complexity	-.00	.01	-.01	.01	.01	.02	.01	.02	---	---
Race	.07	.21								
Gender	1.84**	.20								
Cox & Snell R-squared	.06		.02		.01		.03		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.29. The Effects of Dopamine Genes on Arrest Status for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.02	.19	.17	.25	-.08	.49	-.40	.39	---	---
DRD2	-.03	.19	.21	.24	-1.66	1.01	-.21	.39	---	---
DRD4	-.18	.21	.11	.25	-.97	.62	-.44	.50	---	---
Control Variables										
Age	-.11	.07	-.13	.09	-.14	.18	-.05	.15	---	---
Cognitive complexity	-.01	.01	-.01	.02	-.03	.03	-.01	.03	---	---
Race	-.02	.29								
Gender	1.99**	.31								
Cox & Snell R-squared	.06		.01		.02		.02		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.30. The Effects of Dopamine Genes on Arrest Status for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.03	.19	.00	.25	-.25	.40	.21	.56	---	---
DRD2	-.08	.19	-.49*	.26	.31	.39	.46	.50	---	---
DRD4	-.01	.21	-.28	.28	.11	.41	.41	.43	---	---
Control Variables										
Age	-.06	.07	-.02	.09	-.11	.17	.06	.20	---	---
Cognitive complexity	.00	.01	-.02	.02	.05	.03	.04	.03	---	---
Race	.13	.32								
Gender	1.65**	.27								
Cox & Snell R-squared	.06		.02		.01		.05		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

variable in the negative binomial regression equations. Table 5.31 depicts the results of the direct effects models, where the dopamine polymorphisms and the delinquent peers scale are the independent variables of interest. The measure of delinquent peers is the strongest and most consistent predictor of marijuana use—it is statistically significant for all of the models in Table 5.31. Three equations also reveal that the dopamine genes have a significant direct effect on marijuana use. Specifically, DRD2 is negatively associated with marijuana use for the full sample and the white male sample, while DRD4 is negatively related to marijuana use for black females.

The negative binomial regression equations are next calculated separately for the low delinquent peers group and for the high delinquent peers group to examine whether the effects of the dopamine genes vary by peer context. As shown in Table 5.32, none of the dopamine genes are related to marijuana use for the full sample of respondents in the low delinquent peers group. The models were not calculated for the gender and race subsamples because there was very little variability in the dependent variable. This lack of variability prevented the negative binomial equations from converging and providing interpretable results. Given that the delinquent peers measure taps into the drug-using behaviors of the respondents friends, it should not be too surprising that adolescents without drug-using friends report virtually no involvement with marijuana use. Analyses based on the high delinquent peers group, however, provided interpretable results. As revealed in Table 5.33, DRD4 is a significant and positive predictor of marijuana use for the full sample and DRD2 is positively related to marijuana use for the black male subsample. While comparing the results of Table 5.32 with those of Table 5.33 is difficult, the findings point to the likelihood that GxEs between dopamine genes and delinquent peers in the etiology of marijuana use.

Table 5.31. The Direct Effects of Dopamine Genes and Delinquent Peers on Frequency of Marijuana Use

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.26	.18	.12	.33	-.35	.24	-.05	.54	-.57	1.26
DRD2	-.79**	.18	-.87**	.33	-.31	.24	.43	.44	-.85	1.01
DRD4	.20	.19	-.06	.34	.38	.27	.05	.39	-1.82*	1.04
Socialization Variable										
Delinquent peers	.75**	.05	.89**	.09	.69**	.06	.98**	.15	.93**	.32
Control Variables										
Age	.07	.08	.04	.12	.16	.10	-.03	.24	.15	.36
Cognitive complexity	.03**	.01	.09**	.02	-.01	.02	.05	.03	-.06	.05
Race	.78	.31								
Gender	.67**	.22								
Pseudo R-squared	.11		.11		.14		.23		.10	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.32. The Effects of Dopamine Genes on Marijuana Use for the Low Delinquent Peers Group

	Full Sample		White Males†		White Females†		Black Males†		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.08	.73	---	---	---	---	---	---	---	---
DRD2	-.27	.91	---	---	---	---	---	---	---	---
DRD4	-.94	.95	---	---	---	---	---	---	---	---
Control Variables										
Age	.76	.34	---	---	---	---	---	---	---	---
Cognitive complexity	-.08	.05	---	---	---	---	---	---	---	---
Race	-.41	1.23								
Gender	-.1.23	1.09								
Pseudo R-squared	.06		---		---		---		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
†The negative binomial models that were calculated by the gender and race subgroups failed to converge. Part of the reason for the lack of interpretable results is that less than one percent of each subgroup indicated they had used marijuana in the previous month.

Table 5.33. The Effects of Dopamine Genes on Frequency of Marijuana Use for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.05	.23	.57	.40	-.18	.34	.38	.83	1.34	1.16
DRD2	-.02	.22	-.46	.32	.04	.35	1.16**	.57	-.18	.80
DRD4	.54**	.22	.27	.35	.55	.34	.66	.53	.71	.66
Control Variables										
Age	.14	.09	.24*	.15	.09	.13	.77**	.27	-.37	.38
Cognitive complexity	.02	.01	.05	.02	-.01	.02	.10*	.06	-.01	.03
Race	-.28	.37								
Gender	.74**	.26								
Pseudo R-squared	.01		.02		.00		.05		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.34 presents the findings for the negative binomial regression equations predicting marijuana use with the dopamine genes and with the family risk scale. The measure of family risk is significantly and positively related to marijuana use for the full sample and for black females. In addition, the dopamine genes have significant direct and positive effects on marijuana use in all five of the models. DRD4 is predictive of marijuana use for the full sample, DAT1 is predictive of marijuana use for white males, DRD4 is predictive of marijuana use for white females, DRD2 is predictive of marijuana use for black males, and DRD4 is predictive of marijuana use for black females.

The analysis proceeds by calculating the negative binomial regression models separately for the low-risk family group and for the high-risk family group. Table 5.35 shows the results garnered for the low-risk family subsample. DAT1 is positively related to marijuana use for white males and DRD4 is positively related to marijuana use for both white and black females. The results for the high-risk family group are found in Table 5.36. In comparison with the findings for the low-risk group, Table 5.36 reveals that DRD4 is positively predictive of marijuana use for the full sample and negatively related to marijuana use for black females. Moreover, DRD2 maintains a significant and positive association with marijuana use for black males and DAT1 is positively related to marijuana use for black females.

Summary of the Effects of the Dopamine Genes on Marijuana Use

The findings reported in Tables 5.31 through 5.36 revealed considerable support for the role of dopamine genes in the prediction of marijuana use. In total, the dopamine genes had eight significant direct effects on marijuana use across the models. An additional nine GxEs were detected between the dopamine genes and the two measures of the social environment.

Table 5.34. The Direct Effects of Dopamine Genes and Family Risk on Frequency of Marijuana Use

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.31	.23	.83**	.40	-.08	.36	1.20	.80	-.13	1.17
DRD2	.01	.23	-.48	.33	.01	.35	1.07*	.57	1.03	.91
DRD4	.63**	.22	.17	.36	.70**	.35	.48	.65	1.28**	.64
Socialization Variable										
Family risk	.38**	.14	.07	.21	.42	.26	1.13*	.59	1.01**	.40
Control Variables										
Age	.35**	.09	.48**	.16	.24*	.32	.76**	.25	.07	.34
Cognitive complexity	.01	.01	.04**	.02	-.02	.02	.06	.05	-.01	.03
Race	-.84**	.38								
Gender	.90**	.27								
Pseudo R-squared	.02		.02		.01		.07		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.35. The Effects of Dopamine Genes on Marijuana Use for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	.65	.43	1.56**	.54	-.21	.68	2.46	2.04	-3.09	2.95
DRD2	.05	.43	-.19	.52	.52	.60	-.04	1.56	1.52	1.77
DRD4	.58	.39	.39	.55	1.55**	.76	.85	1.25	1.94*	1.16
Control Variables										
Age	.49**	.17	.57**	.28	.25	.29	.71*	.41	.28	.66
Cognitive complexity	.02	.03	.05*	.03	-.05	.04	.04	.10	.12	.12
Race	-1.54**	.58								
Gender	.89	.48								
Pseudo R-squared	.03		.03		.02		.08		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.36. The Effects of Dopamine Genes on Marijuana Use for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Dopamine Genes										
DAT1	-.31	.30	-.45	.59	-.22	.40	.99	1.08	2.58*	1.46
DRD2	.00	.26	-.24	.41	-.12	.40	1.97**	.82	-.48	.97
DRD4	.47*	.26	.41	.45	.53	.40	.98	.70	-2.60**	1.20
Control Variables										
Age	.26	.11	.35*	.19	.21	.15	1.23	.43	-.57	.41
Cognitive complexity	.00	.02	.02	.03	-.02	.03	.09	.06	-.07**	.04
Race	.16	.46								
Gender	.35	.33								
Pseudo R-squared	.01		.01		.01		.07		.11	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

In sum, the dopamine genes had significant effects both directly and interactively on marijuana use and these effects tended to cut across the race- and gender-specific equations.

Alcohol Abuse

Table 5.37 contains the results for the OLS regression equations predicting scores on the alcohol abuse scale with the dopamine genes and with the measure of delinquent peers. In line with the findings for the previous dependent variables, the delinquent peers scale exerts the strongest and most consistent effect on alcohol abuse. The dopamine genes also directly contribute to alcohol abuse. For example, DAT1 is negatively related to alcohol abuse for white females and DRD2 is positively related to alcohol abuse for black males, while DRD2 is negatively related to alcohol abuse for black females.

The results of the OLS equations predicting alcohol abuse for the low delinquent peers sample are found in Table 5.38. In this table only one significant finding for the dopamine genes is found: DAT1 is positively related to alcohol abuse for white males. The remaining dopamine polymorphisms in all of the remaining models are statistically insignificant.

Table 5.39 depicts the results for the high delinquent peers group and shows a somewhat different pattern of results. In comparison with Table 5.38, Table 5.39 shows that the dopamine genes have four independent and significant effects on alcohol abuse for the high delinquent peers group. Specifically, DAT1 is negatively related to alcohol abuse for the full sample and for white females. In addition, DRD2 maintains a significant association with alcohol abuse for black males and for black females. Taken together, the findings thus far suggest that GxEs are more consistent predictors of alcohol abuse than are the direct effects of the dopamine genes.

Table 5.40 portrays the findings for the OLS regression equations predicting scores on

Table 5.37. The Direct Effects of Dopamine Genes and Delinquent Peers on Alcohol Abuse

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.18	-.03	.05	.01	-.54	-.10**	.48	.08	-.11	-.03
	(.15)		(.28)		(.20)		(.44)		(.23)	
DRD2	.04	.01	-.08	-.01	.13	.02	.70	.14*	-.35	-.14**
	(.14)		(.28)		(.20)		(.41)		(.17)	
DRD4	-.15	-.02	-.17	-.02	-.13	-.02	-.58	-.10	-.13	-.05
	(.14)		(.29)		(.21)		(.43)		(.19)	
Socialization Variable										
Delinquent peers	.19	.13**	.28	.17**	.13	.10**	.18	.14*	.13	.17**
	(.03)		(.07)		(.05)		(.11)		(.06)	
Control Variables										
Age	-.15	-.07**	-.25	-.10**	-.19	-.10**	.14	.07	.15	.15**
	(.07)		(.11)		(.08)		(.17)		(.07)	
Cognitive complexity	.04	.11**	.05	.11**	.04	.12**	.05	.15**	.01	.05
	(.01)		(.02)		(.01)		(.03)		(.01)	
Race	-1.26	-.13**								
	(.23)									
Gender	1.16	.15**								
	(.17)									
R-squared	.08		.04		.03		.08		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.38. The Effects of Dopamine Genes on Alcohol Abuse for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.17	.03	.69	.11*	-.30	-.05	.32	.07	.10	.05
	(.19)		(.37)		(.29)		(.46)		(.20)	
DRD2	.10	.02	.10	.01	.39	.07	-.33	-.08	-.14	-.08
	(.19)		(.40)		(.30)		(.47)		(.16)	
DRD4	-.00	-.00	.47	.07	-.34	-.05	-.71	-.15	-.08	-.05
	(.20)		(.40)		(.31)		(.52)		(.16)	
Control Variables										
Age	-.12	-.06*	-.23	-.10*	-.16	-.08	.20	.13	.07	.10
	(.07)		(.11)		(.10)		(.16)		(.06)	
Cognitive complexity	.04	.12**	.04	.08	.04	.11**	.06	.24**	.02	.22**
	(.01)		(.03)		(.02)		(.03)		(.01)	
Race	-1.49	-.17**								
	(.30)									
Gender	.92	.13**								
	(.23)									
R-squared	.08		.02		.03		.13		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.39. The Effects of Dopamine Genes on Alcohol Abuse for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.55	-.08**	-.50	-.06	-.75	-.13**	.92	.13	-.29	-.07
	(.22)		(.43)		(.28)		(.76)		(.47)	
DRD2	.01	.00	-.13	-.02	-.07	-.01	1.62	.28**	-.57	-.18*
	(.20)		(.39)		(.28)		(.66)		(.34)	
DRD4	-.32	-.05	-.63	-.08	-.04	-.01	-.74	.12	-.18	-.05
	(.21)		(.41)		(.28)		(.67)		(.40)	
Control Variables										
Age	-.13	-.05	-.18	-.06	-.21	-.10*	-.04	-.02	.30	.22**
	(.09)		(.16)		(.11)		(.31)		(.15)	
Cognitive complexity	.04	.09**	.06	.12**	.04	.12**	.03	.05	-.01	-.07
	(.01)		(.03)		(.02)		(.05)		(.02)	
Race	-1.10	-.11**								
	(.35)									
Gender	1.42	.18**								
	(.25)									
R-squared	.07		.03		.04		.10		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

the alcohol abuse scale with the dopamine genes and with the measure of family risk for the full sample and for the race and gender subsamples. As revealed in Table 5.40, family risk is only significantly related to alcohol abuse for black females, and this effect is in the opposite direction than would be expected. However, the dopamine genes do exert statistically significant and direct effects on the alcohol abuse scale. For instance, DAT1 has a negative effect on alcohol abuse for white females, whereas DRD2 is positively related to alcohol abuse for black males and negatively related to alcohol abuse for black females.

The results for the OLS equations predicting alcohol abuse for the low-risk family group are presented in Table 5.41. In the equations, only one significant genetic effect is found: DRD2 is negatively associated with alcohol abuse for black females. Table 5.42 contains the results for the models employing the high-risk family group. In these models, DAT1 is a positive predictor of alcohol use for white females and DRD2 is positively related to alcohol use for black females.

Summary of the Effects of the Dopamine Genes on Alcohol Abuse

The dopamine genes exerted six significant direct effects on the alcohol abuse scale. These direct effects were observed even after partitioning out the effects of delinquent peers and family risk along with some key control variables. In addition, eight GxEs were detected between the dopamine polymorphisms and the two measures of the social environment (i.e., delinquent peers and family risk) in the OLS models employing the alcohol abuse scale as the dependent variable. In summary, the dopamine genes had important direct effects on alcohol abuse across different race and gender subcategories. However, the genetic effects tended to be even stronger when paired with certain environments—evidence in favor of the role of GxEs in the explanation of alcohol abuse.

Table 5.40. The Direct Effects of Dopamine Genes and Family Risk on Alcohol Abuse

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.11	-.02	.18	.02	-.49	-.09**	.59	.10	-.06	-.02
	(.15)		(.28)		(.21)		(.44)		(.24)	
DRD2	.04	.01	-.16	-.02	.15	.03	.67	.13*	-.39	-.15**
	(.14)		(.29)		(.21)		(.41)		(.18)	
DRD4	-.19	-.03	-.15	-.02	-.16	-.03	-.48	-.09	-.11	-.04
	(.15)		(.30)		(.21)		(.43)		(.20)	
Socialization Variable										
Family risk	.11	.03	.31	.06	.12	.04	-.29	-.07	-.20	-.12*
	(.09)		(.19)		(.12)		(.31)		(.11)	
Control Variables										
Age	-.05	-.02	-.12	-.04	-.14	-.07*	.13	.06	.20	.19**
	(.05)		(.10)		(.08)		(.15)		(.07)	
Cognitive complexity	.03	.10**	.05	.10	.04	.12**	.04	.11	.00	.03
	(.01)		(.02)		(.01)		(.03)		(.01)	
Race	-1.38	-.15**								
	(.23)									
Gender	1.21	.16**								
	(.18)									
R-squared	.07		.02		.03		.06		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.41. The Effects of Dopamine Genes on Alcohol Abuse for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.15	-.02	-.17	-.02	-.37	-.07	.88	.13	-.10	-.03
	(.19)		(.35)		(.27)		(.63)		(.35)	
DRD2	.05	.01	.09	.01	.04	.01	.72	.12	-.44	-.16*
	(.18)		(.37)		(.28)		(.57)		(.25)	
DRD4	-.23	-.04	.19	.03	-.45	-.08	-.68	-.10	-.23	-.07
	(.20)		(.37)		(.29)		(.67)		(.31)	
Control Variables										
Age	-.11	-.05*	-.24	-.10*	-.24	-.12**	.27	.12	.29	.25**
	(.07)		(.13)		(.10)		(.22)		(.10)	
Cognitive complexity	.02	.05*	.03	.07	.03	.08	.01	.02	-.01	-.06
	(.01)		(.03)		(.02)		(.04)		(.01)	
Race	-1.25	-.14**								
	(.29)									
Gender	1.09	.15**								
	(.23)									
R-squared	.05		.01		.03		.06		.10	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.42. The Effects of Dopamine Genes on Alcohol Abuse for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.06	-.01	.66	.08	-.67	-.12**	-.09	-.03	-.13	-.06
	(.23)		(.46)		(.32)		(.43)		(.27)	
DRD2	.03	.01	-.42	-.06	.42	.07	.74	.24*	-.09	-.05
	(.23)		(.44)		(.32)		(.41)		(.23)	
DRD4	-.13	-.02	-.66	-.08	.23	.04	-.29	-.10	.02	.01
	(.23)		(.49)		(.32)		(.36)		(.21)	
Control Variables										
Age	.02	.01	.05	.02	.03	.01	-.15	-.12	.01	.01
	(.09)		(.17)		(.13)		(.15)		(.09)	
Cognitive complexity	.06	.16**	.07	.14**	.06	.17**	.07	.37**	.03	.29
	(.01)		(.03)		(.02)		(.02)		(.01)	
Race	-1.59	-.15**								
	(.38)									
Gender	1.35	.17**								
	(.28)									
R-squared	.10		.04		.05		.17		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Indirect Effects of the Dopamine Genes

Tables 5.43, 5.44, and 5.45 contain the results of the OLS regression equations examining the indirect effects of the dopamine genes. Essentially, the indirect effects models are testing for gene X environment correlations (rGE). Table 5.43 predicts the delinquent peers scale with the dopamine polymorphisms, net of the effects of the control variables. As revealed in Table 5.43, DAT1 has as significant and positive effect on the delinquent peers scale for white males and for black males. DRD4 is also a significant and positive predictor of delinquent peers for black males. These results can be interpreted to mean that as the number of DAT1 risk alleles increases so too does the number of delinquent friends for white males and for black males. Additionally, for black males, as the number of DRD4 risk alleles increases so too does the number of delinquent friends. These significant genetic effects provide empirical evidence of a measured genetic polymorphism predicting a measure of the social environment.

Table 5.44 portrays the findings of the OLS regression models predicting the family risk scale with the dopamine genes. As shown in Table 5.44, there are five significant genetic effects on family risk. Specifically, DRD4 is positively related to family risk for the full sample, for white females, and for black males. For white males, DAT1 and DRD2 are significantly associated with the family risk scale. For all of the dopamine measures, as the number of risk alleles increases, family risk also increases.

Finally, Table 5.45 contains the results of the OLS regression models predicting cognitive complexity with the dopamine genes. Five different significant genetic effects are found in Table 5.45. DRD2 has a negative impact on cognitive complexity for the full sample and for both white males and black males. Similarly, DRD4 has a negative effect on cognitive complexity for the full sample and for black females. Lastly, DAT1 is negatively associated

Table 5.43. The Indirect Effects of Dopamine Genes on Delinquent Peers at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.16	.04	.36	.08**	-.11	-.03	.59	.13*	.18	.04
	(.10)		(.16)		(.15)		(.32)		(.30)	
DRD2	.07	.02	-.04	-.01	.19	.04	.08	.02	.10	.03
	(.09)		(.16)		(.15)		(.30)		(.23)	
DRD4	.12	.03	.14	.03	.01	.00	.51	.12*	.11	.03
	(.10)		(.17)		(.15)		(.31)		(.25)	
Control Variables										
Age	.46	.29**	.46	.29**	.49	.31**	.49	.31**	.29	.21**
	(.04)		(.06)		(.05)		(.11)		(.09)	
Cognitive complexity	-.03	-.13**	-.03	-.11**	-.04	-.13**	-.02	-.09	-.04	-.20**
	(.01)		(.01)		(.01)		(.02)		(.01)	
Race	-.75	-.11**								
	(.15)									
Gender	.17	.03								
	(.12)									
R-squared	.11		.10		.11		.14		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.44. The Indirect Effects of Dopamine Genes on Family Risk at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	.04	.02	.10	.06*	-.05	-.03	.10	.07	.17	.08
	(.04)		(.06)		(.07)		(.11)		(.15)	
DRD2	.05	.03	.10	.07*	.10	.05	-.03	-.03	-.17	-.11
	(.04)		(.06)		(.07)		(.10)		(.11)	
DRD4	.09	.06**	-.04	-.02	.15	.08**	.29	.22**	.13	.08
	(.04)		(.06)		(.07)		(.10)		(.13)	
Control Variables										
Age	.07	.11**	.10	.17**	.07	.10**	.03	.06	.02	.03
	(.01)		(.06)		(.05)		(.04)		(.05)	
Cognitive complexity	-.00	-.01	.01	.01	-.00	-.03	-.00	-.01	-.00	-.00
	(.01)		(.00)		(.00)		(.01)		(.01)	
Race	-.14	-.06**								
	(.06)									
Gender	-.04	-.02								
	(.05)									
R-squared	.02		.04		.02		.06		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.45. The Indirect Effects of Dopamine Genes on Cognitive Complexity

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Dopamine Genes										
DAT1	-.03	-.00	-.69	-.05	-.20	-.01	-.42	-.02	4.09	.17**
	(.37)		(.55)		(.55)		(1.3)		(1.6)	
DRD2	-1.59	-.10**	-1.83	-.12**	-.87	-.06	-3.78	-.24**	-1.27	-.07
	(.36)		(.55)		(.54)		(1.2)		(1.3)	
DRD4	-.74	-.04**	-.11	-.01	-.88	-.06	-.78	-.05	-2.50	-.13*
	(.37)		(.58)		(.56)		(1.2)		(1.4)	
Control Variables										
Age	.56	.09**	.61	.11**	.60	.11**	-.09	-.00	.83	.11
	(.13)		(.20)		(.20)		(.45)		(.52)	
Race	-7.34	-.29**								
	(.15)									
Gender	.65	.03								
	(.44)									
R-squared	.12		.03		.02		.06		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

with cognitive complexity for black females.

Summary of the Indirect Effects of the Dopamine Genes

Behavioral geneticists have long argued that measures of the environment are inextricably tied to an individual's genotype (Harris, 1995, 1998; Rutter, 2006). However, very little empirical research has examined whether certain genetic polymorphisms are predictive of environmental measures (i.e., an rGE). The findings reported in Tables 5.43, 5.44, and 5.45 revealed significant genetic effects on two measures of the social environment (i.e., delinquent peers and family risk) and on a measure of cognitive complexity. Taken together, these findings are supportive of the importance of rGEs when studying the effects of certain environmental measures.

The Serotonin Transporter Polymorphism (5HTT)

The next sets of tables examine the effects that the serotonin transporter polymorphism (5HTT) has on seven different measures of criminal and delinquent behavior. The analytical strategy that will be used to estimate the effects of 5HTT on antisocial conduct is identical to the one used in the previous section with the dopaminergic polymorphisms. For each dependent variable, a direct effects model will be calculated by employing the measure of delinquent peers as the socialization item in the multivariate equations. Next, interactive models will be estimated to determine whether there is a significant GxE between 5HTT and delinquent friends in the etiology of misconduct. Following these models, the exact same equations will be calculated using the family risk measure instead of the delinquent peers scale. Direct effects models and interactive models will be calculated with the family risk scale as a covariate. Finally, a series of

regression equations will be estimated to examine the indirect effects of 5HTT. Specifically, delinquent peers, family risk, and cognitive complexity will be entered into the regression equations as dependent variables. These models will essentially test whether the 5HTT polymorphism has an effect on measures of the social environment and on a measure of cognitive abilities. All of the analyses will be estimated for the full sample and separately for white males, white females, black males, and black females.

Wave I Delinquency Scale

Table 5.46 presents the findings of the OLS regression equations predicting the wave I delinquency scale with the 5HTT polymorphism and with the measure of delinquent peers. As can be seen in Table 5.46, the delinquent peers measure exerts a positive and statistically significant effect on the delinquency scale for all five of the multivariate equations. In contrast, however, the 5HTT gene does not maintain a significant relationship with the dependent variable in any of the equations.

Although 5HTT does not have a direct effect on the wave I delinquency scale, it is possible that 5HTT may interact with delinquent peers to predict delinquent involvement. Table 5.47 and 5.48 examine this possibility by testing for a series of GxEs. Table 5.47 estimates the multivariate equations for the low delinquent peers group and Table 5.48 estimates the OLS models for the high delinquent peers group. The results presented in these two tables reveal that the 5HTT gene does not have any significant effects on the wave I delinquency scale for any of the samples in either Table 5.47 or Table 5.48. Thus, the analysis indicates that there is not a GxE between delinquent peers and the 5HTT polymorphism when predicting delinquent involvement at wave I.

Table 5.46. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Delinquency at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.01	-.00	.17	.02	-.10	-.02	.13	.01	-.18	-.03
	(.15)		(.25)		(.21)		(.63)		(.46)	
Socialization Variable										
Delinquent peers	.88	.46**	.95	.47**	.81	.47**	1.02	.46**	.72	.38**
	(.04)		(.07)		(.06)		(.17)		(.13)	
Control Variables										
Age	-.47	-.15**	-.43	-.13**	-.49	-.18**	-.26	-.07	-.63	-.24**
	(.07)		(.11)		(.09)		(.26)		(.17)	
Cognitive complexity	.02	.03	.00	.00	.01	.02	.09	.15**	.02	.05
	(.01)		(.02)		(.02)		(.04)		(.02)	
Race	.78	.06**								
	(.28)									
Gender	1.17	.12**								
	(.21)									
R-squared	.21		.21		.20		.20		.17	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.47. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave I for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.04	-.01	.17	.04	-.29	-.07	.92	.13	-.61	-.11
	(.16)		(.26)		(.20)		(.77)		(.50)	
Control Variables										
Age	-.11	-.06	-.15	-.08	-.08	-.05	.07	.03	-.19	-.10
	(.06)		(.11)		(.08)		(.29)		(.17)	
Cognitive complexity	-.04	-.04	-.03	-.08	-.02	-.07	-.01	-.02	.02	.08
	(.01)		(.02)		(.02)		(.05)		(.02)	
Race	.54	.07**								
	(.27)									
Gender	.85	.13**								
	(.21)									
R-squared	.03		.01		.02		.02		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.48. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave I for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.07	-.01	-.17	-.02	.14	.02	-.68	-.07	.37	.05
	(.27)		(.44)		(.38)		(1.1)		(.81)	
Control Variables										
Age	-.61	-.16**	-.46	-.11**	-.77	-.21**	.34	.08	-1.06	-.33**
	(.12)		(.21)		(.18)		(.49)		(.33)	
Cognitive complexity	.02	.03	.01	.02	.01	.02	.20	.29**	-.02	-.04
	(.02)		(.03)		(.03)		(.08)		(.04)	
Race	.30	.02								
	(.53)									
Gender	1.54	.13**								
	(.37)									
R-squared	.04		.01		.04		.09		.11	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.49 contains the results of the OLS equations predicting delinquency at wave I with the 5HTT gene and the family risk scale. Similar to the findings reported in Table 5.46, Table 5.49 shows that the 5HTT polymorphism is not a significant predictor of the wave I delinquency scale in any of the multivariate models. As expected, the family risk scale has a statistically significant and positive effect on the wave I delinquency scale for each of the models presented in Table 5.49.

Tables 5.50 and 5.51 estimate the OLS equations separately for the low-risk family group and the high-risk family group to test for GxEs in the prediction of delinquency at wave I. The results for the low-risk family group, presented in Table 5.50, show that 5HTT is negatively predictive of the wave I delinquency scale for the full sample and for white males. The 5HTT gene failed to reach statistical significance for the remaining models in Table 5.50. Additionally, Table 5.51 depicts the results for the high-family risk group and shows that the 5HTT gene is not significantly associated with the wave I delinquency scale in any of the models. Taken together, Tables 5.50 and 5.51 show that there is evidence of a GxE between family risk and 5HTT, but this effect is limited to white males.

Summary of the Effects of 5HTT on the Wave I Delinquency Scale

Tables 5.46 through 5.51 examined the direct and interactive effects of the serotonin transporter polymorphism (5HTT) on the wave I delinquency scale. The results of the analyses did not reveal any evidence showing that the 5HTT gene had a significant direct effect on delinquent involvement as measured at wave I. Across ten different direct effects models, the 5HTT polymorphism did not reach statistical significance once. The multivariate equations also did not detect a statistical interaction between 5HTT and delinquent peers.

Table 5.49. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Delinquency at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.22	-.03	-.30	-.04	-.04	-.01	-.54	-.06	-.29	-.04
	(.16)		(.28)		(.23)		(.67)		(.47)	
Socialization Variable										
Family risk	1.15	.23**	.96	.16**	1.20	.28**	1.31	.19**	1.28	.31**
	(.12)		(.22)		(.15)		(.53)		(.27)	
Control Variables										
Age	-.13	-.04*	-.01	-.00	-.20	-.07**	.09	.02	-.41	-.16**
	(.07)		(.12)		(.10)		(.26)		(.17)	
Cognitive complexity	-.02	-.04	-.04	-.06*	-.02	-.04	.05	.08	-.01	-.03
	(.01)		(.02)		(.02)		(.04)		(.02)	
Race	.25	.02								
	(.30)									
Gender	1.30	.13**								
	(.23)									
R-squared	.07		.03		.09		.05		.12	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.50. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave I for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.42	-.07**	-.89	-.13**	-.14	-.03	-.11	-.01	.20	.03
	(.20)		(.35)		(.25)		(.75)		(.58)	
Control Variables										
Age	-.05	-.02	.07	.03	-.06	-.03	-.01	-.00	-.49	-.22**
	(.08)		(.15)		(.11)		(.30)		(.20)	
Cognitive complexity	-.01	-.03	.00	.00	-.02	- .06	-.01	-.01	-.02	-.05
	(.01)		(.03)		(.02)		(.05)		(.03)	
Race	.33	.03								
	(.35)									
Gender	1.49	.17**								
	(.27)									
R-squared	.04		.02		.01		.00		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.51. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave I for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.05	.01	.47	.06	.16	.02	-1.69	-.16	-1.05	-.14
	(.29)		(.45)		(.44)		(1.4)		(.87)	
Control Variables										
Age	-.18	-.05	-.01	-.00	-.39	-.11**	.34	.08	-.26	-.08
	(.13)		(.20)		(.20)		(.55)		(.36)	
Cognitive complexity	-.03	-.06	-.10	-.16**	-.02	-.04	.15	.25*	-.00	-.01
	(.02)		(.04)		(.03)		(.08)		(.04)	
Race	-.02	-.00								
	(.57)									
Gender	.91	.08**								
	(.41)									
R-squared	.01		.03		.02		.09		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

However, there was some evidence revealing that the 5HTT gene interacted with family risk to predict the wave I delinquency scale. In summary, the 5HTT gene did not have any direct effects delinquent involvement at wave I, but there was a statistically significant interaction between family risk and 5HTT for white males.

Wave II Delinquency Scale

The next set of OLS regression equations estimates the effects of 5HTT and delinquent peers on the wave II delinquency scale. Table 5.52 presents the results of the direct effects models. Across all of the models, the 5HTT polymorphism fails to significantly predict the dependent variable. Similar to the results garnered with the wave I delinquency scale, the measure of delinquent peers is a strong and robust predictor of the dependent variable in all of the models in Table 5.52.

The results for the OLS equations predicting delinquency at wave II for the low delinquent peers group are presented in Table 5.53. Again, the 5HTT gene does not maintain a significant relationship with the delinquency scale in any of the models. Table 5.54 portrays the results for the regression models generated when analyzing the high delinquent peers group. For the full sample, for white males, for white females, and for black males, the 5HTT does not significantly impact the wave II delinquency scale. For black females, however, the 5HTT gene exerts a statistically significant and positive effect on the wave II delinquency scale. More specifically, as the number of risk alleles for black females (in the high delinquent peers group) increases, so too does their involvement in delinquency.

Table 5.55 shows the findings of the direct effects models predicting involvement in delinquency at wave II with the 5HTT polymorphism and with the measure of family risk.

Table 5.52. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Delinquency at Wave II

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.01	-.00	.04	.01	-.16	-.03	.24	.04	.50	.10
	(.12)		(.21)		(.17)		(.47)		(.36)	
Socialization Variable										
Delinquent peers	.44	.31**	.44	.30**	.41	.32**	.61	.37**	.41	.29**
	(.03)		(.06)		(.05)		(.13)		(.10)	
Control Variables										
Age	-.43	-.19**	-.35	-.14**	-.56	-.27**	-.11	-.04	-.54	-.28**
	(.05)		(.09)		(.08)		(.20)		(.13)	
Cognitive complexity	.01	.03	.00	.01	.02	.06*	.01	.02	.01	.05
	(.01)		(.02)		(.01)		(.03)		(.02)	
Race	.23	.03								
	(.23)									
Gender	.42	.06**								
	(.17)									
R-squared	.10		.08		.12		.13		.14	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.53. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave II for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.11	-.03	.01	.00	-.26	-.07	.56	.12	-.54	-.13
	(.15)		(.27)		(.20)		(.52)		(.39)	
Control Variables										
Age	-.13	-.08**	-.05	-.03	-.19	-.12**	.14	.08	-.28	-.21**
	(.06)		(.11)		(.08)		(.21)		(.13)	
Cognitive complexity	-.01	-.04	-.05	-.12**	.00	.01	-.02	-.07	.02	.09
	(.01)		(.02)		(.02)		(.03)		(.02)	
Race	-.21	-.03								
	(.26)									
Gender	.33	.06*								
	(.20)									
R-squared	.01		.02		.02		.02		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.54. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave II for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.06	.01	-.11	-.02	-.00	-.00	-.03	-.01	1.64	.29**
	(.19)		(.31)		(.27)		(.89)		(.60)	
Control Variables										
Age	-.70	-.25**	-.53	-.19**	-.96	-.36**	-.01	-.00	-.95	-.35**
	(.09)		(.15)		(.13)		(.41)		(.26)	
Cognitive complexity	.02	.06*	.03	.07	.03	.06	.03	.06	.01	.02
	(.01)		(.03)		(.02)		(.06)		(.03)	
Race	.32	.03								
	(.39)									
Gender	.53	.06*								
	(.27)									
R-squared	.06		.04		.13		.00		.21	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Similar to the findings reported in Table 5.52, the 5HTT gene fails to have a significant direct effect on the wave II delinquency scale. The family risk measure emerges as a strong and consistent predictor of delinquent involvement at wave II. The only model where family risk is insignificant is the analysis for black males; in all of the other equations, the family risk measure is positively associated with the wave II delinquency scale. In other words, as family risk increases, delinquency also increases.

Tables 5.56 and 5.57 depict the results of the OLS regression equations predicting delinquency at wave II for the low-risk family group and the high-risk family group, respectively. In Table 5.56, the 5HTT polymorphism has a significant and negative effect on delinquency for the full sample and for white males. 5HTT is insignificant in the remaining models in Table 5.56. As shown in Table 5.57, the 5HTT gene is not significant in any of the equations for the high-risk family group. When Tables 5.56 and 5.57 are compared, there is evidence indicating a significant GxE between family risk and 5HTT for white males.

Summary of the Effects of 5HTT on the Wave II Delinquency Scale

The results for the wave II delinquency scale are strikingly similar to those found when employing the wave I delinquency scale as the dependent variable. Specifically, the 5HTT polymorphism did not have a significant direct effect on the wave II delinquency scale. However, when the effects of the 5HTT gene were examined in different social environments, there was limited evidence suggesting that 5HTT plays a role in the etiology of delinquency at wave II. The multivariate models indicated a significant interaction between 5HTT and delinquent peers and between 5HTT and family risk in the prediction of self-reported delinquency at wave II.

Table 5.55. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Delinquency at Wave II

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.12	-.02	-.28	-.05	-.12	-.03	.02	.00	.59	.12
	(.13)		(.21)		(.18)		(.49)		(.36)	
Socialization Variable										
Family risk	.54	.14**	.48	.11**	.64	.19**	.11	.02	.52	.17**
	(.09)		(.17)		(.12)		(.45)		(.21)	
Control Variables										
Age	-.24	-.11**	-.15	-.07*	-.37	-.17**	.17	.07	-.44	-.23**
	(.05)		(.09)		(.08)		(.20)		(.13)	
Cognitive complexity	-.00	-.00	-.07	-.02	.01	.03	-.00	-.01	-.01	-.03
	(.01)		(.02)		(.01)		(.03)		(.02)	
Race	-.02	-.00								
	(.23)									
Gender	.43	.06**								
	(.17)									
R-squared	.03		.02		.06		.01		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.56. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave II for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.26	-.05*	-.72	-.13**	-.08	-.02	-.05	-.01	.57	.11
	(.15)		(.28)		(.20)		(.57)		(.45)	
Control Variables										
Age	-.16	-.08**	-.08	-.04	-.26	-.15**	.22	.09	-.46	-.27**
	(.06)		(.12)		(.08)		(.83)		(.16)	
Cognitive complexity	-.02	-.07**	-.03	-.08	-.00	- .01	-.04	-.10	-.03	-.12
	(.01)		(.02)		(.02)		(.04)		(.02)	
Race	-.02	-.00								
	(.28)									
Gender	.53	.08**								
	(.21)									
R-squared	.02		.02		.03		.02		.10	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.57. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave II for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.08	.01	.26	.05	-.14	-.02	.04	.01	.56	.11
	(.21)		(.31)		(.34)		(.96)		(.61)	
Control Variables										
Age	-.35	-.14**	-.25	-.10*	-.54	-.20**	.18	.06	-.35	-.16
	(.09)		(.14)		(.15)		(.40)		(.26)	
Cognitive complexity	.03	.08**	.02	.06	.03	.07	.06	.15	.04	.13
	(.02)		(.03)		(.03)		(.06)		(.04)	
Race	-.10	-.01								
	(.41)									
Gender	.22	.03								
	(.29)									
R-squared	.02		.01		.05		.02		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Wave III Delinquency Scale

Table 5.58 presents the results of the OLS regression equations predicting the wave III delinquency scale with the 5HTT gene and with the measure of delinquent peers. The results reveal that the 5HTT gene does not have a significant direct effect on delinquency at wave III for the full sample or for any of the gender/race subsamples. The delinquent peers has a significant and positive effect on delinquency at wave III for the full sample, for white males, and for white females; the coefficients for the delinquent peers scale are insignificant for black males and for black females.

Table 5.59 shows the effects of the 5HTT gene on delinquency at wave III for respondents in the low delinquent peer category. Similar to the results for the direct effects models, the 5HTT gene does not have a statistically significant impact on the wave III delinquency scale in any of the models presented in Table 5.59. Table 5.60 contains the results for the high delinquent peers group. The 5HTT gene only has a significant negative impact for the models using the black males subsample; in all of the remaining equations, 5HTT is an insignificant predictor of wave III delinquency.

Table 5.61 shows the direct effects of the 5HTT polymorphism and family risk on delinquency at wave III. The measure of family risk has a significant and positive effect on the wave III delinquency scale for all of the subsamples except for black males. The 5HTT gene, however, does not have significant direct effect on delinquency at wave III in any of the direct effects equations.

Tables 5.62 and 5.63 contain the findings for the analyses based on the low-risk family group and the high-risk family group, respectively. In both tables, the 5HTT gene is not significantly related to the wave III delinquency scale in any of the equations. These models

Table 5.58. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Delinquency at Wave III

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.01	.00	.02	.01	.02	.01	-.02	-.01	.02	.01
	(.05)		(.11)		(.05)		(.20)		(.13)	
Socialization Variable										
Delinquent peers	.07	.11**	.13	.16**	.05	.13**	-.04	-.06	-.01	-.01
	(.02)		(.03)		(.01)		(.05)		(.04)	
Control Variables										
Age	-.13	-.14**	-.27	-.21**	-.05	-.09**	.03	.03	-.09	-.14*
	(.02)		(.05)		(.02)		(.08)		(.05)	
Cognitive complexity	.01	.07**	.03	.11**	.00	.01	.01	.07	.01	.07
	(.00)		(.01)		(.00)		(.01)		(.01)	
Race	.07	.02								
	(.10)									
Gender	.71	.21**								
	(.07)									
R-squared	.07		.06		.02		.01		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.59. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave III for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.06	-.03	-.21	-.06	-.07	-.07	.43	.14	.19	.11
	(.08)		(.18)		(.05)		(.33)		(.16)	
Control Variables										
Age	-.10	-.10**	-.28	-.21**	-.00	-.00	.12	.10	-.03	-.05
	(.03)		(.07)		(.02)		(.13)		(.05)	
Cognitive complexity	.01	.06*	.02	.08	.00	.03	-.03	-.01	.00	.05
	(.01)		(.01)		(.00)		(.02)		(.01)	
Race	.14	.04								
	(.14)									
Gender	.75	.23**								
	(.11)									
R-squared	.06		.06		.01		.03		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.60. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave III for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.06	.03	.14	.05	.11	.06	-.47	-.25**	-.13	-.06
	(.07)		(.15)		(.08)		(.21)		(.22)	
Control Variables										
Age	-.14	-.13**	-.18	-.13**	-.11	-.14**	.00	.00	-.19	-.23**
	(.04)		(.07)		(.04)		(.09)		(.09)	
Cognitive complexity	.01	.07**	.03	.11**	-.00	-.01	.02	.14	.01	.10
	(.01)		(.01)		(.01)		(.01)		(.01)	
Race	-.10	-.02								
	(.15)									
Gender	.69	.21**								
	(.10)									
R-squared	.06		.03		.02		.03		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.61. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Delinquency at Wave III

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.01	-.00	-.05	-.02	.03	.02	-.01	-.00	.03	.02
	(.06)		(.12)		(.05)		(.20)		(.13)	
Socialization Variable										
Family risk	.14	.08**	.17	.07*	.12	.13**	.02	.01	.21	.20**
	(.04)		(.09)		(.03)		(.17)		(.07)	
Control Variables										
Age	-.12	-.12**	-.24	-.18**	-.04	-.06	-.02	-.02	-.10	-.14*
	(.02)		(.05)		(.02)		(.08)		(.05)	
Cognitive complexity	.01	.06**	.02	.08**	-.00	-.01	.01	.06	.01	.08
	(.00)		(.01)		(.00)		(.01)		(.01)	
Race	.05	.01								
	(.10)									
Gender	.76	.22**								
	(.08)									
R-squared	.07		.04		.02		.00		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.62. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave III for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.07	-.03	-.08	-.03	-.03	-.03	-.29	-.12	.00	.00
	(.07)		(.15)		(.05)		(.23)		(.11)	
Control Variables										
Age	-.10	-.11**	-.21	-.17**	-.04	-.08**	.07	.08	-.11	-.25**
	(.03)		(.06)		(.02)		(.09)		(.04)	
Cognitive complexity	.01	.08**	.03	.11**	.00	.01	.02	.11	.01	.10
	(.01)		(.01)		(.00)		(.02)		(.01)	
Race	.09	.03								
	(.12)									
Gender	.78	.25**								
	(.09)									
R-squared	.08		.04		.01		.03		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.63. The Effects of the Serotonin Transporter Gene (5HTT) on Delinquency at Wave III for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.08	.03	-.02	-.01	.11	.06	.45	.16	.06	.02
	(.10)		(.19)		(.10)		(.39)		(.29)	
Control Variables										
Age	-.13	-.11**	-.25	-.16**	-.03	-.04	-.14	-.12	-.07	-.07
	(.04)		(.08)		(.04)		(.16)		(.12)	
Cognitive complexity	.00	.02	.01	.05	-.03	-.03	-.01	-.06	.01	.08
	(.01)		(.02)		(.01)		(.02)		(.02)	
Race	-.05	-.01								
	(.19)									
Gender	.70	.19**								
	(.13)									
R-squared	.05		.03		.01		.05		.01	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

thus reveal that there is not a significant GxE between family risk and the 5HTT gene in the creation of delinquency at wave III.

Summary of the Effects of 5HTT on the Wave III Delinquency Scale

The preceding regression models predicted the wave III delinquency scale with the two measures of the social environment and the 5HTT genetic polymorphism. After controlling for the effects of delinquent peers and family risk, the 5HTT gene did not exert a significant direct effect on wave III delinquency in any of the models. In the interactive effects models, only one significant GxE was detected. Specifically, the 5HTT gene interacted with delinquent peers in the prediction of the wave III delinquency scale for black males in the high delinquent peers group.

Number of Police Contacts

Table 5.64 shows the results of the negative binomial regression equations predicting number of police contacts with the 5HTT gene and with the measure of delinquent peers. As shown in Table 5.64, the measure of delinquent peers has a statistically significant and direct effect on police contacts for the full sample, for white males, and for white females. The delinquent peers coefficient fails to reach statistical significance for black males and black females. The 5HTT gene fails to significant predict police contacts for any of the samples analyzed.

Table 5.65 contains the results of the models predicting number of police contacts for the low delinquent peers group. The 5HTT polymorphism has a significant positive effect on police contacts for black males, but not for any of the other gender/race subsamples. Table 5.66

Table 5.64. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Number of Police Contacts

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.11	.09	.06	.11	.18	.19	.41	.25	-.50	.71
Socialization variable										
Delinquent peers	.14**	.02	.12**	.03	.23**	.05	.09	.06	.10	.16
Control Variables										
Age	-.19**	.04	-.17**	.05	-.43**	.09	-.01	.10	.14	.23
Cognitive complexity	.00	.01	.00	.01	.01	.02	.02	.02	-.04	.04
Race	-.03	.17								
Gender	1.60**	.13								
Pseudo R-squared	.07		.02		.06		.02		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.65. The Effects of the Serotonin Transporter Gene (5HTT) on Number of Police Contacts for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.15	.14	.15	.18	-.10	.29	.65**	.31	-.35	1.73
Control Variables										
Age	-.15**	.06	-.16**	.07	-.40**	.15	.04	.12	.54	.56
Cognitive complexity	-.01	.01	-.02	.01	-.01	.02	.02	.02	-.10	.11
Race	-.00	.24								
Gender	1.78**	.21								
Pseudo R-squared	.08		.01		.04		.03		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.66. The Effects of the Serotonin Transporter Gene (5HTT) on Number of Police Contacts for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.05	.11	-.03	.15	.36	.25	.01	.41	-.89	.89
Control Variables										
Age	-.16**	.05	-.12*	.06	-.35**	.12	.08	.21	-.17	.27
Cognitive complexity	.01	.01	.01	.01	.01	.02	.03	.03	-.01	.04
Race	-.17	.24								
Gender	1.45**	.17								
Pseudo R-squared	.05		.01		.03		.01		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

reveals that the 5HTT gene is not significantly related to police contacts for any of the negative binomial regression equations. Taken together, Tables 5.65 and 5.66 show a significant interaction between 5HTT and delinquent peers in the prediction of police contacts, but this GxE is confined only to black males.

Table 5.67 presents the results of the negative binomial regression models predicting number of police contacts with the 5HTT gene and with the measure of family risk. The family risk scale exerts a statistically significant positive effect on police contacts for the full sample, for white males, for white females, and for black females; the family risk coefficient for black males is insignificant. As revealed in Table 5.67, the 5HTT gene does not have a significant direct effect on police contacts in any of the negative binomial regression models.

Table 5.68 shows the results of the negative binomial models predicting number of police contacts for the low-risk family group. Similar to the findings for the direct effects models, the 5HTT gene is not a significant predictor of police contacts in any of the models. Likewise, and as a shown in Table 5.69, the 5HTT polymorphism does not exert a significant effect on police contacts for the high-risk family group. The findings reported in Tables 5.68 and 5.69 do not reveal any evidence of a GxE between 5HTT and family risk when using police contacts as the dependent variable.

Summary of the Effects of 5HTT on Number of Police Contacts

The results of the negative binomial regression equations revealed that the 5HTT gene had very limited effects in the prediction of police contacts. For example, the 5HTT polymorphism did not exert a significant direct effect on police contacts for any of the direct effects models presented in Tables 5.64 and 5.67. There was one significant GxE between

Table 5.67. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Number of Police Contacts

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.05	.09	-.05	.11	.30	.19	.22	.24	-.68	.69
Socialization variable										
Family risk	.24**	.06	.18**	.08	.24*	.13	.21	.18	.66**	.26
Control Variables										
Age	-.12**	.04	-.11**	.05	-.29**	.09	.03	.10	-.01	.22
Cognitive complexity	.00	.01	-.00	.01	-.01	.02	.02	.02	-.03	.03
Race	-.11	.16								
Gender	1.65**	.14								
Pseudo R-squared	.06		.01		.02		.01		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.68. The Effects of the Serotonin Transporter Gene (5HTT) on Number of Police Contacts for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.02	.12	-.08	.14	.25	.29	.15	.32	-.74	1.08
Control Variables										
Age	-.16**	.05	-.17**	.06	-.25**	.13	-.01	.14	-.07	.29
Cognitive complexity	.01	.01	.01	.01	-.01	.02	.02	.02	-.02	.03
Race	-.09	.22								
Gender	1.88**	.19								
Pseudo R-squared	.08		.01		.02		.00		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.69. The Effects of the Serotonin Transporter Gene (5HTT) on Number of Police Contacts for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.07	.13	-.03	.16	.33	.26	.48	.38	-.36	.92
Control Variables										
Age	-.06	.05	.00	.07	-.34**	.13	.17	.16	.21	.30
Cognitive complexity	-.00	.01	-.02	.01	.02	.02	.02	.02	-.04	.05
Race	-.14	.25								
Gender	1.34**	.19								
Pseudo R-squared	.04		.00		.03		.03		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

delinquent peers and 5HTT, but this GxE was limited to black males.

Ever Arrested

Table 5.70 presents the results of the binary logistic regression equations predicting arrest status (yes/no) with the 5HTT gene and with the measure of delinquent peers. For all of the models excluding black females, the delinquent peers scale has a statistically significant and direct effect on arrest status. In contrast, in every model the 5HTT gene failed to reach statistical significance. These findings can be interpreted to mean that the 5HTT gene does not have a significant direct effect on arrest status when controlling for the effects of delinquent peers.

However, it could be the case that 5HTT interacts with delinquent peers to predict arrest status. To examine this possibility, Table 5.71 estimates the models for the low delinquent peers group and Table 5.72 estimates the models for the high delinquent peers group. In Table 5.71, the 5HTT gene is not a significant predictor of arrest status in any of the models. For the analyses using the high delinquent peers group, the 5HTT gene emerges as a positive predictor of arrest status for white females. The results reported in Tables 5.71 and 5.72 indicate that there is a significant GxE between 5HTT and delinquent peers when predicting arrest status for white females.

Table 5.73 contains the results of the binary logistic regression equations predicting arrest status with the 5HTT gene and with the family risk scale. The measure of family risk exerts a statistically significant and positive effect on arrest status for the full sample, for white males, and for black males. In contrast, the family risk measure is insignificant for white females and black females. Additionally, the 5HTT polymorphism has a significant positive effect on arrest status for white females. The 5HTT gene does not exert a significant direct effect in any of the

Table 5.70. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Arrest Status

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.17	.12	.12	.15	.43	.27	.05	.29	-.24	1.17
Socialization Variable										
Delinquent peers	.23**	.03	.23**	.04	.27**	.07	.15**	.08	.36	.27
Control Variables										
Age	-.22**	.05	-.22**	.07	-.27**	.13	-.11	.13	-.97**	.53
Cognitive complexity	.01	.01	.00	.01	.02	.02	.01	.02	.00	.05
Race	.18	.22								
Gender	1.84**	.20								
Cox & Snell R-squared	.08		.05		.02		.02		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.71. The Effects of the Serotonin Transporter Gene (5HTT) on Arrest Status for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.12	.20	.13	.26	-.07	.48	.32	.39	---	---
Control Variables										
Age	-.20**	.08	-.25**	.11	-.31	.23	-.05	.16	---	---
Cognitive complexity	-.01	.01	-.03	.02	-.01	.04	.01	.03	---	---
Race	.36	.33								
Gender	2.00**	.36								
Cox & Snell R-squared	.06		.03		.01		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.72. The Effects of the Serotonin Transporter Gene (5HTT) on Arrest Status for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.12	.14	.03	.18	.69**	.34	-.44	.47	1.21	1.64
Control Variables										
Age	-.13*	.07	-.13	.08	-.16	.16	.03	.19	-2.37*	1.31
Cognitive complexity	.01	.01	.02	.01	.02	.03	-.00	.03	.01	.07
Race	-.14	.30								
Gender	1.70**	.25								
Cox & Snell R-squared	.06		.01		.01		.01		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.73. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Arrest Status

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.04	.12	-.10	.15	.52*	.27	-.02	.28	-.19	1.10
Socialization Variable										
Family risk	.29**	.08	.32**	.11	.23	.15	.37*	.20	.32	.48
Control Variables										
Age	-.10*	.05	-.10	.06	-.11	.12	-.03	.11	-.77	.48
Cognitive complexity	-.00	.01	-.01	.01	.01	.02	.01	.02	-.01	.04
Race	.03	.21								
Gender	1.81**	.20								
Cox & Snell R-squared	.06		.02		.01		.02		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.74. The Effects of the Serotonin Transporter Gene (5HTT) on Arrest Status for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	-.13	.17	-.28	.21	.59	.40	-.30	.40	---	---
Control Variables										
Age	-.11*	.07	-.15*	.09	-.08	.17	-.04	.15	---	---
Cognitive complexity	-.01	.01	-.01	.02	-.03	.03	-.01	.03	---	---
Race	-.10	.29								
Gender	1.93**	.30								
Cox & Snell R-squared	.05		.01		.01		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.75. The Effects of the Serotonin Transporter Gene (5HTT) on Arrest Status for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.17	.16	.05	.20	.48	.36	.27	.43	.54	1.32
Control Variables										
Age	-.05	.07	-.02	.09	-.14	.16	.09	.19	-1.91	1.11
Cognitive complexity	.01	.01	-.01	.02	.05	.03	.03	.03	-.02	.06
Race	.13	.32								
Gender	1.62**	.27								
Cox & Snell R-squared	.06		.00		.01		.03		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

other statistical models.

To test for GxEs in the prediction of arrest status, the models are next estimated separately for the low-risk family group and for the high-risk family group. Table 5.74 contains the results for the logistic regression equations using the low-risk family group. In these models, the 5HTT fails to significantly predict arrest status. These insignificant findings are also replicated in Table 5.75 with the high-risk family group. The findings reported in Tables 5.74 and 5.75 do not provide any evidence of a GxE between 5HTT and family risk when predicting arrest status.

Summary of the Effects of 5HTT on Arrest Status

Tables 5.70 through 5.75 examined the direct and interactive effects of the 5HTT gene on arrest status. The results of these models revealed two broad findings. First, the 5HTT gene only had one direct effect on arrest status and this significant finding was for white females (Table 5.73). Second, only one significant GxE (between 5HTT and delinquent peers) was detected and, again, this finding was observed only for white females. In summary, the 5HTT has no effect on arrest status for white males, black males, and black females; however, the 5HTT gene did have one direct and interactive effect on arrest status for white females.

Marijuana Use

Table 5.76 contains the results of the negative binomial regression equations predicting frequency of marijuana use with the 5HTT polymorphism and with the measure of delinquent peers. As shown in Table 5.76, the delinquent peers scale is the strongest and most consistent predictor of marijuana use. Indeed, it has a statistically significant and positive effect in each of

Table 5.76. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Frequency of Marijuana Use

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	-.27*	.15	-.41	.27	.20	.20	-.16	.44	1.00	.70
Socialization Variable										
Delinquent peers	.70**	.04	.86**	.09	.70**	.06	1.03**	.14	.46**	.14
Control Variables										
Age	.08	.08	.05	.12	.11	.11	-.11	.22	-.10	.29
Cognitive complexity	.03**	.01	.09**	.02	-.01	.02	.05	.03	-.03	.05
Race	.38	.31								
Gender	.64**	.21								
Pseudo R-squared	.11		.11		.14		.23		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.77. The Effects of the Serotonin Transporter Gene (5HTT) on Frequency of Marijuana Use for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	.20	.56	.77	1.08	-.01	.73	.80	1.37	---	---
Control Variables										
Age	.83**	.34	1.69*	.89	.44	.63	-.08	.60	---	---
Cognitive complexity	-.07	.05	.02	.10	-.04	.07	.02	.10	---	---
Race	-.14	1.22								
Gender	-1.13	.90								
Pseudo R-squared	.06		.17		.01		.04		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of marijuana use for black females in the low delinquent peers group precluded the model from converging.

Table 5.78. The Effects of the Serotonin Transporter Gene (5HTT) on Frequency of Marijuana Use for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	-.42**	.16	-.60**	.27	-.31	.25	-.73	.57	.70	.50
Control Variables										
Age	.16*	.09	.23	.14	.12	.14	.61*	.32	-.17	.28
Cognitive complexity	.02**	.01	.06**	.02	-.01	.02	.11**	.06	-.00	.02
Race	-.18	.34								
Gender	.54**	.25								
Pseudo R-squared	.01		.01		.00		.03		.01	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

the five models estimated in Table 5.76. Additionally, in the full sample model, the 5HTT polymorphism has a statistically significant and negative direct effect on marijuana use. In the remaining four models, however, the 5HTT gene fails to reach statistical significance.

Table 5.77 presents the results of the negative binomial regression equations predicting frequency of marijuana use for the low delinquent peers group. The 5HTT is not a significant predictor of marijuana use for any of the models estimated with the low delinquent peers sample. Table 5.78 contains the results of the statistical models estimated with the high delinquent peers group. The 5HTT polymorphism has significant negative effects on marijuana use for the full sample and for white males. The findings in Tables 5.77 and 5.78 indicate a significant GxE between 5HTT and delinquent peers in the etiology of marijuana use.

Table 5.79 displays the results of the negative binomial regression equations predicting frequency of marijuana use with the 5HTT gene and with the measure of family risk. The measure of family risk has a statistically significant direct effect on marijuana use for the full sample for white females, for black males, and for black females. Most importantly, however, are the significant findings for the 5HTT gene. As shown in the top row of Table 5.79, the 5HTT gene exerts a statistically significant direct effect on marijuana use for all of the negative binomial regression models. For black females the relationship between 5HTT and marijuana use is positive, whereas in the remaining models the 5HTT gene is inversely related to marijuana use.

Tables 5.80 and 5.81 examine the effects of the 5HTT gene for respondents in the low-risk family group and for respondents in the high-risk family group, respectively. Table 5.80 shows that the 5TT gene is a negative predictor of marijuana use for the full sample and for white males. However, the 5HTT gene is not significantly related to marijuana use in any of the

Table 5.79. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Frequency of Marijuana Use

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	-.58**	.17	-.94**	.32	-.48*	.26	-1.13**	.56	1.23**	.47
Socialization Variable										
Family risk	.35**	.14	.23	.22	.43*	.25	1.45**	.44	.79**	.31
Control Variables										
Age	.38**	.09	.40**	.15	.31**	.14	.74**	.22	.12	.25
Cognitive complexity	.01	.01	.06**	.02	-.02	.02	.06	.05	-.02	.03
Race	-.55*	.32								
Gender	.58**	.26								
Pseudo R-squared	.02		.02		.01		.07		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.80. The Effects of the Serotonin Transporter Gene (5HTT) on Frequency of Marijuana Use for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	-.51*	.28	-1.37**	.44	-.56	.52	-1.67	-1.13	.96	.70
Control Variables										
Age	.57**	.16	.49**	.25	.24	.32	.74**	.29	.18	.37
Cognitive complexity	.04*	.02	.06**	.03	-.03	.03	.05	.08	.01	.06
Race	-.98**	.49								
Gender	.49	.44								
Pseudo R-squared	.02		.03		.01		.09		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.81. The Effects of the Serotonin Transporter Gene (5HTT) on Frequency of Marijuana Use for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Serotonin Gene										
5HTT	-.28	.22	.03	.41	-.37	.31	-1.04	.67	-.99	.99
Control Variables										
Age	.31**	.11	.35**	.18	.31*	.16	.79*	.43	-.55	.46
Cognitive complexity	.00	.02	.02	.03	-.02	.03	.10	.07	-.02	.04
Race	.11	.46								
Gender	.36	.33								
Pseudo R-squared	.01		.01		.01		.03		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

models estimated for the high-risk family group. Taken together, Tables 5.80 and 5.81 reveal a significant GxE between the 5HTT gene and family risk in the etiology of marijuana use for white males.

Summary of the Effects of 5HTT on Marijuana Use

The negative binomial regression models presented in Tables 5.76 through 5.81 revealed considerable support for the role of the 5HTT gene in the etiology of marijuana use. Across all of the models, there were six significant direct effects of the 5HTT polymorphism on frequency of marijuana use. Similarly, and just as important, were the results garnered from the interactive models. In total, four different statistical interactions were detected between the 5HTT gene and delinquent peers and between the 5HTT gene and family risk.

Alcohol Abuse

Table 5.82 presents the results of the OLS regression equations predicting scores on the alcohol abuse scale with the 5HTT gene and with the measure of delinquent peers. The delinquent peers scale exerts a statistically significant and positive effect on alcohol abuse for all of the models estimated in Table 5.82. However, the 5HTT is a not statistically related to alcohol abuse in any of the models found in Table 5.82.

Tables 5.83 and 5.84 examine whether the 5HTT gene interacts with the measure of delinquent peers to predict scores on the alcohol abuse scale. Table 5.83 contains the results of the OLS regression equations for the low delinquent peers group. As shown in Table 5.83, the 5HTT gene is not a statistically significant predictor of alcohol abuse in any of the models. Similarly, Table 5.84 reveals that the 5HTT gene fails to predict a significant amount of variation

Table 5.82. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Delinquent Peers on Alcohol Abuse

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.11	.02	.11	.02	.19	.04	-.11	-.02	.27	.10
	(.12)		(.23)		(.17)		(.38)		(.19)	
Socialization Variable										
Delinquent peers	.19	.13**	.28	.17**	.13	.10**	.20	.15*	.12	.16**
	(.03)		(.06)		(.05)		(.10)		(.05)	
Control Variables										
Age	-.15	-.06**	-.25	-.10**	-.20	-.10**	.17	.09	.13	.12*
	(.05)		(.10)		(.08)		(.16)		(.07)	
Cognitive complexity	.04	.12**	.06	.12**	.04	.13**	.05	.14*	.01	.04
	(.01)		(.02)		(.01)		(.02)		(.01)	
Race	-1.22	-.13**								
	(.23)									
Gender	1.15	.15**								
	(.17)									
R-squared	.08		.04		.03		.06		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.83. The Effects of the Serotonin Transporter Gene (5HTT) on Alcohol Abuse for the Low Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.03	-.01	.02	.00	-.06	-.01	-.01	-.00	.02	.01
	(.17)		(.33)		(.25)		(.40)		(.18)	
Control Variables										
Age	-.11	-.05	-.20	-.08	-.16	-.08	.15	.10	.07	.10
	(.07)		(.13)		(.10)		(.16)		(.06)	
Cognitive complexity	.04	.13**	.05	.10*	.04	.11**	.06	.27**	.02	.24**
	(.01)		(.03)		(.02)		(.02)		(.01)	
Race	-1.48	-.17**								
	(.29)									
Gender	.94	.13**								
	(.23)									
R-squared	.08		.02		.02		.10		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.84. The Effects of the Serotonin Transporter Gene (5HTT) on Alcohol Abuse for the High Delinquent Peers Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.23	.04	.07	.01	.42	.09	-.26	-.04	.50	.15
	(.17)		(.32)		(.23)		(.68)		(.36)	
Control Variables										
Age	-.13	-.05	-.20	-.07	-.25	-.11**	.19	.07	.24	.18**
	(.08)		(.16)		(.11)		(.31)		(.14)	
Cognitive complexity	.04	.11**	.06	.13**	.05	.14**	.03	.07	-.02	-.09
	(.01)		(.03)		(.02)		(.05)		(.02)	
Race	-1.07	-.11**								
	(.35)									
Gender	1.38	.17**								
	(.25)									
R-squared	.06		.02		.04		.01		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

in alcohol abuse for the high delinquent peers group. Taken together, Tables 5.83 and 5.84 do not reveal any evidence indicating an interaction between the 5HTT gene and delinquent peers in the prediction of alcohol abuse.

Table 5.85 contains the results of the OLS regression equations predicting scores on the alcohol abuse scale with the 5HTT gene and with the measure of family risk. The family risk scale is significantly predictive of alcohol abuse only for white males. Likewise, the 5HTT gene has a significant and positive direct effect on the alcohol abuse scale for black females; however, it is insignificant in the remaining four models.

Tables 5.86 and 5.87 estimate the OLS regression models separately for the low-risk family group and for the high-risk family group, respectively. The only significant finding for the 5HTT gene in Table 5.86 is for black females. Specifically, the 5HTT gene has a significant positive effect on alcohol abuse for black females residing in low-risk families. Table 5.87 depicts the results of the regression models using the high-risk family group. The 5HTT gene does not have a significant effect on the alcohol abuse scale in any of the models presented in Table 5.87.

Summary of the Effects of 5HTT on Alcohol Abuse

Tables 5.82 through 5.87 revealed the direct and interactive effects that the 5HTT gene had on alcohol abuse. The findings reported in these tables showed that the 5HTT gene had a significant direct effect on the alcohol abuse scale for black females. Similarly, the multivariate models revealed a statistically significant GxE between the 5HTT polymorphism and family risk in the prediction of alcohol abuse for black females. Taken together, the 5HTT gene had effects on the alcohol abuse, but these effects were limited to black females.

Table 5.85. The Direct Effects of the Serotonin Transporter Gene (5HTT) and Family Risk on Alcohol Abuse

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.04	.01	-.10	-.02	.21	.04	-.25	-.05	.32	.11*
	(.12)		(.23)		(.17)		(.37)		(.19)	
Socialization Variable										
Family risk	.10	.03	.32	.06*	.12	.04	-.29	-.07	-.17	-.10
	(.09)		(.19)		(.11)		(.29)		(.11)	
Control Variables										
Age	-.05	-.02	-.12	-.05	-.15	-.07**	.20	.10	.17	.17**
	(.05)		(.10)		(.08)		(.15)		(.07)	
Cognitive complexity	.04	.11**	.06	.11**	.05	.13**	.04	.12	.00	.02
	(.01)		(.02)		(.01)		(.02)		(.01)	
Race	-1.36	-.15**								
	(.23)									
Gender	1.20	.16**								
	(.17)									
R-squared	.07		.02		.02		.03		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.86. The Effects of the Serotonin Transporter Gene (5HTT) on Alcohol Abuse for the Low-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	.13	.02	.26	.04	.10	.02	-.64	-.11	.50	.15*
	(.16)		(.30)		(.22)		(.54)		(.28)	
Control Variables										
Age	-.11	-.05*	-.22	-.09*	-.24	-.12**	.37	.16**	.24	.21**
	(.07)		(.12)		(.09)		(.21)		(.10)	
Cognitive complexity	.03	.07**	.04	.09*	.03	.09*	.02	.04	-.01	-.08
	(.01)		(.02)		(.02)		(.04)		(.01)	
Race	-1.18	-.13**								
	(.28)									
Gender	1.08	.15**								
	(.22)									
R-squared	.05		.02		.02		.04		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.87. The Effects of the Serotonin Transporter Gene (5HTT) on Alcohol Abuse for the High-Risk Family Group

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.09	-.02	-.59	-.09	.36	.07	.28	.10	-.03	-.02
	(.19)		(.37)		(.28)		(.35)		(.22)	
Control Variables										
Age	.02	.01	.06	.02	-.01	-.00	-.03	- .02	.01	.02
	(.09)		(.17)		(.13)		(.15)		(.09)	
Cognitive complexity	.06	.15**	.07	.13**	.06	.17**	.06	.32**	.03	.29
	(.01)		(.03)		(.02)		(.02)		(.01)	
Race	-1.64	-.16**								
	(.38)									
Gender	1.35	.17**								
	(.27)									
R-squared	.10		.03		.03		.12		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Indirect Effects of 5HTT

Tables 5.88, 5.89, and 5.90 contain the results of the OLS regression equations examining the indirect effects of the serotonin transporter gene (5HTT). The indirect effects models are testing for gene X environment correlations (rGE). Table 5.88 presents the results for the regression models predicting scores on the delinquent peers scale with the 5HTT gene. As revealed in Table 5.88, the 5HTT gene is negatively related to the delinquent peers scale for the full sample and for white males. The effects of 5HTT dissipate from statistical significance when estimated for the other gender/race subsamples.

Table 5.89 presents the result of the OLS regression equations predicting scores on the family risk scale with the 5HTT gene. These models reveal that the 5HTT gene does not have a significant direct effect on family risk for any of the models estimated in Table 5.89. That is, the measure of family risk, at least as measured in the Add Health data, is not affected by the presence of different 5HTT alleles.

Lastly, Table 5.90 contains the results for the OLS regression models predicting cognitive complexity with the 5HTT gene. The 5HTT polymorphism has a negative effect on cognitive complexity for white males.

Summary of the Indirect Effects of the 5HTT Gene

The indirect effects models examined whether the 5HTT had a significant impact on the delinquent peers scale, on the measure of family risk, and on the cognitive complexity variable. In total, three different indirect effects were found for the 5HTT polymorphism. First, 5HTT was significantly related to the delinquent peers scale for the full sample. Second, the 5HTT polymorphism exerted a statistically significant effect on the delinquent peers scale for white

Table 5.88. The Indirect Effects of the Serotonin Transporter Gene (5HTT) on Delinquent Peers at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.14	-.04*	-.40	-.11**	.09	.02	-.20	-.05	.02	.01
	(.08)		(.13)		(.13)		(.28)		(.25)	
Control Variables										
Age	.46	.29**	.47	.29**	.48	.30**	.53	.34**	.26	.19**
	(.03)		(.06)		(.05)		(.11)		(.09)	
Cognitive complexity	-.04	-.14**	-.04	-.13**	-.04	-.14**	-.02	-.09	-.04	-.21**
	(.01)		(.01)		(.01)		(.02)		(.01)	
Race	-.78	-.12**								
	(.15)									
Gender	.15	.03								
	(.12)									
R-squared	.11		.10		.10		.13		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.89. The Indirect Effects of the Serotonin Transporter Gene (5HTT) on Family Risk at Wave I

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.00	-.00	.02	.02	-.01	-.01	.03	.02	-.04	-.02
	(.03)		(.05)		(.06)		(.10)		(.12)	
Control Variables										
Age	.06	.11**	.09	.17**	.06	.09**	.04	.07	.02	.03
	(.01)		(.02)		(.02)		(.04)		(.05)	
Cognitive complexity	-.00	-.02	.00	.00	-.00	-.04	-.00	-.01	.00	.01
	(.00)		(.00)		(.00)		(.01)		(.01)	
Race	-.13	-.05**								
	(.06)									
Gender	-.03	-.02								
	(.05)									
R-squared	.01		.03		.01		.01		.00	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.90. The Indirect Effects of the Serotonin Transporter Gene (5HTT) on Cognitive Complexity

	Full Sample		White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Serotonin Gene										
5HTT	-.42	-.03	-.91	-.07*	-.26	-.02	-.32	-.02	.70	.03
	(.32)		(.47)		(.46)		(1.2)		(1.4)	
Control Variables										
Age	.56	.09**	.51	.09**	.61	.11**	.11	.02	.87	.12*
	(.13)		(.20)		(.19)		(.45)		(.50)	
Race	-8.32	-.33**								
	(.55)									
Gender	.63	.03								
	(.44)									
R-squared	.12		.01		.01		.00		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

males. Third, the 5HTT gene had a significant and negative impact on the cognitive complexity scale. In summary, the 5HTT has some small effects on measures of the social environment, indicating three significant rGEs.

The Monoamine Oxidase A Promoter Polymorphism (MAOA)

The final series of multivariate models will examine the direct and interactive effects that the monoamine oxidase A promoter polymorphism (MAOA) has on a seven antisocial outcomes. The analytic strategy to estimate the direct and interactive effects of MAOA will be very similar to the ones employed for the dopaminergic polymorphisms and for the serotonin transporter polymorphism (5HTT). The only difference is that the models are not estimated for the full sample. Remember that MAOA is a located on the X chromosome. Since males have only one X chromosome, whereas females have two X chromosomes, the coding of the MAOA genes were slightly different between males and females (see Chapter 4). As a result, the models were only calculated for the race/gender subsamples.

In line with the previous models, each dependent variable will be regressed on the MAOA gene, the delinquent peers scale, and the key control variables. In order to examine whether MAOA interacts with delinquent peers, each model will be calculated separately for respondents in the low delinquent peers group and for respondents in the high delinquent peers group. Second, each outcome measure will be predicted with the MAOA gene, the family risk scale, and the control variables. Next, to test whether the effects of MAOA are conditioned by family risk, each model will be estimated first for low-risk families and then for high-risk families. These models will provide specific information about whether MAOA interacts with family risk to predict a range of antisocial outcomes. Lastly, the indirect models will be

calculated to determine whether MAOA is implicated in gene X environment correlations (rGEs). Specifically, the delinquent peers scale, the family risk measure, and cognitive complexity will be used as dependent variables in three separate regression models. The MAOA gene will be the independent variable of interest. If MAOA is a significant predictor of either delinquent peers or family risk, then an rGE will have been detected. Thus, the proceeding analyses will provide a comprehensive examination of the direct, indirect, and interactive effects of MAOA.

Wave I Delinquency Scale

Table 5.91 contains the results of the OLS regression models predicting the wave I delinquency scale with the MAOA gene and with the measure of delinquent peers. As can be seen in Table 5.91, the measure of delinquent peers is the strongest predictor of delinquency for white males, white females, black males, and black females. However, the MAOA gene is not a significant predictor of delinquency at wave I for any of the race/gender subsamples.

In order to explore the possibility that the MAOA gene interacts with delinquent peers in the etiology of delinquency, the OLS regression equations were calculated separately for the low delinquent peers group and for the high delinquent peers group. The results of these models are presented in Tables 5.92 and 5.93. Table 5.92 shows that the MAOA gene is not a significant predictor of the wave I delinquency scale for any of the models calculated with the low delinquent peers group. Similarly, Table 5.93 reveals that MAOA is not related to delinquent involvement for the models calculated with the high delinquent peer group. Taken together, the multivariate models do not provide any evidence indicating a GxE between MAOA and delinquent peers in the prediction of the wave I delinquency scale.

Table 5.91. The Direct Effects of MAOA and Delinquent Peers on Delinquency at Wave I

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.18	.02	.04	.01	.23	.02	.04	.01
	(.37)		(.22)		(.84)		(.38)	
Socialization Variable								
Delinquent peers	.95	.47**	.82	.47**	1.02	.46**	.69	.36**
	(.07)		(.06)		(.17)		(.13)	
Control Variables								
Age	-.43	-.13**	-.48	-.17**	-.25	-.07	-.65	-.25**
	(.11)		(.09)		(.26)		(.17)	
Cognitive complexity	-.02	-.00	.01	.01	.09	.15**	.01	.03
	(.02)		(.02)		(.04)		(.02)	
R-squared	.20		.21		.21		.16	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.92. The Effects of MAOA on Delinquency at Wave I for the Low Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.13	.02	-.05	-.01	-.06	-.01	.09	.02
	(.38)		(.20)		(1.1)		(.41)	
Control Variables								
Age	-.14	-.07	-.07	-.05	.01	.00	-.20	-.11
	(.11)		(.08)		(.30)		(.17)	
Cognitive complexity	-.04	-.09	-.02	-.07	.01	.03	.02	.08
	(.02)		(.02)		(.05)		(.03)	
R-squared	.02		.01		.00		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.93. The Effects of MAOA on Delinquency at Wave I for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.07	.01	-.06	-.01	1.01	.08	.28	.04
	(.64)		(.40)		(1.5)		(.68)	
Control Variables								
Age	-.45	-.11**	-.75	-.20**	.39	.09	-1.07	-.33**
	(.21)		(.18)		(.51)		(.32)	
Cognitive complexity	.01	.02	.01	.01	.20	.29**	-.02	-.06
	(.03)		(.03)		(.08)		(.04)	
R-squared	.01		.04		.08		.12	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

The results for the OLS regression equations predicting the wave I delinquency scale with the MAOA gene and with the family risk scale are presented in Table 5.94. As revealed in this table, the measure of family risk maintains a significant and positive association with the dependent variable in all of the equations listed in Table 5.94. Of particular interest are the findings for the MAOA polymorphism. The top row of Table 5.94 shows that the MAOA gene is not related to delinquent involvement at wave I for any of the race/gender subsamples.

Table 5.95 presents the result for the models predicting the wave I delinquency scale for the low-risk family group. Similar to the findings for direct effects model (Table 5.94), the MAOA gene is not related to the dependent variable in any of the models. As shown in Table 5.96 for the models estimated for the high-risk family group, MAOA is negatively related to the wave I delinquency scale for white females; MAOA is not a significant predictor of the dependent variable for any of the other models. The findings from Tables 5.95 and 5.96 reveal a GxE between MAOA and family risk in the prediction of delinquent involvement at wave I, but this interaction is confined only to white females.

Summary of the Effects of MAOA on the Wave I Delinquency Scale

Tables 5.91 through 5.96 estimated the direct and interactive effects that the MAOA gene had on the wave I delinquency scale. The results of these models did not provide any evidence that different alleles of the MAOA gene had a significant direct effect on delinquent involvement. Indeed, MAOA was not statistically related to the wave I delinquency scale in any of the direct effects models. Similarly, for the majority of the interactive models, MAOA was not a significant predictor of the dependent variable. However, for white females in high-risk families, MAOA was significantly related to the wave I delinquency scale. Taken together, there is some evidence suggesting that MAOA interacts with family risk to predict delinquency for

Table 5.94. The Direct Effects of MAOA and Family Risk on Delinquency at Wave I

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.23	.02	-.29	-.04	-.28	-.02	-.20	-.03
	(.42)		(.24)		(.93)		(.39)	
Socialization Variable								
Family risk	.94	.16**	1.21	.28**	1.29	.18**	1.29	.31**
	(.23)		(.06)		(.54)		(.27)	
Control Variables								
Age	-.01	-.00	-.20	-.07*	.08	.02	-.43	-.16**
	(.12)		(.10)		(.27)		(.17)	
Cognitive complexity	-.04	-.07*	-.02	-.04	.06	.10	-.01	-.04
	(.02)		(.02)		(.47)		(.02)	
R-squared	.03		.09		.04		.13	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.95. The Effects of MAOA on Delinquency at Wave I for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.75	.07	.11	.02	.12	.01	-.18	-.03
	(.54)		(.27)		(1.0)		(.49)	
Control Variables								
Age	.09	.03	-.06	-.03	-.02	-.01	-.50	-.22**
	(.15)		(.11)		(.30)		(.20)	
Cognitive complexity	.01	.01	-.03	-.06	.00	.00	-.02	-.05
	(.03)		(.02)		(.05)		(.03)	
R-squared	.01		.01		.00		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.96. The Effects of MAOA on Delinquency at Wave I for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.26	-.02	-.75	-.10*	-1.83	-.12	-.14	-.02
	(.65)		(.44)		(2.1)		(.73)	
Control Variables								
Age	.00	.00	-.36	-.10*	.26	.06	-.27	-.09
	(.20)		(.20)		(.58)		(.36)	
Cognitive complexity	-.10	-.16	-.03	-.05	.16	.25*	-.01	-.02
	(.65)		(.03)		(.08)		(.04)	
R-squared	.03		.02		.08		.01	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

white females.

Wave II Delinquency Scale

Table 5.97 shows the results of the OLS regression equations predicting the wave II delinquency scale with the MAOA polymorphism and with the measure of delinquent peers. Consistent with prior research (Warr, 2002), the delinquent peers scale emerged as a significant predictor of delinquent involvement at wave II for all of the models estimated in Table 5.97. Additionally, MAOA is not a significant predictor of the delinquency scale for white males, for white females, and for black males; however, MAOA exerts a positive and statistically significant effect on delinquency at wave II for black females.

The results of the OLS regression equations predicting the wave II delinquency scale for the low delinquent peers group are presented in Table 5.98. The MAOA gene fails to reach statistical significance for any of the models estimated in Table 5.98. The models depicted in Table 5.99, which predict delinquent involvement at wave II for the high delinquent peer group, reveal a slightly different pattern of results. Although MAOA is insignificantly related to the dependent variable for white males, white females, and black males, it is positively related to the wave II delinquency scale for black females. The results presented in Tables 5.98 and 5.99 thus reveal a significant GxE between MAOA and delinquent peers in the prediction of the wave II delinquency scale.

Next, the results of the OLS regression models predicting the wave II delinquency scale with the MAOA polymorphism and with the measure of family risk, net of the effects of statistically controls, are presented. As shown in Table 5.100, the family risk scale exerts a statistically significant effect on the wave II delinquency scale for white males, white females,

Table 5.97. The Direct Effects of MAOA and Delinquent Peers on Delinquency at Wave II

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.17	-.02	-.14	-.03	.50	.06	.68	.16**
	(.30)		(.17)		(.65)		(.29)	
Socialization Variable								
Delinquent peers	.44	.30**	.41	.32**	.58	.35**	.40	.28**
	(.06)		(.05)		(.13)		(.10)	
Control Variables								
Age	-.34	-.14**	-.56	-.27**	-.07	-.03	-.53	-.28**
	(.09)		(.08)		(.20)		(.13)	
Cognitive complexity	.00	.00	.02	.06	.00	.01	.01	.04
	(.02)		(.01)		(.03)		(.02)	
R-squared	.08		.12		.12		.16	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.98. The Effects of MAOA on Delinquency at Wave II for the Low Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.03	-.01	-.07	-.02	.44	.07	.49	.15
	(.40)		(.20)		(.71)		(.31)	
Control Variables								
Age	-.05	-.02	-.19	-.12**	.13	.07	-.30	-.22**
	(.11)		(.08)		(.20)		(.13)	
Cognitive complexity	-.05	-.12	.00	.01	-.02	-.05	.02	.08
	(.02)		(.02)		(.03)		(.02)	
R-squared	.02		.01		.01		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.99. The Effects of MAOA on Delinquency at Wave II for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.28	-.03	-.34	-.06	.63	.06	1.14	.23**
	(.46)		(.28)		(1.2)		(.52)	
Control Variables								
Age	-.54	-.19**	-.94	-.35**	.03	.01	-.86	-.35**
	(.15)		(.13)		(.42)		(.26)	
Cognitive complexity	.03	.07	.02	.06	.02	.04	-.00	-.01
	(.03)		(.02)		(.06)		(.03)	
R-squared	.04		.13		.00		.18	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

and black females. In addition, the MAOA gene has a statistically significant and positive direct effect on the wave II delinquency scale for black females. Specifically, black females with more MAOA risk alleles, on average, are more involved in delinquency during adolescence.

Tables 5.101 and 5.102 contain the results of the OLS regression models predicting the wave II delinquency scale separately for low-risk families and for high-risk families, respectively. As shown in Table 5.101, MAOA continues to maintain a statistically significant and positive relationship with the wave II delinquency scale for black females. Consistent with the direct effects models, however, MAOA is not related to the dependent variable in any of the other models. Table 5.102 shows that the MAOA gene is not significantly associated with delinquent involvement at wave II for any of the race/gender subsamples. The findings reported in Tables 5.101 and 5.102 find evidence in favor of a GxE between MAOA and family risk in the prediction of the wave II delinquency scale for black females.

Summary of the Effects of MAOA on the Wave II Delinquency Scale

Tables 5.99 through 5.102 presented the results of the direct and interactive effects models predicting the wave II delinquency scale with the MAOA polymorphism, with the measure of delinquent peers, and with the family risk scale. The results reveled that the MAOA gene did not have a direct or interactive effect on delinquent involvement for white males, for white females, and for black males. The MAOA gene did, however, have a significant direct effect on delinquency for black females. In addition, MAOA interacted with delinquent peers and with family risk to predict the wave II delinquency scale for black females. In summary, the direct and interactive effects of MAOA on delinquent involvement at wave II were confined only to black females.

Table 5.100. The Direct Effects of MAOA and Family Risk on Delinquency at Wave II

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.24	-.03	-.29	-.06	.33	.04	.59	.14**
	(.31)		(.19)		(.68)		(.30)	
Socialization Variable								
Family risk	.47	.11**	.64	.19**	.12	.02	.49	.17**
	(.17)		(.12)		(.46)		(.21)	
Control Variables								
Age	-.15	-.06	-.36	-.17**	.18	.07	-.42	-.22**
	(.09)		(.08)		(.20)		(.13)	
Cognitive complexity	-.01	-.02	.01	.03	-.01	-.01	-.01	-.03
	(.02)		(.01)		(.03)		(.02)	
R-squared	.02		.06		.01		.10	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.101. The Effects of MAOA on Delinquency at Wave II for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.13	.02	-.04	-.01	.61	.08	.73	.17*
	(.42)		(.21)		(.77)		(.38)	
Control Variables								
Age	-.06	-.03	-.26	-.15**	.24	.10	-.43	-.25**
	(.12)		(.08)		(.23)		(.16)	
Cognitive complexity	-.03	-.08	-.01	-.02	-.04	-.10	-.03	-.12
	(.02)		(.02)		(.04)		(.02)	
R-squared	.01		.02		.03		.12	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.102. The Effects of MAOA on Delinquency at Wave II for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.66	-.08	-.55	-.09	-.74	-.07	.38	.09
	(.42)		(.34)		(1.5)		(.51)	
Control Variables								
Age	-.24	-.10*	-.53	-.20**	.04	.01	-.37	-.17
	(.14)		(.15)		(.42)		(.26)	
Cognitive complexity	.02	.05	.03	.07	.05	.13	.04	.13
	(.03)		(.03)		(.06)		(.04)	
R-squared	.02		.05		.02		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Wave III Delinquency Scale

Table 5.103 contains the results of the OLS regression models predicting the wave III delinquency scale with the MAOA gene and with the measure of delinquent peers. For white males and for white females, the delinquent peers scale is a statistically significant predictor of delinquent involvement at wave III. In contrast, there is not a significant relationship between delinquent peers and the delinquency scale for black males and for black females. As revealed in Table 5.103, the MAOA gene exerts a statistically significant and negative direct impact on the wave III delinquency scale; however, this significant effect is limited only to white males.

Tables 5.104 and 5.105 examine the effects of the MAOA gene for the low delinquent peers group and for the high delinquent peer group, respectively. Table 5.104 shows that the MAOA gene does not have a significant effect on the wave III delinquency scale for any of the models estimated for the low delinquent peers sample. Table 5.105, on the other hand, reveals that the MAOA gene exerts a significant negative effect on the wave III delinquency scale for white males in the high delinquent peers group. These findings indicate a GxE between delinquent peers and MAOA in the creation of delinquency for white males.

Table 5.106 portrays the results of the OLS regression equations predicting the wave III delinquency scale with the MAOA gene and with the family risk scale. As shown in Table 5.106, the family risk scale exerts a significant positive effect on delinquent involvement at wave III for white males, for white females, and for black females. In contrast, the MAOA gene fails to have a significant direct effect on the dependent variable for any of the models calculated.

Tables 5.107 and 5.108 contain the results for the models estimated for low-risk families and high-risk families, respectively. As depicted in Table 5.107, the MAOA gene does not have a significant effect on the wave III delinquency scale for any of the models calculated for

Table 5.103. The Direct Effects of MAOA and Delinquent Peers on Delinquency at Wave III

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.31	-.07*	.06	.05	.28	.09	.14	.09
	(.16)		(.05)		(.23)		(.11)	
Socialization Variable								
Delinquent peers	.14	.17**	.04	.11**	-.01	-.02	-.00	-.01
	(.03)		(.01)		(.05)		(.04)	
Control Variables								
Age	-.27	-.21**	-.06	-.10**	.03	.04	-.09	-.14*
	(.05)		(.02)		(.07)		(.05)	
Cognitive complexity	.03	.11**	.00	.02	.02	.14*	.01	.07
	(.01)		(.00)		(.01)		(.01)	
R-squared	.06		.02		.03		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.104. The Effects of MAOA on Delinquency at Wave III for the Low Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.03	.01	.04	.04	.26	.08	.04	.13
	(.26)		(.05)		(.37)		(.13)	
Control Variables								
Age	-.29	-.22**	-.00	-.00	.05	.06	-.02	-.04
	(.07)		(.02)		(.10)		(.05)	
Cognitive complexity	.02	.09	.00	.03	.01	.10	.00	.05
	(.01)		(.00)		(.02)		(.01)	
R-squared	.05		.00		.02		.00	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.105. The Effects of MAOA on Delinquency at Wave III for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.55	-.13**	.08	.05	.30	.12	.26	.15
	(.21)		(.08)		(.30)		(.18)	
Control Variables								
Age	-.19	-.14**	-.11	-.14**	.02	.02	-.20	-.24**
	(.07)		(.04)		(.10)		(.09)	
Cognitive complexity	.02	.10**	.00	.01	.02	.18	.01	.10
	(.01)		(.01)		(.02)		(.01)	
R-squared	.04		.02		.04		.08	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.106. The Direct Effects of MAOA and Family Risk on Delinquency at Wave III

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.13	.05	.05	.04	.10	.03	.11	.07
	(.18)		(.05)		(.25)		(.11)	
Socialization Variable								
Family risk	.21	.19**	.12	.14**	-.10	-.05	.21	.20**
	(.07)		(.03)		(.15)		(.07)	
Control Variables								
Age	-.10	-.14**	-.04	-.07*	-.01	-.00	-.10	-.14**
	(.05)		(.02)		(.07)		(.05)	
Cognitive complexity	.01	.08	.00	.01	.02	.11	.01	.08
	(.01)		(.00)		(.01)		(.01)	
R-squared	.07		.02		.02		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.107. The Effects of MAOA on Delinquency at Wave III for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.30	-.07	.02	.02	-.08	-.02	-.04	-.04
	(.22)		(.05)		(.32)		(.10)	
Control Variables								
Age	-.21	-.17**	-.05	-.11**	.07	.08	-.11	-.25**
	(.06)		(.02)		(.09)		(.04)	
Cognitive complexity	.03	.12**	.00	.05	.02	.11	.01	.09
	(.01)		(.00)		(.02)		(.01)	
R-squared	.05		.01		.02		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.108. The Effects of MAOA on Delinquency at Wave III for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.30	-.06	.10	.06	.42	.15	.33	.16
	(.27)		(.10)		(.42)		(.24)	
Control Variables								
Age	-.24	-.16**	-.02	-.02	-.10	-.13	-.08	-.08
	(.08)		(.04)		(.12)		(.12)	
Cognitive complexity	.01	.05	-.00	-.03	.01	.06	.01	.08
	(.02)		(.01)		(.02)		(.01)	
R-squared	.03		.01		.06		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

respondents in low-risk families. The results presented in Table 5.108 also show that the MAOA gene does not have a significant effect on the wave III delinquency scale for the high-risk family group.

Summary of the Effects of MAOA on the Wave III Delinquency Scale

Tables 5.103 through 5.108 contained the results for the direct and interactive effects models predicting delinquent involvement at wave III with the MAOA gene and with the measure of delinquent peers and with the family risk scale. Across all of the models, two models revealed a statistically significant association between MAOA and the wave III delinquency scale. First, MAOA had a significant direct effect on delinquency at wave III for white males. Additionally, MAOA interacted with the measure of delinquent peers to predict variation in the wave III delinquency scale for white males. The MAOA gene did not have a significant direct effect or significant interactive effects for any of the other race/gender subsamples.

Number of Police Contacts

Table 5.109 shows the results of the negative binomial regression equations predicting number of police contacts with the MAOA gene and with the delinquent peers scale. As shown in Table 5.109, the measure of delinquent peers has a significant effect on number of police contacts for white males and for white females; the delinquent peers coefficient is insignificant for black males and for black females. Moreover, MAOA has a significant direct effect on number of police contacts for black males and for black females. The relationship between MAOA and police contacts is positive for black males, but negative for black females.

Tables 5.110 and 5.111 portray the results for the negative binomial regression equations

Table 5.109. The Direct Effects of MAOA and Delinquent Peers on Number of Police Contacts

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	.01	.16	.16	.20	.81**	.35	-.94*	.52
Socialization Variable								
Delinquent peers	.12**	.03	.22**	.05	.09	.06	.08	.15
Control Variables								
Age	-.17**	.05	-.46**	.10	-.03	.10	-.00	.23
Cognitive complexity	-.00	.01	.01	.02	.02	.02	-.03	.04
Pseudo R-squared	.02		.06		.02		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.110. The Effects of MAOA on Number of Police Contacts for the Low Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	.07	.27	.30	.29	1.10**	.48	-.37	1.06
Control Variables								
Age	-.15**	.07	-.41**	.15	.00	.12	.40	.47
Cognitive complexity	-.02	.01	-.01	.02	.01	.02	-.08	.10
Pseudo R-squared	.01		.04		.04		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.111. The Effects of MAOA on Number of Police Contacts for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.06	.20	-.02	.27	.49	.54	-1.04	.68
Control Variables								
Age	-.12*	.06	-.40**	.12	.08	.20	-.28	.31
Cognitive complexity	.01	.01	.03	.02	.02	.03	-.02	.04
Pseudo R-squared	.01		.03		.01		.06	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

predicting number of police contacts for the low delinquent peers group and for the high delinquent peers group, respectively. Table 5.110 shows that the MAOA polymorphism has a significant positive effect on number of police contacts for black males in the low delinquent peers group. However, the MAOA gene does not maintain a significant association with number of police contacts for any of the other models estimated in Table 5.110. As shown in Table 5.111, the MAOA gene is not a significant predictor of number of police contacts for the high delinquent peer group in any of the negative binomial regression models. Taken together, Tables 5.110 and 5.111 reveal a significant GxE between MAOA and delinquent peers in the prediction of number of police contacts for black males.

Table 5.112 contains the results of the negative binomial regression equations predicting number of police contacts with the MAOA gene and with the family risk scale. The results garnered from these models show that the family risk scale has a significant and positive effect on number of police contacts for all of the models estimated with the exception of the one calculated for black males. In addition, MAOA has a significant and negative direct effect on number of police contacts for black females.

Table 5.113 portrays the findings of the negative binomial regression equations predicting number of police contacts for low-risk families. As shown in the top row of Table 5.113, the MAOA gene is statistically insignificant across all of the models calculated for low-risk families. Table 5.114 contains the results of the models garnered using the high-risk family group. In these models, MAOA emerges as a negative predictor of number of police contacts for black females; however, the MAOA gene is not statistically significant in the remaining models presented in Table 5.114. Taken together, the results found in Tables 5.113 and 5.114 indicate a significant GxE between MAOA and family risk in the prediction of police contacts for black

Table 5.112. The Direct Effects of MAOA and Family Risk on Number of Police Contacts

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.05	.16	.03	.21	.43	.34	-.82*	.47
Socialization Variable								
Family risk	.19**	.09	.29**	.13	.24	.18	.58**	.23
Control Variables								
Age	-.11**	.05	-.32**	.09	.02	.10	-.06	.22
Cognitive complexity	-.00	.01	.01	.02	.02	.02	-.02	.03
Pseudo R-squared	.01		.03		.01		.11	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.113. The Effects of MAOA on Number of Police Contacts for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.17	.22	-.22	.34	.55	.44	---	---
Control Variables								
Age	-.18**	.06	-.37**	.15	-.04	.13	---	---
Cognitive complexity	.01	.01	.01	.02	.02	.02	---	---
Pseudo R-squared	.01		.03		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of police contacts for black females precluded the model from converging.

Table 5.114. The Effects of MAOA on Number of Police Contacts for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	.07	.23	.21	.26	.29	.62	-1.68**	.81
Control Variables								
Age	-.00	.07	-.29**	.12	.12	.17	-.08	.37
Cognitive complexity	-.02	.01	.02	.02	.03	.02	-.04	.06
Pseudo R-squared	.00		.02		.01		.12	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

females.

Summary of the Effects of MAOA on Number of Police Contacts

Tables 5.109 through 5.114 presented the direct and interactive effects of the MAOA gene, of the delinquent peers scale, and of the family risk measure on number of police contacts. The results of the these models indicated that MAOA had a significant direct effect on police contacts for black males and for black females. Additionally, MAOA interacted with the measure of delinquent peers to predict variation in number of police contacts for black males. Finally, there was a significant GxE between MAOA and family risk for black females. In summary, MAOA had both direct and interactive effects in the prediction of police contacts, but these effects were generally confined to black males and black females.

Ever Arrested

Table 5.115 contains the results of the binary logistic regression equations predicting arrest status (yes/no) with the MAOA gene and with the measure of delinquent peers. The delinquent peers scale is a significant predictor arrest status for white males, for white females, and for black males. In addition, the MAOA polymorphism has a significant direct effect on arrest status for white males.

Tables 5.116 and 5.117 estimate the logistic regression equations separately for the low delinquent peer group and the high delinquent peers group, respectively. Table 5.116 shows that the MAOA gene does not have a significant effect in any of the models employing the low delinquent peers group. Similarly, and as shown in Table 5.117, the MAOA gene also does not have a significant effect in any of the models estimated with the high delinquent peers group.

Table 5.115. The Direct Effects of MAOA and Delinquent Peers on Arrest Status

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.39*	.23	.15	.28	.70	.43	1.51	1.1
Socialization Variable								
Delinquent peers	.23**	.04	.27**	.07	.15**	.08	.37	.30
Control Variables								
Age	-.22**	.07	-.32**	.13	-.10	.13	-1.00**	.55
Cognitive complexity	.00	.01	.03	.02	.01	.02	.01	.06
Cox & Snell R-squared	.05		.02		.04		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.116. The Effects of MAOA on Arrest Status for the Low Delinquent Peers Group

	White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.40	.41	.24	.48	.89	.62	---	---
Control Variables								
Age	-.23**	.11	-.31	.23	-.08	.17	---	---
Cognitive complexity	-.03	.02	-.01	.04	.01	.03	---	---
Cox & Snell R-squared	.03		.01		.03		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.117. The Effects of MAOA on Arrest Status for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.34	.27	.10	.35	.49	.61	1.62	1.5
Control Variables								
Age	-.14	.08	-.22	.16	.05	.20	-2.27*	1.3
Cognitive complexity	.02	.01	.03	.03	-.01	.03	.01	.07
Cox & Snell R-squared	.01		.01		.01		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.118 presents the results of the logistic regression equations predicting arrest status with the MAOA gene and with the family risk scale. In line with prior research (Loeber and Stouthamer-Loeber, 1986), the family risk scale has a significant positive effect on arrest status for white males, for white females, and for black males. Additionally, MAOA exerts a significant and negative direct effect on arrest status for white males. In the remaining three models, however, MAOA is not associated with the dependent variable.

Table 5.119 contains the results of the logistic regression equations estimated for respondents in low-risk families. The top panel of Table 5.119 shows that the MAOA gene has a significant negative effect on arrest status for white males. Similar to the results garnered for the direct effects model, MAOA continues to remain insignificant for the other three gender/race subsamples. Table 5.120 reveals the results for the analyses based on the high-risk family group. The MAOA gene is not a significant predictor of arrest status for any of the models estimated for the high-risk family samples.

Summary of the Effects of MAOA on Ever Arrested

Tables 5.115 through 5.120 presented the results of the direct and interactive effects of the MAOA polymorphism, of the delinquent peers scale, and of the family risk measure on arrest status. In regards to the MAOA gene, two significant findings emerged. First, MAOA had a significant direct effect on arrest status for white males. This finding was observed even when controlling for the effects of delinquent peers, family risk, and a number of key covariates. Second, the multivariate equations also revealed a significant GxE between MAOA and family risk for white males. Taken together, MAOA has significant direct and interactive effects on arrest status for white males.

Table 5.118. The Direct Effects of MAOA and Family Risk on Arrest Status

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.45**	.23	.08	.28	.52	.41	1.46	1.1
Socialization Variable								
Family risk	.33**	.11	.28**	.15	.42**	.20	.27	.53
Control Variables								
Age	-.11*	.06	-.16	.12	-.02	.11	-.80	.49
Cognitive complexity	-.01	.01	.02	.02	.00	.02	-.01	.08
Cox & Snell R-squared	.02		.01		.03		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.119. The Effects of MAOA on Arrest Status for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females†	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.69**	.35	-.27	.48	.47	.52	---	---
Control Variables								
Age	-.16*	.09	-.24	.19	-.06	.15	---	---
Cognitive complexity	-.01	.02	-.01	.03	-.01	.03	---	---
Cox & Snell R-squared	.02		.01		.01		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black females in the low-risk family group failed to converge. The lack of variability in the dependent variable stemming from a low base rate of arrests for black females precluded the model from converging.

Table 5.120. The Effects of MAOA on Arrest Status for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.26	.30	.34	.35	.43	.71	1.34	1.5
Control Variables								
Age	-.01	.09	-.09	.16	.08	.19	-2.02	1.3
Cognitive complexity	-.01	.02	.04	.03	.03	.03	-.00	-.08
Cox & Snell R-squared	.00		.01		.02		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Marijuana Use

Table 5.121 contains the results of the negative binomial regression equations predicting frequency of marijuana use with the MAOA gene and with the measure of delinquent peers. In the multivariate equations, the delinquent peers scale is a strong predictor of marijuana use in all five of the models estimated in Table 5.121. In addition, MAOA has a significant and negative direct effect on marijuana use for white males.

The results of negative binomial equations calculated for the low delinquent peers group are presented in Table 5.122. As shown in the top row of Table 5.122, the MAOA gene has a significant effect on marijuana use for white males and for white females; however, the relationship is positive for white males, while it is negative for white females. Table 5.123 contains the result of the multivariate models estimated for the high delinquent peers group. In only one model—the model calculated for black males—is MAOA a positive predictor of marijuana use. The results thus suggest significant GxEs between MAOA and delinquent peers in the prediction of marijuana use for white males, for white females, and for black males.

Table 5.124 presents the results of the negative binomial regression equations predicting frequency of marijuana use with the MAOA gene and with the family risk scale. The family risk scale is significantly related to marijuana use for white females, for black males, and for black females. Moreover, the MAOA gene has a significant and positive direct effect on marijuana use for black males.

Tables 5.125 and 5.126 contain the results of the models calculated for the low-risk family group and for the high-risk family group, respectively. Table 5.125 shows that the MAOA gene does not maintain a significant association with marijuana use for any of the models calculated for respondents from low-risk families. However, as indicated in Table 5.126,

Table 5.121. The Direct Effects of MAOA and Delinquent Peers on Frequency of Marijuana Use

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.90**	.38	.16	.21	.54	.59	-.34	.48
Socialization Variable								
Delinquent peers	.86**	.08	.68**	.06	.99**	.14	.50	.15**
Control Variables								
Age	.07	.12	.14**	.10	-.04	.23	.01	.29
Cognitive complexity	.09	.02	-.01	.02	.05	.04	-.04	.03
Pseudo R-squared	.11		.14		.22		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.122. The Effects of MAOA on Frequency of Marijuana Use for the Low Delinquent Peers Group

	White Males		White Females		Black Males†		Black Females‡	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	2.74**	1.38	-2.39**	1.31	---	---	---	---
Control Variables								
Age	1.35**	.66	.17	.54	---	---	---	---
Cognitive complexity	-.00	.08	-.02	.06	---	---	---	---
Pseudo R-squared	.23		.07		---		---	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
† The negative binomial model for black males in the low delinquent peers group failed to converge. The lack of variability in the dependent variable precluded the model from converging.
‡ The negative binomial model for black females in the low delinquent peers group failed to converge. The lack of variability in the dependent variable precluded the model from converging.

Table 5.123. The Effects of MAOA on Frequency of Marijuana Use for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.64	.45	.31	.28	2.10**	.74	.55	.41
Control Variables								
Age	.20	.15	.11	.14	.70**	.32	-.05	.28
Cognitive complexity	.05	.02	-.02	.02	.18**	.06	-.00	.03
Pseudo R-squared	.01		.00		.05		.01	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.124. The Direct Effects of MAOA and Family Risk on Frequency of Marijuana Use

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.60	.48	.43	.28	1.63**	.74	.58	.42
Socialization Variable								
Family risk	.04	.20	.45*	.25	1.32**	.44	.59*	.32
Control Variables								
Age	.43	.15	.29**	.14	.74**	.23	.26	.28
Cognitive complexity	.05	.02	-.03	.02	.08	.05	-.02	.03
Pseudo R-squared	.01		.01		.08		.03	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.125. The Effects of MAOA on Frequency of Marijuana Use for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.67	.75	.66	.55	1.44	1.35	.28	.69
Control Variables								
Age	.66**	.30	.35	.33	.76**	.31	.36	.48
Cognitive complexity	.06**	.03	-.04	.04	.06	.08	.02	.08
Pseudo R-squared	.02		.01		.08		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

Table 5.126. The Effects of MAOA on Frequency of Marijuana Use for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Genetic Polymorphism								
MAOA	-.45	.57	.26	.31	2.10*	1.11	-.23	.61
Control Variables								
Age	.35	.17	.28**	.15	.92*	.51	-.61	.53
Cognitive complexity	.01	.04	-.03	.03	.17**	.07	-.01	.04
Pseudo R-squared	.01		.01		.04		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed

MAOA has a significant positive effect on marijuana use for black males from high-risk families. The MAOA gene does not have a significant effect on marijuana use for any of the other models calculated in Table 5.126.

Summary of the Effects of MAOA on Marijuana Use

Tables 5.121 through 5.126 presented the direct and interactive effects that the MAOA polymorphism, the measure of delinquent peers, and the family risk scale had on frequency of marijuana use. The MAOA gene had significant direct effects on marijuana use for white males and for black males. Four significant GxEs were also detected in the multivariate models. Specifically, MAOA interacted with delinquent peers for white males, for white females, and for black males to predict frequency of marijuana use. MAOA also interacted with family risk to explain a significant amount of variation in the dependent variable for black males. In summary, MAOA had significant direct and interactive effects on frequency of marijuana use for both black and white respondents.

Alcohol Abuse

Table 5.127 contains the results of the OLS regression equations predicting scores on the alcohol abuse scale with the MAOA gene and with the measure of delinquent peers. Across all of the models in Table 5.127, the delinquent peers scale is a strong and consistent predictor of alcohol abuse for all of the race/gender subsamples. In addition, the MAOA gene has a negative effect on alcohol abuse for white males and a positive effect on alcohol abuse for white females.

Tables 5.128 and 5.129 present the models calculated for the low delinquent peers group and for the high delinquent peers group, respectively. The top row of Table 5.128 shows that

Table 5.127. The Direct Effects of MAOA and Delinquent Peers on Alcohol Abuse

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.85	-.09**	.41	.08**	.45	.06	.02	.01
	(.33)		(.17)		(.53)		(.16)	
Socialization Variable								
Delinquent peers	.28	.17**	.14	.11**	.20	.15*	.12	.17**
	(.06)		(.05)		(.11)		(.05)	
Control Variables								
Age	-.25	-.09**	-.21	-.10**	.17	.08	.12	.12*
	(.10)		(.08)		(.15)		(.07)	
Cognitive complexity	.06	.12**	.05	.13**	.05	.15**	.01	.04
	(.02)		(.01)		(.03)		(.01)	
R-squared	.05		.03		.06		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.128. The Effects of MAOA on Alcohol Abuse for the Low Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.59	-.07	.60	.12**	.45	.08	-.07	-.05
	(.49)		(.26)		(.56)		(.14)	
Control Variables								
Age	-.18	-.08	-.17	-.09	.15	.10	.04	.07
	(.13)		(.10)		(.16)		(.06)	
Cognitive complexity	.05	.10*	.04	.10*	.07	.28**	.02	.25**
	(.03)		(.02)		(.03)		(.01)	
R-squared	.02		.03		.11		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.129. The Effects of MAOA on Alcohol Abuse for the High Delinquent Peers Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-1.08	-.11**	.21	.04	.34	.04	.16	.06
	(.47)		(.24)		(.93)		(.30)	
Control Variables								
Age	-.22	-.07	-.24	-.11**	.17	.06	.26	.19*
	(.15)		(.11)		(.32)		(.14)	
Cognitive complexity	.06	.12**	.05	.14**	.03	.05	-.02	-.10
	(.03)		(.02)		(.08)		(.02)	
R-squared	.03		.03		.01		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

MAOA has a significant positive effect on white females from the low delinquent peers group. The MAOA coefficient is insignificant for the remaining three models presented in Table 5.128. Table 5.129 contains the results of the analyses using the high delinquent peers sample. MAOA has a significant negative effect on alcohol abuse for white males; however, MAOA failed to reach statistical significance for white females, for black males, and for black females in the high delinquent peers group.

Table 5.130 portrays the results of the OLS regression equations predicting scores on the alcohol abuse scale with the MAOA gene and with the family risk scale. As shown in Table 5.130, the family risk scale has a small effect on alcohol abuse for white males, but the measure of family risk is not significant for any of the other three models. The MAOA gene, in contrast, has a positive effect on alcohol abuse for white males and a negative effect for white females.

Tables 5.131 and 5.132 present the results of the models estimated for respondents from low-risk families and for respondents from high-risk families, respectively. Table 5.131 shows that MAOA has a significant negative effect on alcohol abuse for white males from low-risk families. Similarly, Table 5.132 also shows that MAOA is negatively related to alcohol abuse for white males from high-risk families. Since the effect of MAOA is invariant across family risk levels, these findings are not evidence of a GxE, but instead indicate a robust main effect of MAOA that is not conditioned by the familial environment. Table 5.132 also reveals that MAOA has a significant positive effect on the alcohol abuse scale for white females.

Summary of the Effects of MAOA on Alcohol Abuse

Tables 5.127 through 5.132 presented the direct and interactive effects of MAOA, of the delinquent peers scale, and of the measure of family risk in the prediction of alcohol abuse.

Table 5.130. The Direct Effects of MAOA and Family Risk on Alcohol Abuse

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.86	-.09**	.45	.09**	.34	.05	-.01	-.00
	(.34)		(.18)		(.53)		(.16)	
Socialization Variable								
Family risk	.33	.07*	.11	.04	-.29	-.07	-.16	-.10
	(.19)		(.11)		(.30)		(.11)	
Control Variables								
Age	-.11	-.04	-.15	-.07*	.19	.09	.16	.16**
	(.10)		(.08)		(.15)		(.07)	
Cognitive complexity	.05	.11	.05	.13**	.04	.13	.00	.02
	(.02)		(.01)		(.03)		(.01)	
R-squared	.03		.03		.03		.04	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.131. The Effects of MAOA on Alcohol Abuse for the Low-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-.77	-.09*	.20	.04	.05	.01	.11	.04
	(.44)		(.24)		(.76)		(.24)	
Control Variables								
Age	-.22	-.09*	-.25	-.13**	.33	.14	.25	.22**
	(.12)		(.10)		(.22)		(.10)	
Cognitive complexity	.04	.08	.03	.09*	.03	.07	-.01	-.08
	(.02)		(.02)		(.04)		(.01)	
R-squared	.02		.03		.03		.05	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.132. The Effects of MAOA on Alcohol Abuse for the High-Risk Family Group

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	-1.00	-.10*	.83	.16**	.84	.20	-.17	-.10
	(.54)		(.28)		(.55)		(.18)	
Control Variables								
Age	.06	.02	.04	.02	.02	.02	.01	.01
	(.17)		(.13)		(.15)		(.09)	
Cognitive complexity	.07	.14**	.06	.17**	.05	.29**	.03	.28**
	(.03)		(.02)		(.02)		(.01)	
R-squared	.03		.06		.13		.09	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Four significant direct effects and three significant GxEs emerged in the multivariate equations. Specifically, the MAOA gene had significant direct effects on alcohol abuse for white males and for white females. Additionally, scores on the alcohol abuse scale were predicted by interactions between MAOA and delinquent peers for white males and for white females, and by an interaction between MAOA and family risk for white females.

Indirect Effects of MAOA

Tables 5.133, 5.134, and 5.135 contain the results of the OLS regression equations examining the indirect effect of the MAOA polymorphism. These models are testing for gene X environment correlations (rGEs) between the MAOA gene and two measures of the social environment: delinquent peers and family risk. Moreover, statistical analyses are estimated to determine whether the MAOA polymorphism explains a significant amount of variation in the cognitive complexity measure.

Table 5.133 presents the results of the OLS regression equations predicting scores on the measure of delinquent peers. As can be seen in the top row of Table 5.133, the MAOA gene does not have a significant effect on delinquent peers for white males, for white females, for black males, and for black females. Table 5.134 shows the results of the regression models predicting scores on the family risk scale. Similar to the findings reported in Table 5.133, the MAOA polymorphism does not maintain a significant association with the dependent variable in any of the models estimated in Table 5.134. Finally, Table 5.135 reveals that the MAOA gene does not have a significant effect on the measure of cognitive complexity for any of the models calculated. Of all the indirect effects models presented in Tables 5.133, 5.134, and 5.135, none of the them revealed a significant rGE.

Table 5.133. The Indirect Effects of MAOA on Delinquent Peers at Wave I

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.10	.02	-.16	-.04	-.03	-.01	-.16	-.05
	(.20)		(.13)		(.39)		(.21)	
Control Variables								
Age	.47	.29**	.48	.30**	.51	.33**	.28	.20**
	(.06)		(.05)		(.11)		(.09)	
Cognitive complexity	-.04	-.12**	-.04	-.13**	-.02	-.08	-.04	-.20**
	(.01)		(.01)		(.02)		(.01)	
R-squared	.10		.10		.12		.07	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.134. The Indirect Effects of MAOA on Family Risk at Wave I

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.05	.03	.07	.05	-.07	-.04	.08	.06
	(.07)		(.06)		(.13)		(.10)	
Control Variables								
Age	.09	.17**	.06	.10**	.03	.06	.02	.02
	(.02)		(.02)		(.04)		(.05)	
Cognitive complexity	.00	.00	-.01	-.05	.00	.02	.00	.00
	(.00)		(.00)		(.01)		(.01)	
R-squared	.03		.01		.01		.00	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Table 5.135. The Indirect Effects of MAOA on Cognitive Complexity

	White Males		White Females		Black Males		Black Females	
	b	*Beta*	*b*	*Beta*	*b*	*Beta*	*b*	*Beta*
Genetic Polymorphism								
MAOA	.07	.00	.52	.04	-.07	-.00	.07	.00
	(.69)		(.47)		(1.6)		(1.1)	
Control Variables								
Age	.54	.10**	.62	.11**	.13	.02	.93	.13*
	(.20)		(.20)		(.45)		(.50)	
R-squared	.01		.02		.00		.02	

*Significant at the .10 level, two-tailed
**Significant at the .05 level, two-tailed
Note: Standard errors in parentheses

Summary of the Indirect Effects of the MAOA Gene

A series of OLS regression models were calculated to examine whether the MAOA gene was related to measures of the social environment and to a measure of cognitive complexity. The results of these statistical models were reported in Tables 5.133 through 5.135 and revealed no evidence that the MAOA gene had a significant impact on the social environment. Indeed, across twelve different models, the MAOA gene was not statistically significant in one of them. As a result, it does not appear that the MAOA polymorphism is implicated in the creation of delinquent peer selection and the creation of family risk. Cognitive complexity also does not appear to be under the control of the MAOA gene. Taken together, there was no evidence indicating that MAOA was involved in an rGE for delinquent peers or for family risk.

Summary

This chapter empirically assessed the direct, indirect, and interactive effects of the dopaminergic polymorphisms, of the serotonin transporter gene (5HTT), and of the monoamine oxidase promoter polymorphism (MAOA) on seven different measures of antisocial behavior. The results of these models revealed that some of the polymorphisms, in some of the statistical models, and with some of the dependent variables, had significant direct effects on delinquent and criminal behavior. Gene X environment correlations (rGEs) were calculated by examining whether the genetic polymorphisms were statistically related to measures of the social environment. Specifically, the genetic polymorphisms, in some of the models, were significantly predictive of associating with delinquent peers and of residing in high-risk families. The dopamingeric genes also had significant effects on the measure of cognitive complexity. Finally, a series of GxEs were calculated to determine whether the effects of the genetic polymorphisms

were conditioned by different social environments. The results of the interactive statistical models revealed significant GxEs, whereby the genetic polymorphisms interacted with delinquent peers and with family risk to predict a range of antisocial behaviors. In summary, the genetic polymorphisms had significant direct, indirect, and interactive effects on the seven different measures of criminal and delinquent behavior. However, these effects varied depending upon the measure of antisocial behavior used, the model estimated, and the sample employed.

CHAPTER 6

DISCUSSION

Social explanations of crime and criminality have historically monopolized the field of criminology (Gottfredson and Hirschi, 1990; Walsh, 2002; Walsh and Ellis, 2004; Wilson and Herrnstein, 1985). Sociologically-informed theories of crime, for example, highlight the saliency of parents, peers, schools, siblings, neighborhoods, subcultures, and other social institutions in the etiology of crime, delinquency, and deviancy (Anderson, 1999; Gottfredson and Hirschi, 1990; Patterson, 1982; Sampson and Groves, 1989; Sampson and Laub, 1993; Sampson, Raudenbush, and Earls, 1997; Warr, 2002). At the same time, criminology has shunned the prospect that antisocial behaviors are influenced by genetic or biological factors (Walsh, 2002). As a direct result, very little empirical research has examined the contributions of genetic polymorphisms on the development of chronic problem behaviors (for notable exceptions see Caspi et al., 2002; Foley et al., 2004; Haberstick et al., 2005). The current dissertation took a cautious first step in this direction and examined the effects that five different genetic polymorphisms had on seven measures of antisocial behavior. Analysis of the Add Health data revealed that the dopaminergic, serotonergic, and MAOA genes had significant direct, indirect (rGE), and interactive (GxE) effects on a number of different delinquent and criminal behaviors.

This chapter is designed to provide a summary of the major findings garnered from the multivariate equations calculated in Chapter 5. Toward this end, the current chapter is divided into three main sections. First, the chapter will begin by summarizing the direct, indirect, and interactive effects that each of the five polymorphisms had on the measures of antisocial

behavior. In doing so, evidence relating to each of the three research questions posed in Chapter 3 will be offered. Second, the major limitations of this dissertation will be addressed and directions for future research will be discussed. Third, the implications of the findings will be explicated. Specific attention will be devoted to what the research findings mean for the field of criminology.

Summary of Research Findings

Chapter 5 reported the results of the statistical models that estimated the direct, indirect, and interactive effects of the five polymorphisms on a number of different measures of antisocial behavior. Table 6.1 was constructed to provide a concise way of summarizing the major findings that cut across the statistical models. The left hand column of Table 6.1 reveals the name of the polymorphism that is being summarized. The remaining three columns summarize the direct, indirect, and interactive effects for each of the genes examined. The cells in Table 6.1 reveal the number of significant findings detected out of the total number of models estimated. The percentages contained within the parentheses reveal the percentage of significant findings garnered with respect to each polymorphism. The last row of Table 6.1 reports a summary of the findings for all of the genetic polymorphisms pooled together. In order to facilitate the presentation of the major findings, the results of the statistical models will be discussed as they bear on each of the previously stated research questions (see Chapter 3).

Research Question One: Do the dopaminergic, serotonergic, and MAOA genetic polymorphisms exert a direct effect on a range of antisocial outcomes?

The left hand column in Table 6.1 shows a summary of the direct effects models predicting the seven different measure of antisocial behavior. For these models, the dependent

Table 6.1. Summary of Findings for the Genetic Polymorphisms

	Number of Direct Effects	Number of Indirect Effects	Number of Interactive Effects
DAT1	7/69	4/15	16/65
% Sig.	(10%)	(27%)	(25%)
DRD2	10/69	4/15	20/65
% Sig.	(14%)	(27%)	(31%)
DRD4	8/69	6/15	8/65
% Sig.	(12%)	(40%)	(12%)
5HTT	8/70	3/15	13/69
% Sig.	(11%)	(20%)	(19%)
MAOA	14/56	0/12	14/54
% Sig.	(25%)	(0%)	(26%)
Total	47/333	17/72	71/318
% Sig.	(14%)	(24%)	(22%)

variable was regressed on the genetic polymorphism(s), one of the two socialization measures (either delinquent peers or family risk), and the control variables. The results of these statistical models provide detailed information about whether or not the polymorphisms had a direct effect on delinquent/criminal behavior.

Research question one was concerned with whether the genetic polymorphisms would exert a statistically significant direct effect on a range of delinquent and criminal behaviors. As shown in Table 6.1, all of the genetic polymorphisms exerted statistically significant direct effects on the various measures of antisocial behavior. For example, 10 percent (7/69) of all the direct effects models estimated revealed statistically significant direct effects of DAT1 on the outcome variables. Similar results were garnered for DRD2 (14 percent), for DRD4 (12 percent), and for 5HTT (11 percent). MAOA had the highest percentage of direct effects, with 25 percent of all the models revealing a statistically significant direct effect of MAOA on antisocial behavior.[18] Overall, 14 percent of all the models revealed significant direct effects for the genetic polymorphisms.

The following list of results highlights the main genetic findings for each dependent variable in the direct effects models:

- **Wave I delinquency scale**: 4 significant dopamine effects, no significant serotonin effects, and no significant MAOA effects

[18] Recent work by Meyer-Lindenberg and colleagues (2006) provides one reason why the MAOA polymorphism had considerably more significant direct effects than any of the other genes. Using MRI images, they found brain structure and brain functioning varied depending on the type of MAOA allele the person possessed. Specifically, male carriers of the low expression allele had reductions in the volume of the cingulated gyrus, of the bilateral amygdalae, of the insula, and of the hypothalamus, when compared to carriers of the high expression allele. In addition, differences in orbitofrontal volume and differences in hippocampus activity were also detected between the low expression group and the high expression group. These differences in brain structure and activity that correspond to different allelic sequences of the MAOA gene may begin to explain the direct effect of MAOA on antisocial behavior.

- **Wave II delinquency scale**: 2 significant dopamine effects, no significant serotonin effects, and 2 significant MAOA effects
- **Wave III delinquency scale**: 3 significant dopamine effects, no significant serotonin effects, and 1 significant MAOA effect
- **Police contacts**: 2 significant dopamine effects, no significant serotonin effects, and 3 significant MAOA effects
- **Arrest status**: No significant dopamine effects, 1 significant serotonin effect, and 1 significant MAOA effect
- **Marijuana use**: 8 significant dopamine effects, 6 significant serotonin effects, and 2 significant MAOA effects
- **Alcohol abuse**: 6 significant dopamine effects, 1 significant serotonin effect, and 4 significant MAOA effects

These findings are in line with previous research indicating that genetic influences tend to have small and inconsistent *direct* effects on misbehavior (Raine, 1993; Rowe, 2002; Rutter, 2006). For example, in Caspi et al.'s (2002) seminal work, the results of their analysis revealed that MAOA did not have a significant direct effect on antisocial behavior. The lack of significant direct effects, however, should not be too surprising. Recall that most personality traits and most behaviors are influenced by multiple genes acting together and by multiple genes interacting with the environment (Ridley, 2003; Rowe, 2002; Rutter, 2006). Linear statistical models, such as those estimated in many of the direct effects equations, are unable to detect interactions that occur between genes and the social context.

Statistical models are also not able to determine whether a gene is active or inactive. For example, genes can be switched "on" or turned "off" by other genes (referred to as promoter

genes). Without taking into account the possibility that one gene is "on" for one person, but "off" for another person, it is not possible to determine accurately the effect that a particular gene has on a specific behavior. This may very well be the case for the genetic polymorphisms examined in this dissertation. In some Add Health respondents, the risk allele may be very active, whereas in another person, this same risk allele may remain dormant. Only when geneticists are able to determine whether a particular gene is "on" or "off" will we begin to know the "true" direct effects that genes have on behavior.

In a similar vein, and as will be discussed in more detail momentarily, genetic effects are oftentimes only visible when they are paired with certain environmental conditions (Caspi et al., 2002; Moffitt, 2005; Plomin, DeFries, and Loehlin, 1977; Rutter, 2006). Although the statistical models controlled for the effects of delinquent peers and family risk, these two measures are by no means the only environments that could impact an adolescent. Schools, neighborhoods, and other social settings, for instance, vary significantly from person-to-person. These environments also have the capacity to affect genetic expression (Clark and Grunstein, 2000; Hamer and Copeland, 1998; Ridley, 2003; Rutter, 2006; Moffitt, 2005; Walsh, 2002). The statistical models estimated in this dissertation did not include measures of all these different types of environments. As a result, if some unmeasured environment affected expression of the genetic polymorphisms included in the analysis, then the unmeasured environment may actually have attenuated the effects of the genetic polymorphisms.[19]

[19] A measure of neighborhood disadvantage is available in the Add Health data. However, given the large number of missing cases for this scale, it was not possible to include it in the multivariate equations. Preliminary analyses revealed that the risk alleles for some of the polymorphisms varied significantly across different levels of neighborhood disadvantage. Follow-up statistical tests did not reveal a significant interaction between the neighborhood disadvantage scale and the genetic polymorphisms when predicting the wave I delinquency scale. Future research should explore the close nexus between neighborhoods, genes, and antisocial behavior.

Even so, most behaviors and most personalities are not created by the possession of a single gene (Rutter, 2006). As discussed in Chapter 2 (see also Figure 2.5), behavioral geneticists generally agree that antisocial behaviors are created by polygenic effects, where multiple genes influence the development of a particular behavior or trait. In this case, one gene will have only a small influence on the phenotype—sometimes too small of an effect to detect through traditional multivariate techniques. When a statistically significant direct effect for a genetic polymorphism is observed, it tends to be relatively weak in magnitude (Rutter, 2006). The results of the analysis also bear this point out: the dopaminergic, serotonergic, and MAOA genes, when significant, had relatively small effects on the measures of antisocial behavior (Betas typically ranged between .07 and .24.).

The genetic polymorphisms also had differential effects on white and black Add Health respondents. In general, the genetic effects had stronger and more consistent effects on the outcome measures for blacks than for whites. For example, of all the statistically significant dopamine direct effects, 59 percent were found when analyzing the black sample.[20] Perhaps this finding is not too surprising given that prior population genetic research has established that distributions of alleles vary significantly among people from different racial and ethnic backgrounds (Allele Frequency Database, 2006; Chang et al., 1996; Chen, Burton, Greenberger, and Dmitrieva, 1999; Ding et al. 2000; Ding et al., 2002; Gelernter et al., 1998; Gelernter et al., 1999; Gelernter, Kranzler, and Cubells, 1997; Harpending and Cochran, 2002; Kang, Palmatier, and Kidd, 1999; Sarich and Miele, 2004). Additional analyses were conducted to determine whether the mean number of risk alleles differed between whites and blacks. Table 6.2 contains

[20] The significant effects for the full sample were excluded when tabulating the total number of significant direct effects. Also, the sample size for blacks was much smaller than the sample size for whites. Thus, there is reason to believe that if the sample sizes were comparable, the differences between blacks and whites would become even more pronounced.

Table 6.2. Mean Differences in Risk Alleles between White and Black Add Health Participants (*t*-Tests)

Gene	White Mean	Black Mean	*t*-Value
DAT1	1.52	1.65	-4.15*
DRD2	.60	.68	-6.57*
DRD4	.43	.53	-3.24*
5HTT	.88	.52	9.78*
MAOA (males)	.36	.58	-5.59*
MAOA (females)	.68	1.01	-5.97*

*Significant at the .05 level, two-tailed
Note: Chi-square tests also revealed that the risk alleles varied significantly between white and black Add Health respondents.

the results of the t-tests examining differences in the average number of risk alleles for the five genetic polymorphisms. As shown in the table, all of the genetic polymorphisms varied between blacks and whites. Specifically, blacks, on average, had more risk alleles for DAT1, DRD2, DRD4, and MAOA, whereas whites possessed more serotonin risk alleles.

The results of the t-tests reinforce prior research arguing that race is more than just a socially-constructed concept. Geneticists have long recognized that people can be classified into different racial groups based on analyzing the alleles of less than one hundred polymorphisms (Sarich and Miele, 2004). It is important to point out, however, that blacks and whites do not have different genes; they have the same genes, but the allelic frequencies that are visible in different racial samples vary quite extensively. Taken together, researchers examining the genetic origins to antisocial behavior need to analyze data separately by homogenous racial categories (Cardon and Palmer, 2003).

In summary, the genetic polymorphisms had significant direct effects on the seven measures of antisocial behavior. The genetic effects, however, varied depending upon the dependent variable predicted. The results thus support the hypothesis that the genetic polymorphisms will have some significant direct effects, but for most of the models, the genes will not exert a significant direct effect on crime and delinquency.

Research Question Two: Do the dopaminergic, serotonergic, and MAOA genetic polymorphisms have indirect effects on a range of antisocial outcomes?

The middle column of Table 6.1 contains a summary of the results of the indirect effects models testing for gene X environment correlations (rGEs). In these models, the measure of delinquent peers, the family risk scale, and the cognitive complexity variable were included as dependent variables in a series of ordinary least squares (OLS) regression models. The

dopaminergic, serotonergic, and MAOA polymorphisms were entered into the statistical models as predictor variables. An rGE was detected in the models where the genetic polymorphisms were statistically significant predictors.

Table 6.1 reveals that all of the genetic polymorphisms except for MAOA had significant indirect effects. Indeed, 27 percent of the models revealed significant indirect effects for DAT1 and for DRD2. The 5HTT gene was statistically significant in 20 percent of the indirect effects models. The highest percentage of significant indirect effects, however, was found for the DRD4 polymorphism. In 40 percent of all the indirect models estimated, DRD4 was statistically significant. Across all of the genetic polymorphisms, including MAOA, 24 percent of the indirect effects models detected a statistically significant rGE.

The following list highlights the main genetic findings for each of the dependent variables used in the indirect effects models:

- **Delinquent peers**: 3 significant dopamine effects, 2 significant serotonin effects, and no significant MAOA effects
- **Family risk**: 5 significant dopamine effects, no significant serotonin effects, and no significant MAOA effects
- **Cognitive complexity**: 6 significant dopamine effects, 1 significant serotonin effect, and no significant MAOA effects

The findings in regard to the indirect effects models are particularly important because they provide some of the first empirical evidence revealing an rGE between a measured genetic polymorphism and a measure of the social environment. Although behavioral geneticists have long theorized about the importance of rGEs, there has not been any empirical evidence (analyzing a measured gene) to back up their claims (DiLalla, 2002; Harris, 1998; Plomin,

DeFries, and Loehlin, 1977; Rutter, 2006; Scarr, 1992; Scarr and McCartney, 1983; Walsh, 2002, 2005;). The results generated from the indirect effects models, however, provide initial confirmation of the integral role of rGEs in the etiology of human behavior.

Although it might be tempting to casually gloss over the findings for the indirect models—or worse yet, trivialize them—this would be a serious oversight. In the multivariate models, measures of the environment and the genetic polymorphisms were included in the same statistical equations. Since scores on the environmental measures were partially influenced by the genetic polymorphisms, the genetic effects were somewhat attenuated. For example, DAT1 predicted affiliation with delinquent peers (an rGE). In general, however, the DAT1 gene was not predictive of delinquent involvement, but the measure of delinquent peers was a strong correlate of youthful delinquency. Perhaps one of the main reasons that the genetic polymorphisms have relatively small effects on different phenotypes is because the variance accounted for by genetic effects often overlaps with measures of the social environment (Rutter, 2006). The results provide support of this possibility by showing that genetic polymorphisms are associated with different criminogenic environments (Scarr, 1992; Scarr and McCartney, 1983; Walsh, 2002).

Analysis of the Add Health data thus revealed genetic influences on the environment. These findings add to a small, but emerging line of research showing that the environment and genes are inextricably tied together (DiLalla, 2002; Moffitt, 2005; Ridley, 2003; Rowe and Rodgers, 1997; Rutter, 2006). It is important to point out, however, that only five different genes were included in the statistical models. There is reason to believe that other genes—genes that were not available in the Add Health data—would also have effects on various measures of the social environment. As behavioral geneticist have long recognized, environments are largely a

reflection of an individual's genotype—a genotype that somewhat determines which environments we experience and how we experience them (Caspi and Moffitt, 1995).

As for now, the findings generated from the indirect effects models demonstrate the importance of examining rGEs. Without doing so, it would have appeared as if the two measures of the social environment—that is, the measure of delinquent peers and the family risk scale—exerted independent effects on antisocial behavior. In reality, and in line with behavioral genetic research, these "independent" environmental effects contain high dosages of genetic influences—influences that make it impossible to assume that the environment is not molded by genes (Ridley, 2003; Rutter, 2006; Scarr and McCartney, 1983).

***Research Question Three*: Do the dopaminergic, serotonergic, and MAOA genetic polymorphisms interact with the social environment to predict involvement in antisocial activities?**

The last column in Table 6.1 presents a summary of the findings for the results of the interactive effects models testing for gene X environment interactions (GxE). For the interactive models, the effects of the genetic polymorphisms were examined in different environments. Specifically, the effects of the polymorphisms were estimated for a low delinquent peers group versus a high delinquent peers group and for respondents from high-risk families versus respondents from low-risk families. GxEs were detected when genes had significant effects in one environment (e.g., low-risk environments), but failed to have a significant effect in the other environment (e.g., high-risk environments).[21]

[21] In Table 6.1, the denominator for the interactive effects model was calculated in accordance with the following procedure. The results of the high versus low delinquent peers group (and the high- versus low-risk families) were compared to each other. If the genes had different effects depending upon the group they fell in, then a GxE was observed. If the effects of the genetic measures were significant in both models or insignificant in both models, then a GxE was not detected. Essentially, two models were being compared to determine if one GxE is evident. As such, the denominator is a count of how many models were being compared; it is not a count of the number of models estimated.

The final research question (i.e., research question three) was concerned with whether the dopaminergic, serotonergic, and MAOA genetic polymorphisms would interact with the social environment to predict involvement in antisocial activities. It was hypothesized that the genetic polymorphisms would interact with delinquent peers and with family risk to predict the seven different measures of delinquent and criminal behaviors. Additionally, it was hypothesized that the genetic effects would be much stronger and much more consistent in the interactive models when compared with the direct effects models.

As revealed by Table 6.1, all of the genetic polymorphisms interacted with the two measures of the social environment to predict variation in the outcome measures. Over 20 percent of all the interactive models for DAT1, DRD2, and MAOA detected a significant GxE. Additionally, 12 percent of all the DRD4 interactive models were significant, and 19 percent of all the interactive models for 5HTT were statistically significant. Altogether, 22 percent of all the interactive models detected a GxE. These findings are generally supportive of the first hypothesis.

The following list of results highlights the main genetic findings for each of the dependent variables in the interactive effects models:

- **Wave I delinquency scale**: 4 significant dopamine effects, 2 significant serotonin effects, and no significant MAOA effects
- **Wave II delinquency scale**: 7 significant dopamine effects, 3 significant serotonin effects, and 2 significant MAOA effects
- **Wave III delinquency scale**: 8 significant dopamine effects, 1 significant serotonin effect, and 1 significant MAOA effect

- **Police contacts**: 5 significant dopamine effects, 1 significant serotonin effect, and 2 significant MAOA effects
- **Arrest status**: 3 significant dopamine effects, 1 significant serotonin effect, and 1 significant MAOA effect
- **Marijuana use**: 9 significant dopamine effects, 4 significant serotonin effects, and 4 significant MAOA effects
- **Alcohol abuse**: 8 significant dopamine effects, 1 significant serotonin effect, and 3 significant MAOA effects

When comparing the results of the direct effects models with those of the interactive effects models, it is immediately obvious that the second research question is also supported. For example, the interactive effects models have a much more consistent effect on a range of antisocial behaviors. For every polymorphism, a higher percentage of significant findings were detected for the interactive effects models than for the direct effects models. When the results of the direct effects models were pooled together, 14 percent of all the analyses detected a significant direct effect. In contrast, 22 percent of all the interactive effects models were statistically significant.

Analysis of the Add Health data thus support prior empirical and theoretical research revealing the importance of GxEs in the study of crime and criminals (Caspi et al., 2002; Foley et al., 2004; Rutter, 2006; Walsh, 2002).[22] Many of the genetic effects reported in Table 6.1 were only detected when certain environmental stimuli were present. Without examining how genetic influences ebbed and flowed in different environments, many significant genetic effects would have been masked. The results of the interactive effects models are a testimony to the

[22] Prior research has tended to focus solely on white respondents making it nearly impossible to compare the results of the findings for blacks to those reported in prior published literature.

importance of examining how genes act on the environment and how the environment acts on genes to create antisocial behaviors.

As mentioned previously, part of the reason that criminologists and sociologists have been antagonistic to biogenic explanations of crime is because they are viewed as deterministic. The findings garnered from analysis of the Add Health data should help alleviate some of these concerns. For the interactive effects models, the only time that a particular genetic polymorphisms exerted an effect on the outcome measure was when it was paired to a specific environment; change the environment and the genetic effect evaporates. Only by examining the complex interplay between genes and the environment will a richer understanding of criminal and delinquent behavior be realized (Raine, 1993; Rutter, 2006; Walsh, 2002).

Too many social scientists still view the nature versus nurture debate as alive and well (Pinker, 2002; Ridley, 2003). Publications revealing strong genetic influences on phenotypes only add more fuel to the fire; subsequent failed replications of a genetic effect leave social scientists perplexed. The problem, however, is that the nature-nurture debate is outdated and has already been answered: human development is the result of nature and nurture working *together*. Genes have strong influences on the environment (as revealed by rGEs), while the environment has an equally important effect in conditioning the effect of particular genes (as revealed by GxEs). In order to gain a richer understanding of the causes of crime, criminological research needs to begin to examine the ways in which the environment and genes interact to produce serious, violent, and aggressive behaviors. As the findings in this dissertation show, both the environment and genes make substantial contributions to the study of offending behaviors.

Limitations and Directions for Future Research

The current dissertation provides empirical evidence showing that many types of antisocial behaviors, for many different types of people, are influenced by genetic factors. Before proceeding, however, it is important to discuss the main limitations of this dissertation. The first main limitation of this study is that not everyone in the Add Health data was genotyped.[23] This drawback precludes the ability to infer whether the results for the rGEs and for the GxEs would necessarily hold for a larger sample of respondents. However, two qualifications caution against explaining the findings away in terms of data limitations. First, prior research analyzing the Add Health data has found that the distribution of alleles for certain of the genetic polymorphisms to be very similar to those found in the general population (Hopfer et al., 2005). These results hint at the possibility that the genetic sample is a representative cross-section of American adolescents and young adults. Second, the sample size for the Add Health data is much larger than those usually employed in genetic studies. Quantitative genetic studies often employ clinical samples that are not an adequate representation of the population at large. The Add Health data provides an important exception to the general rule of using convenient samples of patients. Future research would benefit by replicating the analyses reported here with a different sample of respondents.

The second main limitation of the analyses is that respondents in the Add Health sample are mainly adolescents and very young adults. The truncated age range of the Add Health data precludes the ability to examine how particular genetic polymorphisms may affect life-course transitions that occur during adulthood. However, recent research by Sampson and Laub (2005; Laub and Sampson, 2003) provides circumstantial evidence hinting at the possibility that gene X

[23] The Add Health research design team recognizes the importance of genotyping a large, nationally-representative sample of respondents. Currently, efforts are underway to genotype all Add Health respondents during wave IV interviews.

environment correlations are at work in the desistance process. In their work, Sampson and Laub argue that "human agency," or choice, is a major reason that some individuals choose to abstain from offending behaviors, even after a very lengthy involvement in crime (Laub and Sampson, 2003; Sampson and Laub, 2005). For example, Sampson and Laub (2005:14) advocate "a life-course view that emphasizes human agency and choice over the life span, underscoring how people construct their lives..." Furthermore, they "want to ask the hard question of how men with a criminal past go about prospectively *creating* their own trajectories." Laub (2006:244) continues by arguing that "individuals, whether criminal actors or not, make choices and are active participants in the construction of their lives."

For Sampson and Laub, human agency plays a key role in facilitating the desistance process. But a close reading of their work (as evidenced in the above quotes) reveals that their definition of "human agency" closely parallels the logic of active rGEs, where people engage in "niche-picking" based, in large part, on their genetic predispositions (Scarr, 1992; Scarr and McCartnely, 1983; Walsh, 2005). Of course, this is an empirical question that can ultimately be answered through data analysis. But for now, the point remains that gene-environment interplay (e.g., rGEs) has the potential to explain or at least shed some light on the process that lead not only to criminal involvement, but also to desistance.

The third main limitation of this study is that many of the measures used to operationalize traditional criminological theories (e.g., strain, social bonding, and low self-control) were not controlled for in the statistical models. These measures were not included in the analyses because there was not reason to believe that the genetic polymorphisms shared variation with measures typically used by criminologists.[24] A variable can be omitted from multivariate

[24] The skeptical reader may point out the possibility that certain of the genetic polymorphisms could be related to the development of self-control (Wright and Beaver, 2005). To explore this possibility, and following prior research

equations without misspecification, as long as the omitted variable is not related to the independent and dependent variables (Hanushek and Jackson, 1977). Given the lack of correspondence between the polymorphisms and measures of major theoretical constructs, misspecification does not appear to bias the results.

The fourth and final main limitation of this dissertation is that only five different genetic polymorphisms were examined. These genes only represent the tip of the genetic iceberg. But, as the functionality of more and more genes is discovered, the potential for additional "risk genes" to explain variation in antisocial behavior is very likely. For example, Rujesco and colleagues (2003) recently discovered that variants of the COMT polymorphism were differentially related to the development of aggressive personality traits. Future research is needed to examine how different genes—genes that have not yet been implicated in the etiology of psychopathology—may be associated with various forms of antisocial conduct.

Even with these limitations in mind, the statistical models provided a very conservative test of the genetic effects on a range of antisocial outcomes. Most of the extant genetic literature fails to use multivariate statistical models to control for extraneous influences. In this dissertation, the effects of some very potent criminogenic environments—delinquent peers and family risk—along with the effects of key control variables were held constant. Only after partitioning out the effects of these other influences were the genetic effects estimated. Moreover, in some models (i.e., the dopaminergic models), the genetic polymorphisms competed with each other to explain variation in the dependent variables. Nonetheless, when using these

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analyzing the Add Health data (Perrone, Sullivan, Pratt, and Margaryan, 2004), a measure of low self-control was created. Next, bivariate correlations were calculated to determine whether there were any significant correlations between the genes and the self-control scale. The results did not reveal any significant correlations. In order to preserve degrees of freedom, the self-control scale was not included in the multivariate models. Future research may wish to explore whether GxEs are about to account for the development of self-control.

conservative statistical methods, significant direct, indirect, and interactive effects were found for the dopaminergic genes, the serotonin transporter gene, and the MAOA polymorphism.

Implications for Criminology

The results of this dissertation reveal that genes influence not only behaviors, but also measures of the social environment. Still, we are left with the resonating question of how these findings affect criminology and criminologists. While not exhaustive, three different implications are offered. First, in order to stay abreast of the mushrooming body of research revealing strong genetic influences on all types of behaviors and personality traits, criminology must make room for biosocial explanations to crime and criminality. Biosocial criminology has the capacity to explain many of the "brute" facts of crime, including the age-crime curve, racial and gender gaps in delinquent/criminal involvement, and the persistence of criminal behavior over long periods of time (Ishikawa and Raine, 2003; Niehoff, 1999; Raine, 1993; Walsh, 2002, 2004). To pretend that biogenic factors have no bearing on criminal behavior is to turn a blind eye to a perspective that has the very real possibility of providing criminologists with a rich perspective from which to study antisocial behaviors (Walsh, 2002).

Second, and relatedly, criminologists need to shed their ideological allegiance to sociology and embrace an interdisciplinary approach to the study of crime (Laub, 2006)—an approach that includes biological and genetic influences (Walsh, 2000). By narrowly focusing on how social factors promote criminal activities, social scientists have ignored a plethora of biological, genetic, and neuropsychological research revealing the complexity of human behavior. Biosocial criminology incorporates findings from these disciplines and merges them with those of the more traditional soft science explanations of crime (Walsh, 2000, 2002;

Wilson, 1998). A multidisciplinary approach to the study of human behavior has the potential to make great strides towards unpacking the origins and causes of serious violence (Walsh, 2002; Walsh and Ellis, 2003; Wilson, 1998; Wright and Beaver, 2005). Walsh (2002:221) captured the possible contributions that a biosocial perspective has when he stated that "biosocial criminology is *an* answer, not *the* answer to progress in criminology."

Third, the nature/nurture distinction needs to be abandoned in favor of more refined explanations that accurately reflect the close interplay between genes and the environment (Rutter, 2006; Walsh, 2002). Pigeon-holing a statistical variable as either an environmental measure or a genetic measure misses the picture: the social environment is so deeply intertwined with genes that it is nearly impossible to parcel out the two effects. Even measures that are thought to be tapping the social environment are probably heavily influenced by biological and genetic factors (Beaver, Wright, and DeLisi, 2006; Cleveland, Wiebe, and Rowe, 2005; Walsh, 2002).

For example, associating with delinquent peers and being raised in a criminogenic family—measures thought to be purely social—are two of the more commonly invoked explanations for delinquent involvement (Akers, 1998; Gottfredson and Hirschi, 1990; Laub and Sampson, 1988; Loeber and Stouthamer-Loeber, 1986; Patterson, 1982; Warr, 2002). Analysis of the Add Health data (in Chapter 5), however, revealed a strikingly different possibility: affiliating with antisocial friends and residing in a high risk family were partially a reflection of an individual's genotype. The point is that most environments—especially those studied by criminologists—have strong genetic underpinnings to them; trying to argue otherwise goes against mounds of evidence revealing that genes and the environment are too tightly wrapped to

examine separately (Ridley, 2003). A biosocial perspective is needed to explain how genes and the environment interlock to produce crime.

Conclusion

For far too long, criminologists have remained true to their sociological roots and marginalized biosocial explanations of crime (Walsh, 2002; Walsh and Ellis, 2004). "When criminology looks to single disciplines such as sociology, psychology or economics," John Laub argued (2006:248) in his 2005 Sutherland Address to the American Society of Criminology, "the field does not advance in large part because those disciplines seek to establish institutional hegemony by imposing their research agenda on the field of criminology." Laub (2006:248) continued his Sutherland Address by posing the following question: "What is so wrong with drawing on other disciplines…if they add to our understanding of crime?" Biosocial criminologists have followed Laub's advice and drawn from diverse fields of inquiry to provide rich explanations of crime and delinquency (Beaver and Wright, 2005; Benson, 2002; DeLisi, 2005; Raine, 1993, 2002a; Reiss et al., 2000; Rowe, 2002; Rutter, 2006; Walsh, 2002; Wright and Beaver, 2005).

Still, biosocial research is often ridiculed, trivialized, or ignored outright (Daly and Wilson, 1988; Walsh, 2002; Walsh and Ellis, 2003). There can be little doubt, however, that as the 21st century marches on, biosocial criminology will hold the key to uncovering the dynamic processes that unfold and contribute to the development of antisocial behaviors. Until a biosocial approach to the study of crime is accepted, traditional theories of crime will remain underdeveloped, incomplete, and impoverished. With the recent mapping of the human genome and with the almost daily discoveries about the function of certain genes, the time is ripe to

embrace biosocial explanations to the study of crime and delinquency (Beaver and Wright, 2005; Raine, 1993; Walsh, 2002; Wright and Beaver, 2005).

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Appendix A. Description of Add Health Measures and Scales Used in the Analyses

Independent Variables

Genetic Polymorphisms

Dopamine Transporter Gene (DAT1):

1. The number of 10-repeat alleles the participant possesses

Dopamine Receptor Gene (DRD2):

1. The number of A1 alleles the participant possesses

Dopamine Receptor Gene (DRD4):

1. The number of 7-repeat alleles the participant possesses

Serotonin Transporter Gene (5HTT):

1. The number of short alleles (484 base pair) the participant possesses

Monoamine Oxidase A Promoter Gene (MAOA):

1. The number of low-activity alleles the participant possesses

Socialization Measures

Delinquent Peers at Wave I: Of your three best friends, how many:

1. Smoke at least one cigarette a day?
2. Smoke pot more than once a month?
3. Drink alcohol at least once a month?

Family Risk at Wave I:

1. How close do you feel to your mother?
2. How much do you think your mother cares about you?
3. Most of the time, your mother is warm and loving toward you
4. Your mother encourages you to be independent
5. When you do something wrong that is important, your mother talks about it with you and helps you understand why it is wrong
6. You are satisfied with the way you and your mother communicate with each other
7. Overall, you are satisfied with your relationship with your mother

In the past 4 weeks, have you and your mother:

8. Gone shopping?
9. Played a sport?
10. Gone to a religious service or church-related event?
11. Talked about someone you are dating or a party you went to?
12. Gone to a movie, play, museum, concert or sports event?
13. Had a talk about a personal problem you were having?
14. Talked about your school work or grades?
15. Worked on a project for school?
16. Had a serious argument about your behavior?
17. Talked about other things you are doing in school?

Control Variables

Age
Race
Gender
Cognitive Complexity

Dependent Variables

Delinquency Scales

Delinquency Scale at Wave I: 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. Lie to your parents or guardians about where you had been?
4. Take something from a store without paying for it?
5. Get into a serious physical fight?
6. Hurt someone badly enough to need bandages or care from a doctor or nurse?
7. Run away from home?
8. Drive a car without its owner's permission?
9. Steal something worth more than $50?
10. Go into a house or building to steal something?
11. Use or threaten to use a weapon to get something from someone?
12. Sell marijuana or other drugs?
13. Steal something worth less than $50?
14. Take part in a fight where a group of your friends were against another group?
15. Act loud, rowdy, or unruly in public?

Delinquency Scale at Wave II: 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. Lie to your parents or guardians about where you had been?
4. Take something from a store without paying for it?
5. Run away from home?
6. Drive a car without its owner's permission?
7. Steal something worth more than $50?
8. Go into a house or building to steal something?
9. Use or threaten to use a weapon to get something from someone?
10. Sell marijuana or other drugs?
11. Steal something worth less than $50?
12. Act loud, rowdy, or unruly in public?
13. Take part in a fight where a group of your friends were against another group?
14. Have you been initiated into a named gang?

Delinquency Scale at Wave III: In the past 12 months, how often did you:

1. Deliberately damage property that didn't belong to you?
2. Steal something worth more than $50
3. Go into a house or building to steal something?
4. Use or threaten to use a weapon to get something from someone?
5. Sell marijuana or other drugs?
6. Steal something worth less than $50?
7. Take part in a fight where a group of your friends were against another group?
8. Buy, sell, or hold stolen property?
9. Use someone else's credit or bank card without their permission or knowledge?
10. Deliberately write a bad check?
11. Use a weapon in a fight?
12. Carry a handgun at school or work?

Involvement with the Criminal Justice System

Number of Police Contacts:

1. How many times have you been stopped or detained by the police for questioning about your activities? Do not count minor traffic violations.

Ever Arrested:

1. Have you ever been arrested or taken into custody by the police?

Drug and Alcohol Abuse Scales

Marijuana Use at Wave I:

1. During the past 30 days, how many times did you use marijuana?

Alcohol Abuse at Wave III: During the past 12 months, how many times have you:

1. Had problems at school or work because you had been drinking?
2. Had problems with your friends because you had been drinking?
3. Had problems with someone you were dating because you had been drinking?
4. Been hung over?
5. Gotten sick to your stomach or thrown up after drinking?
6. Gotten into a sexual situation that you later regretted because you had been drinking?
7. Gotten into a physical fight because you had been drinking?
8. Been drunk at school or work?