

THE EFFECT OF PARENTAL INFLUENCE AND THE DOPAMINE TRANSPORTER  
(DAT1) GENE ON EARLY ANTISOCIAL BEHAVIOR IN CHILDREN:  
DETERMINING RISK FOR DELINQUENCY USING A GxE

by

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An Abstract

of a thesis submitted in partial fulfilment  
of the requirements for degree of  
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## ABSTRACT

by

Trevor A Gonzales

This study examined the effect of negative maternal behaviors and the child's dopamine transporter polymorphism on early signs of delinquency in children. Using data from the Fragile Families and Child Wellbeing Survey, conducted by Princeton University, the child's DAT1 polymorphism will be examined and tested as part of a gene environment interaction (GxE) to determine a possible relationship between environments with exposure to negative maternal behaviors, and different DAT1 polymorphisms in the child, to predict early signs of delinquency. Four different maternal behaviors were examined; alcohol use, drug use, punishment of the child, and lack of maternal attachment. Each of these behaviors was examined along with the DAT1 gene of the child using two models for each variable. The first examined the behavior and gene independently, the second included a variable for a GxE between them. The GxEs between the maternal behaviors and the DAT1 genes did not have a significant impact on delinquency in any of the tests. Maternal drug use, punishment of the child, and child's lack of attachment to mother were all significantly related to increased delinquency.

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## CHAPTER I INTRODUCTION

Research on juveniles has shown that delinquent behavior is impacted by childhood development and social learning (Akers, 2011; Ary, Tildesley, Hops & Andrews, 1993). Exposure to negative environments creates a greater risk of adopting antisocial or delinquent behavior through developmental learning processes (Akers, 1998; 2011). The sensitivity to these influences is mediated by genetic polymorphisms, which can alter susceptibility to environmental influence and serve as either a risk or protective factor (Scarr & McCartney, 1983). This research will examine the effect of negative parental behaviors and the dopamine transporter polymorphism on early signs of delinquency in children. Using data from the Fragile Families and Child Wellbeing Survey, conducted by Princeton University, the child's DAT1 polymorphism will be examined and tested as part of a gene environment interaction (GxE) to determine a possible relationship between environments with exposure to negative parental behaviors, and different DAT1 polymorphisms in the child, to predict early signs of delinquency.

Learning is the driving determinant of human behavior and development. The way we interact with our physical and social environment is derived from the process of a variety of learning experiences (Akers, 1998; 2011). This process starts with experiences, which can result in rewards or punishments, then subsequent behavior is adapted based on these outcomes and expectations of future outcomes (Akers, 1985; Tittle, Antonaccio & Botchkovar, 2012). Learning is individualized and accomplished through varying methods and processes. Stimuli and environments may impact individuals differently based on their genes, which alter the effects of environmental influences, creating a variety of possible behaviors and personalities resulting



from similar experiences and environments (Belsky & Pluess, 2009; Scarr & McCartney, 1983; Wright, Schnupp, Beaver, Delisi & Vaughn, 2012).

For young children especially, parental relationships and family bonds are the primary learning environment and shape behavior based on these learning conditions, relationships and stimuli (O'Connor, Matias, Futh, Tantam & Scott, 2013). At an early age, behavior is learned and imitated by observing family and peers, and the environmental stimuli associated with different actions and behaviors (Akers, Krohn, Lanza-Kaduce & Radosevich, 1979; O'Connor et al., 2013). Future behavior is formed based on both the anticipation of rewards or punishments associated with a specific behavior (Akers, 1998; 2011; Yun & Kim, 2015) and modeling actions from others through observational learning (DeMartino, Rice & Saltz, 2015). By observing the behavior of others, individuals can identify likely outcomes of different actions and imitate behaviors that have a high probability of resulting in reward. This imitation results in learning of behaviors by copying actions of others in similar environments (Akers, 2011; Yun & Kim, 2015). Individuals modify behavior based on the expected stimuli associated with actions and behaviors, to increase anticipated rewards and avoid unwanted consequences (Akers et al., 1979; Akers, 1985; DeMartino et al., 2015). This process of behavior guided by anticipation of punishment or reward is known as operant conditioning (Watson, 1969).

The current study seeks to examine the intersection of the tenets of social learning theory (Akers, 1998) and genetic risk to investigate childhood antisocial behavior. Four hypotheses will be tested, and each will examine the impact of one of four parental influences on child behavior that, when paired with certain DAT1 genes, may lead to a higher risk of antisocial behavior in early childhood. Certain environmental factors, such as parenting styles and parental bonds, may have a more prominent impact on the development of children when paired with specific genes

of the child (Caspi et al., 2002). Therefore, in a family environment with poor parenting, where children are exposed to negative parental behaviors, it follows that children would be more likely to engage in antisocial or deviant behavior when they possess high-risk genes. By expanding the knowledge of how specific genes and environments interact, this research will identify potential predisposed risks for delinquency. Identifying parental behavior that can influence delinquency in children, when occurring in concert with certain genes, will create a better understanding of how parenting techniques can be shaped to prevent delinquent behavior.

## CHAPTER II LITERATURE REVIEW

Learning transpires through a variety of modes and processes, including experiences, observations, and resulting changes to the environment. The process of learning takes place as individuals acquire or adapt new behaviors to increase anticipated benefits. These behaviors are learned from the individual's social environment, primarily from close personal associations (Akers, 2011). Learned behavior has historically been thought of to be entirely a product of exposure to different environments, but recent research has identified certain biological factors which may impact one's potential to learn (Scarr & McCartney, 1983; Schultz, 1998). The experience of these learning stimuli is individualized based on unique genes. Genes define the biological impact different stimuli will have and how they will be internally perceived, and different variations of genes may make an individual more or less able to predict rewards from behaviors, or determine potential risks associated with their actions (Schultz, 1998). Learning occurs as part of a process that can be moderated by the predisposition to reinforcement and environmental influences of the individual (Schultz, 1998). By changing the way these learning processes are experienced, genes account for variation in learning within similar environments and can influence how stimuli affect future behavior (Watts & McNulty, 2014). Learning is a process that takes multiple steps and influences before a behavior is adopted, used and repeated. This process of learning can be applied to understanding how criminal behavior is developed, just like any other behavior, and can be partially explained and understood as a result of genetic influence on the ability to learn and the sensitivity to environmental influences.

## Social Learning Theory

Social learning theory is considered to be one of the most cited and empirically supported criminological theories (Akers, 2011). Social learning theory seeks to explain the causes, changes, and continuation of delinquent and criminal behavior (Yun & Kim, 2015). It explains not just the factors that contribute to the development of criminal behavior, such as deviant peer groups or associations, but also mitigating and protective factors from the social environment (Yun & Kim, 2015). Burgess and Akers (1966) derived their modern conceptualization of social learning from Sutherland's (1947) differential association theory, which proposed that criminal behavior is learned the same way as any other behavior, predominantly from intimate social groups. Differential association-reinforcement theory, created in the mid-1960s, retained the concepts of differential associations and definitions from Sutherland's differential association theory, and added the ideas of differential reinforcement and imitation (Bandura, 1962; Skinner, 1963). The theory has since been modified into one of the most frequently cited theories in criminology (Cochran, Maskaly, Jones & Sellers, 2017).

Sutherland's original theory described criminality as a behavior that was learned, like any other behavior (e.g., tying one's shoes, language, or driving) primarily from interaction with members of an intimate association, group, or family through differential association. Differential association is the interaction with significant others, such as peers, groups, and family, that results in the learning and adoption of their values, techniques, and attitudes regarding criminal and delinquent behavior (Akers, 2011; Yun & Kim, 2015; Cochran et al., 2017). Through close association with individuals, both indirect and direct, their values and beliefs are observed and learned, which can act as a protective or risk factor based on the relationship and their definitions (Yun & Kim, 2015). "Definitions" refer to the meaning

individuals attach to their behavior, and how it affects learning based on if it is favorable or unfavorable for crime. Sutherland's theory focused primarily on definitions, which define a person's own values, beliefs, attitudes, or meanings assigned to different behaviors and actions (Akers, 2011; Cochran et al., 2017). Close association with individuals who maintain definitions that favor, or reward criminal behavior will result in the learning of these values and therefore increase the propensity or likelihood of criminality. The frequency, duration, priority, and intensity of an individual's relationships and associations determines the effect differential association has on that individual's risk for criminal behavior through social learning of techniques, ideals, and values (Yun & Kim, 2015).

Definitions can be general or specific and are learned from associations with other individuals. These definitions can be either favorable or unfavorable for criminal behavior. Criminal behavior is developed when definitions learned through interactions that favor criminal behavior outweigh the definitions in favor of social conformity to the law (Yun & Kim, 2015). An individual's attitude or belief regarding a certain act determines the definition they attach to it, whether it is an acceptable action or belief or not (Cochran et al., 2017; Rader & Haynes, 2011).

Akers and Burgess elaborated on the criminal learning process by adding the concepts of differential reinforcement and imitation to Sutherland's base of differential associations and definitions. Differential reinforcement describes how individuals perceive the consequences of their actions based on what they have witnessed or experienced in their environment (Akers, 2011). Differential reinforcement is the learning concept that past experiences and observations of consequences formulate expectations of reward or punishment of particular behaviors (Akers, 2011; Tittle et al., 2012). Social learning theory identifies differential reinforcement as the core

learning mechanism for developing behavior (Tittle et al., 2012). Criminal behavior, like other behaviors, is learned through a process of operant conditioning, where behaviors are acquired or modified based on the effects, consequences or outcomes resulting from that behavior in the environment (Akers, 1985; Akers, 1979; Winfree, Mays & Vigil-Bäckström, 1994; DeMartino, Rice, & Saltz, 2015). An individual's choice to actively refrain from, engage in, or continue criminal actions is determined by the perceived probability of reward versus the chances of punishment associate with the action (Akers, 1998; Tittle et al., 2012). If the anticipated rewards of engaging in these behaviors outweigh the potential risks or consequences, individuals are more likely to commit criminal actions. An individual's perception of potential risks and rewards is developed from past experiences, observations of other interactions, and the results of similar actions within the environment; these factors are used to anticipate the outcome of a behavior and weigh the possible risks against the perceived rewards (Akers, 1998; Tittle et al., 2012).

The final theoretical component of Akers and Burgess's social learning theory is imitation. The imitation of others involves the copying or modeling of one's own behavior based on the actions of another, after observing the outcomes of those actions and rationalizing probable consequences (Akers, 2011; Akers, 1998; Yun & Kim, 2015). Individuals imitate the actions and behaviors they observe, particularly when individuals perceive these behaviors to have a high probability of reward. By observing a behavior, legal or illegal, an individual is able to deduce the likely outcomes of adopting this behavior and model subsequent actions based on the perceived consequences or rewards (Akers, 1998; Yun & Kim, 2015). Part of imitation also is the copying of techniques or methods used to complete crimes. In order to engage in criminal behavior, an individual must know how to successfully perform that behavior, whether it

requires certain tools, techniques or skills, and imitation provides a learning process for them to observe these methods (Yun & Kim, 2015).

Akers and Burgess expanded on Sutherland's theory of differential association by theorizing that criminal behavior is also learned from the environment through non-social situations (e.g. reading books or video games) and not just through interaction with peers (Thompson, 2013). Social learning theory describes criminal behavior as a learned process, where the definitions favoring crime, as well as the actions to complete it, are transmitted or learned from individuals (Akers, 2011). Akers and Burgess's continued development of social learning theory primarily focuses on the four main theoretical concepts of differential association, definitions, differential reinforcement, and imitation (Cochran et al., 2017). Akers also outlined the sequence of events that result in criminal behavior, based on the four theoretical concepts. He proposed that individuals are initially exposed through differential association, and if this exposure results in interaction with individuals who have favorable definitions of crime, they adopt these definitions (Akers, 2011; Akers et al., 1979). The individuals then act as a source of imitation, with their behaviors being observed and modeled, they then provide differential reinforcement for these newly learned definitions and behaviors (Akers et al., 1979).

The majority of studies that attempt to empirically test social learning theory offer support of these four primary theoretical concepts; their effect on criminal behavior is especially apparent when applied to peer groups or family and can account for variations in individual criminal or delinquent behavior (Akers, 2011). The four primary concepts of social learning theory provide an explanation of crime when applied individually or in conjunction with one another. Steele, Peralta and Elman (2011) conducted a study examining social learning theory applied to simultaneous use of non-prescription drugs and alcohol, and their research showed

support for the influence of differential association, social reinforcement, and definitions, on the co-use of non-prescription drugs and alcohol. In 1996, Akers and Lee applied social learning theory to adolescent smoking in a longitudinal study that contained variables for differential association, differential reinforcement, definitions, and modeling. Their study attempted to not only identify support for the independent effects of the social learning variables, but also for the theoretical sequence of these variables in the social learning theory. Ultimately the study was supportive of the social learning explanation of deviant behavior; and, although all four social learning variables were significant when applied to adolescent smoking, peer associations had clearer effects than definitions and reinforcement (Akers & Lee, 1996). Social learning theory has gained a great deal of empirical support from a multitude of studies; evidence has been found in favor of both the independent variables of social learning theory, and the more complex explanation of the learning process of criminal behavior (Akers, 2011; Akers & Jensen, 2006; Akers & Lee, 1996).

Social learning theory, originally proposed by Akers as differential association-reinforcement theory, has been extensively tested, gaining support and empirical validity for its four primary theoretical components (Akers, 1998; Akers, 2011; Akers et al., 1979; Akers & Jensen, 2006). Tests of social learning theory have found repeated evidence in favor of its propositions, especially the learning process of criminal behavior (Akers, 1998; Akers, 2011). A key source of learning experiences for children is the parental environment, through which children initially learn to cope with disputes, manage emotions, interact with others, and maintain social bonds from experiences and observations (O'Connor et al., 2013).

Therefore, it follows that many tests of social learning theory have focused on examining the child-parent relationship and its influence on antisocial behavior. O'Connor, Matias, Futh,



Tantam and Scott (2013) examined the effects of implementing a social learning theory-based intervention strategy to promote parent-child relationships. The intervention group showed an increase in favorable relationship behaviors compared to the group that was not treated, these results seem to support the use of a behavioral intervention plan, based on social learning theory, to increase positive aspects of parent-child relationships. Ary, Tildesley, Hops and Andrews (1993) conducted a study over one year that observed the influence of parent, sibling, and peer use (imitation) and attitudes (definitions) regarding alcohol use by adolescents. Both the parents attitude of adolescent use, as well as their modeling of alcohol use showed a strong relationship to change in the use of alcohol by adolescents. These results indicate that parental behavior impacts adolescent behaviors through social learning concepts of imitation and definitions (Ary et al., 1993). Adolescent alcohol use was also influenced by peer and sibling imitation and attitudes, but not to the extent of parental influence.

#### Gene x Environment Interactions and the Role of Dopamine

Parental influence and behavior creates the social environment children are exposed to at their earliest stages; and when this environment is paired with high risk genotypes, children are at risk for increased delinquency (Nilsson, Comasco, Hodgins, Orelund & Åslund, 2015). Genes determine the development of behavior, mediate and moderate the impact of experiences, and how stimuli affect individual perceptions and attitudes (Scarr, 1992; Scarr & McCartney, 1983). Previous research has identified at-risk genes that may be linked to increased antisocial behavior, ADHD, delinquent behavior, and criminal actions (Cornish et al., 2005; Jackson & Beaver, 2012; Nilsson et al., 2015; Tong et al., 2015;).

Genes determine physical and behavioral predispositions of individuals, and act as the initial primer for all actions and reactions of an individual (Wright et al., 2012; Scarr &

McCartney, 1983). The term *genotype* refers to the different possible combinations of alleles found within individual genes. The ability of genes to possess varying individual alleles makes them *polymorphic*, meaning that they can have several forms, where each polymorphism may result in different characteristics. Genes determine how brain processes, such as neurotransmission, are carried out and achieved. They also define the blueprint for brain structure. How well one's brain functions is influenced by both brain structure and neurotransmission – a brain with deficits in structure or neurotransmission will not function as well as a properly developed brain. The development of behavior, as well as the processing of emotions and decisions are all due to brain function (Scarr & McCartney, 1983). In this way, genes help initially determine behavior and emotions of the individual.

Genes predispose individuals to possess higher protective or at-risk attitudes, altering how they interact with their environment and what types of behaviors they engage in (Nilsson, et al., 2015; Wright et al., 2012; Jackson & Beaver 2012; Scarr & McCartney, 1983). The risk associated with genes does not reflect the likelihood the gene will cause an individual to engage in or resist antisocial behavior, but rather it refers to the susceptibility of that individual to environmental influences and how these will affect behavior (Wright et al., 2012). If an environment transmits greater risky or antisocial behavior, and the individual has a highly sensitive gene type, their risk for these behaviors is increased not because of the gene, but because of its relationship with the environment. A high-risk gene, which increases the sensitivity to stimuli, does not cause an individual to commit crime, but when they are in an environment which teaches and reinforces criminal actions, they are more likely to succumb to these influences and adopt this behavior due to the combination of the gene and the stimuli. Past research has shown evidence that genes, such as those that regulate serotonin levels in the brain,

may impact the degree a person exhibits antisocial behavior (Moore, Scarpa & Raine, 2002; Tung & Lee, 2017). A meta-analysis of the serotonin transporter gene (5HTTLPR) and the monoamine oxidase A gene (MAOA-uVNTR) found that specific genetic variants of these genes were significantly associated with antisocial behavior; that risk was dependent on the availability of serotonin, which is regulated by these genes along with several others (Ficks & Waldman, 2014). The difference in sensitivity of genes is due to the variation in the way the genes transmit, activate, and process neuron signals in the brain, which determines how rewards are measured and predicted (Schultz, 1998), how reinforcers of behavior are interpreted, and how individuals react to a variety of stimuli (Ellis & Boyce, 2011).

#### Gene x Environment Interaction

Recent findings in the field of biosocial criminology suggest that environmental factors may increase delinquency based on an individual's genes (Boardman, Menard, Roettger, Knight, Boutwell, & Smolen, 2014). High-risk combinations of genes and environmental stimuli can create a higher likelihood of adverse effects than mere exposure to genetic risk or environmental risk alone. Genes moderate an individual's ability to experience the environment and develop behavior (Scarr & McCartney, 1983; Belsky & Pluess, 2009; Rutter, 2006). Individual differences in behavioral responses to a similar environment can be attributed to genetic variations, which is referred to as a Gene x Environment Interaction (GxE) (Rutter, 2006; Rutter, 2007; Wright et al., 2012). The interplay of both components in the GxE determines behavior, such that the environment determines the factors influencing behavior, and the genes moderate the sensitivity to these factors (Belsky & Pluess, 2009; Scarr & McCartney, 1983; Scarr, 1992). Individuals who have specific genotypes that are more sensitive to environmental forces will have a greater risk for antisocial behavior when exposed to negative environments.

GxEs that contain high sensitivity genes can dramatically increase the adversity and problems experienced by individuals who are exposed to negative and antisocial environments (Berry, Deater-Deckard, McCartney, Wang & Petrill, 2013; Scarr & McCartney, 1983).

A GxE explains the individualized ways that behavior can be developed within the same environment, with the genotypes as the driving factors of behavior, since they alter susceptibility to these environments (Rutter, 2006; Scarr & McCartney, 1983; Wright et al., 2012). For example, individuals with genetically less melanin will be more sensitive to UV rays, and therefore are more likely to experience sunburn when spending time outside. Genes provide differential basis for the environment's impact – in this case, a person with fair skin is more likely to experience sunburn when experiencing the same environment as a person with darker skin. The interplay of the environment and genotypes is not a parallel relationship with equal weights of influence. Scarr and McCartney (1983) stated that genes and environments affect each other as well as the development of behavior, but the relationship is not dichotomous since genes alter the effects of environment, and vice-versa. In some instances, an environmental force may be the primary developer of behavior due to its impact, in other scenarios the genetic predispositions of an individual may cause them to pursue certain environments or alter how they are affected by them. The environment determines the types of stimuli and experiences an individual is exposed to, but genes determine how this exposure affects subsequent behavior, and genes can also play a role in the types of environments an individual engages in or experiences (Scarr, 1992; Scarr & McCartney, 1983).

To demonstrate this concept, researchers found that genes related to delinquency are expressed based on the environment, with low social control environments amplifying the effects of the genes and high social control environments resisting the effects (Liu, Li, & Guo, 2015).

Using an analysis of three dopamine genes, Barnes and Jacobs (2012) found that the effect of genetic risk factors on violent behavior was much more predominant when also accompanied by exposure to disadvantaged neighborhoods or violence. An individual's genes may not have a profound effect on antisocial or criminal behavior on their own but may increase susceptibility or sensitivity to certain risk environments as part of a GxE (Belsky & Pluess, 2009; Scarr & McCartney, 1983). In 2002, Caspi and colleagues conducted a landmark study to identify the mediating factors in the development of antisocial behavior among maltreated children and uncover why some children would develop antisocial behavior following this experience and others would not. The authors found that the effects of maltreatment on the development of antisocial behavior were moderated by polymorphisms of the MAOA gene, which metabolizes neurotransmitters in order to be reused in neurotransmission. Low MAOA expression (i.e., inefficient MAOA) resulted in higher risk of antisocial behavior when childhood maltreatment was also present, beyond the risk of either condition (either genotype or environment) alone. In summary, the study concluded that childhood maltreatment results in a greater risk of antisocial behavior based on the expression of the MAOA gene as part of a GxE. This was the first study to identify a measured gene that interacted with a risk environment to explain behavior, and research has replicated the finding.

Findings from a host of replications (e.g., Ducci et al., 2008; Fergusson et al., 2012; Foley et al., 2004; Nilsson et al., 2006; Wakschlag et al., 2010) and two meta-analyses (Byrd & Manuk, 2014; Kim-Cohen et al., 2006) support the GxE uncovered by Caspi and his colleagues (2002). Successful replications have examined a range of adverse behavioral and clinical outcomes, including conduct disorder (Caspi et al., 2002; Cicchetti et al., 2012; Foley et al., 2004; Wakschlag et al., 2010; Widom & Brzustowicz, 2006), antisocial personality disorder

(Cicchetti et al., 2012; Ducci et al., 2008; Widom & Brzustowicz, 2006), delinquency and criminal behavior (Åslund et al., 2011; Beaver et al., 2013; Caspi et al., 2002; Fergusson, Boden, Horwood, Miller, & Kennedy, 2012; Nilsson et al., 2006; Weder et al., 2009), and various measures of aggression (Cicchetti et al., 2012; Frazetto et al., 2007; Hart & Marmorstein, 2009; Weder et al., 2009). It is possible that other genes are interacting with the environment to produce yet-undiscovered effects on behavior. One of these such areas is within the dopaminergic system, which has implications for how the brain rewards actions or feelings and encourages individuals to repeat activities it finds desirable.

### Dopamine

The dopaminergic system maintains the levels of dopamine in the brain through the processing and removal of proteins in the synaptic cleft of the brain using several dopaminergic genes (Colzato, Zmigrod & Hommel, 2013; Hollerman & Schultz, 1998). Dopaminergic polymorphisms have been linked to increased developmental disorders, antisocial personalities, and substance use (Qadeer, Amar, Mann & Hasnain, 2017; Watts & McNulty, 2015). The dopaminergic system is responsible for an individual's sensitivity to rewards, meaning they may be influenced differently by the environment based on their genetic threshold for experiencing pleasure (Watts & McNulty, 2014). Dopamine enforces learning through increased or decreased rates of cyclic bursts of proteins in the brain, depending on the prediction of rewards or punishments associated with the stimuli (Hollerman & Schultz, 1998). If a behavior produces a reward, the dopamine levels in the brain increase. Dopamine is especially effective when the reward was not predicted, this results in increased firing of dopamine bursts in the synaptic cleft, but if the behavior results in a negative outcome, the dopamine bursts pause, causing reduced levels (Hollerman & Schultz, 1998). The increase or decrease of dopamine levels resulting from

behavioral outcomes then acts as a predictor of reward in the brain, meaning behaviors that increase dopamine levels will be associated with rewards, and behaviors that cause dopamine to decrease or remain inactive results in avoidance of similar actions (Hollerman & Schultz, 1998).

Dopamine neurons are triggered based on event predictability and attach expectations of reward or absence of reward to environmental stimuli (Schultz, 1998). If the reward is greater than predicted, the dopamine neurons are triggered; if the reward is as expected, the neurons stay constant; and if the reward is less than expected, the neurons are depressed (Schultz, 1998). Rewards or lack thereof resulting from behavior acts as a reinforcement for subsequent actions since dopamine attaches on appetitive value to the stimuli experienced. Depending on the result of the behavior, dopamine neurons will increase or decrease activation, influencing future behavior to achieve rewards and higher neuron activation through reinforcement learning (Schultz, 1998). When the dopamine levels in the brain drop, risky or dangerous behavior may be used to increase these levels as a method of compensation (Hollerman & Schultz, 1998; Eisenegger et al., 2013). Increased risk-taking behavior, instigated by environmental stressors, is influenced by the polymorphisms of the dopamine transporter gene DAT1, which is a key moderator of the dopaminergic system (Watts & McNulty, 2014).

Genes such as DAT1 do not act as determinants of criminal propensity; instead they affect the susceptibility of an individual to the influences of their environment and determine how future behavior is developed as a result of these influences (Nilsson et al., 2015). Because genes regulate the individualized formation of behaviors, more sensitive genes confer a higher risk of antisocial behavior when exposed to unfavorable environments. Some genes make individuals more likely to succumb to negative influences, these influences create antisocial behavior. Genes determine how reactions to similar environments will differ between

individuals; and if the genes predispose the individual to higher sensitivity, they are less able to resist negative influences of bad environments. The dopamine (DAT1) transporter gene codes for a protein that is responsible for the reuptake of dopamine in the brain. When the protein's levels are irregular, it can alter risk taking and pleasure-seeking behaviors as a result of a chemical imbalance in the brain (Colzato et al., 2013; Mata, Hau, Papassotiropoulos & Hertwig, 2012). This imbalance can result in dangerous behavior, depressed emotional states, and increased risk taking.

### DAT1 Transporter Gene

Dopamine is stored in the synaptic cleft of the brain before use and is moved to and from the synaptic cleft by the DAT1 gene, which acts as a transporter when dopamine is released and then removed (Eisenegger et al., 2013). The amount of striatal dopamine available in the synaptic cleft of the brain is determined by the amount of DAT1 transporter proteins that are available for the reuptake of dopamine after its release (Eisenegger et al., 2013). Dopamine levels in the brain are dependent on the amount of DAT1 that is available to transport it to the portion of the brain where it is activated and broken down. The efficiency of the different DAT1 genotypes to recirculate dopamine helps to determine the extent to which certain stimuli will create increases or decreases of the dopamine levels in the brain (Colzato et al., 2013). Polymorphisms in dopamine genes between individuals can therefore account for varying dopamine levels and the degree of environmental influence on behavior. DAT1 has a variety of polymorphic genes that can range from tandem repeats with 3 to 13 copies of the nucleotide, down to the smallest and most numerous type of polymorphisms which consist of variation of a single nucleotide (Chen, Lin, Chiang, Su & Wang, 2014; Cornish, et al., 2005; Haefel et al.,



2008; Qadeer et al., 2017; Vandenberg et al., 1992). Variation of the dopamine genotype can alter gene expression and drive pleasure seeking behavior.

Polymorphisms in the DAT1 transporter gene can alter the genes functionality. This can lead to certain genes increasing or decreasing the likelihood for antisocial behavior in an individual and subsequently being considered high-risk genes (Haeffel et al., 2008; Watts & McNulty, 2015). Certain polymorphisms of the DAT1 gene are more or less efficient at removing dopamine carrier proteins from the brain, resulting in differing levels of endogenous striatal dopamine depending on the gene (Eisenegger et al., 2013). Decreased dopamine in the brain, due to more efficient DAT1 genotypes or environmental lack of stimuli, may result in increased antisocial or delinquent behavior. The neurotransmission of dopamine signaling in the brain is more quickly terminated with increased DAT1 gene expression.

Polymorphisms of the DAT1 gene can be determined by the difference of a single nucleotide or a repeating pattern of nucleotides. The most numerous type of genetic variations found among individuals are the single nucleotide polymorphisms (SNPs) (Haeffel et al., 2008). SNPs identify differences of individual nucleotides within a gene, in the DAT1 gene the nucleotides can be either T (thymine), or C (cytosine) (Haeffel et al., 2008). SNPs within the DAT1 gene can affect the functionality of the gene by altering its processes and efficiency (Haeffel et al., 2008). Behavioral problems have been linked to certain SNPs of the DAT1 gene (Haeffel et al., 2008; Zhou et al., 2008). In a 2008 study done by Haeffel and colleagues, maternal rejection was found to significantly increase the likelihood of children suffering from depression when they possessed the TT genotype, compared to the CC or CT varieties. The study concluded that the SNP (40184) of the DAT1 gene acted as part of a GxE with maternal rejection, increasing depression significantly more among children exposed to maternal rejection

when they possessed high risk genotypes. Another study found that the SNP (40184) of the DAT1 gene was also significantly associated with attention deficit hyperactivity disorder (ADHD) and conduct disorder (CD) among juveniles (Zhou et al., 2008).

Different alleles of DAT1 have been linked to varying rates of developmental and behavioral issues (Guo, Roettger & Shih, 2007). When DAT1 has a lower gene expression, individuals engage in reduced risk-taking behavior as a way to pursue dopamine balance in the brain (Cornish et al., 2005; Hadi et al., 2015; Watts & McNulty, 2014). The increased efficiency of certain polymorphisms at removing dopamine from the brain is in part due to the difference of the binding site density of different genotypes. High rates of binding site density result in excessive removal of dopamine by the DAT1 transporter, which results in insufficient dopamine levels for developmental learning (VanNess et al., 2005; Watts & McNulty, 2014). Increased density at the DAT1 binding site makes it easier for the dopamine transporter proteins to attach and remove dopamine from the synaptic cleft back into the surrounding areas (VanNess et al., 2005). This effectively ends the reward signaling in the brain, so the individual loses feelings of pleasure more rapidly with increased binding site density of DAT1. Some DAT1 alleles will have lower binding site density and be able to cycle dopamine from the synaptic cleft at a more stabilized rate, resulting in lower risk polymorphisms when the alleles are present (VanNess et al., 2005; Watts & McNulty, 2014). The increased biological effectiveness of some allelic variations at dopamine cycling makes them an at-risk gene for increased behavioral problems.

Some polymorphisms pose potential problems for increased criminality due to their ability to clear dopamine from the synaptic cleft at an expedited rate, which can cause individuals to pursue legal or illegal sources of pleasure to increase dopamine levels (Watts & McNulty, 2014). For example, individuals who carry the 10R polymorphism of the DAT1 VNTR

gene report almost twice the levels of violent and serious delinquent behavior as individuals who carry only 9R polymorphisms (Guo, Roettger & Shih, 2007), and individuals who carry the TT polymorphism of the DAT1 SNP (40184) reported higher levels of ADHD and depression (Haeffel et al., 2008; Zhou et al., 2008). Increased behavioral issues including substance use, antisocial behavior, and psychiatric disorders and developmental issues like ADHD and autism have all been associated with DAT1 polymorphisms of the dopaminergic genes (Qadeer et al., 2017; Tong et al., 2015; Cornish et al., 2005).

The relationship between the dopamine transporter gene DAT1 and criminality has been examined at length, with findings indicating a clear relationship between the different DAT1 genotypes and behavioral problems (Guo et al., 2007; Vaughn, DeLisi, Beaver & Wright, 2009; Waldman et al., 1998). For example, Waldman and colleagues (1998) used four different analytical methods to test the relationship between DAT1 and ADHD in children. The study identified the 480-bp (10R) allele as being the high-risk allele and found that the symptoms of ADHD were significantly higher in children as the number of risk alleles increased, showing a relationship between DAT1 and ADHD symptoms. This study replicated previous findings that posit a relationship between high-risk DAT1 polymorphisms and increased ADHD symptoms. The individual DAT1 polymorphisms have also been examined as potential risk factors for antisocial behavior. In 2007, Guo, Roettger and Shih performed a study testing the trajectories of serious and violent delinquency. They found that carriers of the 10R polymorphism of DAT1 had a trajectory twice as high for serious delinquency as those who possessed a 9R genotype. Their research found similar results for violent delinquency, with the trajectory of 10R polymorphisms again being twice as high. DAT1 polymorphisms have also been identified as a significant predictor of chronic criminal behavior, with empirical evidence supporting a possible

relationship of DAT1 and criminal behavior (Vaughn et al., 2009). This relationship was also found to be influenced by the delinquent peer social environment the individual was exposed to (Vaughn et al., 2009). These findings indicate a significant GxE between DAT1 and delinquent peers on chronic criminality.

The current study of genes' effect on learning and behavior is somewhat inconclusive but shows support for potential relationships between specific risk genes and increases in antisocial behavior. Learning is a process that is initiated by exposure to certain environments, and the way these environments are perceived and experienced is mediated by genes (Schultz, 1998). The question of the current study is the degree to which the environment and genes independently determine behavior, and how this impact changes when their relationship is considered in a GxE. Parental behavior is the primary environmental influence children are exposed to and plays a significant role in the development of early behavior. Rafferty, Griffin and Lodise's (2011) longitudinal analysis found that increased positive parenting practices such as low family conflict, more family resources, and supportive parenting increased cognitive gain of children from infancy till age three, when the study ceased.

Recent research has examined parenting influence on behavior, but also how different genes may mediate the effects these parenting practices have on behavioral development. Hiemstra, Engels, Barker, Schayck and Otten, (2013) tested how genes of the dopaminergic system and smoking-specific parenting practices, such as parental rules of smoking, frequency of communication, and quality of communication, moderate smoking onset of adolescents. This study found that only quality of communication had a direct effect on the onset of adolescent smoking, with genes and frequency of communication having no direct effect. Some studies have found parental influence to be moderated by specific gene polymorphisms of the child. Paternal

alcohol use has been shown to predict serious alcohol problems among males, when certain high-risk polymorphisms of the DAT1 gene are also present in the child (Vaske, Beaver, Wright, Boisvert & Schnupp, 2009). There was no relation to alcohol problems between the high-risk allele and males with non-alcoholic fathers, or between males with alcoholic fathers who possess the low-risk allele. This relationship shows a specific interaction between a parental influence and a specific risk gene on the development of behavior. Recent studies remain inconclusive but show support for the interactive effects of genes and parental behaviors on the learning of behavior among juveniles. This research will expand on previous studies by testing the relationship between variables of negative parental influences and the mediating effects of the DAT1 polymorphisms of the child.

## CHAPTER III METHODOLOGY

### Sample

This research used data collected by the Fragile Families and Child Wellbeing Study (FFCWS) conducted by Princeton University. The FFCWS is a longitudinal birth cohort data collection of about 5,000 children and their parents, approximately three-fourths of which were unmarried at the time of the child's birth. Being unmarried parents classifies these families as at-risk or fragile. Parents were initially interviewed in the hospital when the child was born, and follow-up interviews were completed when the child reached three, five, and nine years old. The study includes data from both biological parents and other caregivers, and from the children and their teachers at later waves. Characteristics of the parents, including health, deviant behavior, economic status, relationships, and demographics were all recorded. Children were asked similar questions regarding health, development, and relationships. The 9-year wave also included a buccal swab in order to include genetic information from the mother and child. The polymorphism this study will be interested in is the dopamine transporter gene DAT1.

### Measures

*DAT1 genotypes.* During the in-home interview portion of the 9-year follow-up of the FFCWS data, buccal samples were collected from approximately 3,000 participating children for genetic analysis. The FFCWS examined the single nucleotide polymorphism (SNP) of DAT1, which is located on intron 14 (rs40184) of the 3' untranslated (UTR) region of the gene. SNPs of the DAT1 gene (rs40184) have possible homozygous genotypes of T/T and C/C, or a heterozygous genotype of C/T. This study examines the dopamine transporter (DAT1) gene and

how different genotypes may affect the delinquent behavior of the children as part of a GxE relationship. Past research has shown that carriers of the T nucleotide of DAT1 are significantly more susceptible to a number of behavioral and psychological problems when confronted with adverse circumstances (Haefffel et al., 2008; Cummins et al., 2012).

The FFCWS describes their genotyping process as follows:

The SNP rs40184 was genotyped as described by TaqMan® SNP Genotyping Assays Protocol (Applied Biosystems, Foster City, CA). TaqMan® PCR (denaturation: 95°C, 10 m, annealing: 95°C, 15 s; extension: 60°C, 60 s; for 40 cycles) was performed on genomic DNA from working aliquots using TaqMan® Genotyping Master Mix and SNP Genotyping Assays from Applied Biosystems (Foster City, CA), assay number or C\_\_2960969\_10. Data were analyzed using Sequence Detection Systems v.2.3 for 7900HT RT-PCR Machine by Applied Biosystems. (Fragile Families, 1998, p. 5)

DAT1 genotypes in the child were coded based on the combination of the paternal and maternal repeat alleles, and given a value based on the level of risk for criminal behavior associated with each pair: CC = 1 (low risk), CT = 2 (medium risk), and TT = 3 (high risk).

*Parental alcohol use.* One item from the mother survey at the 3, 5 and 9-year waves was used to determine the use of alcohol in the last 12 months (see Appendix A). The item asks how many days in the past 12 months 4 or more drinks had been consumed in a single day. This item measures the mothers' drinking frequency and behavior in the last 12 months prior to the survey. The FFCWS defines a drink of alcohol as a bottle of beer, a wine cooler, a glass of wine, a shot of liquor, or a mixed drink. Respondents' answers for the item at each wave will be given a score of either 1 = less than once a month, 2 = about once a month, 3 = a few times a month, 4 = a few times a week, or 5 = every day or almost every day. The total score for the 3, 5 and 9-year waves

will be summed to determine each respondents' final measure for alcohol use longitudinally throughout the study. This value will range from 3 (low alcohol use) to 15 (high alcohol use).

*Parental drug use.* Frequency of the biological mothers' drug use was measured at the 3, 5, and 9-year waves of the FFCWS data. One item was used to determine the frequency of illicit drug use within the last 12 months. Respondents were asked to identify how often in the last 12 months they had used one or more illicit drugs. The frequency one or more of these drugs was used will be converted to a scale measuring the average use at each wave where: 1 = None, 2 = Less than once a month, 3 = About once a month, 4 = A few times a month, 5 = A few times a week, 6 = Every day or almost every day. The scores at each wave will then be added to create a final measure of the frequency of drug use over the duration of the FFCWS, ranging on a scale of 3 (little/no drug use) to 18 (high drug use).

*Mother-child relationship.* Both the child and primary caregiver (PCG) were asked questions about the closeness and quality of their relationship with each other at the 9-year wave. PCGs were asked three questions regarding how well they view themselves as a parent and how close they feel to the child. Data were collected from children during the 9-year wave, in which the child reported on four items regarding their relationship with their mother. Although data were collected from any PCG, whether or not they were related, due to the current study's focus on genetics, only data from PCGs identified as the child's biological mother were used.

*Maternal Attachment.* Two variables were created to capture the mother-child relationship quality. The first measured the mother's evaluation of her relationship with her child. Three items were collected during the 9-year wave which asked mothers to rate the quality of their relationship with their child (see Appendix A). Responses were taken on a scale of 1 to 4, with higher scores reflecting less favorable relationships. The three-item scale was confirmed



through the use of factor analysis (Cronbach's Alpha = .579). The three items were summed together to create a final measure of at-risk mother-child relationships.

*Child Attachment.* The second variable measured the child's evaluation of their relationship with their mother. During the 9-year wave, the child responded to six items about their relationship with their biological mother. Factor analysis was used to confirm the use of these six items for a scale of child attachment to mother (Cronbach's Alpha = .547). Children reported on how close they felt to their mother and how well they share ideas (see Appendix A). Items were scored 1 to 4, such that higher scores reflected less favorable relationships. The six items were summed together to create a final measure of at-risk child-mother relationships.

*Parental punishment of child.* Parental punishment of the child was measured using the primary caregiver survey conducted at the 3, 5 and 9-year waves. This survey includes a discipline scale which includes 14 items at each wave that inquire about methods and frequency of punishment (see Appendix A). Factor analysis was utilized to confirm the use of all 14 items in the scale (Cronbach's alpha = .903). Each item represented frequency of a punishment, such that 1 = this has never happened, 2 = it happened, but not in the past year, 3 = once in the past year, 4 = twice, 5 = 3-5 times, 6 = 6-10 times, 7 = 11-20 times, and 8 = more than 20 times in the past year. Frequencies at each wave were determined, and then punishment was summed across all waves to create a final measure ranging from 14 (no punishment of child), to 112 (high/extreme punishment of child).

*Child behavioral problems/delinquency.* Child behavior problems were determined through the use of the child self-report survey included in the 9-year wave of data. Children were asked a series of 17 items which prompted the child to report having engaged in various types of delinquent behaviors, such as smoking marijuana, stealing something, or running away from

home (see Appendix A). Items asked children to respond either “yes” or “no” regarding whether they had engaged in the specific delinquent or antisocial. The responses were coded so that “no” = 0 and “yes” = 1. The final measure of child behavioral problems/delinquency was created by summing the scores of all 17 items, for a possible score ranging from 0 to 17. The responses are recorded so that lower scores equate to less problematic behavior, and higher scores mean the child has a higher prevalence of antisocial behavior problems.

### Current Analysis

The analyses for this study utilized gene x environment interaction (GxE) to test for relationships between DAT1 genotypes of the child and the parental environment and determine if the parental environment will moderate the impact of genetic risk for antisocial behavior in the child. The four parental environments of interest in the current study are alcohol abuse, illicit drug abuse, punishment, and parent-child relationship. A series of analyses were used to test the impact of each environment on child antisocial behavior. These analyses utilized Ordinary Least Squares (OLS) regression to examine both the direct impact of and an interaction between the DAT1 gene and each environmental variable on the child’s antisocial behavior. OLS regressions were used since they are simple analyses that uses explanatory variables to predict values for a another variable of interest, and can be used to determine the strength of the relationships between the variables (Hutcheson & Sofroniou, 1999). For each parental environment, two models were conducted. The first examined direct effects, and the second added an interaction term to represent the GxE. This highlighted relationships between variables that interact to moderate antisocial behavior. The GxE was calculated so that the scores of the genotype risk factor and the parental behavior score are multiplied with higher total values representing greater risk for antisocial behavior (Nikolas, Klump & Burt, 2012).

## Hypotheses

H1: Children who have parents that engage in excessive alcohol consumption are more likely to exhibit antisocial behavior based on their DAT1 genotype.

H2: Children who are raised by parents that use illicit drugs are more likely to exhibit antisocial behavior, based on their DAT1 genotype.

H3: Harsh or severe punishments used by the parent increase the child's risk of antisocial behavior, when paired with certain DAT1 genotypes.

H4: The quality of the parent's relationship with the child, when the relationship is negative or unhealthy, increases the child's risk of antisocial behavior based on their DAT1 genotype.

## CHAPTER IV RESULTS

### Introduction

The following chapter details the results of the current study. Four hypotheses were tested using OLS regression by building two models, in order to examine both direct and interaction effects of a parental behavior and the child's DAT1 gene on the child's delinquency. The results for each hypothesis are presented below. Two models were used for each hypothesis, the first included variables for the parental environment, the DAT1 gene of the child, and the delinquency of the child. The second model included these variables, but also added a variable for the interaction of the parental behavior and the DAT1 gene to explore how this interaction might impact delinquency.

Before presenting the results of these analyses, the demographic characteristics of the sample are described in Table 1. The final analytic sample included 2,880 cases, due to the reduced number of respondents who were successfully genotyped for the SNP rs40184. From the genotyped subsample, 52.4% of the children were male, 47.6% were female, 30.8% of the children's mothers were white, and 69.2% were non-white. At the 9-year wave, age ranged in years from 8.7 to 11.9, with a mean age of 9.3. Descriptive information for the outcome variables can be found in Table 1. Of the genotyped sample, 19.8% carried the high-risk TT genotype, 49.7% carried the medium risk CT genotype, and 30.5% carried the low risk CC genotype. Correlations between all key variables can be found below (see Table 2).

Table 1: Descriptive Information

Variable	Percent	n
Child's Sex		
Male	52.4	2568
Female	47.6	2329
Mother's Race		
White	30.8	1480
Non-white	69.2	3327
DAT1 Genotype		
CC	30.5	879
CT	49.7	1430
TT	19.8	571
	Mean	SD
Age	9.30	.40
Scale Range:	8.67 – 11.92	
Mother's Alcohol Use	6.31	2.80
Scale Range:	3 – 14	
Mother's Drug Use	3.56	1.67
Scale Range:	3 – 18	
Parental Punishment	153.78	35.34
Scale Range:	44 – 262	
Child's Attachment	10.95	3.15
Scale Range:	6 – 24	
Mother's Attachment	4.82	1.47
Scale Range:	3 – 11	
Child Delinquency	1.24	1.77
Scale Range:	0 – 17	

Table 2. Correlation Matrix

		X1	X2	X3	X4	X5	X6	X7	X8	X9	X10
DAT1 Gene	X1	1.00									
Child Delinquency	X2	.013	1.00								
Maternal Alcohol Use	X3	.019	.097	1.00							
Maternal Drug Use	X4	.039	.098**	.348**	1.00						
Punishment of Child	X5	.022	.227**	.144	.159**	1.00					
Child's Attachment to Mother	X6	-.032	.146**	.209	.041*	.046	1.00				
Maternal Attachment to Child	X7	-.021	.023	.430**	.053**	.096**	.154**	1.00			
Child's Age	X8	-.017	-.018	-.145	-.040*	-.082**	-.065**	.043*	1.00		
Child's Gender	X9	-.004	.202**	-.037	.005	.137**	.091**	.012	-.019	1.00	
Maternal Race	X10	.043*	.098**	.410**	-.018	.041	-.023	-.065**	-.004	.002	1.00

\* Significant at the .05 level, two-tailed; \*\* Significant at the .01 level, two-tailed

The first hypothesis proposed that maternal alcohol use and the DAT1 genotype of the child effects the child's delinquency. An OLS regression was used to test this hypothesis (see Table 3). The regression did not find a statistically significant relationship between the DAT1 genotype of the child and the child's delinquency or between maternal alcohol use and the child's delinquency. The second model did not find a statistically significant relationship for the GxE effect of DAT1 and maternal alcohol use on the child's delinquency. The sex of the child was the only control variable that was statistically significant on delinquency, but only in the second model ( $B = .248, p < .05$ ).

Table 3: OLS Regression Examining Mother's Alcohol Use and Child's DAT1 Gene on Childhood Delinquency

	Model 1	Model 2
	b/Beta	b/Beta
DAT1	-.587/-.158	.537/.144
<i>SE</i>	.468	1.169
Alcohol Use	.084/.108	.352/.452
<i>SE</i>	.107	.276
Alcohol Use x DAT1	---	-.169/-.477
<i>SE</i>		.161
<i>Controls</i>		
Age	-.440/-.065	-.339/-.050
<i>SE</i>	.864	.869
Sex	1.061/.240	1.096/.248*
<i>SE</i>	.544	.544
Race	-.712/-.156	-.708/-.155
<i>SE</i>	.640	.639
R <sup>2</sup>	.125	.141
n	65	65

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

The second hypothesis proposed that maternal drug use and the DAT1 genotype of the child effects the child's delinquency. An OLS regression was used to test this hypothesis (see Table 4). The regression did not find a statistically significant relationship between the DAT1 genotype of the child and the child's delinquency. The relationship between maternal drug use and the child's delinquency was statistically significant in the first model ( $B = .094, p < .001$ ) and in the second model ( $B = .151, p < .05$ ). There was not a statistically significant relationship between the GxE effect of DAT1 and maternal drug use on the child's delinquency. The relationship between the sex of the child and the child's delinquency was statistically significant in the first model ( $B = .197, p < .001$ ) and in the second model ( $B = .197, p < .001$ ). The relationship between mother's race and the child's delinquency was statistically significant in the first model ( $B = .099, p < .001$ ), and in the second model ( $B = .099, p < .001$ ).

Table 4: OLS Regression Examining Mother's drug Use and Child's DAT1 Gene on Childhood Delinquency

	Model 1	Model 2
	b/Beta	b/Beta
DAT1	.008/.003	.115/.046
<i>SE</i>	.051	.121
Drug Use	.098/.094***	.157/.151*
<i>SE</i>	.021	.065
Drug Use x DAT1	---	-.030/-.075
<i>SE</i>		.031
<i>Controls</i>		
Age	-.125/-.026	-.126/-.027
<i>SE</i>	.095	.095
Sex	.697/.197***	.696/.197***
<i>SE</i>	.071	.071
Race	.377/.099***	.380/.099***
<i>SE</i>	.077	.077
R <sup>2</sup>	.058	.059
n	2346	2346

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

The third hypothesis proposed that parental punishment and the DAT1 genotype of the child effects the child's delinquency. An OLS regression was used to test this hypothesis (see Table 5). The regression did not find a statistically significant relationship between the DAT1 genotype of the child and the child's delinquency. The relationship between parental punishment and the child's delinquency was statistically significant in the first model ( $B = .188, p < .001$ ) and in the second model ( $B = .294, p < .001$ ). There was not a statistically significant relationship between the GxE effect of DAT1 and parental punishment on the child's delinquency. The relationship between the sex of the child and the child's delinquency was statistically significant in the first model ( $B = .199, p < .001$ ) and in the second model ( $B = .199, p < .001$ ). The relationship between mother's race and the child's delinquency was statistically significant in the first model ( $B = .101, p < .001$ ), and in the second model ( $B = .100, p < .001$ ).



Table 5: OLS Regression Examining Punishment of Child and Child's DAT1 Gene on Childhood Delinquency

	Model 1	Model 2
	b/Beta	b/Beta
DAT1	-.022/-.009	.417/.162
<i>SE</i>	.066	.287
Punishment	.010/.188***	.015/.294***
<i>SE</i>	.001	.004
Punishment x DAT1	---	-.003/-.207
<i>SE</i>		.002
<i>Controls</i>		
Age	.020/.004	.023/.004
<i>SE</i>	.143	.143
Sex	.713/.199***	.713/.199***
<i>SE</i>	.093	.093
Race	.386/.101***	.385/.100***
<i>SE</i>	.098	.098
R <sup>2</sup>	.098	.100
n	1386	1386

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

The final hypothesis proposed that child/mother relationship and the DAT1 genotype of the child effects the child's delinquency. Two sets of OLS regressions were used to test this hypothesis. The first set of models analyzed the quality of the child's relationship with their mother and the child's DAT1 gene on childhood delinquency (see Table 6). The regression did not find a statistically significant relationship between the DAT1 genotype of the child and the child's delinquency. The relationship between the child's lack of attachment to their mother and the child's delinquency was statistically significant in the first model ( $B = .122, p < .001$ ) and in the second model ( $B = .118, p < .05$ ). There was not a statistically significant relationship between the GxE effect of DAT1 and the child's lack of attachment to their mother on the child's delinquency. The relationship between the sex of the child and the child's delinquency was statistically significant in the first model ( $B = .113, p < .001$ ) and in the second model ( $B = .113,$

p < .001). The relationship between mother's race and the child's delinquency was statistically significant in the first model (B = .191, p < .001), and in the second model (B = .191, p < .001).

Table 6: OLS Regression Examining Child's Lack of Attachment to Mother and Child's DAT1 Gene on Childhood Delinquency

	Model 1	Model 2
	b/Beta	b/Beta
DAT1	.035/.014	.019/.008
<i>SE</i>	.047	.170
Child Attachment	.069/.122***	.066/.118*
<i>SE</i>	.011	.030
Child Attachment x DAT1	---	.001/.008
<i>SE</i>		.015
<i>Controls</i>		
Age	-.016/-.004	-.016/-.004
<i>SE</i>	.086	.086
Sex	.673/.191***	.673/.191***
<i>SE</i>	.067	.067
Race	.430/.113***	.430/.113***
<i>SE</i>	.072	.072
R <sup>2</sup>	.069	.069
n	2608	2608

\* p < .05; \*\* p < .01; \*\*\* p < .001

The second set of models analyzed lack of maternal attachment to the child and the child's DAT1 gene on childhood delinquency (see Table 7). The regression did not find a statistically significant relationship between the DAT1 genotype of the child and the child's delinquency. The relationship between lack of maternal attachment to the child and the child's delinquency was not statistically significant in either model. There was not a statistically significant relationship between the GxE effect of DAT1 and the mother's attachment to the child on the child's delinquency. The relationship between the sex of the child and the child's delinquency was statistically significant in the first model (B = .203, p < .001) and in the second model (B = .203, p < .001). The relationship between mother's race and the child's delinquency

was statistically significant in the first model ( $B = .109, p < .001$ ), and in the second model ( $B = .110, p < .001$ ).

Table 7: OLS Regression Examining Lack of Maternal Attachment to Child and Child's DAT1 Gene on Childhood Delinquency

	Model 1	Model 2
	b/Beta	b/Beta
DAT1	.036/.014	-.032/-.013
<i>SE</i>	.049	.167
Maternal Attachment	.024/.020	-.003/-.002
<i>SE</i>	.023	.066
Maternal Attachment x DAT1	---	.014/.036
<i>SE</i>		.033
<i>Controls</i>		
Age	-.100/-.022	-.101/-.023
<i>SE</i>	.086	.086
Sex	.718/.203***	.718/.203***
<i>SE</i>	.068	.068
Race	.418/.109***	.420/.110***
<i>SE</i>	.074	.074
R <sup>2</sup>	.055	.055
n	2539	2539

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$

## CHAPTER V DISCUSSION

The current study's aim was to broaden the understanding of how genes and learning interact to develop behavior. The goal of this study was to identify the impact genes have on behavior and how the interplay between genes and the environment result in learning these behaviors. Genes and environment both contribute to the learning and formation of behavior, but do not always act independently since they can also impact the effects of each other.

Determining the impact and interplay of these influences can expand the understanding of how behaviors are developed and how learning is affected by variation of individual's genes and environments. If the impact of genes on antisocial behavior can be better understood, the formation of these behaviors may be more easily prevented by modifying other influences in accordance with the risk of the individual's genes.

OLS regressions were used to test the effects of genes and parental behaviors on delinquency in children. The interaction between the genes and the parental behavior were also analyzed as a GxE to test for possible combinations of gene and parental influences that may lead to increased antisocial behavior. The results of the regressions showed no statistical evidence of direct effects of the DAT1 gene on the behavior of the child. There was also no statistical evidence supporting the effects of a GxE of DAT1 and parental behavior on delinquency. The relationship between the child's genes and delinquency was not statistically significant for any of the four hypotheses tested. The lack of significance of the DAT1 gene may indicate that the influence of parental behavior outweighs genetic impact of the DAT1 gene in the formation and learning of delinquency. The contribution of parental behaviors as an environmental influence could be so significant that it determines delinquency regardless of the

genetic factors. This does not necessarily mean that DAT1 has no impact on behavior, but it does indicate that environmental forces exert a greater influence on learning.

Although certain genes may contribute to the development of behavior, the results of this study did not find a significant impact of the DAT1 gene on delinquency in early childhood. The current study also shows no supporting evidence for the effects of DAT1 with negative parental behaviors, as part of a GxE, on delinquency. The indication of the study is that the DAT1 SNP rs40184 is not a likely candidate gene for predicting delinquency in children, and that it does not interact with the maternal parenting of the child to develop behavior. Although it is possible that certain GxEs exist, and that genes impact the process of learning, the current study shows support for greater importance of environmental influences from maternal parenting. An individual's predisposed genotype may put them at a greater risk of antisocial behavior, but environmental influences such as maternal parenting could supersede the effects of the gene and determine delinquency regardless of genetic risk.

Overall the findings did offer support for social learning theory, which proposes individuals learn behavior through a series of processes and interactions as part of an intimate group or relationship (Akers, 2011). Some of the negative maternal behaviors examined in the study were significantly related to child delinquency. Mother's drug use, child's quality of relationship with mother, and punishment of child were all significantly correlated to child delinquency at the  $p < .001$  level. These results corroborate the assumptions of social learning theory, specifically the notions that behavior is learned at an early age primarily from parents, and that the behavior of parents influences child behavior (Akers, 2011). Social learning theory posits that if the child is exposed to definitions that favor drug use or other types of crime, by their mother or other intimate groups, they are more likely to adopt these definitions and engage

in these activities as the definitions favorable to crime outnumber those that are not (Akers, 2011). This study indicates support for the notion that exposure to definitions favorable to crime increases the risk of delinquency for the child. The quality of the child's relationship with their mother was also predictive of delinquency, with lower quality resulting in higher rates of delinquency among children.

The indication of these findings, coupled with the fact that higher degrees of punishment were related to child delinquency, is that the influence of the child's social environment, such as relationships, is predictive of childhood delinquency. Although there was no support found for the interaction of genes on behavior, social learning from parental environments does appear to be a predictor of delinquency and behavior. The relationship between parental behavior and delinquency can be explained by using social learning theory. Social learning theory argues that delinquency is learned like any other behavior, it is created and maintained as a result of favorable definitions to crime, adoption of criminal values, reinforcement of criminal actions, and imitation of behavior (Akers, 2011). The results corroborate social learning theory's explanation of the causes of delinquency. Parental behavior directly influences children and transfers values and definitions through observations and reinforcement. This study shows evidence of parental behavior's direct impact on childhood delinquency, and what type of behaviors may put the child at greater risk for delinquency. This means that parental influences determine the child's learning environment and are predictive of delinquency. By modifying parental behavior, such as harsh punishments and drug use, the risk for the child's delinquency can be decreased.

The control variable for the mother's race was significant in each test except for the regression examining maternal alcohol use and the DAT1 gene of the child. The results indicated

that children with non-white mothers have a higher rate of delinquency than children with white mothers. According to the FBI's uniform crime report (UCR), juveniles arrested in 2016 were predominantly white (62.1%), with African American juveniles accounting for almost half as many arrests (34.7%) (Federal Bureau of Investigation, 2016). The disparity between the UCR findings and the findings of the current study may be due to the fragile family status and overrepresentation of minority families in the data set. The sex of the child was also significant in every test except the regression examining alcohol use and the DAT1 gene but was significant in the model where the GxE variable was added. The results showed a significant relationship between the sex of the child and increased delinquency with males having much higher rates and being at greater risk. This result is consistent with the FBI's 2015 UCR data which showed males accounted for 73.1% of arrests compared to females only comprised the other 26.9% (Federal Bureau of Investigation, 2016).

The current study was subject to a variety of limitations due to both the sample population and the design of the analytical models. The sample used in this study was originally gathered by the FFCWS and specifically focused on fragile families. The sample was disproportionately composed of unwed or single parents, since they were the focus of the original study. Minorities were also overrepresented in the data, accounting for over two thirds of the total sample size. Many of the participants are also likely to be of low socio-economic status, since the data is skewed toward urban single parent families. This disproportionate representation of single parent households and minorities in the sample limits the generalizability of the final results. The sample demographics are not proportional to the general population, and this creates limitations of generalizability when making inferences about the population based on the study. Although the FFCWS provided a large sample size and longitudinal data, it was not

originally intended as part of a criminological study, and answers regarding delinquency and behavior are given by each respondent without validation from the researchers. This means that respondents could falsify answers and retain information about delinquency or criminal conduct, decreasing the accuracy of the study.

Some limitations were also due to the design and methods of the current study. This research examined only one gene of the dopaminergic system, this provides a very narrow insight into the effects of genes on behavior in general. The research was also done examining only maternal influences and behaviors. By analyzing only variables of the mother's behavior the results do not reflect the entire family environment the child may be exposed to. It is possible the father's behavior is the primary influence on the child, and that this behavior is learned and adopted independently of the mother's. The use of only one parent's behavior and one gene restricts the possible variables and analyses of the study and could lead to potential relationships being undetected. Although three waves of longitudinal data were used in the current study, the data were only available up to nine years after the child was born. This means the children of the sample were still relatively young during each wave of data and may not have fully developed their behavior. Delinquency tends to peak once the child reaches their teenage years (Moffitt, 1993); because these children were still several years away from that, they may not have begun to exhibit signs of delinquency that may manifest later. Some of the variables may indeed effect delinquency, but they might simply react over a longer period of time; at this juncture, delinquent values may not yet have been transmitted. Future research should attempt to use data that includes the primary years of delinquency, and test for longitudinal effects of environmental factors on delinquency, to see if the effects of some forces are delayed and result in delinquency later in life.



Despite these limitations, the study does contribute to the current research by expanding understanding of how genes affect behavior and delinquency. The study showed that the DAT1 gene examined was not a predictor of delinquency and did not significantly interact with parental behavior. Certain parental behaviors were found to predict delinquency independently from the genes of the child. The findings showed a relationship between negative parental behaviors and child delinquency, adding to the body of research that examines parenting's effect on the development of behavior. These results indicate that parenting behaviors and actions do have an effect on the child's risk of delinquency and reducing these negative actions may subsequently reduce this risk. Parents have a direct effect on the development of the child's behavior, and this research shows how this effect can produce delinquency. Future research should attempt to use a more nationally representative sample for analysis so that results may be more accurately generalized. Including a broader range of genes for GxE analyses would increase the understanding of how these interactions help to develop and predict behavior. If more genes are included in future studies, a possible relationship between one or more of these genes and delinquency may be identified. Examining potential genetic contributors to learning creates an interesting analysis of the relationship between natural biology and learning theory. Utilizing both physical and social sciences to formulate an explanation of learning and behavior may help to answer several questions that remain inconclusive when explained by learning theories alone.

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APPENDIX A  
FFCWS Items Comprising Study Variables

Maternal Attachment to Child

1. Would you say you are...
  - An excellent {caregiver/parent}
  - A very good {caregiver/parent}
  - A good {caregiver/parent}
  - Not a very good {caregiver/parent}
2. How close do you feel to {CHILD}? Would you say...
  - Extremely close
  - Quite close
  - Fairly close
  - Not very close
3. How well do you and {CHILD} share ideas or talk about things that really matter?  
Would you say...
  - Extremely well
  - Quite well
  - Fairly well
  - Not very well

Child's Attachment to Mother

1. Does your mom talk over important decisions with you? Would you say that this statement is never true, sometimes true, often true, or always true?
2. Does your mom listen to your side of an argument?
3. Does your mom spend enough time with you?
4. Does your mom miss events or activities that are important to you?
5. How close do you feel to your mom? Would you say...
  - Extremely close
  - Quite close
  - Fairly close
  - Not very close?
6. How well do you and your mom share ideas or talk about things that really matter?
  - Extremely well
  - Quite well
  - Fairly well
  - Not very well

Parental Punishment

How many times in the past year did you (READ ITEM)? Was it once in the past year, twice, 3-5 times, 6-10 times, 11-20 times, more than 20 times in the past year, it happened but not in the past year, or has this never happened?

1. Explain to (CHILD) why something (he/she) did was wrong
2. Put (CHILD) in "time out" (or sent (CHILD) to (his/her) room)
3. Shook (CHILD)

4. Hit (him/her) on the bottom with something like a belt, hairbrush, a stick or some other hard object
5. Gave (him/her) something else to do instead of what (he/she) was doing
6. Shouted, yelled, or screamed at (CHILD)
7. Spanked (him/her) on the bottom with your bare hand
8. Swore or cursed at (him/her)
9. Said you would send (him/her) away or would kick (him/her) out of the house
10. Threatened to spank or hit (him/her) but did not actually do it
11. Slapped (him/her) on the hand, arm, or leg
12. Took away privileges from (him/her)
13. Pinched (him/her)
14. Called (him/her) dumb or lazy or someother name like that

### Child Delinquency

The next questions are about things you might have done either at school or somewhere else. For each activity read, please tell me "Yes" or "No". Have you ever...

1. Purposely damaged or destroyed property that wasn't yours?
2. Taken or stolen something that didn't belong to you from another person or from a store?
3. Taken some money at home that did not belong to you, like from your mothers' purse or from your parents' dresser?
4. Cheated on a school test?
5. Had a fist fight with another person?
6. Hurt an animal on purpose?
7. Gone into somebody's garden, backyard, house or garage when you were not supposed to be there?
8. Run away from home?
9. Skipped school without an excuse?
10. Secretly taken a sip of wine, beer, or liquor?
11. Smoked marijuana, grass, pot, weed?
12. Smoked a cigarette or used tobacco?
13. Been suspended or expelled from school?
14. Written things or sprayed paint on walls or sidewalks or cars?
15. Purposely set fire to a building, a car, or other property or tried to do so?
16. Avoided paying for things such as movies, bus or subway rides, or food?
17. Thrown rocks or bottles at people or cars?

APPENDIX B  
IRB Approval Form

Exempt Review  
3/26/2018  
Protocol Number: 1100

Dear Trevor Gonzales:

Your research project, 'The effects of negative parental behavior on children with at risk DAT1 alleles: Using a GxE to predict antisocial behavior in children', was approved by the University of Central Missouri Human Subjects Review Committee on 3/26/2018.

If an adverse event (such as harm to a research participant) occurs during your project, you must IMMEDIATELY stop the research unless stopping the research would cause more harm to the participant. If an adverse event occurs during your project, notify the committee IMMEDIATELY at [researchreview@ucmo.edu](mailto:researchreview@ucmo.edu).

The following will help to guide you. Please refer to this letter often during your project.

- If you wish to make changes to your study, submit an “Amendment” through Blackboard under the “Amendment and Renewals” tab. You may not implement changes to your study without prior approval of the UCM Human Subjects Review Committee.
- If the nature or status of the risks of participating in this research project change, submit an “Amendment” through Blackboard under the “Amendment and Renewals” tab. You may not implement changes to your study without prior approval of the UCM Human Subjects Review Committee.
- When you have completed your collection of data, please submit the “Final Report” found on Blackboard under the “Final/Renewal Report” tab.

If your protocol contained a consent form and the consent form was approved, you will receive an additional e-mail. The e-mail will contain a copy of your consent form with an approval stamp in the top right corner. Do not begin data collection until you receive a copy of your consent form with an approval stamp. Note: One year after your protocol's approval date, a request for renewal OR a final project report is required.

If you have any questions, please feel free to contact me at [researchreview@ucmo.edu](mailto:researchreview@ucmo.edu).

Sincerely,



Kathy Schnakenberg  
Program Administrator/Research Compliance Officer  
Office of Sponsored Programs and Research Integrity  
University of Central Missouri