I, Jamie Newsome, hereby submit this original work as part of the requirements for the degree of Doctor of Philosophy in Criminal Justice.

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ABSTRACT

Criminologists have emphasized the importance accurately predicting which youths will engage in criminal behaviors. The vast majority of empirical investigations have identified a host of individual characteristics and environmental factors that heighten one’s risk for delinquency. Despite continued efforts to improve prediction, outcomes for some individuals are inconsistent with that which is predicted. Some youth appear to be more resilient to the risks they encounter, while others possess a heightened vulnerability. Outcomes among these cases are not well understood. The primary aim of this dissertation is to assess the extent to which genetic and environmental factors contribute to individual variation in response to risk. Based on the differential susceptibility perspective, individuals are hypothesized to vary in their sensitivity to the conditions to which they are exposed. Furthermore, this perspective proposes that differences in sensitivity are the result of both genetic and environmental factors.

Biometrical genetic modeling is employed to investigate the genetic and environmental contributions to differential response to risk for delinquency. Results obtained from a subsample of twins from the National Longitudinal Study of Adolescent Health indicate that both genetic and environmental influences contribute to variation in the response to risk. Additionally, the magnitude of the effects differs between males and females, with additive genetic influences having a stronger influence in males and common environmental influences having a stronger effect in females. The differences between vulnerable youth and the overall population are largely due to genetic factors; however, the observed differences between resilient youths and the population appear to be due to environmental factors. The theoretical and policy implications of these findings are discussed.
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CHAPTER I
INTRODUCTION

For decades, criminological research has attempted to identify the causes and correlates of delinquency. Loeber and Dishion (1983) argue that prediction research is important for two reasons. First, the ability to accurately predict which youth are at the greatest risk for delinquency allows practitioners to intervene early, before the individual has the opportunity to become a chronic offender. Second, Loeber and Dishion (1983) propose that prediction research can be useful in constructing and reformulating criminological theories. As described by Weisburd and Piquero (2008), if constructs are unable to explain criminal behavior as predicted by a particular theory, it may be necessary to falsify or revise the theory or to seek out new explanations. Additionally, Wikström (2008) has discussed the need to advance criminological theory through empirical investigations designed to further specify the relationships between predictors and crime. The need to advance theory and devise appropriate prevention and intervention strategies has led to the identification and study of several risk factors for criminal and antisocial behavior among youth.

The vast majority of previous research on delinquency emphasizes the importance of social and environmental risk factors. Parents are often cited as particularly influential in the development of delinquency in their children (Farrington, 1990; Gottfredson & Hirschi, 1990; Patterson, 1982; Sampson & Laub, 1993), as well as number of family characteristics, such as family size and socioeconomic status (Hirschi, 1969; Sampson & Laub, 1993). Peers are also believed to be a major contributor to adolescent criminal behavior (Akers, 1998; Bandura, 1986; Burgess & Akers, 1966; Hirschi, 1969). It has even been argued that neighborhood
characteristics (Sampson & Groves, 1989; Sampson, Raudenbush, & Earls, 1997; Wilson, 1987) and cultural differences (Anderson, 1999) could put youth at a greater risk for delinquency.

Other studies have examined a variety of personal characteristics that might increase one’s likelihood of becoming delinquent. Intelligence (Garlick, 2002; Hirschi & Hindelang, 1977; Ward & Tittle, 1994), neuropsychological deficits (Moffitt, 1993), school performance (Hirschi, 1969; Sampson & Laub, 1993), self-control (Gottfredson & Hirschi, 1990), temperament (Sampson & Laub, 1993), personality (Eyesenck, 1977; Hare, 1999; Lykken, 1995), and early problem behavior (Caspi & Silva, 1995; Loeber, 1982; Moffitt, 1990, 1993; Olweus, 1979; Patterson, DeBaryshe & Ramsey, 1989; Robins, 1978; Sampson & Laub, 1993; White, Moffitt, Earls, Robins, & Silva, 1990) have all been implicated as potential risk factors.

Although less common, researchers have also investigated the influences of biological factors on behavior. There is now a rapidly growing body of evidence that suggests genetic differences between individuals account for a significant proportion of variation in behavior, including antisocial behaviors (Moffitt, 2005; Rhee & Waldman, 2002). Moreover, a number of traits identified as risk factors for delinquency also appear to be influenced by genetic factors (Baker, Bezdjian, & Raine, 2006). For example, genetic differences between people appear to account for some of the heterogeneity in intelligence (Bouchard & McGue, 1981; Deary, Spinath, & Bates, 2006; Plomin & Petrill, 1997), low self-control (Beaver, Eagle Schutt, Boutwell, Ratchford, Roberts, & Barnes, 2009; Beaver, Ratchford, & Ferguson, 2009; Beaver, Wright, DeLisi, & Vaughn, 2008; Boisvert, Wright, Knopik, & Vaske, 2012; Wright & Beaver, 2005; Wright, Beaver, DeLisi, & Vaughn, 2008), impulsivity (Bezdjian, Baker, & Tuvblad, 2011), externalizing behaviors (Gjone & Stevenson, 1997; Gjone, Stevenson, Sundet, & Eilertsen, 1996; Haberstick, Schmitz, Young, & Hewitt, 2005; Silberg et al., 1994; Schmitz et...
Despite this growing evidence errors in prediction remain (Blumstein, Farrington, & Moitra, 1985; Loeber & Dishion, 1983; Loeber, Dishion, & Patterson, 1984; O’Donnell, Hawkins, & Abbott, 1995). These errors can be divided into two categories: false positives and false negatives. The first category represents cases that are predicted to be delinquent, yet do not engage in criminal acts. False negatives, on the other hand, are individuals that are delinquent even though they are predicted to have limited or no involvement in crime. A portion of these errors can be attributed to characteristics of the sample under examination, measurement error, the omission of key variables in the model, or the statistical techniques employed by the researcher (Farrington, 1985; Farrington & Tarling, 1985; Gottfredson & Tonry, 1987). However, research suggests that some of these cases represent distinct groups, and the errors in prediction should not necessarily be considered random or unimportant (Stein, Vadum, & Srabin, 1970).

Several authors have emphasized the potential value in examining cases whose outcomes deviate from that which is predicted (Marks, 1964; Reckless, Dinitz, & Murray, 1957; Stein et al., 1970; Sullivan, 2011; Weisburd & Piquero, 2008). Marks (1964) previously advocated this approach on the grounds that it could lead to the discovery of causal or moderating variables that, when incorporated into a statistical model, could improve predictive efficacy. In addition to improving prediction, inspection of deviant cases can aid in the advancement of criminological theory. A recent demonstration by Sullivan (2011) reveals the utility of analyzing deviant cases in refining explanations of criminal behavior. Rather than dismissing these cases, he shows that their careful inspection provides an avenue for using theory and data together to further develop
criminological theories. Weisburd & Piquero (2008) also expound on the need for criminologists to consider things that remain unexplained by traditional theories, not only as a means of scientific development, but also in terms of creating a solid foundation on which theoretically driven policies can be conceived.

One area of research that has focused on deviant cases specifically has characterized individuals as resilient or vulnerable (Blum, McNeely, & Nonnemaker, 2002; Garmezy, 1991; Garmezy, Masten, & Tellegen, 1984; Masten, 2001; Masten & Garmezy, 1985; Rutter, 1979, 1985, 1990, 1993; Sameroff & Seifer, 1983; Werner, Bierman, & French, 1971; Werner & Smith, 1977; 1982; 1992). Resilient individuals are those who manage to achieve positive outcomes despite being at-risk. Essentially, these are false positive cases. With regard to criminal behavior among adolescents, individuals that fall into this category may encounter multiple risk factors for delinquency but still manage to avoid unlawful behavior. In contrast, another group of individuals are categorized as vulnerable. Those in this category are false negatives and ultimately exhibit more adverse outcomes than predicted. These vulnerable youth may participate in criminal acts with no or only minimal exposure to the typical risk factors associated with delinquency.

The study of resilience and vulnerability has indicated that the factors predisposing one to either trajectory could be biological, environmental, or both (Blum et al., 2002; Werner & Smith, 1982, 1992). Variables associated with resilience are often referred to as “protective” or “promotive” factors (Farrington & Welsh, 2007). They either reduce an individual’s probability of offending, or eliminate or mitigate the effects of risk factors. Factors associated with vulnerability are those that increase one’s probability of poor adjustment, either through the introduction of a risk factor or by exacerbating the effects of existing adverse conditions (Luthar
& Zelazo, 2003). Such factors can include manifest risk factors (e.g., abuse or neglect) or latent factors, such as genetic predispositions that make one more sensitive than others to the effects of external risk factors.

Like risk factor research, investigations into the origins of resilience and vulnerability tend to focus on individual characteristics and environmental contributions to behavior, which vary depending upon the outcome of interest (Rutter, 1993). In general, the variables associated with resiliency often represent opposite extremes of commonly examined risk factors (Farrington & Welsh, 2007). At the individual level, high cognitive functioning, self-efficacy, self-esteem, positive temperament, strong self-regulation, and a positive outlook have been designated as protective factors (Garmezy, 1985; Masten & Powell, 2003; Stouthamer-Loeber, Loeber, Farrington, Wikstrom, & Wei, 2002; Stouthamer-Loeber et al., 1993; Werner & Smith, 1982, 1992; White, Moffitt, & Silva, 1989). Strong relationships with parents, relatives, mentors, and prosocial peers also appear to promote resiliency among otherwise at-risk youths (Masten & Powell, 2003; Stouthamer-Loeber et al., 2002; Stouthamer-Loeber et al., 1993; Werner & Smith, 1982, 1992). Contextual factors such as quality schools, connections to prosocial organizations, neighborhood safety, enhanced community supervision, and the availability of social services also appear to foster resiliency in at-risk youth (Jarrett, 1997, 1999; Masten & Powell, 2003; Stouthamer-Loeber et al., 2002; Stouthamer-Loeber et al., 1993).

Research on vulnerable cases, however, is scarce. These cases tend to be ignored by researchers and create a considerable dilemma for criminologists. Specifically, if known risk factors are not contributing to delinquency among these cases, this may suggest that there is no theoretical explanation for their unlawful actions within the field of criminology. As argued by Wikström (2008), failing to identify the causes of their illegal behavior renders it impossible to
prevent. This further highlights the importance of the deviant case approach, as this may provide a means of better understanding the origins of both resilience and vulnerability, which can in turn be used to advance criminological theories and improve intervention strategies.

The heart of this issue is that some people vary in their exposure to risk factors and their responses to those risks. Many respond as would be predicted, but those that appear to be resilient or vulnerable do not. One possible explanation for this differential response to risk is that individuals may vary in their sensitivity to environmental conditions (Belsky, 1997, 2005; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009a, 2009b; Boyce & Ellis, 2005; Ellis & Boyce, 2008; Ellis, Essex, & Boyce, 2005; Rutter, 2003). As an example, two children in the same family may both suffer from physical abuse but could display very different developmental outcomes later in life. One may experience depression, use illegal drugs, or become violent. The other child may go on to graduate college, have a successful career, and start a family. According to this view, some people are harder than others and able to thrive in a variety of conditions (Belsky, 1997, 2005; Ellis & Boyce, 2008). Although this has been applied more often in the study of resilience, this perspective could contribute to our understanding of vulnerability as well. For youth that are more sensitive to the effects of their experiences, even minimal exposure to risk factors could produce an antisocial behavioral response.

The differential susceptibility perspective further proposes that the differences in the genetic makeup of these two children may help explain the contrasting outcomes between them (Belsky, 1997). A number of studies have demonstrated that genetic variation between individuals may be partially responsible for these differences in sensitivity (Cadoret, Cain, & Crowe, 1983; Caspi et al., 2002; Jaffee et al., 2005; Kim-Cohen & Gold, 2009; Kim-Cohen et al.,
In light of this evidence, investigating the extent to which genetic and environmental factors account for variation in response to risk may aid in advancing our understanding of the relationship between risk and behavior.

**Statement of the Problem**

The available evidence strongly indicates that differences in the ways that individuals respond to risk factors are influenced by genetics; however, these studies typically examine variation in relation to specific genes. No study to date has estimated the overall extent to which genes and the environment contribute to the variation in responses to risk for delinquency. Therefore, the primary aim of this dissertation will be to determine the influence of these factors on differential responses to risk. This will be accomplished by assessing variation in responses to risk using a continuum in which resilience is found at one extreme and vulnerability at the other. Using behavior genetic modeling techniques, the variance in differential responses will be decomposed into genetic, shared environmental, and unique environmental components.

Examination of crime statistics consistently show that males are involved in more crime, both violent and property offenses, than females (Federal Bureau of Investigation, 2011). It has also been suggested that gender differences may exist in resilience and vulnerability (Emery & O’Leary, 1982; Fergusson & Horwood, 2003; Hetherington, 1989; McGloin & Widom, 2001; Rutter, 1990, 2003; Rutter & Silberg, 2002; Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006). While gender differences in criminal behavior, resilience, and vulnerability between have been observed, the causes of these differences are still under investigation. Zahn-Waxler and colleagues (2006) explain that sex differences could be due to variation in both the types and amount of risk and protective factors each group encounters. It has also been argued that differences may be due to varying biological processes between boys and girls, such as variation
in genetic factors that make one sex more vulnerable than the other (Rutter, 2003; Rutter & Silberg, 2002; Zahn-Waxler et al., 2006).

To further explore the possibility of sex differences in responses to risk for delinquency, this dissertation will address two additional questions. First, are there qualitative differences between males and females in the genetic and environmental influences in differential responses factors for delinquency? This question seeks to find whether the same genetic and environmental factors that are working in one sex are the same as those working in the other. Second, are there quantitative differences between males and females in differential responses to risk factors for delinquency? The available research suggests that the heritability of resilience may differ between males and females (Boardman, Blalock, & Button, 2008; Waaktaar & Torgersen, 2012). This indicates a need to further examine whether there is a significant difference in the magnitude of genetic and environmental effects between the sexes.

Finally, the study of resilience is appealing to researchers because it focuses on the positive aspects of individuals’ lives and provides direction for intervention and prevention efforts (Masten & Powell, 2003). Nonetheless, it is important to examine both resiliency and vulnerability. As previously noted, criminal behavior among low-risk adolescents is not well understood. This group of offenders increases the risk to society and, without an understanding of the origins of their behavior, it is not possible to prevent or appropriately respond to it. In an effort to better understand both resiliency and vulnerability, the final goal of this dissertation will be to estimate the genetic and environmental influences on these responses separately. DeFries-Fulker regression analyses allow for the isolation and examination of extreme cases. Using this statistical technique, the cases at each extreme of the differential response continuum will be
analyzed in order to determine if genetic factors account for differences between the overall population and resilient and vulnerable groups.

**Conclusion**

Predicting delinquent behavior among adolescents has proven to be a difficult endeavor. Although a number of potential risk factors have been examined, predictions are often inaccurate. This has led some researchers to question why some individuals are able to overcome the risks that they encounter and others are seemingly more susceptible to engaging in delinquent behavior despite being low risk. Those researching the contributions to resilience and vulnerability often look to the same factors commonly used in the prediction of delinquency to explain the heterogeneity in responses. It has previously been argued that people may vary in their sensitivity to their environment, and that this variation may be due to genetic differences (Belsky, 1997, 2005; Boyce & Ellis, 2005; Ellis et al., 2005). However, few studies have examined the potential for genetic factors to influence resilience and vulnerability. In an effort to increase our understanding of the origins of these behaviors, this dissertation will seek to answer the following questions:

1. To what extent are genetic and environmental factors contributing to the differential response to risk for delinquency?
2. Are there qualitative differences in genetic and environmental effects between males and females in the differential response to risk?
3. Are there quantitative differences in genetic and environmental effects between the sexes in the differential response to risk?
4. To what extent are genetic and environmental factors influencing resilience?
5. To what extent are genetic and environmental factors influencing vulnerability?
To examine these questions, restricted-use data from the National Longitudinal Study of Adolescent Health (Add Health) will be analyzed using behavior genetic modeling techniques. The Add Health researchers collected data on a nationally representative sample of youth residing in the United States in 1994. Information was gathered on numerous aspects of youths’ lives, providing the opportunity to examine both the criminogenic risks they encountered and their behavioral responses to those risks. Additionally, the Add Health researchers collected data on a subsample of related individuals. Since a measure of genetic relatedness between individuals is required to assess both genetic and environmental influences on behavior, this property of the Add Health data make it an ideal dataset for the current study.

Many scholars have neglected to closely examine those cases in which predicted behavior and observed behavior diverge; however, there may be real value in such an undertaking. Gaining an understanding of why some youth are resilient, and manage to perform better than predicted, could prove to be valuable in developing prevention and intervention strategies. Additionally, identifying the causes of vulnerability, or delinquency among youth that do not appear to be at-risk, can be used to extend and refine existing criminological theories. Given that the vast majority of criminological theories and empirical investigations have failed to consider the possible role of genetic factors, the aim of this study is to explore genetic influences on differential responses to risk for delinquency.
CHAPTER II

RESILIENCE AND VULNERABILITY: DIFFERENCES IN RESPONSE TO RISK

Traditionally, researchers seeking to better understand crime and delinquency have taken a “risk factor approach.” While criminology has discovered numerous correlates of crime, less progress has been made in understanding how these correlates are related to criminal behavior (Farrington, 1988, 1992, 2000, 2002; Farrington & Welsh, 2007; Moffitt & Caspi, 2006; Wikström, 2008). This study takes a contemporary approach to understanding risk and behavior by examining individual differences in the response to risk factors through: (1) the application of a modern theoretical framework capable of explaining delinquent and non-delinquent behavior at varying levels of risk; (2) the identification and close examination of vulnerable and resilient individuals; and (3) the use of statistical techniques that account for biological and environmental influences on behavior (see Chapter 3).

The chapter will begin with an explanation of the cumulative risk model used to assess risk for delinquency. This discussion will be followed by an overview of three existing theories that consider both nature and nurture in human development, and the evidence pertaining to criminal behavior that has emerged under these frameworks. Finally, the chapter will conclude with a review of the literature indicating the importance of investigating possible differences in genetic and environmental influences for males and females.

Assessing Risk for Delinquency

Much of the existing research on risk for delinquency focuses on the relationship between single risk factors and criminal behavior. Some scholars, however, have argued that a more comprehensive assessment of risk is necessary (Greenberg, Speltz, DeKlyen, & Jones, 2001; Loeber, Slot, & Stouthamer-Loeber, 2006, 2008; Mullis, Cornille, Mullis, & Huber, 2004). The
more inclusive strategy for measuring risk is based on an ecological model in which individual development is hypothesized to be under the simultaneous influence of multiple factors (Bronfenbrenner, 1979; Tolan & Guerra, 1994). Although there is some variation across studies, potential risks can generally be categorized into three domains: individual, family, and contextual (Farrington & Welsh, 2007). Individual-level risk factors are one’s traits or characteristics that contribute to his or her likelihood of becoming a delinquent. A second domain of risk factors is found at the family-level. Factors within this category include both the qualities of a family such as family size, and various aspects of family functioning such as parent-child interactions. The final risk domain is contextual risk factors and includes items that are part of the larger social system in which the youth is situated. These more distal risks can be found in peer groups, the school environment, or the community in which one resides.

Factors across each of these domains can co-occur and may be interconnected (Bronfenbrenner, 1979). For example, schools in impoverished communities may lack the resources to provide safe productive learning environments, which may negatively impact the development of children within that school. The deleterious effects of poor community and school environments may further add to risk resulting from individual characteristics, such as impulsivity or low intelligence. As a result, investigations targeting a single risk factor or domain may present an incomplete picture of one’s overall risk.

Rather than examining the individual effects of unique risk factors, some authors have investigated whether the accumulation of risks is predictive of delinquency. The cumulative risk approach involves determining the total number of risk factors to which individuals are exposed (Gerard & Buehler, 2004; Loeber et al., 2006, 2008). This is often accomplished by dichotomizing continuously measured risk factors in such a way that individuals with a score
exceeding a specified threshold value (typically the highest or lowest quartile in a distribution) receive a score of one, indicating a risk is present. Those with a score outside of the range associated with risk receive a score of zero for the given factor. The scores across all dichotomized measures are then added together to generate a total risk index.

An important assumption underlying the use of the cumulative risk method is that a given outcome is influenced by the number of risks, rather than the specific risks, one encounters (Atzaba-Poria, Pike, & Deater-Deckard, 2004; Gerard & Buehler, 1999, 2004). Relatedly, the relationship between cumulative risk and behavioral outcomes is often assumed to be linear, but this assumption has not been adequately tested (Atzaba-Poria, et al., 2004; Gerard & Buehler, 1999, 2004; Greenburg, et al., 2001). It is possible that the relationship may take a quadratic or exponential form. Rather than risk increasing incrementally with each added factor, the probability of a delinquent outcome may rapidly increase with every risk encountered or once an adolescent is exposed to a particular number of risks (Rutter, 1979; Sameroff, Bartko, Baldwin, & Seifer, 1998). Under such circumstances, the total effect of the risk factors is more than simply the sum of all their individual effects. Moreover, this model assumes that each risk factor, or in some cases each risk domain, has an equal impact on development, and that there are no significant interactions between risk factors (Gerard & Buehler, 2004; Greenberg, et al., 2001).

To better understand the complex relationships between risk factors and development, a number of scholars have tested the assumptions of the cumulative risk model. For example, Herrenkohl and associates (2000) examined the cumulative effects of risk factors present during late childhood and early adolescence on violent behavior at age 18. Risk factors from five domains (i.e., individual, family, school, peer, and community) were measured at ages 10, 14,
and 16 years. At each age, individuals that encountered no or only one risk factor were categorized as very low-risk. Those that were exposed to two or three risk factors were considered low-risk. A third group that experienced four or five risks was labeled moderate-risk. Youths that encountered more than five risk factors were considered high-risk. At each age, the odds of engaging in violent behavior at 18 years increased as the number of risks increased regardless of the specific risks one encountered, suggesting risk factors may have additive effects.

Saner and Ellickson (1996) investigated the cumulative effects of risk factors across four domains (i.e., demographic factors, negative life events, prior behavioral problems, and environmental/institutional variables) on three measures of violent behavior during adolescence. As the number of risks an individual encountered in each domain increased, so did the odds of persistently hitting others, predatory or serious violence, or any violent behavior. This pattern was consistent across males and females, and suggests that risks for violent behavior occur across various aspects of one’s life and act in an additive manner.

Cumulative risk has also been found to negatively impact behavior early in life. Deater-Deckard, Dodge, Bates, & Pettit (1998) tested whether a cumulative risk model predicted parent-reported, teacher-reported, or peer-reported externalizing behaviors in childhood. A total of 20 risk variables across 4 domains (i.e., individual, parental, peer, and sociocultural) were used to create an overall cumulative risk score as well as a cumulative risk score for each domain. The results of this study indicated that the number of risk factors a child encountered predicted differences in externalizing behaviors, accounting for 10-16% of the variance. Each of the four domains made a unique and significant contribution to the prediction of externalizing behaviors.
These findings indicate that risk factors across domains have a negative effect on early development, irrespective of the specific risks one encounters.

Atzaba-Poria, Pike, and Deater-Deckard (2004) also found that risk factors acted in a cumulative manner to predict externalizing behavior problems using a sample of 125 English and Indian children (7 to 9.6-years old). Risk factors were categorized into three domains: individual, immediate environment (e.g., relations with peers, siblings, and parents), and those factors that may influence the child indirectly (e.g., parental job spillover). The cumulative risk variable explained 42% of the variance in externalizing behavior ($p < .001$). In a second series of models, the unique effect of each risk domain was assessed (Atzaba-Pori, et al., 2004). All three domains contributed externalizing behavior problems when the other domains were not included in the model; however, only the accumulation of micro-level factors had a unique effect ($\beta = .42, p < .001$) when all domains were included. When each domain was permitted to have a unique rather than cumulative effect, the variance explained by the model was slightly reduced to 37%. Overall, the level of externalizing behavior problems increased as children accumulated more risks, regardless of the specific risks encountered. There were no significant interactions between risk and ethnicity in any of the models, suggesting that the relationships observed between cumulative risk and behavioral outcomes were the same across cultures.

In another study using a cumulative risk model, van der Laan, Blom, & Kleemans (2009) investigated whether situational risk factors relating to the crime, such as risk of being caught or the presence of co-offenders, contributed to serious delinquency when cumulative risk was taken into account. Cumulative risk factors were classified into three domains: individual, family, and school. Factors across each domain and situational risk factors all contributed to delinquency. Furthermore, the accumulation of risks across the three domains significantly increased the
likelihood of serious delinquency (van der Laan, et al., 2009). The authors concluded that both situational and traditional risk factors are important to consider, although the accumulation of risks had a stronger association with serious delinquency than situational factors.

Examinations of cumulative risk factors have also included risks present very early in the life course. For example, Green, Gesten, Greenwald, and Salcedo (2008) examined the ability of birth-related risk factors, early problem behaviors, academic factors, and demographic characteristics to predict delinquency into adolescence. Continuous risk measures were dichotomized and the riskiest 20 percent received a score of 1 and all others a score of 0, indicating no risk. The dichotomized risk factors were summed and the overall cumulative risk scale was then trichotomized into low risk (0 risk factors), medium risk (1-2 risk factors), and high risk (3+ risk factors). A one-way ANOVA revealed significant differences between the groups in the number of delinquency referrals \(F(2, 2075) = 78.89, p = 0.00\). The average number of referrals in the low and medium risk groups was 0.32 and 0.83, respectively. The high risk group had an average number of 2.43 delinquency referrals, nearly eight times more than the low risk group and three times more than the medium risk group.

Ribeaud and Eisner (2010) also examined the cumulative effects of early risk factors on behavior using a sample of youth from Switzerland. Subjects were part of the Zurich Project on the Social Development of Children, which began in 2004 when participants were approximately 7-years old. Additional waves of data collection were conducted in 2005, 2006, and 2008. Four broad domains of risk factors were divided into nine subdomains, and included a total of 47 variables in all. The overall cumulative risk variable was strongly correlated with aggression at age 11 \(r = .52, p < .001, OR = 4.55\), although the effect sizes were significantly stronger among males \(r = .52, p < .001\) than females \(r = .42, p < .001\). The effects of cumulative risk were
stronger than any other single risk factor or the cumulative effects within each of the nine subdomains (Ribeaud & Eisner, 2010). This was true among the entire sample of youth, and when males and females were analyzed separately. It should be noted, however, that the effects of cumulative risks by domain were strongest among individual level factors (.3 < r < .5), moderate among family, peer, and school factors (.2 < r < .3), and weakest among the most distal factors such as sociodemographic variables (r < .1).

Other scholars have also examined the functional form of the relationship between cumulative risk and delinquency. Appleyard, Egeland, van Dulmen, & Sroufe (2005) tested whether a linear or quadratic (i.e., threshold) measure of cumulative risk better explained variation in behavioral outcomes. This study also examined the timing of encountering risks to determine if those experienced early in life (i.e., birth to 64 months) have a different impact than those experienced later (i.e., during grade school years). Five risk factors comprised of multiple items were included in the cumulative risk measure: child maltreatment, inter-parental violence, family disruption, life stress, and socioeconomic status. Early cumulative risk was predictive of externalizing behaviors at 16 years (β = .27, p < .01), even after controlling for the effect of cumulative risk during middle childhood. Results indicated that a linear model was more appropriate than a quadratic model. There was also a significant effect of the accumulation of risks across developmental periods, with children that had more risks overall being significantly more likely to have externalizing problems than children with fewer risks (F (8, 162) = 10.63, p ≤ .001).

Gerard and Buehler (2004) examined the effects of cumulative environmental risk and externalizing behavior problems in adolescence. Cumulative risk was comprised of 14 risk variables that were dichotomized and summed. The functional form of the relationship between
cumulative risk and externalizing behaviors was determined to be linear ($\beta = .36, p < .001$), and on average externalizing behaviors increased .76 for each unit increase in risk (Gerard & Buehler, 2004). The quadratic model was not significant. Additionally, a measure of affected risk domains was created by summing the number of domains in which an individual encountered at least one risk factor in family, peer, school, or neighborhood contexts. Results of an ANCOVA analysis indicated that youths who had risks in three or four domains had significantly higher average externalizing behavior scores ($F (12, 131) = 33.4, p < .001$) than those with fewer affected domains.

Although the evidence in regards to the assumptions of the cumulative risk model remains somewhat limited, a consistent finding that has emerged is that youths who encounter multiple risk factors are more likely to experience maladjustment. These findings have been consistent across gender (Sameroff et al., 1998; Fergusson & Woodward, 2000), racial groups (Sameroff et al., 1998; Atzaba-Poria, et al., 2004), single- or dual-parent homes (Sameroff et al., 1998), and neighborhoods of varying socioeconomic status (Stouthamer-Loeber, et al., 2002; Wikstom & Loeber, 2000). Still, even comprehensive measures of risk do not account for outcomes across all cases. This suggests that there may be individual differences between people that remain unconsidered. Given that risk factors across domains and across every stage of life have been examined, researchers have been forced to seek alternative explanations for unexpected outcomes.

**Resilience and Vulnerability: Efforts to Improve Prediction in Delinquency**

Some youth that experience very low levels of risk engage in delinquent acts, for which there are at least two possible explanations. First, the assessment of risk may be inadequate (Fergus & Zimmerman, 2005). In this situation, variables associated with increased risk could
be missing or measured inappropriately. A second possibility is that these individuals are more vulnerable than others. Vulnerability may be indicative of a greater sensitivity to stress or an underlying predisposition for the outcome under investigation (Belsky, 1997, 2005; Boyce & Ellis, 2005; Ellis, et al., 2005). Given that cumulative measures of risk are designed to capture most, if not all, variables that increase one’s risk, it becomes increasingly difficult to investigate the factors that may be missing. As a result, these cases tend to be ignored.

Comprehensive risk assessments also frequently identify cases in which youths who are exposed to numerous risk factors manage to avoid delinquent outcomes (Fergus & Zimmerman, 2005; Rutter, 1993, 2007a, 2007b; Werner & Smith, 1982, 1992). Individuals in this category are believed to be resilient to the effects of risk. While not entirely immune from their experiences with risk factors, they tend to avoid responding to risks by acting out or misbehaving. Because of this, researchers have been encouraged to consider whether additional factors may work to decrease the likelihood of negative outcomes. This has led to another adjustment in the assessment of risk in which the possible protective and promotive effects of some variables are also considered (Pollard, Hawkins, & Arthur, 1999; Stouthamer-Loeber, et al., 1993; Stouthamer-Loeber, et al., 2002; van der Laan, Veenstra, Bogaerts, Verhulst, & Ormel, 2010).

Scholars have debated over the definition of and the manner in which protective factors operate in the risk-behavior relationship (Farrington & Welsh, 2007). It is possible that some variables have a curvilinear relationship with particular outcomes so that one end of a variable’s distribution is associated with increases in risk and the other with reductions in risk. For example, if one had favorable attitudes towards crime, such as marijuana use, his or her risk of engaging in crime may increase; however, if the person had attitudes that were disapproving of
crime his or her risk would decrease. For those with neutral attitudes, their risk would neither increase nor decrease. Another possibility is that some variables have purely promotive effects and only work to decrease risk, but do not increase risk at any level (Farrington & Welsh, 2007). Further still, some variables may only moderate the effect of existing risk factors (Rutter, 1985). While the empirical evidence on the role of protective factors is still somewhat incomplete, the available research does indicate that protective variables are an important component of assessing overall risk.

One of the earliest studies of delinquency that considered both risk and protective effects was conducted by Stouthamer-Loeber and her colleagues (1993). Rather than dichotomizing each variable, these researchers trichotomized variables to reflect risk, protection, and neutral effects of individual measures. They found that 12 variables had both protective and risk effects and 7 had risk effects only. No variables in the study had a purely protective effect against delinquency.

In a later study, Stouthamer-Loeber, Loeber, Farrington, Wikström, and Wei (2002) extended their earlier work by exploring whether a cumulative risk score that accounted for both risk and protective effects improved prediction over the accumulation of risk alone. Two samples from the Pittsburgh Youth Study were examined longitudinally; one from ages 13-19 years and a younger sample from 7-13 years. Single variables were trichotomized if differing effects were observed (-1=protective, 0=neutral, 1=risk). For these measures, the three categories were created using the 25th and 75th percentiles as cutoff scores. A total of 44 and 40 variables across 6 domains, including child behaviors, child attitudes, school, peers, family, and demographic characteristics, were included for the older and younger samples, respectively. Of those variables, 28 and 17 were significant predictors of persistent serious delinquency. In both
samples, approximately one third of the variables had both risk and protective effects. The scores for all variables in each domain were summed to create six domain summary scores, which were also trichotomized using the 25th and 75th percentile cutoffs (Stouthamer-Loeber, et al., 2002). A total risk score (0 to 6), a total protective score (-6 to 0), and an overall risk-protective score (-6 to 6) were calculated by adding the trichotomized domain summary scores.

The results of this study showed that for both the younger and older samples, those with higher risk scores had a greater probability of persistent serious delinquency (Stouthamer-Loeber, et al., 2002). Similarly, those with more protective factors were less likely to be delinquent. The combined risk-protective score was linearly related to persistent serious delinquency. Furthermore, combining risk and protective scores improved prediction in both samples significantly with 72% of cases in the older sample and 80% in the younger sample being correctly classified (Stouthamer-Loeber, et al., 2002).

Also using a risk-protective cumulative model, van der Laan, Veenstra, Bogaerts, Verhulst, & Ormel (2010) investigated the relationship between risk and delinquency among participants of the Tracking Adolescents’ Individual Lives Survey (TRAILS). TRAILS was initiated in 2001 and is a prospective study of 2,230 Dutch preadolescents. Three domains were created (i.e., child temperament, family environment, and school environment) using 12 variables. Following the procedures used by Stouthamer-Loeber and her colleagues (1993, 2002), each variable and domain was trichotomized to create an overall risk-protective score (scores range from -3 to 3). Youths were classified as non-delinquents, minor delinquents, or serious delinquents. The results of van der Laan and colleagues (2010) analyses revealed that adding promotive effects from all three domains significantly improved prediction over risk effects alone ($\Delta \chi^2 (16, 2,085) = 47.34; p < .01$). Family risk reduced the likelihood of non-
delinquency, while temperament and family promotive effects increased non-delinquency. Among minor delinquents, the only significant effects were promotive effects in the temperament and family domains. Cumulative risk in the temperament and school domains and the promotive effects of temperament were significant predictors of serious delinquency in the expected directions. Additionally, van der Laan and associates (2010) examined the proportion of non-delinquents and serious delinquents across the distribution of the combined risk-protection scale. A linear relationship was observed. Non-delinquents experienced more promotive effects and fewer risks across domains, while serious delinquents had fewer promotive and more risk effects.

Pollard, Hawkins, and Arthur (1999) examined the effects of risk and protective factors on several adverse outcomes in a sample of adolescents from five states. Twenty risk factors and eight protective factors were assessed across four domains (i.e., community, school, family, and individual/peer). Aggregate risk and aggregate protection scores were calculated by averaging z-scores across all risk or protective factors, rather than dichotomizing and summing the measures. The results showed that youth with the highest levels of risk had the poorest outcomes. These findings were consistent across alcohol use, marijuana use, academic achievement, taking a gun to school, arrests, and attacking others to hurt them. The results for protective effects were less consistent. Higher aggregate protection scores were associated with lower levels of problem behaviors, but the effects of aggregated protection were stronger among higher risk individuals (Pollard et al., 1999). Further analysis of this relationship revealed a significant interaction between the risk and protective scales for every outcome ($p < .001$), where aggregate protection worked to moderate the adverse effects of risk.
In another study, Sprott, Jenkins, and Doob (2005) tested whether a strong school bond had a protective effect for Canadian youth at-risk for delinquency. The cumulative measure of risk included eight factors relating to the individual, their family, and their community. Findings indicated that a significant interaction between cumulative risk and school bond at age 10 to 11-years was predictive of both violent \((b = .066, p < .001)\) and nonviolent delinquency \((b = .103, p < .001)\) at age 12 to 13 years. In both models individuals with strong school bonds had lower levels of delinquency. The protective effects of school bonds were strongest among youths with three or more risks.

Wikström and Loeber (2000) have also examined the role of context in the relationship between risk and delinquency. Cumulative risk-protective scores were created for boys that had participated in the Pittsburgh Youth Study. Six variables were trichotomized using the 25\(^{th}\) and 75\(^{th}\) percentiles as cutoff scores, where -1 indicated a protective effect, 1 indicated the presence of a risk factor, and 0 neither effect. These values were summed to create an overall range of -6 to 6. The summed scale was again trichotomized in order to classify boys as high risk (scores = 3 to 6), high protective (scores = -6 to -3), and balanced (scores = -2 to 2). Additionally, neighborhoods in which the boys resided were also categorized based on the level of disadvantage (i.e., disadvantaged, advantaged, middle-range). Results of this study showed that for high-risk youth, the level of disadvantage did not make a difference in levels of serious offending.

The cumulative effects of risk and protective factors have also been examined over time. Stoddard, Zimmerman, and Bauermeister (2012) investigated whether cumulative risk and cumulative protective factors fluctuate through the course of adolescence, and the association of these influences with violence in a predominantly African American sample of youth. Both
indices included variables from individual, peer, parental, and family domains that were measured on four occasions between mid-adolescence and young adulthood. Growth curve models were estimated to assess the change in cumulative risk, cumulative promotive factors, and violence. The best fitting model for cumulative risk was cubic \( b = .02, p \leq .05 \), indicating that the number of risk factors increased initially in adolescence then stabilized before again increasing as youths transitioned to early adulthood (Stoddard, et al., 2012). A quadratic model provided the best fit for cumulative promotive factors over time \( b = -.21, p \leq .001 \). The analyses of violent behavior revealed that the best fitting model was linear \( b = -.03, p \leq .001 \), and that violent behavior decreased with age. Additional analyses indicated that over time, increasing cumulative risk was associated with increases in violence \( b = .11, p \leq .001 \), the accumulation of promotive factors decreased violence \( b = -.01, p \leq .05 \), and the interaction between the two was significant \( b = -.01, p \leq .05 \). To further assess the interaction, four groups were created: High Risk-High Promotive, High Risk-Low Promotive, Low Risk-High Promotive, and Low Risk-High Promotive. Stoddard and colleagues (2012) found that among high risk youth, higher levels of promotive factors seemed to reduce overall violence scores.

Taken together, the evidence from studies that incorporate thorough measures of risk, or risk and protective effects, indicates that delinquency is a complex behavior resulting from influences across multiple aspects of development. However, even when researchers employ thorough measures of various influences, there are still errors in prediction. Although the extent to which these errors are made are rarely reported, the studies that have done so suggest that the number of false positives and false negatives warrant further investigation. For example, in the study by Herrenkohl and his colleagues (2000), the cumulative risk measured at ages 10, 14, and 16 years accurately predicted violence at 18 in 80.4%, 84.4%, and 83.9% of cases, respectively.
Interestingly, between 1.2% and 3.3% were false positives and, despite being predicted to, did not engage in violent behavior. Even more striking, between 12.9% and 18.4% were false negatives. These cases were violent at age 18 despite predictions to the contrary.

Measures that include both risk and protective effects reveal similar patterns. In Stouthamer-Loeber and associates’ study (2002), a full model including both risk and protective effects correctly classified 72% of cases in the older sample and 80% of cases in the younger sample. Looking at only at-risk individuals, more than 10% of the older sample and a small percentage of the younger sample that had a risk score of zero were persistent serious delinquents. When the risk and protective factors were combined, between approximately 10 and 40% of youth that had predominately promotive or balanced scores were persistent serious delinquent. Similarly, in the study conducted by van der Laan and colleagues (2010), up to 10% of individuals at each level of predominantly promotive scores were found to be serious delinquents. The percentage of non-delinquents at each score above zero was as high as 15%.

In order to fully account for variation in development, comprehensive measures of risk may need to further expand. As noted previously, some of these errors may represent cases that are resilient while others are seemingly more vulnerable. Although researchers have made attempts to explain resilience (i.e., false positives), this research is still somewhat limited. There is also a need to identify sources of delinquency among youth that do not appear to be at-risk but are seemingly more vulnerable (i.e., false negatives). What is often left out of cumulative risk assessments is a genetic component. The following section explains the theoretical basis for considering both genetic and environmental factors. It also includes a review of the empirical literature that provides solid evidence for genetic effects on antisocial behaviors.
Theoretical Foundations and Evidence of Genetic Influences

Three theoretical frameworks suggest that genetic differences between individuals contribute to variation in their responses to risk factors (Belsky & Pluess, 2009a, 2009b). The dual-risk model contends that some people, for genetic reasons, are more sensitive or vulnerable than others when they confront known risk factors. These individuals are tainted by a heightened genetic risk that, when coupled with environmental risks, makes them more likely to experience maladaptive outcomes (Rutter, 2006a). Two evolutionary-based models also implicate the role of genetic factors in explaining variation in response to risk. Biological sensitivity to context theory and differential susceptibility theory assert that natural selection has shaped human development in such a way that some individuals are particularly sensitive to environmental conditions (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & Van IJzendoorn, 2011). In both models, genetic factors influence one’s ability to adaptively respond to his or her situations. Rather than limiting the potential of genetic influences to increasing the risk for problematic outcomes, these perspectives maintain that genes can contribute to both antisocial and prosocial behaviors.

Dual-Risk/Diathesis-Stress

Previous research investigating the relationship between genes and criminal behavior has been rooted in a dual-risk or diathesis-stress framework (Sameroff, 1983; Zuckerman, 1999). Typically, this approach involves identifying particular genes that influence the production and functioning of neurotransmitters, and assessing the moderating effects of those genes on an environmental stressor in predicting a trait or behavior of interest (Belsky & Beaver, 2011). Genes that code for different tasks in neurotransmission are selected because these neurobiological processes are responsible for communication between different parts of the brain.
and largely influence several aspects of human functioning (Raine, 1993; Wright, Tibbetts, & Daigle, 2008). Theoretically then, genetic variation results in behavioral variation through neurobiological mechanisms. When a significant interaction is found between a particular genetic variant and an environmental stressor, it is indicative of individual differences in sensitivity to that condition (Rutter, 2003; Rutter & Silburg, 2002). Those that possess a more sensitive genotype are considered to be at an increased risk for displaying the outcome under examination, particularly when exposed to specific negative life events. That is, these individuals are vulnerable. In contrast, when faced with the threat in question, those that do not possess the risky genotype are able to avoid the negative outcome. In short, they are resilient.

**Evidence supporting the dual-risk model.** Several studies have found support for the dual-risk model. Caspi and his associates (2002) peaked scholarly interest in this approach with their investigation of the interactive effects of maltreatment and the MAOA gene on antisocial outcomes. While maltreatment during childhood is a risk factor for later delinquency, they recognized that not all maltreated children develop into criminals. To further understand why individuals experience these different outcomes, they tested whether variation in the MAOA genotype moderates the effects of maltreatment. The MAOA gene was selected because it codes for the MAOA enzyme, which is responsible for the degradation of several neurotransmitters in the brain. If an individual were to possess a genotype that resulted in low MAOA activity, an overabundance of neurotransmitters would be present resulting in a tendency toward aggressiveness. In light of this, the authors hypothesized that those with high MAOA activity may be better suited to control antisocial impulses resulting from maltreatment than those with low MAOA activity (Caspi et al., 2002).
Among a sample of males from New Zealand, the results indicated that MAOA genotype did not influence exposure to maltreatment or have a main effect on antisocial behavior (Caspi et al., 2002). However, males that had the low MAOA activity genotype that experienced maltreatment were significantly more likely to engage in antisocial behavior \( (b = 0.68, p < 0.001) \) than those with the high activity MAOA genotype \( (b = 0.24, p = 0.03) \). Furthermore, this pattern of significant \( G \times E \) effects held across four indices of antisocial behavior including adolescent conduct disorder, adult violent convictions, self-reported violent disposition, and informant-based reports of antisocial personality disorder symptoms (Caspi et al., 2002).

Since Caspi and his colleagues (2002) published their findings, a number of other studies have emerged that provide further evidence of significant interactions between MAOA and adverse conditions (Kim-Cohen et al., 2006). One study examined the moderating effect of MAOA on the relationship between childhood adversity (i.e., parental neglect, exposure to interparental violence, and inconsistent discipline) and conduct disorder among a sample of 514 white male twins between 8 and 17 years old (Foley et al., 2004). Consistent with the findings obtained by Caspi and associates (2002), the results indicated that possessing the low-activity MAOA genotype increased the risk for conduct disorder only within adverse childhood environments \( (OR = 1.69, p = 0.04) \).

Widom & Brzustowicz (2006) also obtain results consistent with earlier studies using a cohort sample of abused and neglected children and a comparison group matched on sex, race, age, and family social class. The results of this investigation provided additional evidence suggesting that the effects of environmental risks may be moderated by genetic factors. Specifically, a significant interaction between MAOA genotype and abuse and neglect was predictive of juvenile violent and antisocial behavior. The interaction was significant for white
youth possessing the low activity genotype ($b = .39, p < .001$), but not those with the high MAOA genotype. Similarly, the interaction was a significant predictor of lifetime violent and antisocial behavior among only white individuals with the low activity genotype ($b = .34, p < .01$).

Kim-Cohen and her colleagues (2006) found additional support for the moderating effects of the MAOA genotype. Boys selected to participate in the Environmental Risk Longitudinal Twin Study (E-Risk) were included in the study ($N = 975$). The effect of experiencing physical abuse on a composite measure of mental health problems (i.e., antisocial behavior, ADHD, and emotional problems) was significantly stronger among boys with the low activity MAOA genotype ($b = 1.45, p < 0.001$) than boys with the high activity MAOA genotype ($b = 0.61, p = 0.005$).

The effects of MAOA and maltreatment on aggression, rule-breaking, and inattention was examined by Weder and associates (2009). The three outcomes were measured using the Achenbach Teacher Report form in which several items on the aggression and rule-breaking scale are delinquent acts (e.g., stealing). Initial results suggested that the interaction between MAOA genotype and maltreatment was not a significant predictor of aggression or rule-breaking behavior; however, the distributions for aggression scores suggested that the low MAOA genotype may have an effect for children that had experienced moderate trauma. In an exploratory analysis, Weder and her colleagues (2009) examined the relationship further and found that the interaction term was significant among children with low to moderate trauma histories ($\chi^2 = 3.8, p < .05$), but not among children that had a history of extreme trauma. The children that had the most traumatic experiences consistently had elevated aggression scores across all MAOA genotypes.
In a more recent study, Derringer, Krueger, Irons, & Ianco (2010) found evidence of a significant G×E interaction between MAOA genotype and childhood sexual assault, with low activity genotypes increasing risk. The interaction was a significant predictor of both adult antisocial behavior ($b = 0.47, p = 0.014$) and conduct disorder ($b = 0.576, p = 0.007$). The interaction was not significant in models predicting alcohol, tobacco, or cannabis use. The interactive effect of MAOA with harsh discipline was also examined across the same five outcomes, although none of the interactions were significant. However, another study found a significant interaction between MAOA and physical discipline on levels of delinquency among males (Edwards et al., 2010). Males with the low-activity allele were more sensitive to the effects of physical discipline ($b = .124, p < .05$) than males with high-activity alleles ($b = .065, p < .05$). The pattern of results for females was in the expected direction, but did not reach statistical significance.

Fergusson, Boden, Horwood, Miller, & Kennedy (2011) conducted an examination of the interaction between MAOA genotype and childhood sexual and physical abuse, and interparental violence on five antisocial behaviors using a sample of 398 males from the Christchurch Health and Development Study (CHDS). The results of this study showed a significant G×E effect on the total number of self-reported property or violent offenses between 16 and 30 years of age, the number of conduct disorder symptoms between 14 and 16 years of age, and hostility symptoms averaged across scores obtained at 18, 21, and 25 years of age. When the forms of abuse were analyzed separately, the interactions between MAOA and childhood sexual abuse were significant predictors of all five antisocial outcomes. The interactions between MAOA and physical abuse were significant in models predicting self-reported property and violent offenses and hostility scores, but not official reports of convictions or conduct disorder symptoms.
In addition to the moderating effects on measures of abuse and neglect, recent evidence also suggests that one’s genotype may interact with other risk factors to increase the likelihood of antisocial outcomes. Tikkanen and colleagues (2010) examined alcohol consumption, childhood physical abuse, and MAOA genotype in relation to violent recidivism among 174 Finnish male offenders. The offenders had a history of violence and alcoholism, and many were diagnosed with antisocial personality disorder, borderline personality disorder, or both. A significant interaction was observed between MAOA genotype and heavy drinking as well as MAOA genotype and childhood physical abuse. Offenders who possessed the high-activity MAOA allele were significantly more likely than those with the low-activity allele to engage in impulsive violent crimes in an 8-year follow-up if they had engaged in heavy drinking ($OR = 5.2; p = .004$) or had a history of childhood physical abuse ($OR = 5.3; p = .003$).

In another study, Beaver, DeLisi, Vaughn, & Wright (2010) investigated whether genetic risk moderates the effect of neuropsychological deficits on delinquency and low self-control, a strong predictor of criminal behavior (Pratt & Cullen, 2000). This inquiry was conducted on a subsample of 767 white males that were participants of the National Longitudinal Study of Adolescent Health (Add Health). The interaction between MAOA genotype and neuropsychological deficits was a significant predictor of low self-control at Wave I, when participants were in 7th through 12th grade, and one year later at Wave II ($b = .04, p = .05$ at both waves). The interaction was also a significant predictor of delinquency at Wave II, even after controlling for low self-control ($b = .02, p = .05$).

Lee (2011) examined the interactive effects of MAOA genotype and deviant peer affiliation on overt (e.g., physical aggression) and covert (e.g., property offenses) antisocial behaviors over a 6-year period. In regards to overt antisocial behavior, a significant interaction
was observed \((\beta = 1.57, p < 0.01)\) in which those with the high activity allele were more likely to exhibit higher levels of overt antisocial behavior in response to deviant peer affiliation. Individuals who possessed the low activity allele experienced more change in overt antisocial behavior over the 6-years of observation. In contrast, the models examining the effects of deviant peers, MAOA genotype, and their interaction on covert antisocial behaviors did not indicate that genotype had main or interactive effects.

Some evidence also suggests that community-level risk factors may interact with some genotypes to increase the likelihood of criminal outcomes. Hart & Marmorstein (2009) examined the interaction between MAOA and neighborhood child saturation (percentage of population under age 15) in predicting adolescent aggression. They reasoned that MAOA is associated with sensitivity to social rejection; therefore, in areas of high child saturation, aggressive behaviors would be more common and socially accepted and less likely to elicit peer rejection. A significant interaction was found between MAOA genotype and neighborhood child saturation. Males who possessed the short allele and lived in neighborhoods high in child saturation evidenced greater increases in aggression over a one year period than those with the same allele that lived in neighborhoods of lower saturation. The interaction was significant above and beyond the main effects of other neighborhood characteristics including poverty, urbanicity, and racial heterogeneity.

Although the majority of studies that have employed a dual-risk framework to better understand criminal behaviors have focused on the MAOA genotype, some research has also examined other genotypes. Again using a sample of participants from the Add Health study, DeLisi, Beaver, Vaughn, & Wright (2009) examined the interaction between DRD2 and having a criminal father in predicting delinquent outcomes among African American females across three
waves of study. They examined violent and serious delinquency at Waves I and II, and the number of police contacts at Wave III. During Wave I, the interaction between DRD2 and having a criminal father was a significant predictor of serious delinquency ($b = 1.00; p = .043$) and violent delinquency ($b = 1.16; p = .043$). At Wave II, the interaction remained significant in the model predicting violent delinquency ($b = 2.45; p = .000$), but was not significant for serious delinquency. The interaction between DRD2 genotype and having a criminal father was also a significant predictor of the number of police contacts at Wave III ($b = 2.25; p = .042$). Overall, African American females carrying more risk alleles (i.e., A1 alleles) were significantly more likely to be involved in delinquent and criminal behaviors.

In another study using the Add Health data, Beaver, Gibson, Jennings, and Ward (2009) examined the interaction between DRD2 genotype and religiosity on serious and violent delinquency. These authors hypothesized that youths that experienced a religious upbringing would have greater inhibitions toward delinquent behaviors. The results of this study demonstrated a significant interaction between DRD2 genotype and a lack of religiosity for both serious ($b = .102, p = .021$) and violent delinquency ($b = .094, p = .047$) at Wave I. In an effort to establish temporal ordering, a second set of analyses were conducted that included religiosity measured at Wave I and both measures of delinquency at Wave II, approximately one year later. The interaction between DRD2 genotype and a lack of religiosity did not reach statistical significance in the model predicting serious delinquency, but interaction was a significant predictor of violent delinquency at Wave II ($b = .122, p = .035$).

Beaver, Gibson, DeLisi, Vaughn, & Wright (2012) examined the interactive effects of two dopamine receptor genes (i.e., DRD2 and DRD4) and neighborhood disadvantage on violent delinquency. The results from negative binomial regression models indicate that both genotypes
interact with neighborhood conditions to predict violent behavior. Individuals living in
disadvantaged neighborhoods that possess the A1 allele of DRD2 and the 7-repeat allele of
DRD4 engage in violent behaviors more frequently \( (b = .33 \text{ and } b = .36, \text{ respectively}) \) than those
living in adequate neighborhoods.

A study conducted by Brody, Beach, Philibert, Chen, & Murry (2009), tested whether
variation in the serotonin transporter gene 5-HTTLPR interacts with participation in the Strong
African American Families (SAAF) program to influence alcohol use, marijuana use, and sexual
behavior during adolescence. The SAAF program was designed to prevent youth from engaging
in these delinquent and risky behaviors. An important feature of this study is that participants
were randomly assigned to experimental and control groups, which is extremely rare in studies
of G\( \times \)E interactions. This is particularly beneficial because, in addition to ruling out traditional
threats to validity, an experimental design also eliminates the possibility of gene-environment
correlations. These occur when a particular genotype influences the probability of one’s
exposure to a particular environmental condition. For example, individuals with a genetic
predisposition for high intelligence may seek out environments that are intellectually stimulating,
while those that do not have this predisposition may avoid such environments. In traditional
studies of G\( \times \)E interactions, the possibility of a gene-environment correlation must be assessed
statistically, which is unnecessary in this design.

Following previous research, Brody and his associates (2009) classified the alleles for 5-
HTTLPR as either “short” or “long,” with the possible genotypes being short/short, short/long, or
long/long. If an individual possessed one or two short alleles he or she was considered to be at-
risk. The results of their study revealed that youth with the genetic risk that did not receive the
intervention program initiated risk behaviors at a higher rate than any other group. Additionally,
participation in the SAAF program had a protective effect, even among youth with a genetic liability.

Reif and colleagues (2007) examined interactions between 5-HTT, MAOA, and dopamine transporter (DAT) genotypes, and adverse childhood environment in predicting violent behavior. The results indicate that 5-HTTLPR interacts with childhood environmental risk factors to predict violent behavior for the short/short and short/long genotypes. The long/long genotype had a protective effect against adversity in childhood. There were no significant interactions for MAOA or DAT with childhood adversity, but carrying both the short MAOA allele and the short/short or short/long genotypes for 5-HTTLPR most strongly predicted violent behavior.

Beaver, Sak, Vaske, & Nilsson (2010) created a composite measure of genetic risk by combining the total number of genetic risk alleles an individual possessed across three dopamine genes (i.e., DRD2, DRD4, DAT1), 5-HTT, and MAOA. Using a sample of African American males, they assessed the interactive effect of cumulative genetic risk and parent-child interactions on serious and violent delinquency across three waves spanning adolescence to early adulthood, the number of police contacts (lifetime), and a composite antisocial behavior index (lifetime). Significant interactions between genetic risk and parent-child relations were found to predict serious and violent delinquency at Wave I ($b = .33, p = .000; b = .24, p = .008$), violent delinquency at Wave II ($b = .35, p = .023$), serious and violent delinquency at Wave III ($b = .56, p = .007; b = .59, p = .002$), lifetime police contacts ($b = .21, p = .035$), and lifetime antisocial behavior ($b = .50, p = .009$).

In a recent study by Cicchetti, Rogosch, & Thibodeau (2012), the moderating effects of MAOA, 5-HTTLPR, and tryptophan hydroxylase (TPH1; alleles G and T) on childhood
maltreatment in predicting early antisocial behavior were assessed. Antisocial behavior was measured using self, peer, and adult-counselor reports. Additionally, four subtypes of maltreatment (i.e., neglect, emotional maltreatment, physical abuse, and sexual abuse) and three measures of the timing and frequency of maltreatment (i.e., onset, chronicity, and recency) were examined. In a series of ANCOVA models including TPH1 genotype, significant G×E interactions were observed for self-reported antisocial behavior ($F = 4.14, p = .04$), peer ratings of antisocial behavior ($F = 3.28, p = .038$), and adult reported antisocial behavior ($F = 3.03, p < .0376$). Post hoc analyses consistently indicated that those with the GT or TT genotypes that experienced maltreatment had significantly higher antisocial behavior scores than individuals that did not experience maltreatment. Those that possessed the GG genotype generally had more favorable outcomes.

The next series of models examined how maltreatment and 5-HTTLPR genotype contributed to antisocial behavior (Cicchetti et al., 2012). Again, significant interactions were observed for self-reported ($F = 3.91, p = .02$), peer-reported ($F = 2.60, p = .035$), and adult-reported antisocial behavior ($F = 2.93, p = .05$). Children with the short/short genotype consistently had higher antisocial behavior scores, and possessing the long/long genotype appeared to serve as a protective factor.

The final series of ANCOVA models estimated the effects of MAOA genotype and maltreatment for male subjects. A significant interaction was observed for self-reported antisocial behavior ($F = 4.30, p = .04$), but not peer or adult-reported antisocial behavior. Consistent with previous research, males with the low activity MAOA genotype that had experienced maltreatment reported higher levels of antisocial behavior than those with the high activity genotype (Cicchetti et al., 2012).
While many studies indicate that genetic differences can increase one’s risk of being involved in criminal behaviors, others have not found support for this claim. For example, Haberstick and associates (2005) attempted to replicate the findings obtained by Caspi and his colleagues (2002), using a sample of youth from the United States. Although they did observe a trend in the expected direction, the interaction between MAOA genotype and maltreatment was not significant across three different measures of conduct problems.

In another attempt to replicate the study by Caspi and his associates (2002), researchers examined the influence of MAOA genotype, maltreatment, and violent victimization on six measures of serious violent behavior (Huizinga et al., 2006). These measures included a composite measure of antisocial behavior, antisocial personality disorder symptomology, a disposition toward violence, conduct disorder, arrests for violent offenses as an adult, and a life-course measure of antisocial behavior spanning adolescence and early adulthood. Across all of these outcome measures, the interactions between MAOA genotype and maltreatment and MAOA genotype and victimization were not significant.

Beaver, Nedelec, Wilde, Lippoff, & Jackson (2011) examined whether MAOA genotype interacts with a protective-risk factor index to predict incarceration or anger and hostility. The interaction was not significant in models predicting the probability of ever being incarcerated. For males, however, the interaction between MAOA genotype and the protective-risk factor index was a significant predictor of anger and hostility scores. Among males with the low MAOA activity genotype, anger and hostility scores showed greater increases as the protective-risk score increased \( (b = .12, p < .05) \) relative to the group with the high MAOA genotype \( (b = .03, \text{n.s.}) \).
Although some studies of criminal behaviors have not observed statistically significant interactions between genetic and environmental factors, considerable support for the dual-risk model overall has been acquired. Some scholars, however, argue that particular genes may not merely increase vulnerability (Belsky, 1997, 2005; Boyce & Ellis, 2005). Instead, theoretical developments based on an evolutionary perspective suggest that individuals with “risky” genotypes may vary in their responses to the environment in both unfavorable and favorable ways. This reasoning serves as a foundation for two related theories that claim individual variation in response to adversity is genetically influenced: biological sensitivity to context and differential susceptibility.

**Biological Sensitivity to Context**

The basic logic of evolutionary theories is simple: all living things share an innate goal of dispersing their genetic material into future generations (Belsky, 1997, 2005; Belsky & Pluess, 2009a, 2009b). This requires that natural selection favor traits that promote survival and reproduction. Individuals encounter a number of environmental situations that may threaten their survival. Such events activate a stress response system in the body, resulting in automatic physiological responses that allow one to react to a situation in such a way that the likelihood of survival is optimized (Boyce & Ellis, 2005). The stress response system is housed in the central and the peripheral nervous systems, and involves reactions such as the release of different neurotransmitters in various parts of the brain, a redirection of energy to vital organs, and increases in heart rate and breathing. Because these responses tend to increase activity in some parts of the body and impede functioning in others, prolonged or repeated activation of this system is believed to contribute to the development of a number of mental or physical disorders (Boyce & Ellis, 2005). Although humans today encounter a great deal of stress-inducing
situations, it is likely that our ancestors experienced threats of greater severity and frequency (Nesse & Young, 2000). As a result, what may have been a useful mechanism in terms of promoting survival in the evolutionary past has come to be viewed as a weakness among modern humans.

The biological sensitivity to context theory (BSCT) maintains that individuals differ in their sensitivity to environmental conditions and the reactivity of their stress response system (Boyce & Ellis, 2005; Ellis & Boyce, 2008; Ellis, et al., 2005). The variation in reactivity is argued to be due to the mutual effects of genes and environmental conditions. Boyce & Ellis (2005) explain that the manner in which many components of the stress response system function is heritable. Some individuals may be born with a predisposition towards high reactivity, while others are predisposed towards lower reactivity levels. Furthermore, this system is shaped or “calibrated” through exposure to different environmental conditions early in the life course (Boyce & Ellis, 2005).

Children that are highly reactive by nature and who are repeatedly exposed to trauma or high-stress situations are hypothesized to develop even greater sensitivity to adversity (Boyce & Ellis, 2005; Ellis & Boyce, 2008). In a volatile environment, enhanced sensitivity to threats could serve to improve one’s ability to recognize and manage potential dangers. The repetition of this process may ultimately lower the threshold for anticipating new threats in future situations. In this scenario, individuals would acquire a greater awareness and sensitivity to stressors, but are also more likely to experience negative psychological and physiological outcomes as a result of the extensive reactivity (Boyce & Ellis, 2005).

It is also hypothesized that children born with highly reactive phenotypes may be the most likely to benefit from highly supportive or predictable conditions (Boyce and Ellis, 2005).
In such low-stress environments, particularly when coupled with quality parenting, children with high sensitivity could gain tremendously from beneficial experiences. When placed in an ideal environment, these youth use their heightened awareness and sensitivity to their benefit, and are able to take full advantage of the promotive aspects of their situation. In the long run, these children fare better, in terms of health and behavioral adjustment, than less sensitive children (Boyce & Ellis, 2005).

Although some children may be predisposed to be more sensitive to the conditions of their environment, others are born with low reactivity phenotypes. According to BSCT, these individuals are likely to experience normative outcomes, particularly when they are exposed to moderately stressful environments (Boyce & Ellis, 2005). Since they are not highly reactive, it is less likely that the threatening or advantageous experiences they encounter will have a substantial impact on their development. Under somewhat difficult conditions, children that have low reactivity levels would be resilient. Conversely, under extremely threatening or supportive conditions, the benefits of possessing a low reactivity phenotype would be lost (Boyce & Ellis, 2005). Without the ability to recognize and respond to various aspects of their environments, these children may be less likely to survive dangerous confrontations or exhibit meaningful gains from positive experiences.

BSCT postulates that differences in biological sensitivity to context (BSC) are the result of natural selection favoring conditional adaptations (Boyce & Ellis, 2005). The social, physical, and economic conditions of the future are uncertain at the time each generation is born; therefore, it is unlikely that any strategy for ensuring survival and reproduction would be uniformly beneficial across all generations. Although modern humans retain the same genetic material as their ancestors, the genetically-influenced traits that may have been advantageous for
one’s ancestors might not be favorable in the conditions of today. Therefore, the capability of a single genotype to take on a range of phenotypes (i.e., phenotypic plasticity) would be most advantageous.

Boyce and Ellis (2005) propose that individuals pick up on cues embedded in their environment early in life and adapt to meet the demands of their conditions. Particularly harsh or supportive environments should work to either maintain or develop increased levels of sensitivity. This adaptation would be beneficial in terms of survival and reproduction, allowing one to recognize and respond to threats in the worst scenarios, and enhancing aspects of development in the best scenarios. Those that live longer and have more resources also have more opportunities to reproduce. In contrast, lower sensitivity would be desirable when exposed to environments that are neither overly adverse nor overly supportive because the negative aspects of being highly sensitive, including mental and physical difficulties, are avoided (Boyce & Ellis, 2005).

While genes interact with the environment in a systematic way to calibrate sensitivity levels, according to BSCT genes also have the potential to influence BSC through two additional means (Boyce & Ellis, 2005). First, genetic factors can determine reaction norms, or phenotypic “bookends.” Levels of BSC can be viewed across a spectrum ranging from low to high, and although genes and the environment work together to place one at the optimum level for their particular conditions, one’s genes may also place limits on the range of levels that can be achieved. For example, two children with equivalent environmental conditions may vary in their levels of BSC if their reaction norms are genetically restricted to a higher or lower location on the BSC spectrum. Relatedly, genotypic influences may also determine the breadth or width of reaction norms (Boyce & Ellis, 2005). As a result, some children are more malleable and can
take on a greater range of sensitivity levels. These individuals are subject to greater environmental influence. Others will possess a more narrow range of reaction norms, limiting the potential for the environment to have a substantial effect. Those with narrow reaction norms will therefore develop over a more fixed trajectory.

Also based on an evolutionary perspective, differential susceptibility theory provides another explanation for variation in response to the environmental conditions. There are slight differences between the two theories in reasoning and points of emphasis; however, the two are highly compatible and make similar predictions regarding the influences of genes and the environment (Ellis et al., 2011). In light of the substantive overlap, differential susceptibility theory will first be explained followed by a review of the evidence supporting these theories.

Differential Susceptibility

Belsky’s (1997, 2005) differential susceptibility theory (DST) is heavily founded on the same assumptions as BSCT. Humans, like other organisms, are designed to distribute their genetic material in subsequent generations. In order to be successful in this regard, natural selection must favor features that promote reproduction and survival. Reproduction can occur through two methods. The most obvious method is direct reproduction, when an organism produces immediate descendants such as children and grandchildren (Belsky & Pluess, 2009a, 2009b). Since biological relatives share some of their genetic material, reproduction can also be indirect. This occurs when a portion of one’s genetic material is passed through the successful reproduction of a relative with some genetic overlap, such as a sibling. Furthermore, since the future is uncertain, a mechanism for ensuring survival and reproduction under a variety of conditions is likely to have been preserved through natural selection. These assumptions form the basis of DST.
It is impossible to know whether traits that were advantageous in previous generations will remain useful in subsequent generations because the future is largely unpredictable (Belsky, 1997, 2005; Belsky & Pluess, 2009a, 2009b). In light of this, the optimal evolutionary strategy would be for individuals to be capable of taking on a range of traits to meet the needs of their environment. As an illustrative example, a trait such as aggressiveness may have been desirable in more primitive generations because it provided a means of defense, ensuring one’s survival. Furthermore, the capability to protect one’s self may also increase the likelihood of reproducing. In one respect, the longer an individual can survive, the more opportunities he or she may have to find a mate and produce offspring. Violent displays in particularly volatile environments may also increase one’s status and resources, ultimately making one a more desirable mate. While aggressive behavior is widely considered undesirable in modern societies, Belsky (1997) argues that there are some ecological niches in which such a trait would still be beneficial, such as a community marked by extreme deprivation. As a result, natural selection may have preserved the capacity for some individuals to take on a variety of aggressive phenotypes to match the demands of their environments. As described in BSCT, this phenotypic plasticity provides opportunities for conditional adaptation where individuals can improve their chances of survival and reproduction (Belsky, 1997, 2005).

Once two individuals have successfully reproduced, it is necessary to ensure the survival of the offspring and maximize the likelihood of it reproducing. Parents attempt to engage in rearing strategies that foster survival and reproduction; however, without knowing what the future will hold, parents are unable to adjust their childrearing techniques to give their offspring the best chance of meeting these two goals (Belsky, 1997, 2005; Belsky & Pluess, 2009a, 2009b). As a result, it is possible for parents to misguide their offspring in ways that are
counterproductive. Belsky (2005) claims that from an evolutionary perspective, it would make sense for mating pairs to produce progeny with differing levels of sensitivity or malleability because it would disburse risk and increase the probability that at least one of the offspring would reproduce. In the event that the parenting strategy is indeed ideal for preparing children for the future, the children that are more sensitive would be likely to follow parental guidance, and survive and reproduce. In contrast, when the parenting strategy is misguided children that are less malleable would resist parental efforts to shape development, making them more likely to survive and reproduce (Belsky & Pluess, 2009a, 2009b). In both situations the parents and the siblings would all benefit in terms of reproduction, either directly or indirectly.

Belsky (1997, 2005) maintains that genes play a central role in this process in multiple ways. Certain individuals are likely to have a genetic predisposition towards poor functioning, as many traits have been found to be highly heritable (Plomin, 2004). According to DST, individuals will also vary in their sensitivity to the environment and this variation is largely influenced by genetic factors (Belsky et al., 2007). Rather than presuming that particular genes work only to increase one’s vulnerability for maladaptive outcomes, as assumed under a dual-risk model, DST proposes that some genotypes are associated with greater phenotypic plasticity (Belsky et al., 2007; Belsky et al., 2009). Under this framework, individuals that possess a more sensitive genotype will be more likely to experience outcomes that mirror the quality of their rearing environments. Sensitive individuals situated in adverse contexts will be likely to display negative behaviors, while those situated in particularly beneficial contexts will be likely to experience positive outcomes. Still, others may be less sensitive to the environment and are more heavily influenced by their genetic predispositions. In short, individuals will differ in the amount of influence genes and the environment has on their development. This will produce
variation in behavioral outcomes, even if different individuals shared the exact same environments and experiences.

Given the compatibility of BSCT and DST, these two theories have recently been combined to form an evolutionary-neurodevelopmental theory of behavior and development (Ellis et al., 2011). According to this theoretical advancement, individuals differ in their sensitivity to the environment and those that have the greatest sensitivity are more receptive to both positive and negative conditions. Genetic susceptibility factors are hypothesized to enhance neurobiological susceptibility, which is observed through behavioral indicators. Variation in susceptibility to environmental influences exists both between and within individuals, and may change over the life course (Ellis et al., 2011). Furthermore, this variation in neurobiological sensitivity is considered adaptive based on an evolutionary perspective and remains a characteristic of modern humans because it has promoted both survival and reproductive fitness. While the key components of each theory remain intact, the marriage of these two theories results in a more complete theory that emphasizes the importance of both biological and behavioral aspects of human development.

Evidence supporting the differential susceptibility model. Although the dual-risk model has been the dominant paradigm for examining the role of genes and environment in development, some evidence has emerged in support of the differential susceptibility perspective. Much of the available research under this framework has examined the effects of the DRD4 genotype and some measure of parenting. For example, Bakermans-Kranenburg & van IJzendoorn (2006) investigated the interactive effects of DRD4 genotype and maternal sensitivity on externalizing behaviors in childhood, a precursor to criminality (Farrington, 1991; Loeber, 1982; Moffitt, 1990, 1993; Nagin & Tremblay, 1999; Olweus, 1979; White, et al., 1990).
To assess sensitivity, mothers and their infants were videotaped in their homes for 1.5-hours on six occasions. Mothers were rated on their awareness and interpretation of signals from their babies, and the promptness and appropriateness of their responses. Two years later, mothers were asked to complete the Child Behavior Check List to evaluate their child’s behavior. The interaction between DRD4 genotype and sensitivity at 10 months was a significant predictor of externalizing behaviors at 39 months ($F = 6.24, p = .02$). Children with the 7-repeat DRD4 allele exhibited the highest levels of behavioral problems if mothers were insensitive, but this group also displayed the fewest externalizing behaviors when maternal sensitivity was high. Among children that did not possess the 7-repeat allele, maternal sensitivity did not have an effect on externalizing behavior scores.

In another study, Bakermans-Kranenburg, IJzendoorn, Pijlman, Mesman and Juffer (2008) used an experimental design to test whether genetic differences between toddlers contributed to the effectiveness of an intervention designed to reduce externalizing behaviors. The Video-feedback Intervention to promote Positive Parenting (VIPP) was implemented among the experimental families ($n = 74$) to increase parental sensitivity and positive discipline interactions. Family interactions were recorded and female interveners visited families in their homes six times to provide feedback on parenting strategies using sections of the recorded interactions illustratively. Simultaneously, families in assigned to the control group ($n = 83$) received phone calls in which the mothers were asked about the development of their child. During these calls, no parenting advice was given. Mothers were asked to complete the Child Behavior Checklist during screening, the pre- and posttests, and during a follow-up one year after the posttest.
Bakermans-Kranenburg and her colleagues (2008) observed a significant three-way interaction between the intervention, DRD4 genotype, and time of observation \( (F = 4.47, p = .04) \). The intervention was effective in decreasing externalizing behaviors at the follow-up evaluation for children that possessed the 7-repeat DRD4 allele. The intervention was not effective among children that did not have this allele. Examination of the three components of externalizing behavior showed a significant reduction in oppositional behavior for children in the experimental group with the 7-repeat allele \( (F = 4.34, p = .04) \), but not overactive or aggressive behaviors. The effect was significantly stronger in this group when mothers showed the greatest improvement in sensitive discipline strategies.

Although these studies demonstrate support for the DST in terms of behavioral measures of human functioning as emphasized in Belsky’s (1997, 2005) original statement of the theory, the studies do not evaluate the physiological aspect of functioning as emphasized by Boyce & Ellis (2005). In an effort to assess whether gene-environment interactions influence behavior through physiological mechanisms, Bakermans-Kranenburg and her colleagues (2008) conducted another study with the families participating in the VIPP program examining change in children’s cortisol levels. Cortisol is a chemical released in the hypothalamic-pituitary-adrenal (HPA) axis, a component of the body’s stress response system. A meta-analysis of 70 studies indicates that higher levels of cortisol in preschoolers was associated with higher levels of externalizing behaviors, while lower levels were associated with increased externalizing behaviors in school-aged children (Alink et al., 2008). Based on these findings, the authors hypothesized that children 1-3 years of age whose mothers participated in the VIPP program would have lower levels of cortisol and externalizing behaviors, particularly when these children carried at least one 7-repeat DRD4 allele.
In the randomized controlled trial, the interaction between the experimental condition and DRD4 genotype was significant in the model predicting cortisol production ($F = 7.24, p < .01$). Comparisons of the four groups (i.e., experimental and control groups split by genotype) indicated that children with the 7-repeat allele that were in the experimental group showed the lowest levels of cortisol after the intervention. The intervention did not make a significant difference for children that did not have the 7-repeat allele (Bakermans-Kranenburg et al., 2008). Taken together, these studies support the combined differential susceptibility model by demonstrating that gene-environment interactions shape both physiological and behavioral outcomes.

Prior research has also investigated whether DRD4 genotype may interact with participation in the SAAF program in a manner consistent with the differential susceptibility model (Beach, Brody, Philibert, & Lei, 2010). Families participating in the study were randomly assigned to intervention and control groups. The SAAF program participants attended seven meetings in the community that emphasized different aspects of family functioning, including parenting skill building, that may reduce substance use among youth. Substance use for the prior month was measured every year when youths were between 11 and 14 years of age. The results of a negative binomial regression showed a significant interaction between DRD4 genotype and the intervention ($\gamma_{S\times G} = -1.04, p < .01$). Examination of the group means over time indicated that among youths in the intervention group that possessed the 7-repeat allele of DRD4, growth in substance use was reduced ($\gamma_{SAAF} = -.69, p < .01$) while those in the control group reported increases in substance use. Furthermore, this pattern was not observed among youths that did not carry the 7-repeat allele of DRD4.
In addition to investigating how genes interact with intervention programs, researchers have also investigated potential interactions with other risk factors for antisocial outcomes. For example, using a sample of 514 white male twins from the Virginia Twin Study for Adolescent Behavioral Development, researchers investigated the joint effects of MAOA genotype and childhood adversity (i.e., neglect, interparental violence, and inconsistent discipline) on conduct disorder (Foley et al., 2004). The results of this study indicated that males with the low MAOA activity allele were at an increased risk for conduct disorder if they had experienced adversity ($OR = 1.69, p = .04$). However, boys with this genotype also had a significantly lower risk for conduct disorder if they had not experienced adversity during childhood. Although it often remains unnoticed, this same pattern emerged in the study by Caspi and his colleagues (2002). Boys with the low activity MAOA genotype scored the lower than all other groups on antisocial behaviors when they had not been exposed to maltreatment.

Investigating the cumulative effects of multiple alleles, Simons and associates (2011) tested whether some individuals were more or less aggressive as a result of more distal environmental conditions. Both positive and negative aspects of the social environment were considered. Favorable aspects of the environment included supportive parenting, school environment, religious participation, and neighborhood informal social control. Adverse conditions were comprised of harsh parenting, experiences of racial discrimination, neighborhood victimization, and involvement with violent peers. These measures were combined to form a measure of the social environment spanning the spectrum between favorable and adverse conditions. Additionally, the relationship between genetic and environmental factors and aggression was hypothesized to be mediated a hostile orientation, as indicated by a
hostile view of relationships, anger, and the perceptions associated with a reputation for toughness.

Simons and colleagues (2011) found that the cumulative measure of plasticity alleles and social environment produced a significant interaction in models predicting hostile view of relationships ($b = .188, p \leq .01$), chronic anger ($b = .728, p \leq .05$), belief in toughness ($b = .561, p \leq .05$), and aggression ($b = .193, p \leq .01$). Furthermore, the combined effect of the two plasticity genes was compared to possessing just one was investigated for each of the outcomes. The coefficients for those possessing both plasticity alleles were significant and substantially greater than the estimates for individuals that only carried the DRD4 or 5HTTLPR plasticity alleles across all four measures ($p \leq .05$). Finally, in a structural equation model Simons and his colleagues (2011) demonstrated the mediating effects of hostile orientation on the relationship between the gene-environment interaction and aggression. An indirect relationship was observed, where one’s genetic plasticity significantly increased his or her hostile attitudes when the social environment was adverse and decreased it when the environment was favorable.

In another study, Simons and colleagues (2012) investigated whether adoption of a street code mediated the relationship between a gene-environment interaction and aggression. In this study, DRD4, 5HTTLPR, and MAOA were combined to form a measure of cumulative genetic plasticity. Four environmental measures were created to assess whether the environment was hostile or demoralizing: harsh/inept parenting, caregiver antisocial behavior, racial discrimination, and neighborhood crime and the absence of social control. The two-way interaction between cumulative genetic plasticity and hostile/demoralizing environment was a significant predictor of adopting the street code ($b = .961, p \leq .01$) and aggression ($b = .351, p \leq .01$). Closer inspection of these relationships suggested that individuals with more plasticity
alleles were more likely to adopt a street code if exposed to hostile/demoralizing conditions, but also less likely to adopt the street code if they were not exposed to these conditions compared to those with fewer plasticity alleles. Moreover, the G × E effect worked indirectly through adoption of a street code to influence aggression.

Given the existing evidence suggesting that genes may interact with the environment to both enhance and hinder development, Bakermans-Kranenburg & Van IJzendoorn (2011) recently conducted a meta-analysis to compare the effect sizes of both positive and negative outcomes. The study focused on the effects of dopamine genes (i.e., DRD2, DAT, and DRD4), and included 12 studies. Of these studies, nine reported effect sizes related to vulnerability, or susceptibility to adverse environments, and six reported effect sizes related to positive outcomes. The combined effect size for negative outcomes was $r = .37 \ (p < .001)$ for those carrying risk alleles and $r = .10 \ (p = .26)$ for those without risk alleles. The difference between these effects was significant ($Q_{\text{contrast}} = 5.24, \ p = .02$), suggesting that those with risk alleles for dopamine genes were more vulnerable to adverse environmental conditions than those without risk alleles. A similar pattern emerged for positive outcomes, with $r = .31 \ (p < .001)$ for those with risk alleles, compared to $r = -.03 \ (p = .53)$ for those without. Again, the difference was significant ($Q_{\text{contrast}} = 13.84, \ p < .01$), indicating that children with “risk” alleles fared better than others when situated in a supportive environment. Susceptible children were also found to be equally susceptible to positive and negative environmental conditions.

Overall, the available evidence provides support for the differential susceptibility model with regard to behavioral development. Although not contradicting the findings produced under the dual risk perspective, the studies reviewed here do indicate that genes may work to influence one’s sensitivity to their environment rather than simply increasing their risk. In terms of
criminal behavior, this perspective may provide an explanation as to why some individuals seem to engage in delinquency despite being low-risk and others are able to develop in a prosocial manner even under high-risk conditions. Those that are highly sensitive may exhibit antisocial behaviors when they encounter only a small number of risk factors. On the other hand, individuals that have lower sensitivity levels may be able to overcome a greater number of risk factors.

The Effects of Gender

The research produced under both the dual risk and differential susceptibility frameworks provides clear evidence of the importance of both genes and the environment in terms of behavioral development. Some studies also indicate that the influence of these factors, both in terms of the specific genes and environments involved and the magnitude of their effects, may vary between males and females. Findings from these studies highlight the importance of investigating potential sex effects in genetically informed analyses of behavior. Although sex differences have been observed across a number of phenotypes, the research reviewed here will focus on antisocial and analogous behaviors.

Evidence of sex-specific effects comes from behavioral and molecular genetic studies. Behavioral genetic studies decompose the variance in a trait into genetic and environmental parts. For example, in a study assessing the genetic and environmental contributions to externalizing behaviors in 2,292 twins in Virginia, Silberg and colleagues (1994) found evidence of gender differences at ages 8-11 years. Using structural equation modeling techniques, they found that the heritability estimate for males was substantially greater in males ($a^2 = .38$) than females ($a^2 = .13$), while the shared environmental contributions to externalizing behaviors was

1 A description of qualitative and quantitative sex differences and procedures for assessing these effects is provided in chapter three.
stronger in females ($c^2 = .62$) than in males ($c^2 = .46$). The unique environmental effects were also somewhat stronger in females ($e^2 = .25$) than in males ($e^2 = .16$).

Other studies have suggested that the genetic effects are stronger in females. Eley, Lichtenstein, & Stevenson (1999) decomposed the variance in aggressive and non-aggressive antisocial behaviors in Swedish and British samples of twins. The Swedish sample was comprised of 1,022 7-9 year old twin pairs, and the British sample was included 501 pairs ages 8-16. In both samples, the effects on non-aggressive antisocial behaviors varied for males and females. Among Swedish males, additive genetic effects accounted for 30% of the variance, while common and unique environmental effects accounted for 44% and 26%, respectively. For females the additive genetic effects were stronger (41%), and the common and unique environmental effects were weaker (37% and 22%, respectively). In the British sample, additive genetic factors did not account for any of the variance in non-aggressive antisocial behavior among males ($c^2 = .64$, $e^2 = .36$). In contrast, genetic effects accounted for nearly half the variance for females ($a^2 = .47$, $c^2 = .27$, $e^2 = .26$). No sex-specific effects were found in the models for aggressive behaviors.

Another behavioral genetic study by Vierikko, Pulkinen, Kapiro, Viken, and Rose (2003) sought to determine whether the same genetic and environmental factors influenced aggression, and if the magnitude of the effects differed, among males and females in a sample of 1,652 12-year old twins. The behavior of the youth was rated by parents and teachers. After decomposing the variance in teacher-reported aggression, Vierikko and colleagues (2003) found that the magnitude of the effects differed between sexes. For females, the proportion of the variance attributed to additive genetic, common environment, and unique environment was 54%, 37%, and 9%, respectively. For males, the additive genetic effects were substantially weaker (27%),
and the common environmental effects were stronger (15%). Additionally, there were male-specific common environmental influences that accounted for a large proportion of the variance (51%) in aggression in males, but not females. The remaining 7% of the variance for males was due to unique environmental factors.

In the decomposition of the variance in parent-reported aggression, Vierikko and associates (2003) observed a somewhat different pattern of results. Among females, the additive genetic effects still accounted for 54% of the variance, and there was a slight decrease in common environmental effects (25%) and an increase in unique environmental effects (21%). For males, additive genetic factors accounted for only 14% of the variance in aggression, 75% was attributed to common environmental factors, and 11% was due to the effects of the environment unique to each twin. Based on both teacher and parent reports, the magnitude of genetic and environmental influences appears to differ between males and females, with males being affected more by environmental factors and females more by genetic influences. Additionally, using teacher reports, some overlap in genetic and environmental influences was observed between sexes, but males appear to be influenced by other common environmental factors specific to boys only. No sex-specific effects were observed in models using parent reports.

Other scholars have used molecular genetic data to examine how gender can interact with specific genes to influence behavior. For example, Sjöberg and colleagues (2007) examined the relationship between MAOA genotype, housing type (i.e., single family or multifamily), sexual abuse, and adolescent criminality (i.e., total criminality, stealing, vandalism, and violence) in females. A significant interaction was observed between MAOA genotype and sexual abuse in the models predicting total criminality ($F = 2.732, p < .05$), stealing ($F = 3.534, p < .10$), and
vandalism ($F = 4.315, p < .05$). In contrast to the empirical literature indicating that the low activity allele interacts with adversity to predict criminal behavior among males (Caspi et al., 2002; Foley et al., 2004), the results of this study indicated that the high activity allele was risky for females.

Another study investigated the relationship between MAOA and childhood adversity on conduct disorder (Prom-Wormley et al., 2009). A weak but significant interactive effect was observed ($OR = .77, p = .05$) for females with the high activity MAOA genotype that experienced high levels of childhood adversity. However, after this model was reanalyzed using a ridit transformation to account for differences in the sample size at each level of adversity, the interaction was no longer significant. While these findings should be interpreted with caution, the pattern of results is suggestive of sex differences in G × E effects.

In another attempt to better understand the development of conduct disorder, researchers examined the interaction between MAOA genotype and prenatal exposure to cigarettes (Wakschlag et al., 2010). They found a significant interaction for both males and females, but in different directions. The low activity genotype was associated with an increase in parent-reported CD symptoms among males ($b = -.178, p = .032$), but for females the high activity genotype increased risk ($b = .245, p = .002$).

Aslund and associates (2011) examined MAOA genotype and maltreatment predicting four different measures of delinquency among 17-18 year old males and females. The interaction was a significant predictor of overall delinquency ($F = 14.56, p < .001$), vandalism ($F = 4.97, p = .007$), stealing ($F = 19.66, p < .001$), and violence ($F = 21.27, p < .001$). Boys with the short variant had a higher risk of delinquency when exposed to maltreatment, and girls with
at least one long variant of the polymorphism had a higher risk for delinquency when exposed to maltreatment.

Schwartz and Beaver (2011) also tested whether the interaction between MAOA and perceived prejudice was predictive the probability of being arrested. Males and females were analyzed separately. In the model predicting the probability of arrest for males, the interaction between MAOA genotype and perceived prejudice was significant ($b = 1.15$). Males with the low activity genotype that reported perceptions of prejudice had a greater chance of being arrested ($OR = .56$) compared to males that did not report perceptions of prejudice ($OR = .32$) or males with the high activity genotype regardless of perceived prejudice ($OR = .35-.38$) The interaction was not a significant predictor of the probability of being arrested for females.

Relatedly, Brody and colleagues (2011) employed the differential susceptibility framework to investigate the interactive effects of perceived discrimination and 5HTTLPR genotype on conduct problems in a sample of African American adolescents. Adolescents were observed at ages 15, 16, and 17. An unconditional growth curve model indicated that there was significant variability in the slope mean for conduct problems across the three waves. In a conditional model, the interaction between 5HTTLPR genotype and perceived discrimination was significant ($\beta = .76, p < .01$). Inspection of the plotted slopes for each genotype revealed that individuals carrying at least one short allele had greater conduct problems than those with two long alleles when perceived discrimination was highest, and the lowest when perceived discrimination was lowest. When the models were estimated separately for males and females, the pattern of findings held for males ($\beta = 1.22, p < .01$) but the interaction term was not significant for females.
Li and Lee (2010) have also explored a potential $G \times E$ interaction involving 5HTTLPR genotype. Their study examined the moderating effects of this gene on the relationship between maltreatment and antisocial behavior among boys and girls. A latent class analysis of antisocial behavior revealed a 2-class solution for females and a 3-class solution for males between 12 and 20 years of age. For females, 8.8% were classified in a class characterized by exclusive covert antisocial behavior (e.g., vandalism, breaking and entering, selling marijuana), with the remaining girls having low probabilities of antisocial behavior. For males, 85% exhibited minor antisocial behaviors, 10.6% were classified as being exclusively covert, and 4.5% had high probabilities of covert and overt antisocial behavior (e.g., threatening with a weapon). A significant interaction between 5HTTLPR genotype and maltreatment was predictive of group membership for girls in the exclusive covert group ($\beta = -1.98$, $p < .01$). Girls that had two short alleles who had experienced maltreatment were 12 times more likely to be classified in the exclusive covert than those with one or zero short alleles (Li & Lee, 2010). The interaction term was not significant in the models for males.

Belsky and Beaver (2011) directly tested the differential susceptibility hypothesis in a recent study of the effects of genes and parenting quality on self-regulation. In this study, self-regulation was selected as the outcome measure because substantial evidence suggests that this trait is strongly related to development (Pratt & Cullen, 2000). Those with high self-regulation tend to function in a prosocial manner, while those with low self-regulation are more likely to exhibit antisocial behaviors. Additionally, rather than examining the influence of a single gene on development, Belsky and Beaver (2011) created a measure of cumulative genetic plasticity. This measure was a summation of the total number of plasticity alleles (i.e., alleles commonly associated with increased risk in dual-risk models) an individual possessed across DAT1, DRD2,
DRD4, 5HTTLPR, and MAOA. The results of ordinary least squares regression analyses indicated that the interaction between cumulative genetic plastic and parenting quality was significant among males ($b = .95$, $p < .01$), but not females. In a post hoc analysis, the males with the most plasticity alleles were more heavily influenced by the quality of the parenting they received in both positive and negative ways. These boys scored highest on self-regulation when parenting was supportive and lowest when parenting was least supportive. The effects of parenting quality decreased as the number of plasticity alleles decreased, and those with the fewest plasticity alleles appeared unaffected by the quality of parenting to which they were exposed.

Although behavioral and molecular genetic studies have both observed differences in the effects of genes and various environmental factors on antisocial behaviors for males and females, some studies have not found these differences (Boisvert, Vaske, Wright, & Knopik, 2012; Eley et al., 1999; Rhee & Waldman, 2002; Slutske et al., 1997; Taylor, McGue, & Iacono, 2000; Taylor, McGue, Iacono, & Lykken, 2000; van den Oord, Verhulst, & Boomsma, 1996; van Hulle, Rogers, D’Onofrio, Waldman, & Lahey, 2007). Previous literature has not produced consistent findings regarding the relationship between genes, gender, environmental risks, and behavioral development. Given the limited and somewhat inconsistent results of these studies, more research investigating the role of gender is warranted.

**Conclusion**

The risk factor approach to understanding delinquency has shifted over the last two decades, from investigations of the effects of single risk factors on delinquency to research on the cumulative effects of multiple risk factors. Despite the use of comprehensive measures of risk covering different domains, a number of youth do not exhibit predicted outcomes (Fergus &
Zimmerman, 2005). As a result, many scholars have searched for explanations as to why some youth are able to overcome the risks they encounter (Blum, et al., 2002; Rutter, 1979, 1985, 1990, 1993; Stouthamer-Loeber, et al., 1993; Stouthamer-Loeber, et al., 2002; Werner & Smith, 1992). Even these efforts have not produced a solid understanding of resiliency. Furthermore, youths that are delinquent despite being low-risk have not received adequate attention in the empirical literature. Without knowledge of the factors that contribute to their delinquency, and an understanding of how such factors work together to shape behavior, efforts at preventing or reducing their criminality are unlikely to be productive (Wikström, 2008).

While it is apparent that individuals respond to the risks that they encounter in various ways, explanations for the variation in responses are limited. The differential susceptibility perspective (Belsky, 1997, 2005; Belsky, et al., 2007; Belsky & Pluess, 2009a, 2009b; Boyce & Ellis, 2005; Ellis & Boyce, 2008; Ellis, et al., 2005) provides a solid theoretical framework to guide research seeking to better understand cases that develop prosocially or antisocially in the face of varying degrees of risk. Rather than limiting explanations for differential responses to social factors, the differential susceptibility model emphasizes the importance of both genes and the environment. The role of these factors can be assessed using behavioral genetic modeling techniques, to which the focus is turned in the following chapter.
CHAPTER THREE

BIOMETRICAL GENETICS

Biometrical genetics is a theoretical framework designed to estimate genetic and environmental influences that result in observed differences between individuals. While it has long been debated whether nature or nurture is responsible for these differences, the development of biometrical genetics has provided researchers with the opportunity to empirically investigate the joint contribution of these factors. This framework demonstrates that genetic influences are comprised of additive, dominant, and epistatic effects, while environmental influences are decomposed into shared and unique environmental effects.

Genetic and environmental contributions to variation in human traits are commonly assessed through structural equation modeling (SEM) or regression-based analytical techniques. SEM can be employed to estimate these effects for a particular trait using principles derived from biometrical genetics to specify the model. A benefit of the SEM approach is that it is highly versatile and can be extended to test various hypotheses (Neale, 2009). The most basic model is univariate, which decomposes the variance of a single trait into genetic and environmental components. One extension of the univariate model is the sex-limitation model, which is useful for testing for sex differences among genetic and environmental contributions to variation in a trait. A popular regression-based approach, the DeFries-Fulker (DF) method, can be used alternatively or in addition to some SEM models. While less complex than SEM, it also allows the genetic and environmental influences among the extremes of a distribution to be estimated.

It is essential to begin the chapter with a detailed overview of biometrical genetics that serves as the foundation on which the analytical strategies are formulated. This discussion will
then be followed by a brief overview of SEM, with a particular emphasis on the univariate and sex-limitation models that will be used to estimate genetic and environmental influences on variation in response to risk factors. The third section provides an explanation of the DF method. Because DF regression makes it possible to isolate and examine extreme cases, this technique will be used to investigate the etiology of vulnerability and resilience to risk factors separately. Finally, the chapter will conclude with a concise review of the assumptions underlying biometrical genetic modeling.

**Biometrical Genetic Modeling**

**Historical Development**

The work of Gregor Mendel in the 19th century has served as the foundation of modern studies of heredity. Through the study of pea plants, he derived three laws of genetics: segregation, dominance, and independent assortment. Mendel’s first law, segregation, states that hereditary factors are discrete (Carey, 2003; Neale & Cardon, 1992). Each organism possesses two alleles, one of which will randomly be transmitted to an offspring. Mendel’s law of dominance refers to the interaction between two alleles at the same locus (i.e., location of a gene on a chromosome) where one allele is expressed and the other is not. Mendel’s third law, independent assortment, asserts that discrete genetic factors for a single trait are transmitted independently of those transmitted for another trait.

Mendelian laws were initially applied to discrete traits (e.g., color of peas), but were later used by Ronald Fisher in his formation of the polygenic model (Fisher, 1918). The basic premise of this model is that a single observable trait, or phenotype, may be influenced by a number of genes, each of which follows Mendel’s laws. Fisher showed that the contribution of several genes to a phenotype generates several phenotypic categories. As the number of
influential loci increases, the number of phenotypic categories also increases. The distinction
between categories is clouded by environmental influences, giving the distribution a continuous
appearance (Evans, Gillespie, & Martin, 2002). For example, height may be influenced by
multiple genes at different loci, some that increase height and others that decrease height. The
combination of these genes within an individual will primarily determine his or her height, and
variation in genotypes will generate differences in height between people. Those with the same
genotype would be expected to have the same height; however, this phenotype may also be
influenced by environmental factors, such as nutrition. Since environmental factors are unlikely
to be precisely the same across all individuals, there will be slight variation among individuals
with the same genotype (Carey, 2003). The distribution of the majority of biological and
behavioral traits among humans will approximate a normal curve, making it possible to
decompose the phenotypic variance into genetic and environmental components through the
analysis of the covariance between related individuals (Evans et al., 2002; Fisher, 1918; Mather

Decomposition of Phenotypic Variance

An individual’s phenotypic value is his or her score on a given scale. The phenotypic
value ($P$) results from both genotypic and environmental influences:

$$P = G + E$$

(Eq. 1)

where $G$ is the genotypic value and $E$ is the environmental value. A genotypic value is the
average phenotypic value for all individuals with a particular genotype (Evans et al., 2002;
Neale, 2009). Using the previous example, the genotypic value for height would be the average
height of all people with the same combination of influential genes. Fisher (1918) demonstrated
Figure 3.1. A biallelic single locus in a randomly mating population. The genotypic effect of the \(bb\) genotype is \(-a\). The genotypic effect of the \(BB\) genotype is \(a\). The \(Bb\) genotype can fall on either side of 0 and the genotypic effect of \(Bb\) is \(d\), which is the degree of dominance at the locus. In this figure, the value of \(d\) would be negative. (Source: Neale, 2009 with kind permission from Springer Science+Business Media B. V.).

that the genotypic value can be further decomposed into additive genetic effects (\(a\)) and dominant genetic effects (\(d\)).

To illustrate, suppose that for a single locus associated with height there are two possible alleles, \(B\) and \(b^2\) (see Figure 3.1). \(B\) is known to increase height, and \(b\) to decrease height. The existence of two alleles would allow for three different allelic combinations, or genotypes. Both the \(bb\) and \(BB\) genotypes are considered homozygous because they each have two copies of the same allele. The \(Bb\) genotype is considered heterozygous because it is comprised of two different alleles. For each genotype, a mean phenotypic score (i.e., genotypic value) can be calculated. Furthermore, a grand mean, the distance between the two homozygous genotypes, and the deviation of the heterozygous genotype from the grand mean can be determined. The genotypic value for \(bb\) is \(\mu - a\) (one’s height is less than average), while the genotypic effect of \(BB\) is \(\mu + a\) (one is taller than average).

\(^2\) This example is illustrative of gene action at a single locus with only two alleles; however, it should be noted that it is possible that more than two alleles exist at a single locus. Also, as previously stated, it is commonly assumed that many phenotypes are polygenic and that the effect of each locus is relatively small (Neale, 2009). When this occurs, the effect at each locus contributes to the overall phenotypic value that is observed for each individual.
The genotypic effect of $Bb$ depends on the degree of dominant gene action for the individual alleles, and is $\mu + d$. If neither $B$ nor $b$ is dominant, the alleles are assumed to act additively and $d$ will equal 0. When this occurs, the genotypic effect of $Bb$ is half the sum of the genotypic effect of the homozygous genotypes (Evans et al., 2002; Neale & Cardon, 1992). When the value of $d$ is greater than 0 the $B$ allele has a dominant effect over $b$, and when $d$ is less than 0 the $b$ allele has a dominant effect over $B$. If an allele has complete dominance, the genetic effect of the heterozygous genotype will equal that of the genotype that is homozygous for that allele. For example, if $B$ had complete dominance over $b$, the average height of individuals with the $Bb$ genotype would equal that of those with the $BB$ genotype.

Estimates of $\mu$, $a$, and $d$ could be acquired through the collection of genotypic and phenotypic measures from a representative sample of the population of interest. It is important to note that variation in a phenotype observed in a population depends on $a$, $d$, and the frequency that each allele is observed (Neale & Cardon, 1992). If, for example, $B$ did have complete dominance over $b$, over time there would be less variation in height for the given population.

A simple Punnett square can be used to determine the proportions of genotypes that can be expected to be observed in a population in which mating is random. Keeping with the previous example, the Punnett square shown in Table 3.1 displays the possible allelic frequencies when both parents possess a heterozygous genotype. According to Mendel’s law of segregation, each parent will pass one allele randomly to an offspring each time the parents reproduce. Probabilistically, each allele would then have a 50% chance of being passed from parent to offspring. This produces the three genotypes $BB$, $Bb$, and $bb$, with the probabilities of their occurrence (i.e., genotypic frequencies) being .25, .5, and .25, respectively. Assuming mating is random, and that there are no population changes (e.g., mutation, natural selection), these allelic
Table 3.1 Punnett square illustrating all possible genotypes and the probability of their occurrence that results from the mating of two parents possessing a \( Bb \) genotype

<table>
<thead>
<tr>
<th>Female Parent</th>
<th>Male Parent</th>
</tr>
</thead>
<tbody>
<tr>
<td>( B(0.5) )</td>
<td>( B(0.5) )</td>
</tr>
<tr>
<td>( b(0.5) )</td>
<td>( B(0.5) )</td>
</tr>
<tr>
<td></td>
<td>( BB(0.25) )</td>
</tr>
<tr>
<td></td>
<td>( Bb(0.25) )</td>
</tr>
<tr>
<td></td>
<td>( Bb(0.25) )</td>
</tr>
<tr>
<td></td>
<td>( bb(0.25) )</td>
</tr>
</tbody>
</table>
| and genotypic frequencies will remain constant across generations. This is known as Hardy-Weinberg Equilibrium.

Genotypic frequencies are useful for the calculation of the population mean and variance of a particular phenotype, as demonstrated here:

<table>
<thead>
<tr>
<th>Genotype ((i))</th>
<th>( BB )</th>
<th>( Bb )</th>
<th>( bb )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency ((f))</td>
<td>( \frac{1}{4} )</td>
<td>( \frac{1}{2} )</td>
<td>( \frac{1}{4} )</td>
</tr>
<tr>
<td>Mean ((x))</td>
<td>( a )</td>
<td>( d )</td>
<td>(-a)</td>
</tr>
</tbody>
</table>

These values can then be used to estimate the average effect of the locus on the phenotype of interest \( (\mu_A) \) using the following formula:

\[
\mu_A = \sum f_i x_i
\]

\[
= \frac{1}{4} a + \frac{1}{2} d - \frac{1}{4} a
\]

\[
= \frac{1}{2} d.
\]

(Eq. 2)

The variance of the genetic effects \( (\sigma_A^2) \) around the mean can be calculated by taking the sum of the products of the frequencies of the genotypes and their squared deviations from the population mean, as demonstrated in the following formula:
The total variance then, for a single locus with equal allele frequencies, is the sum of its additive genetic variance \( V_A = \frac{1}{2} a^2 \) and dominant genetic variance \( V_D = \frac{1}{4} d^2 \). These two genetic components of variance can also be calculated for phenotypes in which more than one locus is influential; however, the specification of the additive and dominant components must be slightly altered. Following Mendel’s third law, independent assortment, the individual effects of each locus are summed in the calculation of the mean and the variance, as shown in equations four and five below.

\[
\mu = \frac{1}{2} \sum_{i=1}^{k} d_i
\]

(Eq. 4)

\[
\sigma^2 = \frac{1}{2} \sum_{i=1}^{k} a_i^2 + \frac{1}{4} \sum_{i=1}^{k} d_i^2
\]

\[
= V_A + V_D
\]

(Eq. 5)

Recall that the formulae previously denoted apply under circumstances in which allelic frequencies are equal in the population. However, the additive and dominant genetic effects can be redefined to account for unequal allele frequencies and differences in allelic frequencies.
across polygenic loci (Neale & Cardon, 1992). Consider again the three genotypes, $BB$, $Bb$, and $bb$. The genotypic frequencies in the population are $P$, $Q$, and $R$, respectively. The allelic frequency of $B = P + \frac{Q}{2} = p$, and the allelic frequency of $b = R + \frac{Q}{2} = q$ (see Table 3.2). These frequencies will also be in Hardy-Weinberg equilibrium, and the mean and variance\(^3\) are then obtained using equations six and seven.

\[
\mu = p^2a + 2pqd - q^2a = (p - q)a + 2pqd
\]

(Eq. 6)

\[
\sigma^2 = p^2a^2 + 2pqd^2 + q^2a^2 - [(p - q)a + 2pqd]^2
\]

\[
= p^2a^2 + 2pqd^2 + q^2a^2 - [(p - q)^2a^2 + 4pqad(p - q) + 4p^2q^2d^2]
\]

\[
= p^2a^2 + 2pqd^2 + q^2a^2 - [(p^2 - 2pq - q^2)a^2 + 4p^2q^2d^2]
\]

\[
= 2pq[a^2 + 2(q - p)ad + (1 - 2pq)d^2]
\]

\[
= 2pq[a^2 + 2(q - p)ad + (q - p)d^2 + 2pqd^2]
\]

\[
= 2pq[a + (q - p)d]^2 + 4p^2q^2d^2
\]

(Eq. 7)

In the preceding equation for the variance (Eq. 7), the additive genetic variance is defined as

\[
2pq[a + (q - p)d]^2
\]

and the dominance genetic variance is defined as

\[
4p^2q^2d^2
\]

\(^3\) Because the calculation of the mean for uneven allele frequencies is more complex than the previous formula, it is considerably more difficult to sum weighted deviations from the mean in order to calculate the variance. As a result, the formula for the variance has been rearranged.
These definitions are used for $V_A$ and $V_D$ because it is possible to regress genotypic effect on gene content. Gene content refers to the number of copies of an allele, or the “dose” that is present in a particular genotype. As demonstrated in Table 3.2, the gene content for the $bb$, $Bb$, and $BB$ genotypes are 0, 1, and 2, respectively. Using the values presented in Table 3.2, the means and variances, as well as the covariance of gene content and the genotypic effect, can be calculated. These values can then be used to define the slope as:

$$\beta_{yx} = \frac{\sigma_{xy}}{\sigma^2_x}$$

$$= \frac{2pq[a + (q - p)d]}{pq}$$

$$= a + (q - p)d.$$  

(Eq. 8)

The variance in the genotypic effect can then be partitioned into the variance due to the regression and the residual variance. The variance due to the regression is the additive genetic component of variance, and is that which is passed from parent to offspring (Evans et al., 2002). The residual variance is the dominance component of the genetic variance, and is the result of an interaction between alleles at the same locus.

When only a single locus is at work, the total genetic variance is the sum of the additive and dominance components; however, it is possible that multiple loci may be contributing to a phenotype. When this occurs, a third type of variance may exist, known as epistasis (Neale & Cardon, 1992). Epistatic effects arise when there is an interaction between more than one loci. These interactions can occur between the additive effects at each locus, the dominant effects at each locus, or both. The epistatic variance is that which is not explained by additive and dominance components of variance. Although it is possible to estimate epistatic effects, they are typically not estimated (Eaves, 1988; Evans et al., 2002; Neale & Cardon, 1992).
The heritability of a trait is the proportion of the variation of the observed phenotype in a population that is accounted for by genotypic variation in the population. The combined additive and dominance components (i.e., the proportion of the total variance in a phenotype accounted for by genetic influences) are known as broad heritability ($H^2$) (Evans et al., 2002). Narrow heritability ($h^2$) accounts only for the additive genetic proportion of the variance. Since both $H^2$ and $h^2$ are proportions, they can range from zero to one (Carey, 2003). For example, a heritability estimate of zero for differential response to risk would indicate that genetic factors do not account for individual differences in responses. A heritability estimate of one, on the other hand, would indicate that variation in responses to risk factors is completely due to genetic effects. Again, it should be noted that it is possible that, through mechanisms such as natural selection or genetic mutation, populations may change over time. Therefore, the parameters $H^2$ and $h^2$ are specific to the populations from which they are derived.

Recall that the phenotypic value ($P$) is the sum of its genotypic and environmental parts ($G + E$). While it has already been made clear that the genotypic component can be further divided into additive and dominance effects, it is also important to note that the environmental component can be further divided into shared environmental ($\sigma^2_s$) and specific environmental ($\sigma^2_e$) parts. The shared environmental influences are those events and/or experiences that affect all members of a particular family. For example, members of the same family may all experience the same household socioeconomic status. Specific or unique environmental influences, however, include those experiences that are unique to each individual within the family. Examples of specific environmental influences may include experiences with peers, school, or work. It is these four parameters; additive genetic, dominant genetic, shared
environment, and unique environment that are estimated using behavioral genetic modeling techniques.

**Measuring Covariation**

The four variance components discussed previously are often estimated through the examination of kinship pairs. The classic twin design is useful in isolating the effects of genetic and environmental influences on a phenotype (Jinks & Fulker, 1970; Mather & Jinks, 1982). The aim of this design is to compare the degree of phenotypic resemblance of monozygotic (MZ; identical) twins that are reared together to that of dizygotic (DZ; fraternal) twins reared together. MZ twins share exactly the same genetic material, whereas DZ twins share on average half their genetic material. DZ twins are therefore no more similar to one another than non-twin siblings in terms of their genetic makeup; however, DZ twins share the same pre- and post-natal environments, just as MZ twins. This results in a unique “natural experiment” in which environmental influences are controlled between individuals within a twin pair regardless of zygosity. If MZ twins show a greater degree of similarity for a specific trait or behavior than DZ twins, it can only be explained as the result of greater genetic similarity.

In order to use data on related pairs of individuals such as twins, the principles described previously must be used to determine the covariances between relatives (Carey, 2003; Neale & Cardon, 1992). This is done to ensure that the model is not underidentified (discussed in more detail below). One method of computing the covariance between pairs of relatives is to begin by constructing a table such as Table 3.3. The first column lists all possible combinations of genotypes that could be observed in a pair. The next two columns display the additive and dominance effects of the genotypes for each of the individuals within the pair. The deviations of
Table 3.3 Genotypes, gene effects, deviations, cross-products, and frequencies for MZ, DZ, and unrelated sibling pairs (Source: Neale, 2009 with kind permission from Springer Science+Business Media B. V.)

<table>
<thead>
<tr>
<th>Genotype Pairs</th>
<th>Effects</th>
<th>Deviations</th>
<th>Product</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>$x_{1i}$</td>
<td>$x_{2i}$</td>
<td>$x_{1i} - \mu$</td>
<td>$x_{2i} - \mu$</td>
<td>$(x_{1i} - \mu)(x_{2i} - \mu)$</td>
</tr>
<tr>
<td>$BB$</td>
<td>$BB$</td>
<td>$a$</td>
<td>$a - d/2$</td>
<td>$a - d/2$</td>
</tr>
<tr>
<td>$BB$</td>
<td>$Bb$</td>
<td>$a$</td>
<td>$d$</td>
<td>$a - d/2$</td>
</tr>
<tr>
<td>$BB$</td>
<td>$Bb$</td>
<td>$a$</td>
<td>$-a$</td>
<td>$a - d/2$</td>
</tr>
<tr>
<td>$Bb$</td>
<td>$BB$</td>
<td>$d$</td>
<td>$a$</td>
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each of these effects from the grand mean are shown in the next two columns. The product of the deviations is listed next, followed by the population frequencies of MZ, DZ, and unrelated sibling pairs. Since the genotypes of MZ twins will always be identical, there are only three possible genotypes pairs (i.e., $BB$, $Bb$, and $bb$) and their frequency is equal to that of each genotype (i.e., $p^2$, $2pq$, and $q^2$). The frequencies for unrelated siblings are the product of each genotype as they are paired at random. The population frequencies for DZ twins are calculated by listing all possible parental types and the proportions for all paired genotypes they can produce. The proportions are then summed for each frequency for each pair. Once these values are obtained, the covariances for each type of sibling pair is calculated by taking the frequency-weighted sum of the cross-products as shown below in equations 9-11:

$$\text{Cov (MZ)} = 2pq[a + (q - p)d]^2 + 4p^2q^2d^2$$

$$= V_A + V_D$$

(Eq. 9)

$$\text{Cov (DZ)} = pq[a + (q - p)d]^2 + p^2q^2d^2$$

$$= \frac{1}{2}V_A + \frac{1}{4}V_D$$

(Eq. 10)

$$\text{Cov (U)} = 0$$

(Eq. 11)

Once the expected means, variances, and covariance are determined, the values can be used to estimate genetic and environmental contributions to a phenotype using SEM.

**Structural Equation Modeling**

SEM is a statistical method commonly used to analyze twin and family data. In SEM, a number of structural equations can be derived to examine the relationship between several latent (i.e., unobserved) and observed variables. With regard to genetic analyses, the four variance
components; additive genetic, dominance genetic, shared environment, and unique environment, are latent variables since it is not possible to measure these factors directly. The phenotype of interest, differential response to risk for delinquency, is the observed variable in this study.

With SEM, it is possible to display the model graphically using a path diagram. In a path diagram the latent measures are drawn as circles and the observed measures are drawn as squares. These measures are connected by two different types of paths: causal, shown as a single-headed arrow from the causal variable to the outcome variable; and correlational, shown as a double-headed arrow between two related measures. A double-headed arrow from a single measure to itself is often used to indicate the variance of the measure. These path diagrams can then be used to determine the predicted variances and covariances between measures through path tracing\(^4\) (Neale & Cardon, 1992).

In order to use the path tracing approach, a set of rules must be followed (Neale & Cardon, 1992; Neale, 2009). First, begin at one of the variables of interest and trace backwards along any number of single-headed arrows. If a double-headed arrow is encountered, change direction, and then continue to trace forward along single-headed arrow paths to the variable of interest. Second, multiply all the paths along the chain of paths to determine the path effect. These procedures must be repeated for all unique chains. Finally, the path effects can be summed to identify the predicted covariance (Neale & Cardon, 1992).

Once the structural equation model is specified and the expected covariance is obtained, the remaining parameters, or pathway coefficients, can be estimated by fitting a model that uses raw data. The difference between the predicted and observed covariance are quantified, and a measure of how well the model fits the raw data is produced. The significance of each of the

---

\(^4\) It is also possible to calculate the expected values through matrix specifications.
parameters can be tested by fixing the pathway to zero and comparing the fit of the model to the full model.

As with any statistical technique, several assumptions underlie the application of SEM, several of which are common to multiple regression (Kline, 2011). First, the relationship between predictors and outcomes are assumed to be linear, as opposed to another functional form. Second, the residuals are assumed to be normally distributed and homoscedastic (i.e., the variances are equal across all levels of the predictors). If the data is not normally distributed or there are influential outliers, this assumption may be violated. Third, to avoid obtaining biased estimates, there should be no measurement error in the predictors. Fourth, any predictors not included in the model are assumed to be uncorrelated with those that are included. Failing to include all relevant variables results in specification error, and can also bias the estimates.

In addition to these assumptions, the researcher must correctly specify the directionality of the relationship (Kline, 2011, 2012). Directionality may be problematic when the true direction of the relationship between a predictor and the outcome variable is in the opposite direction, or a reciprocal relationship exists in which the two variables are causes and effects of each other (Kline, 2012). With regard to behavioral genetic models, these relationships are specified based on biometrical genetic theory. Variation is hypothesized to result from genetic and environmental differences between individuals.

A final concern in SEM is identification (Kline, 2012). A model is considered identified when it is possible to generate a single estimate for each parameter in the model. Briefly, a model must be just identified or overidentified, but it cannot be underidentified. A model is just identified when the number of parameters to be estimated is equal to the number of observed statistics, and it is overidentified when the number of observed statistics exceeds the number of
parameters to be estimated. When there are fewer observed statistics than estimated parameters the model is considered underidentified. This is particularly problematic because any number of solutions could be applied to an equation, and attempts at estimating parameters will fail (Kline, 2012).

**Univariate Model**

The univariate model is a basic application of SEM to the analysis of twin and family data. The purpose of this design is to derive estimates of the genetic and environmental influences on a single phenotype (see Figure 3.2). All four variance components are included in the path diagram shown for this model because, according to biometrical genetic theory, each of these factors could contribute to variation in the population. It is important to note, however, that when using a classical twin design the dominance and shared environmental components cannot be estimated simultaneously (Neale & Cardon, 1992). The reason for this is that information for these four variance components is derived from three observed statistics, the overall phenotypic variance, the covariance between MZ twins, and the covariance between DZ twins. As it is not possible to estimate four variance components using three observed statistics, a decision must be made about which variance components to estimate. It can be shown that dominance effects inflate the correlation between MZ twins relative to DZ twins, and conversely, shared environmental effects inflate the correlation between DZ twins relative to MZ twins (Martin et al., 1978). According to biometrical genetic theory, the expected covariance for MZ and DZ twin pairs is:

\[
\text{cov}_{MZ}(x, y) = \sigma_A^2 + \sigma_D^2 + \sigma_C^2
\]

(Eq. 12)

\[
\text{cov}_{DZ}(x, y) = 0.5\sigma_A^2 + 0.25\sigma_D^2 + \sigma_C^2.
\]

(Eq. 13)
Figure 3.2 Path diagram of a univariate model for MZ and DZ twins. The observed phenotypes \((P_1, P_2)\) are caused by a linear combination of the latent variables A (additive genetic), D (dominant genetic), C (common environment), and E (unique environment). The path coefficients \(a, d, c,\) and \(e\) are estimated. According to biometrical genetic theory, the correlation between additive genetic effects between pairs \((\alpha)\) is 1 for MZ twins and 0.5 for DZ twins. The correlation between dominant genetic effects between pairs \((\beta)\) is 1 for MZ twins and 0.25 for DZ twins. The correlation between shared environmental effects is 1 for both MZ and DZ twin pairs. The unique environmental effects are, by definition, not correlated between twins.
If these covariances are expressed as a ratio, the weighted average of each term can be shown:

\[
\frac{\text{cov}_{DZ}}{\text{cov}_{MZ}} = 0.5 \frac{\sigma_A^2}{\text{cov}_{MZ}} + 0.25 \frac{\sigma_D^2}{\text{cov}_{MZ}} + 1 \frac{\sigma_C^2}{\text{cov}_{MZ}}.
\]

(Eq. 14)

The weighted terms indicate that if additive genetic influences are the only contribution to the similarity between twins, the ratio would be exactly one half. If the ratio is greater than one half, shared environmental influences must be contributing to the observed similarity because this term increases the ratio toward one. However, if the ratio is less than one half, it indicates that a dominance effect is occurring because this will decrease the ratio toward one quarter. The information can then be used to determine whether it is more appropriate to estimate the effects of genetic dominance or shared environment.

The simplest way to make this determination is to examine cross-twin correlations, which are calculated by correlating the score of one twin with their co-twin. If the correlation between MZ twins is less than twice the DZ correlation, shared environmental effects should be estimated (i.e., an ACE model). On the other hand, if the correlation between MZ twins is more than double the correlation between DZ twins, dominant genetic effects should be estimated (i.e., an ADE model; Grayson, 1989).

Although both dominant genetic and shared environmental effects cannot be estimated simultaneously using a sample of MZ and DZ twins reared together, this does not suggest that both of these factors are not contributing to the phenotypic variance. In order to estimate both effects, additional data must be included. Suitable data may come from twins reared apart (Jinks & Fulker, 1970) or from other relatives, such as parents of twins (Fulker, 1982).

Once the appropriate model has been selected, a fully saturated model is estimated in which the correlation between the latent variables is specified according to biometrical genetic
theory (Neale & Cardon, 1992). As can be seen in Figure 3.2, the correlations for additive and dominant genetic effects are 1 for MZ twin pairs, and .5 and .25 for DZ pairs, respectively. The correlation between shared environmental effects is fixed to 1 for both MZ and DZ twin pairs, and the unique environmental effects are left uncorrelated. The regression coefficients $a$, $d$ or $c$, and $e$ are free to vary, but are assumed to be the same for both members of a twin pair. The mean phenotypic value and the variances are assumed to be equal across members of a twin pair, as well as between MZ and DZ pairs, because they are sampled from the same population. These assumptions are tested by fitting the full model and two submodels, one in which the means and variances are equated across members of a pair, and a second model in which the means and variances are equated across zygosity. The goodness of fit across these models is determined by examining chi-squared ($\chi^2$) statistics and the Akaike Information Criterion (AIC) value (Akaike, 1987). If placing these constraints on the model does not significantly reduce the fit as indicating by a non-significant p-value, it can be concluded that the assumptions have not been violated.

Once the assumptions of the equality of means and variances have been tested, it is also possible to test the significance of each of the variance components (Neale & Cardon, 1992). Similar to the process by which the equality assumptions are tested, a series of nested models are estimated in which the various parameter estimates are constrained to zero and the change in model fit is assessed. For an ACE or ADE model, the significance of shared environment or dominant genetics is tested by constraining the parameter to zero and estimating the effects of the remaining parameters (i.e., AE model). When estimating an ACE model, the significance of additive genetic effects can be tested by setting the $a$ parameter to zero and estimating the remaining two parameters (i.e., CE model). When estimating an ADE model, the $a$ parameter
cannot be fixed to equal zero because dominant genetic effects cannot be fully responsible for variation within a trait; therefore, a DE model should not be estimated. A third submodel is then estimated in which the combined significance of additive genetic and either dominant genetic or shared environment is tested (i.e., E model). The $e$ parameter is never dropped because this component captures any error in the model, and fixing this parameter to zero would result in a model that predicts perfect twin pair correlations for MZ twins (Neale & Cardon, 1992). The relative goodness of fit is assessed by examining the chi-square and AIC values. Again, if placing the constraints on the model does not significantly reduce the fit of the model, a nonsignificant p-value will be observed. In the event that multiple nested models do not reduce the fit, the model with the lowest AIC value is determined to be the best-fitting model. Finally, the parameter estimates for the best-fitting model can be standardized to produce easily interpretable values of the contribution each latent factor has on the variance of the phenotype.

**Sex-Limitation Model**

The basic univariate model can be extended to empirically test whether qualitative and/or quantitative differences exist across males and females (Eaves, Last, Young, & Martin, 1978; Neale & Cardon, 1992). Qualitative differences indicate whether or not the genetic and environmental factors that influence a phenotype are the same for males and females. Quantitative differences are examined to determine if the magnitude of the effects differ between sexes.

In order to examine qualitative and quantitative differences across sex, the sample of twins must be classified by both zygosity and sex. As a result, the following five groups can be compared: MZ males ($\text{MZ}_m$), MZ females ($\text{MZ}_f$), DZ males ($\text{DZ}_m$), DZ females ($\text{DZ}_f$), and DZ opposite-sex ($\text{DZ}_o$) pairs. The intraclass correlation coefficients can be calculated for each of
these groups, which are used as preliminary indicators of sex differences (Neale & Cardon, 1992). If the intraclass correlations differ between male and female pairs, this suggests that there may be quantitative differences between sexes. Furthermore, if the intraclass correlation observed among DZ_o pairs is considerably less than the correlations observed for DZ_m and DZ_f pairs, this indicates that qualitative differences between males and females may exist.

The full sex-limitation model incorporates all five types of twin pairs and permits quantitative and qualitative sex differences (Hatemi, Medland, & Eaves, 2009; Heath, Jardine, & Martin, 1989; Neale & Cardon, 1992). As shown in Figure 3.3, the correlations between additive genetic effects and common environmental effects are permitted to vary based on pair type and the $a$, $c$, and $e$ parameters are estimated separately for males and females. Similar to the univariate model described previously, the sex limitation model assumes additive genetic effects to have a correlation of 1 for MZ twin pairs and .5 for same-sex DZ twin pairs.

The inclusion of opposite-sex DZ twin pairs is key to examining sex effects because these pairs are exposed to the same home environments, but experience these conditions as members of the opposite sex (Hatemi et al., 2009; Neale & Cardon, 1992). Therefore, the additive genetic correlation between DZ_o pairs could range from 0, indicating completely different genes are expressed in males and females, to .5 if there are no qualitative sex differences. This can be assessed by first estimating a model in which $r_g$ for DZ_o pairs is free to vary between 0 and .5. To avoid underidentification, the $r_g$ and $r_c$ parameters cannot be estimated simultaneously. Instead, the $r_c$ must be fixed to 1 when the additive genetic correlation is being tested. Next, a nested model is estimated in which $r_g$ is fixed to .5, just as for same-sex DZ pairs. This model is then compared to the model in which $r_g$ is estimated (Hatemi et al., 2009; Neale & Cardon,
Figure 3.3 Path diagram of a univariate ACE model for opposite-sex DZ twins. The path coefficients a, c, and e are estimated separately for males and females. The correlation between additive genetic effects ($r_g$) between pairs is 1 for MZ twins and 0.5 for same-sex DZ twins. The additive genetic correlation for opposite-sex DZ pairs will range from 0 to .5, depending on qualitative sex effects. The correlation between shared environmental effects ($r_c$) is 1 for both MZ and like-sex DZ twin pairs. The shared environmental correlation between opposite-sex DZ pairs will range between 0 and 1, depending on differences in common environmental experiences.
1992). If fixing the parameter does not reduce the fit of the model, it can be concluded that the same genetic influences are operating across sexes.

Additionally, it is possible to examine the extent to which the same shared environmental influences are operating in males and females for a phenotype of interest. In the full model, the $r_c$ parameter is fixed at 1 and the $r_g$ parameter is fixed to .5 for all same-sex DZ pairs, while $r_c$ is estimated and $r_g$ is fixed to .5 for opposite-sex DZ pairs (Neale & Cardon, 1992). A model in which the $r_c$ parameter is fixed to 1 for these pairs can then be compared to the full model. If the restricted model is not found to be significantly different from the full model, it can be concluded that the same shared environmental experiences are influencing the phenotype for males and females (Neale & Cardon, 1992).

In the models that test for qualitative differences, quantitative differences are permitted for males and females because the $a$, $c$, and $e$ pathways are not constrained to be equal for males and females (Neale & Cardon, 1992). In order to determine if quantitative differences exist, each of the pathways are equated across sex and the model is then compared to the full sex-limitation model. The magnitude of these effects can be assumed to be the same for males and females if equating the pathways does not significantly reduce the fit of the model.

**Regression-Based Behavioral Genetic Modeling**

DeFries and Fulker (1985, 1988) have put forth a multiple regression model (i.e., the DF method or DF analysis), that provides an alternative approach to the analysis of twin data. With this technique it is possible to estimate the heritability of a trait on a full sample of twins, although it is commonly used to estimate the heritability of a trait among cases at the extremes of a distribution. The DF method produces estimates equivalent to those obtained using SEM, but this approach does not require any specialized software and very little training for researchers.
already familiar with ordinary least squares regression (Cherney, DeFries, & Fulker, 1992; Labuda, DeFries, & Fulker, 1986; Rodgers & McGue, 1994; Smith & Hatemi, 2012).

Like models estimated using SEM, the DF model is based on the principles of biometrical genetics. The analysis involves regressing the score of one twin on their co-twin, and is based on the assumption that if a trait is influenced by genetic factors, the mean score of MZ twins will regress less toward the population mean than the mean score of DZ twins, as shown in Figure 3.4 (DeFries & Fulker, 1988). This is because greater genetic similarity between MZ twins would result in greater phenotypic similarity if genetic factors are at work. On the other hand, if shared environmental factors are influential, the mean scores of MZ and DZ twins will be similar to each other and will regress toward the population mean because both MZ and DZ twins are assumed to share these experiences with their co-twin to the same extent. In the event that a trait is influenced entirely by unique environmental experiences, the mean scores of MZ and DZ twins will not correlate with their co-twin and their means will regress to the population mean (DeFries & Fulker, 1988).

A basic DF regression model is based on the following equation:

\[ Y_1 = b_0 + b_1 Y_2 + b_2 R + e \]

(Eq. 13)

where \( Y_1 \) is the score of one twin for a given trait, \( Y_2 \) is the score for the other twin in the pair, and \( R \) represents the degree of genetic relatedness between the twins (MZs=1, DZs=.5). There are several methods of determining which twin will be entered as the dependent variable (\( Y_1 \)) and which the independent variable (\( Y_2 \)). First, the model may be fit to selected data. This method is commonly referred to as single-entry because each pair is entered as one case in the data, and one twin is selected as a proband (i.e., a twin selected due to a deviant or extreme score); this twin is entered as a predictor of their co-twin’s score (DeFries & Fulker, 1985, 1988).
Figure 3.4 Distributions with MZ and DZ co-twin means plotted relative to probands. The top left distribution displays the proportion of the population selected as probands based on exceeding a given cutoff value. The top right distribution displays the means for MZ and DZ co-twins relative to probands if genetic factors are influential. The bottom left distribution displays the co-twin means if shared environmental factors are influential. The bottom right distribution displays the co-twin means if unique environmental experiences are completely responsible for differences between twins.
A more common method is known as double-entry because each pair is entered as a case twice, once in which a twin is assigned to be the first twin and once in which the assignments are reversed (Cherney et al., 1992; LaBuda et al., 1986; Rodgers & McGue, 1994). This allows for each twin to be entered once as the dependent variable and once as the predictor. In an extremes analysis, this procedure allows both twins the opportunity to be selected as a proband.

As is obvious, this approach increases the value of N. Although double-entering the data will not bias the estimates, it does bias the standard errors (Cherney et al., 1992; Smith & Hatemi, 2012). Adjustments can be made to obtain accurate standard errors. A common correction is to multiply the standard errors by the square root of the degrees of freedom for the double-entered data divided by the degrees of freedom for the single-entered data, or \(\sqrt{2}\) (Cherney et al., 1992). It is also possible to implement a generalized method of moment (GMM), random effects, or Huber-White correction (Kohler & Rodgers, 2001; Smith & Hatemi, 2012).

Once the data are double-entered, extreme cases can be selected using a specified cutoff criterion (DeFries & Fulker, 1985, 1988). Ideally, the cutoff criteria would be determined based on previously established standards, such as meeting the diagnostic criteria for a disorder such as ADHD. When such a criterion has not been established, the researcher must decide what the cutoff score will be. Standard deviations (e.g., \(\leq -1\) SD; \(\geq +1\) SD) or percentiles (e.g., \(\leq 25^{th}\) percentile; \(\geq 75^{th}\) percentile) are often used as starting points in research. Pairs in which one twin meets the criteria are entered into the analysis once, pairs in which both twins meet the criteria are entered twice (each twin is the proband one time), and pairs in which neither meets the criteria are omitted.
After selecting probands, the data can be transformed so that $b_2$ from Equation 14 will estimate group heritability ($h_g^2$) directly (DeFries & Fulker, 1988; LaBuda et al., 1986). Rather than indicating the degree to which genetic factors account for individual differences in a trait, group heritability is an estimate of the proportion of the difference between the extreme group and the overall population that is due to genetic factors. All individual scores are transformed using the following formula:

$$\frac{(x - \bar{x}_0)}{(\bar{x}_1 - \bar{x}_0)}$$

(Eq. 15)

where $\bar{x}_0$ is the mean of the population and $\bar{x}_1$ is the mean of the probands (Purcell & Sham, 2003). To account for any differences that may exist between MZ and DZ proband means, the transformation should incorporate zygosity-specific means\(^5\). Transforming the data in this way results in a proband mean of one, a population mean of zero, and a co-twin mean between zero and one. The regression can then be conducted using the transformed data, and the significance of the group heritability estimate can be evaluated.

**Assumptions in Biometrical Genetics**

Both SEM and regression-based techniques founded on the principles derived from biometrical genetic theory rely on three central assumptions. The first is referred to as the Equal Environmental Assumption (EEA). According to the EEA, MZ pairs are believed to encounter the same degree of environmental similarity as DZ twin pairs, which is why the shared environmental components are correlated at one regardless of the zygosity of the pair in the models described here (Neale & Cardon, 1992; Scarr, 1968; Scarr & Carter-Saltzman, 1979). If MZ twins were treated more similarly than DZ pairs, perhaps due to greater physical similarity,

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\(^5\) When the means are not significantly different, it is possible to use the grand mean of the probands for all transformations.
then it is possible that any greater similarity observed among MZ pairs could be the result of these experiences rather than genetic similarity. However, in order for this to occur, the excess in similar treatment must be related to the trait of interest (Neale & Cardon, 1992). For example, dressing identical twins alike as young children is unlikely to be related to criminal behavior in adulthood, in which case the EEA would not be violated.

A second assumption of the classical twin design is that mating occurs at random (Neale & Cardon, 1992). Assortative mating occurs when mated pairs exhibit greater similarity for a given trait than would be observed by chance (Crow & Felsenstein, 1968). Although this may be observed for various reasons, if individuals choose mates that are phenotypically similar to themselves, it is possible that the mates are also more similar genetically. If this occurs, the genetic correlation assumed to be .5 among DZ pairs could, in fact, be greater (Neale & Cardon, 1992).

Finally, the third assumption underlying the classical twin design is that there are no gene-environmental interactions ($G \times E$) influencing the trait under examination (Eaves, Last, Martin, & Jinks, 1977; Jinks & Fulker, 1970; Neale & Cardon, 1992). This is apparent in the models presented here because no correlations are shown between genetic and environmental components in the path diagrams. When $G \times E$ interactions are present, various genotypes differ in their sensitivity to environmental conditions. This can be further investigated by including twin pairs in which both twins have been exposed to the particular environmental stimuli; twin pairs in which neither twin has been exposed; and twin pairs in which one twin has and the other has not been exposed (Evans, et al., 2002). It is also possible to explore the possibility of $G \times E$ interactions when genotypic data is available.
Conclusion

Statistical analyses based on biometrical genetics, such as SEM and DF-regression, allow for the empirical investigation of genetic and environmental influences on a variety of human traits. The literature reviewed in chapter two provides clear evidence of variation in how individuals respond to risk factors for delinquency; however, the extent to which genetic and environmental factors contribute to that variation is currently unknown. Knowledge pertaining to the degree of genetic and environmental influence can be used to guide the search for additional risk and protective factors, and perhaps explain some of the existing errors in prediction research.

A univariate model will be used to better understand the differential response to cumulative risk for delinquency. The variance in a continuous measure of responses, where vulnerable and resilient cases are found at opposite extremes, will be decomposed into genetic and environmental elements. The univariate model will be extended to test for both qualitative and quantitative sex effects in differential response to risk. Finally, many researchers have focused their attention on resilient youths but very few have examined those characterized as more vulnerable. It is possible that these two very different responses to risks have different origins. Therefore, the DF method will be employed to isolate both types of cases and investigate the genetic and environmental influences on these outcomes.
CHAPTER 4

RESEARCH METHODOLOGY

Research designs used to investigate genetic and environmental influences on behavior require the use of data that include kinship pairs. The National Longitudinal Study of Adolescent Health (Add Health) includes a subsample of related individuals and a broad range of measures that are reflective of the participants’ traits and behaviors, including delinquency and individual-level risk factors commonly found in criminological research. These data also include measures of the environment, such as family and neighborhood factors, to which subjects have been exposed. The aim of this chapter is to review the methodology employed in the original Add Health study, and the particular measures used in the current investigation.

The National Longitudinal Study of Adolescent Health

Data for this investigation come from the Add Health restricted-use files (Harris, 2009). Add Health is considered to be the most comprehensive prospective longitudinal study of American youth. To date, four waves of data have been collected over a span of 14-years (see Figure 4.1). The initial wave of data collection was conducted between September 1994 and December 1995 when participants were in grades 7 through 12. Wave II took place between April and August of 1996. Five years later, between August 2001 and April 2002, the third wave of data collection occurred. The final wave of data collection was conducted in 2007-2008, when participants were 24 to 34 years old.

The Add Health researchers administered questionnaires to youths, their parents, siblings, peers, and partners, and school administrators (Harris et al., 2009). This strategy allowed researchers to gather information regarding various aspects of each participant’s life during
adolescence and adulthood. The following section will provide an in-depth discussion of the research design and sampling techniques employed in the Add Health study and the various components of the data.

Research and Sampling Design

A multistage stratified random sampling method was used to select participants for the Add Health study (Harris et al., 2009). Initially, a sampling frame of 26,666 high schools listed
in the Quality Education Data, Inc. (QED) database was used to identify schools for inclusion in the study. Schools that were classified as either public or private with an eleventh grade and a minimum enrollment of 30 students were eligible for inclusion in the study. These schools were stratified into clusters based on the following criteria: (1) the enrollment size of the school (<125, 126-350, 351-775, or >775); (2) school type (public, private, or parochial); (3) geographic location (Northeast, South, West, or Midwest); (4) urbanicity (rural, urban, or suburban); and (5) percentage white students (0%, 1-66%, 67-93%, or 94-100%) (Tourangeau & Shin, 1999). A total of 80 high schools were then selected from these clusters, of which 28 declined to participate in the Add Health study. For each of the declining schools, an additional high school was selected from the same stratum as a replacement (Harris et al., 2009).

If a selected high school did not include a seventh or eighth grade, administrators were asked to identify middle schools that had a seventh grade from which at least five graduates were promoted to that particular high school each year (Harris et al., 2009). These institutions were known as “feeder” schools, and one feeder school for each high school was recruited to participate in the study (unless the high school spanned grades 7 through 12). A total of 56 feeder schools were selected to participate. The probability of a feeder school being selected was proportional to the percentage of incoming freshman that came from that feeder school (Harris et al., 2009). If a school opted not to participate, a replacement was then selected. These sampling procedures resulted in a total of 132 schools in the final sample.

**Data Collection**

Although the Add Health study has included four waves of data collection, the data used in this study come from only the first and third waves. Risk and delinquency measures were all
taken from the first wave of data collection, and genetic measures of zygosity from wave three. In light of this, only these waves are reviewed here.

**Wave I.** Youths that attended one of the 132 selected schools were eligible to participate in the in-school survey between September 1994 and April 1995, during Wave I of the Add Health study. Individuals in grades 7 through 12 obtained parental consent prior to participating. Researchers provided questionnaires that youth self-administered during a 45- to 60-minute class period. Students that were not present during the class period in which the questionnaire was administered did not participate. A total of 90,118 youth completed the in-school questionnaire, which included topics related to characteristics of the individual, the education and employment of parents, home and family dynamics, risk behaviors, expectations for the future and self-esteem, personal health, friendships, and activities (Chantala & Tabor, 1999).

All students enrolled in one of the sampled schools were eligible for selection to participate in an in-home interview during Wave I. Individuals in each school were stratified by grade level and gender. Approximately 17 youth were randomly selected from each stratum, generating a core sample of 12,105 youth (Harris et al., 2009). This core sample is a nationally representative sample of adolescents in grades 7 through 12 during 1994 and 1995.

In addition to the core sample, a number of special subsamples were also selected and included in the Wave-I in-home interviews (Harris et al., 2009). First, an ethnic subsample was selected based on self-reported information collected during the in-school questionnaire. The subsample included 1,038 African American youths that had a parent with a college degree, 334 Chinese youth, 450 Cubans, and 437 Puerto Ricans. Second, a disabled subsample of 589 students was also selected based on self-reported physical disabilities indicated on the in-school questionnaire. Third was a saturated subsample that included all students that were enrolled in
16 schools. This subsample was comprised of 2 schools with an enrollment of more than 3,300 students and 14 schools with an enrollment of 300 or less students. It was designed to provide researchers the opportunity to examine peer groups within a school setting. Fourth, a genetic subsample that included pairs of siblings that resided in the same households was selected (Harris et al., 2009). A total of 2,658 twins, 208 non-twin siblings of twins, 1,611 full siblings, 1,177 half siblings, and 491 youths that shared a household but were not genetically related (e.g., step-siblings, adopted children, foster children) were included in the genetic subsample.

The Wave I in-home interviews took place between April and December 1995 with a total of 20,745 adolescents (Chantala & Tabor, 1999). The in-home surveys addressed several topics including personal health status and care, aspirations for the future, education, employment experiences, romantic relationships, criminal behavior, substance use, peer networks, and family composition and dynamics (Harris et al., 2009). Interviews typically lasted one to two hours, and were conducted in the youth’s home using Computer-Assisted Personal Interview (CAPI) and Audio Computer-Assisted Self-Interview (ACASI) methods. When using CAPI, an interviewer would enter a participant’s response to questions into a secure laptop computer, allowing for greater efficiency and security. The ACASI method was used to collect information on topics of greater sensitivity, such as sexual relationships and activities, substance use, and delinquent behaviors. To maintain the participants’ privacy and reduce interviewer or parental influence, individuals were permitted to listen to pre-recorded questions through headphones and enter responses directly (Couper, Singer, & Tourangeau, 2003; Ghanem, Hutton, Zenilman, Zimba, & Erbelding, 2005).

During Wave I, data were also collected from parents or caregivers of the adolescents. A brief paper and pencil questionnaire was completed by 17,700 caregivers, typically the mother
These were used to gather information regarding heritable and other health conditions, marriages, neighborhood characteristics, involvement in volunteer and school activities, education and employment, family finances, parent-adolescent relations, and parental involvement with the peers of their children.

**Wave III in-home interview.** From August 2001 to April 2002, when participants were between 18 and 28 years of age, Add Health researchers administered the Wave III in-home interviews. A total of 15,170 subjects who had participated in the Wave I in-home interviews were included at Wave III. Participants that were outside of the United States at the time of the interviews were ineligible; however, researchers did attempt to interview individuals that were incarcerated. Of the 132 incarcerated participants, 29 were interviewed.

Questionnaires were administered using CAPI and ACASI methods, similar to the earlier waves (Harris et al., 2009). Many of the questions remained unchanged from the previous interviews, but additional topics were included at Wave III since all of the participants had reached adult status. Subjects were asked to report on various aspects of their upbringing and adult life, including relations with parents and siblings, history of maltreatment, marriage and cohabitation, sexual experiences, pregnancies, employment, physical and mental health, spirituality, substance use, and participation in criminal and violent behaviors.

Approximately half of the original sample was selected to be evaluated for partner recruitment, based on the relationship history provided by the subject. In order to be eligible, a partner had to be current, of the opposite sex, at least 18 years old, and in a relationship with the subject for a minimum of three months (Harris et al., 2009). A total of 1,507 romantic partners of Add Health respondents were interviewed during Wave III, of which one-third was married, one-third was cohabitating, and one-third was dating.
During Wave III, subjects who had indicated that they had a co-twin or full sibling, and were added to the genetic subsample during Wave I were eligible to participate in a DNA subsample. Researchers asked 3,787 participants to submit a saliva sample for genetic analysis, of which 2,574 complied. The DNA analysis was carried out by researchers at the University of Colorado and identified a number of genetic polymorphisms (i.e., a dopamine transporter gene, two dopamine receptor genes, a serotonin transporter gene, cytochrome P450, and monoamine oxidase). Researchers also used DNA to assess the zygosity of same-sex twin pairs.

**Analytical Sample**

Research investigating the genetic and environmental contributions to behavior requires the use of a sample of pairs of individuals of varying degrees of genetic relatedness. In order to meet this criterion, the analyses include all twin pairs that participated in the Add Health study as part of the genetic subsample. The Add Health data are available in a public-use format and a restricted-use format, which includes sensitive and confidential information not available in the public-use files. In order to access the genetic subsample, one must obtain the permission of the Inter-University Consortium for Political and Social Research (ICPSR) under the sponsorship of the Data Sharing for Demographic Research archive (DSDR). Researchers must sign a contract that stipulates how the data must be stored and analyzed. This investigator was granted access to the restricted-use Add Health data under the supervision of Dr. John P. Wright, and the contract was approved by the Institutional Review Board-Social and Behavioral Sciences at the University of Cincinnati.

Following a classical twin design, all non-twin sibling pairs were removed from the genetic subsample prior to analyzing the data. This left a final analytical sample of 1,568 individual twins (N=784 twin pairs), including 289 monozygotic (MZ) twin pairs, 452 dizygotic
(DZ) twin pairs, and 43 twin pairs with unknown zygosity (Rowe & Jacobson, 1998). Those pairs in which zygosity could not be determined were classified as dizygotic. This approach increases the likelihood of overestimating the environmental contributions to behavior, making it a more conservative method (Rowe & Jacobson, 1998). Individuals were randomly assigned as twin one or twin two, with the exception of the opposite-sex twin pairs in sex-limitation models where males were coded as twin one and females as twin two.

This subsample of twins is considered to be nationally representative based on the sampling techniques used by Add Health researchers (Harris, Halpern, Smolen, & Haberstick, 2006). As part of the in-school interviews during Wave I, randomly selected participants were asked whether or not they had a twin sibling. If a subject confirmed being a twin, his or her co-twin was then automatically asked to participate. Because participants were first selected through stratified random sampling techniques for the national sample, and not on the basis of being a twin, the representativeness in the twin subsample was maintained (Harris et al., 2006).

**Measures**

In order to investigate the genetic and environmental influences on responses to risk for delinquency, three measures are required. First, when analyzing twin and family data, a measure of genetic relatedness is needed for each pair of relatives. Because the current investigation is limited to a subsample of twins, the zygosity of each twin pair is used to measure the degree of genetic similarity. Second, a single measure that captures variation in behavioral responses to risk is needed. To obtain such a measure, standardized residual scores are derived from three ordinary least squares regressions with cumulative risk entered as a predictor and overall delinquency, violent delinquency, and nonviolent delinquency as dependent variables. Standardized residuals provide a method of establishing the degree of error in prediction, both in
terms of over- and underestimating delinquent behavior. Finally, a measure of sex is included to assess sex effects where appropriate (0 = female, 1 = male). This measure is based on self-reports during the in-school survey that were later confirmed by researchers during the in-home interview at Wave I.

**Zygosity**

During the Wave I in-school survey, individuals were asked whether or not they are a twin. Those that self-reported being a twin were automatically included in the genetic subsample and were asked to participate in the in-home interviews. Twins were asked if they were identical (i.e., MZ) or fraternal (i.e., DZ). All opposite sex pairs were classified as DZ, but if both members of a twin pair were the same sex, zygosity was determined based on responses to four questions that assessed their degree of similarity in terms of physical appearance and the frequency with which strangers, teachers, and family members confused one twin with the other (Rowe & Jacobson, 1998). An average score of confusability was created using responses from both twins, which was used to classify the majority of pairs as MZ or DZ.

Zygosity could not be determined for twin pairs with average confusability scores falling above the designated cut-off for DZ classification and below the cut-off for MZ classification. These pairs were classified as MZ or DZ based on DNA testing during Wave III. Individuals were asked to provide specimens for buccal cell DNA analysis. The samples were genotyped by researchers at the Institute for Behavioral Genetics at the University of Colorado to confirm the zygosity status of the twin pairs. A total of 11 unlinked short tandem repeat (STR) genetic markers (i.e., D1S1679, D2S1384, D3S1766, D4S1627, D6S1277, D7S1808, D8S1119, D9S301, D13S796, D15S652, D20S481) and a sex-determining locus (i.e., amelogenin) were compared between twin one and twin two in each pair (Harris et al., 2006). In order for a twin
pair to be classified as MZ, the twins were required to be 100% concordant on all genotypes at all 12 loci. This zygosity confirmation strategy indicated the correct zygosity for 16 pairs that had initially been incorrectly assigned and 18 pairs that had originally been classified as undetermined using questionnaire data. Furthermore, this technique demonstrated that 91% of respondents had correctly determined their zygosity (Harris et al., 2006).

**Cumulative Risk**

The risk factors included in the cumulative measure of risk are classified into three broad domains: individual, familial, and environmental. Previous research suggests that individuals are influenced by factors across domains (Bronfenbrenner, 1979; Tolan & Guerra, 1994) and that increases in the accumulation of risk factors are associated with increases in the probability of negative outcomes, including delinquent behavior (Fergusson & Lynskey, 1996; Kolvin, Miller, Fleetin, & Kolvin, 1988; Pollard, et al., 1999; Sameroff, et al., 1998; Stouthamer-Loeber, et al., 2002). A number of studies have used cumulative risk indices to examine the impact of exposure to multiple risk factors on delinquent and violent behavior during adolescence (Gerard & Buehler, 2004; Ribeaud & Eisner, 2010; Stoddard, et al., 2012).

Following previous research, each risk measure was dichotomized so that those individuals that were deemed at-risk for each item received a score of 1 and all others received a score of 0 (Apilayard, et al., 2005; Esbensen, Peterson, Taylor, & Freng, 2009; Farrington & Loeber, 2000; Gerard & Buehler, 2004; Sameroff, et al., 1998; Stouthamer-Loeber et al., 1993; Stouthamer-Loeber et al., 2002; van der Laan, et al., 2009; van der Laan, et al., 2010). Cutoffs were typically at or above the 75th percentile, or at or below the 25th percentile if the lower end of the distribution posed a greater risk. Although the threshold designating the presence of risk may vary across measures, such thresholds have not been empirically established. Therefore,
operationalizing risk using the 25th and 75th percentiles will capture risk at the extreme of a distribution without being so extreme that the risk is only present in a small number of cases (Stouthamer-Loeber, et al., 2002).

The dichotomized scores are then added to generate a cumulative risk score for each individual. To maintain the size of the sample, cases were required to have valid scores on at least 10 of the 14 variables. Descriptive statistics and cutoff criteria for all of these items are shown in Table 4.1. Bivariate correlations between all of the risk measures and all three measures of delinquency are shown in Table 4.2. For measures comprised of multiple items, a complete list of items used in the creation of each variable is presented in Appendix A.

**Individual-level risk items.** A total of seven measures are used to assess individual-level risk. These include school performance, attachment to school, intelligence, problem solving skills, coping skills, current marijuana use, and current cigarette use. Although previous research has also indicates that low self-control (Pratt & Cullen, 2000) and abuse or neglect (Hawkins, et al., 2000; Widom, 1989a, 1989b) may both be risk factors for delinquency, these measures were not be included in the cumulative risk score. Self-control was not measured in the original Add Health study, and items previously used to create a measure of self-control were used in the creation of other variables. A retrospective measure of abuse and neglect was included in the third wave of data collection; however, very few cases in the twin subsample reported a history of those experiences.
<table>
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<tr>
<th>Risk Measure</th>
<th>Range</th>
<th>M (SD)</th>
<th>Number of Items</th>
<th>Cronbach’s Alpha</th>
<th>Risk Criterion</th>
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<td>Individual-level items</td>
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<tr>
<td>School performance</td>
<td>1-4</td>
<td>2.19 (.76)</td>
<td>4</td>
<td>.79</td>
<td>≥ 2.75</td>
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<td>Attachment to school</td>
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<td>-.07 (5.38)</td>
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<td>.78</td>
<td>≥ 3.16</td>
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<td>Intelligence</td>
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<td>-</td>
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<tr>
<td>Problem solving</td>
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<td>8.89 (2.53)</td>
<td>4</td>
<td>.74</td>
<td>≥ 10</td>
</tr>
<tr>
<td>Coping skills</td>
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<td>8.35 (2.19)</td>
<td>3</td>
<td>.44</td>
<td>≤ 10</td>
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<td>Marijuana use</td>
<td>0-1</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Cigarette use</td>
<td>0-1</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
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<tr>
<td>Family-level items</td>
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<td></td>
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<td>Attachment to parents</td>
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<td>15.86 (4.15)</td>
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<td>.68</td>
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<td>Parental involvement</td>
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<td>-</td>
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<td>Parental engagement</td>
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<td>.88</td>
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<td>Parental supervision</td>
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<td>.61</td>
<td>≥ 20</td>
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<td>Environmental-level items</td>
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<td>Delinquent peers</td>
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<td>2.46 (2.64)</td>
<td>3</td>
<td>.76</td>
<td>≥ 4</td>
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<td>Social support</td>
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<td>.79</td>
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<td>Neighborhood safety</td>
<td>0-1</td>
<td>-</td>
<td>1</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>Cumulative Risk (N=1492)</td>
<td>0-12</td>
<td>3.74 (2.45)</td>
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<th>Risk Score</th>
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<th>5</th>
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<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
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<td>Distribution %</td>
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<td>16.2</td>
<td>14.1</td>
<td>14.0</td>
<td>14.8</td>
<td>12.3</td>
<td>8.6</td>
<td>6.2</td>
<td>4.2</td>
<td>2.1</td>
<td>1.5</td>
<td>.4</td>
<td>.1</td>
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<tr>
<td></td>
<td>X1</td>
<td>X2</td>
<td>X3</td>
<td>X4</td>
<td>X5</td>
<td>X6</td>
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<td>X13</td>
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<tr>
<td>X1</td>
<td>Overall Delinquency</td>
<td>1 (1.16**)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>X2</td>
<td>Violent Delinquency</td>
<td>.83** (.73**)</td>
<td>1 (.91**)</td>
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<tr>
<td>X3</td>
<td>Nonviolent Delinquency</td>
<td>.92** (.83**)</td>
<td>.54** (.47**)</td>
<td>1</td>
<td></td>
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<tr>
<td>X4</td>
<td>Cumulative Risk</td>
<td>.36** (.39**)</td>
<td>.27** (.28**)</td>
<td>.34** (.38**)</td>
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<tr>
<td>X5</td>
<td>School Performance</td>
<td>.22** (.26**)</td>
<td>.22** (.24**)</td>
<td>.18** (.20**)</td>
<td>.45**</td>
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<tr>
<td>X6</td>
<td>Attachment to School</td>
<td>.27** (.30**)</td>
<td>.24** (.25**)</td>
<td>.24** (.27**)</td>
<td>.47**</td>
<td>.31**</td>
<td>1</td>
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<tr>
<td>X7</td>
<td>IQ</td>
<td>-.06* (-.07*)</td>
<td>-.12** (-.12**)</td>
<td>-.01</td>
<td>-.02</td>
<td>-.24**</td>
<td>-.30**</td>
<td>-.04</td>
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<tr>
<td>X8</td>
<td>Problem Solving</td>
<td>.08** (.11**)</td>
<td>.02 (.04)</td>
<td>.10** (.12**)</td>
<td>.27**</td>
<td>.11**</td>
<td>.24**</td>
<td>-.05</td>
<td>1</td>
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<tr>
<td>X9</td>
<td>Coping Skills</td>
<td>-.11** (-.12**)</td>
<td>-.15** (-.16**)</td>
<td>-.06* (-.06*)</td>
<td>-.00</td>
<td>-.18**</td>
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<td>.29**</td>
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<td>1</td>
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<tr>
<td>X10</td>
<td>Marijuana Use</td>
<td>.31** (.28**)</td>
<td>.18** (.14**)</td>
<td>.33** (.32**)</td>
<td>.41**</td>
<td>.16**</td>
<td>.16**</td>
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<td>-.01</td>
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<tr>
<td>X11</td>
<td>Cigarette Use</td>
<td>.28** (.29**)</td>
<td>.20** (.20**)</td>
<td>.27** (.29**)</td>
<td>.43**</td>
<td>.18**</td>
<td>.21**</td>
<td>.03</td>
<td>.05*</td>
<td>-.05*</td>
<td>.37**</td>
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<tr>
<td>X12</td>
<td>Attachment to Parents</td>
<td>-.16** (-.19**)</td>
<td>-.13** (-.15**)</td>
<td>-.15** (-.18**)</td>
<td>-.65**</td>
<td>-.14**</td>
<td>-.16**</td>
<td>.15**</td>
<td>-.03</td>
<td>.08**</td>
<td>-.13**</td>
<td>-.09**</td>
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<tr>
<td>X13</td>
<td>Involvement</td>
<td>-.10** (-.11**)</td>
<td>-.09** (-.10**)</td>
<td>-.08** (-.09*)</td>
<td>-.47**</td>
<td>-.17**</td>
<td>-.09**</td>
<td>.18**</td>
<td>-.07**</td>
<td>.07*</td>
<td>-.06*</td>
<td>.51**</td>
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<tr>
<td>X14</td>
<td>Engagement</td>
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<td>.10** (.12**)</td>
<td>.16** (.19**)</td>
<td>.65**</td>
<td>.14**</td>
<td>.25**</td>
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<td>.17**</td>
<td>.14**</td>
<td>-.78**</td>
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<td>X15</td>
<td>Supervision</td>
<td>.08** (.12**)</td>
<td>.05* (.08**)</td>
<td>.08** (.12**)</td>
<td>.53**</td>
<td>.08**</td>
<td>.13**</td>
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<td>.06*</td>
<td>.06*</td>
<td>-.74**</td>
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<tr>
<td>X16</td>
<td>Peers</td>
<td>.37** (.37**)</td>
<td>.28** (.26**)</td>
<td>.36** (.36**)</td>
<td>.47**</td>
<td>.22**</td>
<td>.23**</td>
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<td>.47**</td>
<td>.53**</td>
<td>-.14**</td>
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<tr>
<td>X17</td>
<td>Social Support</td>
<td>-.22** (-.26**)</td>
<td>-.14** (-.18**)</td>
<td>-.23** (-.25**)</td>
<td>-.47**</td>
<td>-.19**</td>
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<td>.29**</td>
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<tr>
<td>X18</td>
<td>Neighborhood Safety</td>
<td>.07** (.08**)</td>
<td>.09** (.08*)</td>
<td>.05 (.05*)</td>
<td>.16**</td>
<td>.15**</td>
<td>.16**</td>
<td>-.14**</td>
<td>.03</td>
<td>-.04</td>
<td>.02</td>
<td>-.02</td>
<td>-.12**</td>
</tr>
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*p<.05
**p<.01

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**School performance.** A measure of academic achievement was created based on the average grades received in English, mathematics, history, and science during the most recent grading period. Individuals reported the letter grade they earned in each subject (1 = A, 2 = B, 3 = C, 4 = D or lower). If an individual did not take all of these subjects, or if the course was not graded using traditional letter grades, the average of available scores was used. Scores ranged from 1 to 4, with higher scores reflecting worse performance. The average for the subsample of twins was 2.19 (SD = .76). Individuals falling at or above the 75th percentile (2.75) were considered at-risk on the dichotomized measure included in the cumulative risk index.

**Attachment to school.** During the in-home interview at Wave I, nine questions asked adolescents to report how often they had trouble at school and the degree to which they felt connected to the school (α = .78). The response categories for four of the items ranged from 0 = never to 4 = everyday, while response categories for the remaining 5 items ranged from 1 = strongly agree to 5 = strongly disagree (see Appendix A). As a result of two set of response categories being employed, all items were standardized.

Factor analysis using principle components extraction (no rotation) indicated that a two factor model fit the data well (Kaiser-Meyer-Olkin (KMO) = .799; two eigenvalues greater than 1); however, further inspection of factor loadings suggested a single factor may provide a good fit. Because nearly all of the items produced a higher loading on the first factor⁶, a second factor analysis was conducted in which the number of factors was fixed to one. The results of this analysis were virtually identical to the first model. Additionally, the reliability analysis indicated that removing any item from the scale would reduce Cronbach’s alpha. Therefore, all items were summed to create a single measure of attachment to school. Scores among the twin subsample

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⁶ One item (“How often did you have trouble getting your homework done?”) produced a slightly greater loading on the second factor (.575) than on the first factor (.519).
range from -10.30-21.52, and the mean is -.07 (SD = 5.38). This measure is dichotomized for the cumulative risk index so that participants with a score of 3.16 (75<sup>th</sup> percentile) or greater are considered at-risk.

**Intelligence.** Participants of Add Health were administered the Add Health Peabody Picture Vocabulary Test (PPVT), which is an abridged version of the PPTV-Revised. During testing, an interviewer would read a word to the participant who would then select an illustration from four choices that best reflects the meaning of the word. Raw scores are included in the analysis and range from 0-87, with a mean of 63.48 (SD = 10.7). Individuals that scored at or below 57 (25<sup>th</sup> percentile) are considered at-risk, and were given a score of 1 on the dichotomized measure.

**Problem solving and coping skills.** During the in-home interviews at Wave I, participants were asked to indicate the degree to which they agreed with seven statements relating to how they respond to problems using a 5-point Likert scale (1 = strongly agree, 5 = strongly disagree). Factor analysis revealed that a two factor model fit the data (KMO = .74, two eigenvalues greater than one). Inspection of the factor loadings suggested that the first factor was tapping into *problem solving skills*. This factor was comprised of four items that related to one’s strategy or approach to solving a problem (factor loadings ranged from .71-.78). Responses to these four items were summed to create a measure of problem solving skills (α = .74). Scores range from 4-20, and the mean is 8.89 (SD = 2.53) with higher scores reflecting greater difficulty solving problems. Youth that scored at or above 10 (75<sup>th</sup> percentile) on this measure are considered at-risk.

A second factor was extracted that included three items relating to how one deals with problems emotionally. These items asked youths to indicate whether they avoid dealing with
problems, if they are upset by problems, or if they make decisions based on “gut feelings” rather than thinking about the consequences of the alternatives. To create a measure of coping skills, these three measures were combined (α = .44) so that higher scores indicated better coping. Although the reliability of this scale is low, bivariate correlations with each measure of delinquency are in the expected direction and significant (-.06 < r < -.16), suggesting that this measure has predictive validity. The range of scores is 3-15, with a mean of 8.35 (SD=2.19). Youths with a score at or below 10 are considered at-risk, and received a score of 1 for this item on the cumulative risk index.

**Marijuana use.** At Wave I participants were asked a series of questions regarding the use of marijuana. Those that had confirmed using marijuana at least once in their lifetime were asked, “During the past 30 days, how many times did you use marijuana?” Responses ranged from 0-900. Among the subsample of twins, 87% reported that they had either never used marijuana, or that they had not used in the past 30 days. These individuals received a score of 0 (not a current marijuana user) on the cumulative risk index. A total of 13% indicated that they had used within the previous month, and received a score of 1 (current marijuana user).

**Cigarette use.** During the in-home interview at Wave I respondents were asked, “During the past 30 days, on how many days did you smoke cigarettes?” Responses ranged from 0 to 30 days. As a measure of current smoking behavior, the responses were recoded so that individuals who reported smoking cigarettes on one or more days received a score of 1 and those that indicated that they did not use cigarettes during the past 30 days received a score of 0.

**Family-level risk items.** Four measures are included to reflect family-level risk factors. These include attachment to parents, parental involvement, parental engagement, and parental supervision. It is important to note that for each of the four measures, items relating to both the
mother and father are included as opposed to constructing these variables separately for each parent. When the father was missing from the home, a score of 0 was entered for each of the paternal items. This approach has been used previously by Gerard and Buehler (2004) and offers three potential benefits. First, while many studies tend to emphasize the importance of maternal influence on the development of a child this strategy accounts for the importance of both parents. Second, including items relating to both parents in each variable provides a more accurate reflection of the parental environment. Adolescents with only one parent in the household will have lower scores on all items, while those with two parents will have higher scores indicating greater interaction with parents. Third, this approach assists in addressing the issue of missing data when no father figure is present in the household (approximately 30% of households sampled). Therefore, where factor analysis revealed that two factors should be used (one including all items relating to one parent and another including all items for the other), the factor analysis was forced to extract one factor.

**Attachment to parents.** The degree to which each adolescent was attached to their parents was measured by summing four items that asked how close the individual felt to his or her mother and father and how much he or she felt that his or her mother and father cared about them (α = .68). Factor analysis confirmed that each of the items loaded adequately on the factor, with loadings ranging from .59 to .83. The scores for this measure range from 4 to 20, with lower scores reflecting lower levels of attachment. The average score was 15.86 (SD = 4.15). Individuals with a score of 12 (25th percentile) or lower are considered at-risk and received a score of 1 on the cumulative risk index.

**Parental involvement.** Participants were asked whether or not they had participated in 10 different activities, such as shopping, attending various events, or talking about subjects related
to their daily lives with their mothers during the four weeks prior to the in-home interview. Participants were also asked if they had engaged in these same activities with their father during this period. For each item the youth indicated he or she did with his or her mother or father a score of 1 was given. All the items were summed to create an overall measure of parental involvement. A similar measure was employed previously by Gerard & Buehler (2004). Scores ranged from 0-19, with higher scores indicating greater parental involvement. The mean score is 5.69 (SD = 3.38), and any youth scoring 3 or less (25th percentile) is considered at-risk.

**Parental engagement.** Eight items on the in-home questionnaire at Wave I asked youth to report how much they agreed with statements regarding how loving their mother and father were, how well they communicated with their parents, and how satisfied they were with their relationships with each parent. Factor analysis confirmed that a single factor model fit the data (KMO = .84), with loadings ranging from .53-.89. These items were summed to create a measure of parental engagement (α = .88). Scores range from 7 to 35, with higher scores indicating lower engagement. The average score is 15.85 (SD = 6.39). Adolescents that scored a 20 (75th percentile) or greater on this scale are considered at-risk.

**Parental supervision.** Participants were asked to report the frequency that their mother and father are home before and after school using a 5-point Likert scale (1 = always, 5 = never). Factor analysis indicated that a single factor provided an adequate fit (KMO = .66) with loadings ranging from .35 to .76. Responses to four items were summed to create a measure of parental supervision, with higher scores indicating lower levels of supervision (α = .61). The range of values is 4-30, with a mean of 16.36 (SD = 5.28). If a youth received a score of 20 (75th percentile) or greater, he or she is considered at-risk.
Environmental-level risk items. Three different measures are included in the cumulative risk index to assess risk at the environmental level. These include delinquent peers, social support, and neighborhood safety.

Delinquent peers. Previous research using the Add Health data has operationalized delinquent peers using a three-item index (Beaver et al., 2009; Beaver & Wright, 2005). Adolescents were asked how many of their three closest friends use cigarettes, alcohol, or marijuana. Responses to these three questions were summed, creating an index ranging from 0-9 and a mean of 2.46 (α = .76). Individuals scoring at or above the 75th percentile (4) are considered to be at-risk on the cumulative risk index.

Social support. Seven items were summed to create a social support scale (α = .79). Adolescents were asked to report how much they felt others care about, understand, and pay attention to them, and how much they felt they have fun with their family. Factor analysis confirmed that a single measure fit the data (KMO = .84). Scores range from 7 to 35, with higher scores indicating greater perceived support. The average score is 28.34 (SD = 4.05). Those with a score of 26 or less (at or below the 25th percentile) are considered at-risk on the dichotomized measure.

Neighborhood safety. The safety of the respondent’s neighborhood is based on his or her response to the following question, “Do you usually feel safe in your neighborhood?”. Responses were coded 0 = yes and 1 = no. This measure has been used previously by Gerard & Buehler (2004) in a cumulative risk index.

Delinquency

Individual responses to risk factors can vary tremendously (Rutter, 2006a; 2006b). With respect to delinquency, some adolescents may respond to adversity in an aggressive or violent
manner, while others act out in nonviolent ways. To account for this, three measures of delinquent behaviors are used in the creation of the differential response to risk measures: overall delinquency, violent delinquency, and nonviolent delinquency.

**Overall delinquency.** Previous research using the Add Health data has measured overall delinquency using 14-items from the in-home questionnaire at Wave I (Boisvert, et al., 2012; Boisvert, Wright, Knopik, & Vaske, 2011; Haynie, 2001, 2002; Haynie, Giordano, Manning, & Longmore, 2005; Haynie & Osgood, 2005; Pearce & Haynie, 2004). Questions asked participants to report the frequency with which they engaged in various behaviors, such as stealing, fighting, or selling drugs over the past 12 months (see Appendix A). These items were summed to create an overall delinquency scale (α = .84). Scores among the subsample of twins range from 0-40, and the mean is 2.46 (SD = 4.19). Because the measure was skewed, it was log transformed (ln(x+1)) prior to analysis. The scores of log transformed overall delinquency range from 0-3.71 with a mean of .80 (SD = .87). Table 4.2 presents the correlations pre- and post-transformation (correlations with log transformed delinquency are in parenthesis). The pattern of relationships remained intact after overall delinquency was log transformed.

**Violent delinquency.** Violent delinquency at Wave I was operationalized by summing six items taken from the overall delinquency scale (α = .74). This same measure has been employed in a number of previous studies using the Add Health data (Boisvert et al., 2011; Haynie & South, 2005; Knoester & Haynie, 2005). For the twin subsample, scores range from 0-16 with a mean of 1.01 (SD = 1.95). Violent delinquency was also log transformed due to the skew of the distribution. The range of scores post-transformation was 0-2.83 with a mean of .45 (SD = .63). There were no significant changes to the correlations between violent delinquency and the risk measures following the transformation.
Nonviolent delinquency. A measure of nonviolent delinquency previously used by Boisvert and colleagues (2011) was created by summing eight items used in the overall delinquency scale (α = .79). Among twins, scores range from 0-24 and the mean is 1.45 (SD = 2.79). The distribution of nonviolent delinquency was also skewed, and the measure was log transformed. Scores of the transformed scale range from 0-3.22 with a mean of .53 (SD = .75). The pattern of correlations with the log of nonviolent delinquency matched the original scale with the exception of neighborhood safety, which reached statistical significance post-transformation although the magnitude remained small (r = .05).

Differential Response to Risk

The operationalization of differential response to risk was guided by research on resilience and vulnerability, since these concepts capture some of the errors in delinquency prediction. The conceptualization and operationalization of resilience has varied across previous studies (Luthar, Cicchetti, & Becker, 2000; Luthar & Cushing, 1999). Despite this heterogeneity, within their definitions of resilience studies typically include two constructs: (1) exposure to some adverse or risky condition(s); and (2) evidence of positive adjustment (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Luthar & Cushing, 1999). These constructs account for false positives, and this logic is extended here to also account for false negatives on the same continuum.

Recent research has operationalized resilience and vulnerability in a single measure using residual scores. For example, a study by Kim-Cohen, Moffitt, Caspi, & Taylor (2004) examined genetic and environmental influences on resilience and vulnerability to socioeconomic deprivation among a sample of 1,116 twin pairs during childhood. For both cognitive and behavioral outcomes, resilience and vulnerability were measured on a continuum using
Table 4.3 Ordinary Least Squares Regressions Predicting Delinquency (log transformed)

<table>
<thead>
<tr>
<th></th>
<th>Overall Delinquency</th>
<th>Violent Delinquency</th>
<th>Nonviolent Delinquency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>SE</td>
<td>β</td>
</tr>
<tr>
<td>Cumulative Risk</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>.12*</td>
<td>.01</td>
<td>.38</td>
</tr>
<tr>
<td>R²</td>
<td>.15</td>
<td>.08</td>
<td>.14</td>
</tr>
</tbody>
</table>

*p<.000

standardized residual scores from regressions predicting each outcome using deprivation as the predictor. In another study, Boardman, Blalock, & Button (2008) examined sex difference in the heritability of psychological resilience among a sample of 998 pairs of adult twins. The residual for positive affect, after controlling for numerous stressors, was used as a measure of resilience.

The primary focus of this study is to examine “off diagonal” cases, or all individuals that are more or less delinquent than would be predicted based on their level of risk. To assess this, the approach used to operationalize vulnerability and resilience by Kim-Cohen and colleagues (2004) is employed here. For each of the three log-transformed measures of delinquency, ordinary least squares regressions were conducted with cumulative risk entered as the predictor. The results of the regressions are presented in Table 4.3. The standardized residual scores were obtained for each regression, and represent a continuum with vulnerability at one end and resilience at the other. Individuals with an actual delinquency score higher than their predicted score have a positive residual score, reflecting vulnerability. Those with actual scores lower than their predicted score have a negative residual, reflecting resilience. With regard to overall delinquency, violent delinquency, and nonviolent delinquency the distribution for resilience and

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7 Ancillary analyses were conducted to assess whether the model would be better specified using an alternative regression approach. These analyses revealed that the OLS regression was most appropriate. Additionally, the estimates were very similar when MZ and DZ twins were analyzed separately.
A histogram of each of measure of differential response to risk is shown in Figure 4.2. All three distributions follow a similar pattern, and although each is standardized with a mean of 0 and standard deviation of 1, inspection of these distributions reveals two notable patterns. First, a large number of individuals tend to cluster between 0 and -1, which may indicate that many youths show at least slight resiliency. Second, the positive scores are more dispersed than the negative scores, suggesting that the degree of error is greater among vulnerable cases. Overall, the distributions seem to reflect that there may be two distinct groups; one that is resilient and one that is vulnerable. Additionally, it is important to note that none of the vulnerability ranges from -2.24 to 3.23, -1.62 to 3.68, and -1.98 to 3.48, respectively.
distributions follow a truly normal distribution, which should be considered when interpreting the results.

Sex Differences in Key Measures

Previous research has indicated that males are consistently involved in more delinquent behaviors than females (Federal Bureau of Investigation, 2011). One explanation that has been put forth is that males and females may differ in their exposure to risk factors (Belknap, 1996; Chesney-Lind, 1989). Additionally, there is some evidence to suggest that there may be sex differences in the heritability of resilience (Boardman et al., 2009; Waaktaar & Torgersen, 2012). In light of these findings, and because gender differences will be examined in this study, the mean of cumulative risk, each measure of delinquency, and each measure of differential response to risk was examined separately for males and females.

The means and standard deviation of each measure for males and females is shown in Table 4.4. The average cumulative risk score for males was 3.79 and the average for females was 3.69. Although the average was slightly higher for males, the difference was not significant. Both genders appeared to be exposed to the same amount of risks. Males were also involved in more delinquent behaviors across all measures of delinquency (.56 - .96) than females (.34 - .64), and the differences were significant across each measure ($p < .001$). It should be noted that for each measure of differential response to risk, the average scores for males were all positive (.15 - .19) suggesting a tendency toward vulnerability. For females, the average scores were all negative (-.15 - -.19), indicating greater resilience. The differences across all measures of differential response to risk were significant ($p < .001$).
Table 4.4 Difference in Risk, Delinquency, and Response to Risk in Males and Females

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th></th>
<th>Females</th>
<th></th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Cumulative Risk</td>
<td>3.79</td>
<td>2.41</td>
<td>3.69</td>
<td>2.48</td>
<td>.82</td>
</tr>
<tr>
<td>Overall Delinquency</td>
<td>.96</td>
<td>.91</td>
<td>.64</td>
<td>.81</td>
<td>7.19***</td>
</tr>
<tr>
<td>Violent Delinquency</td>
<td>.56</td>
<td>.68</td>
<td>.34</td>
<td>.56</td>
<td>6.64***</td>
</tr>
<tr>
<td>Nonviolent Delinquency</td>
<td>.64</td>
<td>.80</td>
<td>.42</td>
<td>.68</td>
<td>5.56***</td>
</tr>
<tr>
<td>Differential Response to Risk for Overall Delinquency</td>
<td>.19</td>
<td>1.05</td>
<td>-.19</td>
<td>.90</td>
<td>7.51***</td>
</tr>
<tr>
<td>Differential Response to Risk for Violent Delinquency</td>
<td>.17</td>
<td>1.08</td>
<td>-.17</td>
<td>.87</td>
<td>6.73***</td>
</tr>
<tr>
<td>Differential Response to Risk for Nonviolent Delinquency</td>
<td>.15</td>
<td>1.07</td>
<td>-.15</td>
<td>.90</td>
<td>5.75***</td>
</tr>
</tbody>
</table>

Note: All measures of delinquency are log transformed.

***p < .001

Plan of Analysis

The majority of analyses for the current research are conducted in R version 2.14.1 with the extension package OpenMx version 1.2.0-1924 (Boker et al., 2011). OpenMx is a structural equation modeling software commonly used in biometrical genetic modeling. Univariate models are estimated in order to examine the genetic, shared environmental, and unique environmental contributions to each of the three continuous measures of differential response to risk. OpenMx is also used to test for both qualitative and quantitative sex effects, as described in chapter three.

While high-risk cases that manage to overcome the odds have received a great deal of empirical attention, little is known about cases in which low-risk individuals engage in delinquent behaviors. It is possible that genetic and environmental influences work in different ways to account for resilience than for vulnerability. For example, genetic factors may contribute more to one’s vulnerability while environmental factors have a greater effect on
resilience. Furthermore, some research suggests that the most extreme cases in a distribution are under greater genetic influence than those closer to the average (Gjone, et al., 1996). In order to isolate each of these traits, and to explore possible differences in genetic influences among the most extreme cases, DF models for both constructs are conducted in Stata 12 (StataCorp, 2011).
CHAPTER V  
RESULTS

The following provides a review of the findings from the analyses described previously. Behavioral genetic models are decompose the variance in a phenotype into genetic and environmental components based on the difference in similarity observed between identical and fraternal twin pairs. Recall from chapter three that both the univariate behavioral genetic model and the sex-limitation models begin with an inspection of the cross-twin correlation coefficients, in which the score for one twin is correlated with the score of their co-twin. Additionally, the sex-limitation models are an extension of the basic univariate model. In light of these considerations, the chapter will begin with a review of the cross-twin correlations for all classifications of twin pairs. Next, the results from the basic univariate model are explained. This is followed by a discussion of the results of the sex-limitation models. Finally, the chapter will conclude with an overview of the DeFries-Fulker regression models used to assess the genetic and environmental contributions to resilience and vulnerability at the extremes of the distribution.

Cross-twin Correlation Coefficients

The cross-twin correlation coefficients are shown in Table 5.1. The first two rows of the table reflect the correlations for all MZ and DZ twins, regardless of sex\(^8\). Across all three measures of differential response to risk, the correlations were stronger for identical twin pairs than for fraternal twin pairs. Correlations ranged from .43 to .47 for MZ twins and .25 to .30 for DZ twins. All of the correlations were significant \((p < .01)\). As discussed in chapter three, dominant genetic and shared environmental effects cannot be estimated simultaneously in a

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\(^8\) In the basic univariate models, opposite sex DZ twin pairs were included and randomly assigned as twin one or twin two. Males were assigned as twin one in the sex limitation models.
classical twin design, and the cross-twin correlations provide a means of assessing whether an ACE or ADE model should be fit to the data. For each measure, the correlation between DZ twins is more than half the correlation observed in MZ twins, indicating than ACE model is the most appropriate for the analysis.

The cross-twin correlation coefficients can also provide a preliminary assessment of qualitative and quantitative sex-effects. As shown in Table 5.1, the correlations for differential response to risk for overall delinquency among MZ and DZ female twin pairs are nearly the same, suggesting no genetic influences on this trait for females. Among the male twin pairs, however, the cross-twin correlation is higher among MZ pairs ($r = .45$) than for DZ pairs ($r = .28$). This indicates that genetic influences may contribute to variation in males. With respect to differential response to risk for violent delinquency, the cross-twin correlations among both male and female MZ pairs ($r = .44$ and .45, respectively) is stronger than those observed among DZ pairs ($r_{DZ_m} = .28$, $r_{DZ_f} = .39$), and the difference is greater among male pairs. This pattern of

<table>
<thead>
<tr>
<th>Pair Type</th>
<th>Number of Pairs</th>
<th>Measure of Differential Response to Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Overall Delinquency</td>
</tr>
<tr>
<td>MZ</td>
<td>289</td>
<td>.471**</td>
</tr>
<tr>
<td>DZ</td>
<td>492</td>
<td>.268**</td>
</tr>
<tr>
<td>MZ$_F$</td>
<td>145</td>
<td>.447**</td>
</tr>
<tr>
<td>DZ$_F$</td>
<td>140</td>
<td>.443**</td>
</tr>
<tr>
<td>MZ$_m$</td>
<td>144</td>
<td>.451**</td>
</tr>
<tr>
<td>DZ$_m$</td>
<td>148</td>
<td>.282**</td>
</tr>
<tr>
<td>DZ$_os$</td>
<td>204</td>
<td>.140</td>
</tr>
</tbody>
</table>

Note: DZ reflects correlations for all DZ pairs, with males and females randomly assigned as twin one and two. DZ$_{os}$ reflects correlations for opposite sex DZ twin pairs, with males entered as twin one.

* $p < .05$; ** $p < .01$
correlations indicates that genetic effects may be influential among males and females, but the magnitude of the effects may be greater among males. The cross-twin correlations for differential response to risk for nonviolent delinquency show a different pattern. Among females, there is a stronger correlation among DZ twin pairs ($r = .43$) than MZ pairs ($r = .38$). The opposite pattern was found among male pairs, where the correlation among MZ pairs ($r = .44$) was stronger than that for DZ pairs ($r = .29$). Similar to differential response to risk for overall delinquency, these correlations suggest that while genetic factors may be influential among males, they may not be operating in females. Inspection of the cross-twin correlation coefficients for opposite sex DZ pairs can also be useful in assessing whether there are qualitative differences between males and females. Among the opposite sex DZ pairs, the correlations range from $.14$ to $.21$ and are somewhat lower than those observed among DZ$_m$ or DZ$_f$ pairs. Overall, the pattern of correlations observed in this sample suggests that both quantitative and qualitative differences may exist between males and females.

**Univariate ACE Model of Differential Response to Risk**

The basic univariate model begins with the estimation of a fully saturated model in which the correlations between additive genetic and shared environmental variables are specified, but the remaining parameters are free to vary. The assumptions of equal means and variances across members of each twin pair and across zygosity are then tested by imposing these constraints on the model and comparing the fit statistics to those of the saturated model. As shown in Table 5.2, both assumptions are met in the univariate ACE model for differential response to risk for overall delinquency. Imposing these constraints on the model did not produce a significant change in the fit of the data as indicated by the nonsignificant p-values.

The analysis proceeded by testing the significance of each variance component through a
Table 5.2 Univariate ACE Model for Differential Response to Risk for Overall Delinquency

<table>
<thead>
<tr>
<th>Model</th>
<th>ep</th>
<th>-2LL</th>
<th>df</th>
<th>Δ-2LL</th>
<th>Δdf</th>
<th>p</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated</td>
<td>10</td>
<td>4082.40</td>
<td>1467</td>
<td></td>
<td></td>
<td></td>
<td>1148.40</td>
</tr>
<tr>
<td>Equated Means and Variances Across Twin Order</td>
<td>6</td>
<td>4086.27</td>
<td>1471</td>
<td>3.87</td>
<td>4</td>
<td>.42</td>
<td>1144.27</td>
</tr>
<tr>
<td>Equated Means and Variances Across Twin Order and Zygosity (ACE)</td>
<td>4</td>
<td>4090.43</td>
<td>1473</td>
<td>8.03</td>
<td>6</td>
<td>.24</td>
<td>1143.43</td>
</tr>
<tr>
<td>AE</td>
<td>3</td>
<td><strong>4091.97</strong></td>
<td>1474</td>
<td><strong>1.54</strong></td>
<td>1</td>
<td>.22</td>
<td><strong>1143.97</strong></td>
</tr>
<tr>
<td>CE</td>
<td>3</td>
<td>4096.92</td>
<td>1474</td>
<td>6.48</td>
<td>1</td>
<td>.01</td>
<td>1148.92</td>
</tr>
<tr>
<td>E</td>
<td>2</td>
<td>4198.54</td>
<td>1475</td>
<td>99.11</td>
<td>2</td>
<td>.00</td>
<td>1239.54</td>
</tr>
</tbody>
</table>

ep = number of estimated parameters, -2LL = negative two log likelihood, df = degrees of freedom, Δ-2LL = difference in -2LL, Δdf = difference in degrees of freedom, AIC = Akaike’s Information Criterion

series of nested models. This is accomplished by constraining the parameter estimates to zero, and comparing the chi-squared statistics and the Akaike Information Criterion values (AIC) to the values in the ACE model. The values presented in Table 5.2 indicate that constraining the c parameter to zero in the AE model does not significantly reduce the fit (p = .22). The a parameter is then fixed to zero in the CE model. This constraint does significantly reduce the fit of the model (p = .01). Finally, the significance of both the a and c parameters is tested by fixing them both to zero. Constraining both values significantly changes the fit (p = .00), indicating both parameters cannot be dropped from the model. Recall that the e parameter can never be dropped because it captures both unique environmental influences and error. Therefore, the AE model is determined to be the best-fitting model. Although the AIC value is slightly smaller in the ACE model, the AE model is more parsimonious without significantly reducing the fit.

The next univariate model decomposes the variance in differential response to risk for violent delinquency. As can be seen in Table 5.3, the results of these analyses are very similar to
Table 5.3 Univariate ACE Model for Differential Response to Risk for Violent Delinquency

<table>
<thead>
<tr>
<th>Model</th>
<th>ep</th>
<th>-2LL</th>
<th>df</th>
<th>Δ-2LL</th>
<th>Δdf</th>
<th>p</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated</td>
<td>10</td>
<td>4095.33</td>
<td>1470</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1155.33</td>
</tr>
<tr>
<td>Equated Means and Variances Across Twin Order</td>
<td>6</td>
<td>4095.89</td>
<td>1474</td>
<td>0.56</td>
<td>4</td>
<td>.97</td>
<td>1147.89</td>
</tr>
<tr>
<td>Equated Means and Variances Across Twin Order and Zygosity (ACE)</td>
<td>4</td>
<td>4103.25</td>
<td>1476</td>
<td>7.92</td>
<td>6</td>
<td>.24</td>
<td>1151.25</td>
</tr>
<tr>
<td>AE</td>
<td>3</td>
<td>4104.46</td>
<td>1477</td>
<td>1.21</td>
<td>1</td>
<td>.27</td>
<td>1150.46</td>
</tr>
<tr>
<td>CE</td>
<td>3</td>
<td>4109.70</td>
<td>1477</td>
<td>6.45</td>
<td>1</td>
<td>.01</td>
<td>1155.70</td>
</tr>
<tr>
<td>E</td>
<td>2</td>
<td>4198.06</td>
<td>1478</td>
<td>94.81</td>
<td>2</td>
<td>.00</td>
<td>1242.06</td>
</tr>
</tbody>
</table>

ep = number of estimated parameters, -2LL = negative two log likelihood, df = degrees of freedom, Δ-2LL = difference in -2LL, Δdf = difference in degrees of freedom, AIC = Akaike’s Information Criterion

...those produced in the previous model. Constraining the c parameter to zero did not decrease the fit of the model to the data (p = .27); however, the constraints imposed in the CE and E models did produce significantly worse fits. Again, the AE model was determined to be the best-fitting model.

The results from the final univariate model, which decomposed the variance in differential response to risk for nonviolent delinquency, are shown in Table 5.4. For this measure, constraining the a parameter to zero significantly decreased the fit of the model (p = .03); however, constraining the c parameter did not reduce the fit (p = .12). The model in which both a and c were constrained to zero significantly reduced the fit (p = .00), indicating that the most parsimonious model providing an adequate fit to the data is the CE model.

The path estimates, standard errors, and the standardized coefficients for each of the best-fitting models are shown in Table 5.5. For differential response to risk for overall delinquency,
Table 5.4 Univariate ACE Model for Differential Response to Risk for Nonviolent Delinquency

<table>
<thead>
<tr>
<th>Model</th>
<th>ep</th>
<th>-2LL</th>
<th>df</th>
<th>Δ-2LL</th>
<th>Δdf</th>
<th>p</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated</td>
<td>10</td>
<td>4095.05</td>
<td>1471</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1153.05</td>
</tr>
<tr>
<td>Equated Means and Variances Across Twin Order</td>
<td>6</td>
<td>4100.64</td>
<td>1475</td>
<td>5.59</td>
<td>4</td>
<td>.23</td>
<td>1150.64</td>
</tr>
<tr>
<td>Equated Means and Variances Across Twin Order and Zygosity (ACE)</td>
<td>4</td>
<td>4103.09</td>
<td>1477</td>
<td>8.05</td>
<td>6</td>
<td>.23</td>
<td>1149.09</td>
</tr>
<tr>
<td>AE</td>
<td>3</td>
<td>4107.98</td>
<td>1478</td>
<td>4.88</td>
<td>1</td>
<td>.03</td>
<td>1151.98</td>
</tr>
<tr>
<td>CE</td>
<td>3</td>
<td><strong>4105.49</strong></td>
<td>1478</td>
<td>2.39</td>
<td>1</td>
<td>.12</td>
<td><strong>1149.49</strong></td>
</tr>
<tr>
<td>E</td>
<td>2</td>
<td>4200.89</td>
<td>1479</td>
<td>97.80</td>
<td>2</td>
<td>.00</td>
<td>1242.89</td>
</tr>
</tbody>
</table>

ep = number of estimated parameters, -2LL = negative two log likelihood, df = degrees of freedom, Δ-2LL = difference in -2LL, Δdf = difference in degrees of freedom, AIC = Akaike’s Information Criterion

Table 5.5 Path Estimates and Standardized Variance Components for Best-fitting Univariate ACE Models

<table>
<thead>
<tr>
<th></th>
<th>a</th>
<th>c</th>
<th>e</th>
<th>a²</th>
<th>c²</th>
<th>e²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Differential Response to Risk for Overall Delinquency</td>
<td>.68</td>
<td>-</td>
<td>.73</td>
<td>.46</td>
<td>-</td>
<td>.54</td>
</tr>
<tr>
<td></td>
<td>(.03)</td>
<td></td>
<td>(.03)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Differential Response to Risk for Violent Delinquency</td>
<td>-.67</td>
<td>-</td>
<td>.74</td>
<td>.45</td>
<td>-</td>
<td>.55</td>
</tr>
<tr>
<td></td>
<td>(.03)</td>
<td></td>
<td>(.03)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Differential Response to Risk for Nonviolent Delinquency</td>
<td>-</td>
<td>.59</td>
<td>.81</td>
<td>-</td>
<td>.35</td>
<td>.65</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(.03)</td>
<td></td>
<td>(.02)</td>
<td></td>
</tr>
</tbody>
</table>

Standard errors shown in parentheses.

46% and 54% of the variance is due to additive genetic and unique environmental influences, respectively. A similar pattern is observed in the best-fitting model for differential response to risk for violent delinquency, where 45% of the variance is due to additive genetic influences and 55% is due to unique environmental influences. Genetic factors did not contribute to variation in differential response to risk for nonviolent delinquency. In the best-fitting model, common and unique environmental influences accounted for 35% and 65% of the variance, respectively.
Sex-Limitation Models

Inspection of the cross-twin correlation coefficients by zygosity and gender suggest both qualitative and quantitative differences may exist between males and females. Testing for these effects begins with fitting a model in which the $a$, $c$, and $e$ path coefficients for males and females are free to vary, and the additive genetic and common environmental correlations between opposite-sex DZ twins are permitted to vary, one at a time, as described in chapter three. To test for qualitative differences, the correlation that is free to vary is equated to that of same-sex DZ twin pairs. If equating the correlations does not reduce the fit of the model, it can be assumed that there are no qualitative differences between males and females. Once potential qualitative differences have been examined, the path estimates between males and females are equated to test for quantitative differences. If constraining the pathways to be equal does not significantly reduce the fit of the model, no quantitative differences exist between the sexes.

Beginning with differential response to risk for overall delinquency, model one in Table 5.6 is the model in which the shared environmental correlation between opposite-sex DZ pairs is free to vary. This parameter is constrained to 1.0, just as with same-sex DZ twin pairs, in model two. This constraint did not significantly reduce the fit of the model ($p = .36$), indicating that the same common environmental factors that influence males also influence females. Model three permits the additive genetic correlation between opposite-sex DZ twins to vary, and it is constrained to equal .5 in model four. Again, this does not significantly reduce the fit of the model ($p = .61$); therefore, it can be concluded that the same genes are influencing males and females.

Model five in Table 5.6 equates the path estimates for males and females to test whether quantitative differences exist between males and females. Because no qualitative differences
<table>
<thead>
<tr>
<th>Model</th>
<th>ep</th>
<th>-2LL</th>
<th>df</th>
<th>AIC</th>
<th>ΔLL</th>
<th>Δdf</th>
<th>p</th>
<th>Comparison Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Full Model</td>
<td>9</td>
<td>4067.54</td>
<td>1468</td>
<td>1131.54</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>(rc free)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 rc = 1</td>
<td>8</td>
<td>4068.36</td>
<td>1469</td>
<td>1130.36</td>
<td>0.83</td>
<td>1</td>
<td>.36</td>
<td>1</td>
</tr>
<tr>
<td>3 Full Model</td>
<td>9</td>
<td>4068.10</td>
<td>1468</td>
<td>1132.10</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>(rg free)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 rg = .5</td>
<td>8</td>
<td>4068.36</td>
<td>1469</td>
<td>1130.36</td>
<td>0.26</td>
<td>1</td>
<td>.61</td>
<td>3</td>
</tr>
<tr>
<td>5 Homogeneity</td>
<td>5</td>
<td>4086.11</td>
<td>1472</td>
<td>1142.11</td>
<td>17.74</td>
<td>3</td>
<td>.00</td>
<td>4</td>
</tr>
<tr>
<td>(m=f)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Heterogeneity</td>
<td>6</td>
<td>4069.41</td>
<td>1471</td>
<td>1127.41</td>
<td>1.05</td>
<td>2</td>
<td>.59</td>
<td>4</td>
</tr>
<tr>
<td>(af=0, cm=0)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ep = number of estimated parameters, -2LL = negative two log likelihood, df = degrees of freedom, Δ-2LL = difference in -2LL, Δdf = difference in degrees of freedom, AIC = Akaike’s Information Criterion

were observed in models one through four, the additive genetic and shared environmental correlations between opposite-sex DZ pairs remained .5 and 1.0, respectively, and subsequent models were compared to model four. Equating the $a$, $c$, and $e$ pathways for males and females significantly reduced the fit of the model ($p = .00$), indicating that the magnitude of the effects between males and females differs.

The cross-twin correlations suggested that genetic factors may be influential among males, but would unlikely have an influence among females. Inspection of the path estimates in Table 5.7 further suggests this pattern, as the variance attributable to additive genetic effects for females in model four is zero. In the same model, the common environmental influence on males explains only 2% of the variance. As a result, these two parameters were fixed to zero in model six and the fit of this model was compared to model four. The values presented in Table 5.6 demonstrate that dropping these paths significantly improved the fit of the model as indicated.
Table 5.7 Path Estimates and Standardized Variance Components for Sex Limitation Models of Differential Response to Risk for Overall Delinquency

<table>
<thead>
<tr>
<th>Model</th>
<th>Femaless</th>
<th>Males</th>
<th>Femaless</th>
<th>Males</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$a$</td>
<td>$c$</td>
<td>$e$</td>
<td>$a^2$</td>
</tr>
<tr>
<td>1</td>
<td>.00</td>
<td>.65</td>
<td>.70</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td>(1.07)</td>
<td>(.04)</td>
<td>(.03)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>.00</td>
<td>.65</td>
<td>.70</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td>(1.33)</td>
<td>(.04)</td>
<td>(.03)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>.23</td>
<td>.62</td>
<td>.70</td>
<td>.06</td>
</tr>
<tr>
<td></td>
<td>(.67)</td>
<td>(.20)</td>
<td>(.06)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>.00</td>
<td>.65</td>
<td>.70</td>
<td>.00</td>
</tr>
<tr>
<td></td>
<td>(1.33)</td>
<td>(.04)</td>
<td>(.03)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>.59</td>
<td>.28</td>
<td>.75</td>
<td>.35</td>
</tr>
<tr>
<td></td>
<td>(.11)</td>
<td>(.18)</td>
<td>(.03)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>-</td>
<td>.65</td>
<td>.70</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>(.04)</td>
<td>(.03)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Standard errors shown in parentheses.

by the nonsignificant p-value ($p = .59$) and the lower AIC value. The path estimates presented in Table 5.7 show that for females, 46% of the variance in differential response to risk for overall delinquency is due to common environmental influences, and 54% is due to unique environmental influences. For males, 43% of the variance is attributed to additive genetic effects and 57% to unique environmental effects.

The results of the sex limitation model for differential response to risk for violent delinquency are shown in Table 5.8. Again, no qualitative sex differences were observed, as evidenced by the nonsignificant p-values in models two and four. Therefore, the test for quantitative differences (model five) was conducted with the additive genetic and shared environmental correlations between opposite-sex DZ pairs equated to those of the same-sex DZ pairs (.5 and 1.0, respectively). Equating the path estimates for males and females significantly reduced the fit of the model ($p = .00$), indicating that the magnitude of genetic and environmental
Table 5.8 Sex Limitation Model Differential Response to Risk for Violent Delinquency

<table>
<thead>
<tr>
<th>Model</th>
<th>ep</th>
<th>-2LL</th>
<th>df</th>
<th>AIC</th>
<th>ΔLL</th>
<th>Δdf</th>
<th>p</th>
<th>Comparison Model</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>4085.14</td>
<td>1471</td>
<td>1143.14</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>rc = 1</td>
<td>4086.51</td>
<td>1472</td>
<td>1142.51</td>
<td>1.37</td>
<td>1</td>
<td>.24</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>Full Model (rg free)</td>
<td>4085.29</td>
<td>1471</td>
<td>1143.29</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>rg = .5</td>
<td>4086.51</td>
<td>1472</td>
<td>1142.51</td>
<td>1.23</td>
<td>1</td>
<td>.27</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>Homogeneity (m=f)</td>
<td>4100.26</td>
<td>1475</td>
<td>1150.26</td>
<td>13.75</td>
<td>3</td>
<td>.00</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>Heterogeneity (cm=0)</td>
<td>4086.51</td>
<td>1473</td>
<td>1140.51</td>
<td>0.00</td>
<td>1</td>
<td>1.00</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>Heterogeneity (af=cm=0)</td>
<td>4088.92</td>
<td>1474</td>
<td>1140.92</td>
<td>2.40</td>
<td>2</td>
<td>.30</td>
<td>4</td>
</tr>
</tbody>
</table>

ep = number of estimated parameters, -2LL = negative two log likelihood, df = degrees of freedom, Δ-2LL = difference in -2LL, Δdf = difference in degrees of freedom, AIC = Akaike’s Information Criterion

effects differs between males and females.

Inspection of the cross-twin correlation coefficients shows a greater difference between MZ and DZ males than for MZ and DZ females, which suggests that genetic effects may be greater among males. This is further suggested by the path estimates generated in model four, as shown in Table 5.9. For males, the common environmental component is estimated to be zero; therefore, this parameter was dropped in model six. Removing this parameter did not reduce the fit of the model as indicated by the nonsignificant p-value and reduced AIC value. For females, while not estimated to be zero, the additive genetic effects estimated in model four were low ($a^2 = .12$). To test the significance of additive genetic effects among females, this parameter was dropped in model seven. As shown in Table 5.8, removing both the additive genetic parameter for females and the common environmental parameter for males did not significantly reduce the
Table 5.9 Path Estimates and Standardized Variance Components for Sex Limitation Models of Differential Response to Risk for Violent Delinquency

| Model | Females | | | | | Males | | | | |
|-------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|---------|
|       | $a$     | $c$     | $e$     | $a^2$   | $c^2$   | $e^2$   | $a$     | $c$     | $e$     | $a^2$   | $c^2$   | $e^2$   |
| 1     | .35     | .57     | .68     | .14     | .35     | .51     | .45     | .46     | .81     | .19     | .20     | .61     |
|       | (.25)   | (.13)   | (.04)   |         |         |         | (.23)   | (.19)   | (.04)   |         |         |         |
| 2     | .36     | .58     | .69     | .12     | .36     | .51     | .66     | .00     | .79     | .41     | 0       | .59     |
|       | (.33)   | (.16)   | (.05)   |         |         |         | (.05)   | (.28)   | (.04)   |         |         |         |
| 3     | .40     | .54     | .68     | .17     | .32     | .50     | .51     | .40     | .80     | .24     | .15     | .61     |
|       | (.20)   | (.13)   | (.04)   |         |         |         | (.20)   | (.22)   | (.04)   |         |         |         |
| 4     | .34     | .58     | .69     | .12     | .36     | .51     | .66     | .00     | .79     | .41     | 0       | .59     |
|       | (.33)   | (.16)   | (.05)   |         |         |         | (.05)   | (.28)   | (.04)   |         |         |         |
| 5     | .58     | .28     | .75     | .34     | .08     | .58     | .58     | .28     | .75     | .34     | .08     | .58     |
|       | (.11)   | (.18)   | (.03)   |         |         |         | (.11)   | (.18)   | (.03)   |         |         |         |
| 6     | .33     | .58     | .69     | .12     | .36     | .51     | .66     | -       | .79     | .41     | -       | .59     |
|       | (.18)   | (.09)   | (.04)   |         |         |         | (.05)   | (.04)   |         |         |         |         |
| 7     | -       | .65     | .71     | -       | .46     | .55     | .66     | -       | .79     | .41     | -       | .59     |
|       | (.05)   | (.03)   |         |         |         |         | (.05)   | (.04)   |         |         |         |         |

Standard errors shown in parentheses.

The fit of the model ($p = .30$). It should be noted, however, that model six provided a slightly better fit as indicated by the lower AIC value. As a result, model six is considered the best-fitting model. The path estimates for model six show that for females, 12% of the variance in differential response to risk for violent delinquency is accounted for by additive genetic factors, and 36% and 51% of the variance was due to common and unique environmental factors, respectively. For males, however, 41% and 59% of the variance can be attributed to additive genetic and unique environmental factors, respectively.

The final series of sex limitation models tests whether qualitative or quantitative sex effects are influencing differential response to risk for nonviolent delinquency. The results of these models are shown in Table 5.10. Again, constraining the additive genetic correlation to .5 and the common environmental correlation to 1.0 among opposite-sex DZ twin pairs in models
Table 5.10 Sex Limitation Model Differential Response to Risk for Nonviolent Delinquency

<table>
<thead>
<tr>
<th>Model</th>
<th>Comparison Model</th>
<th>ep</th>
<th>-2LL</th>
<th>df</th>
<th>AIC</th>
<th>ΔLL</th>
<th>Δdf</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Full Model (rc free)</td>
<td>9</td>
<td>4087.24</td>
<td>1472</td>
<td>1143.24</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>rc = 1</td>
<td>8</td>
<td>4087.49</td>
<td>1473</td>
<td>1141.49</td>
<td>0.26</td>
<td>1</td>
<td>.61</td>
</tr>
<tr>
<td>3</td>
<td>Full Model (rg free)</td>
<td>9</td>
<td>4087.49</td>
<td>1472</td>
<td>1143.42</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>rg = .5</td>
<td>8</td>
<td>4087.49</td>
<td>1473</td>
<td>1141.49</td>
<td>0.08</td>
<td>1</td>
<td>.78</td>
</tr>
<tr>
<td>5</td>
<td>Homogeneity (m=f)</td>
<td>5</td>
<td>4099.56</td>
<td>1476</td>
<td>1147.56</td>
<td>12.07</td>
<td>3</td>
<td>.01</td>
</tr>
<tr>
<td>6</td>
<td>Heterogeneity (af=0)</td>
<td>7</td>
<td>4087.49</td>
<td>1474</td>
<td>1139.49</td>
<td>0.00</td>
<td>1</td>
<td>1.00</td>
</tr>
<tr>
<td>7</td>
<td>Heterogeneity (af=0, cm=0)</td>
<td>6</td>
<td>4093.90</td>
<td>1475</td>
<td>1143.90</td>
<td>6.40</td>
<td>2</td>
<td>.04</td>
</tr>
</tbody>
</table>

ep = number of estimated parameters, -2LL = negative two log likelihood, df = degrees of freedom, Δ-2LL = difference in -2LL, Δdf = difference in degrees of freedom, AIC = Akaike’s Information Criterion

two and four did not significantly reduce the fit of the models. This indicates that there are no qualitative differences between males and females. In light of this, these parameters remained fixed in models five, six, and seven. The path estimates for males and females were equated in model five to test for quantitative differences. The significant p-value indicates that equating these parameters significantly reduces the fit of the model (p = .01) and that quantitative differences exist between the sexes.

Inspection of the cross-twin correlations suggests that additive genetic factors are unlikely to contribute to variation among females because the correlation among female DZ pairs is greater than the correlation among female MZ pairs. For males, the cross-twin correlations suggest that genetic factors may be important as the correlation among MZ males is greater than that of DZ males. Furthermore, the path estimates for model four shown in Table 5.11 suggest
Table 5.11 Path Estimates and Standardized Variance Components for Sex Limitation Models of Differential Response to Risk for Nonviolent Delinquency

| Model | Females | | | | | | | Males | | | | |
|-------|---------|--------|--------|--------|--------|---|--------|---------|--------|--------|---|--------|---------|--------|--------|---|--------|---------|--------|--------|---|
|       | $a$     | $c$    | $e$    | $a^2$  | $c^2$  | $e^2$ |       | $a$     | $c$    | $e$    | $a^2$  | $c^2$  | $e^2$ |
| 1     | .00     | .62    | .72    | .00    | .43    | .57   | (.38) | (.05)   | (.03)   | (.05)   | (.05)   |
| 2     | .00     | .62    | .72    | .00    | .43    | .57   | (.35) | (.05)   | (.03)   | (.12)   | (.18)   | (.04)   |
| 3     | .09     | .62    | .72    | .01    | .42    | .57   | (.39) | (.06)   | (.03)   | (.30)   | (.41)   | (.05)   |
| 4     | .00     | .62    | .72    | .00    | .43    | .57   | (.35) | (.05)   | (.03)   | (.12)   | (.18)   | (.04)   |
| 5     | .46     | .43    | .77    | .21    | .19    | .60   | (.13) | (.11)   | (.03)   | (.13)   | (.11)   | (.03)   |
| 6     | -       | .62    | .72    | -      | .43    | .57   | (.05) | (.03)   | (.03)   | (.09)   | (.11)   | (.04)   |
| 7     | -       | .62    | .72    | -      | .43    | .57   | (.05) | (.03)   | (.03)   | (.05)   | (.04)   |           |

Standard errors shown in parentheses.

the same pattern. Among females, the additive genetic influences are estimated to be zero. In order to test the significance of this parameter, it was dropped in model six. Fixing the female $a$ pathway to zero improved the fit of the model as indicated by the nonsignificant p-value ($p = 1.00$) and lower AIC value. Among males, the path estimate for common environmental effects in model four was somewhat low ($c^2 = .09$). To test the significance of common environmental influences among males, this parameter was dropped in model seven. As shown in Table 5.10, fixing both the additive genetic effects for females and the common environmental effects for males to zero significantly reduced the fit of the model ($p = .01$). Therefore, the best-fitting model is model six. The estimates presented in Table 5.11 show that for females, 43% and 57% of the variance in differential response to risk for nonviolent delinquency can be attributed to common and unique environmental influences, respectively. For males, additive genetic factors
accounted for 32% of the variance, while common and unique environmental factors accounted for 9% and 59%, respectively.

**DeFries-Fulker Extremes Analyses**

In the final stage of the analyses, DF regression is used to investigate the heritability in extreme groups. After selecting probands, or individuals selected due to extreme scores, the data can be transformed so that the mean of the probands is equal to 1 and the mean of the unselected sample is 0. When the basic DF regression model is estimated on transformed data, a direct estimate of group heritability ($h_g^2$) can be obtained. Mean scores for probands and their co-twins form the basis of DF regression and are presented for each model along with the regression coefficients.

Beginning with differential response to risk for overall delinquency, the cutoff criteria and mean scores before and after transforming the data are presented in Table 5.12. Recall that scores exceeding zero reflect vulnerability and scores below zero reflect resilience. Probands were initially selected based on these criteria to isolate the two outcomes. Then, extreme vulnerability was determined based on scoring more than one standard deviation above the mean and extreme resilience was defined as falling below one standard deviation below the mean. The first two rows, therefore, represent all vulnerable cases and extreme vulnerability. The bottom two rows represent resilience and extreme resilience.

The proband means for MZ and DZ twins are expected to be similar prior to transformation; however, scores were transformed by zygosity-specific proband means to account for the slight differences observed. The mean for MZ co-twins is expected to be closer to the proband mean than the DZ co-twin mean if the trait is influenced by genetic factors. This pattern is observed across all four selected samples, and indicates that genetic factors are
The results for the DF regression for each extreme group are shown in Table 5.13. Recall that the co-twin score is regressed on the proband score and a measure of the degree of genetic relatedness. In transformed data, the coefficient for the proband score captures everything that makes twins alike independent of genetic similarity, and the coefficient associated with genetic relatedness is a direct estimate of group heritability.

In the first model presented in Table 5.13, the estimate of group heritability of .53 is significant. This indicates that the genetic contribution to the difference between vulnerable cases and the population is 53%. The second model presented in Table 5.13 was estimated among cases where the proband exhibits more extreme case of vulnerability. The heritability estimate for this group was .51 ($p < .01$), revealing strong genetic influences on the difference between the extreme group and the population. Both models signify the importance of genetic influences on involvement in overall delinquency beyond the amount predicted based on cumulative risk.

The third and fourth models investigate the group heritability of resilient and extremely resilient youth. In the third model, the estimate of group heritability was .38 and significant.

Table 5.12 Proband and Co-twin Differential Response to Risk for Overall Delinquency Means Pre- and Post-Transformation

<table>
<thead>
<tr>
<th>Selection Criteria</th>
<th>Proband Means</th>
<th>Co-twin Means</th>
<th>Transformed Means</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MZ</td>
<td>DZ</td>
<td>MZ</td>
</tr>
<tr>
<td>Proband Score &gt; 0</td>
<td>1.04</td>
<td>.90</td>
<td>.52</td>
</tr>
<tr>
<td>Proband Score &gt; 1</td>
<td>1.60</td>
<td>1.50</td>
<td>.83</td>
</tr>
<tr>
<td>Proband Score &lt; 0</td>
<td>-.76</td>
<td>-.75</td>
<td>-.35</td>
</tr>
<tr>
<td>Proband Score &lt; -1</td>
<td>-1.31</td>
<td>-1.26</td>
<td>-.50</td>
</tr>
</tbody>
</table>
Table 5.13 DeFries-Fulker Extremes Regression Differential Response to Risk for Overall Delinquency

<table>
<thead>
<tr>
<th>Selection Criteria</th>
<th>b (SE)</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
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</tr>
<tr>
<td>(n=640)</td>
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<td></td>
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<tr>
<td>Proband Score</td>
<td>.38 (.08)</td>
<td>.00</td>
<td>.22 -.54</td>
</tr>
<tr>
<td>Relatedness ( h_g^2 )</td>
<td>.53 (.18)</td>
<td>.00</td>
<td>.17 -.88</td>
</tr>
<tr>
<td>Constant</td>
<td>-.40 (.15)</td>
<td>.00</td>
<td>-.69 -.12</td>
</tr>
<tr>
<td>Proband Score &gt; 1</td>
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<td>(n=279)</td>
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<td></td>
</tr>
<tr>
<td>Proband Score</td>
<td>.40 (.15)</td>
<td>.01</td>
<td>.09 -.70</td>
</tr>
<tr>
<td>Relatedness ( h_g^2 )</td>
<td>.51 (.19)</td>
<td>.00</td>
<td>.13 -.89</td>
</tr>
<tr>
<td>Constant</td>
<td>-.39 (.19)</td>
<td>.03</td>
<td>-.76 -.03</td>
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<td>Proband Score &lt; 0</td>
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<tr>
<td>(n=808)</td>
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<td>Proband Score</td>
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<td>.07 -.43</td>
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<tr>
<td>Relatedness ( h_g^2 )</td>
<td>.38 (.18)</td>
<td>.03</td>
<td>.02 -.73</td>
</tr>
<tr>
<td>Constant</td>
<td>-.16 (.16)</td>
<td>.30</td>
<td>-.47 -.15</td>
</tr>
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<td>Proband Score &lt; -1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=240)</td>
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<tr>
<td>Proband Score</td>
<td>.20 (.26)</td>
<td>.44</td>
<td>-.31 -.70</td>
</tr>
<tr>
<td>Relatedness ( h_g^2 )</td>
<td>.24 (.23)</td>
<td>.29</td>
<td>-.21 -.69</td>
</tr>
<tr>
<td>Constant</td>
<td>-.06 (.28)</td>
<td>.83</td>
<td>-.61 -.49</td>
</tr>
</tbody>
</table>

Note: Huber/White standard errors are presented.

\( p < .05 \). This suggests that the difference between resilient youth and the population is moderately influenced by genetic factors. The last model presented in Table 5.13 estimates the group heritability for extremely resilient youth. The heritability estimate among this group is not significant, and that genetic factors do not contribute to differences between the group of extremely resilient youths and the overall population.

The second series of DF regressions examines vulnerability and resilience with respect to differential response to risk for violent delinquency. Proband scores were selected using the
same cutoff criteria applied in previous extremes analyses. The mean scores for probands and their co-twins are shown in Table 5.14. In each selected sample, the mean for MZ co-twins is closer to the proband means than the mean for DZ co-twins. This pattern suggests that genetic factors could be contributing to vulnerability and resilience.

The coefficients produced in the extremes regressions for differential response to risk for violent delinquency are shown in Table 5.15. In the first model, which estimates the group heritability of all vulnerable cases, significant genetic influences accounted for differences between those that display more violent behavior than predicted and the population ($h_g^2 = .52, p < .01$). The second model examines extremely vulnerable youth. The estimate of group heritability is reduced to .33 and is not significant, suggesting that the difference between the population and youth that are much more violent than predicted based on cumulative risk is not accounted for by genetic factors.

The third and fourth models in Table 5.15 examine the group heritability among resilient and extremely resilient youths. The group heritability for resilient youths is estimated to be .34 and approaches significance ($p = .06$). The final model isolates extremely resilient youth. Group heritability was slightly stronger in this model (.45) but was nonsignificant. Taken together, the
Table 5.15 DeFries-Fulker Extremes Regression Differential Response to Risk for Violent Delinquency

<table>
<thead>
<tr>
<th>Selection Criteria</th>
<th>b (SE)</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
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<tr>
<td>(n=558)</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Proband Score</td>
<td>.34 (.08)</td>
<td>.00</td>
<td>.18 - .50</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.52 (.19)</td>
<td>.00</td>
<td>.15 - .88</td>
</tr>
<tr>
<td>Constant</td>
<td>-.36 (.16)</td>
<td>.02</td>
<td>-.68 - -.05</td>
</tr>
<tr>
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</tr>
<tr>
<td>(n=264)</td>
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<td></td>
</tr>
<tr>
<td>Proband Score</td>
<td>.46 (.15)</td>
<td>.00</td>
<td>.16 - .75</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.33 (.20)</td>
<td>.09</td>
<td>-.05 - .72</td>
</tr>
<tr>
<td>Constant</td>
<td>-.37 (.22)</td>
<td>.09</td>
<td>-.80 - .06</td>
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<td>(n=896)</td>
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<tr>
<td>Proband Score</td>
<td>.19 (.12)</td>
<td>.11</td>
<td>-.04 - .42</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.34 (.19)</td>
<td>.06</td>
<td>-.02 - .71</td>
</tr>
<tr>
<td>Constant</td>
<td>-.08 (.17)</td>
<td>.65</td>
<td>-.42 - .26</td>
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<tr>
<td>(n=144)</td>
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<tr>
<td>Proband Score</td>
<td>.51 (.53)</td>
<td>.33</td>
<td>-.53 – 1.56</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.45 (.30)</td>
<td>.13</td>
<td>-.15 – 1.05</td>
</tr>
<tr>
<td>Constant</td>
<td>-.50 (.57)</td>
<td>.38</td>
<td>-1.62 - .62</td>
</tr>
</tbody>
</table>

Note: Huber/White standard errors are presented.

The final series of DF regressions examines vulnerability and resilience with respect to differential response to risk for nonviolent delinquency. The proband and co-twin means pre- and post-transformation are shown in Table 5.16. In the samples selected for scores based on vulnerability and extreme vulnerability, the mean scores of MZ co-twins are closer to the proband means than the means scores of DZ co-twins, which suggests genetic factors may
Table 5.16 Proband and Co-twin Differential Response to Risk for Nonviolent Delinquency Means Pre- and Post-Transformation

<table>
<thead>
<tr>
<th>Selection Criteria</th>
<th>Proband Means</th>
<th>Co-twin Means</th>
<th>Transformed Means</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MZ</td>
<td>DZ</td>
<td>MZ</td>
</tr>
<tr>
<td>Proband Score &gt; 0</td>
<td>1.14</td>
<td>1.08</td>
<td>.49</td>
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<td>Proband Score &gt; 1</td>
<td>1.84</td>
<td>1.67</td>
<td>.89</td>
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<td>Proband Score &lt; 0</td>
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</tr>
<tr>
<td>Proband Score &lt; -1</td>
<td>-1.33</td>
<td>-1.31</td>
<td>-.42</td>
</tr>
</tbody>
</table>

Table 5.17 DeFries-Fulker Extremes Regression Differential Response to Risk for Nonviolent Delinquency

<table>
<thead>
<tr>
<th>Selection Criteria</th>
<th>b (SE)</th>
<th>p</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>(n=534)</td>
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</tr>
<tr>
<td>Proband Score</td>
<td>.36 (.08)</td>
<td>.00</td>
<td>.19 -.52</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.25 (.19)</td>
<td>.19</td>
<td>.12 -.63</td>
</tr>
<tr>
<td>Constant</td>
<td>-.19 (.16)</td>
<td>.24</td>
<td>-.50 -.12</td>
</tr>
<tr>
<td>Proband Score &gt; 1</td>
<td></td>
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</tr>
<tr>
<td>(n=261)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proband Score</td>
<td>.20 (.17)</td>
<td>.23</td>
<td>-.3 - .54</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.34 (.20)</td>
<td>.09</td>
<td>-.06 -.73</td>
</tr>
<tr>
<td>Constant</td>
<td>-.06 (.21)</td>
<td>.78</td>
<td>-.46 -.35</td>
</tr>
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<td>Proband Score &lt; 0</td>
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<td></td>
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</tr>
<tr>
<td>(n=922)</td>
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</tr>
<tr>
<td>Proband Score</td>
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<td>.00</td>
<td>.15 -.51</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>.12 (.19)</td>
<td>.53</td>
<td>-.26 -.50</td>
</tr>
<tr>
<td>Constant</td>
<td>-.07 (.16)</td>
<td>.53</td>
<td>-.37 -.24</td>
</tr>
<tr>
<td>Proband Score &lt; -1</td>
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</tr>
<tr>
<td>(n=122)</td>
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<td></td>
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</tr>
<tr>
<td>Proband Score</td>
<td>.15 (.44)</td>
<td>.73</td>
<td>-.73 - 1.03</td>
</tr>
<tr>
<td>Relatedness ($h_g^2$)</td>
<td>-.01 (.29)</td>
<td>.96</td>
<td>-.59 -.56</td>
</tr>
<tr>
<td>Constant</td>
<td>.17 (.51)</td>
<td>.72</td>
<td>-.83 - 1.18</td>
</tr>
</tbody>
</table>

Note: Huber/White standard errors are presented.
account for some of the differences between these selected groups and the population. The same pattern is observed in the sample selected due to resilience; however, in the last row of the table, the co-twin means are equal, suggesting that genetic factors do not account for differences between extremely resilient cases and the population.

The results from the DF regressions are shown in Table 5.17. The group heritability estimated in each of the models is substantially lower than the models examining the extremes of differential response to overall and violent delinquency. Furthermore, none of the estimates are significant. These findings indicate that the differences between youths that are involved in more or less nonviolent crime than predicted based on cumulative risk cannot be attributed to genetic factors.
CHAPTER VI

CONCLUSIONS AND FUTURE DIRECTIONS

Prior research has uncovered a vast array of risk factors for delinquency; however, errors in predicting delinquent behavior persist. In some instances, youths exhibit more or less delinquent behavior than would be predicted based on the risks they encounter. A closer examination of individuals whose actual behavior deviates from their predicted behavior can be instrumental in improving prediction, advancing criminological theory, and developing appropriate interventions (Marks, 1964; Stein et al., 1970; Sullivan, 2011; Weisburd & Piquero, 2008). The recently proposed differential susceptibility theory contends that individuals may respond differently to the conditions to which they are exposed, and that these contrasting reactions may be due to genetic variation between people (Ellis et al., 2011). Although a number of studies have examined the role of specific genotypes in the relationship between risk and behavior, no study to date has examined the overall extent to which genetic factors contribute to variation in response to cumulative risk for delinquency.

In an effort to extend the literature regarding this issue, this dissertation encompassed three broad objectives. First, the primary aim of the research was to assess the degree to which genetic and environmental factors contribute to differential response to risk. Second, because prior research indicates that males and females differ in both their amount of delinquent involvement and in their response to risk factors (Federal Bureau of Investigation, 2011; Moffitt, et al., 2001), this dissertation explored the qualitative and quantitative sex differences between males and females. Finally, since far less attention has been given to youths that are involved in more delinquency than would be expected compared to those who are resilient (Garmezy et al., 1984; Masten, 2001; Masten & Garmezy, 1985; Rutter, 1990; Werner & Smith, 1982, 1992), this
study fills a gap in the literature by isolating resilient and vulnerable youths and examining these cases separately. The following provides an overview of the findings, limitations, and implications of this research.

**Summary of Findings**

**Univariate and Sex-Limitation Models**

The results from the basic univariate models suggest that the genetic and environmental influences on responses to risk are dependent upon the characteristics of the delinquent behavior examined. Variation in response to risk for any delinquent behavior is influenced by additive genetic (45%) and unique environmental factors\(^9\) (54%). Similarly, variation in violence in response to risk is attributed to additive genetic (46%) and unique environmental (55%) influences. For both of these measures of delinquent responses to risk, shared environmental factors did not contribute to the variation. Interestingly, the opposite pattern was observed when analyzing differences in nonviolent in delinquent outcomes in response to risk. Specifically, shared (35%) and unique (65%) environmental influences accounted for all the variation in this trait.

When gender differences were taken into consideration, a somewhat different pattern of results emerged. Although no qualitative differences were observed by gender, large differences were found in the magnitude of effects. For females, additive genetic factors did not influence responses to risk for overall and nonviolent delinquency. Shared environmental factors accounted for a large portion of the variation in response to risk for these delinquent outcomes (43-46%). The remaining variance was explained by unique environmental factors (54-57%). Additive genetic effects did, however, explain a small portion of the variance in violent

\(^9\) Recall from chapter three that the unique environmental component also includes the error in the model.
responses to risk in females. Specifically, 12%, 36%, and 51% of the variance was attributed to additive genetic, shared environmental, and unique environmental factors in females.

Among males, for overall and violent delinquent responses to risk, additive genetic influences had considerable effects (41-43%), but shared environmental factors had no effect. The remaining variance was attributed to unique environmental factors (57-59%). Variation in nonviolent delinquent responses to risk was partially due to additive genetic factors (32%), but shared environmental (9%) factors had a small significant effect. The remaining variance was attributed to unique environmental influences (59%).

Three features of the results found in the basic and sex-limitation univariate models warrant discussion. First, the different findings for males and females demonstrate the importance of investigating potential differences in the etiology of behaviors in each gender. The results of the basic univariate models that decompose the variance in response to risk for overall and violent delinquency are consistent with the estimates observed among males in the sex-limitation models; however, the results are inconsistent with the estimates for females. Generalizing the findings in the univariate models to both sexes would have overestimated the genetic effects and underestimated the shared environmental effects in females. Additionally, the results of the basic univariate model for response to risk for nonviolent delinquency underestimated the shared environmental influences in females. Among males, the model underestimated the additive genetic influences and overestimated the shared environmental contributions to the variation. Failing to consider possible sex differences would have masked important sources of variation in each group.

Second, these findings suggest that the manner in which females respond to the risks they encounter is largely determined by environmental factors, but males are largely influenced by
genetic factors. Sex differences in genetic effects have been observed in molecular genetic studies that examine antisocial outcomes in response to particular risk factors among individuals with specific genotypes. Prom-Worley and colleagues (2009) found that the interaction between MAOA genotype and childhood adversity among females was not significant in models predicting conduct disorder. In another study by Schwartz & Beaver (2011), the interaction between MAOA genotype and perceived prejudice was a significant predictor of arrest in males, but not in females. Similarly, Brody and associates (2011) found that the interaction between 5HTTLPR genotype and perceived discrimination was a significant predictor of conduct problems in African American adolescent males, but not females. Belsky & Beaver (2011) created a more inclusive measure of genetic factors that covered five genes, and tested whether cumulative genetic plasticity interacted with parenting quality to influence the development of self-regulation. They found that the interaction was only significant among males. While these studies test for gene-environment interactions, the findings suggest that genetic factors may play a more important role in shaping the way males—but not females—respond to risks for delinquency.

Zahn-Waxler and colleagues (2006) argue there are several possible explanations for differences in genetic and environmental influences on traits across gender. One explanation is that males and females may have different biological mechanisms of gene expression. An individual may possess a particular gene, but differences in biological processes may be stronger in one gender and weaker in the other, resulting in differences in the expression of a particular trait. In other words, the process that activates a gene in one sex may not activate it in the other. Second, males and females may have different thresholds of genetic risk that must be met before a particular trait emerges (Moffitt et al., 2001; Zahn-Waxler et al., 2006). Recall from chapter
three that many traits are polygenic, or influenced by several genes. If females had a more extreme threshold, they may require the possession of more genes associated with an increased risk before a trait is observed. A third possibility is that chromosomal differences lead to important differences in physiological processes (Rutter, 2003; Zahn-Waxler et al., 2006). Females possess two X chromosomes, one of which is activated. Males, on the other hand, possess one X and one Y chromosome. The possession of a Y chromosome in males results in differences in the production of enzymes, hormones, and neurotransmitters that may influence behavior (Rutter, 2003; Zahn-Waxler et al., 2006). Each of these explanations is plausible, and may account for the gender differences in the genetic and environmental influences on differential response to risk observed in this study.

The third notable aspect of the results is that genetic effects are operating in males and females for differential response to risk for violent delinquency, and shared environmental factors have a meaningful impact on both genders for differential response to risk for nonviolent delinquency. While the differences in the magnitude of effects are substantial between males and females, the general pattern seems to suggest that violent and nonviolent responses to risk may be shaped by different factors. The findings in regard to sex differences and the sources of variation for different types of delinquent responses to risk are consistent with Moffitt’s (1993) theory on development and delinquency.

Moffitt (1993) has argued that two types of offenders exist: adolescence-limited and life-course-persistent. The antisocial behavior exhibited by these two types of offenders is believed to have different origins. Life-course persistent offenders are hypothesized to suffer from neuropsychological deficits resulting from genetic influences, which are believed to be more common among males (Moffitt, 1993; Moffitt et al., 2001). These individuals begin exhibiting
problem behaviors very early in life and continue into adulthood. Adolescence-limited offenders, on the other hand, are involved in delinquent behaviors only during adolescence. These youths experience a “maturity gap,” or stage at which they are physically developed but prevented from developing socially due to expectations placed on them by society (Moffitt, 1993). The lifestyles of life-course-persistent offenders resemble that of adults, and adolescence-limited offenders mimic their behavior as a means of coping until the maturity gap closes. The social influences on adolescent-limited offending have been found to affect both males and females (Moffitt et al., 2001).

Additional evidence supports the hypothesis that adolescence-limited and life-course-persistent offending emerge from different sources of influence. For example, Eley and colleagues (1999) examined a Swedish and a British sample of males and females and found that genetic (approximately 70%) and unique environmental factors contributed to variation in aggressive behavior, but shared environmental factors were not significant. No qualitative or quantitative sex effects were observed. In the models for nonaggressive behavior, additive genetic, shared environmental, and unique environmental influences were all significant, and the genetic effects were reduced by 30-40%. In contrast to the results in the current study, the genetic influences on nonaggressive behaviors were slightly stronger in females than in males and qualitative differences were observed in the Swedish sample (Eley et al., 1999).

A later study found that there was greater continuity in aggressive behaviors than in nonaggressive behaviors (Eley et al., 2003). Early aggressive behaviors were largely due to genetic factors (60%) and the influence of common environmental factors was minimal (15%). The influences on early nonaggressive behaviors were more balanced, with genetic factors accounting for 49% of the variance and common environmental factors accounting for 35%.
During adolescence, both additive genetic and common environmental sources were moderately significant in both forms of antisocial behavior. Interestingly, there was strong continuity in both behaviors from childhood to adolescence, but the continuity of aggressive behaviors was almost entirely due to genetic factors.

The findings from Eley and colleagues (1999, 2003) are partially consistent with the results of this study. For both genders, genetic factors are important in explaining variation in violent responses to risk, which included behaviors such as threatening to or physically harming another person, the use of weapons to threaten or harm someone, or participating in group fights. Recall that the measure of differential response to risk used in this study captured a range of outcomes from delinquency among low risk youths, and the avoidance of delinquency among those that are exposed to numerous risks. The findings with respect to violence indicate that some youths may, in fact, be more vulnerable and likely to use violence even when they are not in high-risk situations. Still, others may not be willing to use violence, regardless of the number of risks they encounter. Differences in the propensity to use violence are largely due to genetic differences, and failing to account for genetic differences may limit efforts to accurately predict violent behavior.

In contrast, among males and females shared environmental factors are important in explaining variation in nonviolent responses to risk such as damaging property, stealing, or selling drugs. This finding further emphasizes the need to examine protective and promotive factors that reduce nonviolent offending among high-risk youths, as the factors that contribute to their resilience appear to be found within their environments. For example, receiving an abundance of attention from caretakers, positive relations with mentors, having prosocial peers, and being connected to prosocial organizations and quality schools have been shown to have
protective effects among high-risk youths (Jarrett, 1997, 1999; Masten & Powell, 2003; Stouthamer-Loeber et al., 2002; Stouthamer-Loeber et al., 1993; Werner & Smith, 1982, 1992). These findings suggest that resilience among youths at-risk for nonviolent delinquency may be linked to relationships with others.

At the other extreme, some youths were involved in more nonviolent crime than would be predicted based on the risk factors examined in this study. The vulnerability exhibited by some youth also appears to be due to environmental factors, indicating that additional risk factors must be explored. Reiss & Farrington (1991) found that nonviolent offending was more likely to involve co-offenders compared to violence, and that siblings are likely to offend together. One possible explanation offered for this is that some youths may require support or encouragement from others in taking risks, particularly the first time they commit a given criminal act. Others have argued that individuals may commit crimes when there is an opportunity for personal gain (Cohen & Felson, 1979; Cornish & Clark, 1986). Moffitt (1993) has also argued that adolescence-limited offenders are more likely to be involved in crimes that have the potential to be beneficial (e.g., theft). It is possible that youths who encounter few risk factors may still take advantage of opportunities to gain money or items that would otherwise be difficult to obtain. The findings obtained in this study may indicate that relationships with others and situational factors could contribute to nonviolent delinquency among low-risk youths.

**Extremes Analysis**

Given that differential response to risk for delinquency was measured on a continuum from resilience to vulnerability, the basic univariate model was not able to assess the degree to which each of these outcomes was due to genetic influences. DF-regression analyses were employed to examine both resilience and vulnerability independently, as well as extreme
categories in each group. Two notable findings emerged. First, vulnerability appears to be under greater genetic influence than resilience. In only one instance was the group heritability estimate of resilience significant. The difference between the population and youths that were involved in less overall delinquency than predicted was moderately due to genetic factors \( h^2 = .38 \). The difference between extremely resilient youth and the population was not due to genetic factors in any of the models.

On the other hand, in the DF models examining differential response to risk for overall and violent delinquency, the majority of the difference between youths that were involved in more crime than predicted and the population was due to genetic factors. Specifically, 53% and 51% of the difference between the population and youths that were vulnerable or extremely vulnerable for overall delinquency, respectively, was due to genetic influences. Differences between youths vulnerable for violent delinquency and the population was due to genetic factors to a similar degree \( h^2 = .52 \).

The results of this study are somewhat consistent with the findings in previous research. Kim-Cohen and her colleagues (2004) examined behavioral resilience and vulnerability to socioeconomic deprivation using the same type of measurement strategy employed in this study. Behavioral resilience and vulnerability was based on residuals from a regression predicting a measure of aggression and delinquency from socioeconomic deprivation. The basic univariate model in the study revealed that 70.5% of the variation in behavioral responses was due to additive genetic factors and the remaining variance was explained by unique environmental factors. The groups of individuals displaying extreme vulnerability and extreme resilience, defined as the bottom and top 25% of the distribution, had group heritability estimates of .71 and .72, respectively. The group heritability estimates for vulnerability in this study were also
strong, although not as strong as the estimates found by Kim-Cohen and associates (2004). The group heritability estimates for resilience, however, are not consistent with previous findings. In only one instance was group heritability significant and the estimate was notably smaller than that observed by Kim-Cohen and colleagues (2004).

The second notable finding in the DF analyses is that even extreme cases of resilience and vulnerability in response to risk for nonviolent delinquency are not influenced by genetic factors. This suggests that involvement in more or less nonviolent delinquency than predicted is entirely due to environmental factors. This is not to say that nonviolent delinquency overall is not genetically influenced; only that differences between the extremes and the overall population are not due to genetic factors. Recall that for males, there was a moderate genetic influence on nonviolent delinquency. Still, the findings from the DF analysis are consistent with the findings from the univariate models, which seem to suggest that there is an important social element to nonviolent delinquency. As noted previously, resilient youths may be involved in relationships that are supportive and assist them in overcoming the risks that they encounter. Vulnerable youths, however, may be in relationships that encourage participation in nonviolent crimes or may take advantage of opportunities for personal gain in various situations.

The results of the extremes analyses should be interpreted with caution. These analyses were conducting using the entire sample of twins, which was not large enough to examine the extremes by gender. Furthermore, on average, females tended to exhibit resilience in response to risk and males tended to be more vulnerable. Given that the correlations for MZ and DZ females were very similar, it is not surprising that resilience and extreme resilience did not appear to be influenced by genetic factors in this sample. The existing research investigating genetic influences on resilience, however, indicates that resilience in response to some risks is heritable.
(Kim-Cohen et al., 2004), and that there may be differences in the magnitude of genetic effects between males and females. For example, Boardman and his colleagues (2009) examined sex differences in psychological resilience to stressors and found that the heritability of resilience was greater among males ($h^2 = .52$) than females ($h^2 = .38$). In a recent study by Waaktaar and Torgersen (2012), the heritability of resilience as a personality trait was estimated for males and females. For boys, 78% of the variation in resilience was attributed to additive genetic effects. For girls, the estimate was slightly lower at 70%.

The results of this study were not consistent with these previous studies, as resilience generally was not influenced by genetic factors. Research on genetic influences on resilience, however, is only beginning to emerge. Each of these studies has taken a different approach to measuring resilience, and assessed resilience to different risks for different outcomes. Additionally, the samples in each of these studies were very different. Kim-Cohen and her colleagues (2004) examined a sample of 5-year old twins from England and Wales born in 1994-1995. Waaktaar and Torgersen (2012) studied twin pairs at age 12-18 years that were born between 1988 and 1994 in Norway. Boardmand and associates (2009) examined a sample of adults ages 25-74 from the United States. The sample in the current study included adolescents between 12 and 18 years of age in the United States. It is plausible that differences in measurement of resilience or in the characteristics of each sample are contributing to the differences in the results. Continued research in this area will be instrumental in uncovering whether resilience and vulnerability are specific to the risks and outcomes under investigation, or whether resilience reflects variation in sensitivity to risks more generally. Moreover, additional research is needed to assess the most appropriate method of measuring resilience and vulnerability.
Limitations

The results of this study should be considered in light of several limitations. First, there are concerns related to the sample that may impact the generalizability of the results. Second, various aspects of the risk, delinquency, and differential response to risk measures used in this study should be observed in light of the potential impact of each on the results. Third, limitations surrounding the study design and analytical approach also exist that, if addressed in future research, could further the understanding of differential response to risk.

Sample

There are two potential limitations in regard to the sample used in this study. First, the Add Health study was intended to be a nationally representative sample of youths in America; however, only the subsample of twins was examined in this study. Twins were first identified through stratified random sampling techniques rather than purposive methods. Co-twins of selected twins were then asked to participate. Add Health researchers used this technique in an effort to select a nationally representative sample of twins (Harris et al., 2006).

Despite the use of a strong sampling strategy, it has been argued that twins could be different than singletons in meaningful ways, thereby limiting the generalizability of the results (Rutter & Redshaw, 1991; Rutter, Simonoff, & Silberg, 1993). For example, pregnancies and deliveries involving twins are more likely to encounter complications, which may result in neurological disorders or delays (Moilanen & Ebeling, 1998; Moilanen et al., 1999; Rutter & Redshaw, 1991). Additionally, twins have been found to develop linguistically at a slower pace than singletons, which is believed to be the result of having fewer opportunities to have one-on-one conversations with parents and being able to communicate nonverbally with their twin sibling (Moilanen & Ebeling, 1998; Rutter & Redshaw, 1991). Twins also are born into a
relationship with their co-twins that may shape their personalities in meaningful ways, particularly when one twin becomes dominant and the other submissive (Moilanen, 1987). Neuropsychological and language deficits and personality factors have been related to criminality, so it is possible that such differences could lead to important differences in the development of delinquency in twins and singletons (Eyesenck, 1977; Hare, 1999; Lykken, 1995; Moffitt, 1993; Rutter & Redshaw, 1991). A recent study by Barnes & Boutwell (2013), however, found that the twins in the Add Health study did not differ significantly from non-twins in terms of delinquent involvement. The results of their study also indicated that the effects of several predictors of delinquency were not different among co-twins, suggesting that the findings produced in studies of twins in the Add Health data are generalizable to the larger population of adolescents.

The second limitation in regard to the analytic sample is its size. Relative to other studies of twins based on data from twin registries, the subsample of twins in the Add Health study is somewhat small. Primarily, this was an issue in the analysis of extreme cases. The sex-limitation models indicated that there were important differences between the genetic and environmental influences in males and females. In light of this, it would have been ideal to examine resilience and vulnerability by gender. Dividing the sample into same-sex twin pairs by gender and then selecting probands based on extreme scores would not have maintained the size of the sample to the degree necessary to produce reliable results. As previously noted, the results from the DF analyses should be considered preliminary, and future research should continue to investigate potential differences in resilience and vulnerability between males and females.
Measurement

There are also a number of limitations surrounding the measurement of the variables included in this study. With respect to the risk measures, there are five central concerns. First, not all relevant risk factors were included in the cumulative risk measure. For example, Gottfredson and Hirschi (1990) claim that self-control is the single-most important predictor of criminality, and there is substantial evidence in support of this claim (Pratt & Cullen, 2000). Formal measures of self-control were not included in the Add Health questionnaires, and the items used in previous studies to measure self-control were used in the creation of other risk factors in this study. Additionally, a number of scholars have argued that maltreatment, abuse, and neglect may play an important role in the development of antisocial behavior (Hawkins et al., 2000; Widom, 1989a, 1989b). Youths were asked to report experiences of abuse or neglect retrospectively at wave three of the Add Health study, but very few individuals responded to these questions or reported such experiences among the subsample of twins. Failing to include all relevant risk factors in the cumulative risk measure would have led to underestimating the overall risk each individual had encountered, and could have contributed to the low $R^2$ values in the regressions predicting delinquency. Because differential response to risk was operationalized as standardized residuals from those regressions, it is possible that the prevalence of vulnerability was overestimated, the prevalence of resilience was underestimated, or both. To reduce this bias, future research should seek to include more comprehensive assessments of risk.

Second, the same measure of cumulative risk was used to predict all three types of delinquency. Third, and relatedly, the significance of predictors across males and females was assumed to be the same in this study. Whether gender-specific risk factors exist and if males and females are equally exposed to risk factors has been debated (Belknap, 1996; Belknap &
Holsinger, 2006; Campbell, 1981; Chesney-Lind, 1989; Chesney-Lind & Sheldon, 1992; Daly &
Chesney-Lind, 1988; Holsinger, 2000). Some studies have, in fact, found differences in the
predictors of overall, violent, and nonviolent delinquency, as well as differences in the predictors
of these outcomes across gender (Alarid, Vulmer, & Cullen, 2000; Booth, Farrell, & Varano,
2008; Daigle et al., 2007; Mazerolle, 1998; Piquero & Sealock, 2004). While the measure of
cumulative risk used in this study was a significant predictor of all three types of delinquency, it
is possible that prediction could have been improved by creating separate risk scores for each
gender and each type of delinquency. An important next step will be to explore this possibility
in an effort to better understand how males and females respond to particular risks, and the
genetic and environmental factors that influence those responses.

A fourth limitation is that the criteria used to select cut-off scores for each risk factor
were based on strategies employed in previous research; however, these may not accurately
reflect the points at which one’s risk actually increases. The actual thresholds may be lower or
higher than those used, and the measure of cumulative risk may have been imprecise, which
would have also biased the residual scores. Farrington and Loeber (2000), however, found that
dichotomizing risk measures does not greatly affect the relationships between predictors and
outcomes and that this approach is suitable for risk factor research.

Similarly, a fifth limitation in regard to risk is related to the overall assessment of risk.
The accumulation of risks may only be problematic once a particular number of risks are
encountered. That is, rather than being linearly related to delinquency, the relationship could
take another functional form (Appleyard et al., 2005). Alternatively, it is possible that
experiencing risks in multiple domains could place one at the greatest risk for antisocial
outcomes (Gerard & Buehler, 2004). Further still, some risk factors or risk domains may be
more influential than others (Ribeaud & Eisner, 2010). Researchers have only recently begun to investigate these possibilities, and the available research seems to indicate that youths who encounter multiple risk factors are more likely to experience adverse outcomes (Sameroff et al., 1998). As more research becomes available regarding the thresholds for each risk factor, and the relationship between cumulative risk and delinquency, it will be important to reexamine the findings presented here.

With regard to the three measures of delinquency, one limitation is that the measures were based on self-reports. Various problems may exist with self-reported measures that impact the reliability and validity of the scales (Thornberry & Krohn, 2000). For instance, subjects may forget previous events or be tempted to respond dishonestly to questions that are particularly sensitive. Respondents may feel embarrassed about their involvement in deviant acts, or fear being criminally charged after admitting to undetected criminal behavior. Additionally, among juveniles, an additional fear might include their parents becoming aware of their unlawful behavior and the resulting consequences.

Thornberry & Krohn (2000) explain that self-reported measures of delinquency are generally valid and reliable, particularly those that (1) include several items; (2) measure involvement in both serious and nonserious crimes; and (3) employ specialized techniques to encourage participation and honesty. Add Health researchers made efforts to meet these standards. In addition to including a variety of items tapping into different aspects of delinquency, Audio Computer-Assisted Self-Administered Interview (ACASI) methods were used to collect the data. This allowed youths to have the maximum amount of privacy while completing the delinquency questionnaire, which has been shown to elicit higher response rates (Couper et al., 2003; Ghanem et al., 2005; Tourangeau & Smith, 1996). While the measurement
of delinquency in this study is acceptable, future studies of differential response to risk for delinquency should investigate whether similar results are found with alternative measures of delinquency, such as official records or parent reports of behavior.

It is important to note that the central measure used in this study, differential response to risk, suffers from the combination of limitations discussed for the cumulative risk and delinquency measures. Strategies for measuring resilience and vulnerability are heavily debated because they require consideration of both exposure to risk and the resulting outcome (Kazdin et al., 1997; Luthar & Cushing, 1999; Luthar et al., 2000). Beyond this basic premise, there is little consensus regarding the best way to assess these aspects of development. The approach taken in this study has not been widely used, and there is the potential for it to capture more than just resilience and vulnerability. Efforts were made to minimize the amount of error in the model, and obtain the purest measure of resilience and vulnerability in response to risk. Despite these efforts, it is probable that the measure is at least minimally compromised, as indicated by the small effects of the cumulative risk measure on delinquency and variance explained in each of the regression models. However, when MZ and DZ twins were analyzed separately, the results were very similar, suggesting that any error that was unaccounted for affected MZ and DZ twin pairs to the same degree. Because the error applies equally across zygosity, it is probable that the difference (though not the size) in cross-twin correlations was unaffected. It is unlikely that the pattern of results observed in this study would be largely biased as a result of this error.

**Study Design and Analytic Strategy**

In addition to the sample and measures analyzed, limitations related to the study design and analytical techniques should also be considered. A cross-sectional design was used, which raises a number of issues. First, the temporal ordering of variables in this study cannot be
established (Shadish, Cook, & Campbell, 2002). It is possible that individuals initiated their involvement in delinquency, which led to changes in some of the risk measures included in the measure of cumulative risk. School performance and attachment, substance use, parental relations, and delinquent peers are risk factors that would be particularly likely to be affected by delinquent behavior.

A second problem related to the use of a cross-sectional design is the inability to distinguish different types of offenders. As previously discussed, Moffitt’s (1993) theory of adolescence-limited and life-course persistent offenders suggests that the two types of delinquents may have different trajectories. With regard to differential response to risk, life-course persistent offenders may be exposed to more risks beginning very early in life. In addition to suffering from neuropsychological deficits, Moffitt (1993) explains that these youths are often born into disadvantaged environments that exacerbate their antisocial behavior. Even among life-course-persistent youth that may desire to change, the accumulation of negative experiences (social and legal) would make such a transition difficult. It is likely that these youths begin life more vulnerable than others and also encounter more risks that they are ill-suited to overcome.

Adolescence-limited offenders, however, are likely to be situated in environments that foster resilience. Even after minor delinquent involvement, it is possible for these youths to resume prosocial lives. Based on the hypotheses put forth by Moffitt (1993), one possibility is that these youths exhibit greater vulnerability during adolescence but are resilient during other stages of their lives. Furthermore, their vulnerability is due to both biological processes and the social environment, but their resilience is attributed to normal cognitive development and a positive environment. Examining the response to risk longitudinally could further our
understanding of development in light of the types of risks one encounters throughout his or her life course, the impact of the accumulation of risks across life stages, the duration of exposure to risks, and factors that promote resilience and vulnerability in different types of offenders.

Finally, there are limitations related to the analytical strategy used in this study. Recall from chapter three that three assumptions underlie statistical analyses based on biometrical genetic theory. First, the equal environments assumption (EEA) states identical and fraternal twin pairs will experience the same degree of shared environmental similarity (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Neale & Cardon, 1992; Scarr, 1968; Scarr & Carter-Saltzman, 1979). If identical twins were treated more alike than fraternal twins, and those similar experiences influenced the trait under examination, the EEA would be violated and genetic influences would be overestimated.

There is some evidence to suggest that identical twins are treated more alike than fraternal twins, violating the first criterion of the EEA (Loehlin & Nichols, 1976; Plomin, Willerman, & Loehlin, 1976). A number of studies, however, have suggested that the greater similarity in the treatment of identical twins does not contribute to the greater similarity observed among identical pairs relative to fraternal (Borkenau, Riemann, Angleitner, & Spinath, 2002; Kendler et al., 1993; Plomin et al., 1976; Scarr, 1968; Scarr & Carter-Saltzman, 1979). A strong piece of evidence that suggests the EEA is not violated in twin studies comes from research comparing similarities between twins based on biologically determined zygosity to those based on perceived zygosity (Kendler et al., 1993; Scarr, 1968; Scarr & Carter-Saltzman, 1979). Sometimes twin pairs are misclassified as identical or fraternal, and are treated as a pair of the opposite zygosity. Findings from these studies indicate that perceived zygosity is unlikely to bias twin studies, and that twin resemblance is more strongly associated with biological zygosity.
Research on twins reared apart also indicates that the EEA is a valid assumption in twin studies. If the EEA were violated, identical twins reared apart would be dissimilar because they would not share any of their environmental experiences (Carey, 2003). In fact, the phenotypic correlation between identical twins reared apart would be near zero, smaller than any correlation of siblings reared together, and would be very similar to the correlations observed for fraternal twins reared apart. Previous research, however, has not conformed to these predictions (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Carey, 2003). Given these findings, it is unlikely that the twins in this study were treated differently based on their zygosity in a manner that influenced the response to risk each individual encountered.

The second assumption underlying the study of twins is that there is no assortative mating (Neale & Cardon, 1992). Assortative mating occurs when individuals choose mates that are like themselves. Recall from chapter three that the additive genetic correlation for fraternal twins is assumed to be .5, and this assumption is based on the frequency with which a particular genotype is observed in a population if mating is random. When mates are selected because they possess a given trait or characteristics, mating is nonrandom and the frequencies would be biased. This would make fraternal twins more alike, but would not affect the correlation among identical twins since all of their genetic material is shared. Statistically, this would result in an overestimation of shared environmental effects and an underestimation of additive genetic effects because the difference in identical and fraternal pair correlations would be reduced (Neale & Cardon, 1992). It is possible that this assumption was violated in this study, but research has shown that bias in estimates of additive genetic and shared environmental influences due to assortative mating is very small (Maes et al., 1998).
The final assumption of twin studies is that there are no gene-environment interactions influencing the phenotype being examined (Eaves et al., 1977; Jinks & Fulker, 1970; Neale & Cardon, 1992). A gene-environment interaction occurs when a given expression of one’s genotype is observed under particular environmental conditions, but not in others. This possibility can be explored by examining pairs in which both twins, one twin, or neither twin in a pair are exposed to a particular environmental condition, or through the inclusion of genotypic information (Evans et al., 2002).

There is evidence to suggest that the manner in which individuals respond to specific risks is influenced by their genotype (Bakermans-Kranenburg et al., 2008; Bakermans-Kranenburg & Van Ijzendoorn, 2011; Beach et al., 2010; Beaver et al., 2009, 2010, 2012; Brody et al., 2009; Caspi et al., 2002; Cicchetti et al., 2012; DeLisi et al., 2009; Edwards et al., 2010; Fergusson et al., 2011; Foley et al., 2004; Hart & Marmorstein, 2009; Kim-Cohen et al., 2006; Lee, 2011; Reif et al., 2007; Simons et al., 2011, 2012; Tikkanen et al., 2010; Weder et al., 2009; Widom & Brzustowicz, 2006). The primary aim of this study, however, was to examine the genetic and environmental influences on the response to overall risk. According to the differential susceptibility perspective, individuals are born with a given sensitivity level based primarily on their genetic makeup, but sensitivity is also influenced by early environmental experiences (Belsky et al., 2007; Ellis et al., 2011). Theoretically then, gene-environment interactions that would influence sensitivity would most likely be present during early childhood and not adolescence. Still, the possibility has not fully been explored during adolescence, and future research should seek to determine whether general sensitivity is influenced by gene-environment interactions occurring during adolescence.
Implications and Conclusion

The findings of this study show that the association between risk and behavior is complex. Several scholars have examined this relationship by examining the impact various risk factors or the accumulation of risk factors has on the development of delinquency. Exploring the possibility that individuals may respond to those risks differently can advance our understanding of how risk factors are related behavioral outcomes (Wikström, 2008). The results of this research reveal that there is substantial variation in the ways in which individuals respond to risk, and this variation is influenced by both genetic and environmental factors.

Many leading criminological theories ignore or dismiss the possibility of genetic influences on behavior in favor of sociological explanations (Beaver, 2008). Even those that acknowledge the prospect of genetic influences have, at times, consciously failed to investigate their role on the grounds that one’s genetic makeup cannot be changed. This perspective is fundamentally flawed. It assumes that all risk factors must be altered in order to create a change in behavior, but this may not be the case. Several risk factors for delinquency, such as being male, cannot be changed. Still, researchers continue to investigate such risk factors. For example, how gender might influence the impact of particular risk factors on the development of delinquency is investigated, not because it is possible to change gender but because it is possible to better understand the relationship between risks and outcomes. This information can, in turn, be used to develop interventions that are appropriate for individual offenders.

Similarly, genetic factors cannot be ignored if the aim is to gain a complete understanding of the development of criminal behavior (Walsh, 2002). Instead, criminologists are encouraged to consider both genetic and environmental factors that may account for the differences in responses to risk. The findings presented here indicate that genetic factors made a
moderate contribution to differences in all forms of delinquency in response to risk for males, and a small contribution to differences in violent responses to risk in females. Environmental factors explained most of the variation in response to risk of all forms of delinquency among females, and a portion of the variation in nonviolent delinquency among males. The considerable difference in results by gender and crime type further illustrates the complexity of the risk-delinquency relationship. Identifying genetic factors that moderate the relationship between risk and delinquency may ultimately improve prediction, particularly in males. The association between risk and delinquency among girls, however, appears to be influenced more by social factors.

Although there are practical and ethical considerations that will limit the use of genetic information in the development of prevention and intervention strategies, these findings indicate that it may not be realistic to assume that blanket approaches to reform offenders will be widely effective. In fact, recent studies have found that some interventions are particularly effective with individuals possessing certain genotypes (Bakermans-Kranenburg et al., 2008; Beach et al., 2010). This suggests that genotype may be an important aspect of responsivity to treatment. Vulnerability for overall and violent delinquency was largely attributed to genetic influences in the current study, suggesting that some individuals are more sensitive to the risks they encounter. Differential susceptibility theory contends that those that are more sensitive may be more responsive to positive and negative experiences, and could display very different outcomes (Ellis et al., 2011). Placing these individuals in appropriate programming could result in positive change, while those that are less sensitive may be less responsive to treatment. An important area for future research then will be to extend beyond identifying programs that are effective, but to determine for whom various programs are effective. Similar to understanding the mechanisms
by which risk and delinquency are related, it will be important to consider the mechanisms underlying exposure to treatment and outcome.
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APPENDIX A

Description of Risk and Delinquency Measures

School Performance

1. At the most recent grading period, what was your grade in English or language arts?
2. And what was your grade in mathematics?
3. And what was your grade in history or social studies?
4. And what was your grade in science?

Response categories: 1=A, 2=B, 3=C, 4=D or lower

Attachment to School

Since the school year started, how often did you have trouble:
1. getting along with your teachers?
2. paying attention in school?
3. getting your homework done?
4. getting along with other students?

Response categories: 0=never, 1=just a few times, 2=about once a week, 3=almost everyday, 4=everyday

How much do you agree or disagree with the following statements:
5. You feel close to people at your school.
6. You feel like you are part of your school.
7. You are happy to be at your school.
8. The teachers at your school treat students fairly.
9. You feel safe in your school.

Response categories: 1=strongly agree, 2=agree, 3=neither agree nor disagree, 4=disagree, 5=strongly disagree

Problem Solving Skills

1. When you have a problem to solve, one of the first things you do is get as many facts about the problem as possible.
2. When you are attempting to find a solution to a problem, you usually try to think of as many different ways to approach the problem as possible.
3. When making decisions, you generally use a systematic method for judging and comparing alternatives.
4. After carrying out a solution to a problem, you usually try to analyze what went right and what went wrong.

Response categories: 1=strongly agree, 2=agree, 3=neither agree nor disagree,
4=disagree, 5=strongly disagree

Coping Skills

1. You usually go out of your way to avoid having to deal with problems in your life.
2. Difficult problems make you very upset.
3. When making decisions, you usually go with your “gut feelings” without thinking too much about the consequences of each alternative.

Response categories: 1=strongly agree, 2=agree, 3=neither agree nor disagree, 4=disagree, 5=strongly disagree

Attachment to Parents

1. How close do you feel to your mother?
2. How much do you think she cares about you?
3. How close do you feel to your father?
4. How much do you think he cares about you?

Response categories: 1=not at all, 2=very little, 3= somewhat, 4=quite a bit, 5=very much

Parental Involvement

Which of the following have you done with your mother/father in the past 4 weeks?
1. gone shopping
2. played a sport?
3. gone to religious or church-related event?
4. talked about someone you’re dating or a party you went to?
5. gone to a movie, play, museum, concert, or sports event?
6. had a talk about a personal problem you were having?
7. had a serious argument about your behavior?
8. talked about your school work or grades?
9. worked on a project for school?
10. talked about other things you’re doing in school?

Response categories: 0=no, 1=yes

Parental Engagement

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

Response categories: 1=strongly agree, 2=agree, 3=neither agree nor disagree, 4=disagree, 5=strongly disagree

**Parental Supervision**

1. How often is she [mother] home when you leave for school?
2. How often is she home when you return from school?
3. How often is he [father] home when you leave for school?
4. How often is he home when you return from school?

Response categories: 1=always, 2=never, 3=some of the time, 4=almost never, 5=never

**Delinquent Peers**

1. Of your 3 best friends, how many smoke at least 1 cigarette a day?
2. Of your 3 best friends, how many drink alcohol at least once a month?
3. Of your 3 best friends, how many use marijuana at least once a month?

Response categories: 0=no friends, 1=one friend, 2=two friends, 3=three friends

**Social Support**

1. How much do you feel that adults care about you?
2. How much do you feel that your teachers care about you?
3. How much do you feel that your parents care about you?
4. How much do you feel that your friends care about you?
5. How much do you feel that people in your family understand you?
6. How much do you feel that you and your family have fun together?
7. How much do you feel that your family pays attention to you?

Response categories: 1=not at all, 2=very little, 3=somewhat, 4=quite a bit, 5=very much

**Overall Delinquency**

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 did not belong to you?
3. take something from a store without paying for it?
4. get into a serious physical fight?
5. hurt someone badly enough to need bandages or care from a doctor or nurse?
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. take part in a fight where a group of your friends was against another group?

Response categories: 0=never, 1=1 or 2 times, 2=3 or 4 times, 3=5 or more times

During the past 12 months, how often did each of the following things happen?
1. You pulled a gun or knife on someone.
2. You shot or stabbed someone.

Response categories: 0=never, 1=once, 2=more than once

**Violent Delinquency**

In the past 12 months, how often did you:
1. get into a serious physical fight?
2. hurt someone badly enough to need bandages or care from a doctor or nurse?
3. use or threaten to use a weapon to get something from someone?
4. take part in a fight where a group of your friends was against another group?

Response categories: 0=never, 1=1 or 2 times, 2=3 or 4 times, 3=5 or more times

During the past 12 months, how often did each of the following things happen?
1. You pulled a gun or knife on someone.
2. You shot or stabbed someone.

Response categories: 0=never, 1=once, 2=more than once

**Nonviolent Delinquency**

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 did not belong to you?
3. take something from a store without paying for it?
4. drive a car without its owner’s permission?
5. steal something worth more than $50?
6. go into a house or building to steal something?
7. sell marijuana or other drugs?
8. steal something worth less than $50?

Response categories: 0=never, 1=1 or 2 times, 2=3 or 4 times, 3=5 or more times