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Analyzing the Biosocial Selection into Life-Course Transitions

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# TABLE OF CONTENTS

## LIST OF TABLES

viii

## LIST OF FIGURES

x

## ABSTRACT

xii

## 1. STATEMENT OF THE PROBLEM

1.1 Research Questions 2

1.2 Outline 3

## 2. INTRODUCTION TO BIOSOCIAL CRIMINOLOGY

2.1 Behavioral Genetic Research Methods 8

2.1.1 Twin Studies – Estimating $h^2$, $c^2$, $e^2$ 10

2.1.2 Family-Based Studies 17

2.1.3 Adoption Studies 18

2.1.4 Nonshared Environment Studies 19

2.1.5 MZ Twin Reared Apart 20

2.1.6 Molecular Genetics 22

2.1.7 Discussion 23

2.2 Behavioral Genetic Research on Delinquent, Criminal, and Antisocial Behavior 24

2.3 Gene-Environment Interplay 30

2.3.1 Gene-Environment Interaction (GxE) 31

2.3.2 Gene-Environment Correlation (rGE) 37

2.3.2.1 Passive rGE 38

2.3.2.2 Evocative rGE 39

2.3.2.3 Active rGE 41

2.4 Summary and Discussion 42

## 3. LIFE-COURSE CRIMINOLOGY: FINDINGS AND THEORETICAL CONSIDERATIONS

46
3.1 Sampson and Laub's Life-Course Theory 48
3.2 Criminological Evidence for Sampson and Laub's Theory 54
  3.2.1 The Onset of Adolescent Delinquency 54
  3.2.2 State Dependence and Continuity in Criminal Behavior 58
  3.2.3 Adult Social Bonds and Changes in Criminal Behavior 59
    3.2.3.1 Marriage and Changes in Criminal Behavior 64
    3.2.3.2 Employment and Changes in Criminal Behavior 69
    3.2.3.3 Military Involvement and Changes in Criminal Behavior 75
    3.2.3.4 Religious and Educational Bonds and Changes in Criminal Behavior 78
3.3 Summary and Discussion 80

4. JUXTAPOSING LIFE-COURSE CRIMINOLOGY WITH BEHAVIORAL GENETICS 87
  4.1 Genetic Influences on Life-Course Transitions 90
    4.1.1 Active rGE: Marriage 91
    4.1.2 Active rGE: Employment 93
    4.1.3 Active rGE: Military 94
    4.1.4 Discussion 95
  4.2 Genetic Influences on Changes in Antisocial Behavior Over Time 96
  4.3 Summary and Discussion 100

5. METHODS 103
  5.1 The Data 103
  5.2 Measures 108
    5.2.1 Adult Social Bonds: Relationships 108
    5.2.2 Adult Social Bonds: Military and Employment 110
    5.2.3 Adult Social Bonds: Community Service and Religion 112
    5.2.4 Delinquency, Drug Use, and Antisocial Behavior 114
    5.2.5 Desistance from Delinquency, Drug Use, and Antisocial Behavior 118
    5.2.6 Control Variables 119
  5.3 Analytic Plan 120
    5.3.1 Research Question 1: Do Genetic Factors Influence Exposure to Adult Social Bonds Among a Nationally Representative Sample of Siblings? 120
    5.3.2 Research Question 2: Are Genetic Factors Important for Explaining Changes in Delinquency, Drug Use, and Antisocial Behavior Over Time? 125
    5.3.3 Research Question 3: Are Genetic Factors Important for Explaining Desistance from Delinquency, Drug Use, and Antisocial Behavior? 127
    5.3.4 Research Question 4: Do Adult Social Bonds Explain Desistance from Delinquency, Drug Use, and Antisocial Behavior? 128
    5.3.5 Research Question 5: Do Genetic Factors Reduce or Eliminate the Relationship between Adult Social Bonds and Desistance? 128
6. FINDINGS

6.1 Research Question 1: Do Genetic Factors Influence Exposure to Adult Social Bonds Among a Nationally Representative Sample of Siblings? 130

6.2 Research Question 2: Are Genetic Factors Important for Explaining Changes in Delinquency, Drug Use, and Antisocial Behavior Over Time? 133

6.3 Research Question 3: Are Genetic Factors Important for Explaining Desistance from Delinquency, Drug Use, and Antisocial Behavior? 135

6.4 Research Question 4: Do Adult Social Bonds Explain Desistance from Delinquency, Drug Use, and Antisocial Behavior? Research Question 5: Do Genetic Factors Reduce or Eliminate the Relationship between Adult Social Bonds and Desistance? 136

7. SUMMARY AND DISCUSSION 140

7.1 Summary of Findings 140

7.2 Limitations of Current Study 144

7.3 Future Directions 147

REFERENCES 181

BIOGRAPHICAL SKETCH 203
LIST OF TABLES

5.1.1 Levels of Genetic Relatedness for Add Health Pairs 151

5.2.1.1 Descriptive Statistics for Add Health Variables 152

6.1.1 DF Models Predicting Life-Course Transitions (Relationships) 153

6.1.2 DF Models Predicting Life-Course Transitions (Military & Employment) 154

6.1.3 DF Models Predicting Life-Course Transitions (Employment) 155

6.1.4 DF Models Predicting Life-Course Transitions (Community Service & Religion) 156

6.2.1 Zero-Order Correlations Between Wave 1 and Wave 3 Delinquency, Drug Use, and Antisocial Behavior 157

6.2.2 Bivariate Cholesky Models Predicting Stability and Change in Delinquency from Wave 1 to Wave 3 158

6.2.3 Bivariate Cholesky Models Predicting Stability and Change in Drug Use from Wave 1 to Wave 3 159

6.2.4 Bivariate Cholesky Models Predicting Stability and Change in Antisocial Behavior from Wave 1 to Wave 3 160

6.3.1 DF Models Predicting Desistance from Delinquency, Drug Use, and Antisocial Behavior 161

6.4.1 SSSM and DF Logit Models Predicting Desistance from Delinquency (Relationships) 162

6.4.2 SSSM and DF Logit Models Predicting Desistance from Delinquency
6.4.3 SSSM and DF Logit Models Predicting Desistance from Delinquency (Employment) 164

6.4.4 SSSM and DF Logit Models Predicting Desistance from Delinquency (Community Service & Religion) 165

7.1.1 Summary of Results 166
LIST OF FIGURES

2.1.1.1 Heuristic Data Showing the Intraclass Correlations for MZ and DZ Twins on a Phenotype that is Completely Explained by Genetic Influences 167

2.2.1 Estimates of Heritability and Environmental Influences on Antisocial Behaviors from Four Meta-Analyses 168

2.3.1.1 Graphical Depiction of the Double Helix 169

2.3.1.2 Graphical Depiction of a GxE 170

2.3.1.3 Graphical Depiction of "Plasticity" Alleles 171

3.1.1 Age-Crime Curve for Total Number of Offenses Committed in All Categories in 2008 172

3.3.1 The Age-Crime Curve and the Relationship between Age and Marriage Probability 173

5.3.1.1 Balloon Diagram of the Univariate ACE Model 174

5.3.2.2 Balloon Diagram of the Bivariate Cholesky Model 175

6.1.1 Cascade of Tetrachoric Correlations for Married Ever Variable 176

6.1.2 Cascade of Tetrachoric Correlations for Employment Variables 177

6.1.3 Cascade of Tetrachoric Correlations for Community Service Variable 178

6.2.1 Genetic and Environmental Influences on Stability and Change in Delinquency, Drug Use, and Antisocial Behavior 179
6.3.1 Cascade of Tetrachoric Correlations for Desistance Variables 181
ABSTRACT

Over the past 30 years, scholars have increasingly focused on the individual-level factors that explain criminal behavior. This line of research has revealed that myriad factors influence the onset of a criminal career, the maintenance of a criminal career, and the desistance from a criminal career. The current study focused on the factors that account for desistance from a criminal career. One of the most prominent contemporary criminological theories posits that exposure to adult social bonds such as marriage, employment, and military involvement explains why a person desists from crime. Criminological research has supported the theory, but has failed to consider the influence of genetic factors on exposure to adult social bonds and, ultimately, desistance from crime. Three key findings emerged from the analysis. First, genetic factors explained a significant proportion of the variance in nearly all of the adult social bonds analyzed. Second, genetic factors explained a significant proportion of the variance in changes in delinquency, drug use, and antisocial behavior from adolescence to adulthood. Third, once genetic factors were controlled, the explanatory power of an adult social bond on desistance from delinquency was often weakened or eliminated. The theoretical implications of these findings are discussed.
CHAPTER 1

STATEMENT OF THE PROBLEM

As a discipline, criminology has been dominated by sociological (i.e., environmental) explanations of human behavior. Since Durkheim famously proclaimed that, "The determining cause of a social fact should be sought among antecedent social facts and not among the states of the individual consciousness" (Walsh, 2002:3 citing Durkheim 1982:134), social scientists have argued that social forces override and are more important than biological and genetic forces. As a result, criminologists assume that the forces of nature and nurture are mutually exclusive and that factors of socialization—i.e., nurture—are most important. This reasoning, however, is no longer tenable (Sameroff, 2010). There is now mounting evidence flowing from an emerging perspective known as biosocial criminology which suggests that genetic factors confound the effects of many classic criminological variables (Beaver, 2009a; Rowe and Osgood, 1984; Walsh, 2002). As a result, the hegemonic status of sociological explanations of behavior is beginning to weaken.

For the last 25 years, a sizable body of research examining biological and genetic influences on many human behaviors has developed. This body of research has revealed that antisocial behavior is influenced by genetic factors. In other words, genetic factors are important for understanding the etiology of criminal and delinquent behaviors (Raine, 1993). This knowledge base has, unfortunately, been ignored by criminologists. Perhaps the most prominent example of this oversight can be found in Gottfredson and Hirschi's (1990) A General Theory of Crime. After reviewing the literature on genetic influences on criminal behaviors, the authors concluded that, "the magnitude of the 'genetic effect,' as determined by adoption studies, is near zero" (p. 60). However, as noted by Raine (1993), Gottfredson and Hirschi ignored a host of
studies which presented evidence to the contrary. Prior to 1993, there were at least a dozen studies that reported evidence of a genetic influence on criminal and delinquent behaviors. Since this time, even more research has emerged. As a result, there are now hundreds of studies that show genetic influences on antisocial behaviors (see the following meta-analyses, Ferguson, 2010; Mason and Frick, 1994; Miles and Carey, 1997; Rhee and Waldman, 2002).

Criminology as a discipline is now at a crossroads (Rafter, 2008). Biosocial research findings must be incorporated into criminological research. The risk of not doing so is that some criminological explanations of human behavior will be incomplete (Beaver, 2009a; Wright and Boisvert, 2009). One way to avoid this outcome, however, is for criminologists to recognize the various benefits that biosocial criminology affords criminology (Wright and Boisvert, 2009). Perhaps the most important benefit is that biosocial research can be employed to falsify extant criminological theories.

1.1 RESEARCH QUESTIONS

One of the most prominent contemporary theories of criminal behavior is Sampson and Laub's (1993) life-course theory of informal social control. To date, almost no research has considered the myriad ways in which biosocial research can inform this theory (Beaver, Wright, DeLisi, and Vaughn, 2008a). This dissertation represents a first step toward filling this gap in the literature. Specifically, Sampson and Laub's theory argued that attachment to adult social bonds causes individuals to desist from crime. At the same time, however, a line of research has linked desistance and exposure to adult social bonds with genetic factors (Kendler and Baker, 2007; Reiss et al., 2000). These findings hint at the possibility that Sampson and Laub's theory has identified nothing more than a spurious relationship; a possibility that will be considered by the current analysis.

The current study will examine five research questions that flow from Sampson and Laub's (1993) theory and from recent biosocial research findings. These five research questions are:

(1) Do genetic factors influence exposure to adult social bonds among a nationally representative sample of siblings?
(2) Are genetic factors important for explaining changes in delinquency, drug use, and antisocial behavior over time?
(3) Are genetic factors important for explaining desistance from delinquency, drug use, and antisocial behavior?
(4) Do adult social bonds explain desistance from delinquency, drug use, and antisocial behavior?
(5) Do genetic factors reduce or eliminate the relationship between adult social bonds and desistance?

Although more attention will be given to each of these questions and the research that has motivated them in later sections, it is important to note two points at this time. First, Research Questions 1-4 have been examined by prior researchers. This means that evidence bearing on these questions currently exists. As will be discussed, however, some of these knowledge bases have been built upon shaky foundations. In other cases, only a limited number of studies have emerged that examined the specific research question. In short, it is important to keep in mind that, while Research Questions 1-4 have been examined by prior researchers, none have been answered with a consensus in the literature. It is also noteworthy that desistance (Research Question 3 and 4) is a form of change in antisocial behavior (Research Question 2). As will be discussed later, however, the two terms are not synonymous and the analytic approaches to modeling desistance differ from those necessary to model changes in behavior.

Second, Research Question 5 represents a novel line of inquiry. No research has examined the impact of adult social bonds on desistance after genetic factors have been controlled. The fact that this research question has gone unexamined by criminologists is not because current evidence suggests the answer is "no." As will be shown in the following chapters, the most likely reason that this question has gone unexamined is because it has never been asked. Criminologists have been quick to write-off the importance of genetic effects on behavioral change (Reiss et al., 2000). This inattention has led to the oversight of some important research questions.

1.2 OUTLINE
The following chapters of this dissertation will present an overview of behavioral genetic research, an overview of Sampson and Laub's (1993) theory, and an overview of the behavioral genetic research that has a bearing on this theory. Much information will be presented and, therefore, it is important to highlight the organizational structure of each chapter. Presented in Chapter 2 is an overview of biosocial criminology. Chapter 2 will begin with a discussion of the standard social science method (SSSM) and the problems that it entails. The discussion will then turn to behavioral genetic research methods and how they can be used to overcome the primary limitation of the SSSM. Behavioral genetic research methods are utilized by biosocial criminologists to examine genetic and environmental effects on various outcomes. The full constellation of behavioral genetic research methods available to researchers will be presented in the second section of Chapter 2. Attention will be directed to the method that will be employed in the current study (i.e., the family-based research design). The final section of Chapter 2 will introduce the concept of gene-environment interplay. The various ways in which genetic and environmental factors interact and affect one another in the development of human behavior are considered in this section.

Chapter 3 turns attention to Sampson and Laub's (1993) theory. The first part of Chapter 3 will introduce the theory as it was presented by Sampson and Laub (1993). The second section of Chapter 3 will review the criminological evidence bearing on this theory. Detailed attention will be given to the line of research that has examined the impact of adult social bonds on desistance from crime. The final section of Chapter 3 will present several theoretical arguments that problematize Sampson and Laub's explanation of desistance. This discussion will pave the way for the findings presented in Chapter 4 and the current study.

Presented in Chapter 4 is an overview of the behavioral genetic research that has a bearing on Sampson and Laub's (1993) explanation of desistance. The chapter opens with an overview of the behavioral genetic research that has examined the genetic influences on exposure to adult social bonds. The second section reviews the behavioral genetic literature that has examined the impact of genetic factors on changes in antisocial behavior over time. It is a common misconception that genetic influences are fixed at conception and, therefore, can only be used to explain stability in behavior. The second section of Chapter 4 will present recent evidence showing that this is not the case. Chapter 4 will close with a more thorough discussion of the five research questions that will be analyzed by the current study.
Chapter 5 outlines the analytic methods that will be used to answer the five research questions presented above. The first section of Chapter 5 discusses the data that will be analyzed. Next, the measures that will be analyzed are presented in detail. Finally, Chapter 5 will present the analytic plan. This section will detail the analytical methods that will be employed to answer each of the five research questions.

The findings from these analyses are presented in Chapter 6. Chapter 6 is broken into four sections. Each section presents the findings for one research question, with the exception of the fourth section, which presents the findings from research question 4 and 5 together.

The final chapter, Chapter 7, contains three sections. The first section will summarize the results presented in Chapter 6. This section will also contextualize the findings within the larger theoretical framework that is presented in Chapters 2 through 4. The second section of Chapter 6 will discuss the major limitations that could not be addressed in the analysis. Finally, the third section will offer a brief discussion the implications of the current work for future research into life-course criminology.
CHAPTER 2
INTRODUCTION TO BIOSOCIAL CRIMINOLOGY

Much of criminological research analyzes individual-level data to test hypotheses about the causes of crime (Beaver, 2008b; Harris, 1995, 1998; Rowe, 1994; Weisburd and Piquero, 2008; Wright and Beaver, 2005). To gather the data necessary to analyze these types of hypotheses, researchers commonly rely on the standard social science method (SSSM; Harris, 1995, 1998). The SSSM can be defined as any method of data gathering and/or data analysis that does not allow the researcher to account for genetic influences. In the case of family-based research, the SSSM directs the researcher to examine one child per household to test their hypotheses. For example, a researcher interested in estimating the impact of parental attachment on involvement in delinquency would first identify a sample of families. After identifying the families to be sampled, the researcher would gather information from one child and one parent—most commonly the mother—from each family. To gather information from more than one child per household is rarely considered, likely due to the statistical assumptions that would be violated (i.e., uncorrelated error terms) and the biases that might arise. For these reasons, criminologists often limit their analysis to one child per household. Once information was gathered from all families, the researcher would then correlate measures of parental attachment with the child's delinquency. Any association between the two variables would be explained as an effect of parental attachment on the child’s involvement in delinquency (i.e., Gottfredson and Hirschi, 1990; Hirschi, 1969; Loeber and Stouthamer-Loeber, 1986; Patterson, 1982).

By some accounts, more than 99 percent of criminological research examining the individual-level factors that explain variation in criminal behavior employs SSSMs (Beaver,
2009a). This figure should not give the impression, however, that SSSMs are the best method for every purpose. In fact, SSSMs may generate results that are systematically biased. The reason for this systematic bias is that SSSMs do not allow the researcher to control for genetic influences on the outcome (i.e., omitted variables bias). In other words, the problem is one of spuriousness with genetic factors acting as the omitted third variable. Since genetic factors cannot be controlled, research employing an SSSM rests on the assumption that the biasing effects of omitting genetic effects are zero. This assumption will not be important if genetic effects actually are zero. However, if genetic effects are nonzero, the results produced by an SSSM may be biased. The question most relevant for the current discussion, then, is whether the genetic effect on criminal behavior is nonzero. Although an extensive review of the literature bearing on this issue will be presented later, suffice it to say that genetic effects are important for predicting variation in nearly all human behaviors studied so far (Plomin, 1990), including criminal and antisocial behaviors (Ferguson, 2010; Mason and Frick, 1994; Miles and Carey, 1997; Rhee and Waldman, 2002; Walters, 1992).

It should be noted that the omission of genetic effects from criminological research will not bias the estimates of the predictor variables—typically environmental variables such as parenting effects—unless they too are correlated with the same genetic factors that predict the outcome (e.g., antisocial behavior). Unfortunately, since nearly all criminological research has been conducted with SSSMs, it is impossible to know whether genetic effects confound the estimates of the variables commonly studied by criminologists (Plomin and Bergeman, 1991). Thus, the important point to bear in mind is that any researcher employing an SSSM to study individual variation in criminal, delinquent, or antisocial behavior is likely omitting an important third variable. This is perhaps most important when considering the effects of parenting on child outcomes. In order to determine whether genetic factors are confounding estimates produced by SSSMs, researchers must utilize different methods that allow both environmental and genetic influences to be estimated simultaneously. In other words, criminologists must begin to address their research questions employing behavioral genetic research methods so that genetic effects can be separated from environmental effects.

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1 This point applies only to individual-level research examining the factors that explain variation in antisocial behavior.
Since genetic factors are important for understanding the etiology of criminal and antisocial behavior, an important question emerges: are the findings from criminological research biased due to their omission of genetic effects? At this point, a simple answer to this question is not available. It is currently unclear how much and to what extent criminological research may be biased by the omission of genetic effects. However, the following discussion will attempt to address this issue. In order to do so, an overview of behavioral genetic research methods will be presented first. This review will provide the reader with a thorough understanding of the concepts and issues germane to behavioral genetic research.

2.1 BEHAVIORAL GENETIC RESEARCH METHODS

Behavioral genetic researchers employ statistical analyses to study the genetic and environmental influences on phenotypes (Walsh and Beaver, 2009). A phenotype can be defined as any measurable trait or behavior such as levels of self-control or involvement in delinquency. Researchers are often interested in predicting differences—or variation—in phenotypes across people. For example, a researcher may wish to explore the variation in levels of self-control across a sample of adolescents. The researcher would calculate the variance that exists in the sample and then use statistical analyses to determine the factors that account for, or explain, the observed variance.

Behavioral geneticists analyze phenotypes by estimating the amount of variance that is explained by environmental and genetic factors. To do so, variance in a phenotype is decomposed into genetic and environmental components. Specifically, the variance in a phenotype is decomposed into three latent components: heritability \( h^2 \), shared environment \( c^2 \), and nonshared environment \( e^2 \). The heritability component \( h^2 \) measures the amount of variance in the phenotype that can be attributed to differences in genetic material between the individuals in the study. For example, a heritability estimate of .25 would mean that one-fourth of the variance in the phenotype is attributable to differences in genetic material between the respondents in the sample. For ease of interpretation, researchers often transform heritability estimates into percentages by multiplying the estimate by 100. Thus, an estimate of .25 can be interpreted as 25 percent of the variance being explained by differences in genetic factors.
The environmental components (i.e., $c^2$ and $e^2$) estimate the amount of variance in the phenotype that can be attributed to environmental factors. It is important to point out that behavioral geneticists distinguish between two types of environmental influences: shared ($c^2$) and nonshared ($e^2$). The shared environment captures any environmental influence that makes two siblings more alike. The logic is simple: any experience that is shared by siblings and makes them more alike is a shared environmental influence. Growing up in poverty and experiencing a divorce are two examples of shared environmental influences. Nonshared environmental influences, on the other hand, capture environmental effects that make siblings different from one another. As the name implies, nonshared environments are events that are not experienced by both siblings. Nonshared environments may also arise when siblings experience the same objective event but have different subjective interpretations or perceptions of the event (Turkheimer and Waldron, 2000). Examples of nonshared environments include exposure to different peer groups, exposure to different teachers, and experiencing different parenting strategies (i.e., parents treating their children differently). Measurement error is also captured by the nonshared environmental component.

In quantitative behavioral genetics, the three components ($h^2$, $c^2$ and $e^2$) are estimated simultaneously as latent traits. In this way, behavioral genetic models do not suffer from the specification problems that afflict SSSMs (i.e., omitted variable biases) because all factors that can influence variation in the phenotype are included in the model. Specifically, 100 percent of the phenotypic variance is always explained by behavioral genetic methods. In order to gain estimates of the three components, researchers must use behavioral genetic research methods. One of the most common strategies analyzes data that include more than one child per household. This is the primary divergence between behavioral genetic methods and SSSMs. Recall that SSSMs only include one child per household. It is for this reason, however, that SSSMs cannot account for genetic influences nor can they be used to estimate the relative impact of shared versus nonshared environmental influences (or at least not using standard behavioral genetic research methods). Instead, SSSMs lump genetic and environmental influences together and do not allow the researcher to examine their relative contributions to variance in the phenotype.

The following section will explain the quantitative genetic theory that underlies behavioral genetic research methods. How behavioral geneticists garner estimates of heritability,
the shared environment, and the nonshared environment will be discussed. Attention will first be
given to the most common methodology used to gain these estimates, twin studies. After a
discussion of twin studies, other behavioral genetic methods will be considered along with the
advantages that each offers in relation to twin studies.

2.1.1 Twin Studies – Estimating $h^2$, $c^2$, and $e^2$

In order to decompose the variance in a phenotype into a heritability component ($h^2$), a
shared environment component ($c^2$), and a nonshared environment component ($e^2$), researchers
must analyze data that includes information from more than one child per household.
Additionally, the researcher must have information on the level of genetic relatedness between
children appearing in the same household. Behavioral geneticists typically satisfy these two
requirements by drawing information from two different types of twin pairs: monozygotic (MZ;
identical) twins and same-sex dizygotic (DZ; fraternal) twins. Studies taking this approach are
referred to as "twin studies."

Twin studies are the most common methodology used by behavioral genetic researchers
because the genetic differences between MZ and DZ twins can be seen as a "natural experiment." Specifically, the logic of twin studies rest on one key piece of information: MZ twins share 100
percent of their genetic material while DZ twins, on average, share 50 percent of their unique
genetic material. Additionally, both types of twins—for the most part—share their environments
as well. For example, both types of twins share a womb (it should be noted, however, that MZ
twins share a placenta which DZ twins do not) and they usually are raised by the same parents.
Thus, MZ twins share twice as much genetic material as DZ twins but the siblings’ rearing
environments are identical. Thus, if MZ siblings resemble one another more closely than DZ
twins, genetic factors are important. For example, imagine a sample of sibling pairs where some
pairs are MZ twins and other pairs are DZ twins. Imagine also that a researcher is interested in
estimating the importance of genetic and environmental factors for explaining the variance in
height among the sampled participants. The researcher would start by estimating the similarity
in height for each MZ twin pair. The next step would be to estimate the height similarities for
each set of DZ twins. Finally, the last step would be to compare the similarity in height among
MZ twin pairs to the similarity in height among DZ twin pairs. If MZ twins more closely
resemble one another than DZ twins, the researcher would have evidence that genetic factors influence height differences within the sample.

In order to measure the similarity between two siblings from a twin pair, researchers typically calculate a correlation coefficient. A correlation coefficient is a number that represents the direction and degree of relatedness between two variables. For example, if respondents that score high on one variable also tend to score high on another variable, those two variables will show a strong positive correlation. The strength and direction of the relationship between two variables, therefore, can be gleaned by observing the correlation between those two variables. Correlation coefficients range from -1.00 (indicating a perfect negative correlation) to 1.00 (indicating a perfect positive correlation). A correlation coefficient of .00 indicates that there is no correlation between the two variables.

Criminologists typically calculate interclass correlation coefficients. Interclass correlations analyze the relationship between two different variables (e.g., height and weight) for each individual in a sample. Behavioral geneticists can use correlation coefficients to assist in the estimation of heritability and environmental influences; however, they must rely on intraclass correlations. Intraclass correlations analyze the relationship between the same variable between two different people (e.g., sibling 1 and sibling 2 from a twin pair). For example an intraclass correlation coefficient could be calculated by correlating twin 1’s height with twin 2’s height. If the intraclass correlation between MZ twins is higher than the intraclass correlation between DZ twins, researchers can conclude that genetic factors are important in explaining the phenotype.

It must also be noted that interclass correlation coefficients are typically squared ($r^2$) to determine the amount of variation in variable 1 that is predicted by variable 2. This process, however, is unique to interclass correlations. When calculating intraclass correlation coefficients, the coefficient is not squared because the issue is not whether twin 1’s score can be predicted by twin 2's score. Instead the issue is whether the twins' scores covary and by how much. The intraclass correlation coefficient is a direct estimation of the proportion of variance that is shared within twin pairs (Jensen, 1971; Plomin, 1990).

Imagine a phenotype that is scripted entirely by genetic factors. In other words, the phenotype is only influenced by genetics and is not influenced by environmental factors. If this were the case, we would expect the intraclass correlation coefficient for MZ and DZ twins to
look like those displayed in Figure 2.1.1. Notice that the intraclass correlation coefficient for MZ twins is 1.00 while the coefficient for DZ twins is .50. This means that 100 percent of the variation in the phenotype is shared for MZ twins. In other words, for MZ twins, knowing twin 1’s score on the phenotype allows the researcher to perfectly predict twin 2’s score on the same phenotype. On the other hand, DZ twins do not share all of the variation in the phenotype. As can be seen, DZ twins only share 50 percent of the variation in the phenotype. For the DZ twins, then, knowing twin 1’s score will not necessarily allow the researcher to predict twin 2’s score.

In reality, the intraclass correlation coefficients are never as clearly distinguished as those presented in this example. In most cases, the intraclass correlation for MZ twins is not a perfect 1.00 and the correlation for DZ twins can be greater than or less than .50. Nonetheless, intraclass correlations can be used to estimate the amount of variance that is explained by genetic and environmental differences.

Once intraclass correlations have been calculated separately for MZ and DZ twins, they can be used to estimate the influence of heritability, the shared environment, and the nonshared environment on phenotypic variance. Equation 2.1 can be used to estimate the amount of variance in a phenotype that is explained by genetic factors.

\[
  h^2 = 2(r_{MZ} - r_{DZ})
\]  

(2.1)

In Equation 1, \( h^2 \) refers to the estimate of the amount of variance explained by genetic factors, \( r_{MZ} \) refers to the intraclass correlation for MZ twins, and \( r_{DZ} \) refers to the intraclass correlation for DZ twins. To estimate the amount of variance explained by genetic factors, a researcher must first calculate an intraclass correlation for MZ twins and then a separate intraclass correlation for DZ twins. Next, the DZ correlation coefficient is subtracted from the MZ correlation coefficient. Finally, this difference is multiplied by 2. The resulting value provides an estimate of the proportion of variance in the outcome that is explained by genetic differences among the respondents in the sample.

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2 All figures and tables can be found at the end of the manuscript.
3 Since DZ twins share only half (.50) of their unique DNA, their intraclass correlation coefficient will only include half of the genetic effects. Since only half of the genetic effects are reflected in the DZ intraclass correlation, the difference between the MZ and DZ intraclass correlation coefficients must be multiplied by 2 (Plomin, 1990; Walsh, 2002).
The intraclass correlation coefficients for MZ and DZ twins can also be used to determine the proportion of variance that is explained by the shared environment (see Equation 2.2):

\[ c^2 = 2(r_{DZ}) - r_{MZ} \]  

(2.2)

In this equation, \( r_{DZ} \) still refers to the intraclass correlation for DZ twins and \( r_{MZ} \) still refers to the intraclass correlation for MZ twins. In order to estimate the amount of variance explained by shared environmental influences, the intraclass correlation for DZ twins is first multiplied by 2 and then the intraclass correlation for MZ twins is subtracted from that value. The resulting value is an estimate of the amount of variance that is explained by shared environmental effects.

Finally, an estimate of the proportion of variance explained by nonshared environmental influences can be gleaned from Equation 2.3.

\[ e^2 = 1 - (h^2 + c^2) \]  

(2.3)

Using the estimates garnered from Equations 2.1 and 2.2, the amount of variance explained by the nonshared environment can also be determined. As shown, the variance explained by genetic factors (\( h^2 \)) is summed with the variance explained by the shared environment (\( c^2 \)) and the resulting value is subtracted from 1. The logic is simple: any variance that is not explained by genetic or shared environmental factors must be due to the nonshared environment. In addition to capturing the effects of the nonshared environment, \( e^2 \) will also capture the variance that is due to measurement error.

Twin-based research designs are, by far, the most common method used by behavioral geneticists. Estimates for heritability, the shared environment, and the nonshared environment can be gained by using the equations introduced above. In recent decades, however, behavioral geneticists have refined these equations to incorporate and account for other, more complex issues and research designs. The logic of twin based research, however, remains the same: if MZ twins resemble one another more closely than DZ twins, genetic factors are important for explaining a portion of the phenotypic variance.

Before moving to a discussion of alternative methods available to behavioral geneticists, it is important to discuss some of the assumptions that underlie twin-based research. Twin based
research designs rely on three key assumptions: the equal-environment assumption (EEA), no assortative mating, and no measurement error. The EEA states that the environmental experiences of MZ twins are no more similar than those experienced by DZ twins. In other words, for behavioral genetic estimates to be accurate, it is necessary to assume that MZ twins are not more alike simply because they have more similar environments. Critics argue that MZ twins look more alike than DZ twins and that these physical similarities may result in their having more similar environmental experiences. In support of the first part of this critique, research has shown that identical twins are often treated more alike than are fraternal twins (Loehlin and Nichols, 1976; Scarr, 1968). Loehlin and Nichols (1976), for example, reported that MZ twins were more likely to be dressed alike, sleep in the same room, and be treated similarly by their parents than DZ twins. In a more recent study, however, Reiss et al. (2000:170) found that reports gleaned from parents about how they treat their children reflect more similarity in treatment than when the adolescent is studied. In other words when parents are asked to report on their own behavior, MZ twins appear to be treated more alike than if the siblings are asked to report on their parent's behavior (see also Goodman and Stevenson, 1991). It is possible this latter finding reflects social desirability biases in parental reports (Plomin and Bergeman, 1991). Nonetheless, assuming that there are some aspects of life that MZ twins experience more similarly than DZ twins, the question becomes whether the more similar treatment experienced by MZ twins has any lasting effect on personality development. If so, heritability estimates may be artificially inflated and shared environmental effects may be artificially deflated.

Behavioral geneticists have carefully considered the potential limitation embodied by the EEA. As a result, several studies have empirically examined the EEA to determine whether it represents a serious flaw of twin-based designs (Cronk et al., 2002; Eaves et al., 2003). Some of the most powerful evidence comes from research that examines twins with mistaken zygosity (Goodman and Stevenson, 1991; Gunderson et al., 2006; Kendler, 1983; Kendler et al., 1993; Scarr and Carter-Saltzman, 1979). Occasionally, twins incorrectly perceive their level of genetic relatedness. For example, MZ twins may incorrectly believe that they are DZ twins or vice versa. Early estimates suggested that approximately one-third of all same-sex twins were misclassified (Scarr and Carter-Saltzman, 1979). More recent studies show that between 4 and 10 percent of twins incorrectly report their zygosity (Gunderson et al., 2006). Although these
cases are infrequent, researchers can analyze them to test the EEA. If the EEA is violated—meaning that MZ twins resemble one another due to more similar treatment from the environment than DZ twins—then misperceptions of zygosity should predict similarity between siblings more so than their actual zygosity level. Specifically, if violations of the EEA influence heritability estimates, DZ twins that believe they are MZ twins should resemble each other more so than DZ twins that correctly perceived themselves as DZs. Although no studies directly examined antisocial behavior, some examined related issues such as psychiatric illnesses, cognitive abilities, and personality characteristics (Kendler et al., 1993; Scarr and Carter-Salzman, 1979). The results from these studies have shown that twins that incorrectly perceive themselves as MZ twins are no more alike than twins that correctly identify themselves as DZ twins (Reiss et al., 2000). In short, these studies have supported the validity of the EEA and heritability estimates are unlikely to be biased by twin-based designs. It is interesting to note, however, that heritability estimates will be deflated as a function of the number of twins with mistaken zygosity appearing in the sample. Thus, while some factors such as violation of the EEA may inflate heritability estimates, there are others that counterbalance these effects by deflating the estimates (Raine, 1993).

One final point regarding the tenability of the EEA should be mentioned. MZ twins may experience more similar environments, but these similarities may reflect MZ twins eliciting more similar environmental responses than DZ twins. This phenomenon will be covered below with our discussion of gene-environment correlations. For now, it should be noted that if environmental similarities are due to MZ twins eliciting more similar environments, the EEA will not be a concern. In this case, the environmental similarities will represent child-driven effects (i.e., child $\rightarrow$ environment) as opposed to environmental effects (i.e., environment $\rightarrow$ child) (see generally Harris, 1995, 1998).

The second assumption that behavioral genetic research relies upon is that assortative mating is not present. Assortative mating occurs when mates select one another based on similar personality traits, similar interests, or similar behaviors. Research has shown, for example, that mates resemble one another on personality traits, antisocial behavior, smoking and drinking habits, and levels of self-control (Agrawal et al., 2006; Boutwell and Beaver, in press; Krueger, Moffitt, Caspi, Bleske, and Silva, 1998; Rhule-Louie and McMahon, 2007). Krueger and colleagues (1998) found that mates tended to resemble one another on involvement in self-
reported delinquency and in their level of exposure to delinquent peer groups. Boutwell and Beaver found that paternal measures of self-control correlated positively and significantly with maternal measures of self-control. Assortative mating poses a potential problem for behavioral genetic research because it increases the genetic similarity of all non-MZ siblings. Specifically, if two mates select one another due to similar levels of self-control (Boutwell and Beaver, in press) and self-control is heritable (Wright and Beaver, 2005), there is a greater probability that the mates will have similar genotypes—at least regarding those genes that affect self-control. If these two mates produce DZ twin offspring, the level of genetic similarity between the twins is likely to be higher than the assumed .50. Thus, to the extent that assortative mating occurs, all non-MZ siblings will have levels of genetic similarity that are higher than that which is assumed by behavioral genetic methods. This may lead to higher concordance rates among non-MZ siblings, which would ultimately translate into deflated heritability estimates.

The third assumption that twin-based methods rely upon is that the phenotype being examined is measured without error. Recall that the estimate of the nonshared environment ($e^2$) captures variation that is due to the nonshared environment as well as variation that is due to random measurement error. This means that a phenotype that is measured with greater reliability will produce more reliable estimates of heritability, the shared environment, and the nonshared environment. Since behavioral genetic methods account for 100 percent of the variance in the phenotype, and the estimates gleaned from these models describe the proportion of variation explained by each factor, a greater degree of measurement error will inflate the nonshared environmental estimate. This means that, as a function of measurement error, less variation can be attributed to heritability and the shared environment. The problem that measurement error imposes is not unique to behavioral genetic methods. It is, however, important that researchers understand the consequences of using unreliable measurement techniques.

These three assumptions underlie all twin-based research. The EEA is commonly cited by critics as a potential pitfall of twin research. Research has supported the tenability of the EEA, making it unlikely that the findings from twin research reflect these purported biases. Nonetheless, to the extent that the EEA is violated, heritability estimates will be inflated and estimates for the shared and nonshared environment will be deflated. Consider, however, the effect that violation of the other two assumptions will have on heritability estimates. Any level of assortative mating will decrease heritability estimates. Research has shown that assortative
mating occurs for many of the traits of interest to biosocial criminologists. The same is true for measurement error; heritability estimates decrease as measurement error increases. It has been established that involvement in crime and delinquency is often measured with error (Thornberry and Krohn, 2000). Thus, when considering the effects of violating each of the three assumptions, the most likely effect is that heritability estimates are slightly deflated (i.e., more conservative estimates).

One potential limitation of twin-based research designs warrants attention. Results from twin-based research may not be generalizable to different types of sibling groups. It may be the case that twin pairs—both MZ and DZ—have experiences that are systematically different than those of regular sibling pairs. Differences in age between regular siblings, for example, may mean that two siblings experience the same event differently. This would not be the case for twin pairs since they are the same age. Similarly, the results from twin-based studies cannot be generalized to samples of singletons (i.e., children that do not have siblings; see Falbo and Polit, 1986).

In response to these issues, behavioral geneticists have developed a number of alternative methods that can be used to estimate the importance of genetic and environmental influences on phenotypic variance. These other methodologies do not rely on MZ/DZ comparisons. Thus, utilizing these alternative methods eliminates the need to rely on the EEA and offers researchers a way to estimate the robustness of findings garnered from twin-based studies. These alternative methodologies, which are discussed below, are known as family-based studies, adoption studies, nonshared environment studies, studies of MZ twins reared apart, and molecular genetic studies.

### 2.1.2 Family-Based Studies

Family-based research designs represent an alternative to twin-based designs. Like twin studies, family studies examine the similarity between siblings raised together. The key difference between family-based studies and twin-based studies is that family studies do not limit their focus to MZ and DZ twins. In this respect, family-based studies are more diverse; MZ twins, DZ twins, full siblings, half-siblings, cousins, and genetically unrelated individuals raised in the same household can all be included in the sample. Family-based designs, therefore, are more generalizable. Additionally, the need to rely on the EEA may be disregarded if MZ twins
are not included as part of the sample. For these reasons, as well as others to be discussed later, the current study will utilize a family-based design.

The equations presented in the previous section can be easily altered to accommodate kinship pairs of varying levels of genetic relatedness. A researcher need only note the level of genetic relatedness for different types of kinship pairs. As previously discussed, MZ twins share 100 percent of their DNA and DZ twins share, on average, 50 percent. Like DZ twins, full siblings share 50 percent of their DNA. For all intents and purposes, DZ twins are just a special type of full siblings—they are full siblings that share a birthday. Since half-siblings have one parent in common, they will share, on average, 25 percent of their DNA. Cousins do not have a parent in common, but their parents are genetically related siblings. For example, if the father for child 1 is the brother of child 2’s father, then child 1 and child 2 should share, on average, 12.5 percent of their genetic material (0.50 x 0.25 = .125).

Family-based studies are less common than twin-based studies because they require additional steps to obtain estimates for each of the three components. For example, since non-twin siblings are included, it is important for researchers to account for differences in age and sex that may exist between sibling pairs. Not controlling for these factors may produce misleading estimates. Also, it is important for researchers employing family-based designs to consider the nature of the relationship between non-related siblings living together. For example, some non-related siblings may be living together because they belong to a group home or are confined to the same juvenile facility. In this case, the siblings are non-related, in the genetic sense of the term, but they are likely to resemble one another due to uncontrolled selection effects. If a researcher did not account for these selection effects between non-related siblings, the final estimates may be biased. Specifically, heritability estimates are downwardly biased and shared environmental estimates are upwardly biased when selection effects cause non-related siblings to resemble one another more closely.

2.1.3 Adoption Studies

Adoption-based research designs represent another way in which behavioral geneticists can estimate heritability and environmental effects without having to rely on the EEA. Like all children, adopted children share 50 percent of their genes with their biological father and 50
percent with their biological mother. Adopted children, however, do not share the environment with their biological parents. Any similarity between adopted children and their biological parents is, therefore, due to shared genetic material. Likewise, adopted children are unlikely to share any genetic material with their adoptive parents but they do share the environment. Any similarities observed between adopted children and their adoptive parents must, therefore, be due to environmental effects (Beaver, 2009a). These effects can be estimated via intraclass correlation coefficients. The correlation between an adopted child and his/her biological parent is a direct estimate of heritability. The correlation between the adopted child and his/her adoptive parents is a direct estimate of shared environmental effects.

Adoption studies are rare in the criminological literature. One reason for this stems from the difficulty of retrieving data from all necessary subjects (i.e., adopted child, adoptive parents, and biological parents). Selective placement by adoption agencies and the timing of the adoption can also be problematic for behavioral genetic research. Specifically, if adoption agencies make an effort to match the adoptive environment with the child’s original environment, it will be difficult to disentangle genetic and environmental effects. Also, if the child is not adopted at birth, environmental and genetic effects will be difficult to separate. However, the extant adoption studies indicate that genetic factors are important in illuminating risk factors for criminal behavior (Mason and Frick, 1994; Mednick et al., 1984; Raine, 1993; Rhee and Waldman, 2002). Often, heritability estimates gleaned from adoption studies are slightly lower than—but within the confidence intervals of—heritability estimates garnered from twin and family studies (Beaver, 2009a; Raine, 1993).

2.1.4 Nonshared Environment Studies

Behavioral genetic research has consistently shown that nonshared environmental influences explain more variance in antisocial behavior than shared environmental influences (Moffitt, 2005; Pike et al., 1996). A unique strategy for identifying specific nonshared environmental influences is to examine environmental differences between MZ twins. Because MZ twins share 100 percent of their genetic material, any divergences in their behaviors must be due to environmental differences. Recall that shared environments work to make siblings more similar. Thus, shared environments cannot explain differences between siblings.
While this approach is acknowledged as the "gold standard" for identifying important environmental influences (Asbury et al., 2003; Beaver, 2008b; Burt, McGue, Iacono, and Krueger, 2006; Caspi et al., 2004; Deater-Deckard et al., 2001; Pike et al., 1996), the logic is quite simple. Since any differences between MZ twins must be due to environmental differences (i.e., nonshared environments), researchers can calculate difference scores between MZ twins to directly measure nonshared environments. More directly, researchers can calculate difference scores between twins on any number of variables, say delinquency and exposure to delinquent peers. If the twin with more delinquent peers reports more involvement in delinquency, this may indicate a nonshared environmental influence on involvement in delinquency.

While the MZ twin difference score approach is useful for examining nonshared environments, it does not allow the researcher to estimate heritability effects. Rather than directly estimating these effects, genetic and shared environmental effects are controlled by the research design. Nonetheless, research using this analytic strategy has revealed a number of environmental factors important for phenotypic expression. This line of research has highlighted the importance of parental expressed emotions in the development of antisocial behaviors among children and adolescents (Beaver, 2008b; Caspi et al., 2004; Pike et al., 1996). Since this analytical approach does not compare MZ and DZ twins, the EEA is not a necessary component.

2.1.5 MZ Twins Reared Apart

Examining MZ twins separated at birth and reared apart (MZA) offers behavioral geneticists many benefits over other research designs. MZAs can be considered a naturally-occurring experiment where two genetically identical people are placed into separate environments. In this way, researchers are able to gain a clearer understanding of nonshared environmental influences on phenotypic variation since MZAs share 100 percent of their genetic make-up but do not share any substantial portion of their environments. Unfortunately, MZAs are extremely rare, making it difficult to use this methodological strategy.

Researchers at the University of Minnesota have an on-going study designed to identify and examine MZAs. Their research has indicated that genetic influences are responsible for roughly 50 percent of the variance in antisocial phenotypes, with the nonshared environment being responsible for the remaining variance. Interestingly, findings flowing from the University
of Minnesota research project show that MZ twins reared together are no more similar than MZAs, further substantiating the importance of genetic and nonshared environmental influences and representing yet another example of the tenability of the EEA for twin-studies (Bouchard et al., 1990). These findings are important for behavioral genetic research for several reasons. First, MZA research designs offer a unique way to analyze the importance of genetic and environmental influences on human development. Second, the findings from these studies have bolstered the findings from research that has employed different research designs. For example, the findings from MZA research have supported the conclusions drawn from twin-based research. Third, and perhaps most importantly, the findings from MZA research support the tenability of the EEA as it applies to twin-based research. Since MZA designs only analyze MZ twins that were raised apart since birth, there is no way that the siblings could have been exposed to identical environments. Thus, any similarity between the siblings must be due to genetic factors. Since the findings from these studies have reached similar heritability estimates, the EEA appears to be a tenable assumption for other twin-based designs.

This does not mean, however, that the MZA research design is free of limitations. There are two critiques that are typically leveled against MZA research. Critics argue that adoptive environments are similar to one another (Beaver, 2009a; Walsh, 2002). For example, twins separated at birth may be reared by different adoptive parents in different households, but due to standardized screening processes carried out by adoption agencies, the environments in which children are placed can be quite homogenous. Walsh (2002:45) estimated the correlation between adoptive homes of MZ twins to be approximately .30. If this is the case, and MZAs are exposed to similar, albeit different, environments, then heritability estimates may be inflated. This critique rests on the assumption that the environments that are similar across adoptive homes actually affect personality development and can account for a portion of observed sibling similarities. Since siblings raised together have a shared environment correlation of 1.0 and the shared environment is nearly always identified as having weak effects, it is questionable whether standardized screening processes lead to significantly inflated heritability estimates.

It is also important to note a key assumption of the MZA design. Specifically, MZ twins, regardless of whether they are reared together or apart, may receive equal treatment from the environment based on their physical similarities. If this were the case, ostensibly nonshared environments would actually be better defined as shared environments. For example, research
has shown that an infant's physical appearance can affect others' reactions to that infant (Harris, 1998). MZAs are likely to physically resemble one another, thus, they may be more likely to receive similar treatment from their environments. Since the MZA design controls shared environments to be zero, these effects would be captured by the heritability component (i.e., inflated heritability estimates). Evidence bearing on this critique can be drawn from Kendler (1983:1419):

…only two studies have compared the personalities of monozygotic twins reared apart and reared together. Both studies found that monozygotic twins reared apart were more similar in personality than monozygotic twins reared together (emphasis added).

This statement suggests that MZ twins reared together may try to emphasize their individuality by acting differently from one another. If this is the case, heritability estimates drawn from twin research will be deflated. At the same time, this reality should lead to increased heritability estimates in MZA designs. Since MZA designs typically lead to heritability estimates that are within the confidence intervals of those gleaned from other designs, it is unlikely that these "similar environments effects" have biased the findings from MZA studies (Beaver, 2009a).

2.1.6 Molecular Genetics

As already discussed, behavioral genetic research typically decomposes the variance in a phenotype into three separate components (i.e., heritability, shared environment, and nonshared environment). One limitation of this research design is that the individual factors that comprise each of the three components remain unidentified. For example, a research study may estimate that genetic factors account for 50 percent of the variance in antisocial behavior. Although this estimate is informative, it does not tell us anything about which genes are driving the effect. In other words, behavioral genetic research designs estimate latent traits that cannot inform scholars about specific risk factors.

With the recent mapping of the human genome researchers are beginning to pull back the "heritability curtain" and identify links between measured genes and phenotypic outcomes. This line of research—referred to as molecular genetics—has already produced a wealth of knowledge. Certain genetic polymorphisms have been linked to well-known predictors of antisocial behaviors such as ADHD (Faraone et al., 2001) and childhood conduct disorder
(Beaver, 2009b; Beaver, Wright, DeLisi, Walsh, et al., 2007). Perhaps more importantly, molecular genetics research has evinced the importance of the environment in "triggering" genetic effects; a process known as gene-environment interaction (GxE). Findings from GxE research show that certain genetic effects are more likely to manifest when combined with environmental risk factors (Beaver, 2008a; Cadoret et al., 1995; Caspi et al., 2002; Moffitt et al., 2006). This line of work has shown that genetic influences can change throughout the life-course as a result of differential environmental exposure. Since molecular genetics research focuses on specific genes and does not rely on MZ and DZ twins for genetic estimates, the EEA is not a concern. We will return to a discussion of GxEs and other forms of gene-environment interplay in a later section of this chapter.

2.1.7 Discussion

This section has reviewed the various methods available to behavioral genetic researchers. As was discussed, each has unique advantages and disadvantages. Some approaches rely on the EEA (e.g., twin-based designs) while others do not (e.g., adoption studies, MZ twin nonshared environments). Some produce estimates for heritability, the shared environment, and the nonshared environment (e.g., twin studies and family studies), while others do not (e.g., molecular genetics). Despite their differences, the estimates garnered from one research design are often quite similar to those produced by other designs (Beaver, 2009a). In this way, behavioral geneticists have been able to triangulate their methods to develop better and more reliable estimates. In the end, however, behavioral geneticists, like criminologists, must consider the research question before determining the analytic strategy to be employed.

Now that the key differences between SSSMs and behavioral genetic methods have been presented, it is important to examine the findings that have emerged from behavioral genetic research. Specifically, to what extent is variation in antisocial and criminal behaviors explained by heritability, the shared environment, and the nonshared environment? The next section will review the evidence bearing on this issue. Since criminologists have been slow to accept and incorporate the findings from behavioral genetic research, it is often necessary to look to disciplines outside of criminology, such as developmental psychology, to find relevant research.
2.2 BEHAVIORAL GENETIC RESEARCH ON DELINQUENT, CRIMINAL, AND ANTISOCIAL BEHAVIOR

Behavioral genetic research can be found across a range of academic foci but has only recently begun to penetrate the criminological discipline. The delay in the emergence of behavioral genetic studies in criminology may be attributable to ideological opposition (Udry, 1995; van den Berghe, 1990; Walsh and Ellis, 2004). Criminologists are typically trained as sociologists and, by virtue of their training, are often resistant to biological or genetic explanations of human behavior. Consistent with this position, Walsh and Ellis (2004) conducted a survey of members of the American Society of Criminology and found that biosocial explanations of crime and deviance were among the least favored by criminologists in 1997. More recently, Wright, Beaver, DeLisi, Vaughn, Boisvert, and Vaske (2008) conducted a systematic review of the criminological discipline and found that very few criminologists specialize in, teach, or even conduct biosocial research. Despite the disregard of behavioral genetic research, the findings from this field are slowly beginning to penetrate the criminological discipline. Some have even argued that biosocial criminology will eventually emerge as a dominant paradigm due to the fact that sociological criminology is growing "stale" (Cullen, 2009).

Recall from the previous section that behavioral genetic research offers quantitative tools that can be used to decompose the variance in a phenotype into genetic and environmental components (see Lemery and Goldsmith, 1999 for a review of behavioral genetic research designs). There is now an extensive line of research that uses these behavioral genetic methods to explain the etiology of antisocial behavior. Since this body of research is vast, one way to summarize the pattern of results is to consider the findings from meta-analyses. Meta-analysis is a tool that researchers often use to summarize a large body of evidence (Cooper, 1998). Researchers can apply quantitative methods to help categorize and synthesize findings from various studies. By using meta-analytic techniques, researches can summarize a large body of evidence by reporting the "average effect size." In the case of behavioral genetic research, an average effect size would refer to the average estimate for each of the three variance components: heritability, shared environment, and nonshared environment. One key benefit of meta-analysis is that studies can be given more "weight" based on methodological rigor. In this
way, the researcher can determine whether studies that employ more rigorous designs lead to different conclusion or different estimates. Meta-analysis is not, however, without limitations. Specifically, meta-analysis combines the findings from studies that may include heterogeneous samples, operationalizations of key variables, and analytic techniques. Additionally, the average effect size is important in summarizing the literature, but this statistic can mask important variation in estimates that exist both within and between studies included in the review. These limitations are important to keep in mind when considering the evidence presented below.

Currently, there are five meta-analyses that have been conducted in order to summarize the behavioral genetic literature focusing on antisocial behavior (Ferguson, 2010; Mason and Frick, 1994; Miles and Carey, 1997; Rhee and Waldman, 2002; Walters, 1992). As can be seen, the meta-analyses were conducted at different intervals in time. As a result, there is limited overlap in the studies that are covered from one review to the next. In addition to these five meta-analyses, there are a number of literature reviews that also summarize the current state of knowledge (Fishbein, 1990; Moffitt, 2005; Raine, 1993).

In one of the first systematic reviews of the behavioral genetic literature bearing on antisocial behavior, Adrian Raine (1993:71) summarized the evidence as of 1993:

One approximate deduction that can be drawn from twin and adoption studies is that genetic influences may well account for roughly half of the explained variance in crime. That is, genetic influences are nontrivial and probably account for as much variance as environmental influences in relation to crime (emphasis in original).

It is quite clear from Raine's comment that the research available in 1993 indicated that genetic differences across individuals was responsible for approximately half of the variance in criminal outcomes. Raine reached this conclusion after reviewing 10 studies using the twin-based method, 2 studies using MZ twins reared apart, and 15 studies using the adoption design.

Raine's (1993) review spoke to the amount of variance in adult crime that can be explained by genetics factors. But, are genetic factors important for explaining juvenile antisocial behavior? Raine's review also addressed this issue. As of 1993, the evidence appeared to indicate that genetic influences were less important for explaining juvenile delinquency than for explaining adult crime. Specifically, of the ten twin studies included in the review, eight indicated a modest genetic effect on juvenile delinquency. Four adoption studies examining juvenile delinquency were also included. Two of these studies presented evidence for genetic
effects on juvenile delinquency. In short, Raine's review indicated that juvenile delinquency is less influenced by genetic factors than is adult crime. This is not surprising when one juxtaposes these findings with the age-crime curve which shows that delinquent behavior in adolescence is virtually normative but that crime in adulthood is not.

Since Raine's (1993) review, there have been five meta-analyses published that offer cogent summaries of the evidence of genetic effects on antisocial behavior. The first meta-analysis of behavioral genetic studies was coauthored by Mason and Frick in 1994. This study analyzed 12 twin studies and 3 adoption studies. The authors started initially with a pool of 70 studies but narrowed the sample to 15 based on five inclusionary criteria. For example, the authors included studies that were published between 1975 and 1991, studies that compared MZ and DZ twins, and studies that examined antisocial behaviors. Studies were removed from the sample primarily due to inconsistent measurement of antisocial behavior between the adoptee and his/her biological parent (this applied to adoption studies only) or because of overlapping samples between two or more studies. The results of the meta-analysis were quite revealing: the average heritability estimate across the 15 studies was .48. Thus, roughly 48 percent of the variance in antisocial behaviors across these 15 studies was explained by differences in genetic factors. Studies that examined severe antisocial behaviors reported larger heritability estimates ($h^2 = .45$) compared to those examining nonsevere antisocial behavior ($h^2 = .00$). Importantly, heritability estimates were not moderated by study characteristics such as the age, gender, or country from which the sample was drawn. Samples drawn from clinical populations appeared to report slightly higher estimates of heritability ($h^2 = .53$) than volunteer samples ($h^2 = .20$).

The second meta-analysis of behavioral genetic research on antisocial behavior was conducted by Miles and Carey (1997). The focus of this meta-analysis was on aggressive behaviors. In all, 24 effect sizes were analyzed. Similar to the approach taken by Mason and Frick (1994), Miles and Carey examined both twin studies ($n = 15$) and adoption studies ($n = 2$). The meta-analytic results indicated that 50 percent of the variance in aggression was explained by differences in genetic material. The authors examined a number of possible moderating influences—including whether studies with greater methodological rigor produced different results—and concluded that age and sex differences across the samples, as well as whether the data were drawn from observations or self-reports were three factors that led to different heritability estimates. Specifically, samples of males and samples containing older respondents
provided larger heritability estimates and smaller estimates of shared environmental influences. The moderating effect of age on heritability estimates is supportive of Raine’s (1993) comment regarding the greater importance of genetic factors for adult crime relative to juvenile delinquency. Additionally, observational ratings of behavior and parental reports of the child’s behavior provided smaller heritability estimates and larger estimates of shared environments as compared to self-reported behavior. The moderating effect of parental versus self-reports may be due in part to parents perceiving a greater degree of similarity among their children and in their treatment of their children (see generally Reiss et al., 2000). It is less clear why the observational ratings produced significantly different heritability estimates. Although heritability estimates appear to be influenced by the methodological decisions and sample composition, it is important to note that the worst fitting models were those that constrained heritability effects to be zero. In other words, heritability estimates may have changed as a result of methodological designs and sample composition, but in no cases were heritability estimates reduced to zero.

The third meta-analysis was published in 2002 by Rhee and Waldman. The focus of this study was antisocial behavior. In order to summarize the behavioral genetic literature focusing on antisocial behavior, the authors restricted their analyses to twin and adoption studies. In all, 51 studies were included in the analysis. Their results indicated that genetic influences accounted for 41 percent of the variance in antisocial behaviors. Like Mason and Frick (1994) and Miles and Carey (1997), Rhee and Waldman examined a number of possible moderating influences including the assessment method used by the original study, the age of the sample, and the method by which zygosity was determined in the original study. These sensitivity analyses indicated that self-reports of antisocial behavior produced smaller heritability estimates, that older samples produced smaller heritability estimates, and that different methods of zygosity determination led to different heritability estimates (i.e., blood testing $h^2 = .47$; questionnaire $h^2 = .43$). The finding that studies relying on self-reports and studies relying on older samples produces smaller heritability estimates stands in contradiction to the findings presented by Mason and Frick (1994) and Miles and Carey (1997). However, their "age" variable reflected the mean or median age of the sample for each study and was coded trichotomously (i.e., children, adolescents, and adults). Furthermore, the authors cautioned that the moderators examined may be confounded with one another. For example, studies that examine reports by
others are more likely to be used for child samples. This may begin to explain the similarity in moderating effects between the age of the sample and the reporting method used. Recognizing this fact, the authors warned that the moderating influence of age should be interpreted with caution. Nonetheless, it should be pointed out that the best fitting model indicated that genetic effects accounted for roughly half of the variance.

The fourth meta-analysis concerning the genetic and environmental effects on antisocial behavior was recently published by Ferguson (2010). Included in his analysis were all studies published between 1996 and 2006. Ferguson's literature search yielded 38 studies which provided 53 separate effect sizes and a combined sample size of 96,918. Estimates revealed that genetic factors accounted for 56 percent of the variance in antisocial behaviors. Additionally, in line with Rhee and Waldman (2002), Ferguson found that heritability estimates were slightly smaller and that nonshared environments were larger for older samples of respondents. This finding, similar to the explanation offered for the results from the Rhee and Waldman review, may reflect the coding scheme used—Ferguson used a trichotomous coding format as well—or may be confounded by other moderators not considered. On the other hand, the finding that heritability effects decrease with age may be an accurate representation of reality. In which case, as Ferguson argued, nonshared environmental effects \((e^2)\) may become more influential in later life due to events such as head injuries or environmentally driven illnesses affecting antisocial behavior.

Finally, the fifth meta-analysis bearing on the genetic-crime relationship was published by Walters (1992) and covered 38 studies. There are several features of this article that are different than those reviewed above. First, unlike the other meta-analyses, this study did not examine heritability coefficients. Instead, a technique for determining the strength and direction of a relationship in a 2 x 2 table know as the phi coefficient was used. Unlike a heritability estimate, the phi coefficient cannot estimate the amount of variation that is due to genetic or environmental factors. Instead, the phi coefficient indicates whether a sibling is more likely to be arrested if his/her sibling has an arrest record. In this way, genetic and environmental effects (both shared and nonshared) are conflated into one estimate. In short, using the phi coefficient is akin to the SSSM discussed above; all of the same limitations apply. It is unclear why the author chose to use a phi coefficient when heritability estimates were already widespread and were available in many of the studies reviewed.
Second, different behavioral genetic research methods were combined without taking into account their differential ability to detect genetic effects. For example, the findings from adoption studies were combined with those from twin and family studies. This is not a problem if heritability estimates are used because heritability estimates are standardized and can be compared across studies. However, concordance rates and phi coefficients will vary as a result of the research design. Phi coefficients are not standardized and, as a result, will fluctuate as a function of the method being used. Third, Walters examined a heterogeneous set of outcome measures. Some studies analyzed arrest records while others analyzed aggression and sociopathy. Since predictable differences exist between objective (i.e., arrest record) and subjective measures (i.e., aggressive behavior) of antisocial behavior, it is unclear how estimates of genetic influence may be biased when all are combined into a single construct. Fourth, family studies were included in the meta-analysis. Although this, in and of itself, is not a limitation of the study, the information that was gleaned from these studies draws the results into question. As outlined above, it is important to account for gender, age, and other differences between family members when employing the family-based research design. Furthermore, it is important to account for the level of genetic relatedness between two siblings when conducting behavioral genetic research. Unfortunately, Walters did not account for any of these effects when calculating the phi coefficient for family studies. As a result, it is unclear whether the phi coefficient for the family studies is an accurate representation of the gene-crime relationship.

Despite the differences in analytic strategy employed by Walters, the results supported the argument that genetic factors influence criminal behavior (phi = .25 and .09). Although the author eschews the importance of heritability estimates in the introductory section of the review, he did ultimately estimate the influence of heritability, the shared environment, and the nonshared environment (Walters, 1992:606). This secondary analysis indicated that heritability estimates ranged between 11 and 43 percent for the studies examined; estimates that fall within the deviation range of estimates garnered from the other four meta-analyses. The degree of accuracy for these estimates is questionable, however, because the author used logical principles that differ from mainstream behavioral genetic research when calculating the estimates. As a result, caution should be used when interpreting the heritability estimates presented in this analysis.
Clearly, much empirical attention has been devoted to understanding the genetic and environmental underpinnings to antisocial behavior. These five meta-analyses combine to cover more than 100 studies and several hundred thousand respondents. Two findings are worthy of further discussion. First, about half (.48, .50, .41, and .56) of the variance in antisocial behavior can be attributed to genetic influences. Second, environmental influences are also responsible for about half (.52, .50, .59, and .44) of the variance in antisocial behaviors. The findings from four of the five meta-analyses are summarized in Figure 2.2.1. The results from Walters's analysis are not included in this figure for the reasons discussed above (i.e., questionable estimation techniques).

Recall, however, that environmental influences are heterogeneous: some are shared between siblings (i.e., shared environment \( c^2 \)) and others are not (i.e., nonshared environment \( e^2 \)). Shared environments make siblings similar to one another. Shared environments typically identified by criminologists include neighborhood factors, parenting effects, and household socio-economic factors. Nonshared environments make siblings different from one another and include factors such as exposure to different peer groups and exposure to different teachers in school. Since environmental influences are not homogenous, a key question logically arises: which are more important for understanding antisocial behavior, shared environments or nonshared environments? Although Figure 2.2.1 does not shed light on this point, we can draw on a recent review by Terrie Moffitt (2005) for insight\(^4\). As summarized by Moffitt (2005), shared environmental influences account for the least amount of variance in antisocial phenotypes, between 15-20 percent. Nonshared environmental influences, on the other hand, account for approximately 30-35 percent of the variance in antisocial phenotypes. Thus, it appears that genetic and nonshared environmental effects are most important for understanding the etiology of delinquency, criminal, and antisocial behavior (Harris, 1995, 1998; Plomin and Daniels, 1987; Plomin and Bergeman, 1991; Rowe, 1990; Turkheimer and Waldron, 2000).

2.3 GENE-ENVIRONMENT INTERPLAY

\(^4\) Not all studies provided estimates for \( c^2 \) and \( e^2 \). Thus, these environmental effects were combined into a single construct for Figure 2.2.1.
Despite the extensive body of research showing the importance of genetic influences on antisocial behaviors, criminologists have yet to incorporate genetic factors into their statistical models. Social factors are viewed as having paramount importance and genetic factors are ignored. This is unfortunate since there is now a considerable amount of research indicating that both nature and nurture are important for understanding human behaviors. Recall from the last section that the results from four meta-analyses show that genetic factors account for roughly half of the variance in antisocial behavior. This means that the environment accounts for approximately half of the variance in antisocial behavior as well. In other words, genetic and environmental factors are important for understanding the etiology of antisocial behavior.

Since behavioral genetic research has shown that genetic and environmental influences are equally important, scholars are beginning to explore the myriad ways in which genes and environments work together in the development of human phenotypes. This line of inquiry is often captured under the heading of "gene-environment interplay." For example, research has shown that many environments previously believed to be "purely" social are at least partially under genetic influence; often referred to as gene by environment correlation (rGE; Moffitt, 2005; Raine, 2002). Other researchers have begun to identify a different form of gene-environment interplay known as gene by environment interaction (GxE; Raine, 2002). The following discussion will focus first on GxE and its importance for criminological research. Specific attention will be given to the logic of GxE and how it relates to antisocial phenotypes. The second section of this discussion will introduce the concepts and logic underlying rGEs.

2.3.1 Gene-Environment Interaction (GxE)

In order to understand GxE, it is first necessary to introduce a number of concepts related to molecular genetics and genetic functioning. Deoxyribonucleic acid (DNA) is the chemical "blueprint" for the formation, development, and functioning of the human body. With the exception of red blood cells, DNA is found in the nucleus of all cells within the body. The DNA "code" is unique to each individual, with the exception of identical twins who share 100 percent of their DNA. DNA comes packaged on what are known as chromosomes. Most individuals have 23 pairs of chromosomes, 46 individual chromosomes in all. Each chromosome consists of different sections of DNA that are wrapped around the chromosome. Structurally, DNA consists
of two genetic fibers (i.e., polynucleotides) that are twisted around one another forming the famous double-helix (see Figure 2.3.1.1). Placed along each of the two polynucleotides (referred to as "sugar phosphate backbone" in Figure 2.3.1.1) are four nucleotides (referred to as "nitrogenous base" in Figure 2.3.1.1): adenine (A), thymine (T), guanine (G), and cytosine (C). The nucleotides from each of the two polynucleotides are bonded together—forming base pairs—by a sugar phosphate. The nucleotides are bonded together in a systematic fashion so that "A" always bonds with "T" and "G" always bonds with "C". Genes are defined as a contiguous set of base pairs working together to code for the production of proteins that will then perform a specialized function. Current estimates place approximately 3 billion base pair sequences in the human genome. This translates into approximately 20,000 genes.

With the exception of genes transmitted on the Y chromosome, individuals inherit two copies of every gene. One set of chromosomes is inherited maternally and the other set is inherited paternally. Recall that most individuals have 23 pairs of chromosomes. One set of chromosomes are referred to as the "sex" chromosomes and are labeled the X chromosome and the Y chromosome. All other chromosomes are referred to as autosomes and are referred to by numerical labels (i.e., 1 through 22). Males receive one Y chromosome from their father and one X chromosome from their mother. Females do not receive a Y chromosome. Instead, females receive two X chromosomes; one from both their mother and father. Genes, therefore, can be found on all 46 chromosomes, including the sex chromosomes. For the vast majority of genes, all members of the population possess the same gene (i.e., there are no allelic differences across individuals). Thus, most genes inherited maternally are identical to those inherited paternally. For some genes, however, more than one copy exists in the population. Alternative copies of genes are referred to as alleles. Genes with two or more alleles (i.e., genes that vary within the population) are referred to as genetic polymorphisms.

Genetic polymorphisms come in three forms. Single nucleotide polymorphisms (SNP)—where a single base pair differs between two alleles—are the most frequently occurring polymorphisms. The second type of polymorphism is known as a microsatellite. Microsatellites occur when one allele is longer in length due to a small number of base pairs being repeated a number of times in one allele but not the other (or the base pair combination is repeated more times in one allele than the other). Finally, minisatellites are the third type of polymorphism. Minisatellites are similar to microsatellites in the sense that the distinguishing feature between
the two alleles is their length. Specifically, one allele is longer than the other due to the repetition of a sequence of base pairs. Minisatellites differ from microsatellites in respect to the number of base pairs that are repeated; minisatellites repeat a larger number of base pairs. The arbitrary cutoff distinguishing a microsatellite from a minisatellites is around 10 or more repeated base pairs.

Recall that current estimates state that the human genome consists of approximately 3 billion nucleotide base pairs and 20,000 genes. For the vast majority of genes, there is only one copy available in the population. However, as outlined above, genes can vary across the population in three ways (i.e., SNP, microsatellite, and minisatellite). Currently, scientists estimate that between .1 and 10 percent of the 3 billion base pairs differ between individuals. This means that only a small proportion of genes in the human genome vary between individuals. Thus, when scholars talk about genetic differences between individuals (e.g., siblings), they are referring to the genes that have more than one allele in the population (i.e., polymorphic genes). When scientists claim that DZ siblings share 50 percent of the genetic material, for example, they are referring to the genetic material that varies in the population (i.e., their "unique" DNA).

A hypothetical example will help to clarify the concept of alleles and genetic polymorphisms. Imagine that a contiguous strand of base pairs work together to code for hair color (i.e., a "hair color gene"). Suppose that two copies of the hypothetical hair color gene exist in the population. These two copies are distinguished by a single nucleotide base pair difference (i.e., a SNP). One allele has the following genetic sequence:

\[\text{AACTGTAACCG} \quad (2.1)\]

The second allele has the following genetic sequence:

\[\text{AACTATAACCG} \quad (2.2)\]

Notice that the two alleles of the hair color gene are identical except for the fifth base pair. The first allele has a G—C base pair while the second allele has an A—T base pair. These two alleles are an example of a SNP because they are identical except for a single nucleotide base pair. Suppose that allele 2.1 codes for blond hair, while allele 2.2 codes for brown hair. From
this example, it is easy to see how a SNP can lead to phenotypic variation within the population; hair color varies because two versions, or alleles, of the hair color gene are available. A person’s hair color, therefore, is dependent on the combination of alleles of the hair color gene that are inherited. Keep in mind that this is a heuristic example and, in reality, hair color is determined by a more complex process.

Before proceeding, it is important to note that genes do not completely determine human behavior. Specific to the current focus, there is no "crime gene." This means that there is no single gene—or group of genes—that, if inherited, will inevitably cause the person to become a criminal. Instead, genetic risk factors, like environmental risk factors, work in a probabilistic fashion. In certain cases, genetic risk factors are only important when combined with environmental risks (i.e., GxE).

Researchers have highlighted a number of genetic polymorphisms that are predictive of antisocial phenotypes. Most polymorphisms germane to antisocial behaviors are tied to the process of neurotransmission which takes place within the brain. The brain is the most complex organ in the human body utilizing approximately 60 percent of the human genome to develop (Pinker, 2002; Rowe, 2002; Wright, Tibbetts, and Daigle, 2008). Since the brain requires a large number of genes to develop, it should not be surprising that polymorphisms coding for the brain can affect its development and its subsequent functioning (Thompson et al., 2001). Since the brain is crucial to human behavior—it governs our every thought, emotion, and action—criminologists have begun to hypothesize about the various ways in which brain development may lead to antisocial behavior (Wright, Boisvert, Dietrich, and Ris, 2009; Wright, Tibbetts, and Daigle, 2008). Some researchers, for example, have considered whether differential structure and functioning of the prefrontal cortex is predictive of antisocial behavior (Yang et al., 2005a, 2005b). Grounding their work in Gottfredson and Hirschi’s (1990) theory of low self-control, Beaver, Wright, and DeLisi (2007) found evidence supporting the hypothesis that self-control, one of the most prominent correlates of criminal behavior, may be regulated by the prefrontal cortex. Researchers have also investigated other regions of the brain showing that variation in white matter along the corpus callosum is predictive of antisocial conduct (Raine et al., 2003). Taken together, it is clear that the brain plays an integral role in the development of human behavior. Thus, it is not surprising that the majority of GxE studies have focused on genes implicated in brain functioning.
The logic of GxE is simple: the effects of a genetic risk factor on the development of a phenotype (e.g., antisocial behavior) will differ across individuals according to their exposure to environmental risk factors or vice versa. In other words, GxE calls for a nonadditive effect between an environmental risk factor and a genetic risk factor in the development of antisocial behavior. For example, a genetic risk factor may have small or negligible effects on criminal behavior when no or a low level of an environmental risk is present. However, when the environmental risk is present, the effects of the genetic risk factor will be substantially increased.

Figure 2.3.1.2 depicts a GxE with heuristic data. This figure presents a simple GxE where there are two possible genotypes (denoted as Genotype 1 and Genotype 2) and three levels of environmental risk (the x-axis). The y-axis of the figure represents the various scores that a person might receive on an antisocial behavior scale. Careful examination of the figure reveals two things. First, the antisocial behavior for respondents with Genotype 2 is higher across all three levels of environmental risk. Second, the slopes of the two lines (i.e., the different genotypes) are different with the slope for Genotype 2 being larger (i.e., steeper). This second point represents GxE. As can clearly be seen, the effect of the environmental risk factor is dependent upon the person's genotype.

It is important to note that a group of researchers have put forth a somewhat different explanation of GxE. Rather than referring to these genes as "risk factors," Belsky and his colleagues (2009) argued that they should be called "plasticity" genes. Although the distinction appears like an academic exercise, the logic is sound and affords an explanation of findings that have, to date, been overlooked or that have been counter to the GxE hypothesis. In short, Belsky et al. argued that certain GxE findings show that individuals with the "risky" gene perform worse (e.g., more antisocial behavior) when exposed to adverse environments. But, at the same time, these individuals perform better when exposed to positive environments. Thus, Belsky et al. posit that the gene is not "risky" but instead "plastic." In other words, individuals with these "plasticity" alleles are more vulnerable to environmental influence, both good and bad. This relationship is shown graphically in Figure 2.3.1.3. As can be seen, the fundamental relationship between the two genotypes is similar to GxE. There is, however, one important difference. The x-axis has been changed to reflect a continuum of environmental influences. In the middle is "0" which can be considered an average environment. To the left of "0" are positive environments and to the right of "0" are negative environments. The notation "sd" has been used to indicate
the standard deviation distance between the environment of interest and the average environment. Notice that respondents with Genotype 2 report the lowest scores on the antisocial behavior scale when exposed to positive environments (i.e., -1 sd and -2 sd). However, as can be seen, these same respondents exhibit the most antisocial behavior when environmental risk factors are high (i.e., +1 sd and +2 sd).

There is now a sizable body of research examining the effect of GxEs in the creation of antisocial phenotypes and related experiences such as victimization (Beaver, Wright, DeLisi, Daigle, et al., 2007; Caspi et al., 2002; Foley et al., 2004; Haberstick, et al., 2005; Jaffee et al., 2005; Kim-Cohen et al., 2006; Vaske, 2009). Caspi and colleagues (2002) were the first to test the GxE hypothesis—in regards to a behavioral phenotype—with a measured gene. Their findings revealed that males with a particular genotype (i.e., alleles for the MAOA gene linked with low MAOA activity) that were also maltreated as children (i.e., the environmental risk factor) were more likely to have been convicted of a violent offense than were males who were maltreated but did not have the genetic risk factor (i.e., respondents who had the high MAOA activity allele). Although only 12 percent of the sample was exposed to both risk factors (i.e., childhood maltreatment and low MAOA activity allele), these respondents accounted for approximately 44 percent of all violent convictions in the sample. A number of studies have attempted to replicate Caspi et al.’s findings. Some findings have supported the GxE interaction between the low MAOA functioning allele and childhood maltreatment (Foley et al., 2004) while others have failed to replicate (Haberstick et al., 2005). A recent meta-analysis, however, reported that the extant literature supports a GxE between MAOA genotype and childhood maltreatment in the prediction of antisocial behavior (Kim-Cohen et al., 2006). Importantly, researchers have shown that MAOA is predictive of other antisocial outcomes such as gang membership (Beaver, DeLisi, Vaughn, and Barnes, 2010) and white collar crime (Beaver and Holtfreter, 2009).

Research has tested GxE hypotheses using other genetic polymorphisms and other environmental risk factors. For example, Beaver, Wright, DeLisi, Daigle and colleagues (2007) examined a GxE between a polymorphism in the DRD2 gene and exposure to delinquent peers in the creation of victimization. The results gleaned from their multivariate models supported a GxE and were particularly robust for white males. The results from Beaver et al. differed from those of Caspi and colleagues (2002). Caspi et al. found that the genetic risk factor increased
antisocial behavior when combined with an environmental risk factor. Beaver et al., on the other hand, found that the genetic risk factor increased victimization experiences when combined with less environmental risk (i.e., less exposure to delinquent peers). The authors offered an explanation for this seemingly counterintuitive finding: persons associating with a greater number of delinquent peers are more likely to be victimized regardless of their individual genotype. However, when exposed to fewer delinquent peers, an individual’s genotype will be more influential in creating victimization opportunities—perhaps via processes known as gene by environment correlation (rGE).

2.3.2 Gene-Environment Correlation (rGE)

Criminologists often argue that variables can be neatly bifurcated into "social" variables and "genetic" variables. This artificial division is often imposed to justify a focus on social variables without having to include or account for genetic effects. Recall that criminologists typically do not account for genetic effects in their statistical models. Behavioral genetic research, however, has shown that many of the so-called social variables are at least partially influenced by genetics. At first glance, the above sentence may seem counterintuitive. How can a social variable—or an environmental factor—be influenced by genetics? A review of the research into gene-environment correlations (rGE) will elucidate this point (DiLalla, 2002; Kendler and Baker, 2007; Scarr, 1992; Scarr and McCartney, 1983; Walsh, 2009).

Research focusing on rGEs has underscored the importance of considering the interrelationships of social and genetic influences for nearly every outcome imaginable. Delinquent peer groups, for example, are often considered a social phenomenon. To be sure, peer groups influence individuals by offering novel behaviors that may be modeled and learned (Akers, 1998). Recently though, behavioral genetic research has revealed that associating with delinquent peer groups—that is selection into delinquent peer groups—may be influenced by genetic factors (Beaver, Shutt, et al., 2009; Beaver, Wright, and DeLisi, 2008; Cleveland et al., 2005; Iervolino et al., 2002; Rowe and Osgood, 1984). Along these lines, researchers have also shown that many environmental variables are partially influenced by genetics (i.e., some portion of the variance is attributable to heritable factors; Kendler and Baker, 2007). To explain and
clarify these research findings, three types of rGEs are reviewed: passive, evocative, and active (Scarr, 1992; Scarr and McCartney, 1983).

2.3.2.1 Passive rGE

The first type of rGE is known as passive rGE. Passive rGE recognizes that parents pass along an environment and genes to their offspring. Since the child’s environment and the child’s genes both originate from the same source (i.e., their parents) the two are likely to be correlated. In this way, one would expect a child's environment to be correlated with their genotype.

Imagine two mates that are aggressive and prone to violence. Assuming that aggression is at least partially heritable, it is likely that any offspring produced by these two mates will also have aggressive tendencies. All else being equal, parents who are aggressive are more likely to act aggressively toward their children. In other words, children born to parents who are aggressive have a greater probability of inheriting aggressive tendencies as well as a greater probability of being exposed to an environment that promotes (or at least allows for) aggressive behavior. Thus, aggression may be partially heritable, but the expression of this behavior may also be influenced by the environment in which the child is placed by his or her parents. Passive rGEs address this issue by highlighting the fact that parental influence on their child’s environment is often conducive to the child’s genetic tendencies. Passive rGEs examine the possibility that parental influence on phenotypic expression is twofold: first, parents pass along genetic tendencies to their children; and second, parents pass along environments that allow for the expression of those genetic tendencies.

Passive gene-environment correlations offer the potential to better understand many of the major correlates of crime and delinquency. Low self-control, for example, is a trait that research has shown to be strongly correlated with delinquency (Pratt and Cullen, 2000). At the same time, recent evidence has linked self-control development with genetic factors (Beaver, Ratchford, Ferguson, 2009; Beaver, Shutt, et al., 2009; Wright and Beaver, 2005). Passive rGEs, therefore, may be at work in the transmission of levels of self-control (Boutwell and Beaver, in press). Imagine two mates, both of whom have relatively low levels of self-control. Since research has shown that low self-control is partially heritable, it is likely that any offspring produced by these two mates will have low self-control due to genetic transmission. It is also
easy to imagine how the child’s home environment could be affected by the parent’s levels of self-control; parents with low self-control are more likely to be ineffective parents (Gottfredson and Hirschi, 1990). In sum, the child will likely inherit genetic tendencies toward low levels of self-control and, at the same time, will inherit an environment that encourages the development of low levels of self-control.

Passive rGEs are the only rGE that leave open the possibility that parenting practices may impinge upon the child's personality development. Some researchers, however, are beginning to question whether the child’s personality is affected by the home environment. One of the most notable examples is Judith Rich Harris's theory of group socialization (Harris, 1995, 1998). Harris crafted her theory around recent advancements made in behavioral genetic research. Specifically, Harris noticed that this line of research continuously showed negligible effects of the shared environment on personality development. Recall that the shared environment captures environmental influences that make siblings more similar to one another. Since, home environments are similar for all children in a household, Harris argued that parenting is a shared environment. Since behavioral genetic research has shown that shared environments are often unimportant in personality development, Harris argued that parents do not shape their children's personality—at least not in a way that has a lasting effect on the child's personality.

Not surprisingly, Harris's dismissal of parenting effects has received much criticism (Pinker, 2002). Whether her theory is correct is an empirical question that researchers must examine. Regardless, the theory has clear implications for passive rGEs and criminological research more broadly. If parents only influence their child's personality via genetic transmission, then it is unlikely that environments provided by parents will have much of an effect on long-term outcomes. Given the importance placed on the home environment by nearly all criminological theories—see for example Akers (1998), Hirschi (1969), Gottfredson and Hirschi (1990), and Sampson and Laub (1993)—researchers must begin to examine these effects in the context of passive rGEs, with an eye toward the findings from behavioral genetic research.

2.3.2.2 Evocative rGE

The second type of rGE is evocative rGE. Evocative rGE occurs when a person evokes certain responses from their environment due to their genetic tendencies or propensities. Recall
Harris’s (1995, 1998) theory of parenting effects. Harris argued that most of the parenting effects observed by research studies could be accounted for by genetic similarities between parent and child. She did, however, concede that some parenting effects may still be observed (i.e., parental socialization variables may remain statistically significant predictors of child outcomes). Interestingly, however, Harris argued that the causal ordering of such effects are in the direction opposite to what most theories predict. Rather than parent-to-child effects, Harris argued that child-to-parent effects (i.e., evocative rGE) are driving the findings from most parenting research. Research bearing on this issue has supported Harris’s claim (Beaver and Wright, 2007; Huh et al., 2006). In a recent study conducted by Huh and colleagues (2006), results revealed that female problem behavior was a more consistent predictor of parenting behavior than parenting was of problem behavior.

Evocative rGEs allow for environmental responses to vary according to an individual’s genetic predispositions. Differential genetic tendencies can "evoke" differential environmental responses. The child born with aggressive tendencies, for example, may be more likely to get into fights at school. In other words, the aggressive child may be more likely to evoke negative reactions from his/her environment.

Behavioral geneticists studying evocative rGEs have typically focused on differential parenting responses to siblings raised in the same household (Ge, Conger, Cadoret, Neiderhiser, Yates, Troughton, and Stewart, 1996; O’Connor, Deater-Deckard, Fulker, Rutter, and Plomin, 1998; O’Connor, Hetherington, Reiss, and Plomin, 1995). Research has shown that children reared in the same family often report dissimilar experiences (Reiss et al., 2000). These differences may be due in part to the ways in which parents react to differential behaviors exhibited by their children. Consider the example where two children are raised in the same household but one child is born with a high level of impulsivity and the other is born with an average level of impulsivity. Clearly, these two children will evoke differential treatment from their parents. In all likelihood, the child with high levels of impulsivity will be disciplined more frequently and more harshly than will the child with average levels of impulsivity. Their differential treatment may be due in part to their diverse genetic predispositions as opposed to some other sociological variant. Currently, limited research has examined the extent to which criminal and delinquent behavior can be explained by an evocative rGE perspective. Given the
emphasis placed on environmental factors by criminologists, however, it is unlikely that this gap in the literature will remain for long.

2.3.2.3 Active rGE

The third type of rGE is active rGE. Active rGE occurs when a person seeks out environments to suit their genetic tendencies or propensities, a phenomenon commonly referred to as "niche-picking" or "self-selection." A person's genetic profile can influence the way in which a person engages and interprets their environment. Individuals may, for example, be more likely to select into, or actively pursue, environments that are compatible with their genetic tendencies (Wright, Tibbetts, and Daigle, 2008). Active rGEs offer a framework for understanding how genetic factors can influence the nonrandom selection into particular environments.

Clearly, the consideration of active rGEs can be informative for researchers interested in understanding deviant behavior by offering the opportunity to examine self-selection into criminogenic environments (Beaver, Barnes, Boutwell, and Cooper, 2009). One of the quintessential criminological findings is that delinquents have more friends that are also delinquent than do non-delinquents (see for a review Akers and Jensen, 2006). Although interpretations of this finding continue to be debated (e.g., Gottfredson and Hirschi, 1990), the most common explanation is that delinquent peer groups influence the behavior of the individuals affiliated with the group (Akers, 1998). Researchers, however, have noted that peer group selection is not a random process (Glueck and Glueck, 1950; Gottfredson and Hirschi, 1990). In fact, peer group selection can be driven by a number of factors including neighborhood factors, school factors, and familial influences. Not until recently, however, have researchers begun to investigate the influence of genetics on delinquent peer group formation (Beaver, Shutt, et al., 2009; Beaver, Wright, and DeLisi, 2008; Cleveland et al., 2005; Iervolino et al., 2002; Kendler, Jacobson, Gardner, Gillespie, Aggen, and Prescott, 2007; Rowe and Osgood, 1984). Beaver, Shutt and colleagues (2009) estimated DeFries-Fulker models and reported that associating with delinquent peers was between 37 and 62 percent heritable. Taking a different approach to the same issue, Beaver, Wright, and DeLisi (2008) sought to determine whether a specific genetic polymorphism was predictive of delinquent peer group formations.
Their findings indicated that the presence of one or more risk alleles on a specific dopamine transporter gene (DAT1) was strongly associated with self-reported delinquent peer associations among males raised in high-risk environments. The findings gleaned from these studies are supportive of an active rGE framework.

Although it is clear that genetics play a role in delinquent peer group exposure, a key question remains: how do genetic predispositions influence environments like delinquent peer groups? To answer this question, we must consider how genetic factors impact an individual. A person's genetic make-up influences all aspects of their life, including the experiences they find enjoyable. A person will seek environments that are enjoyable and will avoid environments that are not enjoyable. Since humans are social animals by nature (Pinker, 2002), it follows that they will seek the companionship of others that enjoy similar experiences. Thus, a person born with a propensity for risk-seeking is likely to socialize with peers that exhibit risky behavior. The overarching point, therefore, is that genes do not code for environmental experiences or exposure. Instead, genes code for brain development, brain functioning, and brain plasticity, all of which can influence personality traits. Based on personality traits, a person may be more or less likely to engage in certain activities, such as hanging out with delinquent others. Thus, active rGEs offer an explanatory framework to the old saying "birds of a feather flock together" (Glueck and Glueck, 1950).

The current discussion of active rGEs has centered primarily on the influence of delinquent peer groups. Active rGEs also have the potential to explain the correlation between antisocial behavior and many other environmental factors. As will be discussed in the following chapters, active rGEs can help to elucidate a number of factors highlighted by some of the most prominent contemporary theories in criminology. In recognition of this potential, the focus of this dissertation will build on the logic of active rGEs in order to more clearly understand the impact of life-course transitions on stability and change in antisocial behaviors.

2.4 SUMMARY AND DISCUSSION

Parenting research is one area of study that is likely to be most affected by the omission of genetic factors. For example, Gottfredson and Hirschi (1990) hypothesized that poor parenting—measured as a lack of attachment, structured discipline, and supervision—causes
children's development of low levels of self-control. A sizable body of research has examined this point, with most reporting findings supportive of the theory (Hay, 2001; Hay and Forrest, 2006; Burt, Simons, and Simons, 2006). This line of research has been built on SSSMs and can, therefore, only confirm that a *correlation* between parenting styles and child levels of self-control exists. Whether the correlation between parenting and self-control is causal or reflects some other association such as child-driven effects or genetic transmission cannot be understood from studies that employ SSSMs (Harris, 1995, 1998; Jaffee et al., 2004; Lytton, 1990). Thus, it is unclear whether the parenting strategies are actually affecting the child or if they merely reflect some other process. Wright and Beaver (2005) found that measures of parental influence no longer had a statistically significant association with the child's levels of self-control once genetic factors were controlled. This finding suggests that passive rGE may be confounding the results produced by parenting research concerning the etiology of self-control.

Passive rGEs are not the only way that genetic factors may confound criminological research findings. Evocative and active rGEs may be just as important for understanding some "classic" criminological variables such as the link between strain and crime (Agnew, 1985, 1992) and the inhibitory effects of social bonds on involvement in crime and delinquency (Hirschi, 1969). In another example, Akers’ (1998) theory of social learning could be expanded to include active rGE. One of the most hotly debated issues in the criminological literature has focused on whether delinquent peer groups cause delinquency or if they merely reflect nonrandom selection processes. Results from behavioral genetic research indicate that self-selection based on heritable traits explains a portion of the variance in exposure to delinquent peers—roughly one-third of the variance is explained by genetic factors (Beaver, Shutt, et al., 2009). Whether the positive correlation between exposure to delinquent peers and criminal behavior remains after these genetic influences are controlled remains an open empirical question (see Beaver, 2008b).

Although the importance of gene-environment interplay has escaped the attention of most criminologists, an encouraging point to bear in mind is that some theorists have covertly built these interactions into their hypotheses. Moffitt (1993), for example, argued that two groups of offenders exist: adolescence-limiteds (AL) and life-course-persistors (LCP). AL offenders do not offend in childhood nor do they offend in adulthood. Instead, AL offending is restricted to the adolescent years and is caused by a disjuncture between a person’s social maturity and their biological maturity. Moffitt argued that AL offenders will seek out LCP companions during
adolescence in order to "mimic" their adult-like behavior (i.e., active rGE). LCP offenders, on the other hand, offend throughout the life course. LCP offending is caused by neuropsychological deficits that are exacerbated by adverse home environments. If neuropsychological deficits are influenced by genetic factors, then Moffitt's hypothesis can be considered a GxE. Also, Moffitt posited that LCP offenders are likely to seek environments that fit their personality (i.e., active rGE). Thus, LCP offenders will be less likely to finish school during adolescence and less likely get a good job in adulthood (i.e., evocative rGE).

The reader should note that, for the current discussion, consideration of rGEs and GxEs were kept separate. This was done solely for heuristic purposes and should not be taken as an indicator that rGEs and GxEs work in a vacuum or are mutually exclusive. It is more likely the case that the two phenomena occur simultaneously to affect most outcomes. Evidence of this point can be found in recent research. Beaver and colleagues (2008) found that the DAT1 genotype predicted delinquent peer affiliation (i.e., active rGE). This finding, however, was restricted to males who were exposed to high-risk home environments (i.e., GxE). As the results from this study clearly indicate, environmental exposures are predicted by heritable characteristics, but these heritable characteristics are probabilistic; they often require an environmental "trigger."

If criminology is to integrate the findings of other scientific disciplines, researchers must begin to incorporate gene-environment interplay into their theories and their empirical models. Dividing variables into "social" and "biological/genetic" is erroneous and leads to misspecified models, theory, and policy decisions. Social variables can be "caused" by genetic factors (i.e., rGE) just as social variables can moderate genetic influences (i.e., GxE). Thus, many of the relationships examined by criminologists may be confounded by genetic effects. Behavioral genetic studies have also underscored the importance in distinguishing between shared and nonshared environments. Criminologists, however, have traditionally ignored this distinction and have unknowingly focused their efforts on exploring the effects of the shared environment, the smallest piece of the puzzle. To evince this point, recall that typical shared environments are parental influences and neighborhood conditions. Shared environmental effects work to make siblings more alike. Additionally, shared environmental influences explain *inter*family differences (i.e., differences from one family to another), but cannot explain *intra*family differences (i.e., differences between siblings). Thus, nonshared environmental influences are
important for understanding phenotypic variation. Criminological research, however, fails to make the distinction between shared and nonshared environments, making it difficult to interpret and determine how environmental effects matter.

In short, criminologists must begin to incorporate genetically-sensitive research designs by utilizing data that includes more than one child per household. Data on more than one child per household will allow criminologists to account for genetic effects en route to a more accurate estimation of environmental effects. This does not mean that criminologists must expend an exorbitant amount of resources collecting new data. In fact, many of the most popular datasets used by contemporary criminologists already include sibling pairs and twins (Beaver, 2009a). Until criminologists begin to employ genetically sensitive research designs, research into the etiology of delinquent, criminal, and antisocial behaviors will likely be omitting a key explanatory variable.

Behavioral geneticists have a number of methods at their disposal for estimating genetic and environmental effects specific phenotypes. This line of inquiry offers a wealth of insight into the complex etiology of antisocial behaviors. Critics, however, are quick to point out the limitations of behavioral genetic research and to discredit the findings flowing from such studies. A popular critique of twin-based studies is that the EEA artificially inflates heritability estimates. As this review has discussed, however, there are other research designs available to behavioral geneticists that do not rely on the EEA. Results garnered from these other methods have been fairly similar to those from twin-based studies. Specifically, regardless of the methodology employed, behavioral genetic research has revealed that antisocial behaviors are at least partially heritable. As a result, molecular genetics research is now beginning to uncover the specific genes operating behind the "heritability curtain." It is now incumbent upon researchers to determine when, why, and for who are genetic influences important. The analyses presented in this dissertation are a first step toward answering some of these questions.
CHAPTER 3
LIFE-COURSE CRIMINOLOGY: FINDINGS AND THEORETICAL CONSIDERATIONS

Biosocial criminology is an emerging paradigm. Indeed, there are now hundreds of studies that examine the genetic underpinnings to antisocial, criminal, and delinquent behaviors (Ferguson, 2010; Mason and Frick, 1994; Miles and Carey, 1997; Rhee and Waldman, 2002; Walters, 1992). Some behavioral geneticists have also begun to examine the extent to which genetic and environmental factors underlie well known predictors of antisocial behavior such as levels of self-control and exposure to delinquent peer groups (Wright, Beaver, DeLisi, and Vaughn, 2008). This body of literature has shown that genetic and biological influences are important for understanding the etiology of most maladaptive behaviors (Moffitt, 2005; Raine, 1993). Results from meta-analyses and review articles suggest that about 50 percent of the variance in antisocial behaviors is attributable to genetic factors. The remaining 50 percent is divided among shared and nonshared environmental influences with the nonshared environment being the most important (Moffitt, 2005).

Despite the vastness of the literature, the consistency with which researchers identify genetic influences, and the robustness of the findings, biosocial criminology lacks a clear theoretical framework. To date, there is no single theory that incorporates all of the findings from biosocial research into a succinct set of propositions and theoretical axioms. This does not mean that theorists have not proffered biosocial theories. To be sure, there are a number of theories that incorporate biosocial arguments into their hypotheses. Notable examples are Ellis (2005), Moffitt (1993), and Robinson (2004; Robinson and Beaver, 2010). Others have taken a different approach and argued that criminology does not need a biosocial theory (Walsh, 2002).
Instead, as Walsh (2002) explained, many criminological theories can easily be revamped to incorporate recent evidence from biosocial research.

The absence of a theoretical framework may explain, at least in part, why the paradigm has not gained traction among criminologists. Indeed, only a small fraction of criminologists conduct research with a biosocial focus (Wright, Beaver, DeLisi, Vaughn, Boisvert, and Vaske, 2008). As a result, criminologists continue to overlook the importance of behavioral genetic research (Walsh and Ellis, 2004). As outlined in previous chapters of this paper, this is a common misperception—that environmental and biological/genetic influences can be neatly bifurcated and studied separately—that can be extremely misleading (Raine, 1993). Large bodies of work based on behavioral genetic methods have shown that almost all human behaviors are the result of genetic and environmental factors. Furthermore, this same line of research has shown that environments are not randomly distributed but can be predicted by genetic factors (i.e., gene-environment correlations).

Does this mean that biosocial criminologists should focus their efforts on developing a biosocial theory that can be falsified by researchers? If biosocial criminology had a theory, would more researchers be inclined to consider this line of inquiry? Unfortunately, these are questions that have no direct answer. As a result, the position that will be taken in the current discussion is that the biosocial paradigm does not need a singular, all-encompassing theory in order to continue to move forward. Instead, this discussion will argue the case that biosocial criminology can progress as a critique of extant criminological theories.

Findings from biosocial criminology may allow researchers to explain some of the most aggravating and anomalous findings that result from tests of contemporary theories. A cursory review of the current criminological literature will reveal, for example, that researchers typically account for less than one-third of the variance in criminal and delinquent behavior (Weisburd and Piquero, 2008). In other words, criminological theories, despite the volume of theoretical statements that exist, are left with some powerful questions unanswered. For example, why do children in the same family turn out so different (Plomin and Daniels, 1987)? Biosocial research offers the opportunity to answer this question. Perhaps the reason that children that grow up in the same family—and are thus exposed to the same parents, the same neighborhood, and likely the same schools and peers—are different from one another is because they have different genetic profiles. Imagine, for the purposes of this argument, that genetic influences are
important for personality development. If this were so, and personalities were partially scripted by genetic factors, might this explain why two people that are exposed to similar environments turn out differently? Findings from biosocial criminology suggest that this may be the case.

The remainder of this chapter will build the theoretical foundation necessary to understand how biosocial research can be seen as a critique of one criminological theory. Specifically, one of the most prominent contemporary explanations of criminal behavior will be presented: Sampson and Laub’s (1993) age-graded theory of informal social control. As such, this chapter has three main objectives. First, detailed attention will be given to this theory. Second, an exhaustive overview of the criminological research surrounding this theory will be presented. Third, the chapter will close with a discussion of the various ways in which this theory might be critiqued by biosocial research.

3.1 SAMPSON AND LAUB'S LIFE-COURSE THEORY

Walsh (2002) argued that biosocial research naturally serves as a critique of criminological theories. Specifically, Walsh showed that anomie and strain theories, differential association and social learning theories, control theories, human ecology and social disorganization theories, critical, and feminist theories could all be reassessed with a biosocial framework. Not only can these theories be integrated with a biosocial focus, most are already consistent with biosocial explanations. Take, for example, learning theories of delinquency (Akers, 1998; Sutherland, 1947). These theories argue that learning occurs through social interactions and is, therefore, a social process. This does not mean that learning cannot also incorporate biological and genetic components. Consider this question: at the most fundamental level, what is learning? It is well documented that learning is a biochemical process which takes place within the brain, and more specifically, within the hippocampal regions of the brain (Wright, Tibbetts, and Daigle, 2008). The brain, as it turns out, is largely scripted by genetic factors. Thus, it is likely that learning is influenced by genetic factors. At the very least, learning is a process that can be explained in terms of biochemicals and the neurological functioning of the brain. Clearly then, learning is both biological and sociological in nature (Miller, Shutt, and Barnes, 2010).
Although biosocial factors can be incorporated into most theories of crime, there is one that will be the focus of this discussion. Over the last decade, criminology has witnessed a shift in theoretical and empirical focus. Criminology was partially built on findings from cross-sectional research that showed the predictive power of differences between people at one point in time (Hirschi and Selvin, 1973). During the past 15 years or so, this focus has shifted and criminologists are becoming increasingly interested in within-individual changes and how these changes can reshape a person's life trajectories. This focus has been driven mainly by the advent and availability of longitudinal research data and a few prominent theoretical statements (Piquero et al., 2003). One of the most prominent contemporary theories, and one that has certainly reshaped the discipline, is Sampson and Laub's (1993; see also Laub and Sampson, 2003) life-course theory of crime.

In 1993, Sampson and Laub published *Crime in the Making: Pathways and Turning Points through Life*. In this monograph, the authors presented and tested an "age-graded theory of informal social control" which was influenced by Hirschi's (1969) social bonding theory. The theory is presented as one that explains the onset, persistence, and desistance from crime and antisocial behavior. In short, the theory was intended to integrate Hirschi's social bonding theory with more recent evidence which indicated that the types and the effects of bonds vary, within individuals, across the life course. Furthermore, the theory was one of the first to incorporate an explanation of both stability and change in offending.

Any discussion of Sampson and Laub's theory must first begin by defining several concepts that are crucial to the theory. The theory is intended to explain involvement in and desistance from crime across the life course. Thus, the theory is focused primarily on explaining within individual changes in behavior over the individual's life span. Two concepts are most important: trajectories and transitions. A trajectory is defined by Sampson and Laub (1993) as, "a pathway or line of development over the life span, such as work life, marriage, parenthood, self-esteem, or criminal behavior. Trajectories refer to long-term patterns of behavior and are marked by a sequence of transitions" (p. 8). In short, a trajectory is the pathway that an individual follows in regards to a certain aspect of life. When considering crime and delinquency, an individual's trajectory can be defined by their history of involvement in delinquency. Having knowledge of an individual's past behavior will help to predict their future involvement in crime and delinquency (Olweus, 1979; Robins, 1978).
Notice that the definition of trajectory provided by Sampson and Laub included the second concept that is crucial to their theory, transitions. Transitions are defined as life events that unfold over short periods of time and can serve as turning points for an individual’s trajectory. Transitions are perhaps best described through examples. Transitions are typically life events that are meaningful to the individual such as graduation from high school or college, getting married, or getting a good job. Clearly, trajectories and transitions are inextricably linked; transitions are the building blocks of a trajectory. In other words, an individual’s trajectory can be defined by the presence or absence of transitions. The mere presence of a transition, however, does not necessitate an alteration in a person’s trajectory. Graduating from high school is a transition (i.e., from student to nonstudent) but it is not considered a "turning point" unless it has some influence on the individual’s trajectory. In other words, transitions by themselves do not necessitate within-individual changes. However, when a transition does lead to within-individual change, it is considered a turning point. Turning points, in short, are life-course transitions that "redirect paths" (Sampson and Laub, 1993:8). Sampson and Laub use the concept of turning points as their jumping off point. Specifically, they posited that an individual's trajectory of crime and deviance, regardless of what has happened in the past, can be derailed by a positive turning point later in life. In this way, their theory is largely focused on within-individual change.

Sampson and Laub (1993) drew primarily on Hirchi’s (1969) social bonding theory to develop their life-course theory. In many ways, Sampson and Laub’s theory can be seen as an extension of Hirschi’s original theory. Hirschi argued that humans will seek to maximize pleasure and to avoid pain. While this concept of human nature was nothing new, Hirschi built off of this idea to derive several logical arguments. First, Hirschi argued that criminological theory need not account for a person's motivation to crime and deviance. Since humans are naturally driven to maximize pleasure, and since most crime is instrumental in nature (i.e., it serves a purpose to the offender), Hirschi inferred that humans are naturally motivated to commit crime. For this reason, criminological theory need not explain motivations, but instead should explain why most people refrain from crime. Indeed, most people are not criminal. Research has consistently shown that a very small proportion of the population is responsible for the majority of all crime (DeLisi, 2005; Moffitt, 1993; Wolfgang et al., 1972). Thus, any theoretical statement about involvement in crime and delinquency should explain why most people refrain
from criminal acts. Hirschi argued that four social bonds (i.e., social ties) could explain why most people refrain from delinquency. These four social bonds are attachment, involvement, commitment, and belief.

Attachment refers to the social relationships that a person has with others. Having more attachment (i.e., more relationships or stronger relationships) acts as a barrier to delinquency because they act as something that will be jeopardized if the individual were to break social convention (i.e., the law). Involvement refers to the amount of time a person spends on conventional activities. The more time one spends in prosocial activities necessarily means that less time is spent on antisocial activities. Commitments, similar to attachment, act as "stakes in conformity" that are jeopardized each time a person commits a delinquent act. In short, commitments are a person's investment in conventional society such as employment or educational opportunities that may be terminated if the person is caught offending. Finally, belief refers to the person's acceptance and adherence to the social order. If a person believes that individuals must suspend some of their rights in order to receive other benefits from society, then it is more likely that the person will abide by laws handed down by society. In short, Hirschi (1969) argued that these four social bonds were the most important elements for explaining why people do not commit crime. Inversely, the weakening of these bonds, any or all of them, may release the individual from their "controlling" effects and may, therefore, allow the individual to commit a crime. Hirschi believed that these social bonds were barriers to criminality and that releasing a person from any of the bonds will increase the probability that a crime will occur.

Sampson and Laub's theory differs from Hirschi's theory in two important ways (Laub, Sampson, and Sweeten, 2006). First, Hirschi's (1969) theory is a static explanation. In other words, Hirschi's theory is not specific to a certain point in the life course. Social bonds that matter in early adolescence are the same as those that matter in late adulthood, according to Hirschi. Sampson and Laub contended that this is not the case. Converging lines of research suggested the contrary: (1) the effects of social bonds change over time; and (2) the types or forms of social bonds that matter change over the life course. Second, and related to the first point, Hirschi's theory did not offer an explanation of the age-crime curve. Research shows that involvement in crime and delinquency increases rapidly in late childhood and early adolescence, peaks somewhere between ages 16 and 21 (depending on the crime type being examined) and
then begins to quickly descend back to pre-adolescent levels. Figure 3.1.1 presents a graphical representation of the age-crime curve using data gleaned from the Uniform Crime Reports in 2008 (U.S. Federal Bureau of Investigation, 2009).

Using social control theory as a springboard, Sampson and Laub (1993:243) proffered a theory that had three goals: (1) explain the onset of adolescent delinquency; (2) explain the persistence of delinquency from adolescence to adulthood; and, (3) explain changes in criminal behavior in adulthood (i.e., desistance). To explain the onset of delinquency, Sampson and Laub highlighted the importance of structural process and family-level influences. The authors argued that structural background characteristics such as exposure to poverty and growing up in a disadvantaged home influence the onset of adolescent delinquency through their effects on parenting practices. Levels of parental discipline, monitoring, and attachment are all thought to be affected by structural background characteristics. These parenting practices, in turn, were hypothesized to have a direct effect on adolescent delinquency. Sampson and Laub also argued that structural background characteristics affect adolescent delinquency through their effects on the child's levels of attachment and performance in school and through the child's involvement with delinquent peers. In other words, Sampson and Laub argued that structural background factors not only affect parenting practices, but they also influence adolescent delinquency through their effects on school and peer outcomes.

In summary, Sampson and Laub theoretically account for the onset of adolescent delinquency in three ways. First, structural background factors such as household crowding and having a parent that is a criminal will influence adolescent involvement in delinquency. These effects, however, are indirect. Second, parents that are harsh disciplinarians or parents who do not monitor their child's behavior are more likely raise a delinquent child. The effects of the structural background factors are mediated by these parental factors. Third, the child's levels of attachment and performance in school and the child's attachment to delinquent peers will also influence involvement in delinquency. School and peer group factors will also mediate the relationship between the structural background factors and delinquency. These three propositions summarize Sampson and Laub's account of the onset of adolescent delinquency.

As explained above, the second goal of Sampson and Laub's theory was to explain continuity in delinquency from adolescence to adulthood. In order to do so, Sampson and Laub note that two explanations are possible: population heterogeneity and state dependence (Nagin
and Paternoster, 1991). Population heterogeneity states that there is a stable propensity to offend that varies from person to person. This argument is synonymous with Gottfredson and Hirschi's (1990) theory of low self-control. Low self-control is argued to be the underlying cause of all criminal and antisocial behavior. On the other hand, state dependence—which is the stance that Sampson and Laub take—argues that individual offending patterns are stable due to the effects of past behavior. Offending behaviors typically reduce a person's access to prosocial opportunities and will often weaken social bonds. For example, delinquent behavior during adolescence may increase the person's likelihood of being arrested and spending time in prison. By virtue of having a criminal record, future opportunities (i.e., employment or relationship opportunities) may be lost. This knifing off of opportunities and the weakening of social bonds, in turn, leads to future delinquent behavior. Thus, antisocial behavior exhibited in adolescence will be correlated with adulthood deviance, but the effect will be indirect; the negative consequences of early deviance will mediate the effect between adolescent and adulthood deviance.

Finally, the third component of Sampson and Laub's theory sought to explain changes in delinquent behavior. In other words, the authors put forth an explanation of desistance. Recall the earlier discussion of turning points. Turning points are events that occur during the life course that have the effect of derailing or altering a person's trajectory. Sampson and Laub drew on this concept of turning points to explain how and why most people desist from crime in adulthood. They argued that certain life-course transitions—marriage, job stability, and military involvement—can act as turning points and can lead a person to desist from criminal activities. Consistent with their focus on social control, the authors argued that getting involved in a good marriage or a good job increase social capital which in turn cause criminal behaviors to appear less attractive. An increase in social capital means that the individual has something to lose if caught committing a crime. Thus, gaining social capital through marriage or employment will reduce the likelihood that a person will continue to offend because the person will have a "stake in conformity." In this way, Sampson and Laub's age-graded theory of informal control is able to account for changes, and, more specifically, fairly rapid changes in offending behavior.

Sampson and Laub's theory can be neatly summarized into three main hypotheses. First, the onset of adolescent delinquency is caused by a lack of familial social bonds in early childhood. These social bonds may be affected by (i.e., they may mediate) structural background
factors such as neighborhood conditions. Second, offending behaviors (i.e., criminal trajectories) are maintained from adolescence to adulthood through the process of cumulative continuity or state dependence. Delinquency in childhood reduces future opportunities and weakens social bonds. The net effect is that the offending trajectory is more likely to remain stable. Third, though offending can be stable due to cumulative continuity processes, change can also be realized. Sampson and Laub pointed out that most offenders eventually desist from crime. To explain this disjuncture in a person's trajectory, the authors argued that life-course transitions can become turning points, which may eventually lead to the cessation from offending. The most important turning points are involvement in a good marriage, holding a stable job, and joining the military (Sampson and Laub, 1993).

3.2 CRIMINOLOGICAL EVIDENCE FOR SAMPSON AND LAUB'S THEORY

Much of the empirical evidence bearing on Sampson and Laub's age-graded theory of informal social control comes from their work. To date the authors have written two books (1993, 2003) and a host of articles testing different hypotheses drawn from the theory. Their life-course theory has become one of the more prominent in criminology (Piquero et al., 2003). This section will review the empirical findings gleaned from tests of the theory. In reviewing this extensive body of literature, the findings will be presented according to the three organizing hypotheses outlined above: (1) factors affecting the onset of adolescent delinquency; (2) factors affecting the stability in offending; and (3) factors affecting changes in offending. The research culled from Sampson and Laub's two books will provide a loose framework for the discussion.

3.2.1 The Onset of Adolescent Delinquency

Much of the empirical evidence bearing on social control theory (Hirschi, 1969) can be extended to Sampson and Laub's (1993) life-course theory. This is especially true regarding the empirical literature examining factors that predict onset of adolescent delinquency. Sampson and Laub discussed the literature bearing on the effects of structural background factors, parenting factors, school factors, and delinquent peer groups on the onset of adolescent delinquency. They argued that the effects of structural background factors such as
socioeconomic status had been largely overlooked in regards to their effects on individual-level measures of adolescent delinquency. However, the few studies that did exist indicated that these factors indirectly affected adolescent delinquency through family factors (Blake, 1989; Kempf, 1993; Laub and Sampson, 1988) or had direct effects on delinquency (Glueck and Glueck, 1950; Hirschi, 1969). As a result, the authors argued these factors would have effects on the onset of adolescent delinquency, but that they would be mediated, at least partially, by the parenting, school, and peer measures.

In regards to the effects of parenting on adolescent delinquency, there was already an extensive body of literature by the time Sampson and Laub crafted their theory. In fact, there were several meta-analyses and reviews that summarized the evidence (Kempf, 1993; Loeber and Stouthamer-Loeber, 1986). As expected, these reviews highlighted the importance of parenting effects on adolescent delinquency. Loeber and Stouthamer-Loeber (1986), in what is considered one of the most comprehensive reviews of this literature, explained that parenting and family factors are among the strongest predictors of adolescent delinquency. In brief, their review indicated that parenting effects were robust to different measurement strategies and different methodological specifications (e.g., comparison groups, experimental study, longitudinal, cross-sectional, etc.). Loeber and Stouthamer-Loeber organized their review in terms of four "paradigms of family influence." First, the neglect paradigm captures the effects of parents that do not spend enough time with their children and are unaware of their child's deviant behaviors. The second paradigm, referred to as the conflict paradigm, captures the effects of parents and children who have a bellicose relationship with one another. The third paradigm, the deviant behaviors and attitudes paradigm, captures the transmission of deviance from parent to child. This transmission, according to Loeber and Stouthamer-Loeber can occur by genetic transmission or by the child modeling the deviant behaviors of the parent. Finally, the fourth paradigm discussed by Loeber and Stouthamer-Loeber was the disruption paradigm. The disruption paradigm focuses on the effects of marital discord on children. Specifically, marital problems between parents can disrupt normative family functioning which may increase the probability that the child will react with delinquent or antisocial behaviors.

Loeber and Stouthamer-Loeber (1986) used these four paradigms (i.e., neglect, conflict, deviant behaviors and attitudes, and disruption) to organize their review of the research. Their findings indicated that, "In both concurrent and predictive studies, at least 70 percent of the
analyses for each paradigm showed a significant effect in the expected direction. Thus family variables and child conduct problems or delinquency were consistently related to each other” (Loeber and Stouthamer-Loeber, 1986:120). These effects did not appear to be moderated by the child's gender, and many parenting effects were stronger when lagged parenting measures predicted the child's delinquency. Their results also indicated that some forms of parental intervention may be effective in reducing the child's antisocial behavior. Interestingly, however, the authors noted the fact that almost no studies controlled for genetic and biological influences. They cautioned that their results may overestimate family effects for this reason. However, they later directly contradict this statement by concluding that the effects of heredity and biology are likely weak and inconsequential (Loeber and Stouthamer-Loeber, 1986:127-128).

As previously mentioned, much of the empirical evidence surrounding the age-graded theory was advanced by Sampson and Laub (1993). In order to test the first part of their theory (i.e., factors influencing the onset of adolescent delinquency), the authors constructed measures of structural background factors, family process factors, and delinquency. Structural background factors included items such as residential mobility, family size, whether the mother was employed, and whether either parent had a criminal record. Family process variables included items such as maternal supervision, parental rejection of the child, and the harshness of parental disciplinary practices. Delinquency was measured via self-reports, parent-reports, teacher-reports, and official records.

The first stage of their analysis was to determine whether the structural background factors predicted the family process variables. Results indicated that the structural background factors were consistently related to each of the family process variables. The second stage of the analysis, therefore, was to determine whether the structural background factors influenced delinquency, and whether these effects were mediated by family process variables. Results from these analyses were supportive of the theory. Specifically, the various models indicated that the structural background factors predicted delinquency, but that these effects, for the most part, were fully mediated by the family process variables. Consider the following: "…the calculation of indirect effects (see Alwin and Hauser, 1981) reveals that of the total effect of all structural background factors on delinquency, 73 percent is mediated by family process" (Sampson and Laub, 1993:83). These substantive conclusions were similar across the different specifications of delinquency (e.g., self-report vs. official reports).
Recall that Sampson and Laub (1993) hypothesized that school process and peer delinquency should affect delinquency and will mediate the effects of the structural background variables. To test these hypotheses, Sampson and Laub performed an analysis that was similar to the one performed on family process variables. Specifically, school process and delinquent peer variables were regressed on the structural background factors. Evidence indicated that the structural background variables were associated with the school and peer variables. Thus, a second analysis was conducted where the school and delinquent peers variables were included in a model predicting delinquency. Once again, the authors found evidence to support their hypothesis: school process and delinquent peer variables mediated much of the effects of the structural background variables on delinquency.

But what about individual-level factors such as genetic and biological influences? As discussed earlier, genetic factors are important for predicting antisocial behaviors (see Chapter 2 of this dissertation). To be fair, Sampson and Laub do not completely ignore these factors. However, their efforts to account for genetic and biological influences were weak at best. Three indicators of the child's temperament—all of which were measured retrospectively during the first wave of data collection when the respondents were 15 years old—were used to account for genetic and biological effects. These three indicators were labeled child difficulty, tantrums, and early onset. Child difficulty was a dichotomous variable which indicated whether the respondent was restless and irritable as a child. The tantrums variable was a measure that tapped how often the respondent engaged in violent temper tantrums as a child. Finally, the early onset variable was a dichotomous variable which indicated whether the respondent had engaged in "unofficial behavior" prior to the age of 8. The criterion used to classify "unofficial behavior" was never clearly defined. Sampson and Laub's description of these three items is brief and puzzling. No research is cited to support the use of these three measures. Further, the degree to which these three measures capture the complex arrangement of genetic and biological influences on antisocial behaviors is even more ambiguous. Nonetheless, Sampson and Laub (1993) relied on these three measures to account for genetic and biological effects. Their analyses revealed that each of the three measures was significantly related to delinquency. Not surprisingly, however, the effects of the family process, school process, and delinquent peers variables were not significantly altered by the inclusion of the child temperament variables. This is evidence,
according to the authors, that the effects of informal social control on delinquency are robust to the inclusion of genetic and biological effects.

Although further discussion will be given to this point in a later section, it bears mentioning that the approach set forth by Sampson and Laub (1993) to control for genetic and biological influences is not an appropriate strategy. Research has shown that genetic influences are complex and are not constant across the life-course (Reiss et al., 2000). In short, genetic factors that influence antisocial behavior in childhood may not be the same genetic factors that influence antisocial behavior in adulthood (Lyons et al., 1995). Thus, controlling for childhood irritability, temper tantrums, and "unofficial behavior" is unlikely to account for much, if any, of the variance in adulthood crime.

3.2.2 State Dependence and Continuity in Criminal Behavior

The second hypothesis garnered from Sampson and Laub's (1993) theory is that state dependence (i.e., cumulative continuity) will explain stability in antisocial behavior (i.e., behavior that extends from adolescence into adulthood). Some of the most direct evidence regarding this hypothesis comes from Nagin and Paternoster (1991, 2000). Building on the body of research showing that antisocial behavior remains relatively stable (i.e., between-individual differences remain stable) throughout the life course, Nagin and Paternoster set out to determine whether these patterns of stability are due to population heterogeneity or state dependence processes. The findings from their first paper (Nagin and Paternoster, 1991) indicated that state dependence processes were more important than population heterogeneity. However, subsequent studies produced by this research team and others have drawn this conclusion into question (Gottfredson and Hirschi, 1990; Nagin and Paternoster, 2000). Some researchers have found evidence that state dependence processes are dominant, while others have found population heterogeneity to be more influential (Gottfredson and Hirschi, 1990). A contentious debate regarding the relative importance of state dependence over population heterogeneity abounds (Nagin and Paternoster, 2000).

As has already been discussed, however, Sampson and Laub (1993) hypothesized that state dependence effects would account for the lion's share of the stability in antisocial behavior. Recall that their argument stated that delinquency in adolescence would jeopardize adulthood
opportunities and would weaken social ties. In this way, Sampson and Laub argued that labeling processes (Becker, 1963; Lemert, 1951) would explain the link between childhood and adulthood deviance. To test this hypothesis, Sampson and Laub estimated the effect of juvenile delinquency on adulthood arrest likelihood. Controlling for a host of individual-level differences (i.e., to rule out the effects of population heterogeneity), their models indicated that juvenile delinquency was a strong predictor of adult arrest likelihood. They concluded, therefore, that state dependence processes explained continuity in antisocial behavior:

…we emphasize a cumulative developmental model whereby delinquent behavior has a systematic attenuating effect on the social institutional bonds linking adults to society (for example, labor force attachment, marital cohesion) (Sampson and Laub, 1993:138).

Criminologists have long been interested the stability of criminal behavior. Some of the earliest studies noted the remarkable stability in antisocial behavior over time (Glueck and Glueck, 1940, 1950; Loeber, 1982; Robins, 1966; West and Farrington, 1973). For example, Olweus (1979) equated the stability of aggression with that of intelligence. Robins (1978:611) famously concluded that "adult antisocial behavior virtually requires childhood antisocial behavior" (emphasis in original). Statements such as these, however, may give the wrong impression about the predictive power of prior behavior. Although it is true that most antisocial adults were antisocial as children (i.e., researchers can retrospectively identify antisocial adults based on childhood characteristics), it is also the case that most antisocial children do not grow up to be antisocial adults (i.e., prospectively identifying antisocial adults based on childhood characteristics is much more difficult). In other words, the research literature bearing on stability of criminal behavior indicates two things: (1) stability is common and most criminal adults were delinquent as adolescents; however, (2) change is possible and most adolescent delinquents do not become criminals in adulthood. As discussed above, Sampson and Laub (1993) relied on state dependence processes to explain stability in antisocial behavior. However, Sampson and Laub were more interested in identifying the factors that lead to change in antisocial behavior. Indeed the bulk of their research has focused on the effects of adult social bonds—marriage, employment, and military involvement—on change and desistance from crime.

3.2.3 Adult Social Bonds and Changes in Criminal Behavior

59
The third hypothesis flowing from Sampson and Laub's (1993) theory is that adult social bonds can lead to changes in behavior and eventually to desistance from crime. Building off of a substantial body of evidence, they note that behavioral continuity across the life course is normative, yet change does occur. Sampson and Laub attribute these changes in criminal trajectories to the inhibitory effects of adult social bonds. Compared to other aspects of criminal career research (i.e., onset, prevalence, and frequency of offending), the desistance process has received far less attention (Loeber and Le Blanc, 1990; Piquero et al., 2003). The remainder of this discussion—and the analyses presented later—will focus on the effect of adult social bonds on changes in criminal behavior.

Sampson and Laub (1993) argued that social bonds were important for predicting intra-individual changes in criminal behavior. However, they do not argue that the mere presence of a social bond (e.g., being married) is sufficient to lead to change. Instead, they argued that the level of attachment to that social bond would be most important element. This means that having a job is not enough to lead a person to desist from criminal activity. Yet, if the person has a job that they are passionate about and that they wish to retain (i.e., more attachment to the job), then this social bond may lead to behavioral change. Sampson and Laub introduced the concept "social capital" (Coleman, 1988) as a way to define their logic. More social capital that is invested in a social bond—being more attached and committed to a marriage—will be a stronger predictor of behavioral change than will the mere presence of the social bond.

Clearly, the third component of Sampson and Laub's theory intends to explain changes in crime over the life-course. More specifically, Sampson and Laub are interested in explaining desistance from crime. It is important, therefore, to first arrive at a definition of desistance. Unfortunately, Sampson and Laub (1993) do not offer a definition of desistance. Others, however, have described desistance as:

- a slowing down in the frequency of offending (deceleration), a reduction in its variety (specialization), or a reduction in its seriousness (de-escalation). The relevant boundary concept for all these subprocesses is the age at termination (emphasis in original; Le Blanc and Loeber, 1998:123).

In other words, the desistance process begins when a person begins to slow their involvement in crime (deceleration and de-escalation). The exact moment when the person has "quit" crime is the precise moment when desistance has occurred. Thus, it is important to note that the
desistance process may involve deceleration, de-escalation, and eventually desistance. The term "desistance," however, refers to the moment when the person has quit crim. Although it is not directly addressed, it would appear from this definition that the discrete time point when a person quits crime occurs the moment after the final crime has been committed (see for a similar position Maruna, 1998:11).

Sampson and Laub (1993) argued that three adult social bonds were of paramount importance for explaining why some offenders desist from crime. These three adult social bonds are marital attachment, commitment to occupational goals, and job stability. Marital attachment was operationalized as, "the general conjugal relationship between the subject and his spouse during the period plus the subject's attitude toward marital responsibility" (Sampson and Laub, 1993:144). Commitment to occupational goals was measured as the individual's expressed educational and economic interests. Additionally, this measure tapped the individual's efforts taken to achieve these goals. Job stability was a measure that tapped employment status and the time spent at the current place of employment. These three adult social bonds were entered into regression equations predicting crime and deviance in adulthood. It is important to note that Sampson and Laub did not measure and examine desistance as a discrete event (i.e., whether the respondent had terminated criminal involvement). Instead, Sampson and Laub analyzed changes in crime, which may best be understood as deceleration or de-escalation. Several patterns emerged across all models estimated. First, marriage (coded as married or not) was not a significant predictor of change in criminal involvement in adulthood. Thus, unmarried men were no more likely to be involved in crime than were married men. Second, marital attachment was associated with changes in criminal behavior. Men who reported being more attached to their spouse were less involved in crime and deviance. This means that the mere presence of the social bond (i.e., marriage) did not predict change. The level of attachment to that bond, however, was an important predictor. Third, commitment to occupational goals was unrelated to changes in criminal behavior. Fourth, job stability predicted changes in criminal behavior. Individuals reporting more job stability were less involved in crime.

Sampson and Laub (1993) performed several sensitivity analyses to examine the robustness of their findings. For example, the effects of job stability and marital attachment were examined after controls for individual background differences were included (i.e., child temperament and adolescent levels of delinquency), after simultaneity effects were modeled (i.e.,
social bonds affect criminal involvement and crime involvement affects social bonds), and after unobserved heterogeneity was controlled. The results from these sensitivity checks supported the original findings. Sampson and Laub therefore concluded that the effects of job stability and marital attachment on changes in criminal behavior were strong and robust predictors of change in criminal behavior. In sum, the estimates presented by Sampson and Laub (1993) indicated that adult social bonds lead to decreases, and possibly even desistance, from crime.

In 2003, Laub and Sampson published a second monograph that sought to address some questions raised by their original work. This second book, Shared Beginnings, Divergent Lives: Delinquent Boys to Age 70, focused primarily on criminal trajectories (do some people persist in their delinquency for their entire life or does desistance eventually occur for everyone?) and the factors that predict changes in criminal trajectories. For the most part, the analyses presented in this book were performed with semi-parametric, group-based trajectory models developed by Nagin (1999). In brief, the 2003 book focused on 3 research questions: (1) what does a criminal trajectory look like when examined across the entire life course; (2) what predicts changes in criminal trajectories; and, (3) what predicts persistence in criminal trajectories?

Laub and Sampson (2003) focused intently on the first research question outlined above: what does a criminal trajectory look like when examined across the entire life course? Specifically, the authors were interested in determining if more than one offender type existed in their sample of delinquent boys. Their results were quite revealing. Desistance from crime was the norm, across all crime types and for all groups of offenders. In other words, no matter what type of crime was examined, or which type of offender an individual was identified as, all offenders eventually desisted. These results, as argued by the authors, stand in contrast to other theoretical approaches which typify offenders into two categories: desisters and nondesisters (see for example Moffitt, 1993). However, it is debatable whether Laub and Sampson's results actually run counter to these theoretical statements (Moffitt, 2006).

To address the second and third research questions (factors accounting for change and stability in criminal trajectories), the authors employed a mixed methodological approach. Specifically, qualitative and quantitative data were both analyzed. For the most part, the information gained from qualitative interviews was also assessed in the quantitative models. Thus, for the purposes of this discussion, only the quantitative results will be discussed. Similar to their 1993 work, Laub and Sampson (2003) sought to address the question of whether
marriage and employment could account for changes in crime. To assess this question, the authors employed growth curve modeling techniques. These models allow a researcher to model longitudinal trajectories of offending. At the same time, the researcher can estimate the effects of time-dependent variables to determine whether they are important for predicting changes in offending trajectories. In line with their earlier work, Laub and Sampson examined whether marriage and employment had an effect on crime involvement. Their findings fell in line with their theoretical arguments and their previous research. Intraindividual changes in marital status predicted decreases in crime. A person that reported getting married evinced a decrease in the number of crimes committed. Additionally, individuals who were married longer exhibited lower levels of crime than those who were unmarried or who were involved in short-term marriages. As for the effects of employment, Laub and Sampson's models showed that individuals who were unemployed for longer periods of time were involved in more crime and intraindividual changes in employment status covaried with contemporaneous levels of crime involvement. Gaining employment predicted a reduction in crime. Finally, military involvement predicted intraindividual changes in crime. When a person was in the military, they were less likely to commit a crime compared to when they were not in the military.

The above discussed findings point to several conclusions. First, the effects of marriage, employment, and military involvement are important predictors of intraindividual changes in crime. This means that within-person changes in crime involvement coincided with changes in marital, employment, or military status. Second, marriage and employment exhibited inter-individual effects, meaning that persons who were married longer and who had longer employment histories committed fewer crimes. It appears, therefore, that these three factors are important for predicting stability and change in offending behaviors. The absence of any of the three adult social bonds means that the person is at greater risk for continued involvement in crime. The presence of any one of the three social bonds, on the other hand, means that the person is likely to reduce criminal involvement. In general, it appears that these social bonds: 1) 'knife off' the past from the present; 2) provide both supervision and monitoring as well as new opportunities of social support and growth; 3) change and structure routine activities; and/or 4) provide the opportunity for identity transformation (Laub, Sampson, and Sweeten, 2006:324).
Outside of the work presented by Sampson and Laub (1993; Laub and Sampson, 2003), a growing body of research has examined the factors that predict desistance from crime (Rhule-Louie and McMahon, 2007; Siennick and Osgood, 2008). The effects of marriage and employment have been the primary focus; however, several other social bonds have emerged as predictors of desistance as well. The following paragraphs will review this literature in order to assess the current state of knowledge surrounding the desistance process (for a discussion of desistance as a process rather than a discrete event, see Bushway et al., 2001; Bushway, Thornberry, Krohn, 2003; Fagan, 1989; Paternoster and Bushway, 2009).  

3.2.3.1 Marriage and Changes in Criminal Behavior

One of the more consistent findings in the criminological literature is the "marriage effect" on desistance from crime (Siennick and Osgood, 2008). Although there is some conflicting evidence, most studies indicate that marital status is inversely related to crime, especially for individuals who are attached to their spouse (Beaver, Wright, DeLisi, and Vaughn, 2008a; Benda and Toombs, 2002; Bersani, Laub, and Nieuwbeerta, 2009; Blokland and Nieuwbeerta, 2005; Horney et al., 1995; King et al., 2007; Laub et al., 1998; Maume et al., 2005; Piquero, MacDonald, et al., 2002; Ragan and Beaver, in press; Sampson and Laub, 1990; Sampson et al., 2006; Shover, 1996; Warr, 1998). This line of literature has examined the marriage effect using different samples and various statistical techniques. For example, after employing inverse probability of treatment weights to the marriage effect, Sampson et al. (2006) estimated that marriage led to more than a 30 percent reduction in criminal offenses. In fact, the authors concluded that their findings were consistent with a causal effect of marriage on desistance processes (Sampson et al., 2006:498).

Horney et al. (1995) estimated a multi-level model similar to that of Laub and Sampson (2003). Their findings indicated that intraindividual changes in marital status—specifically, going from unmarried to married—was associated with decreased odds of committing an assault. There was no relationship between marital status and the odds of committing a property crime or a drug crime. Interestingly, their results indicated that living with a girlfriend increased the odds

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5 See also Vaughan (2007), Uggen and Piliavin (1998), Caspi and Moffitt (1993), and Nagin and Paternoster (1994) for more global considerations of desistance as a concept and a theory of desistance.
of committing drug crimes. The divergence in these findings may reflect the attachment aspect of a marriage. Recall that Sampson and Laub (1993) argued that marital status itself may not have an effect on crime. Only if the person invests social capital (i.e., is attached to spouse and committed to the marriage) will marriage reduce offending. Perhaps, living with a girlfriend reflects an event that looks like a marriage on the surface, but is instead characterized by less investment of social capital (i.e., less commitment). There is some evidence of this point. Specifically, cohabiting with a non-spousal partner offers many benefits, but across a range of outcomes (e.g., sexual satisfaction and mortality rates) married individuals fare better (Waite, 1995).

King et al. (2007) employed propensity score matching to examine the marriage effect. Using the National Youth Survey (NYS) data, the authors predicted each individual's propensity to marry based on 16 covariates (i.e., the data were balanced on these 16 covariates). The results of the propensity score analysis indicated that marriage had a crime reducing effect for males. The models for females indicated that marital status was associated with a reduction in crime; however, these models did not reach statistical significance. The authors performed an additional analysis that examined the marriage effect for males in further detail. The results from this secondary analysis indicated that the marriage effect was primarily confined to those respondents who had the lowest propensity to marry. This finding falls in line with Sampson and Laub's (1993) hypothesis that self-selection into marriage does not fully account for the marriage effect. Those most likely to benefit from marriage are those that are least likely to enter into a marriage.

Not all studies, however, have supported Sampson and Laub's (1993) theory regarding the marriage effect. Some have found support for the marriage effect, but question the causal processes driving the effect (Giordano et al., 2002, 2003; Warr, 1998). Rather than affecting social bonds, Warr (1998) argued that marriage reduces a person's exposure to delinquent peer groups. Thus, opportunities and exposure to crime-encouraging definitions and reinforcement are reduced after marriage. Support for this claim was previously reported by Osgood and Lee (1993). Osgood and Lee found that married persons spent less time with their friends than did non-married persons. To put his hypothesis regarding the relationship between marriage, delinquent peers, and desistance to the test, Warr (1998) analyzed data from the 5th and 6th waves of the NYS. He examined desistance from marijuana use by including only those respondents
who reported marijuana use at wave 5 in the sample. Thus, he could analyze changes in marijuana use from wave 5 to wave 6. Those who quit using marijuana were coded as desisters, while those who continued their marijuana use through wave 6 were coded as non-desisters. A similar coding strategy was followed for the marriage and delinquent peers measure. Those who were unmarried at wave 5 but were married at wave 6 were coded as "1" while all others were coded as "0." In this way, Warr was able to capture changes in marijuana use that coincide (or are at least within the same year) with changes in marital status. Finally, the delinquent peers measure captured changes in delinquent peer exposure from wave 5 to wave 6. Warr found support for his hypothesis: changes in marital status predicted marijuana desistance. This association, however, was completely mediated by the changes in delinquent peer exposure. Maume and colleagues (2005) used Warr's analysis as a springboard and found divergent results. Also using data from waves 5 and 6 of the NYS, their models showed that entering a marriage characterized by high levels of attachment predicted desistance from marijuana. This marriage effect remained after controlling for changes in delinquent peer exposure and changes in definitions favorable to marijuana use. Thus, the marriage effect could not be fully explained by changes in exposure to delinquent peer groups.

There are also several studies that present mixed or conflicting results in regards to the marriage effect (Arnett, 1998; Blokland and Nieuwbeerta, 2005; Capaldi et al., 2008; Giordano et al., 2002; Knight, Osborn and West, 1977; Piquero, Brame, et al., 2002; Simons et al., 2002; Stouthamer-Loeber et al., 2004; Thornberry et al., 1985; Uggen and Kruttschnitt, 1998; Wright and Wright, 1992). In one of the earliest studies to examine the effect of marriage on delinquent and analogous behaviors, Knight et al. (1977) found that marriage was unrelated to delinquency, whether measured as self-reported behavior or official records. Marital status was, however, related to other forms of antisocial behavior such as drug use. Thornberry et al. (1985) found that marital status—after accounting for employment status and whether the respondent had dropped out of high school—was not significantly related to arrest rates for a subsample of males drawn from the Philadelphia Birth Cohort of 1945. Arnett (1998) found that marital status was linked with risk factors for delinquency (e.g., risk taking) but was not significantly predictive of several criminal behaviors such as driving under the influence.

Uggen and Kruttschnitt (1998) examined a sample of high-risk individuals (e.g., welfare recipients, hardcore drug users, recently released ex-offenders, and youth dropouts; data were
taken from the National Supported Work Demonstration Project (NSWDP) who were exposed to a random assignment experiment which offered employment opportunities to those in the treatment condition. Although their aim was not to address the efficacy of the experimental condition, the sample offered an opportunity to examine desistance for a high risk population. Several findings were noteworthy. First, males and females differed in their probability of recidivism. The proportion of males that reoffended was significantly greater than was the proportion of females that reoffended. Second, the survival curve observed for males was significantly different than that observed for females. Males reoffended more often and were quicker to reoffend than females. Third, and directly related to the current discussion, living with a spouse did not have a significant impact on the hazards of recidivism for males or females. In other words, there was no difference in survival times between respondents that reported living with a spouse and those who did not live with a spouse. Thus, this study failed to find evidence of a marriage effect on recidivism.

Giordano and colleagues (2002) analyzed a sample of male and female respondents who were confined to state-level institutions as juveniles. Like Sampson and Laub (1993; Laub and Sampson, 2003), the authors employed a mixed-methodological research strategy drawing on quantitative and qualitative data. The quantitative analyses indicated that "attachment to spouse" did not have a significant effect on crime, whether measured via self-reports or official records. However, when attachment to spouse and job stability were combined into a single item, the results were inversely related with crime. In other words, individuals with higher scores on this "respectability package" (e.g., more attachment to spouse and more job stability) were less likely to be involved in crime.

Piquero, Brame, and colleagues (2002) analyzed a sample of male parolees ranging in age from 16 to 22. The authors first estimated the different latent trajectories of offending exhibited in the sample. Four different trajectories were found. To study the effects of local life circumstances, the authors combined employment and marriage effects into a single construct labeled "stakes in conformity." After breaking the sample apart based on trajectory grouping, the results indicated that the stakes in conformity variable was negatively related to violent and nonviolent crime, however the effects were not statistically significant. Results from supplementary analyses showed that employment, not marriage, was significantly related to reductions in offending (Piquero, Brame, et al., 2002:163).
Stouthamer-Loeber and colleagues (2004) examined the factors that affected various offending patterns among respondents in the Pittsburgh Youth Study. Specifically, the authors divided their sample into three groups: persisters, non-persisters, and desisters. The authors compared percentages of respondents that were in a relationship with a girlfriend or a wife across the three groups of offenders. The results indicated no significant difference. In other words, the desisters were no more likely to be involved in a relationship than were the persisters. Since these results were not specific to marriages, it is unclear how they relate to Sampson and Laub's (1993) theory. Similarly, Simons et al. (2002) found mixed results for the effect of relationship quality on criminal behavior for their sample of young adults. For the female subsample, the quality of a romantic relationship was inversely related to criminal behavior. However, the male subsample failed to display a significant association between relationship quality and criminal behavior. Since this study did not differentiate various types of romantic relationships (i.e., dating, cohabiting, married) it is unclear how these findings inform Sampson and Laub's (1993) theory.

Finally, in a comprehensive study regarding the effect of adult social bonds on crime, Blokland and Nieuwbeerta (2005) examined the effect of marital status on criminal arrests and on self-reported offending across two separate samples of respondents. The first sample consisted of a random drawing of all criminal arrestees in the Netherlands in 1977. The second sample was drawn from a nationally representative study of residents in the Netherlands in 1996. There were a number of layers to the analysis. First, semi-parametric group based modeling procedures were performed separately on the two datasets. The findings revealed four groups of offenders in the criminal arrests dataset. Only two groups of offenders were found in the national sample. Second, after separating the two samples according to group membership, they estimated a multi-level model that allowed for the simultaneous estimation of between and within-individual differences. These analyses showed that between-individual differences in marriage propensity predicted differences in arrests and in self-reported crime across all groups. In other words, respondents—regardless of group membership—who were married for longer periods of time were less likely to be arrested and reported fewer crimes. Interestingly, the within-individual analyses produced less consistent results. When considering the sample of arrestees, marital status was inversely related to arrests for low rate and moderate rate offenders. For sporadic and high rate offenders, however, there was no effect of marital status on arrests.
For the national probability sample, marital status was unrelated to self-reported offending. However, when analyzing the effects of crime in the aggregate, the authors noted that marriage reduced levels of offending, above and beyond the effects of age.

Although there is not a clear consensus in the literature, the majority of the findings flowing from these studies indicated a negative association between marital status and criminal behavior. Perhaps not surprisingly, however, the debate concerning how and why marriage affects desistance has yet to be resolved (Sampson and Laub, 1993; Warr, 1998). As previously discussed, Sampson and Laub contended that getting married leads to changes in social capital. In short, persons who are married have more stakes in conformity than persons that are unmarried. Marriage, for Sampson and Laub, represents a change in a person's lifestyle such that the individual is no longer the central concern. Instead, there is another person who is reliant on the individual and that will be affected by their actions. In this way, entering into a marriage will act as an inhibitory factor for future criminal behavior because the individual has more to lose if caught. It is not that the criminal motivation is removed when one gets married —control theories assume that individuals are always motivated for criminal behavior. Instead, as Sampson et al (2006) stated:

…marriage has the potential to "knife-off" the past from the present in the lives of disadvantaged men and lead to one or more of the following: opportunities for investment in new relationships that offer social support, growth, and new social networks; structured routines that center more on family life and less on unstructured time with peers; forms of direct and indirect supervision and monitoring behavior; or situations that provide an opportunity for identity transformation and that allow for the emergence of a new self or script what Hill (1971) described as the "movement from a hell raiser to a family man."

3.2.3.2 Employment and Changes in Criminal Behavior

In addition to marriage, factors tapping employment status and employment stability are often linked with desistance processes. As previously reviewed, Sampson and Laub (1993; Laub and Sampson, 2003) found that offenders who had a stable job were much less likely to offend. Furthermore, intraindividual differences in job stability and employment status were linked with
changes in offending rates. The findings gleaned from researchers other than Sampson and Laub, however, are far less consistent. A number of studies have found that employment and job stability is inversely related to crime (Berk et al., 1980; Crutchfield and Pitchford, 1997; Lattimore et al., 1990; Sampson and Laub, 1990; Thornberry and Christenson, 1984; Thornberry et al., 1985; Uggen, 1999), others have failed to identify a link between employment and crime (Giordano et al., 2002; Maume et al., 2005; O'Connell, 2003; Warr, 1998), and still others have presented mixed results where employment status is only important for certain individuals or under certain circumstances (Cernkovich and Giordano, 2001; Piquero, MacDonald et al., 2002; Simons et al., 2002; Uggen, 2000; Uggen and Kruttschnitt, 1998). Some have even found that employment is positively related to crime and delinquency (i.e., more crime when employed; Horney et al., 1995). Horney and colleagues (1995) found that employment status was positively related to property crime among their sample of male inmates in Nebraska. Specifically, the authors concluded that, "the odds of committing a property crime increased by 28 percent in the months when men worked" (Horney et al., 1995:667). With the exception of the study by Horney and colleagues, the positive effect of employment on delinquency appears to be limited to the adolescent years and may be more indicative of an underlying propensity of criminality rather than a causal effect of employment (for a review and recent evidence of employment on adolescent delinquency, see Apel et al., 2007).

There is a large body of evidence bearing on the association between employment status and crime involvement or crime rates. Although the results have been inconsistent across studies, it appears that employment status is linked with crime involvement (Chiricos, 1987; Crutchfield and Pitchford, 1997; Thornberry and Christenson, 1984; Thornberry et al., 1985). Thornberry et al. (1985) found that respondents that reported longer bouts of unemployment had higher arrest counts than those reporting fewer problems with unemployment. In another study, Crutchfield and Pitchford (1997) analyzed data drawn from the National Longitudinal Surveys of Youth (NLSY) Labor Market Experience. They found that employment status was linked with both violent and property crime. Additionally, Crutchfield and Pitchford found that respondents that expected to stay at their current job were less likely to be involved in both types of crime. Despite the large body of evidence linking employment status and crime involvement, most of this research does not measure recidivism or desistance. Thus, the relevance of this research for the current purposes is unclear.
One exception is Uggen's (1999) evaluation of an experimental job assignment program. In this study, Uggen examined the relationship between job quality and recidivism among a sample of ex-offenders that participated in a random assignment job placement study (i.e., the NSWDP). Uggen was interested in the effects of job quality since social control theories (e.g., Sampson and Laub, 1993) and strain theories (e.g., Merton, 1938) hypothesize that attachment to a job, rather than a job in and of itself, should have an impact on recidivism. The relationship between job quality and recidivism was negative, in line with Sampson and Laub's (1993) theory. Ex-offenders who obtained employment of a higher quality were less likely to recidivate. Although these bivariate findings are informative, it was important to determine whether the relationship held after selection effects were controlled. Uggen recognized that selection into higher quality employment is not a random process. As a result, he estimated a bivariate probit model which accounted for the selection into employment while simultaneously estimating the effect of job quality on recidivism. The results from this analysis supported the bivariate findings. In short, jobs of higher quality were associated with a lower risk of recidivism. This relationship was observed across different specifications of the crime variable; job quality was inversely related to economic (e.g., burglary, robbery, and drug sales) and non-economic crimes (e.g., assault and arson). Interestingly, the job quality effect was larger for non-economic crimes.

Lattimore et al. (1990) evaluated the impact of an experimental program that offered vocational training and assistance in finding employment to a group of property offenders. Subjects were randomly assigned to receive the program services (i.e., the treatment group) or to receive routine services offered to all offenders (i.e., the control group). Most importantly, the treatment group received community reentry training and job development services while the control group did not. Although the vast majority of offenders did not receive the full range of services (i.e., very few offenders completed all stages of the program), those who were assigned to the treatment group evidenced lower levels of recidivism and longer "survival times." It should be noted, however, that these differences between the treatment and control groups were not statistically significant by conventional standards ($p = .12$). Nonetheless, offenders receiving the vocational training appeared to be more likely to desist from crime.

Berk and colleagues (1980) analyzed data drawn from an experimental program involving young adults who had recently been released from prison ($n = 2,000$). Respondents were randomly assigned to either a treatment or a control group. Unlike most experiments, this
program included four treatment groups and two control groups. The main difference between the treatment groups was the amount of funding that was allocated to each offender. Most respondents appearing in the treatment group received supplemental funds (between $500 and $2,000) similar to unemployment benefits for a certain period of time (between 13 and 26 weeks). Other respondents in the treatment group did not receive funds but were provided job placement opportunities. In order to determine whether the experimental conditions influenced recidivism, the authors analyzed the number of property and nonproperty arrests over a 12 month follow-up. The results indicated that the treatment condition (measured dichotomously as being in the treatment group or not) did not have an effect on arrests of either variety. Simply being assigned to the treatment group had no effect on recidivism. However, when the amount of money given to those in the treatment condition was considered, an effect on recidivism emerged: respondents receiving more unemployment benefits (i.e., more money) were less likely to be arrested for either a property or a nonproperty offense. Additionally, the results revealed that employment status was linked to recidivism. Respondents who were employed for longer periods of time had lower arrest rates for both offense types over the 12 month follow-up.

As mentioned above, some studies have failed to identify a link between crime and employment status. Among their sample of previously incarcerated juvenile delinquents, Giordano et al. (2002) found that job stability was unrelated to self-reports of delinquency and official criminal records. Although the effect of job stability on crime was negative—in line with Sampson and Laub's predictions—it failed to reach statistical significance. Their measure of job stability, however, was different than that of prior researchers. Rather than tapping the respondent's attachment to their job and how long they had held the job, Giordano and colleagues asked the respondents to indicate the likelihood that they would leave or get fired from their job within the next two years. Thus, the extent to which this measure overlaps with measures used by other researchers such as Sampson and Laub is unclear and may have a part in explaining the divergent results. In another study, Warr (1998) found that having a job was unrelated to theft and vandalism but was significantly related (positive effect) to marijuana use and alcohol use: respondents that were employed reported more marijuana and alcohol use. Furthermore, employment status was unrelated to desistance from marijuana use in both Warr's (1998) study as well as Maume et al. (2005).
O'Connell (2003) examined a sample of convicted offenders. Information was drawn from each offender at the time of release from prison, 6 months post-release, and 18 months post-release. Recidivism (i.e., whether the subject was rearrested) was measured at the 18 month follow-up, while information concerning adult social bonds was collected at the 6 month follow-up. The results from structural equation models indicated that employment status (i.e., whether the respondent was working) was not associated with recidivism. Employment status, however, was associated with reduced risk of drug use.

Other researchers have argued that employment status and job stability are related to changes in delinquency, but only under certain circumstances. Cernkovich and Giordano (2001) analyzed two separate samples of young adults. The first sample was drawn from a probability sample of youth living in Ohio. The second sample was drawn from three juvenile institutions in Ohio. Their analysis examined whether involvement in crime was influenced by social bonds while controlling for prior levels of delinquency. Separate analyses were conducted for each sample. The results showed that subjects who reported being more satisfied with their current economic situation—measured as the respondent's level of satisfaction with their current employment or job prospects, their financial situation, their personal achievements, their economic prospects, and their material possessions—were less likely to be involved in crime. This effect, however, was only present when analyzing the sample drawn from the non-institutionalized sample. Their analysis of the institutionalized sample found no link between economic satisfaction and crime.

Piquero, MacDonald, and colleagues (2002) analyzed data from more than 500 parolees and the findings indicated that full-time employment status was unrelated to total arrests, nonviolent arrests, and violent arrests. The authors, however, went one step further by re-estimating their models after disaggregating the sample by race. These analyses indicated that, for the most part, employment status did not predict differences in arrest rates. However, white parolees that were employed full-time were less likely to be arrested for a violent offense. These effects were observed at both the between-individual level as well as at the within-individual level of analysis. In other words, whites that were employed were less likely to be arrested for a violent crime than were whites that were unemployed. At the same time, individuals were less likely to be arrested during times of employment compared to times of unemployment.
In another study, Simons et al. (2002) constructed a job attachment scale that included 23 items. Employing structural equation modeling techniques, the authors found that job attachment was significantly associated with criminal behavior for males but not for females. Specifically, more job attachment predicted less criminal behavior for males. For the female subsample, job attachment did not have a direct effect on criminal behavior, but it did moderate the relationship between prior delinquency and criminal behavior. Specifically, the relationship between past delinquency and criminal behavior was stronger for females with low job attachment.

As discussed above, Uggen and Kruttschnitt (1998) analyzed survival curves for females and males who participated in a random assignment job opportunity experiment (i.e., the National Supported Work Demonstration Project [NSWDP]). Those in the treatment condition were given employment opportunities and those in the control conditions were not given these opportunities. Uggen and Kruttschnitt analyzed whether several variables affected a person’s time to reoffense (i.e., proportional hazards models). Examining time to reoffense separately for males and females revealed several interesting findings. First, indicators of employment history, current employment status, and the perceived risk of losing one’s job if arrested were unrelated to female reoffense patterns. In other words, females who had a longer employment history, females who were working in a job offered by the program, females who were working in a job not offered by the program, and females who perceived a greater risk of losing their job if arrested did not have significantly shorter times to reoffense than those with shorter employment histories, those not working, and those who perceived less of a risk of losing their job if arrested. Second, although indicators of employment were unrelated to female reoffense patterns, they were related to female arrest patterns. Females who were employed—whether or not the job was provided as part of the experiment—had lower hazards of rearrest (i.e., longer times until rearrest). The length of time spent at the current job and the perceived risk of losing one’s job if rearrested was unrelated to rearrest risk for females. Third, the patterns observed for males were slightly different than those observed for females. Males who were employed as part of the experimental program had lower hazards of reoffense. Perceived risk of losing one’s job and length of time employed was unrelated to time to reoffense for males. As for risk of rearrest, males who were employed—whether the job was provided as part of the experiment or not—had lower hazards of arrest risk. In sum, this study extends evidence that partially supports the
hypothesis that employment opportunities will lead to desistance. These factors, however, may be conditioned by the sex of the respondent.

In a more recent study, Uggen (2000) analyzed data from a job experiment (i.e., the NSWDP) to determine whether employment had an effect on crime. As previously noted, the NSWDP was a random assignment experiment where those assigned to the treatment condition were offered minimum-wage jobs (typically construction or service industry work) and those in the control group were interviewed but were not offered employment opportunities. Subjects from both groups were interviewed in nine-month intervals for up to three years. Uggen assessed whether each subject had been rearrested and whether the subject self-reported involvement in illegal activity (i.e., self-report of illegal earnings). At the end of the three year research period, more than half of all subjects had been rearrested. First, the findings indicated that subjects appearing in the treatment group (i.e., those that were given job opportunities) who were 26 years old or younger did not significantly differ in rearrest probability over the three year follow-up period. In other words, those who were given jobs as part of the experiment were no less likely to be rearrested than those in the control group. Second, subjects aged 27 and older had significantly different outcomes. Subjects who were 27 and older that were given job opportunities (i.e., the treatment group) were significantly less likely to be rearrested as compared to the control group (i.e., those not given job opportunities). Third, this same pattern of results held up when the "illegal earnings" variable was analyzed. Fourth, subjects in the treatment group had lower hazards of rearrest. Thus, participants who were provided job opportunities were less likely to be rearrested and to recidivate. Fifth, these effects were robust to controls for race, sex, marital status, work history, prior record, and measures of program participation. In sum, Uggen's study indicated that a job—even one that does not dramatically shift the respondent's socioeconomic standing—can be a turning point in the life course for some offenders, especially older offenders.

3.2.3.3 Military Involvement and Changes in Criminal Behavior

The third adult social bond highlighted by Sampson and Laub (1993; Laub and Sampson, 2003) is involvement in the military. Although there is a growing body of literature bearing on the effects of marriage and employment on desistance, the impact of military involvement has
received very little attention from researchers. For instance, a general search for the keywords "desistance" and "military" was carried out in the Criminal Justice Periodicals Index and no relevant studies were retrieved. There are, however, a few studies that have included military involvement as a predictor of criminal behavior and, therefore, have a bearing on the current discussion (Crutchfield and Pitchford, 1997; Mattick, 1960; Wright, Carter, and Cullen, 2005). Sampson and Laub (1993; Laub and Sampson, 2003) focused much attention on the effects of the military. These findings have already been discussed and will not be reviewed here. Suffice it to say, however, that Sampson and Laub found mixed results for the effects of military involvement on changes in crime. In some analyses military involvement appeared to significantly predict changes in crime, while in other specifications it failed to reach statistical significance. The qualitative information presented by Sampson and Laub, however, highlighted the importance of military involvement for certain respondents.

These mixed results of military involvement on crime hold true for other studies not authored by Sampson and Laub as well. In one of the first studies to examine the effect of military exposure on recidivism, Mattick (1960) presented data drawn from an experimental design that assigned parolees to military services during World War II. From 1940 to 1947, the Illinois State Penitentiary System paroled nearly 3,000 men to military services as opposed to traditional parole services (i.e., community supervision). As a whole, the program appeared to be a success by several objective standards. For example, only 3.4 percent of military parolees violated their parole. This is a remarkable figure when one considers that the population of parolees not sent to the military violated at a rate of 36.5 percent during the same time period. When recidivism rates are examined, the picture is very similar. Men paroled to the military had a recidivism rate of 10.5 percent compared to 66.6 percent for ex-felons not paroled to the military. In short, it appears from this analysis that military involvement may have some crime-reducing effects for ex-felons.

Studies published since Mattick (1960), have, however, been less supportive of a "military effect" on crime. One such study is that by Crutchfield and Pitchford (1997). Details concerning Crutchfield and Pitchford's analysis have already been discussed. Recall that the primary focus of their study was to determine the effects of employment status on involvement in property and violent crime. They did, however, include military involvement as a control variable. The data were analyzed with an ordinary regression model. In other words, the authors
did not account for within-individual differences in military involvement, only between-individual differences were estimated and military service was not randomly assigned, thereby limiting the conclusions that can be drawn from this analysis. The results from their analysis showed that individuals that reported military involvement had higher rates of self-reported violent crime. Military involvement was unrelated to self-reported involvement in property crime.

In another study, Wright, Carter, and Cullen (2005) analyzed a longitudinal dataset of adolescent males who were sophomores in high-school in 1964. More than half of all respondents eventually served time in the military, with the majority of those respondents serving in Vietnam. Most respondents who served in Vietnam voluntarily enlisted (i.e., they were not drafted). The fact that Wright and colleagues used a sample of Vietnam veterans is a distinguishing point. Sampson and Laub's (1993) sample, the Glueck men, were mostly exposed to military service during World War II and the Korean War, both of which involved a draft. Thus, many of the men that entered the military in Sampson and Laub's sample may have been drafted. The conditional term "may" is used here because it is not clear from any of Sampson and Laub's work how many men were drafted and how many willingly enlisted. Although a draft was instituted for the Vietnam War, most respondents in Wright et al.'s sample enlisted without being drafted—approximately 80 percent enlisted. Thus, selection effects into the military may be less of a concern for Sampson and Laub's sample than for Wright et al.'s or Crutchfield and Pitchford's (1997) sample. In other words, military service, for Sampson and Laub's sample may be considered a "random event" that is less affected by self-selection than is the case for Wright et al.'s sample. Additionally, the social climates that surrounded World War II and the Korean War were much different than what surrounded the Vietnam War. As Wright et al. explained, the Vietnam War was followed by much less of an economic "boom" and political support for the Vietnam War was much lower than was seen during World War II and the Korean War. Thus, it is reasonable to expect that exposure to the Vietnam War may have had a different impact on offending careers than did World War II or the Korean War. In short, any divergences between Sampson and Laub's findings and those presented by Wright et al. may reflect some or all of these distinguishing points of the Vietnam War and Wright et al.'s sample.

Several findings from Wright, Carter and Cullen's (2005) study warrant close attention. First, the authors' employed latent growth curve modeling which allows for the estimation of
within-individual changes. These models indicated that individuals who served in Vietnam accelerated their levels of drug use more quickly than did those who did not serve in Vietnam. Second, individuals who served in Vietnam had significantly more military arrests than those not serving in Vietnam. Third, Vietnam service was not directly associated with adult arrest rates that occurred later in life. In all, it appears that service in Vietnam was an accelerating factor for drug use and for certain crime (i.e., military crime). These findings directly contradict Sampson and Laub's (1993; Laub and Sampson, 2003) analyses which showed that military involvement decreased criminal involvement. Perhaps Wright and colleagues' analysis has revealed the complexity of military service and the differential effects that being exposed to military service (e.g., during different political climates) can have on individuals. Because of this, it is questionable whether joining the military can be considered a factor that will inhibit future crime for all people in all situations. Maybe during times of peace, military involvement will serve as a structuring agent for individuals and will become a "script for change." In other cases, especially during times of war, military service may have widely varying effects across individuals (Elder, 1986). Unfortunately, at this point, the literature is too limited to draw any firm conclusions regarding these issues. All that can be said with any certainty is that the effects of military involvement may be contingent on individual background factors as well as on macro-social variables such as political support for the military.

3.2.3.4 Religious and Educational Bonds and Changes in Criminal Behavior

Sampson and Laub (1993; Laub and Sampson, 2003) primarily focused on three adult social bonds: marriage, employment, and military involvement. As reviewed above, a growing body of literature has examined the link between these social bonds and crime involvement. However, these three adult social bonds are not the only adult transitions thought to be important for desistance from crime. Indeed, scholars have found a number of other social bonds to be important predictors of desistance such as religiosity (Benda and Toombs, 2002; Chu, 2007; Chu and Sung, 2009; Giordano et al., 2002: 1036; Giordano et al., 2008) and educational bonds
As noted by Chu (2007), no theoretical accounts of desistance include a discussion of the role of religiosity. Chu noted, however, that religiosity may have a deterrent effect on crime and, as such, may be important for understanding desistance from antisocial behaviors. Drawing on Sampson and Laub's (1993) discussion of social capital, Chu argued that individuals may begin to build bonds to society via their personal relationships (i.e., marriage), their professional life (i.e., employment), and their spiritual life (i.e., religiosity). Chu's analysis indicated that religiosity predicted desistance from drug use among respondents in the NYS. Other evidence supporting Chu's (2007) hypothesis can be drawn from the work of Giordano and colleagues (2002, 2008). Although, Giordano et al. (2002, 2008) failed to find a significant link between religiosity and desistance in their quantitative analyses, their qualitative findings suggested that offenders sometimes "find religion" while in prison. Those who do become religious while incarcerated often reference their faith as being a primary motivator to stop their criminal involvement. In short, religiosity may represent an important "hook for change" for many offenders.

Benda and Toombs (2002) estimated the effect of religiosity on recidivism for male offenders. Religiosity was measured by asking respondents to report the frequency with which they attended church, how often they prayed, how involved they were with church activities, how often they talked about religion, and how often they tried to convert others to their religious preference. Recidivism was measured as a new arrest or a parole violation within three years after being released from an adult boot camp. The results indicated that respondents with higher levels of religiosity had lower hazard rates of recidivism. In other words, respondents that were more religious were less likely to recidivate and they had longer "survival" times when they did recidivate. Finally, Chu and Sung (2009) reported that religiosity was a predictor of desistance from drug use for Blacks but not for White respondents.

It should be noted that there are a host of other variables known to predict desistance. For example, childbearing and parenthood (Ayers et al., 1999; Blokland and Nieuwbeerta, 2005; Giordano et al., 2002; Kreager et al., 2010; Thompson and Petrovic, 2009; Uggen and Kruttschnitt, 1998), serving time in prison (Lanctot et al., 2007; Giordano et al., 2002), and drug use (Ayers et al., 1999; Huebner et al., 2007; Hussong et al., 2004; Schroeder et al., 2007) have been shown to affect desistance processes. However, these variables do not represent adult social bonds in the same sense as do marriage, employment, religious involvement, and education. As a result, a full-length discussion of these variables will not be presented.

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Some scholars have noted that crime is less likely to occur when a person is involved in school or has more educational bonds. Horney et al. (1995) found that within-person changes in crime, especially drug crime, occurred during periods when a respondent reported they were attending school. The direction of these effects revealed that being in school led to a reduction in crime. Employing a similar analytic strategy, Blokland and Nieuwebeerta (2005) reported that individuals that spent more time as a student actually had higher rates of self-reported offending. However, when considering the within-individual results, the relationship between student status and self-reported crime was negative (i.e., less crime during times of student status). Ayers et al. (1999) found that youth who desisted from delinquency had higher schoolwork skills, better grades and test scores, and more commitment to school than did non-desisters. Individuals with more education had lower hazards of recidivism in Benda and Toombs’ (2002) analysis. Uggen and Kruttschnitt (1998) reported that being in school translated into a marked reduction in the hazards of rearrest among their high-risk sample and O’Connell (2003) reported similar findings. Shover and Thompson (1992) analyzed a sample of incarcerated offenders to determine the factors that predicted desistance from crime. Desistance was measured as having no arrests for 36 months post-release. Logistic regression models showed that ex-offenders with a higher level of education were more likely to remain arrest free for the 36 month follow-up period.

3.3 SUMMARY AND DISCUSSION

Sampson and Laub’s theory put forth three organizing hypotheses. The first hypothesis generally stated that the onset of delinquency occurs in adolescence as a result of family process factors. Family process factors include the type of parenting that the child is exposed to and the level of attachment between parent and child. In addition to family processes, school attachment and exposure to delinquent peer groups will influence the onset of adolescent delinquency. Although a long line of research has shown that structural background factors like growing up in poverty are important predictors of delinquency, Sampson and Laub argued that these effects are more distal processes that work through the family process factors to affect delinquency.

The second hypothesis proffered by Sampson and Laub is that stability in delinquency from adolescence to adulthood can be explained by cumulative continuity or state dependence. Cumulative continuity refers to the process of wagering one's future opportunities based on past
behavior. In other words, Sampson and Laub argued that adult crime is a function of adolescent delinquency. Rather than being caused by a similar underlying trait, as is argued by population heterogeneity theorists such as Gottfredson and Hirschi (1990) and Wilson and Herrnstein (1985), Sampson and Laub argued that adult crime is simply a function of past behavior. Adolescent delinquency can knife-off future opportunities making adult crime a more likely outcome.

Finally, Sampson and Laub's third hypothesis states that change in crime can occur in adulthood as a function of the person's exposure to adult social bonds. Adult social bonds have inhibitory effects on future crime by giving the person a stake in conformity. In this way, the social bond becomes a social control mechanism (Hirschi, 1969) that reduces the likelihood that the person will continue a life of crime. In order to contextualize the concept of adult social bonds as a social control mechanism, Sampson and Laub argued that social bonds will act as turning points in the life-course. These turning points will divert criminal trajectories and eventually cause the person to desist from criminal activity. Sampson and Laub highlighted marriage, employment, and military involvement as the three most important adult social bonds.

This chapter reviewed the criminological evidence bearing on Sampson and Laub's theory. Much of this evidence can be drawn from Sampson and Laub's two books (1993 and 2003). Although the first two hypotheses drawn from the theory (i.e., factors affecting the onset of adolescent delinquency and the effect of past behavior on future behavior) have been examined by scholars, this chapter placed greater emphasis on the research that has tested the third hypothesis: whether adult social bonds (i.e., marriage, employment, and military involvement) are turning points that encourage desistance from crime. The rationale for focusing greater attention on the effect of adult social bonds on desistance has not yet been stated explicitly. It is important, therefore, to take a moment and discuss the focal concerns that will motivate the remaining chapters of this dissertation.

As mentioned earlier, only a limited number of studies have examined the effects of adult social bonds on desistance from crime (Piquero et al., 2003). All relevant studies were presented in the preceding section. As was discussed, some evidence supports Sampson and Laub's hypotheses and some evidence runs counter to their predictions. The latter studies, however, raise some interesting questions. For instance, do adult social bonds have uniform affects across all individuals? The answer appears to be "no." Adult social bonds have significant effects in
some samples and under some circumstances but not in others. Another question that this review has raised is whether adult social bonds have instantaneous effects or whether they take time to influence a person's criminal trajectory. Laub et al. (1998) argued that marriage will not reduce crime in and of itself. Instead, Laub and colleagues explained that marriage takes time to affect crime. This argument is consistent with their discussion of social capital (Laub, Sampson, and Sweeten, 2006). In short, the argument states that the person must invest time in the bond (e.g., marriage) and must be attached to the bond (e.g., their spouse) in order for the bond to serve as a turning point.

In all, the preponderance of the evidence indicates that adult social bonds may be related to desistance processes. Some important issues, however, have not been adequately addressed by researchers. The first issue is one of timing. In order for Sampson and Laub's theory of the effect of adult social bonds on desistance to be tenable, it is necessary for adult social bonds to occur prior to the beginning of the desistance process. In short, an adult social bond may well be correlated with deceleration or de-escalation of crime, but if these elements of the desistance process occur prior to the presence of an adult social bond, then the latter cannot cause the former. On the surface, this appears to be a simple issue that could easily be resolved by logic and sound research. Unfortunately, neither has been thoroughly considered.

Consider the age-crime curve. As was discussed, the age-crime curve peaks somewhere around age 18. If desistance is understood as a process rather than a discrete event as many scholars have argued (Bushway et al., 2001; Bushway, Thornberry, Krohn, 2003; Fagan, 1989; Laub and Sampson, 2003; Paternoster and Bushway, 2009), then the age-crime curve tells us that the desistance process begins in early adulthood—when the curve turns downward. But, when do adult social bonds typically set-in? Obviously, an adult social bond must occur at some point in adulthood. Thus, the question becomes, when does adulthood set-in? Of course, there is no hard-and-fast rule governing when a person is considered to have passed from adolescence into adulthood. However, if we accept the legal definition of adulthood as a proxy of this transition, then it would appear that adulthood does not set-in until somewhere between ages 18 and 21. It should now be obvious that the timing of the transition from adolescence to adulthood almost

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7 A similar argument was posed previously by Bushway et al. (2001), but the authors noted that Sampson and Laub (1993) suggested that bonds and delinquency would interact in a complex and dynamic way. This suggestion, according to the authors, releases the adult social bond from the necessity of occurring prior to an individual's initial decline in crime for it to remain an explanatory variable of desistance.
perfectly matches the timing of the onset of desistance from crime as shown by the age-crime curve.

This brings us back to the original question of whether adult social bonds precede the beginning of the desistance process. If adulthood occurs at almost the exact same time in the life-course as desistance, then it will be difficult to discern the temporal ordering of the events. Luckily, there is a small body of research that has investigated the timing of adult social bonds (Fussell and Furstenberg, 2005; Osgood and Lee, 1993). This research shows that many of the adult social bonds that are hypothesized to affect desistance actually appear after desistance processes are already in motion. Osgood and Lee (1993) showed that marriage and full-time employment probabilities (i.e., aggregate measures of the number of people that are married and employed full-time) do not peak until the late 20s/early 30s. For instance, the predicted probability of marriage for an 18 year old is lower than .1, for a 23 year old the predicted probability is less than .4, and the probability for a 28 year old is .6. A similar trend is observed for the probability of full-time employment. Specifically, the predicted probability of full-time employment does not peak until the early 30s (Osgood and Lee, 1993).

More recent estimates from the U.S. Census Bureau (2010) tell the same story. Figure 3.3.1 presents the age-crime curve along with the age-marriage curve (i.e., the proportion of the population that is married)\(^8\). One point is immediately obvious: the age-crime curve peaks before marriage peaks. To be sure, the median age for first marriage was estimated to be 28 for males and 25 for females in 2009 (U.S. Census Bureau, 2009). It is also apparent that the two curves cross somewhere in the mid 20s. In short, crime is on the decline—desistance processes have already begun—well before marriage is likely to occur. If we accept Laub et al.’s (1998) argument that social bonds take time and investment to influence behavior, the temporal ordering of events and direction of causality become even more problematic. Therefore, marriage—and it is likely that stable employment follows this same progression of investment (i.e., social capital)—cannot explain the initial decline nor the deceleration in crime since both begin prior to the onset of most adult social bonds.

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\(^8\) The age-crime curve has been altered to reflect the proportion of the total offenses occurring within each age group, multiplied by 10 to put both curves on a similar measurement scale.
It appears that adult social bonds, at least marriage and employment, may not explain the initial stages of the desistance process, at least at the aggregate level. However, if we discard the understanding of desistance as a process, or if we change the argument to state that adult social bonds accelerate desistance processes, then perhaps adult social bonds can retain some theoretical import. This is an area of research that has not received direct attention. Obviously, the life-course perspective, at least Sampson and Laub's theory, rests on the ability of criminologists to reconcile this issue. Also, it may be the case that the initial decline in crime observed in early adulthood has nothing to do with social bonds and is instead a normative developmental pattern. If this were the case, social bonds would only matter for changes occurring later in the life course or for the discrete event of desistance (i.e., when the person "quits" crime).

Perhaps one way to reconcile the age-crime curve with the age-bond curve is that adult social bonds explain desistance, but only for more serious/frequent offenders. Recall that Sampson and Laub developed their theory out of their observation of the Glueck Men. It is important to remember that 50 percent of their sample consisted of juvenile delinquents and that the majority of Sampson and Laub's analyses were performed exclusively on this "delinquent" subsample. It is possible that these delinquents follow different trajectories of offending and desistance than does the typical (adolescence-limited?) offender. Perhaps the men appearing in their sample follow more of a "persistent offender" trajectory and, as a result, desist from crime much later than the typical offender. Further, perhaps adult social bonds are important for desistance patterns in these "persistent" samples but are less important in the general population.

This argument is consistent with some of Sampson and Laub's findings, but is inconsistent with their theoretical position. Laub et al. (1998) split their sample into four different offending groups. Results showed that marriage and marital quality were important predictors of crime, but only for the groups of offenders that evinced longer criminal careers.

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9 Admittedly, the question is whether adult social bonds lead to within-individual changes in behavior. Aggregate statistics may not be "downwardly" generalizable.

10 See Sampson et al. (2006:486) for an empirical example of this point. The age-crime curve and the age-bond curve is markedly different for the Glueck Men than is found in the general population. Specifically, compare Sampson et al. Figure 1 to Figure 3.3.1 and 3.3.2 from this dissertation. Adding even more complexity to this issue is the analysis by Blokland and Nieuwbeerta (2005) which showed that adult social bonds were less important for more serious/persistent offending groups; findings that are contradictory to Sampson and Laub's (1993) theoretical position.
(compare the results from Group 4 to the results from Groups 1-3). However, Laub and Sampson (2003) are diametrically opposed to typological theories that hypothesize about different offending trajectories (see specifically Moffitt's (1993) theory of adolescence-limited and life-course-persistent offenders). Laub and Sampson (2003:81-113) are very clear on one point: they do not believe that individuals can be classified into different offending trajectories (see also Bersani, Nieuwbeerta, and Laub, 2009). Instead, they argue that each individual will follow a unique trajectory that cannot be predicted prospectively. Perhaps, however, their sample contained a unique offender type and other samples, especially general population samples, include different types of offenders that follow different and identifiable trajectories. It could be that when analyzing a sample of known offenders, more groups are identifiable than when a general population sample is used. This may begin to explain why the marriage effect and the employment effect are not consistent across all studies.

A second way to reconcile the age-crime curve with the age-bond curve is to argue that adult bonds reflect selection effects. In other words, it is possible that individuals most likely to desist are those most likely to get married, get a job, or join the military. If we look closely at the extant literature, we begin to see evidence supportive of this argument. Specifically, in some studies (e.g., Blokland and Nieuwbeerta, 2005) social bonds are important for predicting crime, but only at the between-individual level. When analyses are extended to within-individual differences, the findings are less stable. Assuming that selection effects are operating, what might explain them? Perhaps genetic factors can explain the selection into adult social bonds and the deceleration and desistance from crime.

There is a limited body of research and some theoretical arguments that attempt to deal with the issue of selection into adult social bonds. Wright et al. (1999) found that both self-control and social bonds were important for understanding criminal behavior. Moreover, they found that levels of self-control were mediated by the social bonds. Thus, it appears that self-

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11 Further evidence of this point can be drawn from Blokland and Nieuwbeerta's (2005) analysis. Blokland and Nieuwbeerta analyzed two separate samples. The first sample was drawn from an arrest database. Thus, this sample consists only of known offenders—similar to Sampson and Laub's (1993) delinquent subsample. Blokland and Nieuwbeerta's second sample was a general population sample. The authors performed identical analyses on both samples, but reached markedly different conclusions. Important for the current discussion is that Blokland and Nieuwbeerta found that four offending groups existed in the arrest data. The same number identified by Laub et al. (1998) in their delinquent subsample. Only two groups, however, existed when the general population sample was used. Perhaps a similar phenomenon has occurred with Laub and Sampson's (2003) analysis of the delinquent subsample of the Glueck men.
selection into the local life circumstances of interest can be predicted by personal background factors. Furthermore—and in support of Sampson and Laub's (1993) argument—the inhibitory effect of social bonds does not appear to be spurious to selection effects, at least not completely.

Sampson and Laub obviously took the position that adult social bonds do not merely reflect selection effects. For example, they argued that, "Some changes in the life course result from chance or random events; other changes stem from macro-level shocks largely beyond the pale of individual choice (for example, war, depression, natural disasters, revolutions, plant closings, industrial restructuring)" (Laub and Sampson, 2003:34). They continue this argument by stating that, "Selection into marriage also appears to be less systematic than many think. As we shall demonstrate, many men cannot even articulate why they got married or how they began relationships, which often just seemed to happen by chance." (Laub and Sampson, 2003:45). It is apparent, however, that Sampson and Laub believe that selection effects do, at least partially, confound some of the relationship that their theory is built upon. Consider the following statement: "Selection is surely operating at some level, but most marriages originate in fortuitous contacts rooted in everyday routine activities" (Laub and Sampson, 2003:45). Later, it appears that Sampson and Laub are more open to the idea that selection effects may be important: "As for marriage, a selection argument can be made on the spurious nature of the work-crime connection. It is likely that selection contamination is even greater for employment" (Laub and Sampson, 2003:48).

From the above passages, it is clear the Sampson and Laub are aware that selection effects may underlie some of the associations highlighted by their research and their theory. This issue, however, goes unaddressed by Sampson and Laub (1993) and other criminological researchers. Instead, the authors rely on theoretical arguments to convince the reader that, even if selection effects do matter, there is no way that they could account for all of the effects that marriage or employment has on desistance.
Criminologists have been slow to accept the findings from behavioral genetic research. Rather than offering competing hypotheses and testing them empirically, criminologists often rely on theory and argumentation to dismiss the importance of genetic effects on crime. To date, one of the more common arguments against genetic influences on crime has surrounded desistance research. The argument goes something like this: if genetic effects are important, they can only affect the onset and stability of crime. Since genes and DNA do not change over time, they cannot explain changes in crime across the life course. Thus, genes cannot be important for understanding desistance from crime. This reasoning has led some criminologists to the erroneous belief that controlling for prior levels of delinquency can act as a control for genetic effects.

One of the most prominent examples of this approach is found in Sampson and Laub's (1993) research. Recall that their theory proffered an explanation of the onset of adolescent delinquency, stability in delinquency over time, and changes in crime in adulthood. The authors briefly noted that genetic effects may be important for understanding the onset and stability of delinquency. Since their data did not allow them to directly control for genetic effects, the authors argued that controlling for childhood behavioral problems would be just as effective: they included three control variables (two of which were dichotomous indicators) of childhood behavioral problems. Sampson and Laub offered very little justification for these three measures, and further, they offered no empirical evidence that controlling for childhood behavioral problems will control all genetic effects. Nonetheless, this approach is used in criminology (Burt, Simons, and Simons, 2006; Cernkovich and Giordano, 2001; Simons et al.,
Authors include a control for prior behavior and conclude, without any evidence to support their claim, that genetic effects have been removed from their statistical models (Sampson and Laub, 1993:88).

The problem, however, is that controlling for prior behavior does not mean that genetic effects have been controlled. Although it is true that stability in behavior is often influenced by genetic effects (Reiss et al., 2000), it is erroneous to think that genetic effects cannot also account for changes in behavior (Hoghughi and Forrest, 1986:252). Stated directly, genetic effects can be important for stability and changes in behavior over time (Collins, 2004). But how can genetic effects explain changes in behavior? One way to answer this question is to recall the earlier discussion of gene-environment interplay (see Section 2.3).

The underlying logic of gene-environment interplay is that genetic effects are sensitive to environmental influences. One form of gene-environment interplay shows that genetic effects are sometimes contingent on environmental factors (i.e., gene by environment interaction; GxE). For example, a person with a certain genotype may be predisposed to aggressive behavior. However, it may also be the case that these aggressive tendencies are stifled under certain environmental conditions. For this person, genetic effects can be important for explaining change in behavior over time as a result of the interaction of genetic and environmental factors. In short, GxE provide a framework for understanding how and why genetic effects may "pop up" at certain points in the life course, but may be absent at other times. The second type of gene-environment interplay explains how genetic factors can influence environmental exposure (i.e., gene by environment correlation; rGE). One type of rGE, active rGE, shows how a person's genotype can affect the types of environments they come into contact with. In this way, active rGE shows that people seek out environments that fit their genetic make-up. Musically inclined people join the band and athletic people play sports. If people seek out environments that allow them to express their genetic tendencies, it may be the case that genetic factors remain dormant until the environment allows for their expression. In this way, genetic factors may not be important early in life when parents provide their children with their environments. However, as the child ages into adulthood, certain genetic predispositions may begin to emerge as a result of self-selection into environments conducive to genetic expression.

There are other ways that genetic effects can change across the life course. Genes can be triggered on and off by biological or developmental processes (Reiss et al., 2000). For example,
puberty brings about many biological changes that are thought to trigger certain genetic effects on and others off (Wright, Tibbetts, and Daigle, 2008). Thus, gene-environment interplay is not the only way in which genetic effects can change over time.

There is a nascent body of research that has shown that genetic effects on certain phenotypes increase with age (Bergen et al., 2007). For example, behavioral geneticists have shown that heritability estimates for IQ tend to increase from adolescence to adulthood (Bergen et al., 2007). Similar findings have emerged for antisocial behavior (Lyons et al., 1995). Recall that several meta-analyses have examined the moderating influences of age on heritability estimates. Some of these studies have shown that heritability estimates are larger in older samples (Bergen et al., 2007; Miles and Carey, 1997; but see Mason and Frick, 1994 for null effects; see Rhee and Waldman, 2002 and Ferguson, 2010 for contradictory results). One explanation of this phenomenon may be found in the logic of active rGE. If people seek environments that are compatible with their genetic tendencies (i.e., niche-picking) then it seems logical that genetic factors will emerge or will become more prominent in environments that are conducive to their expression. In other words, people will be more likely to follow their own interests and to act in accordance with their genetic predispositions in environments that allow them to do so. Adulthood often affords the individual the freedom to choose their own environments (Agnew, 2003). As a result, the person will often choose environments that allow for the expression of their genetic predispositions.

But how does this relate to or explain changes in crime over the life-course? If anything, it would seem that the concept of active rGE would be more likely to explain stability in crime as opposed to changes in crime. Although it is likely that active rGEs do explain stability in crime, it may also be the case that active rGEs can elucidate the effect of adult social bonds on desistance from crime. To explain this hypothesis, it is necessary to recall the age-crime curve. The age-crime curve shows us that delinquency is normative in adolescence. In fact, some have hypothesized, and research has shown, that abstaining from delinquency is non-normative and may be indicative of underlying psychosocial problems (Boutwell and Beaver, 2008; Moffitt, 1993; Piquero et al., 2005). If we assume that adolescent delinquency is normative behavior, in that adolescent delinquency is less influenced by genetic factors than is adulthood crime, then we can begin to formulate an understanding of active rGEs and how they can influence changes in delinquency.
Imagine a person who is predisposed to obtaining a good job (i.e., the person is intelligent and is willing to work hard to achieve long-term goals). Imagine also that this same person engaged in delinquency during adolescence. Since adolescent delinquency is normative, this should not be hard to imagine. Finally, imagine that this person obtains a good job when he/she becomes an adult and that the delinquent behavior exhibited in adolescence ceases once adulthood is reached. Sampson and Laub (1993) would argue that the adult social bond (i.e., the good job) caused the desistance from delinquency. But, it is also likely that active rGE can explain the relationship between the bond and desistance.

For this hypothesized relationship to be a tenable explanation of the link between adult social bonds and desistance from crime, several points must be supported by research. First, active rGE must explain why certain people are exposed to adult social bonds and others are not. Sampson and Laub (1993; Laub and Sampson, 2003) argued that exposure to adult social bonds are sometimes the result of "random" or "fortuitous" events that cannot be explained by selection effects. What if, however, random chance had less to do with exposure to adult social bonds than Sampson and Laub lead us to believe? What then might explain selection into adult social bonds? Whether the answer to this question is "active rGEs" will be explored in this chapter. Second, genetic effects must be important for understanding changes in antisocial behavior. The findings bearing on this issue will be outlined in the second section of this chapter. Third, the explanatory power of the adult social bond on desistance must be reduced or eliminated once genetic effects are controlled. To date, there is no research that has addressed this final point. As a result, the last section of this chapter will offer several research questions that must be addressed.

4.1 GENETIC INFLUENCES ON LIFE-COURSE TRANSITIONS

The first question to be considered in this chapter is whether genetic effects explain selection into adult social bonds such as marriage, stable employment, and military service. Before moving to a discussion of the research examining the genetic influences on exposure to adult social bonds, however, it is important to review the concept of active rGE. Recall from Chapter 2 that active rGEs provide a framework to understand why adult social bonds may be influenced by genetic factors (Scarr, 1992, Scarr and McCartney, 1983). Specifically, active
rGEs highlight the importance of niche-picking, or self-selection into certain environments based on one's genotype. Individuals who are more intelligent—intelligence has been shown to be one of the most highly heritable traits—are more likely to stay in school for longer periods of time (e.g., college, graduate school, etc.). A person born with a talent for acting may be more likely to move to a city such as Los Angeles where their particular ability provides more opportunities for success. These are two simple examples of how a person's genotype may influence their environment. The same logic may hold true for exposure to adult social bonds. For example, it may be the case that people born with certain genotypes are more likely to marry, find stable employment, or join the military than people born with different genotypes.

4.1.1 Active rGE: Marriage

Scholars are beginning to explore the genetic underpinnings to adult social bonds. One adult social bond that has received attention from behavioral geneticists is marriage (Beaver, Wright, DeLisi, and Vaughn, 2008a; D'Onofrio et al., 2005; Dick et al., 2006; Johnson, McGue, Krueger, and Bouchard, 2004; McGue and Lykken, 1992; Spotts et al., 2004; Spotts et al., 2005; Trumbetta et al., 2007). Trumbetta et al. (2007) analyzed a twin sample of World War II veterans. Their analyses indicated that genetic factors were important for understanding the variance in marital status. Furthermore, the authors found that a single latent factor representing genetic influences accounted for a portion of the variance in marital status across six decades of life. Heritability coefficients ranged from .09 when respondents were in their 70s to .58 when respondents were in their 20s. In another study, Heath et al. (1989) found that heritability estimates for alcohol abuse were moderated by marital status among a sample of female twin pairs. Specifically, the heritability estimate for alcohol consumption was larger ($h^2 = .77$) for unmarried respondents than for married respondents ($h^2 = .59$). This study showed that heritable influences can be affected by marital status.

Johnson et al. (2004) examined whether certain personality characteristics increased the odds of marriage, whether genetic influences impacted decisions to marry, and whether genetic factors explained the link between personality characteristics and marriage. Using data taken from the Minnesota Twin Registry, the authors were able to analyze a large sample of twins ($N = 4,094$). To answer their first question—whether certain personality characteristics increased the
odds of marriage—the authors estimated a logistic regression model using marital status as the outcome and the various personality characteristics as predictor variables. Their results indicated that a number of personality characteristics predicted marital status. Interestingly, however, there was almost no overlap between the personality characteristics that predicted marriage for males with those that predicted marriage for females. To determine whether marriage was influenced by genetic propensities, the authors utilized their twin-based sample to estimate a heritability coefficient. The results from their model fitting analyses indicated that whether the respondent had ever been married was highly influenced by genetic factors. Specifically, heritable factors explained approximately 70 percent of the variance in marital status. Finally, to address their third research question, the authors decomposed the covariance between marital status and personality type. This analysis allowed the authors to estimate whether the correlation between personality type and marriage were due to shared genetic influences. The results from this analysis indicated that genetic factors accounted for the majority (83 percent for females and 61 percent for males) of the covariance between personality type and marital status.

Other studies have examined the impact of genetic and environmental influences on factors affecting marital quality. One of the hallmark studies in this line of research was published in 1992 by McGue and Lykken. In this study, the authors found that concordance rates of divorce were much higher for MZ twins than for DZ twins. Specifically, MZ twins had odds of divorce that were 6 times higher if their co-twin had been divorced in the past. For DZ twins, the odds were only 2 times higher if their co-twin had been divorced. In a more recent study, Spotts et al. (2005; see also Spotts et al., 2004) examined a sample of female twin pairs to determine whether genetic factors were important for predicting variation in marital quality. Their findings indicated that genetic factors predicted a significant proportion of the variance in marital satisfaction \( (h^2 = .35) \) and marital warmth \( (h^2 = .21) \) as reported on by the wife and the husband. It is interesting to note that the genetic factors were measured for the wife only. Thus, the findings from this study indicated that genetically influenced traits in the wife influence the way in which both the wife and the husband rate the quality of their relationship. Finally, D'Onofrio et al. (2005) found that genetic factors explained 15 percent of the variance in marital instability.

Some scholars have explored the genetic underpinnings to marriage by analyzing specific genes (Beaver, Wright, DeLisi, and Vaughn, 2008a; Dick et al., 2006). Beaver et al. (2008a)
examined five genes to determine whether they predicted marital status. The results from logistic regression analyses indicated that none of the five genes predicted marital status among respondents in the Add Health sample. Although the Beaver et al. were unable to identify the specific genes that predispose individuals to marriage, Dick and colleagues (2006) found that the GABRA2 genotype was associated with marriage. Individuals with the "high-risk" allele were less likely to be married (48 percent vs. 54 percent) and reported less marital stability (39 percent vs. 43 percent).

The findings from the above studies are extremely important to life-course criminology for two overarching points. First, selection into marriage is not simply a random or fortuitous event. Instead, there are genetic factors that are predictors of marital status. It is interesting to note, nonetheless, that the only other component found to influence marital status in some studies was the nonshared environment (e.g., Johnson et al., 2004). This is important because random or fortuitous events, if they are important, would fall into this category. Second, genetic factors likely influence variation in marital status through their effects on personality characteristics of the spouses. In other words, the genetic effects probably work through personality characteristics that make an individual more amenable to the long-term commitment intrinsic in a marriage.

4.1.2 Active rGE: Employment

Some scholars have assessed the genetic linkages to various facets of employment and occupational success (Arvey, Bouchard, Segal, and Abraham, 1989; Keller, Bouchard, Arvey, Segal, and Dawis, 1992; McCall, Cavanaugh, Arvey, and Taubman, 1997; Middeldorp, Cath, and Boomsma, 2006; Tambs, Sundet, Eaves, and Berg, 1992). Arvey et al. (1989) employed the monozygotic twins reared apart (MZA) method of generating heritability estimates. Their focus was on different aspects of employment. To measure these factors, the authors used the Minnesota Job Satisfaction Questionnaire (MSQ). The MSQ taps domains of employment that are both intrinsic (i.e., achievements) and extrinsic (e.g., quality of working conditions). Additionally, each respondent's occupation was rated by using a standardized job assessment scale. This scale tapped the level of complexity involved in each respondent's occupation. Since the MZA design controls for the shared environment, any similarities between siblings is
evidence of genetic effects and any differences tap the nonshared environment (and measurement error). Five of the 20 questions included in the MSQ showed significant heritability effects. Most of the significant items fell into the intrinsic scale, which also showed significant genetic effects. Genetic effects were not significant for the extrinsic scale. Perhaps most telling, however, is that the "general satisfaction" scale showed significant genetic effects. As for the occupational rating scale, 3 of the 4 ranking criteria evinced significant genetic effects. Specifically, the level of complexity of the job, the amount of motor skills needed to perform the job, and the physical demand of the job all showed significant genetic effects. Across all domains of job satisfaction and job ratings, when genetic effects were significant, they accounted for roughly one-third of the variance. Similar estimates were garnered from Tambs et al. (1992) and Middeldorp et al. (2006).

In a similar study, Keller and colleagues (1992) examined MZ and DZ twins reared apart to determine whether genetic influences affected work values. Work values were measured using the Minnesota Importance Questionnaire (MIQ), which taps the respondent's perception of how important things like creativity and moral values are to job success. The 20 items available in the MIQ loaded onto 6 factors tapping the value of achievement, comfort, status, altruism, safety, and autonomy. When model-fitting analyses were performed on these 6 scales, the results indicated that genetic effects were important for all of the outcomes. Heritability estimates ranged between .37 for altruism and .68 for achievement. In short, the authors argued that heritable traits account for about 40 percent of the variance in work values. Another study (McCall et al., 1997) examined the genetic influences on job stability. The results from model-fitting analyses indicated that job-switching (i.e., job instability) was significantly influenced by genetic factors. Approximately 30 percent of the variance in job instability was explained by genetic influences.

Unfortunately, only a small body of research has examined the genetic influences on employment outcomes. There is only a limited amount of evidence, therefore, that can be related to the claims made by Sampson and Laub (1993). Still, the extant research indicates that some of the components of employment that are of interest to Sampson and Laub's theory (e.g., job stability) are influenced by genetic factors.

4.1.3 Active rGE: Military
Finally, there is one study that analyzed the impact of genetic and environmental influence on military involvement (Lyons et al., 1993). Lyons and colleagues examined correlations between MZ and DZ twins regarding decisions to volunteer for service in Vietnam, for actually serving during the War efforts, and for combat experience. The correlations for volunteering for service were .40 for MZ twins and .22 for DZ twins, suggesting substantial (approximately 40 percent) genetic effects. When the researchers examined whether the respondent had actually served in the war, the MZ correlations were .41 and the DZ correlations were .24. As for combat experience, the correlations for MZ twins were .53 and .30 for DZ twins. In all, this study presents evidence that military exposure is partially driven by genetic predispositions of the individual, at least when it comes to volunteering for service, actually serving in the military, and for experiencing combat.

4.1.4 Discussion

As the above review has displayed, many human experiences that have previously been believed to be "purely social" in origin and in their effect are partially influenced by genetic factors. Indeed, research has shown that nearly all environments that have been studied are the product of genetic effects. In their review of the gene-environment correlation literature, Kendler and Baker (2007) analyzed 55 studies to determine the range of heritability effects across different environments that are thought to be important for psychiatric and drug use disorders (i.e., antisocial behavior). The authors divided the sample of studies into eight different sections to reduce heterogeneity. The different sections identified a different environment such as stressful life events and the family environment. Of particular interest to the current review are the sections labeled "stressful life events" and "marital quality." Stressful life events captured things such as traumatic experiences and experiencing a divorce. For this section, heritability estimates ranged from .07 for selection into non-assaultive trauma and .39 for experiencing negative life events. The average heritability estimate for the two studies examining divorce was .35. The second section of interest from Kendler and Baker's review analyzed marital quality. For this section, three separate studies were analyzed. Within these three studies, several domains germane to marital quality were measured: marital satisfaction,
conflict, and warmth. Heritability estimates for these three domains were .28, .13, .17, respectively. In summarizing their results, Kendler and Baker (2007:620) stated:

The literature we have reviewed suggests that genetic influences on measures of the environment are pervasive in extent and modest to moderate in impact. Every aspect of the environment that we were able to examine was significantly influenced by genetic factors. However, the role of genetic influences on these behaviors was far from overwhelming…with most falling between 15% and 35%.

Genetic factors are important for understanding variation in exposure to adult social bonds. Perhaps not surprisingly, however, the environment is responsible for the majority of the explained variance. The most important question to consider now is whether genetic factors contribute to changes in antisocial behavior over time.

4.2 GENETIC INFLUENCES ON CHANGES IN ANTISOCIAL BEHAVIOR OVER TIME

There is a small body of literature that has examined the impact of genetic effects on changes in antisocial behavior over time. Although behavioral genetic research has been around for decades, some of the more complex issues such as whether genes can affect changes in behavior have only recently been explored by scientists. As a result, there is a more limited knowledge base regarding this topic. One of the most comprehensive studies to examine this topic was presented by Reiss and his colleagues (2000) in their book titled *The Relationship Code: Deciphering Genetic and Social Influences on Adolescent Development*. Reiss and his colleagues collected and analyzed an impressive dataset that included sibling pairs from 720 families. The sibling pairs ranged from monozygotic (MZ) twins who share 100 percent of their DNA to unrelated/step siblings which share none of their DNA. Importantly, Reiss et al. interviewed each of the respondents at two separate points in time. At time 1, all respondents were in their adolescent years (mean age was 13.5 years for the oldest sibling) and a series of questions tapping their psychosocial development were administered. The second round of interviews (i.e., time 2) occurred approximately 3 years later. Thus, most of the children were in the later stages of adolescent development (mean age was 16.2 years for the oldest sibling). As a
result of the sampling design, many of the questions that were asked at time 1 were asked again at time 2. This allowed the researchers to assess changes in development from time 1 to time 2.

Although their analysis considered a number of domains important for adolescent development, most important for the current discussion is that they included measures tapping antisocial behavior at both time 1 and time 2. In order to measure antisocial behavior, the authors utilized multiple raters and a number of standardized questionnaires. For example, antisocial behavior was measured by asking the respondent to self-report their behavior, by asking the mother to report on the child's behavior, by asking the father to report on the child's behavior, and by having independent raters observe the child's behavior. By employing multiple raters, the authors were able to limit the effects of shared methods bias. As for the standardized questionnaires, the authors assessed antisocial behavior by employing a number of well established (i.e., psychometrically sound) questionnaires such as the Behavior Problems Index (Zill, 1985).

In order to determine whether genetic factors affect changes in antisocial behavior from time 1 to time 2, the authors employed the logic and statistical tools offered by quantitative behavioral genetics. A description of the statistical method used by Reiss et al. (2000:223) will be offered later. For now, it is only important to understand their findings and what they mean for life-course criminology. The findings can be neatly summarized by four points. First, genetic factors were important for explaining the variance in antisocial behavior at time 1 and separately at time 2. Second, genetic effects were the most important factor for explaining stability in antisocial behavior from time 1 to time 2. In fact, 69 percent of the stability in antisocial behavior was due to genetic factors that influenced behavior at both time points. Third, genetic factors were the most important influence on changes in antisocial behavior from time 1 to time 2. Approximately 60 percent of the changes in antisocial behavior were the result genetic influences. Thus, it is clear that genetic factors can affect changes in antisocial behavior over time. Fourth, environmental factors were important for changes in antisocial behavior. The environmental effects, however, were restricted to nonshared environments. In other words, the shared environment did not explain changes in antisocial behavior.

The findings from Reiss et al. (2000) are an important first step in understanding the influences on behavioral stability. Even more important for the current analysis is that Reiss et al. found that genetic and nonshared environmental influences affect behavioral change. One of
the more interesting findings is that genetic influences can change over time. Indeed, this finding runs counter to most criminologists' understanding of genetic factors. However, the possibility that genetic effects can change over relatively short periods of time is not a new concept for behavioral geneticists. In fact, some research has shown that changes in genetic effects can be observed in as little as six months (Plomin et al., 1993). Unfortunately, exactly which genetic factors and which nonshared environments are affecting change remains unknown. Perhaps the emergent properties of adult social bonds constitute a portion of the nonshared environmental influence on change. This possibility may be true to the extent that adult social bonds actually are random or fortuitous events as Laub and Sampson (2003) maintain. Remember, however, that this possibility would not be borne out in Reiss et al.'s study due to the age range of their participants (all respondents were adolescents at time 1 and time 2), but may hold true for other studies using older samples.

In an earlier study, Lyons et al. (1995) analyzed data drawn from male twin pairs that served in the military during Vietnam (May 1965 through August 1975). As a result of the all male sample, their analysis was restricted to MZ and same-sex DZ twin pairs. Unlike the Reiss et al. (2000) study, Lyons and colleagues analyzed a sample of twin pairs that were much older (mean age 44.6 years and age range 36 to 55 years). The primary variable of interest was antisocial personality disorder (APD) as defined by the DSM-III. A large sample size was achieved (\(N = 3,226\) twin pairs) and all subjects were interviewed by telephone. Their results revealed that genetic and nonshared environmental factors were important for explaining the variance in individual symptoms associated with an APD diagnosis. When the individual symptoms were combined into a single APD scale, the findings revealed that genetic factors were less important \((h^2 = .07)\) and that the shared environment \((c^2 = .31)\) was more important for understanding juvenile APD. When the analysis was extended to adulthood APD, however, the findings reversed; heritability \((h^2 = .43)\) was more important and the shared environment was less important \((c^2 = .05)\).

Like Reiss et al. (2000), Lyons and his colleagues (1995) estimated the effect of genetic and environmental factors on stability and change in APD. A distinguishing factor is that Lyons et al.'s analysis extended well into adulthood. Lyons et al. found that that genetic factors, shared environmental factors, and nonshared environmental factors were all important for explaining stability in APD from adolescence to adulthood. Interestingly, and in contrast to Reiss et al.
(2000), Lyons and his colleagues found that changes in APD from adolescence to adulthood were not influenced by genetic factors. Instead, changes in APD were only attributable to nonshared environmental influences. As mentioned earlier, perhaps this estimate captures the effect of adult social bonds.

There is also some evidence that genetic factors are important for understanding changes in personality traits that are known correlates of crime and delinquency. Beaver, Wright, DeLisi, and Vaughn (2008b) explored the genetic and environmental effects on stability and change in levels of self-control across two waves of data collection. Analyzing twin data drawn from the National Longitudinal Study of Adolescent Health (Add Health), the authors constructed measures of self-control at wave 1 and at wave 2. The authors noted that genetic factors were the predominant influence on differences in levels of self-control at both wave 1 and wave 2. However, their results showed that self-control was not perfectly stable across the two waves ($r = .64$). To determine the factors that accounted for stability and change, the authors employed a behavioral genetic model that could separate out these unique effects. Their findings were quite revealing. First, the majority of the stability in self-control from wave 1 to wave 2 was explained by stable genetic factors (82 percent). The remaining portion of the stability in self-control (18 percent) was explained by nonshared environmental influences. As for changes in self-control from wave 1 to wave 2, the findings revealed that genetic factors were an important factor. Specifically, 20 percent of the variance in changes in self-control was due to unique genetic effects operating only at wave 2. The remaining variance was explained by nonshared environments that were unique to wave 2 (80 percent).

Evidence regarding the effect of genetic factors on changes in antisocial behavior can also be drawn from another study by Beaver, Wright, DeLisi, and Vaughn (2008a). This study estimated the effect of measured genetic factors on desistance from crime using the Add Health data. Statistical models indicated that four of the five genes analyzed predicted desistance from crime. Moreover, these genes interacted with marriage to affect desistance; the genes were more influential in predicting desistance for respondents who were also married. Interestingly, however, these effects were only apparent for males. When females were studied, the genetic factors were unimportant for explaining desistance. These findings suggest that males and females may follow different pathways toward desistance from antisocial behavior (see also Uggen and Kruttschnitt, 1998).
4.3 SUMMARY AND RESEARCH QUESTIONS

We have now arrived at a point where the research available to answer our questions ceases to exist. Thus, the remainder of this chapter will be devoted to briefly summarizing the evidence that was presented above and to identifying the research questions that will guide the analysis that will be presented later.

If we accept the argument that biosocial criminology can be seen as a critique of extant theories, we can start to build a framework for understanding the factors that influence desistance across the life course. Sampson and Laub (1993) proffered a sociological theory which argued for the importance of adult social bonds on desistance in adulthood. However, results from behavioral genetic studies imply that this argument may not be tenable. First, Sampson and Laub argued that exposure to adult social bonds was partially the effect of random events. Although they acknowledged the importance of selection effects, they argued that these factors would not be sufficient to account for the effect of the bonds on desistance. On the other hand, behavioral genetic research has found that approximately one-third of the variance in adult social bonds can be explained by genetic factors. Consider the following statement by Johnson et al. (2004:285):

Most analysts have not credited self-selection [into marriage] with a significant role…It seems possible at least that attraction of a suitable partner, propensity to establish a relationship intended to be permanent, and maintenance of that relationship may have related etiologies and that these etiologies may have their roots in personality. We thus propose the possibility that there are common genetic influences on personality and propensity to marry.

The point Johnson et al. are trying to make is that self-selection into adult social bonds—especially marriage—is likely to be influenced by many factors, not the least of which are genetic factors.

Second, Sampson and Laub are ardent supporters of the effects of state dependence on behavior. This model argues that past crime is correlated with future crime through the "knifing off" of opportunities over time. In other words, a "pure" state dependence theory argues that individual traits, such as genetic factors, do not explain the continuity in crime over time.
Results from behavioral genetic research shows that this is not the case. Instead, genetic factors are extremely important in explaining stability in offending over time. By some accounts, 70 percent of the stability in antisocial behavior can be attributable to genetic factors (Reiss et al., 2000).

Third, Sampson and Laub argued that stability is common, but change does occur. Changes in antisocial behavior were completely attributed to the inhibitory effects of adult social bonds. In fact, the undertone to Sampson and Laub's theory is that adult social bonds are the only factors that "make sense." However, behavioral genetic research has shown that changes in antisocial behavior are affected by genetic influences and nonshared environmental influences.

The above reviews have provided a large amount of information, but two points bear repeating. First, evidence suggests that genetic factors influence exposure to marriage, employment, and the military (i.e., adult social bonds). Second, research also suggests genetic factors influence change in antisocial behavior. What remains unknown, however, is whether the genetic factors that influence changes in antisocial behavior over time are the same genetic factors that influence a person's experiences with adult social bonds. It is at this point that we reach the "end of the line" in available research. As a result, attempting to fill this void in the literature will represent the primary point of departure for this study. Specifically, the focus of the remainder of this dissertation will be toward examining the overlap in genetic effects between desistance and adult social bonds. Toward this end, several research questions will guide the analyses that follow:

(1) Do genetic factors influence exposure to adult social bonds among a nationally representative sample of siblings?
(2) Are genetic factors important for explaining changes in delinquency, drug use, and antisocial behavior over time?
(3) Are genetic factors important for explaining desistance from delinquency, drug use, and antisocial behavior?
(4) Do adult social bonds explain desistance from delinquency, drug use, and antisocial behavior?
(5) Do genetic factors reduce or eliminate the relationship between adult social bonds and desistance?
Extant research has examined research questions 1 – 4. This and the previous chapter have extensively reviewed these bodies of evidence. However, as was pointed out in covering these bodies of literature, there are many questions that have not been directly or adequately addressed. For instance, much of the criminological research on the effect of adult social bonds on desistance has not actually measured desistance. Instead, changes in crime are more frequently measured. Although desistance can be understood as a "slowing down" in crime, the extent to which changes in crime tap desistance is unknown. As such, it is important to lay the foundation for this analysis by re-examining some "old" questions with a lens toward providing clearer answers. Additionally, it is always important to establish the replicability of extant findings with new data and new analytic techniques. Comparing and contrasting the findings from the current analysis with the results from the extant literature bearing on those topics will help bolster the accuracy of the findings. Research question 5 represents a novel line of inquiry. To date, there are no studies that examine the effects of adult social bonds on desistance after shared genetic factors have been removed.
CHAPTER 5

METHODS

The preceding chapters have outlined the biosocial criminology perspective as well as findings from one of the most prominent life-course theories; Sampson and Laub's (1993) age-graded theory of social control. Chapter 4 set forth 5 research questions that will motivate the analysis for this dissertation. The current chapter will provide a detailed overview of the sample that will be analyzed to address each of the research questions. Then, attention will be given to the measures that are available for the current analysis. Finally, this chapter will provide an overview of the statistical methods that will be employed.

5.1 THE DATA

Data for this study came from the National Longitudinal Study for Adolescent Health (Add Health; Udry, 1998). The Add Health is a longitudinal and nationally representative sample of adolescents enrolled in grades 7 through 12. The general focus of the Add Health study was to assess the health and development of American adolescents. In order to do so, three waves of data were collected from approximately 20,000 respondents. To gain information from these adolescents, a sample of high schools was first selected by employing stratified random sampling techniques. In short, high schools acted as the primary sampling unit. A school was classified as a high school if it included an 11th grade and enrolled a minimum of 31 students. Initially, 80 eligible high schools were included in the sample frame. Using this list, schools were stratified by region, urbanicity, school type, ethnic mix, and size. After stratification, schools were selected with a probability proportional to their size. Initially, more than 70
percent of the 80 schools were recruited for inclusion in the study. In order to participate, the school was required to provide a roster of all students and was required to (in most cases) agree to administer the in-school questionnaire during one class period. Not all schools, however, agreed to participate. When a school refused to participate, a replacement school was selected within the stratum that the refusing school had filled. When a school was successfully recruited, information from students in "feeder" schools was also gathered. A feeder school was defined as a school that included 7th grade and provided graduates to the high school of focus. One feeder school for each high school was selected with the probability of selection being proportional to the number of students that it sent to the high school. In some cases, a school included all grades from 7 to 12. In these rare instances, the primary target school acted as its own feeder school. The above described sampling procedure ultimately resulted in 132 schools being included in the study (see Harris et al., 2009 or Kelly and Peterson, 1997 for an overview of the Add Health data and the research design).

Students attending these 132 schools were asked to complete a self-report questionnaire during one class period. This round of data collection began in September 1994 and continued through April 1995. Each school administered the questionnaire during one class period on a designated day. No make-up day was offered to students that were absent on the day of administration. Parents were given prior notification of the survey and the date it would be administered. Participation was strictly voluntary and parents were allowed to advise their children to not participate. The net result of the in-school questionnaire was that more than 90,000 students across the 132 schools provided information.

Starting in April 1995 and continuing through December 1995, the Add Health research team collected more detailed information from a subsample of the students that completed the in-school surveys. Not all 90,000 students that completed in-school surveys also completed the follow-up interview (i.e., the in-home interview). Instead, students listed on each school's roster provided a sample frame from which respondents were chosen to complete an additional survey (i.e., wave 1). All students within a school were stratified by grade and sex and roughly 17 students within each stratum were chosen for in-home interviews. This process led to an average of 200 students being selected from each of the 132 schools for a total "core" sample of 12,105 respondents. In addition to this core sample, several "special" populations were oversampled. For example, based on information gleaned from the in-school component, certain respondents
were chosen to be interviewed in their homes at a higher rate. These special populations included four minority ethnic groups (Blacks from well educated families, Chinese students, Cuban students, and Puerto Rican students), disabled students, and sibling pairs residing in the same household. Overall, these sampling procedures netted wave 1 in-home interviews from 20,745 adolescents. In addition to interviewing the student, each student's primary caregiver was eligible to be interviewed. Information from 17,700 primary caregivers (typically the mother) was included in the wave 1 in-home component of the study. The response rate for the wave 1 in-home surveys was 80 percent (Harris, Halpern, Smolen, and Haberstick, 2006). Respondents ranged between 11 and 21 years of age at wave 1.

The wave 1 in-home surveys, which lasted approximately 90 minutes each, were designed to gain more detailed information about the adolescent, his/her experiences, and his/her rearing environment. For example, information was gathered about the respondent’s personality traits, about their social relationships, and about their behaviors. The surveys were conducted using laptop computers (i.e., computer assisted interviews). For most questions, the interviewer read the question aloud to the respondent who provided verbal responses. This procedure, however, may lead to biased responses on sensitive topics such as sexual experiences. In order to mitigate this possibility, audio computer assisted interviewing procedures were used when sensitive topics became the focus. In these cases, the respondent was provided earphones and questions were read aloud by the computer. The respondent then entered his/her responses directly into the computer.

Approximately one year after the wave 1 in-home interviews were completed (beginning in April 1996 and continuing through August 1996), 14,738 respondents completed a second round of in-home interviews (i.e., wave 2). Respondents who were a senior in high school during wave 1 interviews and those who were oversampled during wave 1 as part of the disabled students sample were not reinterviewed at wave 2. This omission explains a large portion of the apparent attrition from wave 1 to wave 2. It should also be noted that several respondents (n = 29) were interviewed at wave 2 who were not interviewed at wave 1. Since only a small amount of time elapsed between wave 1 and wave 2, and since most of the respondents were still adolescents, the questionnaires remained very similar. However, some questions were added to the wave 2 interviews that did not appear at wave 1. For example, questions referencing sun exposure and nutrition information were added to wave 2 surveys. Questions regarding
background information that is invariant over time were removed from wave 2 interviews (e.g., ethnicity). The response rate for wave 2 surveys was 90 percent (Harris, et al., 2006). Respondents ranged between 11 and 23 years of age at wave 2.

Between 2001 and 2002, approximately six years after wave 1 interviews were conducted, a third round of interviews took place with 15,197 respondents (i.e. wave 3). During this data collection period, most of the respondents were young adults, not adolescents. As a result, the surveys were changed to include more age-appropriate questions. Respondents, for instance, were asked about their employment history, their marital status, and their involvement in criminal behaviors. All respondents from wave 1 were eligible for wave 3 interviews. Efforts were made to locate all respondents and administer interviews. For example, interviews were sometimes conducted in jails if the respondent was incarcerated. As a result, the response rate was 77 percent (Harris, et al., 2006). Respondents ranged between 18 and 27 years of age at wave 3. As will be discussed momentarily, information concerning exposure to adult social bonds will be drawn from wave 3 data. Since the respondents were young adults, the observed variation for many of these adult social bond variables may be truncated.

Nested within the Add Health data is a subsample of sibling pairs residing in the same household. During wave 1 in-home interviews, respondents with an identical twin, a fraternal twin, a half-sibling, or a step-sibling were selected with certainty. In other words, if a respondent identified themselves as a twin, a half-sibling, or a step-sibling during wave 1 interviews, their sibling was automatically added to the sample. A subsample of non-related pairs living in the same household was also included at wave 1. These pairs were not selected with certainty. In all, information from more than 3,000 sibling pairs was included in the genetic subsample (Rowe and Jacobson, 1998).

Twin zygosity information was obtained in a number of steps. First, all mixed-sex twin pairs were classified as DZ twins. Second, same-sex twin zygosity was determined on the basis of self-reported confusability of appearance. Twins were asked to report, for example, whether they were ever confused for one another by strangers, teachers, and family members. This, however, led to a small group of twins with uncertain zygosity. A portion of this group (n = 89) of twins with uncertain zygosity was assessed on the basis of seven molecular genetic markers. If the twins matched on at least five markers, they were classified as MZ twins. The error rate for this type of classification scheme is approximately 4/1000. Since not all twins with uncertain
zygosity were exposed to genetic testing, a small number of twins were assigned a "missing" value for zygosity. The pairs subsample was designated as a "special population." For this reason, greater efforts were made to ensure that all respondents were located and contacted at waves 2 and 3. As a result, higher response rates were realized for the pairs subsample than for the full sample; response rate was 95 percent at wave 2 and 89 percent at wave 3 (Harris et al., 2006).

The sibling pairs subsample was compiled in a fashion that allowed more than one pair of siblings to be included per household. This meant that more than two siblings were sometimes interviewed. In order to remove any possible biases, the current study restricted the sample to two children per household. This process of excluding certain siblings from the sample was carried out using a semi-structured selection method. All MZ and DZ twins were selected with certainty. In other words, if a household had three respondents and two of them were DZ twins, the DZ twins were automatically included and the third sibling was removed. Full siblings, half siblings, and cousins were chosen at a rate that would ensure sample sizes large enough to perform statistical analyses. Non-related pairs living in the same household were only included if they were the only sibling pair interviewed from that household. In line with prior researchers using the Add Health data, however, all non-related pairs were removed from the sample prior to the estimation of statistical analyses (Cleveland, 2003). The non-related pairs were removed because selection effects were unable to be ruled out for many of the pairings. For example, some of the non-related pairs were living together due to assortative mating processes (e.g., step siblings and spouses). In these cases, the genetic relatedness between the two individuals can no longer be assumed to be zero—at least to the extent that genetic selection has occurred (which is an unknown parameter). The result of this self-selection is that inclusion of these pairs may lead to biased parameter estimates. There was one set of DZ triplets. One of the three DZ triplets was randomly excluded from the sample. Recall also that a small number of respondents were added at wave 2. Given the longitudinal focus of the current research, these cases were excluded from the analysis. In other words, the current study analyzed respondents that provided information during wave 1 interviews.

The most frequently used approach to behavioral genetic research methods is the twin methodology. This method compares MZ twins with DZ twins to determine whether MZ twins resemble each other more than DZ twins. The limitations of this approach, however, have
already been discussed and will not be readdressed here. Recall that one approach to overcoming some of the limitations of the twin-based methodology is to perform a family-based study. In other words, one way to overcome the pitfalls of twin-based designs is to include more than just MZ and DZ sibling pairs. Since the Add Health data included information on MZ twins, DZ twins, full siblings, half siblings, cousins, and unrelated pairs living in the same household, a family-based study is possible. Table 5.1.1 presents a breakdown of the sample that will be used for the current study. Information for individuals and sibling pairs is included. Information is presented in this fashion because the sibling data were double entered (i.e., sibling 1 appears in the dataset twice; once as the target sibling and a second time as the reference sibling). This process has become the convention in behavioral genetic analyses because it removes artifactual variance that may arise due to arbitrary ordering of one sibling as "sibling 1" and another as "sibling 2" (Johnson et al., 2004). As a result, double entry is employed in this study and robust standard errors are used to account for clustering. After the removal of non-related pairs (n = 620 individuals), respondents added to the sample at wave 2 (n = 27 individuals), and twins with uncertain zygosity (n = 39 individuals), 4,568 individuals (2,284 sibling pairs) remained available in the sibling pairs dataset. The majority of the siblings fell into the DZ twin/Full-sibling category. The remaining pairs, in order of their contribution to the sample, were categorized as half-siblings, MZ twins, and cousins.

5.2 MEASURES

5.2.1 Adult Social Bonds: Relationships

A growing body of research has developed where the influence of marriage is connected to desistance processes (e.g., Blokland and Nieuwbeerta, 2005; Laub et al., 1998; Maume et al., 2005). As noted in previous chapters, Sampson and Laub (1993) afforded much attention to the effects of marital bonds on desistance from crime. Recall, however, that they argued that marriage alone was not sufficient to explain desistance. Instead, it is important to determine the person's level of attachment to the marriage. The Add Health data provide a measure of each respondent's marital status at wave 3. Unfortunately, direct measures of marital attachment are
not available. There are, however, several measures that tap the respondent's perception of the importance of love and commitment to a long-term relationship.

**Ever Married.** During the third wave of data collection, each respondent was asked to report the number of times they had been married. Since respondents were young adults during wave 3 interviews, very few had been married more than once. Specifically, within the full sample—not the sibling subsample to be used here—fewer than 100 respondents reported more than one marriage. As a result, there was little basis for maintaining the original coding of the variable. Instead, all responses were collapsed into a dichotomous variable with 1 indicating that the respondent had ever been married and a 0 indicating that the respondent had never been married. Nearly 700 respondents reported having ever been married ($n = 684$). Descriptive statistics for this and all of the other variables that will be used in the statistical analyses can be found in Table 5.2.1.1.

**Marital Love.** At wave 3, respondents were asked to indicate how important love is to having a successful marriage or a serious committed relationship. Respondents were asked to respond using a scale ranging from 1 to 10 with 1 meaning that love is not important and 10 meaning that love is extremely important.

**Marital Commitment.** Respondents were also asked to indicate how important making a life-long commitment was to having a successful marriage or a serious committed relationship during wave 3 interviews. Again, responses were coded on a scale of 1 to 10 with 1 indicating that commitment is not important and 10 indicating that commitment is extremely important.

**Composite Marital Scale.** Principal component analysis was carried out on all three of the variables tapping the various domains of relationship status (Ever Married, Marital Love, and Marital Commitment). The results indicated that a single component explained the associations among the three variables. However, the Married Ever variable had a much weaker relationship with the extracted component. Excluding the Married Ever variable also resulted in an increase in the reliability coefficient. As a result, the Married Ever variable was omitted and the Love Scale variable was created by standardizing the values for the Love Importance variable and the
Commitment Importance variable and summing the resulting values ($\alpha = .60$). Higher values indicated that the respondent felt that love and commitment were more important to a long term relationship.

5.2.2 Adult Social Bonds: Military and Employment

Although it has received less attention from scholars, Sampson and Laub (1993) argued that involvement in the military is an important turning point for some offenders. Thus, the current study included a measure of each respondent's involvement with the military. Additionally, Sampson and Laub, as well as a growing body of research, reported that education and employment bonds are important for predicting desistance. For this reason, several indicators of each respondent's involvement and commitment to education and employment were measured.

Military Involvement. At wave 3, all respondents were asked to indicate whether they had ever served time in the military reserves. Responses were coded dichotomously with 1 indicating that the respondent had been in the military and a 0 indicating that the respondent had not been in the military. A relatively small number of respondents in the sibling data had spent time in the military ($n = 97$). The limited variation on this variable may have implications for the analyses performed later.

Employment Status. Respondents were asked to indicate whether they were currently employed at a job that required at least 10 hours of work per week during wave 3 interviews. Responses were coded such that 1 ($n = 2,554$) indicated the respondent was employed at wave 3 and 0 indicated that the respondent was not employed.

Job Stability. All respondents who indicated they were employed during wave 3 interviews were asked several follow-up questions. The first follow-up question asked the respondent to indicate whether they were working at the first paying job they had ever held. This item was coded dichotomously with a 1 ($n = 300$) indicating that the respondent was working at their first job and a 0 indicating that the respondent was no longer working at their first job.
measure, therefore, taps job stability. All respondents with a 1 on this item have maintained a stable employment with their first employer and respondents with a 0 have switched jobs at least once. Respondents that reported no job on the initial employment question were coded as missing.

**Two Jobs.** All respondents who indicated they were employed during wave 3 interviews were asked a second follow-up question that indexed the number of jobs at which they were currently working. A number of respondents indicated working at two jobs \(n = 1,143\) for the full sample and far fewer reported working at 3 or more jobs \(n = 129\) for the full sample. Due to lack of variation, all response categories were collapsed into a dichotomous variable. For this new variable, a 1 indicated that the respondent was working at 2 or more jobs \(n = 304\). A 0 indicated that the respondent was only working 1 job. It should be noted that small number of respondents reported 0 jobs on this follow-up question \(n = 4\). These respondents were assigned a 0 for this variable since they had indicating working at a job on the initial employment "feeder" question. All respondents who reported no job on the initial employment question were assigned a 0.

**Hours Worked.** A third follow-up question was presented to all respondents that reported being employed during wave 3 interviews. Specifically, each respondent was asked to indicate the number of hours a week that they usually work at their primary job. Primary job was defined as the job at which they put in the most hours each week. Responses were coded in whole numbers and indicated the number of hours worked each week. Responses ranged from 0 to 90 with higher values reflecting more hours worked. Respondents that reported no job on the initial employment question were assigned a 0.

**Job Satisfaction.** Respondents who indicated being employed during wave 3 interviews were asked a fourth follow-up question that tapped their level of satisfaction with their job. Again, respondents were asked to reference their primary job when answering this question. Respondents were allowed to answer using a Likert-type scaling system where 1 represented "extremely satisfied" and 5 represented "extremely dissatisfied." For the analysis, this variable
was reverse coded so that higher values indicated more job satisfaction. Respondents who reported no job on the initial employment question were coded as missing.

**Annual Income.** At wave 3, all respondents were asked to indicate their personal income, before taxes, for the previous year. Respondents were asked to include all sources of income such as government assistance, income from employment, and child support. The values for this variable are reported in their raw numbers. Therefore, higher values indicated a larger income in the previous year. The mean value for the current sample was 13,200.63, indicating that the average income was just over 13,000 dollars.

**Education Level.** During wave 3 interviews, respondents were asked to indicate the highest grade or year of school they had completed. As a result of the question wording, responses were coded as years of schooling completed. Responses ranged from 6 to 22 years of schooling completed.\(^{12}\)

**Composite Employment Scale.** A principal component analysis was conducted on five variables tapping the various domains of employment. Specifically, Income Last Year, Job Stability, Work Two Jobs, Hours Work, and Job Satisfaction were analyzed to determine whether they could be combined into a single scale. The results from the principal component analysis failed to identify a single interpretable component to which all five variables contributed. However, Income Last Year and Hours Work loaded onto a single component that explained 27 percent of the variance. As a result, these two variables were standardized and summed into the Work Scale (\(\alpha = .50\)). Higher values indicated a greater degree of involvement in employment activities.

### 5.2.3 Adult Social Bonds: Community Service and Religion

Sampson and Laub (1993) did not focus attention on a person's involvement in the community and their level of religiosity. However, consistent with Hirschi (1969), it may be

\(^{12}\) Since some respondents were as old as 28 years at wave 3, it is not impossible for a respondent to have reported 22 years of education.
important to tap a person's level of attachment to the community in order to understand desistance. Additionally, some researchers have highlighted the importance of religion for desistance from crime (e.g., Chu, 2007; Giordano et al., 2008). This analysis included measures of community involvement and religiosity.

**Community Service.** Respondents were asked whether they had performed any unpaid volunteer or community service work within the past 12 months during wave 3 interviews. Responses were coded such that 1 indicated that the respondent had performed volunteer/community service work \( (n = 1,113) \) and 0 indicated no such work.

**Church Attendance.** During wave 3 interviews, respondents were asked a series of questions that tapped religious involvement. The first question referred to the number of times the respondent had attended church or religious services in the past 12 months. Response categories were "a few times" (coded as 1), "several times" (coded as 2), "once a month" (coded as 3), "2 or 3 times a month" (coded as 4), "once a week" (coded as 5), and "more than once a week" (coded as 6). Higher values indicated greater attendance at church/religious services in the last year.

**Importance of Religion.** A second question concerning religious involvement was asked to each respondent during wave 3 interviews. This question asked respondents to indicate how important religious faith was to them. Responses were coded on a scale ranging from 0 to 3. Higher values reflected more religious importance for the respondent.

**Religiosity.** Respondents were asked to identify the extent to which they were a religious person during wave 3 interviews. Responses ranged from 0 to 3 with higher values indicating that the respondent was more religious.

**Composite Religion Scale.** A principal component analysis was carried out on the Community Service variable and the three religion variables (Attend Church Last Year, Religious Importance, and How Religious). The results from this analysis indicated that the three religion variables loaded onto a single component. The Community Service variable also
When reliability analyses were carried out, it was evident that including this variable with the religion variables reduced the alpha coefficient. As a result, only the three religion variables were standardized and summed into a single Religion Scale ($\alpha = .83$). Higher values indicated that the respondent is either more involved in or places a greater importance on religion.

### 5.2.4 Delinquency, Drug Use, and Antisocial Behavior

Sampson and Laub's (1993) theory focuses almost exclusively on crime and delinquency. It may be the case, however, that adult social bonds are important predictors of drug use and more global manifestations of antisocial behavior as well. In order to incorporate these possibilities, the current analysis included measures of delinquency, drug use, and antisocial behavior.

**Delinquency at Wave 1.** Respondents were asked a host of questions during wave 1 interviews that referenced delinquent behavior. Each respondent was asked to indicate whether and how often they had been involved in 17 different delinquent activities within the last year. Specifically, respondents were asked whether they had painted graffiti, damaged property, lied to their parents, stolen from a store, gotten into a serious fight, hurt someone badly enough to require medical attention, run away from home, stolen a car, stolen something worth more than $50, broken into a house, committed an armed robbery, sold drugs, stolen something worth less than $50, taken part in a group fight, acted loud or unruly in a public place, carried a weapon to school, and used a weapon in a fight. Responses to most of the questions were coded as follows: 0 (i.e., "never"), 1 (i.e., "1 or 2 times"), 2 (i.e., "3 or 4 times"), and 3 (i.e., "5 or more times"). Two questions (i.e., carried a weapon to school and used a weapon in a fight) were coded dichotomously where 1 indicated that the event had occurred and a 0 indicated that the event had not occurred. Principal component analysis indicated that a single latent construct best explained the 17 items. With one exception, the reliability coefficient was reduced if any of the items were removed from the scale. The one exception was for the "lying to parents" item. Dropping this variable from the scale raised the alpha coefficient by a negligible amount (.0002). As such, a
single scale was created by summing the responses to all 17 items ($\alpha = .85$). Higher values indicated more involvement in delinquency.

**Delinquency at Wave 2.** Delinquent behaviors were also measured at wave 2. Since most respondents were still adolescents during wave 2 interviews, all of the behaviors in question remained the same. Respondents were again asked to indicate whether and how often they had painted graffiti, damaged property, lied to their parents, stolen from a store, gotten into a serious fight, hurt someone badly enough to require medical attention, run away from home, stolen a car, stolen something worth more than $50, broken into a house, committed an armed robbery, sold drugs, stolen something worth less than $50, taken part in a group fight, acted loud or unruly in a public place, carried a weapon to school, and used a weapon in a fight. Respondents were asked to report the frequency of these behaviors over the past 12 months and the questions were coded on a scale ranging from 0 to 3 (0 = "never", 1 = "1 or 2 times", 2 = "3 or 4 times", and 3 = "5 or more times"). The questions that asked about taking a weapon to school and using a weapon in a fight were coded dichotomously (0 = "no" and 1 = "yes"). Principal component analysis indicated that all 17 items loaded onto a single item and the reliability coefficient was reduced if any items were removed from the scale ($\alpha = .82$). Thus, all 17 items were combined into a single scale created by summing the responses to each item. Higher values reflected more involvement in delinquency.

**Delinquency at Wave 3.** During wave 3 interviews, respondents were asked a series of questions that referenced criminal activity within the last 12 months. Since most respondents had reached adulthood by wave 3, many of the criminal and delinquent behaviors were changed to reflect more age-appropriate activities and there were slightly fewer items addressed. Specifically, respondents were asked to indicate whether and how often they had deliberately damaged property, stolen something worth less than $50, stolen something worth more than $50, broken into a house, committed an armed robbery, sold drugs, taken part in a group fight, bought or sold stolen property, committed identity theft, written a bad check, used a weapon in a fight, and carried a gun to school or work. Each of the 12 items were coded on a scale ranging from 0 (i.e., "never") to 3 (i.e., "5 or more times"). Principal component analysis indicated that a single underlying construct best explained the structure of the 12 items. With the exception of two
items, the reliability coefficient was reduced when any of the items were removed from the scale. Inclusion of the "sold drugs" question reduced the alpha coefficient by a very small margin that did not warrant its exclusion from the scale (.0005). Similarly, the "writing bad checks" question reduced the coefficient by .0008. This margin of reduction did not support the exclusion of this item from the scale. As a result, all 12 items were summed together to create a scale of delinquent behavior at wave 3 with higher values reflecting more involvement in delinquent activity (α = .69).

**Drug Use at Wave 1.** During wave 1 interviews, respondents were asked whether they had ever used a number of substances. Specifically, respondents were asked to indicate whether they had ever tried a cigarette, drank alcohol, smoked marijuana, used cocaine, tried inhalants such as glue or solvents, and tried other illegal drugs such as LSD and ecstasy. The question referencing cigarette use was originally coded dichotomously (i.e., 0 = "no" and 1 = "yes"). The remaining questions asked the respondent to indicate how old they were when they first used the substance in question. Respondents were asked to select 0 if they had never used the substance. Since age of onset was not the focus of this research, all responses to these items were collapsed into dichotomous variables reflecting whether the respondent had ever used the substance (i.e., 0 = "no" and 1 = "yes"). Principal component analysis indicated that a single component best fit the data. As such, responses to the six items were combined into a scale by summing the responses. The reliability coefficient was reduced when any items were removed from the scale (α = .66). Higher values reflected more substance use.

**Drug Use at Wave 2.** During wave 2 interviews, respondents were asked to report on their drug use since wave 1 interviews. In this way, the wave 2 drug use questions are different from those collected at wave 1 because they have a different reference period. Specifically, respondents were asked whether, since wave 1 interviews, they had tried a cigarette, tried alcohol, used marijuana, tried cocaine, tried inhalants, or used other drugs such as LSD and ecstasy. Responses were coded dichotomously with 1 indicating an affirmative response and a 0 indicating a negative response. Principal component analysis supported the creation of a single scale to summarize the six items. The alpha coefficient was increased when the inhalants item was omitted. This increase, however, was negligible (.0106) and did not support its removal.
from the scale. As a result, the six items were combined by summarizing the responses to each item ($\alpha = .63$).

**Drug Use at Wave 3.** Drug use was measured at wave 3 by asking the respondent to indicate whether, in the last year, they had tried or used six substances. Respondents were asked to indicate whether they had used marijuana, cocaine, crystal meth, other drugs such as LSD and ecstasy, drugs taken intravenously such as heroin, and alcohol over the last 12 months. Responses were coded dichotomously (0 = "no" and 1 = "yes"). Principal component analysis supported the combination of the six items into a single scale. The reliability coefficient was increased when the questions referencing intravenous drugs and alcohol were omitted. However, the increase was not enough to warrant their removal from the scale (the increase was less than .02 for each item). These variables, therefore, were retained as part of the scale. As a result, the scale was created by summing across all six items ($\alpha = .56$). Higher values were indicative of more drug use.

**Antisocial Behavior at Wave 1.** Principal component analysis indicated that the 17 items from the wave 1 delinquency scale and the 6 items from the wave 1 drug scale loaded onto a single component tapping antisocial behavior. As a result, responses to each of the 23 items were first standardized and then averaged together ($\alpha = .88$). Higher values on this new variable reflected more involvement in antisocial behavior. Removal of any of the items resulted in a reduction in the alpha coefficient.

**Antisocial Behavior at Wave 2.** A similar procedure was carried out using the 17 items from the wave 2 delinquency scale and the 6 items from the wave 2 drug use scale. Specifically, principal component analysis indicated that the 23 items loaded onto a single component measuring antisocial behavior. The antisocial behavior scale was created by standardizing the original items and then averaging across all of the variables. Higher values on this new scale reflected more involvement in antisocial behavior at wave 2 ($\alpha = .86$). Removing any of the 23 items from the scale reduced the reliability coefficient.
Antisocial Behavior at Wave 3. Finally, the 12 delinquency items and the 6 drug use items loaded onto a single component as indicated by principal component analysis. The scale's reliability was reduced, with a few exceptions, if any items were removed. The three exceptions were for the items measuring writing bad checks, intravenous drug use, and alcohol use. The reduction in the reliability coefficient by including these items was negligible (reduction was less than .004 for all three items) and did not warrant their exclusion from the scale. As a result, all 18 items were combined by standardizing the original responses and summing across the items ($\alpha = .75$). Higher values reflected more antisocial behavior at wave 3.

5.2.5 Desistance from Delinquency, Drug Use, and Antisocial Behavior

Most criminological research testing Sampson and Laub's theory has not actually measured desistance (two notable exceptions are Maume et al., 2005 and Warr, 1998). Instead, most studies examined within-individual changes in crime involvement over time (e.g., Laub and Sampson, 2003). While change is certainly necessary for desistance to take place, change in crime over time does not necessarily mean desistance has occurred. Thus, the question of whether marriage, employment, and other adult social bonds actually affect desistance from crime largely remains unexamined. To fill this gap in the literature, the current study included measures of desistance from delinquency, drug use, and antisocial behavior.

Desistance from Delinquency. Scholars have struggled with operationalizing desistance (e.g., Beaver et al., 2008a). Some researchers have argued that desistance is a process, implying that desistance can be measured as decreases, but not necessarily a cessation, in crime over time (e.g., Bushway et al., 2001). For others, desistance is a boundary concept that identifies the discrete event at which a criminal career comes to an end (Le Blanc and Loeber, 1998). For the current analysis, desistance from delinquency was measured as a dichotomous variable with 1 indicating that the respondent had desisted and a 0 indicating that the respondent had not desisted. The construction of this variable followed in a three-step process. First, all respondents who had a nonzero score on the wave 1 delinquency scale who also had a 0 on the wave 3 delinquency scale were given a value of 1 (i.e., desisters). Second, respondents who had a 0 on the wave 1 delinquency scale, who had a nonzero score on the wave 3 delinquency scale,
or those who had a 0 on both scales were coded as 0 (i.e., non-desisters). Third, respondents with missing values on either the wave 1 or the wave 3 delinquency scales were coded as missing. This coding scheme led to the identification of 2,073 respondents as desisters (57 percent of the sibling pair subsample) and 1,560 as non-desisters (43 percent of the sibling pair subsample).

**Desistance from Drug Use.** Desistance from drug use was measured in a similar fashion to the delinquency desistance measure. Specifically, the dichotomous variable (1 = desistance and 0 = non-desistance) was constructed by giving respondents a 1 if they had a nonzero value on the wave 1 drug use scale and a 0 on the wave 3 drug use scale. All respondents who had a 0 on the wave 1 drug use scale, a nonzero score on the wave 3 drug use scale, or had a 0 on both the wave 1 and the wave 3 drug use scale were given a 0 on the drug use desistance measure. All respondents with a missing value on either scale were coded as missing. This approach identified 540 respondents as desisters (15 percent of the sibling pair subsample) and 3,103 as non-desisters (85 percent of the sibling pair subsample).

**Desistance from Antisocial Behavior.** The final desistance measure indicated whether the respondent had desisted from all antisocial behavior (i.e., delinquency and drug use). As before, all respondents who had a nonzero score on the wave 1 antisocial behavior scale who also had a 0 on the wave 3 antisocial behavior scale were given a value of 1. Those with a 0 on the wave 1 antisocial behavior scale, who had a nonzero score on the wave 3 antisocial behavior scale, or had a 0 on both scales were coded as 0. Respondents with a missing value on either the wave 1 or the wave 3 antisocial behavior scale were coded as missing. This procedure produced 663 desisters (19 percent of the sibling pair subsample) and 2,906 non-desisters (81 percent of the sibling pair subsample).

5.2.6 Control Variables

Some models required that between-sibling differences in age, gender, and race be controlled in order to garner unbiased estimates. As a result, each respondent's age was included as a continuous variable measured in years. Gender was measured as a dichotomous variable
where 1 indicated the respondent was male and 0 indicated the respondent was female. Race was also coded dichotomously with 1 indicating that the respondent was White and 0 indicating that the respondent was non-White.

5.3 ANALYTIC PLAN

Presented in the previous chapter were five research questions that will be addressed by the current study. These research questions are as follows:

(1) Do genetic factors influence exposure to adult social bonds among a nationally representative sample of siblings?
(2) Are genetic factors important for explaining changes in delinquency, drug use, and antisocial behavior over time?
(3) Are genetic factors important for explaining desistance from delinquency, drug use, and antisocial behavior?
(4) Do adult social bonds explain desistance from delinquency, drug use, and antisocial behavior?
(5) Do genetic factors reduce or eliminate the relationship between adult social bonds and desistance?

This section will describe the analytic methods that will be used to address each of the questions. As such, this section will be organized according to the five research questions.

5.3.1 Research Question 1: Do Genetic Factors Influence Exposure to Adult Social Bonds Among a Nationally Representative Sample of Siblings?

Recall from Chapter 2 of this dissertation that behavioral genetic research methods require information on more than one child per household. Additionally, the level of genetic relatedness between the children in each household must be available. Fortunately, the Add Health data include both of these pieces of information. The Add Health data include information on MZ twins, DZ twins, full-siblings, half-siblings, and cousins who live in the same
household. To address the first research question, the family-based method (see Section 2.1.2) is employed. The family-based method will allow for the estimation of the proportion of the variance in an adult social bond that is due to heritable factors ($h^2$), shared environmental factors ($c^2$), and nonshared environmental factors ($e^2$).

Behavioral geneticists have developed a number of methods for decomposing the variance in a phenotype into the three variance components (i.e., $h^2$, $c^2$, and $e^2$). One of the most popular approaches to biometric modeling—often referred to as "model-fitting"—uses structural equation methods (SEM) of estimation. The most common model used by behavioral geneticists is the ACE model. Factor A corresponds to $h^2$, factor C corresponds to $c^2$, and factor E corresponds to $e^2$. This model is presented in Figure 5.3.1.1. There are several pieces of information that are crucial to understanding how the ACE model produces estimates of $h^2$, $c^2$, and $e^2$. First, notice that the model contains two observed indicators (i.e., Phenotype for Sibling 1 and Phenotype for Sibling 2). As was previously discussed, behavioral genetic methods compare siblings on an outcome to determine whether siblings that share more genetic material resemble each other more closely. For this reason, the ACE model includes information from both sibling 1 and sibling 2 on the same measure. Second, you will notice that A, C, and E are included as latent factors that explain the variance in the phenotype of interest. The path coefficients leading from these latent factors (i.e., a1, c1, e1, a2, c2, and e2) to the observed phenotypes will provide estimates of $h^2$, $c^2$, and $e^2$. Third, note that the A factors have a correlation (i.e., the curved double headed arrows) that can vary from 1.00 to .125. The value for the correlation between the A factors corresponds to the level of genetic relatedness between the two siblings providing data. Thus, when MZ twins are being observed, the correlation is set to 1.00, when DZ twins are being observed the correlation is set to .50, and so on. Fourth, note that the C factors are also correlated, but that this correlation is always set to 1.00. The C factor captures the variance that is due to shared environmental influences. Since, by definition, shared environments are always identical between two siblings, the correlation is always set to 1.00. Fifth, the E factors are left free to vary. By definition, nonshared environmental factors are not shared between two siblings. Therefore, there is no correlation between two siblings on nonshared environmental influences.

Although model-fitting is one of the most popular approaches to gaining behavioral genetic estimates, it is not the only analytic technique that is available. In fact, another way to
gain behavioral genetic estimates is through a regression-based statistic known as the DeFries-Fulker (DF) model. The DF model, like the ACE model, decomposes the variance in a phenotype into $h^2$, $c^2$, and $e^2$. A key difference between the DF model and the ACE model is that the former is based on linear regression techniques (DeFries and Fulker, 1985; Rodgers and Kohler, 2005). Although the two approaches differ in estimation methods, they have been shown to produce almost identical estimates (Cherny et al., 1992). The DF model takes the following form:

$$K_1 = b_0 + b_1(K_2 - K_m) + b_2(R * (K_2 - K_m)) + e$$

In this equation, $K_1$ represents the score on an outcome measure for sibling 1. For example, $K_1$ can represent sibling 1’s scores on the Religiosity scale. $K_2$ represents the score on the same outcome measure, but the value reflects sibling 2’s score on that measure. $K_m$ is the mean for $K_2$ and is used to mean center sibling 2’s score. $R$ is a measure of genetic relatedness for the sibling pair (e.g., $R = 1.0$ for MZ twins, $R = .5$ for DZ twins, etc.). The next term, $R * (K_2 - K_m)$, is a multiplicative interaction term created by multiplying the sibling pair's level of genetic relatedness by sibling 2's score on the variable of interest. In the DF model, the regression coefficients represent the variance component estimates: $b_0$ is the constant and is not typically interpreted, $b_1$ is the unstandardized regression coefficient reflecting the proportion of variance in $K_1$ that is explained by the shared environment, and $b_2$ reflects the proportion of variance in $K_1$ that is explained by genetic factors (i.e., heritability). The error term (i.e., $e$) is an estimate of the proportion of variance in $K_1$ that is due to nonshared environmental influences and measurement error.

In order to address Research Question 1, the DF model will be employed. Specifically, estimates of the proportion of the variance that is due to heritable and environmental influences on each of the adult social bonds presented above will be garnered from DF analysis. For example, to determine whether and how much variation in the Job Satisfaction variable is due to genetic and environmental influences, a DF model utilizing sibling 1’s score on Job Satisfaction as the outcome and sibling 2’s score as the predictor will be employed. The DF model presented above, however, was developed to deal with normally distributed variables. Many of the outcomes of interest to this study, however, are dichotomous indicators. For these variables, it is
not possible to garner direct estimates of $h^2$, $c^2$, and $e^2$. This does not mean, however, that the DF model cannot be used. Instead, the DF model must be altered and estimated with a log-link function:

\[
\log_e[P(K_1)/1-P(K_1)] = b_0 + b_1(K_2) + b_2(R \ast (K_2)) + e
\] (5.2)

In this model, the outcome now becomes the log odds of sibling 1 scoring a 1 on the outcome. $K_2$ still refers to sibling 2’s score on the same variable, and $R$ is still the coefficient of genetic relatedness. Additionally, $b_0$ is still the constant and is not interpreted, $b_1$ is still an estimate of the shared environment, and $b_2$ is still an estimate of heritability, and $e$ still captures the nonshared environment and measurement error. The only limitation to this model is that the regression coefficients no longer correspond to direct estimates of the amount of variance explained. Instead, the coefficient can only be interpreted for its significance level. For example, if $b_1$ is significantly different from zero, then the researcher concludes that the shared environment explains a significant portion of the variance in the outcome. The point estimate, however, does not correspond to the amount of variance explained.

Since the logistic DF model does not allow for the direct estimation of the amount of variance explained by each component, several additional statistics will be presented when considering the dichotomous variables. These additional statistics do not directly correspond to estimates of $h^2$, $c^2$, and $e^2$. They will, however, assist in the interpretation of the regression coefficients. Specifically, probandwise concordance rates, intraclass tetrachoric correlations, and intraclass odds ratios will be presented. The probandwise concordance rate is a statistic that represents the risk of sibling 1 having a 1 on the variable if sibling 2 has a 1. The probandwise concordance rate is estimated by applying the following formula to each level of sibling relatedness (i.e., MZ twins, DZ, twins and full-siblings, half-siblings, and cousins; Plomin, 1990):

\[
\text{Probandwise Concordance} = (2C) / (2C + D)
\] (5.3)

In the above formula, $C$ represents the number of concordant pairs (i.e., both have a 1 on the outcome) and $D$ denotes the number of discordant pairs (only one member has a 1). As can be
seen, the number of concordant pairs is multiplied by 2 and divided by the sum of the number of concordant pairs (multiplied by 2) and the number of discordant pairs.

The intraclass tetrachoric correlation coefficient is another statistic that measures the degree to which family members are concordant on an either/or trait (i.e., a dichotomous variable). The tetrachoric correlation incorporates more information than the probandwise concordance rate. Specifically, the tetrachoric correlation incorporates population incidence. The probandwise concordance rate, on the other hand, only incorporates information from pairs where at least one sibling is affected (Plomin, 1990). For this reason, the tetrachoric correlation is a useful statistic for behavioral geneticists (Lyons et al., 1995).

One potential limitation of the tetrachoric correlation is that it requires the researcher to assume that the binomial variable is an indicator of an underlying normally distributed outcome (Plomin, 1990). Since this assumption may not always be met, it is useful to calculate additional statistics that indicate concordance rates across different levels of relatedness. As a result, a third measure of concordance referred to as the intraclass odds ratio will be presented. When dealing with dichotomies, behavioral geneticists often disaggregate the sample according to genetic relatedness and estimate a logistic regression model for each subsample (see Cho et al., 2006). The logistic model takes the following form:

$$\log_e[P(K_1)/1- P(K_1)] = b_0 + \exp(b_1(K_2))$$  \hspace{1cm} (5.4)

In the above logistic regression formula, $b_0$ is the constant and is typically not interpreted. As can be seen, the coefficient $b_1$ is exponentiated, transforming it to an odds ratio. This formula, therefore, provides an estimate of the odds that sibling 1 has a 1 on the outcome based on sibling 2's scores.

In summary, Research Question 1 will be addressed by employing the DF model. In most cases, the DF model will provide estimates of $h^2$, $c^2$, and $e2$. However, when the outcome of interest is a dichotomy, the logistic DF model will be used. The logistic model will not produce estimates of $h^2$, $c^2$, and $e2$. In order to supplement the logistic DF model the probandwise concordance rate, the intraclass tetrachoric correlation coefficient, and the intraclass odds ratio, will be presented. These additional statistics will help to reinforce the conclusions that are drawn from the logistic DF model.
Recall that when employing a family-based design, it is important to remove any variance that is due to sibling differences in gender, age, and race. Accordingly, this analysis will control for any gender differences, age differences, or racial differences that exist between siblings. In other words, it is important to "control" for these nonshared environments when estimating the DF model. To incorporate measured nonshared environments, Rodgers, Rowe, and Li (1994) presented a modified version of the DF equation. This modified DF equation takes the following form:

\[ K_1 = b_0 + b_1(K - K_m) + b_2(R \times (K - K_m)) + b_3(ENVDIF) + e \] (5.5)

The only difference between this equation and the ones presented earlier is the inclusion of a new variable, ENVDIF, which captures the difference between twins on a measured variable (i.e., gender, age, and race). ENVDIF is calculated by subtracting one twin’s score on a measure from their co-twin’s score on the same measure. In this equation, \( b_3 \) is the unstandardized regression coefficient for the nonshared environment and is typically only interpreted for the direction and significance of the relationship with \( K_1 \). All of the remaining coefficients are interpreted as they are in Equation 5.1. The coefficient estimates for gender, age, and race differences will not be presented. However, it is important to note that these nonshared environmental influences will be controlled in almost all of the analyses.

### 5.3.2 Research Question 2: Are Genetic Factors Important for Explaining Changes in Delinquency, Drug Use, and Antisocial Behavior Over Time?

A growing body of research has shown that genetic and environmental factors are important for predicting changes in antisocial behavior over time. This line of evidence, however, is far from conclusive. Therefore, it is important to replicate these findings using different samples. To date, no research has estimated the effect of genetic and environmental influences on changes in delinquency, drug use, and antisocial behavior using the Add Health data. Thus, this analysis will provide much needed replication of extant evidence.

Estimating the effect of genetic and environmental influences on changes in behavior over time requires more complicated statistical models than when estimating these effects on a
phenotype measured at one time point. Recall from the previous section that the ACE model is often used by behavioral geneticists to estimate the influence of genetic and environmental influences on a phenotype. The ACE model represents a "univariate" model. In this sense, the term univariate refers to the fact that only one phenotype is being considered. Although we have two measures in the ACE model, they represent the same phenotype (i.e., one from sibling 1 and one from sibling 2). In order to estimate models that incorporate changes in antisocial behavior, a variant of the ACE model is necessary. Presented in Figure 5.3.2.1 is a graphical depiction of the bivariate Cholesky model. This model is "bivariate" because it incorporates two phenotypes (or the same phenotype measured at two different time points). In this way, the Cholesky model can estimate the genetic and environmental influences on the covariation in two phenotypes or the covariation in one phenotype measured at different times.

Several points warrant attention with the Cholesky model. First, the now familiar latent factors A, C, and E are included in the model. Like the univariate model presented earlier, A represents genetic factors, C represents shared environmental factors, and E represents nonshared environmental factors. Second, there are two versions of A, C, and E. Specifically, the Cholesky model incorporates estimates of A1, C1, E1, A2, C2, and E2. The difference between the factors with a "1" and those with a "2" is very important. Specifically, A1, C1, and E1 estimate the influence of genetic and environmental influences on the covariance between the two measures. For our purposes, A1, C1, and E1 can be thought of as estimates of the proportion of the stability from Time 1 to Time 2 that is due to genetic (A1), shared environmental (C1), and nonshared environmental (E1) factors. Stability is measured as the correlation between the phenotype at Time 1 with the same phenotype at Time 2. It should be noted that the correlation coefficient can be represented by the following formula:

\[ r = (a11 \times a21) + (c11 \times c21) + (e11 \times e21) \tag{5.6} \]

To estimate the proportion of variance in stability that is due to, say, the shared environment, a researcher would only need to multiply the path coefficients c11 and c21 and divide by the stability coefficient (r).

The third point that is important to note about the Cholesky model is that the factors A2, C2, and E2 estimate the proportion of the variance in changes from Time 1 to Time 2 that are
due to genetic influences, shared environmental influences, and nonshared environmental influences, respectively. This means that a coefficient of change (Δ) can be calculated from the following formula:

\[ Δ = (a22)^2 + (c22)^2 + (e22)^2 \]  \hspace{1cm} (5.7)

The path coefficients a22, c22, and e22 can be squared and added together to represent the amount of change from Time 1 to Time 2 (Δ). In order to estimate the proportion of the variance in changes from Time 1 to Time 2 that are due to genetic influences, one only needs to square the path coefficient for a22 and divide by \(Δ \) \((a22)^2 / Δ\). The same procedure can be used to estimate the proportion of variance in change that is due to shared and nonshared environmental influences.

In order to address Research Question 2, the bivariate Cholesky model will be estimated. This analysis will estimate a bivariate Cholesky model to determine the extent to which genetic and environmental factors influence stability and change in delinquency, drug use, and antisocial behavior.

### 5.3.3 Research Question 3: Are Genetic Factors Important for Explaining Desistance from Delinquency, Drug Use, and Antisocial Behavior?

The current study included three measures of desistance: one for delinquency, one for drug use, and one for antisocial behavior. Each of these three measures will be analyzed with behavioral genetic methods to determine whether genetic factors are important for explaining desistance. Similar to the strategy employed to address Research Question 1, a univariate DF model will be used to estimate the impact of genetic and environmental factors on desistance. Since the desistance measures are dichotomous indicators, the logistic DF model presented in Equation 5.2 will be estimated. Recall, however, that direct estimates of the proportion of variance explained by genetic and environmental factors cannot be gleaned from this model. Instead, whether the factor is a significant influence on desistance is all that can be drawn from the logistic DF model. As a result, several additional statistics will be presented: the
probandwise concordance rate, the intraclass tetrachoric correlation, and the intraclass odds ratio. See Section 5.3.1 for more details on each of these three statistics.

5.3.4 Research Question 4: Do Adult Social Bonds Explain Desistance from Delinquency, Drug Use, and Antisocial Behavior?

The major premise set forth in Sampson and Laub's (1993) theory is that adult social bonds are important for explaining desistance from crime. An extensive body of literature has tested this hypothesis, however, almost no research actually measured desistance. Instead, intra-individual changes in crime were most often the focus. This means that we still know very little about the effect of adult social bonds on desistance from crime. The current study will provide some empirical evidence bearing on this point. A measure of desistance from delinquency, drug use, and antisocial behavior will be analyzed to determine if each of the adult social bonds increase the odds of desistance. Specifically, the following equation will be estimated for each of the adult social bonds:

\[
\log_e\left[\frac{P(K_1)}{1-P(K_1)}\right] = b_0 + \exp(b_1(BOND_{K1})) \quad (5.8)
\]

Two points must be mentioned. First, the results for this model are presented as odds ratios. Thus, any value greater than 1 is interpreted as increasing the odds of desisting from delinquency, drug use, and/or antisocial behavior. Second, when estimating the effect of adult social bonds on desistance, only one sibling from each sibling pair will be assessed. In other words, when addressing Research Question 4, the standard social science method (SSSM) of examining one child per household will be employed. This is done to ensure comparability of findings with the extant literature. Thus, the findings bearing on Research Question 4 are interpreted as between-individual differences in desistance as a function of exposure to adult social bonds.

5.3.5 Research Question 5: Do Genetic Factors Reduce or Eliminate the Relationship between Adult Social Bonds and Desistance?
The final research question will be analyzed using the logistic DF model. The findings bearing on Research Question 4 will lay the foundation for whether adult social bonds affect desistance for the Add Health respondents. The next step, however, is to determine whether these effects remain after shared genetic effects are partialled out of the equation. To estimate the logistic DF model, it will be necessary to use both siblings from each sibling pair. In order to control for shared genetic effects the following equation will be estimated:

\[
\log_e \left[ \frac{P(K_1)}{1 - P(K_1)} \right] = b_0 + b_1(K_2) + b_2(R \ast (K_2)) + b_3(ENVDIF) + e
\]  

(5.9)

The equation presented above is the logistic DF model. One unique feature of this model, however, warrants attention. Specifically, notice that this model includes coefficient $b_3$ which captures the effect of environmental differences between siblings (i.e., a measured nonshared environmental influence). The ENVDIF variable will capture the effects of the adult social bond on desistance. In this way, the effect of the adult social bond becomes a nonshared environmental influence. This allows for the removal of any genetic and shared environmental factors that may explain the link between the adult social bond and desistance. The findings garnered from Research Question 4 will be compared with those garnered from the current equation. If adult social bonds have "unique" effects on desistance, as Sampson and Laub (1993) argue, then we should expect to find positive effects for each adult social bond after the DF models are estimated. If adult social bonds are spurious to genetic factors, then any effect found when using the SSSM (i.e., Research Question 4) should vanish when the logistic DF model is estimated.
CHAPTER 6

FINDINGS

Divided into four sections—each corresponding to a specific research question—this chapter will present the findings from the statistical analyses outlined in the previous chapter. The first section will review the findings from the DF models analyzing the genetic and environmental influences on each of the adult social bonds (Research Question 1). The second section will present the findings from the Cholesky models where changes in delinquency, drug use, and antisocial behavior are analyzed (Research Question 2). The third section presents the findings from the DF models examining the genetic underpinnings to desistance (Research Question 3). Finally, the fourth section reviews the findings from logistic regression models examining the effect of the adult social bonds on desistance before (Research Question 4) and after genetic effects have been controlled (Research Question 5).

Across all analyses, findings from the full sample of siblings will be presented along with findings split by sibling gender. The gender specific analyses are estimated on same-sex siblings where male-male sibling pairs are examined separately from female-female sibling pairs. Behavioral genetic researchers often analyze models disaggregated by gender (Moffitt, 2001), and some criminologists have theorized that the link between social bonds and desistance is gender-specific (Piquero, Brame, and Moffitt, 2005; Uggen and Kruttschnitt, 1998). Thus, it is informative to fit models to the full sample, the male subsample, and the female subsample.

6.1 RESEARCH QUESTION 1: DO GENETIC FACTORS INFLUENCE EXPOSURE TO ADULT SOCIAL BONDS AMONG A NATIONALLY REPRESENTATIVE SAMPLE OF SIBLINGS?
Recall that some of the adult social bonds were measured dichotomously. For these variables, it was necessary to employ the logistic DF formula. When the logistic DF formula was utilized, the term "Logit" was inserted into the table next to the model number. Thus, unless noted differently, the standard DF model was utilized.

Table 6.1.1 displays findings from the DF models that analyzed the genetic and environmental influences on the relationship variables. The ever married variable was measured dichotomously and thus called for the logistic DF formula (Model 1). As can be seen, the estimates indicated that genetic factors were important for explaining marital status. Shared environmental influences were also important and these findings were similar across the gender specific models. The logistic DF estimates cannot be used to determine the exact proportion of variance explained by each factor. As a result, the probandwise concordance rate, the intraclass tetrachoric correlation coefficient, and the intraclass odds ratio, are presented to facilitate a better understanding of the results gleaned from the logistic DF model. The estimates for these supplemental statistics correspond to the full sample of sibling pairs, not the gender specific models. The probandwise concordance rate revealed that MZ twins had the highest concordance rate of any sibling pair type. DZ twins and full-siblings had a higher concordance rate than half-siblings (abbreviated as “HS”) and cousins. These findings are supportive of a genetic influence on marital status. The cascade of intraclass tetrachoric correlations revealed a similar pattern (Figure 6.1.1). Specifically, MZ twins had the highest correlation, followed by DZ twins and full-siblings. Cousins had a larger intraclass correlation than half-siblings. The intraclass odds ratios were also supportive of genetic effects.

The next three models presented in Table 6.1.1 employed the standard DF equation. Thus, the estimates gleaned from these analyses can be interpreted as estimates of the proportion of variance explained by each factor. For instance, Model 2 indicated that genetic influences explained approximately 5 percent of the variance in martial love for the full sample of siblings and the shared environment did not significantly explain any of the variance. When analyzing male-male siblings, neither the heritability coefficient, nor the shared environment coefficient reached statistical significance. When analyzing the female-female siblings, only shared environmental influences explained a significant proportion of the variance.
Model 3 revealed that genetic factors explained a significant proportion of the variance in marital commitment for the full sibling sample \((h^2 = .17)\). The shared environment did not explain any of the variance in marital commitment. The gender specific analyses revealed that genetic factors explained a significant proportion of the variance for the male-male subsample, but not for the female-female subsample. Finally, Model 4 indicated that genetic factors explained 7 percent of the variance in the composite marital scale for the full sibling sample. The gender specific analyses revealed that genetic factors were influential for male-male sibling pairs \((h^2 = .18)\) but not for female-female sibling pairs.

Presented in Table 6.1.2 are four logistic DF models which analyzed the military involvement and employment social bonds. As can be seen in Model 1, genetic factors did not significantly predict military involvement for the full sibling sample nor for the gender specific subsamples. Model 2 found that genetic factors were relevant for explaining variance in employment status for the full sibling sample. Genetic factors were significant in the female-female subsample but not in the male-male subsample. Model 3 analyzed job stability and indicated that genetic factors were relevant for the full sibling sample, for the male-male subsample, and for the female-female subsample. Finally, Model 4 revealed a significant genetic contribution to working two jobs for the full sibling sample and for the female-female subsample. Across all the models, the pattern of concordance rates, tetrachoric correlations, and odds ratios generally supported the conclusions drawn from the logistic DF analyses. Specifically, these supplementary statistics suggested that genetic factors are not important for military involvement, but are relevant for the other three outcomes (Figure 6.1.2).

All five models presented in Table 6.1.3 employed the standard DF model, meaning that the estimates can be interpreted as the proportion of variance explained. As can be seen in Model 1, genetic factors explained between 29 percent (female-female subsample) and 48 percent (male-male subsample) of the variance in education level. Model 2 revealed that levels of job satisfaction were under genetic influence for the full sibling sample and for the gender specific subsamples. Model 3 produced similar results for income levels: 37 percent of the variance is explained by genetic factors in the full sibling sample. A significant proportion of the variance in the number of hours worked was explained by genetic factors for the full sibling sample and for the female-female subsample (Model 4). Finally, Model 5 indicated that genetic influences explained a significant proportion of the variance in the composite employment scale.
Presented in Table 6.1.4 are five models that analyzed the genetic and environmental influences on community service and religious involvement. Model 1 revealed that genetic factors explained a significant proportion of the variance in community service involvement for the full sibling sample and the male-male subsample. The supplementary statistics—concordance rate, tetrachoric correlation, and odds ratio—supported this conclusion (Figure 6.1.3). Models 2 through 5 analyzed religious involvement outcomes. For the most part, these analyses revealed that genetic factors explained a significant proportion of the variance in each outcome for the full sibling sample. For example, Model 3 indicated that 35 percent of the variance in importance of religion was explained by genetic factors. Model 4, on the other hand, found that genetic factors were not statistically significant in explaining variance in religiosity for the full sibling sample. Findings for the gender specific models were more mixed. Genetic factors were important for explaining variance in church attendance, importance of religion, and the composite religion scale for the male-male subsample. For the female-female subsample, genetic factors were only important for explaining variance in church attendance.

The findings presented in this section revealed that many of the adult social bonds measured at wave 3 are significantly influenced by genetic factors. In some cases, genetic factors explained a substantial proportion of the variance (e.g., 38 percent for church attendance). In other cases, genetic factors were important, but only accounted for a small proportion of the variance (e.g., 7 percent for the composite marital scale). The next step in the analysis is to determine whether genetic factors are important for explaining changes in behavior over time.

### 6.2 RESEARCH QUESTION 2: ARE GENETIC FACTORS IMPORTANT FOR EXPLAINING CHANGES IN DELINQUENCY, DRUG USE, AND ANTISOCIAL BEHAVIOR OVER TIME?

Tables 6.2.1 through 6.2.4 present the findings from the longitudinal analysis of changes in delinquency, drug use, and antisocial behavior. Changes in behavior from wave 1 to wave 3 were modeled in these analyses. The first step was to examine the degree of stability in behavior over time. Presented in Table 6.2.1 are stability coefficients (i.e., correlation coefficients) for delinquency, drug use, and antisocial behavior from wave 1 to wave 3. These analyses revealed
that behavior was stable over time, but change was the predominant pattern (no coefficient was over .40).

In order to determine the factors that account for stability and changes in behavior, it was necessary to analyze the data using the bivariate Cholesky model. Recall that the path coefficients estimated by the Cholesky model can be used to estimate the level of genetic and environmental influences on stability and changes in behavior. Findings from the Cholesky analysis on stability and change in delinquency are presented in Table 6.2.2. For ease of readability, the table identifies the factors that account for stability separately from those that account for change. Beginning with the estimates garnered from the full sibling sample, we see that genetic factors (i.e., A) were the predominant influence on stability in delinquency (77 percent). Nonshared environmental factors (i.e., E) were the only environmental influence that accounted for stability (.23); the shared environment (i.e., C) was unimportant. Importantly, the findings also revealed that genetic factors accounted for a proportion of the variance in changes in delinquency: genetic factors accounted for 21 percent of the variance in changes in delinquency. Perhaps not surprisingly, nonshared environmental influences were the most influential factor on changes in delinquency (76 percent).

Although the estimates are slightly different, the substantive conclusions garnered from the male-male subsample and from the female-female subsample are identical to those garnered from the full sibling sample. Specifically, genetic factors accounted for the largest proportion of the variance in stability across both the male-male and female-female subsample analyses. At the same time, genetic factors were important for explaining changes in delinquency for both subsamples. Nonshared environmental influences accounted for the largest proportion of the variance in changes in delinquency for both subsamples, though much more so for females.

Table 6.2.3 presents the findings gleaned from the Cholesky model analyzing stability and change in drug use from wave 1 to wave 3. As can be seen, genetic factors accounted for the largest proportion of the variance in stability for the full sibling sample, for the male-male subsample, and for the female-female subsample (.52, .93, and .51, respectively). Genetic factors were also important for explaining changes in drug use. Specifically, genetic factors accounted for approximately 30 percent of the variance in changes in drug use across all three analyses. As with the delinquency analysis, nonshared environmental factors were important for explaining changes in drug use. Unlike the results from the delinquency analysis, however, the
shared environment proved to be influential for explaining stability and change in drug use. The shared environment accounted for 33 percent of the stability and for 13 percent of the change in drug use for the full sibling sample.

The results from the Cholesky model analyzing stability and change in antisocial behavior (a composite score of delinquency and drug use) from wave 1 to wave 3 can be found in Table 6.2.4. The results from this analysis are similar to those garnered from the analyses of stability and change in delinquency and drug use. To be specific, genetic influences were important for explaining stability and change for the full sibling sample and for the male-male subsample. For example, genetic factors explained 74 percent of the variance in stability and 33 percent of the variance in changes in antisocial behavior for the full sibling sample. Nonshared environmental influences were also important: nonshared environmental factors accounted for 63 percent of the variance in changes in antisocial behavior for the full sibling sample. For the female-female subsample, genetic influences were important for explaining stability, but were less important for explaining change (2 percent). With one exception, the shared environment was relatively unimportant for explaining stability and change in antisocial behavior. The lone exception was that the shared environment accounted for 28 percent of the variance in changes in antisocial behavior for the female-female subsample.

Figure 6.2.1 presents a summary of the results from the Cholesky models. The findings presented in this figure were drawn from the full sibling sample analyses. As can be seen, genetic factors were the preponderant influence on stability in delinquency, drug use, and antisocial behavior. Most important for the current discussion, however, is that genetic factors were also influential on changes in delinquency, drug use, and antisocial behavior.

6.3 RESEARCH QUESTION 3: ARE GENETIC FACTORS IMPORTANT FOR EXPLAINING DESISTANCE FROM DELINQUENCY, DRUG USE, AND ANTISOCIAL BEHAVIOR?

Results from the logistic DF models analyzing the genetic and environmental influences on desistance from delinquency, drug use, and antisocial behavior can be found in Table 6.3.1. Genetic factors explained a statistically significant portion of the variance in desistance from delinquency. This finding was consistent across the gender specific models and observation of
the concordance rates, the tetrachoric correlations (Figure 6.3.1), and the odds ratios generally supported this conclusion (although it should be noted that cousins had a closer association than did half-siblings – a finding counter to what would be expected if genetic factors were the only operative influence). As can be seen, the shared environment did not significantly predict desistance from delinquency.

Genetic effects were not significantly related to desistance from drug use or antisocial behavior for the full sibling sample. In both cases, the shared environment was the dominant factor predicting desistance. The findings from the gender specific subsamples revealed that genetic factors were important for explaining desistance from drug use and antisocial behavior when analyzing male-male sibling pairs. When analyzing female-female sibling pairs, the shared environment was the only significant factor.

Since genetic factors explained a significant proportion of the variance across all three analyses of delinquency desistance (e.g., the full sibling sample, the male-male subsample, and the female-female subsample), the next section will examine the impact of adult social bonds on desistance from delinquency. Genetic factors were not consistently related to desistance from drug use and antisocial behavior, so these outcomes will not be considered for the remainder of the analysis.

6.4 RESEARCH QUESTION 4: DO ADULT SOCIAL BONDS EXPLAIN DESISTANCE FROM DELINQUENCY, DRUG USE, AND ANTISOCIAL BEHAVIOR? RESEARCH QUESTION 5: DO GENETIC FACTORS REDUCE OR ELIMINATE THE RELATIONSHIP BETWEEN ADULT SOCIAL BONDS AND DESISTANCE?

Findings from the previous section revealed that genetic factors were important for explaining desistance from delinquency but not for explaining desistance from drug use and antisocial behavior (see the full sibling sample results). Building on these findings, this final section will analyze the effect of each adult social bond on desistance from delinquency. The first step was to examine the relationship between each adult social bond and desistance from delinquency using a standard criminological approach. Specifically, desistance was regressed on each adult social bond without accounting for genetic effects. Since this approach does not account for genetic influences, it can be considered a standard social science method (SSSM) for
examining individual-level predictors of behavior. The second step of the analysis analyzed the effect of each adult social bond on desistance after accounting for genetic factors. The results from the logistic DF model are interpreted as sibling differences. For example, if the coefficient "X" is positive and significant, the interpretation is that the sibling with more of "X" had a greater likelihood of desisting from delinquency.

The first set of models is presented in Table 6.4.1. These models examined the association between the relationship variables and desistance from delinquency. Model 1 analyzed the link between marital status and desistance. As can be seen, when analyzing this relationship with an SSSM (no control for genetic factors), respondents that reported being married were significantly more likely to desist from delinquency than were unmarried respondents. This finding was consistent in the male-male subsample and the female-female subsample. When the logistic DF model was employed (genetic factors controlled), the predictive power of marital status was reduced but remained statistically significant. Genetic factors accounted for approximately 60 percent of the observed effect of marital status on desistance for the full sibling sample ($\hat{\beta}_{\text{SSSM}} = 0.41; \hat{\beta}_{\text{DF}} = 0.24; 0.24/0.41 = 0.59$).

Model 2 analyzed the relationship between love importance and desistance. When analyzing love importance with an SSSM in the full sibling sample, the social bond was not significantly related to desistance. When the male-male subsample was analyzed, however, the results indicated that love importance was significantly related to desistance. Before controlling for genetic factors, respondents who reported more love importance were more likely to desist from delinquency. Once genetic factors were controlled, this relationship was no longer statistically significant. The reduction in the size of the coefficient, however, was of negligible size and was not statistically significant. For the female subsample, the social bond did not reach statistical significance in either model.

Model 3 examined the association between commitment importance and desistance. When the full sibling sample was analyzed with an SSSM, commitment importance was related to desistance such that respondents who reported more commitment importance were more likely to desist. Once genetic factors were controlled, the relationship was unaffected. For the male subsample, there was no affect of the social bond on desistance in the SSSM nor in the DF model. In the female-female subsample analysis, marital commitment was significantly related to desistance in the SSSM, but not in DF model (the difference in the coefficient was not
statistically significant). Finally, Model 4 analyzed the impact of the composite marital scale on desistance. When analyzed with an SSSM in the full sibling sample, the composite scale was significantly related to desistance. This relationship was reduced and was no longer significant once genetic factors were controlled (the difference in the coefficient was not statistically significant). A similar pattern of findings emerged in the male-male subsample analysis (coefficient differences were not statistically significant). The female-female subsample analysis did not produce a statistically significant result in the SSSM nor in the DF model.

Presented in Table 6.4.2 are models that examined the relationship between military involvement and employment outcomes on desistance. Model 1 revealed that, with one exception, military involvement was unrelated to desistance from delinquency in the SSSM and in the logistic DF model. The lone exception was for the relationship between military involvement and desistance using the logistic DF model in the male-male subsample. Specifically, for males, military involvement was unrelated to desistance in the SSSM. In the DF model, however, the coefficient increased and revealed that the sibling who reported military involvement was more likely to desist. It is unclear whether suppression effects underlie this finding. Model 2 examined the link between employment status and desistance. This analysis indicated that the observed relationship between employment status and desistance was significant in the SSSM model, but not in the genetically sensitive model (i.e., the logistic DF analysis). The difference in the coefficient form the SSSM to the DF model was not statistically significant. Model 3 revealed that job stability was unrelated to delinquency desistance, whether analyzed with the SSSM or the logistic DF model. Finally, Model 4 revealed that working two jobs was not related to desistance in the SSSM models and was only related to desistance in the DF model where the male-male subsample was being examined. Interestingly, the coefficient was significant for the male-male subsample when the DF model was estimated. It is unclear whether this effect was due to suppression effects.

With all but one exception, the models presented in Table 6.4.3 indicated that employment bonds were unrelated to desistance in both the SSSM analyses and the logistic DF analyses. The only exception was for the relationship between annual income and desistance for the male-male subsample in the logistic DF analysis. This model revealed that the sibling with the higher income was more likely to desist from delinquent activity. In no instances did
controlling for genetic effects change the substantive association between the employment bonds and desistance in Table 6.4.3 (because there were no associations in the SSSMs to begin with).

The final set of analyses is presented in Table 6.4.4. These models examined the relationship between the community service and religion variables on desistance. No relationship between the community service variable and desistance was found, whether analyzed with the SSSM or the DF model. As for the religion variables, four social bonds were significant in the SSSM but not in the DF model. Specifically, importance of religion (female-female subsample only), religiosity (male-male subsample only), and the composite religion scale (full sibling sample and male-male subsample) were all significantly related to desistance when analyzed with an SSSM. When these same relationships were considered with the logistic DF model, none reached statistical significance. However, the differences in the coefficients across modeling strategies were not statistically significant. None of the other relationships between the religion variables and desistance reached statistical significance, regardless of whether the SSSM model was used, the DF model was used, the full sibling sample was analyzed, the male-male subsample was analyzed, or the female-female subsample was analyzed.
CHAPTER 7
SUMMARY AND DISCUSSION

This final chapter will serve three purposes. First, a summary of the findings will be presented. Specific findings will be highlighted and discussed within the broader context of Sampson and Laub's (1993) theory. Second, limitations of the current study will be discussed. Although the Add Health data provide numerous advantages and unique methodological elements, several points warrant closer attention. Finally, this chapter will conclude by considering the direction in which research on crime over the life-course is currently headed. The extent to which the findings from this dissertation can chart a new course for life-course criminology will be discussed.

7.1 SUMMARY OF FINDINGS

The previous chapter presented the findings from all the statistical analyses described in Chapter 5. A large amount of information was presented. It will be useful, therefore, to concisely summarize the main findings. Presented in Table 7.1.1 is a summary of the main results presented in the previous chapter. This table is divided into three main panels. The first panel summarizes the findings bearing on Research Question 1. The number of significant heritability estimates ($h^2$) for each of the relationships variables, the military and employment variables, and the community service and religion variables is displayed first. To be specific, the findings presented in the previous chapter revealed that genetic factors influenced 9 out of 12 of the relationships variables, 21 out of 27 of the military and employment variables, and 9 out of 15 of the community service and religion variables. The average heritability estimates were .10,
.31, and .27, respectively. Taken together, these findings indicate that genetic factors influenced nearly all of the social bonds analyzed.

The second main panel in Table 7.1.1 summarizes the findings bearing on Research Question 2 and Research Question 3. Starting with the findings for Research Question 2, the analyses revealed that genetic factors were the dominant influence on stability in delinquency, drug use, and antisocial behavior from wave 1 to wave 3. The most important finding for the current focus, however, was that genetic factors were influential for changes in delinquency, drug use, and antisocial behavior. As can be seen, the heritability estimates regarding changes in these behaviors was .21, .36, and .33, respectively.

Genetic factors were found to be an important influence on changes in behavior from wave 1 to wave 3. This finding hinted at the possibility that genetic factors are important for explaining desistance from delinquency, drug use, and antisocial behavior. This possibility was, therefore, directly examined (Research Question 3). The findings from the logistic DF models analyzing desistance from delinquency revealed that genetic factors were statistically significant. Specifically, in all three of the models considered (full sibling sample, male-male subsample, and female-female subsample), the heritability coefficient was statistically significant. When desistance from drug use and desistance from antisocial behavior were analyzed, the findings were less consistent. As can be seen, in only 1 out of 3 analyses were the heritability components statistically significant for desistance from drug use and for desistance from antisocial behavior.

The final main panel in Table 7.1.1 presents a summary of the findings bearing on Research Question 4 and Research Question 5. The first row summarizes the relationship between the social bonds and desistance from delinquency before genetic factors were controlled (i.e., the SSSM models). Somewhat surprisingly, the pattern of findings from this set of models revealed that most of the social bonds were not related to desistance, even prior to accounting for genetic factors. To be sure, only when considering the relationship variables did the analyses uncover a consistent association between the social bonds and desistance (i.e., the social bond predicted desistance in 8 out of 12 models for the relationship variables). Analysis of the military and employment variables revealed that only 1 social bond was related to desistance from delinquency, and only 3 out of 15 analyses of the community service and religion variables indicated a significant association.
The inconsistencies in the relationship between the social bonds and desistance (i.e., Research Question 4) made it difficult to test whether genetic factors confounded the association between adult social bonds and desistance (i.e., Research Question 5). Since the majority of tests of the relationship between social bonds and desistance did not attain statistical significance in the SSSM, the current study was limited in its ability to determine whether genetic factors confounded the social bond-desistance relationship. It is important, however, to draw further attention to one point. Most of the social bonds were unrelated to desistance in the SSSM models. This finding is problematic for Sampson and Laub’s (1993) theory since it suggests that the relationship between adult social bonds and desistance may not be confounded by genetic factors because the relationship may not exist in the first place. In other words, the current study found little support (outside of the effect of marriage on desistance) for a relationship between adult social bonds and desistance from delinquency, a finding contradictory to Sampson and Laub’s predictions.

In broad strokes, the findings from the current study can be summarized with three statements. First, genetic factors partially predicted exposure to nearly all of the adult social bonds analyzed, including many of those highlighted by Sampson and Laub (1993; e.g., marriage and employment). Second, changes in delinquency, drug use, and antisocial behavior from adolescence to adulthood were influenced primarily by genetic and nonshared environmental factors. Third, many of the adult social bonds were unrelated to desistance prior to controlling for genetic influences. Thus, little support was found for Sampson and Laub’s (1993) theory before accounting for the possible confounding effects of genes. This made it difficult to test whether genetic effects confounded these relationships because they were nonexistent in the first place (i.e., before accounting for genetic effects).

As a side note, one further point is worthy of discussion. In some cases, there were differences in findings across the models split by gender. For example, when analyzing the genetic and environmental influences on marital commitment, it was discovered that heritable influences explained approximately one-third of the variance for males, but heritable influences were not statistically significant for females. Although not the focus of this dissertation, a speculative explanation for this finding is warranted. Evolutionary psychology is often called upon to explain gender differences in behavioral genetic findings (Moffitt et al., 2001; Walsh and Beaver, 2009). It is possible that females have adapted in a way that reduces the variation in the
belief in marital commitment. For example, females may be more likely than males to agree that commitment is an important element of a strong relationship. Males on the other hand have adapted in a way that encourages multiple relationships (i.e., increasing their likelihood of procreating), which leads to decreases in the level of commitment to any one relationship. Thus, it would be more likely that heritable influences would explain variation in marital commitment for males because there is more variation to explain. For females, there is less variation because all females agree with the notion that commitment is important to a relationship, leaving less variation for the model to explain. If this were the case, heritable influences may not be expected to be a statistically significant component for females since there is little variation to explain in the first place. Preliminary support for this hypothesis can be reached with the Add Health data: descriptive statistics confirmed that less variation in marital commitment was observed in the female subsample (variance = 2.53) as compared to the male subsample (variance = 3.65) and that females (mean = 9.43), as compared to males (mean = 9.06), were more likely to rate commitment as important to a relationship.

Taken together, the findings from the current study draw into question many elements of Sampson and Laub's (1993) age-graded theory of informal social control. Sampson and Laub identified exposure to adult social bonds as the predominant factor influencing changes in behavior over the life course. Nowhere in their analyses, however, did Sampson and Laub consider whether genetic factors influenced changes in behavior. As this dissertation has shown, this omission may have led to an overestimation of the influence of adult social bonds on behavioral change. The current study revealed that, in some cases, the relationship between an adult social bond and desistance was weakened when genetic factors were controlled (e.g., the effect of marriage on desistance). In other cases, the relationship was completely explained away (i.e., the relationship was spurious to uncontrolled genetic factors). Although it is impossible to know for sure, a lingering question remains: would a reanalysis of Sampson and Laub's data reveal a pattern of results similar to those found here? This question, unfortunately, can never be answered because the Glueck data used by Sampson and Laub, like many criminological datasets, does not include information on sibling pairs. Analyses like those performed in the current work, therefore, are not feasible with the data analyzed by Sampson and Laub.
7.2 LIMITATIONS OF CURRENT STUDY

There are several limitations facing the current study that must be discussed. The first limitation is that the Add Health data do not include information beyond early adulthood. Specifically, the eldest respondent at wave 3 was 27 years old and the youngest was 18 (median age = 22). The Add Health data, therefore, cannot be used to analyze adult social bonds that occur in middle- or late-adulthood. This is an important shortcoming since Sampson and Laub's (1993) theory does not specify that an adult social bond must be experienced in early-adulthood (Although the case was made that adult social bonds must occur prior to the decline in crime which is observed (in the aggregate) around age 18 years.) In other words, Sampson and Laub do not specify when an adult social bond must occur in order to lead to desistance. Instead, it is only necessary for the adult social bond to precede desistance. Since the Add Health data are truncated in early adulthood, the current study could only analyze a person's likelihood of experiencing a social bond in early adulthood. It will be important for future research to utilize later waves of the Add Health data collection to examine the genetic influences on adult social bonds occurring later in the life course\(^{13}\).

The second main limitation of the current study is also rooted in the truncated age range of the Add Health data. Specifically, since the Add Health respondents had only reached early adulthood during wave 3 interviews, variability on many of the key measures (i.e., the adult social bonds) was limited. A notable example is the military involvement variable. Only 2 percent of all respondents reported that they had served time in the military reserves. This left very little variation to gain reliable estimates—this point may explain why military involvement was one of the few adult social bonds that did not show a significant heritability estimate. Another example is the job stability variable. Respondents were asked to indicate whether they were employed during wave 3 interviews and whether they were still working in the first paying job they had ever held. Although this item certainly taps job stability, the limited age range of the respondents raises two issues. First, respondents may have responded "yes" to this item even if they had only secured the job several days prior to the interview. We would not expect job stability to predict desistance for these respondents since it is most likely that social bonds take

\(^{13}\) The Add Health data collection team has recently announced that wave 4 data, which were collected between 2007 and 2008 (respondents' age range was 24-32 at wave 4), will be released soon.
time to influence behavior (Laub et al., 1998). Second, the employment-crime relationship is likely to be contingent upon age. For example, employment may have crime-reducing effects for adults (Laub and Sampson, 2003) but may have the opposite effect for adolescents (e.g., Apel et al., 2007). Thus, to the extent that wave 3 data have tapped adolescent employment experiences, the relationship between employment bonds and desistance may have been attenuated.

The third main limitation concerns the operationalization of desistance. This limitation is not restricted to the current analysis—scholars have debated the proper operationalization of desistance for decades (Bushway et al., 2001; Bushway et al., 2003; Fagan, 1989; Laub and Sampson, 2003; Le Blanc and Loeber, 1998; Maume et al., 2005; Paternoster and Bushway, 2009; Vaughan, 2007; Warr, 1998). The current study operationalized desistance as a discrete event where the individual reported involvement in delinquency or drug use at wave 1 but not at wave 3. Recall Le Blanc and Loeber's (1998:123) definition of desistance:

\[\text{a slowing down in the frequency of offending (deceleration), a reduction in its variety (specialization), or a reduction in its seriousness (de-escalation).}\]

The relevant boundary concept for all these subprocesses is the age at termination (emphasis in original). This definition makes reference to three concepts that can be thought of as comprising the desistance process. Specifically, if desistance is defined as the discrete event at which a person stops crime (i.e., the age at termination), then deceleration, reduced specialization, and de-escalation are defined as three elements that lead to the occurrence of the desistance event (i.e., they make up the desistance process). Since desistance includes elements such as deceleration and de-escalation, and since some scholars have argued that desistance cannot unequivocally be observed until the respondent has deceased (Laub and Sampson, 2003), researchers often measure changes in crime over time to tap desistance processes. In other words, researchers typically do not analyze direct measures of desistance (i.e., did the respondent desist or not), but instead estimate the impact of adult social bonds on changes in crime (i.e., deceleration or de-escalation).

In an effort to triangulate the findings and to incorporate the different concepts that make up the desistance process, three procedures were used in the current study. First, changes in crime and drug use were analyzed using a bivariate decomposition model (i.e., the Cholesky model) that allowed for the simultaneous estimation of genetic and environmental effects on stability and changes in behavior. Second, a direct measure of desistance was created and
regressed on the social bonds before and after genetic effects had been controlled. Third, a supplementary analysis was conducted where a measure of deceleration was created and regressed on each of the social bonds. Deceleration was operationalized slightly differently than proposed by Le Blanc and Loeber (1998) and was more in line with Bushway and his colleagues' definition (2001). Bushway et al. (2001:500) defined deceleration as "the process of reduction in the rate of offending from a nonzero level to a stable rate empirically indistinguishable from zero." For the current study, deceleration was measured as a decrease in the expected slope of delinquency from wave 2 to wave 3. Specifically, the rate of change in delinquency from wave 1 to wave 2 was first calculated. This rate of change was extended to predict the number of delinquent acts the respondent would report at wave 3. This prediction model was then compared to the respondent's observed number of delinquent acts at wave 3. If the respondent reported more delinquent acts than expected, he/she was assigned a 1 (acceleration in crime). Respondents who committed the same number of delinquent acts as predicted were assigned a 0 (no change in crime). Respondents who committed fewer delinquent acts than predicted were assigned a -1 (deceleration in crime). This new variable, therefore, measured within-individual changes in crime from wave 2 to wave 3. The final step was to regress the deceleration variable on each of the adult social bonds. These analyses failed to identify a significant relationship between deceleration in crime and the presence of an adult social bond.

The fourth limitation facing the current study was that it was unable to determine exactly which genes influence exposure to the adult social bonds, which genes lead to changes in crime, and which genes cause desistance from crime. The current study employed a family-based study to determine the proportion of variance in each of these outcomes that can be explained by genes. This study design, unfortunately, says nothing about the specific genes that are affecting the outcomes. Instead, all genetic effects are wrapped up into the latent heritability estimate. In order to determine the specific genes that are operating behind the heritability curtain, molecular genetic information is necessary. The Add Health data include information on five genes and a few studies have analyzed the link between these genes and some of the social bonds examined here (e.g., Beaver, Wright, DeLisi, and Vaughn, 2008a). Future researchers should explore the link between specific genetic factors, adult social bonds, changes in crime, and desistance from crime to facilitate a better understanding of the relationships identified in this dissertation.
The fifth limitation is that the Add Health research team did not make an effort to reduce the chances of siblings communicating about the survey before both siblings had completed the questionnaire (i.e., there is no indication that siblings were interviewed simultaneously, thereby eliminating the chance that they could communicate about the questionnaire). This may be problematic to the extent that MZ twins were more likely to communicate about the survey than were DZ twins or full siblings (and so on for full siblings and half-siblings and cousins). If genetic relatedness is correlated with the likelihood that siblings communicated about the survey prior to the completion of the survey by both siblings, then heritability estimates may be artificially inflated. This critique is similar to the EEA and rests on many of the same assumptions (i.e., that MZ twins are more likely to communicate about their survey experience than are DZ twins and that this greater propensity to communicate affects survey responses). For this reason, the body of research which has investigated the tenability of the EEA is informative. Recall from Chapter 2 that the EEA has been tested and has been shown to be a valid and tenable assumption (Goodman and Stevenson, 1991; Gunderson et al., 2006; Kendler, 1983; Kendler et al., 1993; Scarr and Carter-Saltzman, 1979). Based on these findings, it is unlikely that communication about the survey between siblings has affected the estimates. Nonetheless, future research should account for this possible biasing influence.

### 7.3 FUTURE DIRECTIONS

A growing body of evidence has revealed the importance of considering the genetic influences on antisocial behavior (Ferguson, 2010; Mason and Frick, 1994; Miles and Carey, 1997; Rhee and Waldman, 2002; Walters, 1992). A subset of this literature has revealed that genetic factors influence stability and changes in antisocial behavior over time (e.g., Reiss et al., 2000). This dissertation built upon the latter body of research in three important ways. First, the findings revealed that the emergence of adult social bonds in early adulthood is influenced (partially) by genetic factors. Thus, factors that are often hypothesized to be "purely" social in their effects (Sampson and Laub, 1993) are actually under genetic influence. Second, changes in delinquent, drug-using, and antisocial behaviors from adolescence to early adulthood are partially the result of changes in genetic factors/effects. Third, the effect of some adult social bonds on desistance from crime may be confounded by omitted genetic factors.
Although the current study is not without limitations (see the above section), these findings highlight two different courses of action that can be taken by criminologists examining factors across the life-course. First, scholars analyzing the factors that influence crime over the life-course can continue ignoring the emerging literature bearing on the importance of genetic influences on changes in behavior over time. This course of action will undoubtedly lead to "more of the same." In other words, ignoring the findings presented in this dissertation and those reported by previous researchers will only serve to reinforce the status quo. To be clear, the status quo refers to the paradigmatic focus of life-course criminology as purely social and operating outside of genetic influences.

The second course of action is that scholars can begin to consider the implications of omitting genetic factors from their models, even when the focus is on changes in behavior. Prior research has shown that genetic factors can explain changes in behavior and the current study has focused on one mechanism through which these genetic factors may operate (i.e., by affecting a person's probability of being exposed to an adult social bond – although the current study found little support for this argument with the exception of the relationship between marriage and desistance). The risk of choosing the former course of action is that criminologists may never realize a complete explanation of crime and delinquency over the life-course. (The worst case scenario is that criminologists will never identify "true" causal effects, only spurious effects).

The simultaneous estimation of genetic and environmental effects provides the opportunity for gaining a clearer understanding of crime and delinquency over the life-course. The findings from this dissertation hint at the possibility that genetic factors can explain some anomalous findings that have heretofore plagued criminologists. For example, Laub and Sampson (2003) briefly discuss the importance of "human agency" in determining one's likelihood of desistance from crime. The discussion is quite limited in regards to the clarity of their argument, but human agency appears to be a typical "black box" concept (i.e., a latent concept which captures unexplained variance in crime and especially desistance—in other words human agency appears to be a scholarly term for measurement error). Human agency, according to Laub and Sampson, is best understood as an immeasurable "will power" that is expressed by offenders who wish to change their life circumstances. Consider the following passage (Laub and Sampson, 2003:55):
Fortunately, as developed in more detail below, what is most striking in the narrative we collected is the role of human agency in processes of desistance from crime and deviance. The Glueck men are seen to be active players in their destiny, especially when their actions project a new sense of a redeemed self. One man told us how he felt when he left prison: "The heck with you [guards and others in authority]. I made a conscious effort—do my time and get the hell out. And don't come back."

Laub and Sampson believe that offenders are active participants in their lives and, thus, active participants in their decisions to desist from crime. By extension, this means that offenders are active participants in the construction of the social bonds to which they are exposed (or not exposed) in adulthood. As discussed by Laub et al. (2006), the difficulty of measuring human agency has precluded any direct tests to date. What Laub and colleagues failed to consider, however, is that gene-environment interplay may offer one way to open the black box of human agency.

Recall that active rGE provides an explanation of niche-picking (Scarr, 1992; Scarr and McCartney, 1983) by arguing that individuals create or select environments that fit their genetic predispositions. Findings from the current study suggest that genetic factors impact a person's likelihood of being exposed to social bonds in early adulthood. At the same time, genetic factors explained, partially, changes in delinquency and why some offenders desisted from delinquency and others did not. As compared to Laub and Sampson's (2003) concept of human agency, the active rGE framework is a more powerful explanation of changes in antisocial behavior over the life course.

A separate matter is whether the current findings can inform policy discussions and, if so, what types of policies might be recommended. A common misperception surrounding behavioral genetic research is that there are no policy implications, or that the policy recommendations flowing from this work are invasive and harmful. For example, the Nazi regime misinterpreted criminal-biological research to justify their eugenic and genocide policies (Rafter, 2008). Many of today’s scholars are fearful that behavioral genetic research will lead to similar types of policies. Interestingly, however, one can just as easily argue that the policy implications of the current study are purely social, and non-invasive. One of the more robust findings from this dissertation was that being married increases a person’s likelihood of desisting from crime. This finding remained even after genetic effects were controlled. A policy
recommendation flowing from the current study, therefore, could focus on the effects of marriage and how to strengthen the marital bond for offenders. Marriage counseling for prisoners soon to be released to the community might be a good place to start. Whatever the specific policy may be, the most important message to take away from this dissertation is that behavioral genetic research can be used to justify and support social policies that affect environmental factors (such as marriage). The interaction of genetic and environmental influences can be used to support many of the same policies already advocated for by "sociological" criminologists.

This dissertation represents one way that behavioral genetic research can be fused with criminological research to arrive at a clearer understanding of well-known, but poorly understood relationships and concepts, like that of human agency. In some cases genetic factors may explain away the association observed between an environmental factor and antisocial behavior. In other cases, genetic factors may weaken the association, and at other times, genetic factors may have no effect on the relationship. The most important point, however, is that criminologists will never know whether their models are omitting an important explanatory variable (i.e., genes) unless an effort is made to control for their effects. Until research similar to that presented here is conducted on a larger scale, scholars will continue to rely on the tenuous assumption that genes do not matter. This assumption is likely to prompt misinformed, misspecified, and, at best, incomplete theoretical statements. The weak foundation upon which this assumption rests in regards to Sampson and Laub's (1993) age-graded theory of informal social control has been elucidated by this dissertation. The extent to which other prominent theories of crime suffer the same deficit is an empirical question that will hopefully spur a new generation of research.
### Table 5.1.1. Levels of Genetic Relatedness for Add Health Pairs

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<th>Pair Type (% Genetic Similarity)</th>
<th>Individuals (Pairs)</th>
<th>Percentage</th>
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Table 5.2.1.1. Descriptive Statistics for Add Health Variables (Full Sample N=4,646)

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Note: All variables are measured at Wave 3 unless noted differently
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<th>Model 4</th>
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<td>Full M F</td>
<td>Full M F</td>
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<td>(.00-.11) (.00-.14)</td>
<td>(.09-.25) (.17-.48) (.00-.14)</td>
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†p<.10, two-tailed; *p<.05, two-tailed
Notes: CI = Confidence Interval; Logit coefficients presented as odds ratios; Full = full sibling sample; M = male-male subsample; F = female-female subsample
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<td>F 11.14</td>
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<td>95% CI (.68-32.72)</td>
<td>(.12-32.38)</td>
<td>(.13-967.76)</td>
<td>(.202-3.44)</td>
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<td>(2.03-6.65) (1.9-10.43)</td>
<td>(2.03-6.65) (1.9-10.43)</td>
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| Heritability         | Full 4.28*     | M 5.29†       | F 6.94†        | Full ---       |
|                      | 95% CI (1.29-14.17) (1.78-35.79) | (.78-35.79) | (.91-52.79) | --- |
| Shared Environment   | --- 1.90*      | --- ---       | --- ---        | --- --- ---    |

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<table>
<thead>
<tr>
<th>Intraclass Odds Ratio</th>
<th>MZ 13.00*</th>
<th>DZ/FS 14.04*</th>
<th>HS ---a</th>
<th>Cousin ---a</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ</td>
<td>3.45*</td>
<td>4.03*</td>
<td>3.78*</td>
<td>3.78*</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>1.62*</td>
<td>1.91*</td>
<td>1.93*</td>
<td>1.93*</td>
</tr>
<tr>
<td>HS</td>
<td>---a</td>
<td>---a</td>
<td>---a</td>
<td>---a</td>
</tr>
<tr>
<td>Cousin</td>
<td>1.45†</td>
<td>---a</td>
<td>.83</td>
<td>---a</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed
Notes: a. Model could not produce solution due to perfect prediction of "failure" on outcome; CI = Confidence Interval; Logit coefficients presented as odds ratios; Full = full sibling sample; M = male-male subsample; F = female-female subsample

154
Table 6.1.3. DF Models Predicting Life-Course Transitions (Employment)

<table>
<thead>
<tr>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education</td>
<td>Job</td>
<td>Annual</td>
<td>Hours</td>
<td>Composite</td>
</tr>
<tr>
<td>Level</td>
<td>Satisfaction</td>
<td>Income</td>
<td>Worked</td>
<td>Employment Scale</td>
</tr>
</tbody>
</table>

Heritability

<table>
<thead>
<tr>
<th>95% CI</th>
<th>.38*</th>
<th>.48*</th>
<th>.29*</th>
<th>.14*</th>
<th>.23*</th>
<th>.15*</th>
<th>.37*</th>
<th>.53*</th>
<th>.25*</th>
<th>.33*</th>
<th>.18</th>
<th>.27*</th>
<th>.34*</th>
<th>.43*</th>
<th>.29*</th>
</tr>
</thead>
<tbody>
<tr>
<td>(.25-.52)</td>
<td>(.25-.70)</td>
<td>(.10-.48)</td>
<td>(.06-.22)</td>
<td>(.11-.34)</td>
<td>(.02-.28)</td>
<td>(.23-.51)</td>
<td>(.41-.64)</td>
<td>(.05-.45)</td>
<td>(.26-.39)</td>
<td>(.00-.44)</td>
<td>(.17-.37)</td>
<td>(.26-.42)</td>
<td>(.28-.58)</td>
<td>(.17-.40)</td>
<td></td>
</tr>
</tbody>
</table>

Shared Environment

<table>
<thead>
<tr>
<th>95% CI</th>
<th>.33*</th>
<th>.25*</th>
<th>.43*</th>
<th>---</th>
<th>---</th>
<th>---</th>
<th>---</th>
<th>---</th>
<th>---</th>
<th>---</th>
<th>---</th>
<th>.15†</th>
<th>---</th>
<th>---</th>
<th>---</th>
</tr>
</thead>
<tbody>
<tr>
<td>(.24-.41)</td>
<td>(.41-.41)</td>
<td>(.31-.56)</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>(.00-.31)</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed

Notes: CI = Confidence Interval; Full = full sibling sample; M = male-male subsample; F = female-female subsample
Table 6.1.4. DF Models Predicting Life-Course Transitions (Community Service & Religion)

<table>
<thead>
<tr>
<th></th>
<th>Model 1 (Logit)</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
<th>Model 5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Community Service</td>
<td>Church Attendance</td>
<td>Importance of Religion</td>
<td>Religiosity</td>
<td>Composite Religion Scale</td>
</tr>
<tr>
<td>Heritability</td>
<td>Full</td>
<td>M</td>
<td>F</td>
<td>Full</td>
<td>M</td>
</tr>
<tr>
<td>95% CI</td>
<td>(2.68-4.73)</td>
<td>(.36-2.41)</td>
<td>(.00-.47)</td>
<td>(.20-.49)</td>
<td>(.00-.36)</td>
</tr>
<tr>
<td>Heritability</td>
<td>.35*</td>
<td>.65*</td>
<td>.13</td>
<td>.40*</td>
<td>.36*</td>
</tr>
<tr>
<td>95% CI</td>
<td>(.26-.109)</td>
<td>(.19-.47)</td>
<td>(.29-.55)</td>
<td>(.13-.30)</td>
<td>(.27-.53)</td>
</tr>
<tr>
<td>Shared Environment</td>
<td>.53†</td>
<td>.28*</td>
<td>.32*</td>
<td>.21*</td>
<td>.40*</td>
</tr>
<tr>
<td>95% CI</td>
<td>(.18-.402)</td>
<td>(.19-.47)</td>
<td>(.29-.55)</td>
<td>(.13-.30)</td>
<td>(.27-.53)</td>
</tr>
</tbody>
</table>

Probandwise Concordance (Percent)

<table>
<thead>
<tr>
<th></th>
<th>MZ</th>
<th>DZ/FS</th>
<th>HS</th>
<th>Cousin</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ</td>
<td>58/119</td>
<td>(.49)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DZ/FS</td>
<td>230/613</td>
<td>(.38)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td>28/100</td>
<td>(.28)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cousin</td>
<td>8/33</td>
<td>(.24)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Intraclass Tetrachoric Correlation

<table>
<thead>
<tr>
<th></th>
<th>MZ</th>
<th>DZ/FS</th>
<th>HS</th>
<th>Cousin</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ</td>
<td>.47*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DZ/FS</td>
<td>.21*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td>.16†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cousin</td>
<td>.06</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Intraclass Odds Ratio

<table>
<thead>
<tr>
<th></th>
<th>MZ</th>
<th>DZ/FS</th>
<th>HS</th>
<th>Cousin</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ</td>
<td>3.93*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DZ/FS</td>
<td>1.81*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS</td>
<td>1.74*</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Cousin</td>
<td>1.20</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed

Notes: CI = Confidence Interval; Logit coefficients presented as odds ratios; Full = full sibling sample; M = male-male subsample; F = female-female subsample
Table 6.2.1. Zero-Order Correlations Between Wave 1 and Wave 3 Delinquency, Drug Use, and Antisocial Behavior

<table>
<thead>
<tr>
<th></th>
<th>Delinquency Wave 3</th>
<th>Drug Use Wave 3</th>
<th>Antisocial Behavior Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full</td>
<td>.23*</td>
<td>.29*</td>
<td>.20*</td>
</tr>
<tr>
<td>M</td>
<td>.28*</td>
<td>.31*</td>
<td>.25*</td>
</tr>
<tr>
<td>F</td>
<td>.16*</td>
<td>.31*</td>
<td>.14*</td>
</tr>
</tbody>
</table>

*<.05, two-tailed
Notes: Full = full sibling sample; M = male-male subsample; F = female-female subsample
Table 6.2.2. Bivariate Cholesky Models Predicting Stability and Change in Delinquency from Wave 1 to Wave 3

<table>
<thead>
<tr>
<th></th>
<th>A Heritability</th>
<th>C Shared Environment</th>
<th>E Nonshared Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Sibling Sample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.77(.54-.99)</td>
<td>.00(.00-.00)</td>
<td>.23(.00-.46)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.21(.21-.33)</td>
<td>.02(.00-.13)</td>
<td>.76(.67-.87)</td>
</tr>
<tr>
<td><strong>Male-Male Subsample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.93(.63-1.00)</td>
<td>.00(.00-.00)</td>
<td>.07(.00-.37)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.37(.07-.58)</td>
<td>.05(.00-.24)</td>
<td>.57(.42-.77)</td>
</tr>
<tr>
<td><strong>Female-Female Subsample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.58(.07-1.00)</td>
<td>.00(.00-.00)</td>
<td>.42(.00-.93)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.15(.00-.25)</td>
<td>.00(.00-.13)</td>
<td>.85(.75-.95)</td>
</tr>
</tbody>
</table>

Note: 95% confidence interval in parentheses
Table 6.2.3. Bivariate Cholesky Models Predicting Stability and Change in Drug Use from Wave 1 to Wave 3

<table>
<thead>
<tr>
<th></th>
<th>A Heritability</th>
<th>C Shared Environment</th>
<th>E Nonshared Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Sibling Sample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.52(.15-.88)</td>
<td>.33(.10-.55)</td>
<td>.15(.00-.35)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.36(.18-.51)</td>
<td>.13(.03-.23)</td>
<td>.51(.43-.61)</td>
</tr>
<tr>
<td><strong>Male-Male Subsample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.93(.71-1.00)</td>
<td>.00(.00-.00)</td>
<td>.08(.00-.29)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.35(.09-.60)</td>
<td>.17(.00-.33)</td>
<td>.48(.36-.64)</td>
</tr>
<tr>
<td><strong>Female-Female Subsample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.51(.00-1.00)</td>
<td>.31(.00-.64)</td>
<td>.18(.00-.45)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.31(.07-.52)</td>
<td>.19(.04-.34)</td>
<td>.50(.40-.63)</td>
</tr>
</tbody>
</table>

Note: 95% confidence interval in parentheses
Table 6.2.4. Bivariate Cholesky Models Predicting Stability and Change in Antisocial Behavior from Wave 1 to Wave 3

<table>
<thead>
<tr>
<th></th>
<th>Heritability A</th>
<th>C Shared Environment</th>
<th>E Nonshared Environment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Sibling Sample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.74(.19-1.00)</td>
<td>.07(.00-.39)</td>
<td>.19(.00-.49)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.34(.16-.45)</td>
<td>.03(.00-.13)</td>
<td>.63(.55-.72)</td>
</tr>
<tr>
<td><strong>Male-Male Subsample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.92(.65-1.00)</td>
<td>.00(.00-.00)</td>
<td>.08(.00-.35)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.53(.38-.62)</td>
<td>.00(.00-.08)</td>
<td>.47(.38-.58)</td>
</tr>
<tr>
<td><strong>Female-Female Subsample</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factors accounting for stability</td>
<td>.65(.18-1.00)</td>
<td>.00(.00-.00)</td>
<td>.35(.00-.82)</td>
</tr>
<tr>
<td>Factors accounting for change</td>
<td>.02(.00-.17)</td>
<td>.28(.16-.34)</td>
<td>.71(.62-.78)</td>
</tr>
</tbody>
</table>

Note: 95% confidence interval in parentheses
### Table 6.3.1. DF Models Predicting Desistance from Delinquency, Drug Use, and Antisocial Behavior

<table>
<thead>
<tr>
<th></th>
<th>Delinquency (Logit)</th>
<th>Desistance from…</th>
<th>Antisocial Behavior (Logit)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Full M F</td>
<td>Drug Use (Logit)</td>
<td>Full M F</td>
</tr>
<tr>
<td>Heritability</td>
<td>1.86* 2.51* 1.86*</td>
<td>1.37 2.11† 1.54</td>
<td>1.09 3.54* .49</td>
</tr>
<tr>
<td>Shared Environment</td>
<td>--- --- ---</td>
<td>2.29* --- 2.98*</td>
<td>2.02* --- 4.32*</td>
</tr>
<tr>
<td>Probandwise Concordance (Percent)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>164/238 (.69)</td>
<td>20/59 (.34)</td>
<td>22/64 (.34)</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>700/1,177 (.59)</td>
<td>70/277 (.25)</td>
<td>88/344 (.26)</td>
</tr>
<tr>
<td>HS</td>
<td>134/251 (.53)</td>
<td>30/91 (.33)</td>
<td>26/91 (.29)</td>
</tr>
<tr>
<td>Cousin</td>
<td>52/82 (.63)</td>
<td>6/32 (.19)</td>
<td>14/38 (.39)</td>
</tr>
<tr>
<td>Intraclass Tetrachoric Correlation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>.38*</td>
<td>.44*</td>
<td>.41*</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>.10*</td>
<td>.29*</td>
<td>.20*</td>
</tr>
<tr>
<td>HS</td>
<td>-.11</td>
<td>.29*</td>
<td>.17†</td>
</tr>
<tr>
<td>Cousin</td>
<td>.19</td>
<td>-.09</td>
<td>.19</td>
</tr>
<tr>
<td>Intraclass Odds Ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>2.82*</td>
<td>4.23*</td>
<td>3.62*</td>
</tr>
<tr>
<td>DZ/FS</td>
<td>1.31*</td>
<td>2.62*</td>
<td>1.92*</td>
</tr>
<tr>
<td>HS</td>
<td>.84</td>
<td>2.51*</td>
<td>1.89*</td>
</tr>
<tr>
<td>Cousin</td>
<td>1.64</td>
<td>.85</td>
<td>2.11</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed

Notes: Logit coefficients presented as odds ratios; Full = full sibling sample; M = male-male subsample; F = female-female subsample
Table 6.4.1. SSSM and DF Logit Models Predicting Desistance from Delinquency (Relationships)

<table>
<thead>
<tr>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ever Married</td>
<td>Marital Love</td>
<td>Marital Commitment</td>
<td>Composite Marital Scale</td>
</tr>
<tr>
<td>SSSM</td>
<td>DF</td>
<td>SSSM</td>
<td>DF</td>
</tr>
<tr>
<td>----------</td>
<td>---------</td>
<td>---------</td>
<td>------</td>
</tr>
<tr>
<td>Full Sibling Sample</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>1.51*</td>
<td>1.28*</td>
<td>1.02</td>
</tr>
<tr>
<td></td>
<td>(1.25-1.82)</td>
<td>(1.10-1.48)</td>
<td>(.96-1.07)</td>
</tr>
<tr>
<td>Male-Male Subsample</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>2.29*</td>
<td>1.71*</td>
<td>1.11*</td>
</tr>
<tr>
<td></td>
<td>(1.60-3.29)</td>
<td>(1.26-2.32)</td>
<td>(1.01-1.21)</td>
</tr>
<tr>
<td>Female-Female Subsample</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>1.35†</td>
<td>1.29*</td>
<td>1.02</td>
</tr>
<tr>
<td></td>
<td>(1.00-1.82)</td>
<td>(1.02-1.64)</td>
<td>(.91-1.13)</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed
Notes: Logit coefficients presented as odds ratios; 95% confidence interval in parentheses
Table 6.4.2. SSSM and DF Logit Models Predicting Desistance from Delinquency (Military & Employment)

<table>
<thead>
<tr>
<th></th>
<th>Model 1 Military Involvement</th>
<th>Model 2 Employment Status</th>
<th>Model 3 Job Stability</th>
<th>Model 4 Two Jobs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SSSM</td>
<td>DF</td>
<td>SSSM</td>
<td>DF</td>
</tr>
<tr>
<td><strong>Full Sibling Sample</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>1.03</td>
<td>1.10</td>
<td>1.13†</td>
<td>1.10</td>
</tr>
<tr>
<td></td>
<td>(.68-1.56)</td>
<td>(.78-1.56)</td>
<td>(.98-1.31)</td>
<td>(.98-1.24)</td>
</tr>
<tr>
<td><strong>Male-Male Subsample</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>1.46</td>
<td>1.60†</td>
<td>1.14</td>
<td>1.10</td>
</tr>
<tr>
<td></td>
<td>(.80-2.65)</td>
<td>(.93-2.74)</td>
<td>(.86-1.50)</td>
<td>(.87-1.39)</td>
</tr>
<tr>
<td><strong>Female-Female Subsample</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>.53</td>
<td>.72</td>
<td>1.07</td>
<td>1.07</td>
</tr>
<tr>
<td></td>
<td>(.21-1.32)</td>
<td>(.33-1.56)</td>
<td>(.84-1.38)</td>
<td>(.88-1.32)</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed

Notes: Logit coefficients presented as odds ratios; 95% confidence interval in parentheses
Table 6.4.3. SSSM and DF Logit Models Predicting Desistance from Delinquency (Employment)

<table>
<thead>
<tr>
<th></th>
<th>Model 1 Education Level</th>
<th>Model 2 Job Satisfaction</th>
<th>Model 3 Annual Income</th>
<th>Model 4 Hours Worked</th>
<th>Model 5 Composite Employment Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SSSM</td>
<td>DF</td>
<td>SSSM</td>
<td>DF</td>
<td>SSSM</td>
</tr>
<tr>
<td>Full Sibling Sample</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>.99</td>
<td>1.01</td>
<td>.98</td>
<td>1.01</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>(.96-1.02)</td>
<td>(.97-1.05)</td>
<td>(.90-1.07)</td>
<td>(.93-1.10)</td>
<td>(1.00-1.00)</td>
</tr>
<tr>
<td>Male-Male Subsample</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>.97</td>
<td>1.01</td>
<td>1.04</td>
<td>1.06</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>(.92-1.04)</td>
<td>(.95-1.09)</td>
<td>(.88-1.22)</td>
<td>(.91-1.24)</td>
<td>(1.00-1.00)</td>
</tr>
<tr>
<td>Female-Female Subsample</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L.C. Transition</td>
<td>1.02</td>
<td>1.01</td>
<td>1.04</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>(.96-1.09)</td>
<td>(.97-1.11)</td>
<td>(.87-1.18)</td>
<td>(.87-1.14)</td>
<td>(1.00-1.00)</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed
Notes: Logit coefficients presented as odds ratios; 95% confidence interval in parentheses
Table 6.4.4. SSSM and DF Logit Models Predicting Desistance from Delinquency (Community Service & Religion)

<table>
<thead>
<tr>
<th></th>
<th>Model 1 Community Service</th>
<th>Model 2 Church Attendance</th>
<th>Model 3 Importance of Religion</th>
<th>Model 4 Religiosity</th>
<th>Model 5 Composite Religion Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SSSM</td>
<td>DF</td>
<td>SSSM</td>
<td>DF</td>
<td>SSSM</td>
</tr>
<tr>
<td>Full Sibling Sample</td>
<td>L.C. Transition</td>
<td>.93</td>
<td>1.06</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.80-1.08)</td>
<td>(.93-1.20)</td>
<td>(.97-1.04)</td>
<td>(.96-1.03)</td>
</tr>
<tr>
<td>Male-Male Subsample</td>
<td>L.C. Transition</td>
<td>.89</td>
<td>.96</td>
<td>1.03</td>
<td>.98</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.68-1.16)</td>
<td>(.77-1.21)</td>
<td>(.97-1.10)</td>
<td>(.92-1.05)</td>
</tr>
<tr>
<td>Female-Female Subsample</td>
<td>L.C. Transition</td>
<td>1.02</td>
<td>1.08</td>
<td>1.01</td>
<td>.98</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(.79-1.33)</td>
<td>(.86-1.34)</td>
<td>(.95-1.07)</td>
<td>(.92-1.04)</td>
</tr>
</tbody>
</table>

†p<.10, two-tailed; *p<.05, two-tailed

Notes: Logit coefficients presented as odds ratios; 95% confidence interval in parentheses
<table>
<thead>
<tr>
<th></th>
<th>Relationships</th>
<th>Military &amp; Employment</th>
<th>Community Service &amp; Religion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of significant (h^2) estimates over the number of models estimated</td>
<td>9/12</td>
<td>21/27</td>
<td>9/15</td>
</tr>
<tr>
<td>Average (h^2) estimate(^{a})</td>
<td>.10</td>
<td>.31</td>
<td>.27</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Delinquency</th>
<th>Drug Use</th>
<th>Antisocial Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>(h^2) estimate for stability(^{a})</td>
<td>.77</td>
<td>.52</td>
<td>.74</td>
</tr>
<tr>
<td>(h^2) estimate for change(^{a})</td>
<td>.21</td>
<td>.36</td>
<td>.33</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Relationships</th>
<th>Military &amp; Employment</th>
<th>Community Service &amp; Religion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of significant (h^2) estimates over the number of models estimated (Desistance)</td>
<td>3/3</td>
<td>1/3</td>
<td>1/3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Relationships</th>
<th>Military &amp; Employment</th>
<th>Community Service &amp; Religion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of significant social bond effects (SSSM) over the number of models estimated</td>
<td>8/12</td>
<td>1/27</td>
<td>3/15</td>
</tr>
</tbody>
</table>

| Number of significant social bond effects after genes controlled (DF Model) | 4/8 | 0/1 | 0/3 |

Note: \(^{a}\) \(h^2\) estimate calculated using the full sibling sample estimates and does not include estimates from DF Logit models; Unless otherwise noted, numbers were calculated by combining full sibling sample, male-male subsample, and female-female subsample estimates.
Figure 2.1.1.1. Heuristic Data Showing the Intraclass Correlations for MZ and DZ Twins on a Phenotype that is Completely Explained by Genetic Influences.
Figure 2.2.1. Estimates of Heritability and Environmental Influences on Antisocial Behaviors from Four Meta-Analyses.
Figure 2.3.1.1. Graphical Depiction of the Double Helix. Copyrighted by the National Health Museum. Available Online at http://www.accessexcellence.org/RC/VL/GG/dna.html
Figure 2.3.1.2. Graphical Depiction of a GxE.
Figure 2.3.1.3. Graphical Depiction of "Plasticity" Alleles.
Figure 3.1.1. Age-Crime Curve for Total Number of Offenses Committed in All Categories in 2008 (U.S. Federal Bureau of Investigation, 2009).
Figure 5.3.1.1. Balloon Diagram of the Univariate ACE Model
Figure 5.3.2.1. Balloon Diagram of the Bivariate Cholesky Model
Figure 6.1.1. Cascade of Tetrachoric Correlations for Married Ever Variable
Figure 6.1.2. Cascade of Tetrachoric Correlations for Employment Variables
Figure 6.1.3. Cascade of Tetrachoric Correlations for Community Service Variable
Figure 6.2.1. Genetic and Environmental Influences on Stability and Change in Delinquency, Drug Use, and Antisocial Behavior
Figure 6.3.1. Cascade of Tetrachoric Correlations for Desistance Variables
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Yang, Yaling, Adrian Raine, Todd Lencz, Susan Bihrlle, Lori LaCasse, and Patrick Colletti.

BIOGRAPHICAL SKETCH

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J.C. Barnes received his Master's degree in Criminology and Criminal Justice from the University of South Carolina in 2006. In the fall of 2007, he began work on his Ph.D. at The Florida State University. He was recognized as the Graduate Student of the Year by the Southern Criminal Justice Association in 2008. His research has covered a range of topics—from laws governing sex offenders to the biosocial underpinnings to adolescent delinquency and victimization. In the fall of 2010, he will begin a position as Assistant Professor of Criminology at The University of Texas at Dallas.