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TESTING MOFFITT’S ADOLESCENCE LIMITED AND LIFE-COURSE-PERSISTENT TAXONOMY UTILIZING A BEHAVIORAL GENETIC DESIGN: AN ADOPTION STUDY OF ADOLESCENT ANTISOCIAL BEHAVIOR

by

Michael Scott Gilson

A Dissertation Submitted to the faculty of the SCHOOL OF FAMILY AND CONSUMER SCIENCES In partial Fulfillment of the Requirements For the Degree of DOCTOR OF PHILOSOPHY WITH A MAJOR IN FAMILY STUDIES AND HUMAN DEVELOPMENT In the Graduate College THE UNIVERSITY OF ARIZONA

2002
As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Michael Scott Gilson entitled Testing Moffitt's Adolescence Limited and Life-Course-Persistent Taxonomy Utilizing a Behavioral Genetic Design: An Adoption Study of Adolescent Antisocial Behavior

and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy

Final approval and acceptance of this dissertation is contingent upon the candidate's submission of the final copy of the dissertation to the Graduate College.

I hereby certify that I have read this dissertation prepared under my direction and recommend that it be accepted as fulfilling the dissertation requirement.

Dissertation Director Jennifer L. Maggs
STATEMENT BY AUTHOR

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ABSTRACT

The notion of adolescent antisocial behaviors being committed by qualitatively different adolescents, those who engage in antisocial behavior only during adolescence due to social influences and pressures and those who persist in antisocial behaviors throughout the lifespan due to pathological characteristics, is quite popular though not previously empirically tested. The present study tested Moffitt’s (1993) dual taxonomy of antisocial behavior utilizing a full adoption design. The sample used in this study came from Cadoret’s Iowa Adoption Studies, 1975-1982 (Cadoret, 1988). Parent reports of antisocial behaviors of adoptees in this sample (N = 387) were utilized to classify adoptees as either Adolescence Limited (AL) (n = 115) or Life-Course-Persistent (LCP) (n = 62). Central questions examined in this study were: 1) Are AL and LCP individuals independent of biological history of either psychopathology or antisocial personality?; 2) Is there a differential genetic influence on AL and LCP individuals?; and, 3) Does genetic influence differ by domain of antisocial behavior examined? Analyses indicated that AL and LCP classification was not independent of biological history of either psychopathology or antisocial personality disorder. Hierarchical regression analyses consistently indicated that AL and LCP classification predicted both parent reports of antisocial behavior and clinical assessments of adoptee antisocial personality. While AL/LCP Classification × Biological History interactions were not significant, logistic regression analyses consistently indicated that LCP individuals were significantly more likely to have a biological parent with a history of either psychopathology or antisocial
ABSTRACT – continued

personality than were AL individuals. Further support for the normative nature of AL antisocial behaviors was demonstrated by the finding that AL individuals were no more likely than those adolescents who did not engage in any antisocial behaviors to have a biological parent diagnosed with either psychopathology or antisocial personality.

Analyses by domains of antisocial behavior revealed no significant difference between groups for aggressive behaviors but that LCP individuals were more likely to engage in substance use during adolescence than were AL individuals. Discussion focuses on the implications that the findings have for both subsequent research and intervention programs.
CHAPTER I
INTRODUCTION

Developmental researchers commonly ascribe different meanings to similar behaviors, depending upon the stage of development in which the acts are committed. The notion of developmentally appropriate behavior is as applicable to the study of antisocial behavior as to the more commonly considered topics of cognition and motor skills. Consider, for example, a case where a 15-year-old individual defaces public property and a case where a 30-year-old commits an identical act. Simply knowing the age of the individuals involved would probably lead laypersons and developmental researchers alike to reach separate conclusions about the causes, severity, and meaning of each offense. While perhaps unfortunate in the case of the teenager, such behaviors are perhaps more understandable and better tolerated. However, in the case of the 30-year-old, the motivation and appropriateness are far more suspect. Placing delinquent behavior in a developmental framework is a view that is readily embraced by not only the general public but the legal system as well. Both have little difficulty meting out differential opinion and punishment.

Recently, however, developmentalists have gone further, arguing that two 15-year-olds who commit an identical delinquent behavior may do so for very different reasons: one adolescent may do so to fit in with peers and one may do so because of pathological characteristics. Understandably, behaviors committed due to these different motivations may hold profound and long lasting developmental consequences. It is at the
heart of this paper that the existence of different "types" of delinquent individuals should be empirically tested and the etiology of these different groups examined.

A popular notion in the developmental literature is that different types of delinquent individuals can be readily identified by such features as age of onset and rates of desistance (Aguilar, Sroufe, Egeland, & Carlson, 2000; Bartusch, Lynam, Moffitt, & Silva, 1997; DiLalla & Gottesman, 1989; McDermott & Nagin, 2001; Moffitt, 1993; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996; Patterson, Forgatch, Yoerger, & Stoolmiller, 1998). Most commonly, these developmental theories specify that some individuals confine their delinquent behavior to adolescence whereas others engage in delinquency at earlier ages and persist throughout their lifespan. Perhaps the two most prominent examples of this theorizing are described in papers by DiLalla and Gottesman (1989) and Moffitt (1993). Each of these papers argues for the presence of qualitatively different individuals engaging in antisocial behavior and each of these will be presented in some detail later in the literature review in Chapter 2. Moffitt's (1993) developmentally based taxonomy for antisocial and delinquent behavior, in particular, has generated intense interest among social and behavioral scientists. Despite the wide popularity of this taxonomy, the very existence of different antisocial or delinquent groups has been little tested. Further, with the notable exception of a paper by Taylor, Iacono, and McGue (2000) the potential to use genetically informed designs as a means to test the existence of a developmentally based taxonomy has been ignored.

The present study uses data from the Iowa Adoption Studies (Cadoret, 1988) to empirically examine this taxonomy. First, this study will seek to identify two groups of
delinquent individuals: a group that limits delinquent behavior to adolescence and a group that both initiates delinquency prior to adolescence and persists in delinquent behavior into adulthood. Having identified these two groups, the present study will seek to examine the etiology of delinquent behavior. A review of the literature shows that the hypothesized existence of different groups of delinquents depends upon a different etiology for each group. A behavioral genetic design, specifically the adoption design, will be employed using a sample of adoptees to test this theory. The biological influence of delinquent behavior for each group will be identified, with the aim of testing whether these two groups are qualitatively different from one another with one group being affected by more environmental influences and the other by more genetic influences.

This paper will now discuss genetic influences, first describing what is meant by heritability. Three methods that behavioral genetic researchers frequently use to obtain estimates of heritability will then be presented.

**Heritability**

A key feature of most behavioral genetic designs is the ability to estimate heritability. Heritability is a concept that is easy enough to define yet is frequently misunderstood. Heritability may be expressed by the following simple equation:

\[ h^2 = \frac{V_g}{V_p}, \]

where \( h^2 \) is the symbol for heritability, \( V_g \) represents genotypic variance and \( V_p \) represents phenotypic variance. Thus, this heritability coefficient, \( h^2 \), is a statistic that
describes the ratio of genetic to phenotypic variance or the proportion of phenotypic variance explained by genetic variance. Phenotypic variation is simply variation in an observable trait or the "measurable, expressed outcome of development" (Rowe, 1994; p. 30). Genotypic variation is variation in the exact genetic makeup of an individual. The degree to which a phenotype is influenced by the underlying genotype provides an index of how heritable a trait is. Necessarily, valid heritability values may range from 0 to 1.0. A heritability coefficient of 0 indicates that variation in an observable characteristic or behavior (phenotype) is not influenced by genetic factors. As \( h^2 \) increases, genetic effects are increasingly responsible for the variation in observable characteristics.

It is important to recognize the limitations of what heritability can tell us about the genetic predisposition of phenotypic traits. Many of the misunderstandings surrounding the concept of heritability are created when these limitations are forgotten. Plomin and colleagues (Plomin, DeFries, McClearn, & Rutter, 1997) remind us of the following three limitations: 1) heritability is neither constant nor immutable; 2) heritability does not refer to one individual; and 3) heritability is like any other statistic in that it is not absolutely precise. Each of these limitations will now be briefly considered.

First, it is important to remember that heritability is neither constant nor immutable. That is, heritability does not exist in a vacuum free from non-genetic effects. This is important because, despite the lack of an explicit acknowledgement of environmental influences, the above equation implicitly acknowledges the presence of environmental influences (very broadly defined) as those that remain after genetic effects are considered. A necessary corollary to this statement then is that heritability estimates
may certainly change over time as an environment changes. An example of this may be readily obtained by looking at studies of intelligence. Numerous studies have found intelligence to have a heritable component with most contemporary studies estimating the heritability of intelligence from between 47% and 58% (Chipauer, Rovine, & Plomin, 1990; Loehlin, 1989). These estimates were derived from a number of studies that included children as well as adults. Perhaps the most interesting finding arising from the study of the heritability of intelligence, at least to a developmentally minded researcher, is that a number of studies have also demonstrated that the effects of shared environment are substantially reduced when an individual reaches adolescence (Jensen, 1998).

Clearly, estimates obtained from behavioral genetic studies are different across development with the accompanying changes in environment and saliency of genetic characteristics. Neale and Cardon (1992) further remind us that heritability can only account for the sources of variation and that heritability by itself cannot reliably account for the processes that lead to the mean level of a phenotype itself. Thus, the expression "height is genetic" would be incorrect. Rather, a high heritability coefficient for height actually indicates that individual differences in height are mainly genetic.

The second limitation that must be remembered is that heritability does not refer to any one individual. Rather, heritability is a descriptive statistic that refers to the population sampled. Continuing with the above height example, a heritability coefficient of $h^2 = .80$ does not mean that 80% of a person's height is genetic and the remainder is due to environmental influences such as nutrition or heels of footwear. Instead, this high heritability coefficient indicates that 80% of the deviation from the mean may be
attributed to genetic effects and 20% to various environmental influences. Lastly, the third limitation that must be remembered is that heritability is no different than any other descriptive statistic in that it contains error. Like other statistics, heritability estimates are sensitive to sample size, measurement error, and other sources of bias and random error.

The first assertion made in this section was that heritability was a very simple concept that could be expressed by a simple equation. While technically correct, this statement requires some modification, needing to be qualified by saying that there are two common types of heritability that may actually be estimated. These are determined by the methods that one uses to estimate heritability. The two types of heritability are broad-sense heritability and narrow-sense heritability. The differences between the two will now be briefly considered.

Broad-sense heritability refers to the sum of all sources of genetic variance — both additive and non-additive. Narrow-sense heritability refers to only additive sources of genetic variance. Additive sources of genetic variation may be thought of in terms of gene “dosage”, that is the more overall genetic similarity, the greater the overall similarity in phenotypic expression. Non-additive sources of genetic variation come from two general sources: dominance effects and epistasis effects. Dominance effects were initially discovered in Mendel’s classical experiments with pea plants. He discovered that the cross of two pure breeding lines of plants often resulted in an offspring that resembled one parent and not the other. This means that if you crossed a “yellow” plant with its homozygous alleles (AA) for color with a “green” plant (aa) the offspring (necessarily Aa) would not be some intermediate color but rather the ‘A’ allele for
"green" would be dominant and expressed in the phenotype and the 'a' allele for "yellow" would be recessive and not expressed. This pea plant study is equally applicable to some human traits as it is to legumes. For example, we know that in the human genotype that the allele for blue eyes is recessive so, for those who have this eye color, it is known that they are necessarily homozygous recessive for this trait.

Epistasis refers to the interaction between gene loci (Plomin, DeFries, McClearn, & Rutter, 1997). This means that the expression of a gene is dependent upon the expression of another gene. In the presence of epistasis of two genes, we would not be able to predict the expression of one gene without knowledge of how the other gene is expressed. This is extremely important to remember when we consider that many characteristics are polygenic or dependent upon the expression of multiple genes (Rowe, 1994).

The only design that allows for the estimation of broad-sense heritability is the twin study. Adoption designs and family studies, with the notable exception of when sufficient numbers of identical and fraternal twins are included, are unable to provide an estimation of the non-additive genetic effects included in broad-sense heritability. Each of these three designs will now be described, ending with the adoption design which will be used in the proposed analyses.

The Twin Design

The twin design represents the most common behavioral genetics design. Central to this design is the fact that two types of twins exist: identical or monozygotic (MZ) twins and fraternal or dizygotic (DZ) twins. MZ twins are twins conceived from the
same fertilized egg that has split into two organisms and thus, MZ twins share 100% of their genes with their co-twin. DZ twins are the result of the release of two separate eggs by the mother that are fertilized by two different sperm cells. They thus share, on average, 50% of their genes with their co-twin, the same amount as full siblings. The twin design compares the resemblance of MZ twins for a particular trait with that of the resemblance of the same-sex DZ twins. It is inferred that if heredity affects a particular trait, MZ twins should be more similar for that trait than are DZ twins. We may obtain an estimate of heritability by simply taking the difference between the MZ and DZ correlations and doubling this: \( h^2 = 2[(r_{MZ} - r_{DZ})] \) (Rowe, 1994).

Behavioral genetic designs tell us much more than just heritability of a trait however; they also partition variance in the environment into two parts, shared (\(c^2\)) and nonshared (\(e^2\)) environments. Shared environment concerns features in the environment that make individuals similar to one another. An example of shared environment includes the number of books in a home that are available to both siblings. Non-shared environment refers to aspects of the environment that make individuals different from one another. An example of non-shared environment includes an accident or injury. In the twin design, differences within pairs of MZ twins are due only to non-genetic factors that are not shared by twins. This is because members of MZ twin pairs are genetically identical with one another. Some examples of twin correlations may prove useful in illustrating how variation may be partitioned between genetic, shared, and nonshared environment. Assume that for trait X, MZ twins correlate .75 and DZ twins correlate .50. For trait Y, the MZ and DZ correlations are \( r = .50 \) and \( r = .25 \), respectively. We may
state that for both of these traits $h^2$ is equal to .50 as it is twice the difference between MZ and DZ twins. In our above example, $(.75-.50) \times 2$ and $(.50-.25) \times 2$ are both equal to .50. We may obtain an estimate of nonshared environment by taking the amount that MZ twins are discordant for a particular trait – again the logic being that any difference in MZ twins must be due to non-genetic factors. So, for trait X the amount of non-shared environment is equal to $1.0 - .75$ or $e^2 = .25$. For trait Y, the amount of non-shared environment is equal to $1.0 - .50$ or $e^2 = .50$. The remainder of the variance is considered to be the effect of shared environment as the sum of shared, nonshared, and genetic estimates must sum to 1.0. For trait X, this leaves us with a shared environment estimate of $c^2 = .25$. Applying the same logic, we estimate that trait Y has no shared environment component or $c^2 = 0$.

The twin study methodology is frequently questioned for two reasons. First, there is the very real concern that assortative mating, also termed homogamy, may raise a DZ correlation and that the presence of nonadditive genetic effects will lower the DZ correlation. Assortative mating is the idea that couples do not randomly mate but rather individuals who are similar on specific traits may be more likely to pair (Karkowski, 2000). Indeed, an abundance of evidence for this phenomenon exists. For example, assortative mating appears to take place on the basis of leisure interests, role performance, and social characteristics (Houtes, Robins, & Huston, 1996). Neale and Cardon (1992) state that within humans there is usually some spousal correlations between phenotypes as varied as religion, education, and SES. Research has recently found that assortative mating is in evidence with respect to antisocial behaviors (Krueger,
Moffitt, Caspi, Bleske, & Silva, 1998). From an evolutionary perspective or what Dawkins (1990) would call a "selfish gene" perspective, assortative mating serves the purpose of increasing an individual's genetic representativeness in the next generation by increasing their genetic similarity with their offspring. The extent to which a DZ twin is more than 50% genetically related to their co-twin is an indication of the amount of assortative mating. As previously discussed, nonadditive genetic effects of dominance and epistasis are known to exist. Identical twins, because they are genetically identical, are said to provide a broad-sense heritability estimate and contain these effects. The presence of these non-additive genetic effects may be one of the main reasons that studies of twins may provide us with different heritability estimates than do other behavioral genetic methods (Plomin, DeFries, McClearn, & Rutter, 1997).

Another concern frequently cited with twin studies is the presence of a special "twin environment." The concern here is that twins may not be representative of the rest of the non-twin populations and that the "equal environments" assumption made by BG researchers is a fallacy. Critics using this line of reasoning state that identical twins experience more similar family environments than do fraternal twins. Diana Baumrind (1993) in her response to Sandra Scarr (1992) states that the equal environment assumption is "untenable" as parents treat MZ twins more similarly and MZ twins dress more alike and spend more time together. Hopper (1992) echoed this sentiment stating that the equal environments assumption is merely a convenience that has rarely been addressed and then only on a superficial level. These concerns, however, do not appear to represent a major threat to the twin design as the equal environments assumption has
been extensively examined in the context of misclassified twins, in direct assessment of environmental differences, and in tests of whether differential environments affect behavior. While these studies have consistently found that MZ twins do experience slightly more similar environments than DZ twins (e.g., Loehlin & Nichols, 1976; Rose, Kaprio, Williams, Viken, & Obremski, 1990), the effects of this differential environment were negligible (e.g., Kendler, Neale, Kessler, Heath, & Eaves, 1994), indicating strong support for the equal environment assumption. Indeed empirical studies consistently indicate that environmental variables that were unequal for identical and fraternal twins simply did not make a difference in cognition, personality, vocational interests, or interpersonal relationships (Kendler, 1983; Plomin, DeFries, McClearn, & Rutter, 1997; Plomin, Willerman, & Loehlin, 1976).

The Family Design

While less often utilized by the behavioral geneticist, the family study design allows researchers to obtain estimates of heritability when data from family members of differing degrees of relatedness are available. Like the twin design, the family design also permits variation to be partitioned into the same three components of heritability, shared environment and non-shared environment. A frequent criticism of the family study is that the inability to experimentally manipulate humans results in differential environmental influences that render estimates unreliable (e.g., Hoffman, 1991). Nevertheless, the family design has repeatedly proven to be useful in estimating limits of genetic and environmental influences (Plomin, DeFries, McClearn, & Rutter, 1997). Further, by utilizing a family study design that is able to include all levels of siblings
rather than focusing solely on twins, researchers are able to take advantage of the existence of a number of different levels of sibling relatedness. The family study design, because it does not rely on the relatively rare twin or adopted participants, is often the behavioral genetic design that allows for the inclusion of the most participants.

A concept central to the family study design is that of the coefficient of relatedness or $r$. This coefficient refers to the fraction of genes in two individuals that are identical by descent, averaged over all loci (Wilson, 2000). Identical twins or monozygotic (MZ) twins, as mentioned previously, share all of their genes with one another so their coefficient of relatedness is 100%, more commonly expressed as $r = 1.0$. Fraternal or dizygotic (DZ) twins as well as full siblings share, on average, half of their genes with their sibling so the coefficient of relatedness is $r = .50$. This same degree of relatedness, $r = .50$, is also found between the parent and child. Half-siblings share one-quarter of their genes with their sibling, or $r = .25$. The coefficient of relatedness may be extended to any biological relative by use of a pedigree and taking the product of the paths in the same way that one would calculate a correlation between two variables using path analysis. Thus, individuals share, on average across all gene loci, 1/8 of their genes with their great-grandfather. This is because they are related to their great-grandfather by $0.50(\text{father}) \times 0.50(\text{grandfather}) \times 0.50(\text{great-grandfather}) = 0.125$ or 1/8. The family study method may be applied to the study of trait heritability by the observation of intraclass correlations among sibling pairs. An intraclass correlation merely refers to the correlation within a particular level of kinship. If a trait is heritable, the family study method will detect and estimate it based on the fact that genetically related individuals
ought to be similar phenotypically for any behavior that is influenced genetically. A heritable pattern of these intraclass correlations shows that as the coefficient of relatedness increases, the intraclass correlation of sibling pairs increases. Further, the family study provides an estimate of the percentage of phenotypic variation that is attributable to genetic variation. Specifically, the intraclass correlation of full siblings must be multiplied by 2 to obtain an estimate of heritability or $h^2$. Similarly, the intraclass correlation of half-siblings must be multiplied by 4 to obtain an estimate of $h^2$.

The basic problem of the family study design is that environmental resemblance often covaries with genetic relatedness such that relatives who are closer genetically may also reside in environments that are more similar. For this reason family studies that use animals are much better at obtaining precise estimates of heritability and environment. This is because aspects of both the environment and genetics may be tightly controlled in animal studies whereas such controls in the study of humans are simply not feasible. Nevertheless, by focusing a human family study on siblings rather than across generations a greater degree of similarity may be assumed in the environment. This is because siblings typically grow up in the same home and develop at the approximate same time in history. Further evidence of the validity of the family study design may be garnered from the observation that numerous behavioral genetic studies that have employed the family study design with humans have produced results that are consistent with other methods such as the twin and the adoption design.
The Adoption Design

The last design frequently used by behavioral geneticists is the adoption design. It should first be noted that there are actually a number of variations on the adoption design. This paper will explain two of these designs, the separated twin design and the design used in this study, the full adoption design. Each of these designs takes advantage of what could be referred to as an experiment of nature (Plomin, DeFries, McClean, & Rutter, 1997) where children are reared in an environment by parents with whom they are typically not biologically related.

The separated twin design, while relatively rare, is a design that should be familiar to many, as stories of twins separated at birth seem to capture the public’s fancy. In this design, participants are twins who are adopted into different homes at or shortly after birth. This design is perhaps the most intuitive adoption design to understand and is one where genetic effects can be directly estimated. Consider an instance where MZ twins are adopted separately at birth and are then reared apart in completely uncorrelated environments. Because the environments in which they are reared are unrelated, any degree of phenotypic correlation between these genetically identical twins must be attributed to broad-sense genetic effects alone. Thus, a correlation of $r = .40$ for a twin pair on a particular trait indicates that 40% of the phenotypic variation is genetic or $h^2 = .40$. This means that the remaining 60% of the phenotypic variation must be attributed to non-genetic or environmental sources. These environmental sources are again very broadly defined and may include anything from prenatal environment to aspects of the individual neighborhoods. Because it is relatively rare that twins are separated at birth,
few studies have utilized this particular design (see Bouchard, Lykken, McGue, Segal, & Tellegen, 1990 for an exception). However, the logic of the design may be applied to another more common form of the design, the full adoption design.

The full adoption design requires that information about the biological parents is available. Formerly, when adoption records were closed, information about biological parents was difficult to obtain. However, adoptions are becoming more open and information of this sort is increasingly available (Cadoret, 1986; Rowe, 1994). Unlike the family study and the twin design, the full adoption design, as used in the present study, does not permit portioning of variance into estimates of heritability, shared environment and non-shared environment. This is because the present study involves only one child per family. This precludes the possibility of deriving simultaneous estimates of $h^2$, $c^2$ and $e^2$ from siblings with different coefficients of relatedness. What the design provides is a direct estimate of genetic influence and a direct estimate of environmental influence. This is because this design essentially creates two separate families for the adopted child. The first is the biological family. Any resemblance between biological parents and their adopted away offspring is solely genetic as they do not share environmental influences but have a coefficient of relatedness of $r = .50$. The second family created is the adopted family. Any similarity between the adoptive parent and the adoptee is solely the result of environmental influences as they have a coefficient of relatedness $r = 0$.

Unfortunately, for the purpose of research, adoption studies are subject to biases introduced by selective placement. Many adoption agencies consciously match adoption
parents with biological parents along dimensions of IQ or SES (Rowe, 1994). If selective placement exists, it represents a potential bias for estimates of both genetic and environment variance. Adoptees may be placed into families that are more environmentally as well as genetically similar to their family of biological origin. This means that in order to obtain valid estimates of hereditability and environment selective placement must be taken into account. One way of doing this is to compare biological families matched for characteristics relevant to the trait studied. Genetic effects may be estimated by subtracting the correlation of unrelated pairs from those of related pairs in the matched families (Rowe, 1994). This may not be as easy to do as it sounds, as the danger in this is that obtaining a good match is not easily determined. A far better way, and an option readily available in the full adoption design, is to determine and control for the degree of selective placement by obtaining relevant information from the biological parents.

Before the present study is further explained, however, it is important to first describe the literature on developmental approaches to antisocial behavior as well as behavioral genetic research on antisocial behavior that provides the foundation for the present study. First, this paper will briefly define what is meant by antisocial behavior and other similar terms frequently used in the literature. Next, this paper will consider the initial arguments for a taxonomy of delinquents who are qualitatively different from one another. Finally, the literature review will consider the subsequent body of research on delinquency leading up to the initial behavioral genetic examinations of a developmental theory of delinquency.
CHAPTER II
LITERATURE REVIEW

Some confusion exists with regards to what is precisely meant by antisocial behaviors. Wolf (1987) states that some of this confusion arises from the rather broad definitions of antisocial that are available from most dictionaries. He further argues that this confusion is amplified when the term is considered from the different lenses of psychiatry, psychology, sociology, and law. Another difficulty inherent in defining antisocial behavior is the necessary reliance upon social norms to frame what is and what is not antisocial. Antisocial behavior can be defined as any behavior that violates one or more social norms beyond the tolerance limits of the social system (Wolf, 1987).

Antisocial behavior, when discussed in the adolescent literature, refers to a wide range of behaviors that typically includes items ranging from the rather innocuous “often argues with adults” to the more serious “initiates physical fights” (e.g., Ge et al., 1996).

Criminality and delinquency represent clear-cut examples of antisocial behaviors. In the case of both criminal and delinquent behaviors, the social norms violated have been anticipated and legal consequences specified. While not all antisocial behaviors can be classified as either delinquent or criminal, it is important to note that delinquent behaviors, by definition, are reserved for antisocial behaviors committed by individuals under the age of 18. Identical behaviors committed by adults are referred to as criminal behaviors. Given that the legal system must reach a general consensus about what constitutes criminal or delinquent behavior, it is hardly surprising that many studies use these as indicators of antisocial behavior. For example, Moffitt (1993) consistently refers
to both antisocial behaviors and antisocial persons. However, she frequently relies upon
delinquent behaviors and delinquency to operationalize what she means by these terms
and at times uses delinquent as a synonym for antisocial. Due to the focus of the present
study on behaviors through the adolescent years, the construct of antisocial behavior will
be operationalized primarily as delinquent behavior.

Moffitt’s Adolescence-Limited and Life-Course-Persistent Taxonomy of Antisocial
Behavior

Perhaps the best known application of developmental theory to the study of
antisocial behavior is presented in a theoretical piece by Moffitt (1993). A quick review
of the literature shows that this paper has been quite popular, having been cited
approximately 520 times in the nine years since publication. The argument put forth by
Moffitt is that two types of antisocial behaviors are committed by two types of
individuals – those who limit their antisocial behavior to adolescence are referred to as
adolescent limited (AL) antisocial individuals, and those who persist in antisocial
behavior across their lifespan, referred to as life-course-persistent (LCP) antisocial
individuals. Moffitt contends that these two groups are fundamentally different from one
another and that AL antisocial behavior and LCP antisocial behavior each have their own
unique theoretical explanation. AL antisocial behavior is viewed as a phenomenon
arising from social pressures and LCP antisocial behavior is a function of individual traits
or characteristics.

Experimentation with risk behaviors during adolescence is increasingly perceived
as a normative component of development that serves both constructive as well as
destructive functions (Chassin, Presson, & Sherman, 1989; Jessor, 1992; Maggs, 1997; Maggs, Almeida, & Galambos, 1995; Moffitt, 1993; Shedler & Block, 1990; Silbereisen & Noack, 1988). Adolescent limited (AL) antisocial behavior, like a number of other adolescent risk behaviors, may be both normative and serve a constructive social function. A cursory examination of the research suggests this may be so. First, ample evidence exists that antisocial behaviors occur with normative regularity during adolescence (Arnett, 1992). For example, Gold and Petronio (1980) reported that 80% of adolescents engage in delinquent behavior. Indeed, delinquent behaviors are so pervasive during adolescence that Seidman (1984) claimed that they simply represent behaviors that are part of a behavioral repertoire and comprise a normative developmental phase for adolescents. This claim is bolstered by FBI arrest records that show arrest rates increase considerably during early adolescence and fall off precipitously by late adolescence (Blumstein, Cohen, & Farrington, 1988; Gottfredson & Hirschi, 1990). Recent FBI data gathered from the 2000 Uniform Crime Reporting Program (http://www.fbi.gov/ucr/00cius.htm) are summarized in Figure 1. This figure provides a contemporary representation of the well-established “age-crime” curve, that is one of the more robust findings in criminology (Gottfredson & Hirschi, 1990). In this figure, arrest frequencies are at their highest around age 17 and fall off after adolescence. Second, evidence suggests that adolescent delinquency while defined as an antisocial behavior may, under certain circumstances, actually serve a constructive prosocial function by eliciting increased peer relations (Newcomb & Bentler, 1988). However, this association
Moffitt (1993) theorizes that AL antisocial behavior is chiefly the result of two factors – social mimicry and the maturity gap. Social mimicry, a term borrowed from ethology, refers to the mechanism by which an individual adopts the social behavior of another in order to obtain a resource or desirable outcome. Moffitt specifies the social currency obtained by AL delinquency as the power and privilege of mature status. A repeated finding that adolescents typically break the law in groups and that they cooperate with one another in delinquent acts provides some indication of the social nature of delinquent behaviors committed during this stage of development (Gold, 1970; Jones & Jones, 2000). This result has been echoed in more contemporary research that has found a positive relationship between delinquency and peer relationships (Engels & Bogt, 2001). The maturity gap refers to the discrepancy between the time an adolescent achieves biological maturity and the socially proscribed time when an adolescent attains mature adult status. It is well documented that adolescents are increasingly achieving biological maturity at earlier ages (e.g., Tanner, 1978; Williams & Dunlop, 1999) for a number of reasons that include nutrition and health care. It is also a function of our society that many of the trappings associated with adult status such as marriage and careers are increasingly delayed by such factors as compulsory education and college
enrollment (Arnett, 2000; Hogan & Astone, 1986). Moffitt’s theory posits that the
discrepancy between the biological and social attainment of maturity has aggravated this
maturity gap, and that delinquent behavior is seen by adolescents as a means to bridge
this gap and obtain mature status. For example, an adolescent who is truant from school
could be viewed as exhibiting maturity by rejecting rules and engaging in an autonomous
behavior. Exercising this autonomy, then, is associated with adult status. A necessary
consequence of this explanation for AL antisocial behavior is the eventual maturing out
of this behavior. This to appears to be supported as, by age 28, approximately 85% of
former delinquents desist from delinquent behaviors (Blumstein & Cohen, 1987).

Life-course-persistent (LCP) antisocial behavior refers to a group of individuals
that fails to limit their antisocial behaviors to adolescence. “As implied by the label,
continuity is the hallmark of the small group of life-course-persistent antisocial persons”
(Moffitt, 1993; p. 679). The LCP individuals begin exhibiting delinquent antisocial
behaviors at earlier ages, often as toddlers (Farrington, 1991), and continue to engage in
antisocial behaviors across their lifespan. Some evidence for the presence of this group
can be garnered from criminal records. For example, it is estimated that 60% to 80% of
all crimes are committed by a small number of recidivists (Loeber, 1982; Piper, 1985).
Other studies have found that the most persistent 5% or 6% of offenders are responsible
for about 50% of all known crimes (Farrington, Ohlin, & Wilson, 1986). Moffitt
describes LCP behavioral continuity using a term initially defined by Kagan (1969),
heterotypic continuity. Heterotypic continuity refers to the fact that LCP individuals do
not merely exhibit continuity of a single behavior but rather exhibit a continuous
underlying trait. This trait then manifests itself in a variety of antisocial behaviors that may expand in both breadth and scope as an individual's resources expand with their development.

Obviously, the explanation of social mimicry and maturity gap cannot be applied to LCP individuals, as the social pressures described above fails to predict either the earlier onset or the failure to desist from antisocial behavior. Instead, Moffitt presents LCP antisocial behavior as behavior committed by individuals who are qualitatively different from individuals engaging in AL antisocial behaviors. Moffitt contends that the origin of LCP antisocial behavior resides in neuropsychological vulnerabilities that predispose individuals to act in an antisocial fashion (e.g., Moffitt, Lynam, & Silva, 1994). What exactly is intended by the term neuropsychological vulnerability is somewhat vague and broadly described. One possibility raised is neurological damage. Studies have found that compromised in-utero neural development has been linked with elevated rates of antisocial personality (e.g., Paulhus & Martin, 1986) and still other studies have found that postnatal brain injuries have been linked with subsequent antisocial behaviors (e.g., Kandel & Mednick, 1991). Another explanation is that compromised neuropsychological health is the result of genetics. Studies have found adult antisocial behaviors have a large heritable component (Martin, Jardine, & Eaves, 1984; Plomin, Nitz, & Rowe, 1990). In Moffitt (1993), this notion is given short shrift as an explanation for LCP antisocial behavior as it is indicated in passing as just one possibility among many. This is unfortunate as an earlier paper by DiLalla and Gottesman (1989) presents an extensive and compelling hypothesis about the role
heritability may play in predicting antisocial behavior outside the context of adolescent limited antisocial behavior. The arguments presented in this paper will now be briefly related.

DiLalla and Gottesman's Lifespan Perspective of Delinquency and Criminality

In their review of family, twin, and adoption studies of antisocial behavior, DiLalla and Gottesman (1989) noted that, while many studies have found evidence for the heritability of antisocial behavior among adult populations, only weak evidence exists for the heritability of antisocial behavior among adolescents. They surmised that these seemingly contradictory findings could be reconciled if individuals who commit antisocial behaviors were conceived of not as a single group but rather as different populations that exhibit similar behavior that is the result of different underlying mechanisms. Specifically, DiLalla and Gottesman (1989) hypothesized the existence of three types of offenders, the continuous antisocials, the transitory delinquents, and the late bloomers.

Continuous antisocials describe those who exhibit delinquent behavior during adolescence and exhibit antisocial continuity by engaging in criminal conduct as adults. This group is analogous to the LCP antisocial group subsequently described by Moffitt (1993). The transitory delinquents describe those who exhibit delinquent behavior during adolescents and desist by adulthood. The behavior of this group is analogous to Moffitt's description of AL antisocial behavior. The late bloomers describe those who refrain from delinquent behavior during adolescence but engage in criminal behavior during adulthood.
DiLalla and Gottesman (1989) hypothesize that each of the three groups has its own unique etiology. Of importance to this paper are the mechanisms hypothesized for the development of transitory delinquents and the continuous antisocials. Transitory delinquent behaviors are thought to arise primarily due to environmental conditions, the most notable being peer influences. Transitory delinquency is viewed as part of a developmental phase that serves to help resolve problems and is outgrown by adulthood when other, more socially acceptable, solutions become available. While not providing a comprehensive description of the developmental processes underlying this phase, DiLalla and Gottesman (1989) indicate that these individuals will cease to commit antisocial acts when the environment changes and ceases to elicit them. On the other hand, continuous antisocial behaviors are hypothesized to be the result of individual traits such as temperament that possess a significant heritable component. Continuous antisocial behaviors are committed throughout the lifespan because of these intrinsic traits that predispose individuals for this behavior.

It is important to note that both DiLalla and Gottesman's (1989) transitory and continuous antisocial groups are hypothesized to commit antisocial acts during adolescence. The problem presented by this taxonomy of antisocial behavior, then, is in distinguishing between these different groups as they present the same behavioral phenotype during adolescence. If these antisocial groups exist, studies that examine adolescent delinquency are at risk for including members of the different antisocial groups into the same analyses (DiLalla & Gottesman, 1989). Attempts at describing or predicting behavior are compromised by the antisocial heterogeneity of the adolescents.
included. For example, suppose a researcher is interested in examining the genetic contributions to delinquent behavior. Simply conducting an analysis of delinquency among all adolescents will not work. The subset of individuals who are genetically predisposed to behave delinquently throughout their lifespan will be obscured by those who are predisposed to behave delinquently only during adolescence due to environmental influences. This is not an entirely new argument. Researchers have long argued that the base rates for delinquency among adolescents are so high that heritability is hard to establish (Gold & Petronio, 1980; Rowe & Rodgers, 1989; Wolfgang, Figlio, & Sellin, 1972). Indeed, the bulk of behavioral genetic research on antisocial behavior appears to support the notion of qualitatively different antisocial groups. This literature will now be reviewed.

**Behavioral Genetic Studies of Antisocial Behavior**

Sufficient anecdotal evidence exists demonstrating the heritability of antisocial behaviors that even the most ardent opponents of genetic explanations of behavior must give pause. A recent book detailing life inside a correctional institution (Conover, 2000) provides a nice anecdotal illustration of this. In this book, a new inmate confronts a man serving a life sentence for murder. The new inmate stares at the man’s face, finally exclaiming, “Dad!” Despite being separated at a young age and having no contact for over twenty-two years the son has joined his father in prison, serving out a sentence for armed robbery. In a similar vein, Fox Butterfield offers an intriguing series of historical accounts of violence and delinquency in his book, *All God’s Children* (1995). Butterfield’s book examines the case of Willie Bosket, one of the more violent and
aggressive inmates housed in America’s correctional facilities, having committed several hundred robberies, twenty-five stabbings, and two murders before the age of 15. Butterfield, like social science researchers, documents the adverse environment associated with violent and criminal behavior such as that to which Willie Bosket was exposed. However, unlike a number of researchers (e.g., Catalano & Hawkins, 1996; Haapasalo & Tremblay, 1994), Butterfield does not confine himself to a contextual argument, instead offering an explanation of aggression that also includes temperament. Butterfield traces the violent history of the Bosket family back to before the Civil War, showing that despite knowledge of the family’s own history, similar patterns of violence played out across generations. Butterfield and Conover’s books make for good reading and provide colorful examples of a potential genetic basis for delinquent and criminal behavior, however, they lack the statistical and methodological rigor required of the social sciences.

Fortunately, behavioral geneticists need not rely on anecdotes as they have conducted numerous studies of antisocial behavior over the past decades. This research has principally focused on two distinct areas, adult behaviors and adolescent delinquency. The bulk of this research has focused on adult criminal behavior. A smaller, though still substantial, body of research has focused on adolescent delinquency. Within the arena of research on adult criminology, the behavioral genetic literature has been in general agreement about the relative influences of environmental and genetic factors. This agreement, however, does not extend to the study of adolescent delinquency. Whereas recent reviews of the literature suggest there is some genetic influence on adolescent
antisocial behavior, much variability and controversy exists in the estimation of the extent of this influence (Carey, 1994; Gottesman & Goldsmith, 1994).

This paper will now present a review of the behavioral genetic literature on adolescent delinquent behaviors followed by a review of the behavioral genetic literature on adult criminal behaviors. Next, this paper will offer an explanation for the discrepancy between these two areas that is in keeping with the notion of a developmentally based taxonomy of antisocial behavior.

Behavioral Genetic Research on Adolescent Delinquency

A number of researchers have concluded that the preponderance of variation in adolescent antisocial behavior may be explained by shared environmental influences (Cloninger & Gottesman, 1987; Lyons et al., 1995; Rowe & Rodgers, 1989; Taylor, McGue, & Iacono, 2000). This is not to say that these researchers have failed to find high concordance rates for delinquent behaviors within twin pairs. A concordance rate simply refers to familial resemblance when dichotomous outcomes are considered. When applied to disorders, the concordance rate can be thought of as an index of risk where the rate indicates the percentage risk for siblings having the disorder (Plomin, DeFries, McClearn, & Rutter, 1997). A review by Cloninger and Gottesman (1987) found average concordance rates of 87% for MZ pairs and 72% for DZ pairs. The problem when attempting to partition variance into genetic and environmental influences is not low concordance rates but rather that the concordance rates for DZ twins are too high to be sufficiently different from the concordance rates of MZ twins. If we refer to these data not as concordance rates but rather, for illustrative purposes, as intra-class correlations we
can see how this is so by applying the formulae initially presented in Chapter 1 (see page 8). The heritability of a continuous trait is simply twice the difference between MZ and DZ twins, or $h^2 = 2[(r_{MZ} - r_{DZ})] = .30$ in this case. Again, treating the concordance rates provided in the review by Cloninger and Gottesman (1987) as intraclass correlations, we can further partition the variance in terms of shared and nonshared environment, keeping in mind that total phenotypic variation is simply the sum of genetic and environmental effects. This may be expressed with the following equation:

$$V_p = h^2 + c^2 + e^2,$$

where $V_p$ again represents total phenotypic variation, $h^2$ again represents heritability, $c^2$ represents shared environment, and $e^2$ represents unshared environment plus measurement error. Just as an algebraic proof can be derived from the MZ and DZ concordance rates so too can an estimation of $c^2$ can be expressed as simply $2*r_{DZ} - r_{MZ}$, or $2*().72 - .87 = .57$. Thus, shared environment ($c^2$) accounts for 57% of the phenotypic variation in adolescent antisocial behavior. The last component of variation, nonshared environment, is simply the proportion of variation remaining. This can be expressed as $1.00 - h^2 - c^2 = .13$. Alternately put, 13% of the total variation is due to nonshared environment ($e^2$) in this case. For a comprehensive and highly readable explanation of how each of these components may be estimated, please see Rowe (1994), "Separating Nature and Nurture."
Not all research, however, has come to the same conclusions about the relative influences of genes and environment. For example, while Rowe (1983) estimated the heritability ($h^2$) of delinquents at .32 and .44 for males and females, respectively, his estimates of shared environment were significantly lower for males and females, $c^2 = .30$, than the estimates in Cloninger and Gottesman (1987). Another study found remarkably similar contributions of heritability (36%), shared environment (31%), and nonshared environment (36%) (Jacobson, Prescott, & Kendler, 2000). Other studies have found that genetic factors contributed most to the phenotypic variation in a study of adolescent delinquency (Eaves et al., 1997; Gjone & Stevenson, 1997; O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998; Rowe, 1986; Rowe & Osgood, 1984; Silberg et al., 1996; Slutske et al., 1997). Still other studies have obtained lower or negligible estimates of heritability of antisocial traits among juveniles. For example, Lyons et al. (1995) obtained an estimate of heritability of only .07 and a shared environment estimate of .31, indicating that the largest proportion of phenotypic variation, .62, was due to nonshared influences. Achenbach (1993) describes two types of adolescent antisocial behaviors: delinquent behaviors that consist primarily of property and status offenses and aggressive behaviors that consist primarily of violent behaviors. In his study he finds aggressive behaviors have a significant heritable component whereas delinquent behaviors do not have a heritable component.

A recent meta-analysis examining genetic and environmental influences on adolescent aggression concluded that genetic and shared environmental influences contributed roughly equally to individual differences in aggression (Miles & Carey,
This result was based upon the analysis of 24 twin and adoption studies of aggressive behavior. A study was included in the analysis if it used any measure of aggression, hostility, or antisocial behavior or if the scale was specifically constructed to predict juvenile delinquency. Heritability was summarized across studies by age and gender. Heritability estimates for aggression among male twins under the age of 18 who were raised together (Gottesman, 1966; Loehlin & Nichols, 1976; Lytton, Watts, & Dunn, 1988; Rowe, 1983; Stevenson & Graham, 1988) accounted for between 20% and 74% of the variability in aggression with an average heritability estimate of 46.4%. Heritability estimates for aggression among female twins under the age of 18 who were raised together (Gottesman, 1966; Loehlin & Nichols, 1976; Owen & Sines, 1970; Rowe, 1983) accounted for between 14% and 72% of the variability in aggression with an average heritability estimate of 42.8%. The conclusion reached across these studies in the meta-analysis was that heritability and common environment are definitely responsible for individual differences in aggression among adolescents (Miles & Carey, 1997).

Miles and Carey’s (1997) meta-analysis also found that observational methods of measuring aggression under laboratory conditions, utilized in two studies (Plomin, Foch, & Rowe, 1981; Rende, Slomkowski, Stacker, Fulker, & Plomin, 1992), gave different results than self-report or parental report. Specifically, they note that observational studies suggested a strong shared environment influence and no genetic influence – a finding that the researchers were at a loss to explain. Several possible explanations for this discrepancy exist. First, research has found that studies of adolescent personality
including aggression that utilize self-report measures invariably find a significant effect of genotype (Plomin & Fulker, 1987). This suggests that the genetic influence may be on responding to questionnaires rather than on the construct being measured meaning that observational data may provide estimates free from biases introduced by self-report measures. Another explanation is that it is self-reports of adolescent antisocial acts that are reliable and valid (Gold & Petronio, 1980). Reliance on either observational or archival data may introduce bias by failing to account for those "clever or lucky individuals who are able to elude arrest [or detection]" (Rowe, 1983, p. 474).

Last, but of great significance, the meta-analysis suggested that genetic and environmental influences change with age. Common or shared environment was found to be very important in juvenile delinquency, whereas genes have been relatively more important for explaining variation in both adult and child criminality (Miles & Carey, 1997; Silberg et al., 1996; Siminoff, 2001; Siminoff, McGuffin, & Gottesman, 1994). This finding again points out the relevance of studying antisocial acts from a developmental perspective and is consistent with the notion of a developmentally based taxonomy.

Clearly, research on the genetic and environmental influences on adolescent delinquency contains sufficient discrepancies to warrant further study. There are a number of potential reasons for these discrepancies. One likely explanation is that antisocial behaviors are present at a very high rate among adolescents (DiLalla & Gottesman, 1989; Gold & Petronio, 1980; O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998; Wolfgang, Figlio, & Sellin, 1972). Differences in concordance between
groups such as those found between MZ and DZ twins are often negligible. The high base rate of delinquent behavior may be occluding a subgroup of adolescents with a genetic predisposition for delinquency (DiLalla & Gottesman, 1989). It is perhaps possible that only a small minority of adolescents are significantly predisposed to commit antisocial behavior and it is this minority that continues to be criminal in adulthood (DiLalla & Gottesman, 1989).

Another possibility for the discrepant results is that antisocial behavior is often operationalized quite broadly to incorporate both delinquent behaviors, encompassing a broad range of behaviors that may include relatively innocuous behaviors such as lying and curfew violations, as well as the more serious violent behaviors. As seen in the study by Achenbach (1993) this presents a problem. Violent, aggressive behavior may, as Moffitt (1993) speculates, tap into a more biologically determined trait. Delinquent behaviors may be more reflective of social influences. Carelessly lumping these two together may produce error and lead to discrepant estimates. An example of this is found in Miles and Carey’s (1997) inclusion of Rowe’s (1983) study of delinquency in their meta-analysis of aggression. This is unfortunate as a number of Rowe’s items include delinquent (e.g., shoplifting, trespassing, lying about age), rather than aggressive acts. A recent study that specifically separated aggressive and delinquent behavior found aggressive behavior to have a heritable component but failed to find this for delinquent behavior (Deater-Deckard & Plomin, 1999). This finding supports the notion that failure to treat aggression and delinquency separately may introduce error into estimates of heritability.
Behavioral Genetic Research on Adult Antisocial Behavior

Whereas the relative influence of genes and environment on adolescent antisocial behavior remains a matter of considerable debate, studies of adult antisocial behavior show far more consistent results. Specifically, research has repeatedly found that genetic influences and not shared environment account for much of the variation in adult antisocial behavior (Miles & Carey, 1997). This result has been found both in adoption studies (Brennan, Mednick, & Gabrielli, 1991; Cadoret, 1982; Cloninger, Sigvardsson, Bohman, & von Knorring, 1982; Crowe, 1974; Hutchings & Mednick, 1975; Mednick, Gabrielli, & Hutchings, 1984; Schulsinger, 1972) and in twin studies (Christiansen, 1977; Cloninger & Gottesman, 1987; Gottesman & Goldsmith, 1994; Lyons et al., 1995; McGuffin & Gottesman, 1985; Nigg & Goldsmith, 1994). Results from both research designs will now be presented.

Adoption Studies. Cloninger et al. (1982) conducted a study of male adoptee criminals in Sweden. They found evidence for both environmental and genetic influences on criminal behavior with genetic factors being the strongest predictor of criminal behavior. Specifically, their study found that men who were adopted into criminal environments but who did not have criminal biological parents were more than twice as likely to be criminal themselves when compared with male adoptees who had no biological or adoptive criminal background. Male adoptees whose biological parents were criminal and who were adopted into non-criminal environments were more than four times as likely to be criminal themselves. What was perhaps most interesting in their study, and a point which emphasizes the importance of both environment and
heredity, was the interaction effect of both criminal environment and genetic backgrounds. Those males who were adopted into a criminal environment and who had a biological parent with a criminal background were more than 14 times as likely to themselves be criminal than those with no biological or adoptive criminal background.

Similar results were also found in a large (N = 14,427) Danish adoption study that looked at male conviction rates (Hutchings & Mednick, 1975; Mednick Gabrielli, & Hutchings, 1984). In this study, a significant relationship was found between biological background and criminality but not for adopted environment. If neither the biological nor the adoptive parents were convicted, 13.5 percent of adopted sons were convicted of a crime. If an adoptive parent was convicted but a biological parent was not, the conviction rate was 14.5 percent, not a statistically significant increase. If, however, adoptive parents are not convicted but a biological parent was, the conviction rates were significantly larger at 20.0 percent. Again, a situation where both an adoptive parent and a biological parent were convicted resulted in the highest conviction rates for sons at 24.5 percent.

Another adoption study by Crowe (1974) focused on a sample of 46 children given up for adoption during infancy whose biological mothers were incarcerated for felony offenses. While no formal diagnosis of antisocial disorder was made for biological mothers, it was assumed that they would be overrepresented for antisocial characteristics based upon their conviction records. A control group of adoptees whose biological parents were not convicted of felonies were also sampled. Significantly more
offspring of felons had adult convictions and were incarcerated than were matched controls providing an indication of a genetic effects.

A review of adoption studies (Cadoret, 1982) concluded that while genetic factors typically explained more variance in adult antisocial behaviors than environmental factors (e.g., 5.4% vs. 3% in Hutchings, 1972), both sources of variation were statistically significant. Cadoret (1982) argues that controversies about whether the environment or genes hold greater influence over antisocial behaviors are meaningless without considering the possibility of an interaction between genetic and environmental influences. He states, "...with two factors – genetic and environmental – one factor might behave differently in the presence of different degrees (or in some cases, presence or absence) of the second factor" (Cadoret, 1982, p. 236). A method for detecting genotype-environment (G × E) interaction using adoption data involves utilizing adoption data where adoptees are separated at birth from their biological parents and placed with genetically unrelated adoptive parents. Data from both biological as well as adoptive parents must be collected. This design, termed the full adoption design, permits estimation of a genetic effect, estimated from the biological parent background, and an environmental effect, estimated from the adoptive parent or adoptive conditions. An interaction term can be created consisting of the product of the adoptive and biological terms. An adoption study of antisocial behaviors among adults (Cadoret, 1985) demonstrated the utility of including a G × E interaction term. The main effects of genetics and environment explained 8% to 10% of the variation in antisocial behaviors by themselves. However, the inclusion of a G × E term increased this to 16% to 18%. 
A potential difficulty with relying on adoption studies is that of restricted range of the adoptive environment. The fact that adopted environments are generally screened so as to avoid particularly difficult or dysfunctional environments limits what can be said about environmental influences. Twin studies are not limited in this manner and present an effective method to deal with the potential problem of restricted range.

Twin Studies. Results from twin studies are consistent with those obtained from adoption studies. A review of twin studies by Cloninger and Gottesman (1987) found that MZ twins have a significantly higher concordance rate for adult antisocial behavior than do DZ twins. The average MZ concordance rate in this review was .51 and the average DZ concordance rate was .22. Based upon these concordance rates, it is possible to generate a statistic known as the liability-threshold model (Falconer, 1965; Smith, 1974) and estimate heritability and shared environment. These estimates show that heritability explains a greater proportion of variation, $h^2 = .54$, than does the influence of shared environment, $c^2 = .20$. Alternatively put, studies in this review found that 54% of the variation in adult antisocial behavior was attributable to genetic factors and 20% of the variation in adult antisocial behavior was attributed to aspects of the environment that made twins similar to one another. More recent studies have found remarkably similar concordance rates. For example, Gottesman and Goldsmith (1994) found an adult concordance rate of 51.5% for MZ pairs and 23.1% for DZ pairs. Lyons et al. (1995) obtained a similar estimate of heritability at $h^2 = .43$ but estimated shared environment at only $c^2 = .05$. One explanation for why these estimates are lower may be the presence of significant measurement error, which, if present, would reduce both $h^2$ and $c^2$ while
inflating estimates of nonshared environment. Indeed, nonshared environment accounted for the majority of variation in this study as $e^2 = .52$. Lyons et al. (1995) suggest that measurement error may be a factor in estimates of adolescent antisocial behaviors where nonshared environment is similarly high ($e^2 = .62$) as the sample was older ($M = 44.6, SD = 2.6$) and participants were asked to recall behavior before the age of 15. A similar rate in error of recall could be in effect for adult antisocial behaviors as participants were asked to recollect behaviors that may have occurred many years earlier during their later teenage years or early twenties.

Interpreting Differences between Adolescent and Adult Antisocial Behavior

While some discrepancies exist in the estimation of genetic and environmental influences on adolescent antisocial behavior, it is generally accepted that genetic influences on this behavior become increasingly important in adulthood (Miles & Carey, 1997). A tempting way to interpret this difference is to adopt a developmental argument. This is an option that has been taken by a number of researchers and typically takes the form, "(shared environmental influences) may be very important in the initiation and early maintenance of aggression but may fade over time" (Miles & Carey, 1997, p. 214). An analogy employed by some researchers (e.g., Lyons et al., 1995) is to the developmental changes noted in the heritability of intelligence. This argument, when applied to intelligence, makes sense as it satisfies a couple of conditions. First, intelligence when applied to a 16 year old is the same construct as intelligence when applied to a 30 year old. Second, it is plausible that familial influences may be reduced and genetic factors increased when an individual leaves the home and enters more fully
into an environment of his or her own making (Scarr & McCartney, 1983; Shanahan, Sulloway, & Hofer, 2000). Certainly this second condition is met when we consider antisocial behavior. It is less clear, however, that the first condition is met. As previously stated (p. 2), a compelling argument could be made that an antisocial act committed by a 16 year old is something that is very different from an antisocial act committed by a 30 year old. Thus, it may not be that shared environmental influences evaporate or that heredity takes hold at an older age but rather that we are examining two different types of behavior that merely look equivalent. This is just what Moffitt (1993) contends, arguing that the majority of antisocial behavior during adolescence may be a socially adaptive response and antisocial behavior during childhood and during adulthood may reflect pathological characteristics.

Comparisons of adolescent behavior with adult behavior will not resolve this matter. What is needed is a longitudinal study of antisocial behavior, where individuals who restrict their antisocial behavior to adolescence can be compared with those who engage in antisocial behavior prior to or after adolescence. The heritability of antisocial behavior during adolescence can be compared to the heritability of this same trait in the same individuals during childhood or adulthood. Genetic analyses can be conducted separately on each group to determine the degree to which heredity and environment are differentially influential. DiLalla and Gottesman (1989) stated that there has yet to be a longitudinal adoption or twin study of criminality beginning in adolescence an continuing through adulthood. This does not appear to have changed with one recent exception.
A longitudinal study by Taylor, Iacono, and McGue (2000) followed twins from age 11 through age 17. The authors of this study proposed that early onset delinquency, operationalized as exhibiting delinquency by age 11 or high aggression scores by age 12, would have a larger genetic component than would later onset delinquency. Their data appeared to tentatively support this conclusion: MZ twins in this group exhibited greater concordance for antisocial behaviors (55% concordant) than did DZ twins (29% concordant) though this failed to reach statistical significance (p = .06). Later onset delinquents did not exhibit differential concordance on the basis of zygosity (MZ = 43%, DZ = 39%). While this study provides some support for the notion of qualitatively different antisocial groups, a number of methodological difficulties limited this study. First, they were only able to collect data on 36 early starter twin pairs and only 41% of the initial sample had data past age 14. This low return rate is, in part, due to the fact that data were still being collected when this paper was written. While the study is suggestive that a greater genetic influence exists for early-onset than late-onset delinquency, the study lacked sufficient power to determine this.

The Present Study

The present study utilizes a behavioral genetic design, the full adoption study, to test Moffitt’s (1993) AL and LCP antisocial behavior taxonomy. According to Moffitt, AL and LCP antisocial behaviors have a different etiology. AL antisocial behavior, it is argued, constitutes the bulk of adolescent antisocial behavior and is characterized by being both transitory and environmentally driven. LCP antisocial behavior, on the other hand, is described as being far less common and is characterized by both its persistence
and its dependency upon individual traits that foster antisocial behaviors. The first goal of the present study is to identify groups that can be categorized as either belonging in the AL or LCP category. Participants will be assigned to the AL group if they limit their delinquent behavior to adolescence. Participants will be assigned to the LCP group if they initiate antisocial behaviors prior to adolescence, commit antisocial behaviors during adolescence, and persist in antisocial behavior into adulthood. Next, this study will seek to examine whether genetic influences on antisocial behaviors are differential for AL and LCP groups.

The present study will contribute to the literature on adolescent antisocial behaviors both theoretically and empirically. This study will join a very small number of papers that have specifically tested Moffitt's (1993) developmentally based taxonomy of antisocial behaviors (e.g., Piquero & Brezina, 2001). Further, with the exception of the previously cited study by Taylor et al. (2000), this study will be the only study to do so utilizing a behavioral genetic design. By applying a behavioral genetic design, this study will be better able to disentangle the two groups if indeed they differ on a heritable trait.

Research Questions

The primary focus of this paper is to examine whether AL and LCP antisocial behaviors are differentially influenced by genetics. In addition, salient features of the sample will be examined in an attempt to distinguish the two groups from one another. Specifically, the following research questions will be examined:

1. Are classifications of LCP and AL antisocial behaviors independent of a biological history of antisocial behavior? That is, does biological parent history differentially predict whether an individual is classified as LCP and AL?
2. Do LCP individuals exhibit a higher genetic influence for antisocial behavior than AL individuals?

3. Does the genetic influence on antisocial behavior differ depending upon the domain of antisocial behavior examined? Specifically, the genetic influences on aggressive behaviors (e.g., fighting) and substance use will be contrasted.

4. Do LCP offenders generally engage in a greater number of offenses than AL offenders? Do LCP individuals commit more aggressive offenses than AL individuals?

5. Are AL individuals more likely to commit some offenses than other offenses? Specifically, are AL individuals just as likely to commit behaviors that confer adult status (e.g., substance use) as LCP individuals and fewer aggressive offenses?
CHAPTER III

METHOD

Sample

The data used in this study come from Cadoret's Iowa Adoption Studies, 1975-1982 (Cadoret, 1988). These data were obtained from the Henry A. Murray Research Center of the Radcliffe Institute for Advanced Study at Harvard University, a national repository for social and behavioral science data on human development (http://www.radcliffe.edu/murray/index.html). The Iowa Adoption Studies consist of three separate studies: the Iowa Children and Family Services study, the Lutheran Social Services Antisocial project, and the Lutheran Social Services Alcohol project. The present study is concerned with the first two of these three studies. The purpose of the Iowa Children and Family Services and Lutheran Social Service Antisocial projects were to assess environmental and genetic influences on subsequent antisocial behavior among adoptees and matched controls.

The Iowa Children and Family Services Study. Data were collected for the Iowa Children and Family Services study in 1975 and included data on adoptee participants with biological family histories of psychiatric dysfunction, the adoptive family, and on matched adoptee controls. Selection criteria for inclusion in this study included that adoptee subjects have one or more biological parents presenting with either a documented psychiatric condition or established behaviors that would permit for or suggest a psychiatric diagnosis. To be eligible, the adoptee had to have been separated from biologic parents at birth and had to have had no subsequent contact with either biological
parent or other members of the biological family. Lastly, the adoptee had to have been
placed permanently in an adoptive home.

The adoption records of 1,646 adoptees obtained from the Iowa Children's and
Family Services Agency of Des Moines, Iowa, dating from 1939 through 1965, were
reviewed in search of adoptees who met the above criteria. One hundred ninety adoptees
met the selection criteria. These adoptees were then matched to a control subject who
was also adopted. Adoptees were matched on age, gender, age of biological mother, and
time spent in foster care before final placement. Adoptees in the control group were also
required to meet the same selection criteria as the adoptees in the experimental group
except that neither biological parent could have any documented history of psychiatric
disorder, or of behaviors that could be diagnostic or suggestive of a psychiatric disorder.
Psychiatric diagnoses of biological parents and family members were based on data
gathered from adoption agency records, and were made by a psychiatrist under blind
conditions with respect to information found about adoptees.

A total of 505 families were initially contacted by mail. Of these, researchers
were unable to locate 24 families and 114 families refused participation in the study. The
final sample consisted of 367 participating families (72.3% response rate) and included
205 males and 162 females. The participants were predominantly Caucasian. The
comparative research design resulted in a final sample where essentially one half of the
participants came from a biological history of either a documented psychiatric condition
or established behaviors that would permit for or suggest a psychiatric diagnosis and one
half of the participants served as members of a demographically matched control group.
who did not have biological history of psychiatric diagnosis. At the time of data collection in 1975, participants in this study were between 10 and 40 with an average age of 21.74 years (SD = 6.69).

Nonparticipating adoptees from the Iowa Children and Family Services study were compared to participating adoptees on age, gender, amount of time the adoptee spent in foster care prior to permanent placement, whether an adoptee was an experimental or control participant, age of biological mother, the type of psychiatric diagnosis of the biologic parents, and adoptive parent socioeconomic status. No significant differences were found on any of these variables (Cadoret, 1988).

The Lutheran Social Services Antisocial Project. Data were collected for the Lutheran Social Services Antisocial project during 1979 and 1980. This project gathered data on adoptee participants with biological family histories of antisocial problems, the adoptive family, and on participants' matched adoptee controls. Sample selection was initiated through review of adoption records from the Lutheran Social Services in Des Moines, Iowa by social workers. Records dating from 1938 through 1962 were examined. The records of adoptees who had been separated from their biological parents at birth and permanently adopted by nonrelatives were selected. Information was recorded about any psychiatric, substance abuse, or behavior problems among any of the adoptee's first and second degree, and extended biological family members. While a biological parent could be comorbid for other disorders, a diagnosis of antisocial personality disorder was required for inclusion in the project.
A total of 391 families were identified as potentially eligible for participating in one of two projects, either the Lutheran Social Services Antisocial project or the Lutheran Social Services Alcohol project. Participants selected for the Alcohol project were not used in this study. Researchers were unable to locate eight of the 391 families. Of the remaining 383 families, 160 families refused participation in either project yielding an overall response rate of 55.9% and a complementary refusal rate of 44.1%. Of the 223 families who agreed to participate, 96 were included as participants in the antisocial behavior project. These participants were predominantly Caucasian and included 66 males and 30 females. Roughly half of the participants in this project were adoptee control participants matched to the target adoptee participants. Participants in this study were between 17 and 39 with an average age 24.44 years ($SD = 6.44$).

Adoptee participants included in the antisocial project had the presence of antisocial behavior among many of their biological family members. Family presence of antisocial behavior was considered "definite" in cases in which two or more of the following behaviors were noted: school behavior problem, running away overnight, incorrigibility, staying out at night beyond permitted time, associating with children of bad reputation, impulsive or reckless behavior, truancy, vandalism, irresponsibility about spending time or money, stealing, bad language, training school record, juvenile court record, police trouble, poor work history, marital problems, repeated fighting, sexual promiscuity, vagrancy, persistent lying or use of aliases, being a convicted felon, participation in rape or incest, excessive sex talk or play or self-exposure, and having a prison record (Cadoret, 1988). Antisocial behavior was defined as "possible" when one
or more of the above behaviors were noted. These definitions were established lieu of formal diagnosis of antisocial personality based on the DSM-III due to lack of detail about behaviors in the adoption records (Cadoret, 1988). Felony convictions were considered to be a valid proxy for antisocial behavior due to a finding that 80% of similar individuals were diagnosable as antisocial (Cadoret & Cain, 1980).

Families who refused participation in the Lutheran Social Services Antisocial and Alcohol research projects were compared to families who did participate. One significant difference between groups emerged. It was found that participating families were more likely to have a biological history of psychiatric problems other than alcohol or antisocial behaviors than were families that refused to participate (Cadoret, 1988).

**Sample Analyzed in This Study**

The sample analyzed in the present study consisted of families from both the Iowa Children and Family Services Study and the Lutheran Social Services Antisocial Project. From these families, adoptees who were under the age of 18 at the time of the interview in the Iowa Children and Family Services Study (n = 67) were not included as they had incomplete data about adolescent behaviors. This resulted in a final sample of 387 and included 161 females (41.6%) and 226 males (58.4%). The age of the adoptees used in subsequent analyses ranged from 17 to 40 (M=24.03; SD = 5.92).

**Procedures**

*The Iowa Children and Family Services Study.* The Iowa Children and Family Services study included an Adoptive Parent Interview consisting of a modified structured questionnaire using 150 items. The Adoptive Parent Interview covered such topics as
physical development, health, and social adjustment, and also included questions pertaining to disorders of conduct, and disorders of emotion throughout childhood and adulthood. Data on all adoptees were obtained from adoptive parents, regardless of the age of the adoptee. Two versions of the Adoptive Parent Interview were used, one for adoptees under 18 years of age and one for adoptees aged 18 or older. The interview for adoptees under the age of 18 contained four parts. Part I contained information on infancy through age 2 covering physical health, development, and temperament. Part II covered preschool ages 2 through 6 and included questions about physical health, development, temperament, and discipline. Part III detailed ages 6 through 12 and covered physical health, development, school, temperament, and discipline. Part IV detailed adolescence age 13 to present and covered physical health, development, school, temperament, and discipline. The interview for adoptees 18 years of age and older contained the same elements save that Part IV detailed ages 13 to 18 and a fifth section, Part V which detailed age 18 to the present, was included. A research assistant who was blind to whether the adoptee in question has a family history of antisocial behavior conducted the Adoptive Parent Interviews over the telephone. The interview routinely required between 40 and 90 minutes to administer.

Biological data were also gathered concerning the adoptee's birth, delivery, and pediatric information over the first few months. Data also included information concerning the biological mother's psychological, and physical condition during pregnancy, labor, and delivery. The socioeconomic status of adoptive families was based on the occupation of the adoptive father. This was based on a seven-point scale from the
Hollingshead Two-factor Index of Social Position-Occupation factor (Hollingshead, 1947).

In cases in which an adoptee was 18 years of age or older, adoptive parents were asked if they would permit their child to be interviewed. If the parents permitted, then the adoptee was sent a letter introducing the study, and requesting an interview. The adoptee interviews were also conducted by telephone. The Adoptee Interview, administered to adoptees 18 years of age and older, was a structured psychiatric interview which included sections on social history; schooling; work history; marital history; social and leisure activities; military history; financial and housing status; housing; and medical history. This interview also included questions designed to screen for any of the following psychiatric syndromes: affective disorder; mania and hypomania; schizophrenia; hysteria; antisocial personality; drug dependence; alcoholism; anxiety neurosis; obsessive compulsive disorder; and mental retardation. These diagnoses were made by a psychiatrist and was also made under blind conditions as to the clinical status of the adoptee's biological parents (Cadoret, 1988).

The Lutheran Social Services Antisocial Project. The Lutheran Social Services Antisocial Project included an Adoptive Parent Interview through which data were gathered about the adoptive family and the adoptee. This interview consisted of five parts. Part I detailed infancy through age 2 and covered physical health, development, and temperament. Part II detailed preschool ages of 2 through 6 and covered physical health, development, temperament, and discipline. Part III detailed grade school ages of 6 through 12 and covered physical health, development, school, temperament, and
discipline. Part IV detailed adolescence age 13 to 18 and covered school, temperament, and discipline. Part V detailed ages 18 to the present and covered physical health, development, marriage, and parenthood. The interviewer was blind to whether the adoptee was a member of the experimental or control group. This interview was conducted face-to-face by a trained research assistant.

The Adoptee Interview included the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (SADS-L; Spitzer & Endicott, 1979) to assess for the presence of clinical conditions. This structured psychiatric interview was administered by a trained social worker. Other questions from the Adoptee Interview were designed to gather data on social history; work history; marital history; social leisure; military experience; finance and housing status; medical history; family environment; and adoption. The Adoptee Interviews were conducted face-to-face by trained research assistants, usually at the home of the adoptee.

Measures

Measures used in this paper were the same across both samples except when otherwise indicated.

Adoptee Antisocial Behaviors. Information on adoptee antisocial behavior was obtained from both adoptee self-report and from adoptive parent reports. Adoptive parents reported on antisocial behaviors at five time points in the adoptee's development: at ages 0 to 2, 2 to 6, 6 to 12, 13 to 18, and from 18 through present. Adoptees reported on antisocial behaviors just once.
Adoptive Parent Reports of Adolescent Antisocial Behaviors (13-18). Adoptive parents were asked a series of 12 yes/no questions about their child's behavior between the ages of 13 and 18. These questions were largely based on an adolescent antisocial scale developed by Robins (1966). Validity of this scale was established by comparing a diagnosis based upon the scale with a diagnosis made when additional information was made available such as after a period of hospitalization. The diagnosis based upon the interview was identical to subsequent diagnoses 89% of the time (Robins, 1966).

Although the questions relied on an adoptive parent's retrospective recall, one advantage of the particular items used was that because of attendant accompanying consequences (i.e., expulsion from school, juvenile court appearance) or because the behaviors could cause a flagrant disruption in the home environment, previous research has shown they were more readily recalled (Cadoret & Cain, 1981). The 12 questions asked were: 1) "Was he ever truant from school?"; 2) "Did he ever have trouble with teachers or administrators?"; 3) "Was he ever suspended or expelled?"; 4) "Was he frequently disobedient or rebellious?"; 5) "Did he ever run away from home?"; 6) "Did he have any experience with drugs or alcohol?"; 7) "Did he ever come to the attention of the juvenile authorities for any reason?"; 8) "Did he belong to any gangs?" 9) "Was he ever involved in physical fights?"; 10) "Was he defiant about obeying rules or carrying out chores?"; 11) "Was he ever insolent at home?"; and, 12) "Did he bully or dominate others, brothers, or sisters?"

Several of these items, specifically numbers 4, 6, and 12, represented additions by Cadoret, Cunningham, and Loftus (1976) to Robin's original scale. Psychometric
properties of these variables will be presented in the results section. Internal consistency of the set of 12 items will be computed within AL and LCP adolescents separately. Convergent and discriminant validity will be assessed to the extent possible through the computation of correlations with additional variables in the data sets. These items will be included in the analyses of adolescent antisocial behaviors as they provide additional information on antisocial behavior in the domains of substance use and aggressive behavior. Two separate subscales sampling the domains of aggressive behavior, and substance use will also be examined. The aggressive behavior subscale will consist of two items: "Was he ever involved in physical fights?"; and, "Did he bully or dominate others, brothers, or sisters?" The substance abuse domain will consist of a single item: "Did he have any experience with drugs or alcohol?"

Adoptee Self Report of Antisocial Behaviors. Adoptees in the Iowa Children and Family Services Study were administered a psychiatric interview in accordance with ICD-9 criteria (Rutter, Shaffer, & Sturge, 1975). In order to assess antisocial behavior, this interview contained a 28-question measure over nine different areas (e.g., "Did you ever play hookie from school?"; "Have you ever been arrested?"; "Have you ever told lies for no particular reason?"). The format of the items were generally yes/no. The complete interview is presented in Appendix A. The psychiatrist administering this interview would stop asking questions from the antisocial component of the interview if the adoptee responded negatively to six of the nine sections. Positive responses in four of the nine areas indicated a probable diagnosis of antisocial personality disorder. Five positive responses indicated a definite clinical assessment of antisocial personality
disorder. Interrater reliability of this measure for this diagnosis, assessed by Cohen's Kappa, was moderate ($\kappa = .48$) and the overall reliability of all ICD-9 Axis-I diagnoses was quite similar to Axis I diagnoses utilizing DSM-III (Gould, Shaffer, Rutter, & Sturge, 1988).

Adoptees in the Lutheran Social Services Antisocial Project were administered the SADS-L that included the Antisocial sub-scale (Spitzer & Endicott, 1979). The antisocial subscale consisted of four criteria evaluated by 21 yes/no items. The four criteria included: poor occupational performance (e.g., "Since you started working have you changed jobs a lot?"); behaviors before the age of 15 (e.g., "When you were younger did you break windows, destroy property?"); behaviors after the age of 15 (e.g., "Since age 15 have you often not paid debts or taken care of other expected financial responsibilities (e.g., child support)?"); and, some evidence of markedly impaired capability to sustain lasting, close, warm and responsible relationships with family friends, or sexual partners (e.g., "Is there anyone that you really feel very close to?") (reverse coded). A positive rating for all four criteria indicates a clinical diagnosis of antisocial personality disorder. The complete interview is presented in Appendix B. This measure was assessed in accordance with the guidelines specified by Feigner et al. (1972). Published reports have indicated the antisocial component of the SADS-L has good test-retest reliability (Guze & Goodwin, 1971) and a high intraclass correlation of $r = .77$ (Keller, et al. 1981).
Sufficient data were available to make a diagnosis of antisocial personality for 71% of the sample utilized in this study’s analyses (n = 271). Of these, 46 met clinical criteria for antisocial personality and 225 did not.

**Adoptee Antisocial Status.** Adoptees were classified as either adolescent-limited (AL) or life-course-persistent (LCP) based upon adoptive parent reports of when the child had engaged in antisocial behaviors. Reports of antisocial behaviors were available from ages 0 to 2, 2 to 6, 6 to 12, 13 to 18, and over 18. Based upon Moffitt’s (1993) definition of AL and LCP antisocial behavior, in the present study, a classification of AL required that adoptive parents indicated that adolescents had engaged in antisocial behavior only during adolescence. Endorsement of an antisocial activity during the ages of 13 to 18 and/or over the age of 18 but not from ages 0 to 2, 2 to 6, and 6 to 12 resulted in a classification of AL. A classification of LCP requires that adoptees indicated that they had engaged in antisocial behavior during adolescence (defined as 13 to 18), over the age of 18, and during at least one other developmental periods. Utilizing these criteria, 177 of 387 adoptees (45.7%) in this study’s sample could be classified as either AL or LCP. Of these, 115 (29.7%) were classified as AL and 62 (16.0%) classified as LCP. Further description of these groups will be presented in the results.

Items used from each of the stages of development will now be presented. These items are necessarily different from the adolescent antisocial behaviors cited above as the operational definitions of the underlying antisocial behavior construct are necessarily different at earlier versus later ages. While different, these items capture what Kagan (1969) and Moffitt (1993) refer to as heterotypic continuity. Reliability coefficients...
(presented in the results section) were calculated for antisocial behaviors before adolescence, during adolescence, and after the age of 18.

Adoptive Parent Reports of Adolescent Antisocial Behaviors (0-2). A code of antisocial behavior for the infancy age period was given based upon adoptive parents’ responses to two items. These items were: “When you first got the child, what was he like? In what ways?” and, “Was he more often contented or discontented as a baby?” A code of difficult and violent reactions for the first item or discontented in response to the second item resulted in classification as antisocial for this period.

Adoptive Parent Reports of Adolescent Antisocial Behaviors (2-6). A code of antisocial behavior for the toddler age period was given based upon adoptive parents’ response to four items. These items were: “What was he like as a toddler?”; “Did he have frequent temper tantrums?”; “Was he a contented or discontented toddler?”; and, “When there were changes in his immediate environment, how did he react?” Codes of violent/difficult, yes, discontented, or violent/difficult resulted in classification as antisocial for this period.

Adoptive Parent Reports of Adolescent Antisocial Behaviors (6-12). A code of antisocial behavior for the elementary school-age period was given based upon adoptive parents’ responses to four items. These items were: “Have you noticed or has the teacher ever complained about any of the following traits” “rebellious or difficult to discipline” “does not get along with classmates”; “reckless” or, “destructive.” Endorsement of any of these categories resulted in a classification of antisocial for this period.
Adoptive Parent Reports of Adolescent Antisocial Behaviors (13-18). A code of antisocial behavior for these two age groups was based upon adoptive parents' endorsement of the items drawn from Robins (1966) as described above.

Adoptive Parent Reports of Adolescent Antisocial Behaviors (18+). A code of antisocial behavior for age 18 and older was based upon adoptive parents' responses to six items. These items were: "Is he generally easy to get along with, or is he easily upset?"; "How does he adjust to new situations and experiences?"; "Does he have any close friends?"; "Does he lie frequently?"; "Has he ever stolen anything?"; Has he ever had trouble with the police?; and, "Has he ever been fired from a job?" Endorsement of any of these items resulted in a classification of antisocial for this period.

Diagnosis of Biological Parents. For the purposes of this dissertation, biological parents were classified into groups diagnosed with antisocial personality disorder, history of psychopathology, or no known disorder. A diagnosis of antisocial personality required a record of three or more antisocial behaviors (e.g., "arrested for writing bad checks" or "convicted of felony") as described by Robins (1966). In the sample analyzed in this study, 70 adoptees had a positive diagnosis of antisocial personality disorder.

In addition to antisocial personality, a diagnosis of psychopathology included behavioral disorders, and severe alcohol or drug abuse/dependency. Behavioral disorders required clinical diagnosis and alcohol abuse/dependency was applied if individuals had two or more social or medical complications of heavy drinking (e.g., "drink all the time" or "lost job due to drinking") or had been hospitalized for alcohol detoxification (Cadoret & Gath, 1980; Cadoret, Troughton, & O'Gorman, 1983). A diagnosis of drug
abuse/dependency required that individuals have two or more social or medical complications from drug use (Cadoret, Troughton, O'Gorman, & Heywood, 1986). In the final sample analyzed in this study, 123 adoptees had a biological parent with a diagnosis of one or more forms of psychopathology. Missing data resulted in the inability to classify 53 adoptees.

Adoptive Home Environment. A dichotomous variable describing quality of adoptive home environment was created. Classification of the adoptive home environment as adverse was based upon the original measure by Cadoret, Cain, and Crowe (1983). This measure held that an adoptive home environment was adverse if either an adoptive parent or an adoptive sibling had a psychiatric disorder or behavioral problem which had required professional attention, if an adoptive parent had an alcohol or drug problem that required treatment, or if adoptive parents were divorced. While divorce occurs with normative regularity in American society, this is not the case among married couples who adopt, here divorce is quite rare and is associated with greater conflict among adopted families (Cadoret, 1985). Similarly, many of these families raised their children in decades where divorce was less prevalent and thus being raised by a divorced parent could be stigmatizing (Glenn, 1991; Hernandez, 1988). In the final sample analyzed in this study, 65 adoptees were adopted into a home that was classified as an aversive environment.

Plan of Analysis

The first hypothesis that was tested was whether LCP and AL antisocial behaviors are independent of genetic history. Genetic influence on LCP and AL antisocial behavior
will be assessed using Chi-square tests of independence and multiple regression. It was
hypothesized that LCP antisocial classification would be dependent upon genetic
influences and that AL antisocial classification would be independent of genetic
influences.

The second hypothesis that was tested predicted that there would be a higher
genetic influence on LCP antisocial individuals for their antisocial behavior than would
be found among AL antisocial individuals. The dependent variable for these analyses
was both adoptive parent reports of adoptee antisocial behavior during adolescence and
adoptee self-report of antisocial behavior during a clinical interview. Hierarchical
multiple regression was utilized when the dependent variable was parent report of
antisocial behaviors during adolescence and logistic regression was utilized when the
dependent variable was diagnosis of antisocial personality based upon adoptee clinical
interviews.

First, a multiple regression equation with biological history of antisocial behavior
and adverse family environment was used to predict the number of adolescent antisocial
behaviors reported by parents. It was hypothesized that genetic history would be a weak
predictor of variation in antisocial behavior during adolescence. The regression equation
tested was:

\[ ASB_x = b_0 + b_1(\text{Genetics}) + b_2(\text{Environment}) + e, \]

where \( ASB_x \) indicates antisocial behavior during adolescence, genetics indicates a
psychiatric diagnosis for a biological parent, and environment refers to whether or not an
adverse adoptive environment existed. The full adoption design also allows for the
influence of environment to be examined. The above equation includes a measure of adverse family environment. It was not hypothesized that adverse family environment would predict antisocial behavior (e.g. Cloninger, et al. 1982), rather, $b_2$ (environment) was entered as predictor so that any effect of biological history had to be above that which was predicted by environment and to allow for subsequent testing of a Genetic × Environment interaction.

In order to test whether genetic influence on antisocial behavior was significantly different between the AL and LCP groups, the next step of the hierarchical multiple regression model included an AL/LCP classification variable. The main effect of AL or LCP classification was tested with the following equation,

$$\text{ASB}_x = b_0 + b_1(\text{Genetics}) + b_2(\text{Environment}) + b_3(\text{AL-LCP}) + e$$

In this equation, the AL-LCP variable was a dichotomous variable. Those individuals classified as AL were given a code of 0 and those classified as LCP were given a code of 1. It was hypothesized that this classification variable would be positively associated with variation in antisocial behaviors.

The next step of this hierarchical equation included the addition of an interaction term. This interaction term created was the product of the genetic history variable and the AL-LCP variable. The equation tested was,

$$\text{ASB}_x = b_0 + b_1(\text{Genetics}) + b_2(\text{Environment}) + b_3(\text{AL-LCP}) + b_4(\text{AL-LCP} \times \text{Genetics}) + e$$

It was hypothesized that evidence of an interaction between biological history and group classification would be found and the interaction term would be significant.
Similarly, a significant G × E interaction, consistent with the previously described adoption studies by Cloninger et al. (1982) and Mednick et al. (1984), was hypothesized for LCP individuals exposed to an adverse environment. The equation tested was:

\[ ASBx = b_0 + b_1(AL-LCP) + b_2(\text{Genetics}) + b_3(AL-LCP \times \text{Genetics}) + b_4(\text{Environment}) + b_5(\text{Genetics} \times \text{Environment}) \]

In order to better test for interactions, all predictors were centered.\(^1\) A parallel set of analyses using adoptee diagnosis of antisocial personality was also conducted. As the dependent variable was a dichotomous (no/yes) variable and all of the predictors were dichotomous, these analyses were conducted utilizing logistic regression.

This study also examined the types of offenses reported by AL and LCP individuals. Of interest in these analyses was the total number of antisocial behaviors as well as the numbers of aggressive antisocial acts and substance use behaviors. Antisocial acts were coded as three separate groups: total antisocial behaviors, aggressive behaviors (e.g., bullying, fighting), and substance use. A 2 × 3 (Classification Group × Antisocial Behavior) MANOVA was utilized to test whether LCP adoptees engaged in a wider variety of offenses than AL adoptees. Antisocial behavior was a within subjects variable. It was hypothesized that LCP adoptees would engage in a greater quantity of antisocial behaviors than would AL adoptees. It was also hypothesized that LCP offenders would engage in more violent and aggressive behaviors than would AL offenders. No difference between AL and LCP offenders was hypothesized for the more status related offense of substance use.

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\(^1\) In the case of dichotomous predictors, regression analyses were conducted using both the centered and uncentered variables. These analyses produced quite similar estimates.
Last, multiple regression was also utilized to test whether the genetic influences on antisocial behavior differed depending upon the domain of behavior examined. It was hypothesized that genetic influences would explain more variation in the antisocial subdomain of aggressive behaviors than in the subdomain of substance use. Two equations were used for these analyses. These were:

\[
\text{ASBA}_{\text{Agg}} = b_0 + b_1(\text{Genetics}) + b_2(\text{Environment}) + e
\]

\[
\text{ASB}_{\text{Subst}} = b_0 + b_1(\text{Genetics}) + b_2(\text{Environment}) + e,
\]

where the dependent variable is aggressive behaviors in the first equation and substance use in the second. As in previous regression analyses, these models also examined Classification group \(\times\) Biological history as well as Biological history \(\times\) Environment interactions.
CHAPTER IV

RESULTS

The results will be organized into four sections. The first section will focus upon the classification of adoptees as Adolescent Limited (AL) and Life-Course-Persistent (LCP) antisocial individuals. The second section provides a simple and direct test of the relationship between group classification and biological history of both psychopathology and antisocial personality utilizing the chi-square statistic. The third section focuses on multiple and logistic regression models examining the relationship of adoptee’s biological history with adolescent antisocial behaviors. Last, the fourth section will examine the relationship of adoptee’s biological history with adolescent antisocial behaviors in the domains of aggressive behaviors and substance use.

Classification of Adoptees as Adolescent-Limited or Life-Course Persistent

Classification of adoptees into AL and LCP groups was dependent upon adoptive parent reports of antisocial behaviors at different ages. Internal consistency was established for three different scales: 1) behaviors committed before adolescence; 2) behaviors committed during adolescence; and, 3) behaviors committed after adolescence. Adolescence was defined as the period between ages 13 and 18. Internal consistency of parental reports of antisocial behaviors was established using the Kuder-Richardson formula 20 (KR-20; Kuder & Richardson, 1937). The KR-20 reliability coefficient is applicable to forms where items are scored in a dichotomous fashion and is analogous to the more generalized Cronbach’s alpha (Anastasi, 1988). Given that antisocial behaviors encompass such a heterogeneous domain, the internal consistency of these scales was
good. The internal reliability of the 11 behaviors assessing childhood antisocial behaviors (behaviors before the age of 13) was $\alpha = .65$. The reliability of the 12 adolescent antisocial behavior items was $\alpha = .80$. The reliability of the seven post-adolescent items was $\alpha = .61$. Interestingly, comparison of the reliability of the adolescent items revealed poor reliability of items among those classified as AL ($\alpha = .45$) and good reliability for those classified as LCP ($\alpha = .80$). One possibility for the discrepancy between these two groups is that endorsement of antisocial behaviors among those classified as LCP was reflective of an underlying antisocial trait but endorsement of items among those classified as AL could be reflective of a number of different possibilities. This notion was tested by conducting separate factor analyses of the 12 items for each group. This analysis appeared to support this idea as behaviors among the LCP group tended to load on one general factor whereas a general factor did not emerge for the AL group.

A complete list of the items in the three antisocial scales is presented in Appendix A. Descriptions of the antisocial items during childhood, during adolescence, and after the age of 18 will now be presented.

**Childhood Antisocial Behavior.** Table 1 presents the frequency of positive reports of antisocial items prior to adolescence for the entire sample as well as for adoptees classified as LCP. This table shows that the majority of parents in the total sample indicated no antisocial behaviors (59.5%). The average number of antisocial items for the entire sample was less than one ($M = 0.77; SD = 1.24$). The average number of antisocial items for the adoptees classified as LCP was greater ($M = 1.92, SD$
= 1.32) than those reported for the rest of the sample; \( t = 8.75, p < .001 \). However, in over 93% of the cases where adoptees were classified as LCP, the number of antisocial behaviors reported was three or fewer.

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A description of the number of positive endorsements for each antisocial item prior to adolescence is presented in Table 2. This table shows that, with a few notable exceptions, the base rate for each behavior was fairly low. The item describing frequent temper tantrums between the ages of 2 and 6 was more frequently endorsed by parents (18.1%) than any other item. For each antisocial item, a greater percentage of parents of adoptees classified as LCP endorsed the antisocial item, though some of these differences did not reach statistical significance. The only exception to this trend was in the case of violent reactions during the ages of two to six. Here, the base rate was quite low for both the total sample and the LCP group (1.9% vs. 1.6%) as to be virtually indistinguishable from one another. It should also be noted that the base rates of antisocial behaviors during the ages of 6 to 12 increase from those of the younger ages. A paired samples \( t \)-test compared the percentage of positive responses before the age of 6 with the percentage of positive responses during the ages of 6 to 12 and found that there was a significantly higher percentage of positive reports during the ages of 6 to 12; \( t = 4.39, p < .001 \). These behaviors were more consistent with the antisocial behaviors reported on during adolescence and after the age of 18. It is therefore compelling support for the
validity of the LCP classification that at least one of these four items was endorsed for the majority (73%) of the LCP group.

Adolescent Antisocial Behaviors. Table 3 presents the frequency of parental reports of antisocial items during adolescence for the entire sample as well as for adoptees classified as AL and LCP. Again, adolescence was defined as ages 13 to 18. This table shows that parents for over one third of the total sample (n = 137) did not endorse any of the antisocial behaviors described for the period of adolescence. The number of adolescent antisocial behaviors were not distributed normally, this will be addressed later in the results (p. 83). The average number of antisocial behaviors endorsed for the entire sample was 2.00 (SD = 2.53). The average number of antisocial items endorsed for the adoptees classified as AL (M = 2.50, SD = 2.10) was greater than those endorsed for the rest of the sample that was not classified as AL, t = 2.79, p < .01. The average number of antisocial items endorsed for the adoptees classified as LCP (M = 4.55, SD = 3.12) was greater than those endorsed for the rest of the sample not classified as LCP, t = 7.28, p < .001. Further, significantly more antisocial items were endorsed for those classified as LCP than those classified as AL, t = 4.58, p < .001. For both the total sample and those classified as AL the relative frequency of adoptees decreased as the number of antisocial behaviors increased. Such distributions are typical of general population surveys of adolescents (e.g., Elliott, Ageton, & Huizinga, 1985). This was not
the case with those classified as LCP as nearly one-third (30.6%) of parents endorsed seven or more antisocial behaviors.

A description of the number of positive endorsements for each antisocial item during adolescence is presented in Table 4. This table shows that being insolent was the most commonly endorsed antisocial item among the total sample as well as those classified as AL and LCP. This item was endorsed by approximately one-third of the total sample (34%). The antisocial item least often endorsed among the total sample was membership in a gang (5.1%). Overall, each antisocial item was endorsed at a low base rate with most items endorsed by less than twenty percent of the total sample. Parents of adoptees classified as LCP were more likely than the total sample to endorse each antisocial item, a significant result when tested by chi-square statistics. Parental endorsement of antisocial behaviors among LCP adoptees occurred at greater than twice the relative frequency for all but three of the antisocial behaviors, namely getting into fights, being insolent, and physically bullying others. Parents of adoptees classified as AL were about as likely to endorse each antisocial item as in the total sample.
Antisocial Behaviors After Age 18. Table 5 presents the frequency of positive parental reports of antisocial items after age 18 for the entire sample as well as for adoptees classified as AL and LCP. First, it should be noted that data for these variables are incomplete. This missing data arises in part from the investigators' failure to consistently ask these items when adoptees were under the age of 20 at the time of the interview. Of the 158 adoptees who were 20 years of age or younger at the time of the interview, valid responses were available for only 61. Examination of the existing data reveals that no post-age-18 antisocial behaviors were endorsed for just over half of the total sample (n = 121). The average number of antisocial items endorsed for this sample was just under one (M = 0.98, SD = 1.32). The average number of antisocial items endorsed for those adoptees classified as AL was quite similar (M = 0.97, SD = 1.30). The average number of items endorsed for those adoptees classified as LCP (M = 2.32, SD = 1.34) was significantly higher than for the rest of the sample; t = 9.79, p < .001.

The five AL cases where parents reported four or more antisocial behaviors merit special attention. Closer examination of these cases revealed that each was classified as AL and not LCP because parents reported no antisocial behaviors prior to adolescence. During adolescence, four of the five cases reported an above average number of antisocial behaviors. For the fifth case, childhood data on antisocial behavior were missing. While a strong argument could be made that these cases represent a persistent
pattern of antisocial behaviors, indeed four of the five meet clinical diagnoses criteria for antisocial personality disorder, and should be reclassified as LCP, a more conservative approach was adopted where these cases were simply dropped from subsequent analyses as outliers. Deletion of these five cases decreased the parental reports of number of antisocial behaviors committed after the age of 18 among the AL group to a mean of 0.66 (SD = .82).

A description of the number of positive endorsements for each antisocial item after age 18 is presented in Table 6. Dropping the five cases as indicated above significantly altered the frequencies of AL antisocial items as these cases were represented multiple times for each item. Therefore, data from the five dropped cases are not included in Table 6.

Table 6 shows that each of the antisocial behaviors after the age of 18 was endorsed at relatively low rates. The most frequently endorsed antisocial item among the total sample (21.1%) was difficulties in getting along with others. The item that was least often endorsed was violent reactions to new situations (2.7%). For each item, the percentage endorsed for those classified as LCP was greater than for the total sample or those classified as AL. Independent t-tests were all significant (p < .05) except in the case of “Does he have any close friends?” (p = .05).
Failures of Classification

A total of 210 adoptees could not be classified into AL or LCP groups for four reasons. First, parents for a significant portion of the total sample (n = 75, 19.4% of the total sample) reported that their child did not engage in any antisocial behaviors. This figure is remarkably consistent with the 20% of adolescents who refrained from any delinquent behavior in the classic study by Gold and Petronio (1980). Second, parents for a significant portion of the total sample endorsed a behavior prior to adolescence but did not endorse any antisocial behaviors during adolescence (n = 73, 18.9%). In over half of these cases, parents reported only one antisocial behavior and in approximately 80% of these cases, parents reported two or fewer behaviors (M = 1.85, SD = 1.27). By far, the item most frequently endorsed prior to adolescence that resulted in an inability to classify as AL or LCP was frequent temper tantrums during the ages of 2 to 6. This item was endorsed by 68 parents. That is, this item was endorsed by 38 more parents in the total sample than among those who were classified into the LCP group. This means that for these 38 families an AL or LCP classification could not be made. It is quite possible that this and other early childhood variables could capture aspects of development besides antisocial behaviors. Evidence that this may be the case may be gathered from the weak correlation between frequent temper tantrums (2 to 6) and self-report of any antisocial behaviors, Φ = .13, p < .05.

Third, a number of parents indicated antisocial behaviors prior to adolescence and during adolescence but did not provide any information or did not have any opportunity to present information about antisocial behaviors after the age of 18 (n = 44, 11.4% of
total sample). With more complete data it is possible that many of these cases could meet the criteria for classification as LCP. However, given the lack of this information, these cases were simply not classified. Last, an additional 18 cases (4.7%) in the total sample could not be classified as parents did not endorse any antisocial behaviors after the age of 18 despite endorsing antisocial behaviors prior to and during adolescence.

Validity of AL and LCP Antisocial Classification

Evidence of the validity of parent reports of antisocial behavior can be gathered from the significant associations between parent reports and clinical diagnoses of antisocial behavior at all ages. The association between parent reports of any antisocial behaviors and adoptee antisocial diagnosis based upon a clinical interview was positive and significant for ages 0 to 2 (Φ = .17, p < .01), 2 to 6 (Φ = .17, p < .01), 6 to 12 (Φ = .16, p < .05), 13 to 18 (Φ = .22, p < .001), and over the age of 18 (Φ = .19, p < .01). Similarly, point-biserial correlations between the adoptee clinical interview and the number of antisocial items endorsed by adoptive parents provide an indication of the concurrent validity of parent reports; parents of adoptees who were diagnosed as antisocial reported a greater number of antisocial behaviors. Table 7 shows that all of the associations between parent reports of the number of behaviors and the adoptee clinical interview were positive and eight of the ten correlations were statistically significant. This table also shows that the relationships between parent reports of antisocial behavior and the clinical interview are highest during adolescence and after the age of 18. These coefficients range from r_pb = .33 (p < .001) to r_pb = .46 (p < .001).
Additionally, correlations were computed between each of the parent reports of antisocial behaviors and SES, as assessed by the occupation factor of the Hollingshead Two-factor Index of Social Position-Occupation (Hollingshead, 1947) of the adoptive family, and with age of adoptee. Significant relationships would be problematic as they would indicate that parental reports were influenced by these parental characteristics or they in fact affected antisocial behavior – a result that was not hypothesized. However, no statistically significant relationships were found between SES and parent reports of antisocial behaviors. Similarly, age of adoptee was largely uncorrelated with parent reports of antisocial behaviors. One notable exception was the association between experience with drugs and age, \( r_{pb} = -0.24, p < .001 \). Rather than raising a warning flag about the veracity of parent reports, this result accurately reflects secular trends in drug use as national data indicate drug use among adolescents peaked in the early 1980s (Johnston, O’Malley, & Johnston, 2002; O’Malley, Bachman, & Johnston, 1984).

**Gender Differences in Antisocial Behaviors**

Further evidence of the validity of parent reports of antisocial behaviors can also be garnered from examination of gender differences. At all ages, parents reported that males committed a greater number of antisocial behaviors than did females. This result is consistent with a large body of literature that finds, “gender differences [in antisocial behaviors] appear to be invariant over time and space” (Gottfredson & Hirschi, 1990; p.
This study found that prior to adolescence, parents reported a higher number of antisocial behaviors for boys \((M = .92, SD = 1.37)\) than girls \((M = .57, SD = 1.02)\), \(t = 2.84, p < .01\). During adolescence parents reported males \((M = 2.42, SD = 2.79)\) committed more antisocial acts than did females \((M = 1.29, SD = 1.71)\), \(t = 4.84, p < .001\). Last, after the age of 18, parents reported males \((M = 1.06, SD = 1.35)\) committed more antisocial acts than did females \((M = .65, SD = .97)\), \(t = 2.70, p < .01\).

It should be noted that despite the consistent presence of mean level gender differences, analyses were not run separately for males and females. This was primarily due to two reasons. First, mean level differences need not imply different processes (e.g., Gilson & Rowe, 2002; Rowe, Vazsonyi, & Flannery, 1994). These two papers demonstrate that while group mean level differences were found for such variables as achievement, delinquency, and sexual behavior, the developmental processes underlying these phenomena were identical across groups. Strong evidence for similarity of process between males and females is also demonstrated in the literature. Rodgers, Buster, and Rowe (2001) demonstrated that genetic influences on delinquency are stable across both gender and racial groups, producing similar estimates – this despite the fact that mean level differences exist. Second, practical limitations with the sample size would make separate analyses unfeasible.

**Test of Independence Between AL and LCP Classifications and Biological History**

The first hypothesis tested predicted that classification of AL and LCP individuals would not be independent of biological history of psychopathology and antisocial behavior. This hypothesis was tested using the chi-square statistic. Specifically, it was
hypothesized that those classified as LCP individuals would be more likely to have a biological history of psychopathology or antisocial behavior than those classified as AL. The first set of analyses, then, examined two $2 \times 2$ contingency tables. In these analyses, a dichotomous variable was created based upon AL and LCP classification. Adoptees classified as AL were given a code of 0 and adoptees classified as LCP were given a code of 1. This variable, representing Moffitt's two groups, was compared with adoptee biological history of psychopathology and with adoptee biological history of antisocial behaviors. In the first $2 \times 2$ table utilizing biological history of psychopathology, those adoptees who did not have any biological parent with psychopathology were given a code of 0. Those adoptees who had a biological parent with history of psychopathology were given a code of 1. Similarly, in the $2 \times 2$ table utilizing antisocial behavior, adoptees whose biological parents did not have any history of antisocial personality were given a code of 0. Those adoptees that had a biological parent with a history of antisocial personality were given a code of 1.

Insert Table 8 about here

Table 8 shows the joint distribution of individuals classified as AL and LCP by biological history of psychopathology. Classification into the two groups was not independent of a biological history of psychopathology as indicated by a significant chi-square, $\chi^2 (1, N = 151) = 8.84, p < .01$. Further, as hypothesized, more LCP individuals had a history of psychopathology than would be expected by chance. The standardized
residual, the standardized difference between the observed and expected frequency that has a mean of 0 and standard deviation of 1, was calculated for each cell. The standardized residual for the joint occurrence of LCP classification and biological history of psychopathology was 1.7. This indicated that the observed frequency of being classified as LCP and having a biological history of psychopathology was 1.7 standard deviations more likely to occur than was expected. Similarly, the observed frequency of being classified as AL and not having a biological history of psychopathology was 1.2 standard deviations more likely to occur than was expected.

Table 9 illustrates a similar joint frequency distribution. However, this time the measure of biological history was limited to antisocial behaviors. As before, AL and LCP classification was not independent of biological history, $\chi^2 (1, N = 172) = 14.22, p < .001$. As expected, more LCP individuals had a biological history of antisocial behaviors than would be expected by chance. The standardized residual for LCP individuals with a biological history of antisocial behaviors was 2.7, indicating that being classified as LCP and having a biological history of psychopathology occurred at a rate that was 2.7 standard deviations above the expected frequency. Again, the observed frequency of being classified as AL and not having a biological history of antisocial personality was 1.1 standard deviations more likely to occur than was expected.

________________________________________________________________________

Insert Table 9 about here

________________________________________________________________________
Influence of Biological Parent Characteristics on Adoptees

Next, two sets of regression analyses were presented examining the influence of biological history and group classification on antisocial behavior. These analyses took advantage of data on two separate outcomes (i.e., number of adolescent antisocial behaviors and diagnosis of antisocial personality disorder in adulthood) and data from multiple reporters (i.e., adoptive parent, adoptee) to provide a more robust test of relationships. Results that were significant across both parent and adoptee reports, for example, provide a powerful test of a relationship as well as support for the concurrent validity of reports. The first set of analyses were conducted using multiple regression. In these equations, parental report of number of antisocial behaviors was the dependent variable and biological history, adverse environment, and group classification were entered as predictors. Next, logistic regression models were utilized. Here, clinical diagnosis of adoptee antisocial behavior was the dependent variable and biological history, adverse environment, and group classification were entered as predictors.

Table 10 presents the intercorrelations of the predictor variables with parent reports of antisocial behaviors and clinical diagnoses.

| Insert Table 10 about here |

This table showed that biological history of psychopathology was positively correlated with parent reports of antisocial behaviors during adolescence ($r_{pb} (318) = .18, p < .01$). However, the association of biological history of psychopathology with clinical diagnosis
of antisocial behavior did not reach significance ($r_{pb} (262) = .12, p = .06$). Biological history of antisocial personality was positively associated with both parent reports of antisocial behaviors ($r_{pb} (369) = .16, p < .01$) and clinical diagnosis of antisocial behaviors ($r_{pb} (269) = .17, p < .01$). AL/LCP classification was positively associated with both parent reports of antisocial behaviors ($r_{pb} (170) = .40, p < .001$) and clinical diagnosis of antisocial behaviors ($\Phi (120) = .30, p < .01$). That is, as hypothesized, classification as LCP was associated with both higher parental reports of antisocial behaviors and a greater likelihood of clinical diagnosis of antisocial behavior.

**Multiple Regression With Parent Reports as Dependent Variable.** Prior to running the multiple regression models, a series of data transformations were made. First, parental reports of the number of antisocial behaviors were not distributed normally. Specifically, the number of antisocial behaviors was positively skewed ($1.69, SE = .13$), and the standard deviation was greater than the mean ($M = 1.95, SD = 2.47$). A square root transformation reduced the skewness of the distribution to 0.41 as well as the standard deviation ($M = 1.03, SD = .94$). The other transformation involved the predictors. As suggested by Jaccard, Turrisi, and Wan (1990), predictors used to create interaction terms were centered to reduce nonessential multicollinearity and thus allow for a fair test of interaction effects.

Table 11 contains both the standardized and unstandardized beta coefficients for the regression models utilizing biological history of psychopathology as a predictor of parent reports of antisocial behaviors. At Step 1, biological history of psychopathology and adverse adoptive home environment were entered. This model showed that variation
in parent reports of antisocial behaviors could be predicted by these variables, $R^2 = .06$, $p < .01$. At this step, both predictors were significant, indicating that both presence of biological history of psychopathology ($\beta = .17$, $p < .05$) and adverse home environment ($\beta = .17$, $p < .05$) increased the number of adolescent antisocial behaviors reported by parents. Step 2 added AL/LCP group classification as a predictor. When this predictor was added both biological history of psychopathology and adverse adoptive home environment ceased to be significant. While the main effect of AL/LCP classification was significant ($\beta = .34$, $p < .001$), the addition, in Step 3, of an interaction term consisting of the product of the centered biological history and AL/LCP classification terms was not significant ($\beta = .09$, $p = .27$).

Insert Table 11 about here

Table 12 contains both the standardized and unstandardized beta coefficients for the regression models utilizing biological history of antisocial personality as a predictor of parent reports of antisocial behaviors. At Step 1, biological history of antisocial personality and adverse home environment were entered. As in the first model, variables entered at this step predicted significant variation in our dependent variable, $R^2 = .06$ ($p < .01$). Unlike the previous model, home environment failed to predict variation in antisocial behavior. Step 2 added AL/LCP classification as a predictor ($\beta = .36$, $p < .001$). Again, addition of this variable affected the relative contributions of the other predictors, rendering biological history nonsignificant. Last, in Step 3, an interaction
term consisting of the product of the centered biological history of antisocial personality and the AL/LCP classification was not significant ($\beta = -0.07, p = 0.37$).

Insert Table 12 about here

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**Logistic Regressions with Adoptee Antisocial Diagnosis as Dependent Variable.**

Table 13 contains logistic regression models utilizing biological history of psychopathology as a predictor of adoptee diagnosis of antisocial personality, a binary outcome. The first model tested included only the history of psychopathology variable and adverse home environment. This model was nonsignificant (see Table 13). Model 2 added AL/LCP classification as a predictor. This model was significant, with AL/LCP classification ($\chi^2 = 7.53, p < .01$) as the only significant predictor in this model.

According to Model 2, being classified as LCP increased the odds of being diagnosed as antisocial by a factor of 3.58 over those classified as AL. Alternatively expressed, LCP classification was associated with a 258% increase in the probability of being diagnosed with antisocial personality disorder over those classified as AL. Model 3 adds an interaction term, History of Psychopathology $\times$ AL/LCP classification, however, this interaction was not significant ($\chi^2 = .48, p = .49$). Logistic regression models do not provide a change in $R^2$ statistic, however, attention to the chi-square statistic can provide an indication of the significance of model change with the inclusion of additional predictors. The change in chi-square between Model 1 and Model 2 ($\Delta \chi^2 = 5.16$) with 1 degree of freedom was significant ($p < .05$), indicating that the inclusion of the AL/LCP
classification variable significantly improved the model. The inclusion of the interaction term in Model 3 did not result in a significant improvement in the model, \( \Delta \chi^2 (1) = 0.49, \ p = .48 \).

Table 14 contains logistic regression models utilizing biological history of antisocial personality as a predictor of adoptee diagnosis of antisocial personality.\(^2\) The first model tested included only biological history of antisocial personality and adverse adoptive home environment. While this model was significant, only biological history and not adverse home environment contributed to the model (see Table 14). Model 1 found that biological history of antisocial personality resulted in a 196% increase in the odds of an adoptee being diagnosed as antisocial (\( p < .01 \)). Model 2 added AL/LCP classification as a predictor. The addition of this variable resulted in biological history no longer being significant (\( \chi^2 = 3.53, \ p = .06 \)). In this model, those adoptees classified as LCP were 220% more likely to be diagnosed as antisocial than those classified as AL (\( p < .01 \)). Model 3 added an interaction term, History of Antisocial Behavior \( \times \) AL/LCP classification, however, this interaction term was not statistically significant (\( \chi^2 = .65, \ p = .42 \)). The inclusion of AL/LCP classification as a predictor in Model 2 yielded a

\(^2\) Model 1 of all logistic regressions were also reanalyzed, restricting the samples to only adoptees classified as AL or LCP. In each instance, parameter estimates were very similar, leading to the same substantive conclusions.
significant increase in the model, $\Delta \chi^2 (1) = 5.60, p < .05$. The inclusion of the interaction term in Model 3 yielded no significant change in the model, $\Delta \chi^2 (1) = 0.73, p = .39$.

Influence of Biological History and Adoptive Environment on Group Classification

An alternative method to test whether biological history influenced antisocial behavior in adoptees was conducted wherein antisocial group classification was the dependent variable. Table 15 contains logistic regression models when biological history of psychopathology was entered as a predictor along with adoptive home environment. In Model 1, the dependent variable was adoptees classified as LCP contrasted with all others. In Model 2, the dependent variable was adoptees classified as LCP contrasted with adoptees classified as AL. In Model 3, the dependent variable was adoptees classified as AL contrasted with adoptees with no reported history of antisocial behaviors. Given that multiple tests of correlated predictors were being conducted, a more conservative alpha level of $p < .01$ was observed. Taking this precaution against Type I error had little impact as noted below.

Presence of biological history of psychopathology increased the odds of being classified as LCP by a factor of 3.14 ($p < .001$) compared to those with no biological
history of psychopathology (see Table 15, Model 1). Alternatively stated, a biological history of psychopathology resulted in a 214% increase in the odds of being classified as LCP. Similarly, when the dependent variable was a contrast between adoptees classified as LCP and AL, biological history of psychopathology increased the odds of being classified by a factor of 2.73 ($p < .01$) compared to those with no biological history of psychopathology (see Table 15, Model 2). Model 3 found no significant effect for biological history of psychopathology when AL adoptees were contrasted with those adoptees whose parents reported they did not engage in any antisocial behaviors. That is, AL adoptees were no more likely to have a biological history of psychopathology than those adoptees who refrained from all antisocial behaviors. In each model, the influence of adoptive environment was not statistically significant.

Results were quite consistent when the same set of analyses were carried out with biological history of antisocial personality replacing biological history of psychopathology (see Table 16). Model 1, contrasting LCP adoptees with all others (OR = 3.76, $p < .001$), and Model 2, contrasting LCP adoptees with AL adoptees (OR = 4.02, $p < .01$), found a significant effect of biological history of antisocial personality. Model 3, contrasting AL adoptees with those adoptees whose parents did not report any antisocial behaviors, again failed to find an influence of biology. For each of the three models, adverse adoptive environment was not statistically significant.

 Insert Table 16 about here
What About Biology x Environment Effects?

Consistently, in the above results, the effect of environment has been dealt with in a rather cursory manner. This is in large part due to the fact that the effect of environment has been statistically significant in only one of the above analyses (see Table 11, Step 1). Nevertheless, despite minimal evidence of any main effect for adverse home environment as presently operationalized, it is still possible that adverse environment, again as operationalized, could interact with biological history. To test for this possibility, interaction terms were created by taking the product of the centered adoptive home environment variable and the two centered biological variables, biological history of psychopathology and biological history of antisocial personality.

The significance of these interaction terms was tested by adding these product terms to the final steps of the above 10 regression models. Biological history x Adoptive environment models were run predicting parent reports of antisocial behaviors (two models), adoptee diagnosis of antisocial behavior (two models), and group classification (6 models). In one of the models, however, the interaction term could not be tested. In this model, where biological history of antisocial personality was regressed on adoptee diagnosis of antisocial personality, the introduction of the interaction term resulted in insufficient sample size. In this model, only two adoptees had both an adverse adoptive home environment and a biological history of antisocial personality. This mean that Biological history x Adoptive environment interactions were tested in nine models (see Tables 17 to 25). For these nine models, no significant interactions were found between biological history and environment. Bivariate correlations were computed between the
interaction terms and biological history of psychopathology, biological history of antisocial personality, and adverse home environment. The largest bivariate correlation was between the interaction term Biological history of psychopathology × Adoptive environment and the main effect of Adoptive environment, \( r = .16, p < .01 \). Therefore, the relatively low correlations indicate that failure to find an effect of an interaction is not merely a function of multicollinearity (Aiken & West, 1991).

Tables 17 and 18 represent the addition of the interaction term to the third step of the models presented in tables 11 and 12, respectively. Table 19 adds the interaction term to the third model that was presented in table 13. Tables 20 to 22 add the interaction term to Models 1 to 3 that are presented in table 15. Tables 23 to 25 add the interaction term to Models 1 to 3 that are presented in table 16.

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Insert Tables 17 to 25 about here

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**Domain-Specific Behaviors**

The next series of analyses performed examined domain-specific antisocial behaviors. Domain specific behaviors were obtained only from parent reports. Table 26 presents the mean number of antisocial behaviors reported by parents. As shown in this table, and as reported previously on page 72, the total number of antisocial behaviors were higher among those adoptees classified as LCP than those classified as AL. This result was consistent with the hypothesis. However, contrary to what was expected, there was no significant difference in mean level of aggressive behaviors between the LCP and
AL groups. Also contrary to what was hypothesized, adoptees classified as LCP engaged in significantly more substance use behaviors than those classified as AL.

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Insert Table 26 about here

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The first set of domain specific regression analyses utilized aggressive behaviors as the dependent variable. These analyses were testing the hypothesis that genetic influences would be greater for aggressive behaviors than other forms of antisocial behavior. Aggressive behavior was operationalized as engaging in either physical fights or bullying behavior during adolescence. As only two aggressive items were included and only 20 parents reported their children engaged in both behaviors, aggressive behavior was given a dichotomous code. Aggressive behavior was coded 0 if neither fighting nor bullying were reported or as 1 if at least one of these two items were reported.

Table 27 contains results from three logistic regression models predicting aggressive behavior by biological history of psychopathology. Model 1 contained biological history of psychopathology and adoptive environment as predictors. Model 2 added AL/LCP classification as a predictor. Model 3 added a Biological history of psychopathology × Adoptive environment interaction term. As shown in Table 26, none of these models were statistically significant.
Table 28 contains three models predicting aggressive behavior with biological history of antisocial personality. Model 1 contained biological history of antisocial personality and adoptive environment as predictors. Model 2 added AL/LCP classification as a predictor. Model 3 added a Biological history of antisocial personality x Adoptive environment interaction term. Again, none of these models predicting aggression were statistically significant.

The final set of analyses utilized substance use as the dependent variable. These analyses examined whether there would be genetic influence on substance use. It was hypothesized that substance use would be more environmentally driven and thus occur with equal frequency among both AL and LCP adoptees. Substance use was measured by parental reports to a single item, “Did he (sic) have any experience with drugs or alcohol?” Table 29 contains three models predicting drug use with biological history of psychopathology. Model 1 contained biological history of psychopathology and adoptive environment as predictors. Model 2 added AL/LCP classification as a predictor. Model 3 added a biological history of psychopathology x adoptive environment interaction term.
While the AL/LCP classification variable was significant in Model 2, none of these models predicting substance use were significant.

Table 30 contains three models predicting substance use with biological history of antisocial personality. Model 1 contained biological history of antisocial personality and adoptive environment as predictors. Model 2 added AL/LCP classification as a predictor. Model 3 added a biological history of antisocial personality × adoptive environment interaction term. The inclusion of AL/LCP classification in Model 2 yielded a significant effect. In this model, classification as LCP predicted a significant increase in the likelihood of using substances, contrary to the hypothesis of no difference. The inclusion of the interaction term in Model 3 was not significant and did not significantly affect the fit of the model, $\Delta \chi^2 (1) = 1.71, p = .19$. 

Insert Table 29 about here

Insert Table 30 about here
CHAPTER V
DISCUSSION

The present study examined antisocial behavior from a developmental perspective. In particular, this study examined the genetic influence on antisocial behavior among a group of adoptees, contrasting a group that engaged in antisocial behavior only during adolescence with a group that engaged in antisocial behavior during adolescence and at other developmental stages. This final chapter discusses the results and their implications. Limitations of the research and directions for future research in this area are presented. Before turning to this discussion, however, a brief review of the rationale and an overview of the organization of the study will be presented.

Rationale and Organization

The study was conducted with the focus of accomplishing two main goals. First, the study was designed to test a popular theory of antisocial behavior. This theory, put forth most completely by Moffitt (1993) and DiLalla and Gottesman (1989) is that two types of antisocial behaviors are committed by two types of qualitatively different individuals. The first of these two groups limit their antisocial behaviors to adolescence. This group engages in what Moffitt refers to as Adolescent Limited (AL) antisocial behavior. In contrast to this group, a small but significant minority of individuals persistently engage in antisocial behaviors from a very early age throughout their lifespan. This group engages in what Moffitt refers to as Life-Course-Persistent (LCP) antisocial behavior. These two groups differ not merely in what stage they commit these behaviors but rather in the underlying motivation for their behaviors. It is theorized that
the motivation behind AL antisocial behavior is social influences. In the case of LCP antisocial behavior, the explanation offered is one of pathology induced by individual characteristics.

Second, the present study attempted to provide an explanation for the body of behavioral genetic research on adolescent antisocial behavior that has produced frequently inconsistent results. While many studies have found consistent evidence for the heritability of antisocial behavior among adult populations, only weak evidence exists for the heritability of antisocial behavior among adolescents (DiLalla & Gottesman, 1989). The present study links this developmental theory of adolescent antisocial behaviors with the inconsistent behavioral genetic results by utilizing an adoption design to examine the genetic influences on antisocial behaviors committed by individuals who limit these acts to adolescence and those who persist across their lifespan. If Moffitt's (1993) theory is correct, the presence of AL and LCP groups could explain the inconsistent evidence of heritability of antisocial behavior among adolescents.

Specifically, because AL behaviors are socially driven there should be little evidence of genetic influence on AL individuals' antisocial behaviors. However, the same theory holds that LCP antisocial behavior is the result of individual characteristics and evidence of genetic influence should be found among this much smaller subgroup of individuals.

The present study applies the rarely utilized full adoption design (Cadoret, 1986; Rowe, 1994) to examine genetic influences on adolescent antisocial behavior. Using archival data from the Cadoret (1988) adoption studies, the present study used information on adoptees who were separated at birth from their biological parents, their
biological parents, and their adoptive family. This study first utilized adoptive parent reports of adoptee antisocial behaviors at three different periods of the lifespan to categorize adoptees as AL or LCP individuals. Next, analyses were conducted to examine genetic influences on AL and LCP behaviors. Last, analyses were restricted to examine antisocial domains of aggression and substance use.

**AL and LCP Classification**

Categorization of adoptee antisocial behavior was contingent upon adoptive parent reports of behaviors. Adoptive parents were utilized as respondents as they were considered the most accurate reporters of childhood behaviors. Further, parent reports were correlated with adoptee self-reports of behaviors. While it was not expected that many antisocial behaviors would be reported prior to adolescence, it was expected that antisocial behaviors would be frequently reported during adolescence. As noted in Chapter 2, previous research has shown that adolescents engage in antisocial behaviors with “normative regularity” (Arnett, 1992). Estimates of how pervasive these antisocial behaviors are during adolescence seem to converge around 80% to 90% (Farrington, 1986; Gold & Petronio, 1980). However, in this sample, only 64% of the parents reported that their children had engaged in any of the antisocial items listed for the ages of 13 to 18. While it is possible that this rate accurately reflects the total number of adoptees who engaged in any antisocial behavior during this period, it is more likely that this figure is considerably lower than reality. Several explanations readily present themselves. First, the list of antisocial behaviors was far from exhaustive. It is quite possible that adolescents in this sample were engaging in antisocial behaviors but not the
behaviors presented as options to the parents during their interview. Second, the questions required that parents respond in a simple yes/no fashion; this forced choice format may have lowered the number of positive reports as illustrated by classical signal detection theory (Nunnally & Bernstein, 1994). For example, parents whose children exhibited only a low level of bullying behavior may, when presented with the yes/no dichotomy, have elected not to respond in the affirmative as they may have felt that such a categorical response would not accurately reflect their child's typical behavior. Third, many of the antisocial behaviors actually committed by adolescents go undetected by parents (e.g., Sourander, Helstelae & Helenius, 1999). Indeed, many of the antisocial items available to parents required that the adoptee have been caught in the antisocial act and parents notified (e.g., suspended, trouble with juvenile authorities). It should also be noted that self-reports of delinquent and criminal behaviors tend always to be higher than arrest records, parent reports, or teacher reports (Gottfredson & Hirschi, 1990; Youngstrom, Loeber, & Stouthamer-Loeber, 2000).

While reports of behaviors were lower than expected, there appears to be considerable evidence that these parent reports are both reliable and valid. First, internal consistency of behaviors, assessed by the Kuder-Richardson formula 20, were quite good, ranging from a low of .61 for the 7 behaviors after the age of 18 to a high of .84 for the 12 items that assessed behaviors between the ages of 13 and 18. Unlike some scales that may focus on a rather homogenous construct, antisocial behavior covers a rather broad range of behaviors. All other things being equal, the more homogenous the construct, the higher the interitem reliability should be (Anastasi, 1988). Given that antisocial behavior
is a fairly heterogenous construct the internal consistency reliability of parent reports is quite good. Evidence for the validity of parental reports may be inferred from the fact that parent reports of adolescent behaviors are highly correlated with adoptees' self-reports of antisocial behaviors (see Table 10). In addition, parent reports of behaviors seem to have been unaffected by other factors such as the time period they occurred and parent characteristics such as SES (see page 78). Were reports associated with these characteristics, it would raise the possibility that reports in part reflected these characteristics.

It is important to note that a large number of adoptees (54%) could not be classified as either AL or LCP. The most frequent reason was that parents reported that their child did not engage in any antisocial behavior during any time period. Little needs to be said about this group as there is little ambiguity about their not belonging in either the AL or LCP group. Obviously, a behavior that occurs with "normative regularity" does not require that everyone engage in that behavior. The presence of this group as well as the frequency, about 20%, is consistent with other research that has found evidence of those who self-reported as having abstained from antisocial behavior (e.g., Gold & Petronio, 1980).

Another similarly-sized group that failed to be classified into one of the two groups, however, is more problematic. The reason this group could not be classified was that they engaged in antisocial behaviors before the age of 13 only. This is problematic because these individuals may not have been classified for a number of different reasons. First, it is possible that these individuals could represent a different type of individual that
engages in antisocial behavior only prior to adolescence and is therefore not represented in the AL/LCP taxonomy. Alternatively, this group could represent a group of adoptees whose parents endorsed a childhood item that did not truly represent antisocial behavior. Last, this group of adoptees could represent a group of individuals that was engaging in AL antisocial behavior prior to the age of 13 but had matured out by these behaviors by age of 13.

Most commonly, this group contained individuals who only reported frequent temper tantrums during the ages of 2 to 6. While previous research has linked temper tantrums among children as young as three with adult criminality (e.g., Stevenson & Goodman, 2001) it is possible that this item does not represent antisocial behavior but, for these individuals, may capture a group of colicky and fussy two-year-olds and not six-year-olds who repeatedly displays angry outbursts. Supporting evidence for this explanation can be found in the rather low correlation between this behavior and adoptees’ self-reports of antisocial behavior. In these instances, the failure to classify these individuals does not represent a threat to the validity of the AL and LCP groups as they clearly do not belong to either group. However, in some instances these individuals could actually be engaging in AL antisocial behaviors. Consider, for example, an adoptee whose parents reported they were reckless and destructive in school during the ages of 6 to 12 but did not report any antisocial behaviors during the age of 13 to 18. If these behaviors were committed during the ages of 11 or 12 and the adoptee “matured” out of these behaviors by the age of 13 they would accurately reflect what is intended by
the AL classification. Additional information would need to be gathered to ensure that these individuals were accurately excluded for the AL group classification.

The end result of the lower than expected reports of antisocial behavior as well as the failure to classify a significant portion of the sample into either the AL or LCP group, in large part caused by low reports of antisocial behavior, contributed to a classification scheme that is fairly conservative. Individuals who engaged in lower levels of antisocial behaviors are probably those that were the most likely to be unclassified and are thus excluded from many of the subsequent analyses. As discussed below, those who engaged in the lowest levels of antisocial behavior were far more likely to be classified as AL. The exclusion of this population resulted in the potential differences between the AL and LCP groups being muted and thus made for more conservative tests of AL/LCP differences.

The above conclusion can be reached because the results showed that individuals classified as LCP engaged in significantly more antisocial behaviors during adolescence than those individuals classified as AL. This difference coupled with the fact that AL/LCP classification was dependent only upon reports of antisocial behaviors committed outside of adolescence indicates that information about adolescent antisocial behaviors can be useful in discriminating between normative and more pathological behaviors. This finding is consistent with early research that found quantity of antisocial behavior during adolescence is predictive of the subsequent development of antisocial personality disorder (Robins, 1966). While not entirely unexpected, this result
nevertheless led to some complications in regression analyses that will be discussed below.

**Genetic Influence on Antisocial Behavior**

In this and in subsequent analyses biological history was defined two ways. The first way biological history was defined was as evidence of biological parent psychopathology that included antisocial behavior, behavioral disorders, and severe drug and alcohol disorders. The second way biological history was defined was as antisocial behavior that met a clinical definition of antisocial personality disorder. Biological history was classified in these two manners arising over concerns of sample cell sizes for some of the analyses when biological history was limited to just antisocial behavior. Indeed, one of the planned analyses utilizing biological history of antisocial behavior could not be conducted due to this issue. Biological history of psychopathology was also included as a predictor as this group consisted of biological parents who were diagnosed with a behavioral disorder as well as with severe drug and alcohol disorders. These biological parents represented a population that often engaged in antisocial behaviors but in insufficient quantity to receive a formal diagnosis of antisocial personality disorder (Cadoret & Cain, 1983). By including this group in analyses the sample size of biological parents engaging in antisocial behaviors could be increased, albeit by including a greater number of biological parents with lower levels of antisocial behaviors. By including biological parents with lower levels of antisocial behaviors the end result is to again conduct more conservative tests.
Support for Differences in AL and LCP Classification Etiology

The first research question investigated by this study was whether AL/LCP classification was independent of the biological parents' history. Both the analysis that utilized the biological history of psychopathology variable and the analysis that utilized the biological history of antisocial behavior produced similar results. Both analyses confirmed the hypothesis that adoptees classified as LCP were more likely to have a biological parent with a history of either psychopathology or antisocial personality. While LCP adoptees were more likely than expected to have a biological history of either psychopathology or antisocial behavior, AL individuals were less likely to have a biological history of either psychopathology or antisocial behavior. These initial findings provided the first indication of a genetic basis for LCP behavior but not for AL behavior.

Having established that biological history was related to classification as either AL or LCP, analyses moved toward an examination of whether genetic influences were higher for those adoptees classified as LCP than for those classified as AL. The first set of these analyses utilized parental reports of adoptee antisocial behaviors during adolescence as the dependent variable. A series of hierarchical regression equations were utilized to examine this question. The biological history variable was significant in the first step of both analyses but was not significant when the AL/LCP classification variable was added. An interaction term, created by taking the product of the AL/LCP classification variable and the biological history variables, was entered into the equation after the main effects. This interaction term was not significant, either when psychopathology or antisocial behavior were used. Thus, these analyses failed to confirm
the second hypothesis that LCP individuals would exhibit higher genetic influences on their antisocial behavior than AL individuals.

A similar set of logistic regression analyses were conducted where adoptee diagnosis of antisocial behavior was the dependent variable. These analyses found a significant main effect for biological history of antisocial behavior, but not for the more conservative analysis utilizing psychopathology. Again, when the classification variable was included in a second model, the contribution of the biological history variable was reduced and became nonsignificant. The interaction term entered in the last model was not significant. Thus, these analyses utilizing the adoptee clinical interview failed to confirm the second hypothesis that LCP individuals would exhibit higher genetic influences on their antisocial behavior than AL individuals.

Why did we fail to find evidence of an interaction? The most straightforward explanation is that we failed to find them because they are not there. However, there is sufficient reason to believe that an interaction could be present but is simply undetected by the above analytic strategy. This is because interactions in non-experimental designs are notoriously difficult to detect when compared to experimental designs with orthogonal factors (McClelland & Judd, 1993). The predictors in this study are not orthogonal. As shown in Table 10, the predictors are significantly correlated. It is well acknowledged that interactions in regression models using correlational designs have very low statistical power (Jaccard, Turrisi, & Wan, 1990). In experimental designs, the treatment and control groups are identical except for the manipulation of the independent variables (e.g., exposure to a condition) and cells have equal numbers of subjects. These
characteristics lead to orthogonal main effects and interactions, which maximize the chances of finding a significant interaction, should one actually exist. In contrast, non-experimental designs must work with the joint distribution of predictors found in the data. With naturally correlated predictors, in contrast to orthogonal factors, there may be relatively few individuals who are high on one predictor (e.g., classified as LCP) and low on another (e.g., biological history). Therefore, in comparison to experimental designs, McClelland and Judd (1993) suggested that interactions are difficult to identify in non-experimental studies and, when detected, typically account for only 1% to 3% of the total variation.

Rather than simply concluding that analyses failed to find evidence of a higher genetic influence among LCP individuals than AL individuals, an alternative analytic strategy was employed where group classification was entered as the dependent variable and biological history was included as a predictor. These analyses attempted to answer a similar question by testing whether there is a main effect of biological history on group classification. The results consistently indicate support for a biological factor, either biological history of antisocial behavior or of psychopathology, as a predictor of LCP classification. We see this both when the dependent variable is LCP classification contrasted with all others as well as when LCP classification is contrasted with AL classification. While this is strong support for genetic influences on LCP behaviors, we do not see any similar evidence of genetic influence on those adoptees classified as AL. When the dependent variable was AL antisocial classification contrasted with those adoptees whose parents report they evidenced no antisocial behavior there is no
significant effect of biological history, indicating that AL individuals were no more likely to have had a biological history of psychopathology or antisocial behavior than those individuals who did not express any antisocial behaviors.

**Genetic Influence on Domain Specific Behaviors**

As hypothesized, adoptees classified as LCP engaged in significantly more antisocial behaviors than those adoptees classified as AL. However, when the specific domains of aggressive behaviors and substance use are examined, the results were not consistent with Moffitt's (1993) theorized motivations for each group's antisocial behaviors. First, there was not a significant difference in either the mean level of aggressive behaviors when analyzed by the two classification groups nor was there any effect of group classification when it was entered as a predictor of aggressive behavior in a regression equation. This last result was also inconsistent with previous research on adolescent antisocial behavior that has found aggressive behaviors to be more heritable than other forms of antisocial behavior (e.g., Achenbach, 1993). One possibility for the failure to find the expected effect is that the aggressive behaviors measured (e.g., bullying) were relatively innocuous compared with those behaviors used in other studies of aggression (e.g., Farrington, 1975; Farrington & Loeber, 2000; Piquero, Paternoster, Mazerolle, Brame, & Dean, 1999; Rushton, 1996). The anticipated result, that aggressive behavior would include "crimes often committed by lone offenders [and include] more victim-oriented offenses" (Moffitt, 1993, p. 695) may not have occurred because the behaviors measured were not severe enough to either be condemned by peers or result in alienation from peers. Indeed, bullying behaviors may have served an instrumental social
fashion such as establishing peer hierarchy or gaining peer acceptance. For example, Dodge, Coie, Pettit and Price (1990) found that among first grades, aggressive males were more popular though this effect faded by the third grade. Another study of early adolescents indicated aggressive peer behaviors may serve to establish peer affiliation during the transition to middle school (Pellegrini & Bartini, 2001). Far from being rare, aggressive behaviors were found in about 20% of the entire sample. In order to find the hypothesized relationship of aggression to LCP classification more serious, and thus more rare, indices aggressive behavior may be necessary.

While it was hypothesized that there would be no difference between the AL and LCP groups in regards to substance use, there was some evidence of both a biological influence on substance use as well as evidence that LCP adoptees were more likely to engage in substance use. Interestingly, biological history of psychopathology which includes substance abuse and dependence as a diagnosis was not significant whereas biological history of antisocial personality was. One explanation for why parents were more likely to report substance use among LCP than AL adoptees could be that LCP substance use could be qualitatively different than AL use. Use among LCP adoptees could be more severe and abusive and thus more likely to be noted. It is possible that AL adoptee substance use was less abusive and more social and thus less likely to be detected by parents. The nature of the present data, however, do not allow these ideas to be tested.

**Gene x Environment Interactions**

No evidence of any Gene x Environment interactions was found in this study. This runs counter to the results of a number of adoption studies that have been previously
described in this paper (e.g., Cloninger et al., 1982; Mednick et al., 1984). Why was there no evidence of interactions here? It is difficult to say. As mentioned previously, interactions are difficult to detect in correlational designs (Jaccard, Turrisi, & Wan, 1990). In this study, the power available to detect interactions was consistently quite low. The effect sizes of the interactions, estimated from equations provided by Aiken and West (1991), were quite small, generally ranging from $f^2 = .01$ to .02 and the power to detect interactions was calculated at between .14 to .20 using GPOWER 2.0 (Erdfelder, Faul, & Buchner, 1996).

Interactions could also be difficult to find in this particular study due to the low quality of the environmental measure and the restricted range of the adoptive environment. First, the only environmental measure utilized in this study was a dichotomous code indicating whether or not an adverse environment was present. This variable was based upon parent report of the presence of certain familial characteristics (e.g., mental illness, divorce). No opportunity was available in the creation of this variable to consider whether or not the presence of a particular characteristic was actually stressful or deleterious to the development of an adoptee. Surely, considerable variation exists in the extent that the presence of a characteristic like mental illness adversely affects a family. The dichotomous coding does not allow for this variation to be expressed (Nunnally & Bernstein, 1994). Additionally, the difficulties of restricted environmental range hamper efforts to find interactions. Adoption environments are generally screened in order to avoid particularly dysfunctional environments (Cadoret, 1985; Rowe, 1994). The effective elimination of particularly harmful environments,
while potentially beneficial for the adoptee, further limits the power of the interaction analyses.

Implications

Increasingly, criminologists and adolescent researchers are considering developmental theories of antisocial behavior (Piquero & Brezina, 2001). The present study represents an important contribution to the study of antisocial behavior by being among the first to utilize a behavioral genetic design to test Moffitt's (1993) developmental taxonomy of antisocial behavior. Indeed, with the notable exception of a paper by Taylor, Iacono, and McGue (2000), the potential to use genetically informed designs as a means to test the existence of such a developmentally based taxonomy has been ignored. The present study tested the popular theory that two distinct groups of antisocial adolescents exist, those with traits that predispose them to a lifelong pattern of antisocial behavior and those that limit their antisocial acts to adolescence. Considerable evidence has been found in this study to validate this theory. Adoptees in this study who limited their antisocial behaviors to adolescence were no more likely to have a biological parent with an antisocial disorder or other form of psychopathology than adoptees who did not engage in any antisocial behavior. On the other hand, adoptees who engaged in antisocial behavior both during adolescence and at other stages in their development were far more likely to have a biological parent with either an antisocial disorder or another form of psychopathology.

Therefore, the results of this study strongly suggest that adolescent antisocial behaviors are committed by two qualitatively different groups of offenders. Those
individuals who engage in antisocial behavior both during adolescence as well as at other stages of development, the Life-Course-Persistent group, appear to have a genetic influence on these behaviors. Those individuals who limit their antisocial behaviors to adolescence, the Adolescent Limited group, appear not to have a genetic influence on these behaviors. The presence of both AL and LCP adolescents and their accompanying different etiologies may explain why behavioral genetic research on adolescent antisocial behavior provides more discrepant findings than similar research on adult antisocial behaviors. Behavioral genetic analyses of adolescent antisocial behavior may well be overstating the heritability of these behaviors for the majority of adolescents but, perhaps more significantly, under representing the role that heritability plays for that minority group of adolescents who persist in antisocial behaviors throughout the lifespan.

Further, validation of the AL and LCP taxonomy could have both profound implications for research as well as programming. First, research studies seeking to explain adolescent antisocial behavior utilizing a general theory of crime, like that offered by Gottfredson and Hirschi (1990), may be oversimplifying the processes that are actually occurring. The behaviors of the majority may successfully occlude the motivations and etiology of antisocial behaviors committed by the LCP subgroup. Further, given that these two groups may be operating under distinctly different motivations for their behavior, research efforts that fail to treat them separately may well be weakening their ability to find significant results. Additionally, this research suggests that subsequent antisocial research acknowledging developmental heterogeneity may be fruitful.
The present study has programmatic implications as well. The majority of adolescents may be exhibiting antisocial behavior not as a pathological or criminal precursor but as a social expression (Jessor, 1992; Maggs, 1997; Silbereisen & Noack, 1988). However, antisocial behavior for a smaller minority of adolescents may be driven by characteristics that are inherited. Far from arguing genetic determinism and giving up on this group of adolescents, the present study suggests that inherited characteristics may predispose these individuals to antisocial behaviors and that intervention efforts should recognize the presence of differences among them and apply different programs. For example, for those who are engaging in AL antisocial behavior, increasing opportunities that provide healthy and constructive opportunities to express autonomy may be most helpful in reducing antisocial behaviors (e.g., Calhoun, Glaser, & Bartolomucci, 2001; Dembo, et al. 2000). This tactic would not, however, be successful for those adolescents that are engaging in LCP antisocial behaviors due to individual characteristics (e.g., Moffitt, Caspi, Harrington, & Milne, 2002). Here, the biggest programmatic benefit may come from such efforts as anger management, emotional regulation, and impulse control. While LCP individuals may be predisposed to engage in antisocial behaviors due to inherited traits, environmental interventions addressing any deficits or deficiencies in these traits may prove effective.

Intervention efforts that fail to acknowledge that their participants may contain a subgroup of LCP adolescents that are qualitatively different may be in danger of not only being ineffective but of providing AL with the kind of deviance training that has been mentioned in previous research (e.g., Dishion, 2000).
Limitations

A number of limitations should be considered when interpreting the findings in this study. These limitations primarily concern issues surrounding the sample, the design and the measures.

First, the limitations of the adoption design must be realized. The full adoption design utilized in this study allows for a direct test of genetic and environmental effects but does not contain sufficient information to partition variation into separate estimates of heritability, shared environment and nonshared environment. It should be noted that the emphasis in this study was to examine whether or not genetic effects were present for each of the AL and LCP groups rather than to provide an estimate of the extent to which genetics and environmental sources contribute to variation in antisocial behavior.

Another limitation of the adoption design is the restricted range found in adoption environments (Cadoret, 1986). Unlike other behavioral genetic designs (e.g., family study, twin design), the adoption design is less likely to feature a full range of environmental conditions. Families seeking to adopt a child are regularly screened beforehand so that families with potentially adverse characteristics may be precluded from adoption. It should also be noted that families that adopt are different from other family forms in that they actively choose to become parents. This is not a prerequisite for other family forms. By actively selecting to become parents, adoptive parents may, on average, exhibit greater parental investment and thus effectively restrict the range of the adoptive home environment.
The measures used in this study rely primarily upon retrospective accounts of behaviors. While it may be argued that the antisocial behaviors studied were salient or had consequences that accompany them that made their recollection reliable (Cadoret & Cain, 1981), other research has found retrospective reports of antisocial behavior are only modestly reliable (e.g., Jacobson, Prescott, & Kendler, 2000). Some reassurance can be taken from the fact that this study utilizes both parental reports and self report to obtain similar results. However, both of these are prone to errors of recall and retrospective accounts of early behavior may well be colored by more recent events (Jacobson, Prescott, & Kendler, 2000).

The measures used in the present study were largely dichotomous. The reports provided by both self-report and parents consisted of whether or not a particular behavior or characteristic exists. These measures failed to acknowledge that considerable variation may exist within positive reports. An adoptee who is suspended from school on one occasion would be treated the same way as someone who has been suspended many times. The net effect of these dichotomous codes is loss of information and potential variability (Nunnally & Bernstein, 1994).

One dichotomous measure worthy of additional discussion is this study’s measure of adoptive home environment. This variable suffers from a number of limitations that make drawing strong conclusions about the importance of environmental influences on antisocial behavior untenable. First, as mentioned above, adoption designs suffer from a problem of restricted range; in general, children are not adopted into particularly aversive family environments. Second, given the broad definitions of aversive environments,
considerable variation could exist within those environments classified as aversive and those classified as non-aversive rendering this variable a poor proxy for environmental influences. While I believe this point has been made on a number of occasions, it nevertheless bears repeating that any conclusions drawn from this study that the influences of environment on antisocial behaviors is negligible misrepresents what can be said from the assessment of environment available for these data. If this variable is so poor, why include it? First, there is the practical matter that this is the only variable that was available from the data set. More importantly, however, it is important to remember that the emphasis in this study was not to identify environmental influences but to determine whether genetic influences were differentially present. In order to provide a more conservative test of genetic influences, environmental influences were included in the regression equations. In retrospect, the fact that environment and the biological history variables were uncorrelated means that this environmental variable could have been omitted and the estimates of genetic influences would not have been significantly affected.

It should be noted that the data used in this study were somewhat older, the adolescent behaviors described occurred between 20 and 40 years ago. Given this fact, some of the antisocial items used (e.g., “Was he ever insolent at home?”) may not be as relevant for describing more contemporary notions of adolescent antisocial behavior. While this means that the measures of antisocial behavior used may need to be changed for present studies this does not mean that the results of this study are hampered by the measures. Indeed, the measures used in this study are far more relevant examples of
antisocial behaviors for the sample studied than would be more contemporary examples of antisocial behaviors (e.g., "Has he participated in a drive by shooting?"). Further, while the specific manifestations of antisocial behaviors may have changed in the past generation or two, the underlying genetic processes could hardly have changed in such an evolutionarily short time span.

Some difficulties inherent in the classification scheme utilized must be acknowledged. First, definitions of adolescence were set by the data as encompassing the ages of 13 to 18. This, however, may not be the best definition of adolescence (e.g., Petersen & Leffert, 1995). Consider, for example, those adoptees who have already entered puberty prior to the age of 13. Antisocial behaviors committed by these 12-year-olds may indeed be adolescent limited but would not be identifiable as such with this data. A definition of adolescence that incorporates such information as pubertal development may provide a better classification of behavior. Second, adult behavior was indicated as behaviors that persisted past the age of 18. This definition of adulthood is similarly unsatisfying (see e.g., Arnett, 2001). Antisocial behaviors committed by a 19-year-old may well fit with what is intended by adolescent limited whereas identical antisocial behaviors committed by a 40-year-old may fit with what is intended by life-course-persistent behaviors yet both are treated the same in these data. Further data that details more precisely when behaviors are committed would aid in the development of more precise classification.

Last, it should also be recognized that some of the adoptees in this study were young enough (i.e., 18) that they could have engaged in AL antisocial behavior but have
not yet matured out of their behavior. It is possible in these instances that AL individuals were misclassified as LCP. This could have decreased the differences between the two groups.

Directions for Future Research

The present study tested Moffitt's (1993) theory of a dual taxonomy by utilizing biological history of antisocial behaviors and psychopathology as a predictor of antisocial behavior in an adoptee sample. This study employed the rarely used full adoption design. In this design, adoptees were separated from their parents at or shortly after birth. Any resemblance between adoptees and their biological parents, then, was the result of genetics. The study found consistent support for differences in the etiology of antisocial behavior, however, what this study has not done is directly test Moffitt’s proposed mechanisms for AL and LCP antisocial behavior. Future research that focuses upon the motivation and context of antisocial behaviors would be of interest in further validating the theory.

The adoption design utilized in this study permits for an examination of direct effects of genetic history and environment. A stronger design for measuring the extent of these influences would be either a twin or a family study. These designs would permit variation in antisocial behaviors to be partitioned to allow for estimates of heritability as well as shared and non-shared environmental influences. Either of these designs would also provide for a fairer test of environmental influences as a less restricted range of family environment would be present. Ideally, future research would also utilize a
prospective longitudinal design to eliminate the potential for and reduce the causal ambiguity of any environmental influences that were found.
### Table 1

**Frequency Distribution of Parent Reports of Antisocial Behaviors During Childhood**

<table>
<thead>
<tr>
<th>Number of Antisocial Behaviors</th>
<th>Total (N = 378)</th>
<th>LCP (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>0</td>
<td>225</td>
<td>59.5</td>
</tr>
<tr>
<td>1</td>
<td>81</td>
<td>21.4</td>
</tr>
<tr>
<td>2</td>
<td>36</td>
<td>9.5</td>
</tr>
<tr>
<td>3</td>
<td>21</td>
<td>5.6</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>2.4</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>0.8</td>
</tr>
<tr>
<td>7+</td>
<td>2</td>
<td>0.6</td>
</tr>
</tbody>
</table>

*Note.* Parents could endorse up to 11 childhood antisocial behaviors.
Table 2

Types of Antisocial Behaviors Reported by Parents Prior to Adolescence

<table>
<thead>
<tr>
<th></th>
<th>Total (N = 378)</th>
<th>LCP (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>Difficult/Violent Temperament (0-2)</td>
<td>26</td>
<td>7.1</td>
</tr>
<tr>
<td>Violent Reactions (0-2)</td>
<td>8</td>
<td>2.2</td>
</tr>
<tr>
<td>Discontented (0-2)</td>
<td>17</td>
<td>4.7</td>
</tr>
<tr>
<td>Difficult/Violent Temperament (2-6)</td>
<td>14</td>
<td>3.8</td>
</tr>
<tr>
<td>Frequent Temper Tantrums (2-6)</td>
<td>68</td>
<td>18.1</td>
</tr>
<tr>
<td>Discontented (2-6)</td>
<td>10</td>
<td>2.7</td>
</tr>
<tr>
<td>Violent Reactions (2-6)</td>
<td>7</td>
<td>1.9</td>
</tr>
<tr>
<td>Rebellious (6-12)</td>
<td>49</td>
<td>13.0</td>
</tr>
<tr>
<td>Doesn’t Get Along (6-12)</td>
<td>37</td>
<td>9.8</td>
</tr>
<tr>
<td>Recklessness (6-12)</td>
<td>51</td>
<td>13.6</td>
</tr>
<tr>
<td>Destructive (6-12)</td>
<td>15</td>
<td>4.0</td>
</tr>
</tbody>
</table>

Note. The age group a specific item referred to follows each item label in parentheses. Due to missing data, the number of valid responses ranged from 361 to 378 for the total sample and from 58 to 62 for the LCP group. AL group not reported as, by definition, no childhood reports were permitted.
<table>
<thead>
<tr>
<th>Number of Antisocial Behaviors</th>
<th>Total (N = 378)</th>
<th>AL (N = 115)</th>
<th>LCP (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>0</td>
<td>137</td>
<td>36.4%</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>83</td>
<td>22.1%</td>
<td>57</td>
</tr>
<tr>
<td>2</td>
<td>44</td>
<td>11.7%</td>
<td>18</td>
</tr>
<tr>
<td>3</td>
<td>32</td>
<td>8.5%</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>29</td>
<td>7.7%</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>13</td>
<td>3.5%</td>
<td>7</td>
</tr>
<tr>
<td>6</td>
<td>11</td>
<td>2.9%</td>
<td>7</td>
</tr>
<tr>
<td>7+</td>
<td>27</td>
<td>7.2%</td>
<td>4</td>
</tr>
</tbody>
</table>

Note. Parents could endorse up to 12 adolescent antisocial behaviors.
Table 4

Types of Adolescent Antisocial Behaviors Reported by Parents

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Total (N = 378)</th>
<th>AL (N = 115)</th>
<th>LCP (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>Truant</td>
<td>59</td>
<td>15.8%</td>
<td>21</td>
</tr>
<tr>
<td>Trouble w/ Teachers</td>
<td>52</td>
<td>13.9%</td>
<td>12</td>
</tr>
<tr>
<td>Suspended</td>
<td>28</td>
<td>7.4%</td>
<td>11</td>
</tr>
<tr>
<td>Disobedient</td>
<td>73</td>
<td>19.5%</td>
<td>26</td>
</tr>
<tr>
<td>Run Away</td>
<td>39</td>
<td>10.4%</td>
<td>14</td>
</tr>
<tr>
<td>Drugs</td>
<td>77</td>
<td>20.5%</td>
<td>30</td>
</tr>
<tr>
<td>Juvenile Authorities</td>
<td>37</td>
<td>9.9%</td>
<td>14</td>
</tr>
<tr>
<td>Gangs</td>
<td>19</td>
<td>5.1%</td>
<td>8</td>
</tr>
<tr>
<td>Fights</td>
<td>76</td>
<td>20.4%</td>
<td>34</td>
</tr>
<tr>
<td>Defiant</td>
<td>92</td>
<td>24.5%</td>
<td>38</td>
</tr>
<tr>
<td>Insolent</td>
<td>127</td>
<td>34.0%</td>
<td>52</td>
</tr>
<tr>
<td>Bully</td>
<td>61</td>
<td>16.3%</td>
<td>25</td>
</tr>
</tbody>
</table>

Note. Due to missing data, the number of valid responses ranged from 372 to 378 for the total sample, 114 to 115 for the AL group, and 59 to 62 for the LCP group.
Table 5

Frequency Distribution of Parent Reports of Antisocial Behaviors After Age 18

<table>
<thead>
<tr>
<th>Number of Antisocial Behaviors</th>
<th>Total (N = 240)</th>
<th>AL (N = 61)</th>
<th>LCP (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>0</td>
<td>121</td>
<td>50.4%</td>
<td>31</td>
</tr>
<tr>
<td>1</td>
<td>56</td>
<td>23.3%</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>34</td>
<td>14.2%</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>13</td>
<td>5.4%</td>
<td>0</td>
</tr>
<tr>
<td>4+</td>
<td>16</td>
<td>6.7%</td>
<td>5</td>
</tr>
</tbody>
</table>

Note. Parents could endorse up to 7 antisocial behaviors after the age of 18.
Table 6

Types of Antisocial Behaviors Reported by Parents After Age 18

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Total (N = 240)</th>
<th>AL (N = 56)</th>
<th>LCP (N = 62)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
</tr>
<tr>
<td>Can't Get Along</td>
<td>50</td>
<td>21.1%</td>
<td>5</td>
</tr>
<tr>
<td>Violent Reactions</td>
<td>6</td>
<td>2.7%</td>
<td>0</td>
</tr>
<tr>
<td>No Friends</td>
<td>39</td>
<td>17.6%</td>
<td>9</td>
</tr>
<tr>
<td>Frequent Lies</td>
<td>38</td>
<td>16.0%</td>
<td>10</td>
</tr>
<tr>
<td>Steals</td>
<td>31</td>
<td>13.1%</td>
<td>4</td>
</tr>
<tr>
<td>Police Trouble</td>
<td>37</td>
<td>15.5%</td>
<td>4</td>
</tr>
<tr>
<td>Fired</td>
<td>35</td>
<td>15.6%</td>
<td>5</td>
</tr>
</tbody>
</table>

Note. Due to missing data, the number of valid responses ranged from 221 to 240 for the total sample, 52 to 56 for the AL group, and 57 to 62 for the LCP group.
Table 7

Point-Biserial Correlations between Adoptee Clinical Interviews and Number of Antisocial Behaviors Reported by Parents

<table>
<thead>
<tr>
<th>Clinical Interview</th>
<th>Any Antisocial Behavior</th>
<th>Clinical Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$p_{pb}$</td>
<td>$N$</td>
</tr>
<tr>
<td>Parent Report of</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antisocial Behaviors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 to 2 years of age</td>
<td>.10</td>
<td>(306)</td>
</tr>
<tr>
<td>2 to 6</td>
<td>.16**</td>
<td>(313)</td>
</tr>
<tr>
<td>6 to 12</td>
<td>.19***</td>
<td>(313)</td>
</tr>
<tr>
<td>13 to 18</td>
<td>.35***</td>
<td>(313)</td>
</tr>
<tr>
<td>18+</td>
<td>.33***</td>
<td>(178)</td>
</tr>
</tbody>
</table>

* $p < .05$. ** $p < .01$. *** $p < .001$. 
Table 8

**Joint Frequency Distribution of AL and LCP Adoptees With Biological History of Psychopathology**

<table>
<thead>
<tr>
<th>Biological History of Psychopathology</th>
<th>No</th>
<th>Yes</th>
<th>Σ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obs. (Exp.)</td>
<td>Obs (Exp.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antisocial Classification</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adolescent Limited</td>
<td>61 (52.2)</td>
<td>34 (42.8)</td>
<td>95</td>
</tr>
<tr>
<td>Life-Course-Persistent</td>
<td>22 (30.8)</td>
<td>34 (25.2)</td>
<td>56</td>
</tr>
<tr>
<td>Σ</td>
<td>83</td>
<td>68</td>
<td>151</td>
</tr>
</tbody>
</table>

*Note. Obs. = observed count. Exp. = expected count. $\chi^2 (1, N = 151) = 8.84, p < .01.*
Table 9

Joint Frequency Distribution of AL and LCP Adoptees With Biological History of Antisocial Behaviors

<table>
<thead>
<tr>
<th>Biological History of Antisocial Behaviors</th>
<th>No</th>
<th>Yes</th>
<th>( \Sigma )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obs</td>
<td>Exp</td>
<td></td>
</tr>
<tr>
<td>Obs (Exp.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antisocial Classification</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adolescent Limited</td>
<td>95</td>
<td>(85.1)</td>
<td>110</td>
</tr>
<tr>
<td>Life-Course-Persistent</td>
<td>38</td>
<td>(47.9)</td>
<td>62</td>
</tr>
<tr>
<td>( \Sigma )</td>
<td>133</td>
<td>39</td>
<td>172</td>
</tr>
</tbody>
</table>

*Note.* Obs. = observed count. Exp. = expected count. \( \chi^2 (1, N = 172) = 14.22, p < .001. \)
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1. 2. 3. 4. 5.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>,67***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.09</td>
<td>24**</td>
<td>.02</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.24**</td>
<td>.29***</td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>.18**</td>
<td>.16**</td>
<td>.06</td>
<td>.40***</td>
</tr>
<tr>
<td></td>
<td></td>
<td>.30**</td>
<td></td>
<td>.30**</td>
<td>.42***</td>
</tr>
</tbody>
</table>

Note.  

n = 122 to 382.  

**p < .01.  *** p < .001.
Table 11

Hierarchical Multiple Regression Models Utilizing Biological History of Psychopathology as a Predictor of Parental Reports of Antisocial Behavior

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.24</td>
<td>0.11</td>
<td>0.17*</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.33</td>
<td>0.16</td>
<td>0.17*</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.13</td>
<td>0.11</td>
<td>0.09</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.26</td>
<td>0.15</td>
<td>0.13</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.48</td>
<td>0.11</td>
<td>0.34***</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.12</td>
<td>0.11</td>
<td>0.09</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.25</td>
<td>0.15</td>
<td>0.13</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.45</td>
<td>0.11</td>
<td>0.32***</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>0.24</td>
<td>0.22</td>
<td>0.09</td>
</tr>
</tbody>
</table>

Note. N = 151. $R^2 = .06$ for Step 1 ($p < .01$); $\Delta R^2 = .11$ for Step 2 ($p < .001$); $\Delta R^2 = .01$ for Step 3 ($p = .27$).

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


Table 12

*Hierarchical Multiple Regression Models Utilizing Biological History of Antisocial Personality as a Predictor of Parental Reports of Antisocial Behavior*

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td>0.37</td>
<td>0.12</td>
<td>0.23**</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.17</td>
<td>0.14</td>
<td>0.09</td>
</tr>
<tr>
<td><strong>Step 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td>0.20</td>
<td>0.12</td>
<td>0.12</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.14</td>
<td>0.13</td>
<td>0.07</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.51</td>
<td>0.10</td>
<td>0.36***</td>
</tr>
<tr>
<td><strong>Step 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td>0.24</td>
<td>0.13</td>
<td>0.15</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.14</td>
<td>0.13</td>
<td>0.07</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.53</td>
<td>0.10</td>
<td>0.38***</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>-0.21</td>
<td>0.24</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

**Note.** N = 172. R² = .06 for Step 1 (p < .01); ΔR² = .12 for Step 2 (p < .001); ΔR² = .01 for Step 3 (p = .37).

* p < .05. ** p < .01. *** p < .001.
Table 13

Logistic Regression Utilizing Biological History of Psychopathology as a Predictor of Adoptee Antisocial Diagnosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>3.20</td>
<td>1.84</td>
<td>(0.94 – 3.57)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.42</td>
<td>1.63</td>
<td>(0.73 – 3.66)</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.34</td>
<td>1.30</td>
<td>(0.54 – 3.15)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.40</td>
<td>1.46</td>
<td>(0.45 – 4.80)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>7.53**</td>
<td>3.58</td>
<td>(1.44 – 8.91)</td>
</tr>
<tr>
<td>Model 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.00</td>
<td>0.99</td>
<td>(0.31 – 3.25)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.39</td>
<td>1.46</td>
<td>(0.44 – 4.79)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>5.44*</td>
<td>5.09</td>
<td>(1.30 – 20.00)</td>
</tr>
<tr>
<td>Bio. Hx. x AL/LCP</td>
<td>0.48</td>
<td>1.91</td>
<td>(0.31 – 11.85)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio. * $p < .05$. ** $p < .01$. 

Table 14

Logistic Regression Utilizing Biological History of Antisocial Personality as a Predictor of Adoptee Antisocial Diagnosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td>8.24*</td>
<td>2.96</td>
<td>(1.35 – 6.49)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.57</td>
<td>1.68</td>
<td>(0.75 – 3.76)</td>
</tr>
<tr>
<td>Model 2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td>3.53</td>
<td>2.74</td>
<td>(0.96 – 7.84)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.64</td>
<td>1.64</td>
<td>(0.49 – 5.49)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>6.22*</td>
<td>3.20</td>
<td>(1.28 – 7.96)</td>
</tr>
<tr>
<td>Model 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td>3.95*</td>
<td>4.25</td>
<td>(1.02 – 17.73)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.67</td>
<td>1.66</td>
<td>(0.49 – 5.57)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>6.74**</td>
<td>4.00</td>
<td>(1.41 – 11.41)</td>
</tr>
<tr>
<td>Bio. Hx. x AL/LCP</td>
<td>0.74</td>
<td>0.41</td>
<td>(0.05 – 3.12)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.

* $p < .05$. ** $p < .01$. 
Table 15

Logistic Regression Utilizing Biological History of Psychopathology as a Predictor of Group Classification

<table>
<thead>
<tr>
<th>Variable</th>
<th>( \chi^2 )</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: LCP vs. Others</td>
<td>15.59***</td>
<td>3.14</td>
<td>(1.73 – 5.70)</td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>14.21***</td>
<td>3.14</td>
<td>(1.73 – 5.70)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.42</td>
<td>1.29</td>
<td>(0.60 – 2.76)</td>
</tr>
<tr>
<td>Model 2: LCP vs. AL</td>
<td>10.85**</td>
<td>2.73</td>
<td>(1.38 – 5.42)</td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>8.25**</td>
<td>2.73</td>
<td>(1.38 – 5.42)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.98</td>
<td>1.99</td>
<td>(0.76 – 5.19)</td>
</tr>
<tr>
<td>Model 3: AL vs. None</td>
<td>2.58</td>
<td>0.85</td>
<td>(0.43 – 1.69)</td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.22</td>
<td>0.85</td>
<td>(0.43 – 1.69)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>2.18</td>
<td>0.49</td>
<td>(0.19 – 1.26)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.

** p < .01. *** p < .001.
Table 16

Logistic Regression Utilizing Biological History of Antisocial Personality as a Predictor of Group Classification

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1: LCP vs. Others</td>
<td>17.63***</td>
<td>18.78***</td>
<td>3.76 (2.07 - 6.85)</td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.01</td>
<td>1.03</td>
<td>(0.49 - 2.15)</td>
</tr>
<tr>
<td>Model 2: LCP vs. AL</td>
<td>14.11**</td>
<td>13.28***</td>
<td>4.02 (1.90 - 8.49)</td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.35</td>
<td>1.31</td>
<td>(0.54 - 3.14)</td>
</tr>
<tr>
<td>Model 3: AL vs. None</td>
<td>5.25</td>
<td>3.81</td>
<td>0.47 (0.22 - 1.00)</td>
</tr>
<tr>
<td>Bio. Hx. Antisocial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.26</td>
<td>0.64</td>
<td>(0.30 - 1.39)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio. ** $p < .01$. *** $p < .001$. 
Table 17

**Multiple Regression Testing Biological History of Psychopathology × Adverse Home Environment Interaction as a Predictor of Parental Reports of Antisocial Behavior**

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.11</td>
<td>0.11</td>
<td>0.08</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.27</td>
<td>0.16</td>
<td>0.14</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.45</td>
<td>0.11</td>
<td>0.32***</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>0.25</td>
<td>0.22</td>
<td>0.09</td>
</tr>
<tr>
<td>Bio. Hx × Home Environment</td>
<td>-0.15</td>
<td>0.30</td>
<td>-0.39</td>
</tr>
</tbody>
</table>

Note. N = 151. $R^2 = .18$.

*** p < .001.
Table 18

**Multiple Regression Testing Biological History of Antisocial Personality \times Adverse Home Environment Interaction as a Predictor of Parental Reports of Antisocial Behavior**

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE B</th>
<th>β</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>0.23</td>
<td>0.13</td>
<td>0.14</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.14</td>
<td>0.13</td>
<td>0.08</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.53</td>
<td>0.10</td>
<td>0.37***</td>
</tr>
<tr>
<td>Bio. Hx. \times AL/LCP</td>
<td>-0.21</td>
<td>0.24</td>
<td>-0.07</td>
</tr>
<tr>
<td>Bio. Hx \times Home Environment</td>
<td>-0.22</td>
<td>0.31</td>
<td>-0.05</td>
</tr>
</tbody>
</table>

Note. \( N = 172. \quad R^2 = .19. \)

*** \( p < .001. \)
Table 19

Logistic Regression Utilizing Biological History of Psychopathology × Adverse Home Environment as a Predictor of Adoptee Antisocial Diagnosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>( \chi^2 )</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>11.62* (df = 5, N = 119)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.04</td>
<td>1.13</td>
<td>(0.34 – 3.79)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.37</td>
<td>2.39</td>
<td>(0.56 – 10.28)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>2.08</td>
<td>2.51</td>
<td>(0.72 – 8.78)</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>0.78</td>
<td>2.34</td>
<td>(0.36 – 15.35)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>1.12</td>
<td>0.26</td>
<td>(0.02 – 3.14)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio. *p < .05.
Table 20

Logistic Regression Utilizing Biological History of Psychopathology × Adverse Home Environment as a Predictor of Life Course Persistent Antisocial Behavior

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>15.85** (df = 3, N = 329)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>13.13***</td>
<td>3.38</td>
<td>(1.75 - 6.52)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.68</td>
<td>1.64</td>
<td>(0.51 - 5.28)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>0.26</td>
<td>0.67</td>
<td>(0.15 - 3.10)</td>
</tr>
</tbody>
</table>

Note. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio. ** p < .01. *** p < .001.
Table 21

Logistic Regression Utilizing Biological History of Psychopathology × Adverse Home Environment as a Predictor of Life-Course-Persistent vs. Adolescence Limited Antisocial Behavior

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>10.85*</td>
<td>(df = 3, N = 151)</td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>7.10**</td>
<td>2.75</td>
<td>(1.31 – 5.79)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.03</td>
<td>2.04</td>
<td>(0.52 – 8.04)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>0.00</td>
<td>0.96</td>
<td>(0.14 – 6.47)</td>
</tr>
</tbody>
</table>

Note. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.
* $p < .05$. ** $p < .01$. 
Table 22

**Logistic Regression Utilizing Biological History of Psychopathology × Adverse Home Environment as a Predictor of Adolescence Limited Antisocial Behavior vs No Antisocial Behavior**

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>3.55 (df = 3, N = 151)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.00</td>
<td>0.99</td>
<td>(0.47 – 2.10)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.12</td>
<td>0.79</td>
<td>(0.21 – 3.03)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>0.95</td>
<td>0.39</td>
<td>(0.06 – 2.62)</td>
</tr>
</tbody>
</table>

*Note.* Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.
Table 23

Logistic Regression Utilizing Biological History of Antisocial Personality × Adverse Home Environment as a Predictor of Life-Course-Persistent Behavior

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>17.80***</td>
<td>(df = 3, N = 382)</td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>16.87***</td>
<td>3.99</td>
<td>(2.06 – 7.73)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.10</td>
<td>1.15</td>
<td>(0.48 – 2.77)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>0.18</td>
<td>0.72</td>
<td>(0.15 – 3.43)</td>
</tr>
</tbody>
</table>

Note. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio. *** p < .001.
Table 24

Logistic Regression Utilizing Biological History of Antisocial Personality × Adverse Home Environment as a Predictor of Life-Course-Persistent vs. Adolescence Limited Antisocial Behavior

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>14.11**</td>
<td></td>
<td>(df = 3, N = 172)</td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>11.28**</td>
<td>4.02</td>
<td>(1.79 – 9.05)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.28</td>
<td>1.31</td>
<td>(0.48 – 3.54)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>0.00</td>
<td>1.00</td>
<td>(0.12 – 8.04)</td>
</tr>
</tbody>
</table>

Note. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio. ** p < .01.
Table 25

Logistic Regression Utilizing Biological History of Antisocial Personality × Adverse Home Environment as a Predictor of Adolescence Limited Antisocial Behavior vs No Antisocial Behavior

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>5.50 (df = 3, N = 185)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>2.40</td>
<td>0.52</td>
<td>(0.22 - 1.19)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.61</td>
<td>0.71</td>
<td>(0.30 - 1.69)</td>
</tr>
<tr>
<td>Bio Hx. × Home Environment</td>
<td>0.24</td>
<td>0.61</td>
<td>(0.08 - 4.52)</td>
</tr>
</tbody>
</table>

Note. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.
Table 26

**Means and Standard Deviations by Adolescent Limited and Life-Course-Persistent Classification for Total Antisocial Behaviors, Aggressive Behaviors, and Substance Use**

<table>
<thead>
<tr>
<th></th>
<th>AL</th>
<th>LCP</th>
<th>F</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Total Antisocial Behaviors</strong></td>
<td>2.35</td>
<td>1.86</td>
<td>4.54</td>
<td>3.17</td>
</tr>
<tr>
<td></td>
<td>32.90***</td>
<td>1,170</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aggressive Behaviors</strong></td>
<td>0.47</td>
<td>0.50</td>
<td>0.53</td>
<td>0.50</td>
</tr>
<tr>
<td></td>
<td>0.57</td>
<td>1,168</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Substance Use</strong></td>
<td>0.25</td>
<td>0.44</td>
<td>0.49</td>
<td>0.50</td>
</tr>
<tr>
<td></td>
<td>10.34**</td>
<td>1,169</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note.** \( N = 170-172. \)

\*\* \( p < .01. \) \*\*\* \( p < .001. \)
Table 27

**Logistic Regression Utilizing Biological History of Psychopathology as a Predictor of Parental Reports of Aggressive Behaviors**

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td>1.42 (df = 2, N = 315)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.16</td>
<td>1.10</td>
<td>(0.68 - 1.78)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.21</td>
<td>1.43</td>
<td>(0.76 - 2.72)</td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td>1.30 (df = 3, N = 149)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.38</td>
<td>0.81</td>
<td>(0.42 - 1.58)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.88</td>
<td>1.58</td>
<td>(0.61 - 4.12)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.07</td>
<td>1.10</td>
<td>(0.55 - 2.20)</td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td>1.40 (df = 4, N = 149)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.46</td>
<td>0.75</td>
<td>(0.32 - 1.74)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.87</td>
<td>1.58</td>
<td>(0.61 - 4.11)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.00</td>
<td>0.98</td>
<td>(0.37 - 2.63)</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>0.10</td>
<td>1.24</td>
<td>(0.31 - 4.95)</td>
</tr>
</tbody>
</table>

**Note.** Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.
Table 28

Logistic Regression Utilizing Biological History of Antisocial Personality as a Predictor of Parental Reports of Aggressive Behaviors

<table>
<thead>
<tr>
<th>Variable</th>
<th>( \chi^2 )</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>0.34 (df = 2, ( N = 366 ))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>0.14</td>
<td>1.12</td>
<td>(0.63 - 1.97)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.20</td>
<td>1.15</td>
<td>(0.64 - 2.07)</td>
</tr>
<tr>
<td>Model 2</td>
<td>0.62 (df = 3, ( N = 170 ))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>0.02</td>
<td>0.95</td>
<td>(0.45 - 2.01)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.03</td>
<td>0.93</td>
<td>(0.41 - 2.11)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>0.59</td>
<td>1.30</td>
<td>(0.67 - 2.50)</td>
</tr>
<tr>
<td>Model 3</td>
<td>1.32 (df = 4, ( N = 170 ))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>0.25</td>
<td>1.32</td>
<td>(1.02 - 17.73)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.03</td>
<td>0.93</td>
<td>(0.40 - 2.12)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>1.17</td>
<td>1.53</td>
<td>(0.71 - 3.28)</td>
</tr>
<tr>
<td>Bio. Hx. x AL/LCP</td>
<td>0.69</td>
<td>0.53</td>
<td>(0.12 - 2.39)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.
Table 29

**Logistic Regression Utilizing Biological History of Psychopathology as a Predictor of Parental Reports of Substance Use**

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Model 1</strong></td>
<td>3.44 (df = 2, N = 319)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>2.70</td>
<td>1.61</td>
<td>(0.91 – 2.83)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.55</td>
<td>1.33</td>
<td>(0.63 – 2.81)</td>
</tr>
<tr>
<td><strong>Model 2</strong></td>
<td>7.40 (df = 3, N = 150)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.01</td>
<td>1.03</td>
<td>(0.50 – 2.14)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.28</td>
<td>1.31</td>
<td>(0.48 – 3.57)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>6.15*</td>
<td>2.54</td>
<td>(1.22 – 5.30)</td>
</tr>
<tr>
<td><strong>Model 3</strong></td>
<td>7.48 (df = 4, N = 150)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Psychopathology</td>
<td>0.02</td>
<td>0.94</td>
<td>(0.35 – 2.51)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>0.28</td>
<td>1.31</td>
<td>(0.48 – 3.56)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>2.38</td>
<td>2.28</td>
<td>(0.80 – 6.46)</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>0.83</td>
<td>1.24</td>
<td>(0.28 – 5.43)</td>
</tr>
</tbody>
</table>

*Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.

* p < .05.
Table 30

Logistic Regression Utilizing Biological History of Antisocial Personality as a Predictor of Parental Reports of Substance Use

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\chi^2$</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>5.11 (df = 2, $N = 370$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>4.22*</td>
<td>1.87</td>
<td>(1.03 - 3.41)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.14</td>
<td>1.43</td>
<td>(0.74 - 2.75)</td>
</tr>
<tr>
<td>Model 2</td>
<td>11.89**  (df = 3, $N = 171$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>0.32</td>
<td>1.26</td>
<td>(0.57 - 2.76)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.91</td>
<td>1.85</td>
<td>(0.77 - 4.45)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>7.77**</td>
<td>2.70</td>
<td>(1.34 - 5.36)</td>
</tr>
<tr>
<td>Model 3</td>
<td>13.60**  (df = 4, $N = 171$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bio. Hx. Antisocial Personality</td>
<td>1.94</td>
<td>2.25</td>
<td>(0.72 - 7.08)</td>
</tr>
<tr>
<td>Adverse Home Environment</td>
<td>1.97</td>
<td>1.88</td>
<td>(0.78 - 4.53)</td>
</tr>
<tr>
<td>AL/LCP Classification</td>
<td>9.35**</td>
<td>3.52</td>
<td>(1.57 - 7.90)</td>
</tr>
<tr>
<td>Bio. Hx. × AL/LCP</td>
<td>1.74</td>
<td>0.35</td>
<td>(0.08 - 1.66)</td>
</tr>
</tbody>
</table>

Note. Ns vary as a result of listwise deletion of missing data. Wald chi-squares were used to test significance of individual parameters. OR = Odds ratio. 95% CI = 95% confidence limits for the odds ratio.
* $p < .05$. ** $p < .01$. 
Figure 1. Age distribution of people arrested in the United States by age in 2000. Adapted from data available through the Uniform Crime Reporting (UCR) Program.
Appendix A
Iowa Children and Family Services Study Psychiatric Interview utilizing ICD-9 Criteria

ANTISOCIAL PERSONALITY SCREEN

Stop at the first 6 negative sections.

A. 1) Did you ever run away from home overnight? (score + only if this is from “parental” home or home of rearing) .................................................................

B. 2) Did you ever play hookey from school? (+ if > 1 time per year except last year of school).................................................................

3) Were you ever suspended or expelled from school? (if so, give reason) ...........

4) Did you ever get into fights at school? (+ if it led to disciplinary action by teachers or principal).................................................................

C. 5) Did you or have you had problems controlling your temper? ....................... 

If yes,

6) When angry, do you get into physical fights? (score + if this has occurred at least 2X or one time if a weapon was used) .................................................................

D. 7) Have you ever been arrested?

8) If yes, how many times? (do not include non-moving traffic violations) ...........

9) List reasons and if convicted (# of convictions 8 = 8 or more times)

................................................................................................................................................
................................................................................................................................................
................................................................................................................................................
................................................................................................................................................
................................................................................................................................................
E. 10) Have you ever quit a job without having another one to go to? ..............

11) Have you ever been fired? ........................................................................

12) Changes jobs frequently? (2X/yr. Or more, except where explained by seasonal, economic or nature of work) .................................................................

F. 13) Have you ever been divorced:
If yes,

14) How many times (if > 3X then +) ..............................................................

15) When married, did you ever separate temporarily because of arguments?... 
If yes,

16) How frequently (if > 3X then +) ..............................................................

17) Did you ever leave your family to financially fend for itself? ....................

18) Did you ever have an extramarital “affair”? .............................................

19) Did you ever strike your spouse in anger? (Score + if > 3X) .................

20) Have you ever struck your children in anger? (Score + if any physical harm was done) .................................................................

G. 21) Have you ever had the desire to “Keep on the move” (i.e., keep moving from place to place)?
If yes,

22) Have you ever done so for more than a couple of months? (Without arranging for work or financial support) .................................................................

H. 23) Have you ever told lies for no particular reason? .................................
24) Have you ever used a name not your own? ........................................... __

25) At any time in your life have you taken pay for sexual purposes? ............ __

26) Have you ever provided customers for one who did so? ....................... __

27) Have you ever had V.D.? (Score + if > 2X) ........................................ __

28) Was there ever a time in your life when you had many different sexual partners? .............................................................. __

AT LEAST ONE SYMPTOM PRESENT PRIOR TO AGE 15

Dx = DEFINITE ANTISOCIAL PERSONALITY DISORDER (5 out of 9 areas checked)

Dx = PROBABLE ANTISOCIAL PERSONALITY DISORDER (4 out of 9 areas checked)
Appendix B

Lutheran Social Services Antisocial Project SADS – L Antisocial Sub-Scale

ANTISOCIAL PERSONALITY

This category is for subjects with a chronic or recurrent disorder characterized by a failure to conform to social norms in many areas, always beginning before the age of 15 and persisting into adulthood, in the absence of severe mental retardation. The diagnosis should not be made in individuals below the age of 18.

If the subject alcohol or drug problem, score as present only those manifestations of Antisocial Personality which cannot be clearly attributed to the alcohol or drug problem. Do not count symptoms limited to a period of Manic Disorder, a depressive disorder, any other episodic disorder, or physical illness.

There are 4 criteria.

I. Since age 15, poor occupational performance over several years as shown by at least 1 of the following (most of this information should have already been obtained).

Note: Poor performance in school for the last few years of school may substitute for this criterion in individuals who, by virtue of their age or circumstances, have not had an opportunity to demonstrate their occupational adjustment.

<table>
<thead>
<tr>
<th>No</th>
<th>Info</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
</table>

*Since you started working have you changed jobs a lot (yes, if 3 or more jobs in 5 years, not accounted for by either the nature of the job, or economic or seasonal fluctuations)?*

X 1 2

*Have you had periods when you were not working (yes, if a total of 6 months or more during 10 years when expected to work and not due to physical illness)?*

X 1 2

*Did you miss a lot of time when you were working (yes, if absenteeism involved an average of 3 days or more per month when either late or absent)?*

X 1 2
Had at least 1 of the above items (or poor performance in school)
1 No (Skip to Personality Traits etc.)
2 Yes
8 Inapplicable

II. Onset in childhood as indicated by a history of 3 or more of the following (at least 1 beginning before age 15):

<table>
<thead>
<tr>
<th>When you were younger...</th>
</tr>
</thead>
<tbody>
<tr>
<td>... did you play hookey from school a lot (more than once per year for at least 2 years not including senior year of high school)?</td>
</tr>
<tr>
<td>X</td>
</tr>
<tr>
<td>... were you ever expelled from school?</td>
</tr>
<tr>
<td>X</td>
</tr>
<tr>
<td>... did people expect you to make better grades than you did (yes, if academic achievement below level expected on the basis of rater's judgement of likely IQ level)?</td>
</tr>
<tr>
<td>X</td>
</tr>
<tr>
<td>... were you always breaking the rules at school or home?</td>
</tr>
<tr>
<td>X</td>
</tr>
<tr>
<td>... were you arrested or sent to a juvenile court because of something you had done?</td>
</tr>
<tr>
<td>X</td>
</tr>
<tr>
<td>... did you run away from home overnight (at least twice while living in a parental or parental surrogate home)?</td>
</tr>
<tr>
<td>X</td>
</tr>
<tr>
<td>... did you lie a lot?</td>
</tr>
<tr>
<td>X</td>
</tr>
</tbody>
</table>
... did you drink a lot before most of the other (boys, girls) of your age?

X 1 2

... did you steal things?

X 1 2

... did you break windows, destroy property (vandalism)?

X 1 2

... did you start having sex long before most of the other (boys, girls) of your age (yes, if unusually early or aggressive sexual behavior)?

X 1 2

Had at least 3 items, at least 1 of which began before age 15
1 No (Skip to Personality Traits, etc.)
2 Yes
8 Inapplicable

III. At least 2 of the following since age 15:

No
Info No Yes

Since age 15 have you...

... been arrested (yes, if 3 or more serious arrests)?

X 1 2

... been divorced or separated (yes, if 2 or more divorces and/or separations whether legally married or not)?

X 1 2

... gotten into fights (physical)?

X 1 2

... often gotten drunk every week?

... often not paid debts or taken care of other expected financial responsibilities (e.g., child support)?

... ever had a period of time when you had no permanent residence or wandered from place to place with no pre-arranged plans (other than vacations)?

Had at least 2 items
1 No (Skip to Personality Traits, etc.)
2 Yes
8 Inapplicable

IV. There is some evidence of a markedly impaired capacity to sustain lasting, close, warm, and responsible relationships with family, friends, or sexual partners. (Thus, individuals who demonstrate the capacity for this kind of relationship are not given this diagnosis.)

0 No information or not sure
1 No
2 Yes
8 Inapplicable
(If 0 or 1 skip to Personality Traits etc.)

Is there anyone that you really feel very close to? Anyone else?

How long have you felt this way?

Do you help them out when they have problems?

Do you keep the same friends for a long time?

Has met the 4 criteria for Antisocial Personality

Yes

No
Appendix C

Antisocial Items asked of Adoptive Parents in Both The Iowa Children and Family Services and Lutheran Social Services Antisocial Project Parent Interview

Pre-adolescent Antisocial Behaviors (11 items, α = .65)

Age 0 to 2
1. When you first got our child, what was he like? In what ways?
2. How did he react to new things and situations such as strange adults, new places, other children or a new bed?
3. Was he more often contented or discontented as a baby?

Age 2 to 6
4. What was he like as a toddler?
5. Did he have frequent temper tantrums?
6. Was he contented or discontented as a toddler?
7. When there were changes in his immediate environment, how did he react?

Age 6 to 12
Have you noticed or has the teacher ever complained about any of the following traits:
8. rebellious or difficult to discipline
9. doesn’t get along with classmates
10. recklessness
11. destructive

Adolescent Antisocial Behaviors (12 items, α = .80)
1. Was he ever truant from school?
2. Did he ever have trouble with teachers or administrators?
3. Was he ever suspended or expelled?
4. Was he frequently disobedient or rebellious?
5. Did he ever run away from home?
6. Did he have any experience with drugs or alcohol?
7. Did he ever come to the attention of the juvenile authorities for any reason?
8. Did he belong to any groups or gangs? (Did they get in trouble?)
9. Was he ever involved in physical fights?
10. Was he defiant about obeying rules or carrying out chores?
11. Was he ever insolent at home?
12. Did he bully or dominate others, brothers, or sisters?

Late Adolescence and Adult Antisocial Behaviors (7 items, α = .61)
1. Is he generally easy to get along with, or is he easily upset?
2. How does he adjust to new situations and experiences?
3. Does he have any close friends?
4. Does he lie frequently?
5. Has he ever stolen anything?
6. Has he ever had trouble with the police?
7. Has he ever been fired from a job?
References


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