From the DEPARTMENT OF MEDICAL EPIDEMIOLOGY
AND BIOSTATISTICS
Karolinska Institutet, Stockholm, Sweden

Genetic and environmental influences on antisocial behavior from childhood to emerging adulthood

Catherine Tuvblad

Stockholm 2006

Published and printed by US-AB, Stockholm, Sweden

ISBN 91-7140-944-0

To Elizabeth and Alice

ABSTRACT

Antisocial behavior, in other words, normative and rule-breaking behavior, is a major problem in societies all over the world. Because many antisocial behavioral problems start in childhood or adolescence, the study of such behavior problems during this developmental period should contribute to an understanding of the etiology of adult psychopathology. Improved understanding of the etiology of antisocial behavior may contribute to better treatment and prevention.

The overall aim of this thesis was to investigate the influence of genetic and environmental factors in the development of antisocial behavior from childhood to emerging adulthood.

The data used in this thesis comes from the Twin study of CHild and Adolescent Development (TCHAD), a Swedish population-based study of 1,480 twin pairs born 1985-1986. The twins and their parents have been contacted on four different occasions (8-9 years, 13-14 years, 16-17 years, and 19-20 years) with good to excellent response rates. Multivariate twin methods were applied to investigate the influence of genetic and environmental effects on antisocial behavior from childhood to emerging adulthood.

The results suggest that the genetic and environmental etiology of antisocial behavior differs between boys and girls. Heritability was higher in girls, whereas the shared environment was more important in boys. These sex differences remained during the developmental period studied. Antisocial behavior that persists from early adolescence to emerging adulthood has strong familial effect in both boys and girls, with a limited influence of the unique environment.

Further, a substantial genetic overlap was found between psychopathic personality traits and antisocial behavior. This genetic overlap could reflect that psychopathic personality has an important role in mediating genetic effects on antisocial behavior. Alternatively, it may indicate a genetic vulnerability to externalizing psychopathology.

Finally, socioeconomic status moderated the influence of genetic and environmental factors on antisocial behavior. Genetic influences on antisocial behavior were more important in adolescents in socioeconomically more advantaged environments, whereas the shared environment was higher in adolescents in socioeconomically less advantaged environments.

Future research should address the causes of the sex differences in the genetic and environmental etiology of antisocial behavior. Another important question to answer is whether the genetic factor in persistent antisocial behavior is also associated with childhood and adulthood psychopathology. A further aspect to explore is if genetic influences associated with psychopathic personality traits are correlated with the emergence of later antisocial behavior. Knowledge from such studies would provide tools needed to identify effective intervention targets.

Key words: antisocial and aggressive behavior, psychopathic personality traits, socioeconomic status, childhood, adolescence, genes, environments, sex differences, development, covariation, interactions

LIST OF PUBLICATIONS

This thesis is based on the following papers, which will be referred to in the text by their roman numerals (I-IV).

I Tuvblad C., Eley T.E., & Lichtenstein P.
The development of antisocial behaviour from childhood to adolescence. A longitudinal twin study.
European Child & Adolescent Psychiatry (2005) 14, *4*, 216-225.

II Tuvblad C., Grann M., Sarnecki J., & Lichtenstein P.
The genetic and environmental etiology of antisocial behavior, from childhood to emerging adulthood.
Submitted for publication

III Larsson H., Tuvblad C., Rijsdijk F.V., Andershed H., Grann M., & Lichtenstein P.
A common genetic factor explains the association between psychopathic personality and antisocial behavior.
Psychological Medicine (2006) Epub ahead of print, 19*,* 1-12.

IV Tuvblad, C., Grann, M., & Lichtenstein, P.
Heritability for adolescent antisocial behavior differs with socioeconomic status: Gene-environment interaction.
Journal of Child Psychology and Psychiatry (2006) 47, *7*, 734-743.

CONTENTS

LIST OF ABBREVIATIONS

A	Additive genetic factor
ADHD	Attention-deficit hyperactivity disorder
AIC	Akaike's Information Criteria
ASB	Antisocial behavior
ASPD	Antisocial personality disorder
C	Shared environmental factor
CBCL	Child Behavior Checklist
CD	Conduct disorder
CI	Confidence interval
df	Degrees of freedom
DSM	Diagnostic and Statistical Manual of Mental Disorders
DZ	Dizygotic
E	Non-shared environmental factor
GxE	Gene-environment interaction
MZ	Monozygotic
OR	Odds Ratio
OS	Opposite-sexed
p	p-value
r_{GE}	Gene-environment correlation
SES	Socioeconomic status
TCHAD	The Twin study of CHild and Adolescent Development
χ^2	Chi-square
YPI	Youth Psychopathy trait Inventory
-2LL	-2(log-likelihood)

1 INTRODUCTION

Antisocial behavior is a major problem in societies all over the world. Violence (including homicide, child abuse, violence by intimate partners, abuse of elderly people, sexual violence, sexual trafficking, suicide, and collective violence) is among the leading causes of death worldwide for people age 15– 44 years (WHO, 2002).

Antisocial behavior not only causes physical and mental health problems for the victims, but also the antisocial individuals themselves are at higher risk for substance abuse and dependence (Miles et al., 2002, White et al., 2001), violence (Rutter et al., 1998), psychiatric disorders (Kim-Cohen et al., 2003), and to experience various types of psychosocial problems (Rutter et al., 2006).

Furthermore, antisocial behavior exacts heavy financial costs to society, including support for victims and costs of incarceration and treatment programs (Scott et al., 2001, Cohen, 1998).

Because many antisocial behavioral problems start in childhood or adolescence, the study of such behavior problems during this developmental period should contribute to an understanding of the etiology of adult psychopathology. Improved understanding of the etiology of antisocial behavior may also contribute to better treatment and prevention.

2 BACKGROUND

2.1 DEFINITIONS OF ANTISOCIAL BEHAVIOR

Antisocial behavior has mainly been studied by three different disciplines. These disciplines are broadly the psychiatric field, the criminological field, and the psychological field (Moffitt, 2005a, Plomin et al., 1990). Even though antisocial behavior is defined and measured slightly different by these three research traditions, what they have in common is the underlying assumption that antisocial behavior is a behavior that violates the rights and safety of others.

2.1.1 The psychiatric field

The psychiatric field examines antisocial behavior in terms of psychiatric diagnoses, that is, conduct disorder (CD) and antisocial personality disorder. These definitions require that the behavior is causing physical harm to others, involves a number of different types of antisocial acts, and has persisted over long periods of time (DSM-IV, 1994).

The characteristic feature of conduct disorder in children and adolescents is a repetitive and persistent pattern of behaviors including aggressive conduct causing physical harm to people and/or animals, nonaggressive conduct causing property loss or damage, deceitfulness, theft, and violation of rules. A diagnosis of conduct disorder requires that three or more of these behaviors must have been present in the last twelve months. Conduct disorder is mainly diagnosed in individuals 18 years or younger. The behavior should also cause significant impairment in social and academic functioning. Conduct disorder can further be divided into two subtypes: childhood-onset type and adolescent-onset type. The childhood-onset type requires an onset of conduct disorder prior to the age of ten. It typically includes individuals that are frequently aggressive towards others, have disturbed peer relationships, and may also have been diagnosed with oppositional defiant disorder in early childhood. Oppositional defiant disorder is a pattern of disobedient and hostile behavior towards authority figures. The adolescent-onset type, on the contrary, does not require an onset prior to age ten. Compared to the childhood-onset type, adolescent-onset individuals tend to be less aggressive and to have more normative peer relationships (DSM-IV, 1994).

The prevalence rates of conduct disorder for children and adolescents range between 1–15 % in community samples (Loeber et al., 2000, Costello et al., 2003). A general finding is that conduct disorder is more prevalent among boys (Costello et al., 1996, Hipwell et al., 2002), and that there is an increasing prevalence with age, in both sexes, especially in the mid-teens (Maughan et al., 2004).

Antisocial personality disorder (ASPD) is a pervasive pattern of disregard for and violation of the rights of others occurring since childhood or early adolescence. Individuals diagnosed with antisocial personality disorder must be at least 18 years old, and have had a history of conduct disorder before age fifteen. Three or more of the following criteria is required: failure to conform to social norms with respect to lawful behaviors, in other words, repeatedly performing acts that are grounds for arrests; deceitfulness, that is, repeated lying or conning others for personal profits; impulsiveness and a failure to plan ahead; irritability and aggressiveness as indicated by reckless disregard for safety of one self and others; and consistent irresponsibility and lack of remorse (DSM-IV, 1994).

The prevalence of antisocial personality disorder in community settings is about 3 % in males and 1 % in females, and in forensic settings about 3–30 % (DSM-IV, 1994). Both conduct disorder and antisocial personality disorder are typically measured as diagnostic criteria categories; either the individual meets the criteria for the disorder, or not.

2.1.2 The criminological field

The criminological field defines antisocial behavior as violation of legal or social norms, referred to as criminality among adults, and as delinquency among juveniles. This definition does not require there to be serious harm, a variety of acts, or persistence. Still, criminologists make distinctions between ‘one-off’ offenders and those offending with high frequency, and between trivial and violent and serious offenders. Criminologists usually measure antisocial behavior through official records (Lilly et al., 1995). In general, the majority of reported crimes are larceny crimes, i.e., various types of theft and burglary, followed by vandalism, crime against the person, traffic crime, fraud, and narcotics offences. For crimes to be included in official records they must be discovered and reported to the police. This is far from the case in respect

of many types of crime, such as petty theft or traffic offences. Consequently, there is hidden criminality that is not seen in the official records. Criminologists therefore also measure crime through victimization surveys, and most commonly through self-reports (The Swedish National Council for Crime Prevention, 2004). When measured with self-reports, the number of different illegal behaviors committed and the frequency with which they have been committed are counted. Depending on age, these legal definitions of antisocial behavior tend to apply to approximately 20–30 % of the population (Moffitt, 2005a).

2.1.3 The psychological field

Antisocial behavior is also conceptualized as aggressive behavior (Plomin et al., 1990). Aggressive behavior is usually studied in the psychology field as a personality characteristic or a trait. Personality traits tend to apply to the entire population; the high end of an 'aggression' scales may indicate a more aggressive individual and the lower end may indicate timidity. As personality traits are assumed to be continuously distributed, they are assessed with a checklist which counts the number of different aggressive behaviors endorsed by the individual.

2.1.4 Definition of antisocial behavior in this thesis: a dimensional approach

In this thesis is antisocial behavior fairly broadly defined; it includes acts that involve breaking the law, irrespective if the individual is caught and prosecuted, and also other types of problem behaviors that are not necessarily illegal. Acts committed by children below the age of criminal responsibility (15 years in Sweden) for which they cannot be prosecuted are also included. The breadth of antisocial behavior so defined, is such that it does not fall into distinct categories, but is better conceptualized as quantitative variations of behaviors that most individuals show to a greater or lesser degree. Antisocial behavior is therefore considered in this thesis as a behavior or trait that is normally distributed and possible to be measured as part of variation in the normal range.

2.2 MULTIFACTORIAL EXPLANATIONS TO ANTISOCIAL BEHAVIOR

It is generally assumed that antisocial behavior has a multifactorial origin. In other words, it is thought to arise from the effects of multiple genes, as well as the effects of

multiple environmental factors. The factors influencing the behavior may be several alleles at one locus, several gene loci, environmental factors, or any combination of these. These factors combine additively to make up the total liability for the behavior (Lahey et al., 2003, Rutter, 2006, Rutter et al., 1998).

2.3 TWIN STUDIES AS A TOOL FOR UNDERSTANDING INDIVIDUAL DIFFERENCES

The main focus in this thesis has been to increase the understanding of the development of antisocial behavior through behavioral genetic methods. Behavioral genetics is concerned with the study of individual differences, that is, detecting the factors that make individuals in the population different from each other. By partitioning the variance into genetic and environmental components, the broad causes of individual differences in a trait is being revealed. In the classical twin design, monozygotic twin pairs, who share all their genes are compared to dizygotic twin pairs, who on average share half of their segregating genes to estimate the contribution of genetic and environmental factors to individual differences in a phenotype of interest (Plomin et al., 2001, Neale & Cardon, 1992), in this case antisocial behavior.

Behavioral genetics has in recent years rapidly moved beyond the initial question of whether behavior or trait is heritable (Dick & Rose, 2002, Kendler, 2001), and an important extension of the univariate twin design is multivariate modeling. Multivariate modeling involves analyzing correlated traits simultaneously (Posthuma & Boomsma, 2005, Boomsma et al., 2002). This type of analyses allows modeling causes of covariation between traits, testing for gene-environment interaction, and analysis of longitudinal data to study developmental patterns.

This thesis has mainly used multivariate twin methods to analyze longitudinal data. The overall aim has been to investigate the development of antisocial behavior from childhood to emerging adulthood.

2.4 THE NATURE OF ANTISOCIAL BEHAVIOR

There are certain patterns regarding antisocial behavior that have been established through empirical research. For example, official statistics and victim surveys show that juveniles account for a large proportion (approximately one fourth to one third) of

all crimes. Self-reports generally suggest that between 50–80 % participate in antisocial behavior at some time during childhood or adolescence. Another finding is that a small proportion of all antisocial individuals (5–7 %) accounts for approximately half of all antisocial acts (Rutter et al., 1998, Loeber & Farrington, 1998, Vermeiren, 2003). It is also well documented that males are much more likely than females to engage in most forms of antisocial behavior (Moffitt et al., 2001, Rutter et al., 2003, Junger-Tas et al., 1994). This sex difference is widest for violent crimes (Smith & Visher, 1980, Rutter et al., 1998), and narrowest for drug and alcohol related crimes (Moffitt et al., 2001). Moreover, antisocial behavior has been found to increase in early adolescence, to peak in mid-adolescence, and then to drop sharply in young adulthood (Moffitt, 1993a). Most of the antisocial acts committed are theft-related, and only a small proportion is aggressive and violent (Ring, 2005, Farrington & Loeber, 2000). There are also some well-established developmental patterns in antisocial behavior. For example, individuals with an early age of onset are more likely to persist in antisocial behavior (Loeber & Farrington, 2000, Stouthamer-Loeber & Loeber, 2002, Simonoff et al., 2004, Robins, 1978, Tremblay et al., 1994).

2.4.1 The influence of genetic and environmental effects on antisocial behavior

There are now a fairly substantial number of studies exploring the role of genes and environments in antisocial behavior. These studies consistently demonstrate that the etiology of antisocial behavior depends both on genetic and environmental effects (Rhee & Waldman, 2002). However, findings regarding the influence of genetic and environmental factors on antisocial behavior tend to differ more across studies than for other psychopathological phenotypes, e.g., attention-deficit hyperactivity disorder (ADHD), depression, schizophrenia, alcoholism (Slutske, 2001). Some studies have reported very low heritability estimates (Lyons et al., 1995), whereas others have reported very high heritability estimates (Ghodesian-Carpey & Baker, 1987, Slutske et al., 1997a, Rowe, 1983). This variation in heritability estimates for antisocial behavior across samples could be due to genetic and environmental differences between populations (Kendler, 2001). But it could also be due to methodological differences, for example the measurement of antisocial behavior (e.g., psychiatric diagnosis, criminality/delinquency, aggression) (Goldstein et al., 2001, Slutske et al., 1997a, Jacobson et al., 2000, Lyons et al., 1995, Gelhorn et al., 2005). The lack of consistency

in the literature regarding the influence of genetic and environmental influences on antisocial behavior warrants further investigation.

2.4.2 The role of sex differences in the genetic and environmental etiology of antisocial behavior

As mentioned above, a much higher proportion of males than females engage in antisocial behavior. This sex difference in prevalence remains consistent over age; it is substantial in childhood, narrows somewhat in adolescence, and increases again in late adolescence and early adulthood (Moffitt et al., 2001, Rutter et al., 2003). Very little is known about the mechanisms that underlie these sex differences. Given this sex differences in prevalence, it is also interesting to examine whether the magnitude of genetic and environmental effects differs in males and females, and whether the genes that influence the liability to antisocial behavior are the same in the two sexes. Earlier behavior genetic studies have been inconsistent regarding sex differences in antisocial behavior, with some reporting significant sex differences (Miles & Carey, 1997), and others reporting no sex differences (Rhee & Waldman, 2002). Therefore, the first aim in this thesis was to examine the role of sex differences in the genetic and environmental etiology of antisocial behavior.

2.4.3 The influence of genes and environments on the development of antisocial behavior

To understand different developmental patterns through which children develop antisocial behavior, many different theories have been suggested. For example, the fact that antisocial behavior peaks in adolescence and that age of onset is related to persistence is the starting point for a developmental taxonomy of antisocial behavior that differentiates the most deviant over the life course from those likely to show temporary difficulties during adolescence. The theory proposes that 'life-course persistent' and 'adolescent-limited' antisocial behavior differs in terms of etiology, developmental course, prognosis, and classification of behavior as pathological versus normative. Life-course persistent antisocial behavior is thought to have a neuro-developmental origin, and to begin at a very young age and continue from adolescence into adulthood. Adolescence-limited antisocial behavior on the other hand, is thought to be restricted to adolescent years and to be more influenced by social peer pressure

(Moffitt, 1993a). DiLalla and Gottesman, (1989) have also suggested a third group called 'late bloomers', who are thought to begin their offending in adulthood.

Another theory aiming at understanding developmental patterns of antisocial behavior posits that aggressive (overt) and nonaggressive (covert) behavior represents different developmental pathways or trajectories toward antisocial behavior (Loeber & Hay, 1997, Achenbach, 1991, Frick et al., 1993). It should be mentioned that a pathway or trajectory in this context is defined as a common pattern of development shared by a group of individuals which is distinct from the behavioral development experienced by other groups of individuals (Loeber & Coie, 2001). The focus of this theory is on the progression along different pathways from more frequent and milder antisocial behavior to less frequent but more severe criminal behavior (Loeber & Hay, 1997). The aggressive pathway is thought to begin with childhood aggression such as bullying and annoying others, followed by physical fighting and ending in violent crime. The nonaggressive pathway begins with minor behaviors in childhood such as lying and shop-lifting, moves on to property damage (vandalism, setting fires), and ends with serious forms of delinquency such as stealing cars, breaking and entering property, selling drugs and fraud. The further progression in a given pathway, the greater the likelihood would be of later involvement in both pathways.

Differences in aggressive and nonaggressive developmental trajectories have also been supported empirically (Tolan & Gorman-Smith, 1998, Stanger et al., 1997). Further support is provided by twin studies, in that aggressive behavior has been found to be highly heritable (Ghodesian-Carpey & Baker, 1987, Hudziak et al., 2003, Edelbrock et al., 1995, Eley et al., 1999), whereas nonaggressive behavior shows a roughly equal influence of genes and shared environment (Edelbrock et al., 1995, Eley et al., 2003, Bartels et al., 2003). Twin studies analyzing aggressive and nonaggressive behavior simultaneously suggest unique genetic and environmental factors, but also common genetic and environmental influences (Gelhorn et al., 2006, Button et al., 2004). Longitudinal twin studies provide great power to clarify the developmental pathways through which genes and environments contribute to the development of antisocial behavior. So far, the influence of genetic and environmental effects on the development of aggressive and nonaggressive behavior in childhood to antisocial behavior in adolescence is less explored.

A related issue is that longitudinal and multivariate twin studies generally report higher heritability estimates than cross-sectional studies. A meta-analysis, including 51 twin and adoption studies, reported a moderately strong heritability of 41 % (additive and non-additive genetic effects) for antisocial behavior measured in various ways (Rhee & Waldman, 2002). In contrast, variation in pervasive antisocial behavior, as agreed by multiple informants, has been reported to be highly heritable (Arseneault et al., 2003, Scourfield et al., 2004b). For example, Arsenault et al. (2003), using data from mothers, teachers, observers and children themselves, demonstrated that heritability was higher (82 %) for a shared latent phenotype than for corresponding single measures of antisocial behavior. Further, antisocial behavior persisting from adolescence to early adulthood (Jacobson et al., 2001), and childhood onset antisocial behavior (Taylor et al., 2000a, Slutske et al., 1997c) have also been found to have a stronger genetic component. Thus, these results suggest that the effect of genes and environments might be different when studied in a developmental perspective.

2.5 FACTORS ASSOCIATED WITH ANTISOCIAL BEHAVIOR

Earlier research has established a number of factors that are associated with antisocial behavior. Some of these factors may either function as risk or protective factors. In other words, it is assumed that they are the same variable at the opposite ends of a continuum. For example, negative parenting is a risk factor, whereas positive parent-child relationship is a protective factor. The larger the accumulation of risk factors, the higher probability of a later negative outcome, such as antisocial behavior (Stouthamer-Loeber et al., 2002). Further, Kraemer and colleagues (Kraemer, 2003) have pointed out that the term 'risk factor' should only be used when a factor predicts an antisocial outcome. That is, risk factors should be distinguished from variables that are correlated cross-sectionally, or correlated after the outcome.

Factors that are associated with antisocial behavior include for example *biological factors* (e.g., low resting heart rate, cortisol levels, serotonin levels, genes) (Ortiz & Raine, 2004, McBurnett et al., 2000, Moore et al., 2002, Caspi et al., 2002); *prenatal factors* (e.g., fetal exposure to alcohol, smoking and/or malnutrition) (Raine, 2002b); *individual level factors* (e.g., impulsivity, attention/hyperactivity problems, aggression, callous and unemotional traits, stimulation-seeking, fearlessness, depressive symptoms,

executive dysfunction, poor verbal ability, substance abuse, early onset menarche) (Vermeiren, 2003, Nigg & Huang-Pollock, 2003, Raine, 2002b, Viding et al., 2005, Lahey et al., 2003, Stattin & Magnusson, 1990, Mustanski et al., 2004, Burt et al., 2006, Caspi et al., 1993); *family factors* (e.g., parental criminality, poor child rearing practices, parental substance use, low socioeconomic status, maltreatment, single-parent household, young motherhood, large family size) (Cottle et al., 2001, Lipsey & Derzon, 1998, Caspi et al., 2002, Farrington et al., 1996, Loeber & Dishion, 1983); *school factors* (e.g., poor academic performance, weak bonding to school) (Hawkins et al., 1998, Loeber & Farrington, 2000); *peer factors* (e.g., delinquent peers and siblings, gang membership, peer rejection) (Farrington & Loeber, 2000); and *contextual factors* (e.g., neighbourhood disadvantage and poverty, availability of weapons, to reside in urban versus rural areas) (Beyers et al., 2001, Sampson et al., 1997, Brooks-Gunn et al., 1993, Lynam, 2000).

Although it is well-established what factors are associated with antisocial behavior, less is known about the underlying mechanisms of how these are related to antisocial behavior. This has led several researchers to conclude that the study of antisocial behavior is 'stuck in the risk factor stage' (Moffitt, 2005b, Hinshaw, 2002, Rutter, 2003). One way to further examine how some of these factors are associated to antisocial behavior is using a genetic sensitive design.

2.5.1 Covariation

As aforementioned, certain individual level factors (e.g., behaviors, disorders, personality traits), are associated with antisocial behavior, i.e., comorbidity or covariation. Generally, comorbidity refers to categorical data and dichotomous measures, whereas covariation refers to dimensional data and continuous measures. It is important to understand the causes of covariation because the individuals exhibiting only one problem behavior are likely to have different prognoses, treatment responses, and risk factors compared with individuals who have multiple problems. Even though covariation among antisocial behavior and various individual level factors has been has been repeatedly found in both epidemiological and clinical samples (Angold et al., 1999b, Biederman et al., 1995, Newman et al., 1996, Kim-Cohen et al., 2003, Young et al., 2000), the causes of such covariation are much less understood. Behavior genetic models can assess the degree to which covariation of antisocial behavior and individual

level factors are due to common genetic influences, common environmental influences, or both.

Several twin studies have been conducted to examine the covariation between antisocial behavior and some of its associated factors. Some studies have examined the association between antisocial behavior and hyperactivity-impulsivity-inattention problems (Nadder et al., 2002, Silberg et al., 1996, Thapar et al., 2001, Dick et al., 2005). Others have investigated the covariation between antisocial behavior and substance use and dependence (Slutske et al., 1998, Button et al., 2006, Hicks et al., 2004, Miles et al., 2002), depression symptoms (O'Connor et al., 1998b), social cognition (Scourfield et al., 2004a), and pathological gambling (Slutske et al., 2001). A few studies have concluded that the association depends on environmental factors, (e.g., reading achievement (Trzesniewski et al., 2006), alcohol dependence (Rose et al., 2004), hyperactivity-impulsivity-inattention problems and oppositional defiant disorder (Burt et al., 2001)). However, the majority of these studies have reported a considerable genetic overlap between antisocial behavior and these different traits.

Another individual level factor that is associated with antisocial behavior is psychopathic personality traits (Walters, 2003, Hart & Hare, 1997). Psychopathy is in its adult manifestation considered a serious personality disorder characterized by a constellation of interpersonal, affective, and behavioral traits. Interpersonally, psychopaths are superficial and egocentric; affectively, they are shallow and callous; and behaviorally they are impulsive and irresponsible (Cleckley, 1941, Hare, 1991).

A substantial amount of literature indicates that psychopathy is a reliable, valid, and meaningful construct in adults. It is well established that psychopaths account for a disproportionate amount of crime, are more likely to commit violent offences, have higher rates of recidivism, and are less motivated and amenable to treatment (Hemphill et al., 1998, Grann et al., 1999, Salekin et al., 1996, Hare et al., 2000).

Studies have shown that psychopathic personality traits can be assessed reliably in adolescence (e.g., Andershed et al., 2002, Forth et al., 2003, Lynam & Gudonis, 2005, Vitacco et al., 2003) and that these traits are relatively stable over the course of development from late childhood into adolescence (Frick et al., 2003). Also, this

personality type seems to be present, measurable and manifested in similar ways in referred and non-referred adolescents (Skeem & Cauffman, 2003).

A previous twin study has examined the genetic and environmental overlap between psychopathic traits and antisocial behavior in adolescence (Blonigen et al., 2005). This study used a self-report normal range personality measure to index the interpersonal-affective (called Fearless Dominance) and impulsive-antisocial dimensions (called Impulsive Antisociality) of psychopathy. They found a strong genetic overlap between Fearless Dominance and externalizing behavior (antisocial behavior and substance use) in males and females, and between Impulsive Antisociality and externalizing behavior, but only in males (Blonigen et al., 2005). The study was cross-sectional; it did not include opposite sex twins and could therefore not test for sex differences. Thus, the association between psychopathic personality traits and antisocial behavior needs to be further investigated.

2.5.2 Gene-environment interaction

It is also possible that some of the factors associated with antisocial behavior act as moderators of the influence of genetic and environmental factors on antisocial behavior, that is, gene-environment interaction.

Evidence of gene-environment interactions for antisocial behavior has been demonstrated in molecular genetic studies (Caspi et al., 2002, Foley et al., 2004, Kim-Cohen et al., 2006). For example, Caspi et al (2002) reported that a functional polymorphism in the gene encoding the neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA) moderated the impact of early childhood maltreatment on the development of antisocial behavior in males. Maltreated boys with a genotype conferring high levels of MAOA expression were found to be less likely to develop antisocial problems than maltreated boys who had a genotype conferring low levels of MAOA expression.

An indirect way to test for gene-environment interaction is to use data from adoption studies. Adoption studies generally demonstrate that the combination of a genetic predisposition (i.e., psychopathology in biological parents) with a high risk environment (i.e., adoptive home environment) leads to greater pathology than what

would be expected from either factor acting alone or both in an additive combination (Crowe, 1974, Cadoret et al., 1983, Bohman et al., 1982, Mednick et al., 1984, Cadoret et al., 1995). A notable example is a study by Cloninger and colleagues (1982). The interaction of congenital (i.e., whether the biological parents were criminal) and postnatal background (i.e., adverse rearing experiences and adoptive placement) was studied in 862 Swedish men adopted at an early age by non-relatives. When both hereditary factors and environmental factors were present, 40 % were found to be criminal, if only genetic factors were present 12.1 % were criminal, if only environmental factors were present 6.7 % were criminal and with neither hereditary nor bad environmental factors being present 2.7 % were criminal.

It is also possible to use twin studies to test for gene-environment interaction (Lau & Eley, 2004, Rowe, 2003). For example, one study found that the heritability of conduct problems was lower in dysfunctional families and higher in families where dysfunction was absent (Button et al., 2005). Further, Rowe and colleagues (1999) reported that the genetic influence on aggression was higher in schools with higher average levels of family warmth. In contrast, both shared and non-shared environmental influences were important in schools with lower average levels of family warmth. The authors concluded that a greater genetic effect is required for the expression of aggression in more benign environments, whereas in more disadvantaged environments, negative family-related factors and context-dependent risks may promote aggressive behavior even among individuals without a genetic predisposition (Rowe et al., 1999). Other twin studies have reported that contextual factors e.g., regional residency (Rose et al., 2001), and socio-demographic characteristics of the community of residence (Dick et al., 2001) moderates the influence of genetic and environmental factors on alcohol use in adolescents. This suggests that contextual factors may also be important moderators for antisocial behavior. Thus, the fourth aim of this thesis was to examine socioeconomic status as a possible moderator of genetic and environmental influences on antisocial behavior.

3 AIM

The overall aim of this thesis has been to investigate the influence of genetic and environmental factors in the development of antisocial behavior from childhood to emerging adulthood. More specifically;

To further investigate the nature of antisocial behavior by

(1) Examining the role of sex differences in the genetic and environmental etiology of antisocial behavior (study I to IV)

(2) Investigating how genes and environments influence the development of antisocial behavior (study I, II)

To investigate some of the factors associated with antisocial behavior by

(3) Exploring the covariation between antisocial behavior and psychopathic personality traits (study III)

(4) Investigating whether socioeconomic status moderates the impact of genetic and environmental influences on antisocial behavior, that is, gene-environment interaction (study IV).

4 METHODS

4.1 SAMPLE

This thesis is based on data from the Twin Study of CHild and Adolescent Development (TCHAD). TCHAD is an ongoing prospective longitudinal study concerning health and behavior in children and adolescents. The sample was derived from the population-based Swedish Twin Registry, which in principle contains information on all twins born in Sweden since 1886 (Lichtenstein et al., 2002). The initial sample consists of all 1,480 twin pairs, born in Sweden between 1st of May, 1985 and 31st of December, 1986, where both in the pair were still living and residing in Sweden at the time of the start of the study.

The twins have been followed-up from childhood (age 8-9), throughout early (age 13-14) and late adolescence (age 16-17), into emerging adulthood (age 19-20). The study includes questions concerning for example socio-demographic factors, physical health, puberty, personality (e.g., psychopathic personality traits), externalizing behavior (e.g., attention deficit hyperactivity disorder, antisocial behavior), internalizing behavior (e.g., fears, phobias), drug and alcohol use, and parent-child relationships.

At *wave 1* in 1994 when the twins were 8-9 years old, the parents received a mailed questionnaire of which 75 % responded (n = 1,103). Non-responders were approached with up to three reminders. A telephone follow-up was conducted with a reduced battery of questions. Combining the larger study and the smaller telephone study, at least some information was collected on 91 % (n = 1,335) of the twin pairs.

At *wave 2* in 1999 when the twins were 13-14 years old, data were again collected from the parents, but this time the twins were also used as informants (parent's response rate = 73 % (n = 1,063); children's response rate = 78 % (n = 2,261)). Complete information from the whole family between wave 1 and wave 2 is available for 911 of the twin pairs. In addition, two smaller sub-studies were conducted at wave 2. In the first one, the children's teachers were contacted via a mailed questionnaire. However, to contact the teachers, written consent had to be obtained from both the children and their parents. This resulted in permission to contact only 1,120 teachers, of which 67 %

(n = 745) responded. The second sub-study was a clinical study of the 271 twin pairs living in the Stockholm area. Of those, 156 twin pairs were examined through a clinical psychiatric interview (K-SADS-PL; (Ambrosini, 2000)) by a child psychiatrist. DNA was also collected from these twins.

At *wave 3* in 2002 when the twins were 16-17 years old, questionnaires were again sent out to both parents and children (parents' response rate = 74 %, (n = 1,067); children's response rate = 82 %, (n = 2,368)). Responses were also obtained from an additional 5 % of the twins with a telephone follow-up, using a reduced battery of questions (n = 2,525 twins). Complete information from the whole family between wave 2 and wave 3 is available for 906 of the twin pairs. Furthermore, the majority of the parent-reported information was supplied by mothers rather than by fathers (range: 75-90 %) (Lichtenstein et al., in press).

At *wave 4* in 2005 when the twins were 19-20 years old a renewed contact was established with these families: ((parents' response rate = 51 %, (n = 1,197); twins' response rate = 59 % (n = 1,698)). At wave 4, consent to contact the parents was obtained from the twins. Also, mothers and fathers were approached separately this time.

4.2 ZYGOSITY DETERMINATION

To determine zygosity of the same-sexed twin pairs, parents (wave 1, 2 and 3) and twins (wave 2 and 3) were asked to complete a series of four questions concerning the twin pairs' physical similarity and the frequency with which people confused them. Algorithms derived from discriminant analyses on 106 like-sexed twin pairs participating in the clinical study described above with known zygosity (based on 16 polymorphic DNA-markers) was used. The algorithms only classify pairs that have a 95 % probability of being correctly classified as monozygotic (MZ) or dizygotic (DZ). Using the algorithm five preliminary zygosity determinations from parents' response (wave 1, 2 and 3) and children's response (wave 2 and 3) were established. A final zygosity was then determined from these five preliminary zygosity assignments. In case of contradiction between any of the five zygosity determinations, zygosity was determined as unknown.

This zygosity classification was used in Study II, III and IV. In Study I, zygosity was determined using parent response from wave 1 and wave 2; and child response from wave 2. During wave 4, DNA from all twins has been collected with oragene® via mail which will allow further refinements of the zygosity classification. At the time of writing this thesis, this DNA has not yet been analysed.

4.3 MEASURES

The main focus in this thesis is on the development of antisocial behavior. Below is a description of how antisocial behavior was measured (Study I to IV). In addition, the other measures used in this thesis, i.e., aggressive and nonaggressive behavior (Study I) and psychopathic personality traits (Study III) are presented. Further, a short description is also given of the moderator variables (socioeconomic status) used in Study IV.

4.3.1 Antisocial behavior

Antisocial behavior at ages 13-14 (wave 2), 16-17 (wave 3), and 19-20 (wave 4) was measured using an extensive self-report questionnaire of 34, 32, and 31-items, respectively. The questionnaire is part of an extensive battery of questions, which has been developed by the Department of Criminology, Stockholm University (Ring, 1999). The items used were initially derived from an instrument used in the project *Delinquent Behavior among Young People in the Western World* comparing self-reports of delinquency in 13 countries (Junger-Tas et al., 1994). The questionnaire served as an indicator of the frequency with which the adolescents had participated in illegal acts in the past twelve months. The questionnaire roughly covered three different areas (Table 1): (i) *Property offences and problem behavior* including 23 items such as shop lifting, breaking and entering, vandalism, motor vehicle theft, several other kinds of thefts and fraud. (ii) *Violent offences* including 5 items about simple assault, fighting, and robbery. (iii) *Drug-related offences* including 3 items about using and selling various types of illicit drugs. The twins were asked to indicate the frequency with which they had engaged in these behaviors, ranging from 0 (never), 1 (1-2 times), 2 (3-5 times), 3 (6-10 times), 4 (11-50 times), to 5 (more than 50 times).

It is well known that antisocial individuals often show a pattern of versatile offending (Klein, 1995). Factor analyses on the antisocial behavior items resulted in a single

factor at each wave with excellent internal consistency (age 13-14: $\alpha = .87$; age 16-17: $\alpha = .92$; age 19-20: $\alpha = .83$); consequently, we analyzed it as a single composite scale. An antisocial behavior score was created by summing across all items. The three scales were skewed, (skewness: 13-14 year: 4.14; 16-17 years: 4.73; 19-20 year: 2.86), and to approximate normal distribution each scale was transformed ($\log_{10}(x+1)$) prior to analyses (skewness: 13-14 year: .99; 16-17 years: .59; 19-20 years: .49).

From wave 2 to wave 3, the questionnaire was revised in the following way: the item 'have you used anabolic steroids?' was deleted as no one reported having done that at wave 2. The two items 'have you used hashish/marijuana?' and 'have you used any other illicit drug, e.g., amphetamine, heroine, ecstasy' etc, were collapsed into one item. From wave 3 to wave 4 the questionnaire was again revised: The items 'have you used any illicit drug, and 'have you sniffed glue' were collapsed into one item.

Reliability and validity of the antisocial behavior measure are discussed under Methodological considerations, Discussion section.

Table 1 Items included in the Antisocial behavior scale, wave 4

Property offences and problem behavior		
Shop-lifting	Pocket-picking	Writing on public walls/surfaces
Steal from school	Purse/bag-snatching	Theft from vending machine
Steal at home	Theft from car	Train/bus fare evasion
Unspecified thievery	Graffiti	Ride motorbike/car without licence
Buy stolen goods	Motorbike theft	Use false ID card
Bike theft	Car theft	Cheat or lie to get money
Sell stolen goods	Arson	Truancy
Burglary	Vandalism	
Violent offences		
Carry a knife	Threaten for money	Hurt someone with a weapon
Beat non-family member	Beat family member	
Drug-related offences		
Use drugs	Sell hard drugs	Sell soft drugs

4.3.2 Aggressive and nonaggressive behavior

The Child Behavior Checklist (CBCL) (Achenbach, 1991) is a widely used measure of general behavioral and emotional problems in children and adolescents. The CBCL consists of eight scales: aggressive behavior, attention problems, nonaggressive (or rule-breaking) behavior, depression/anxiety, social problems, somatic complaints, thought problems, and withdrawn. The CBCL has been shown in several studies to be a reliable and valid instrument for assessment of behavioral and emotional problems in children and adolescents (Achenbach & Rescorla, 2000). The CBCL was completed by parents when the twins were 8-9 years old and the aggressive and nonaggressive behavior scales were used in Study I.

The aggression scale is made up by 20 items (Table 2), including both physically aggressive antisocial behaviors such as destroying one's own and other's belongings, fighting with other children, and attacking others, as well as personality-type items such as argues a lot, brags and boasts, and being stubborn.

The nonaggressive behavior scale consists of 13 items, including more nonaggressive antisocial behaviors such as lying, and/or stealing at home. The items had a three-point response format: 0 if the item is not true, 1 if it is sometimes or somewhat true and 2 if is very true or often true. A score was created by summing across the items. The internal consistencies (Cronbach's alpha) of the two scales used in the study were adequate (aggressive behavior scale $\alpha = .89$, nonaggressive behavior scale $\alpha = .71$).

Table 2 Items included in the Aggressive behavior and Nonaggressive behavior score from the Child Behavior Check List

Aggressive Behavior	Nonaggressive Behavior
Argues a lot	Doesn't seem to feel guilty after misbehaving
Bragging, boasting	Hangs around with others who get into trouble
Cruelty, bullying, or meanness to others	Lying or cheating
Demands a lot of attention	Prefers being with older kids
Destroys his/her own things	Runs away from home
Unusually loud	Sets fires
Disobedient at home	Steals at home
Disobedient at school	Steals outside the home
Easily jealous	Swears or uses obscene language
Gets teased a lot	Thinks about sex too much
Physically attacks people	Truancy, skips school
Screams a lot	Uses drugs for non-medical purposes
Shows off or clowns	Vandalism
Stubborn, sullen or irritable	
Sudden changes in mood or feelings	
Talks too much	
Teases a lot	
Temper tantrums or hot temper	
Threatens people	
Destroys things that belongs to his/her family members or others	

4.3.3 Psychopathic personality traits

The Youth Psychopathic traits Inventory (YPI) is a 50-item youth self-report questionnaire designed to measure the core traits of the psychopathic personality constellation in young people of 12 years or older (Andershed et al., 2002). The YPI questionnaire was completed by the twins when they were 16-17 years old (wave 3), and used in Study III.

Table 3 The ten subscales of the Youth Psychopathic traits Inventory (YPI), with two sample items for each subscale

Subscales	Items included in the YPI
Dishonest charm	I have the ability to con people by using my charm and smile.
	When I need to, I use my smile and charm to use others.
Grandiosity	I am more important and valuable than other people.
	I am better than everyone at almost everything.
Lying	Sometimes I lie for no reason, other than because it is fun.
	I have often gotten into trouble because I have lied too much.
Manipulation	I can get almost anyone to believe anything.
	To get what I want, I often find it efficient to con others.
Callousness	When other people have problems it is often their own fault, therefore one should not help them.
	I often become sad or moved by watching things on TV or film (reversed).
Unemotionality	I don't let my feelings affect me as much as other people do.
	I usually feel calm when other people are scared.
Remorselessness	I have the ability not to feel guilt and regret about things that I think other people would feel guilty about.
	To feel guilty and remorseful about things you have done that have hurt other people is a sign of weakness.
Impulsiveness	It often happens that I do things without thinking ahead.
	I prefer to spend my money right away rather than save it.
Thrill-seeking	I like to do things just for the thrill of it.
	I get bored quickly by doing the same thing over.
Irresponsibility	I have cut class more often than other people.
	It happened several times that I have borrowed something and then lost it.

The instrument primarily focuses on the core personality traits of the psychopathic personality constellation, rather than on the behavioral traits associated with it (Andershed et al., 2002). To avoid response distortion and social desirability effects, items have been framed as potentially positive attributes. That is, the items were phrased so as they should seem neutral or even appealing to those with psychopathic traits (e.g., 'I usually feel calm when other people are scared' instead of 'My emotions are more shallow than others'). As these traits are generally considered by others to be socially undesirable or malignant, this would decrease the likelihood that adolescents possessing these traits would deny having them.

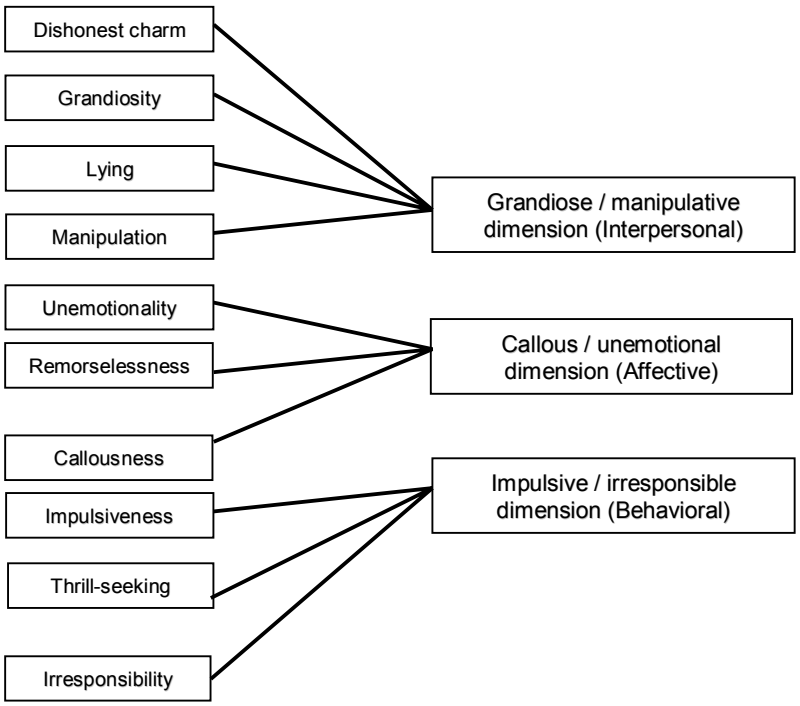
The YPI uses a Likert-type scale (1 = does not apply at all, 2 = does not apply well, 3 = applies fairly well, 4 = applies very well), and measures each psychopathic trait with five items, making up ten subscales. The ten subscales of the YPI with two sample items each are presented in Table 3.

When the YPI was used in a longitudinal community-based sample of 1,279 (response rate = 80 %) 16-year-old adolescents in a medium sized Swedish community, the ten subscales were found to be internally consistent (Cronbach alphas = .66 - .82). The YPI was also validated against self-reported measures of conduct problems, and as expected found to be positively correlated with such problems. Further, a three-factor structure was supported by both exploratory and confirmatory factor analyses (Andershed et al., 2002), and is similar to the three-factor structure reported for adults (Cooke & Michie, 2001).

The three factors of the YPI (Figure 1) consist of: (i) a Grandiose/Manipulative Dimension (Interpersonal): including the subscales Dishonest charm, Grandiosity, Lying and Manipulation, (ii) a Callous/Unemotional Dimension (Affective): including the subscales Callousness, Unemotionality and Remorselessness, (iii) and an Impulsive/Irresponsible Dimension (Behavioral): including the subscales Impulsiveness, Thrill-seeking and Irresponsibility.

In line with this, Larsson and colleagues (2006) have previously shown, using the TCHAD-sample, that the YPI subscales are moderate to adequate internally consistent (Cronbach alphas = .58 - .79). Also, tests showed that a three-factor model fitted significantly better than a one-factor and a two-factor model (Larsson et al., 2006).

Figure 1



The Youth Psychopathic traits Inventory (YPI) consists of three dimensions that include ten subscales.

Furthermore, the YPI has also been reliability-tested and validated using a sample of incarcerated male juvenile offenders (n = 160; ages: 14 to 17) (Skeem & Cauffman, 2003). The ten YPI subscales were found to have a moderate to satisfactory internal consistency ranging from .49 to .85. Validity was determined by correlating the YPI with scores from other valid measures, i.e., concurrent validity. The YPI was reported to be positively correlated with the Psychopathy Checklist-Youth Version (PCL-YV) (Forth et al., 2003, Hare, 2003), and negatively associated with the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1985).

4.3.4 Socioeconomic status

Family socioeconomic status was measured using information on (1) occupational status and (2) educational level, reported by the twins' parents at wave 3 and used in study IV. Occupational status and educational level was categorized by the person with the highest occupation and education in the family. (1) Family occupational status was measured with the SEI (socioeconomic classification) scale (Statistics Sweden, 1995). Fifteen percent of the families in our sample were unskilled and semiskilled workers, 29 % were skilled workers and assistant non-manual employees, 28 % were

intermediate non-manual employees, and 28 % were employed and self-employed professionals, higher civil servants and executives. (2) Family educational level was classified as elementary school (7 % of the families in our sample), secondary school (29 %), junior college (18 %) and university (46 %). Prior to analyses, family occupational status and family educational level were independently transformed ($\log_{10}(x+1)$) due to the positive skewness of their distributions (Lichtenstein et al., 1992, Lichtenstein et al., 1993).

Five variables measured on an aggregate level were used to assess *neighbourhood socioeconomic conditions* in study IV. (1) Ethnic diversity, (2) educational level, (3) unemployment level, and (4) buying power were available on zip code level from Statistics Sweden. (5) Neighbourhood rates of crime were available on a municipality level from the National Council of Crime Prevention. Sweden contains 289 municipalities. Each family in our sample was matched to the variables for its neighbourhood via its zip or municipality codes. There were 1,067 families in the sample, distributed throughout 999 neighbourhoods in the 289 municipalities.

(1) Neighbourhood ethnic diversity was assessed as the proportion of people in each neighbourhood born in countries outside Europe, North America and Australia/New Zealand. (2) Neighbourhood basic educational level was assessed as the proportion of people who had completed at least elementary school and/or secondary school. (3) Neighbourhood unemployment level was assessed as the proportion of unemployed people. (4) Neighbourhood buying power was defined as a family's net income after taxes have been paid and social benefits added. The measure also depends on how many individuals a household contains. For the analyses, we calculated the proportion of families with low buying power. (5) Neighbourhood rates of crime were measured as the total number of crimes reported to the police during 2001. The crime-rate was standardized to the average number of people living in each municipality, i.e., number of crimes reported to the police divided by 100,000 inhabitants.

4.4 REPRESENTATIVENESS OF THE SAMPLE

4.4.1 Attrition

As selective attrition may bias estimates in longitudinal analyses (Heath et al., 1998), we carried out some analyses in order to test whether subjects lost to follow up at later waves differed from responders on measures at previous waves.

No significant differences in sex ratio (OR (odds-ratios) = 0.96; 95 % CI: 0.74 - 1.22) were found between responders to wave 2 compared to non-responders at wave 2. Similarly, there were no differences between the groups in parental ratings of aggressive behavior (OR = 1.02; 95 % CI: 0.68-1.58), or nonaggressive behavior (OR = 1.78; 95 % CI: 0.94 - 3.32) measured at wave 1. However, the result indicated that families with lower socioeconomic status were somewhat more likely to cease to participate (OR = 1.30; 95 % CI: 1.09 - 1.40).

There were no significant differences in sex ratio (OR = 0.69; 95 % CI: 0.48-1.00) between responders at wave 3 compared to non-responders at wave 3. Similarly, there were no differences between the groups in antisocial behavior (OR = 1.22; 95 % CI: 0.77 - 1.93), or family socioeconomic status (OR = 0.42; 95 % CI: 0.11 - 1.58).

At wave 4 however, comparisons of responders and non-responders revealed significant differences in sex ratio (OR = 0.39; 95 % CI: 0.32 - 0.48; more boys than girls dropped out between wave 3 and wave 4), and for antisocial behavior (OR = 1.46; 95 % CI: 1.15 - 1.86), indicating that individuals scoring higher on the antisocial behavior scale at wave 3 were more likely to drop out at wave 4.

4.4.2 Telephone follow-up

At wave 3 when the twins were 16-17 years old, a telephone follow-up to non-responders was conducted with a reduced battery of questions. Twins who responded to the telephone follow-up did not score higher on antisocial behavior compared to twins who responded to the questionnaire ($t_{217} = .62$, $p = .54$) (Tuvblad et al., 2006). However, twins who responded to the telephone interview scored significantly higher on the total score of the selected Youth Psychopathic traits Inventory (YPI) items compared to twins who responded to the questionnaire ($t_{2310} = -5.92$, $p < .001$). In addition, there were also significant differences between responders and non-

responders with regard to socioeconomic status ($\chi^2 = 27.63$, $p < .001$). Thus, twins who responded to the questionnaire more often came from higher socioeconomic status families than the twins who responded to the telephone interview (Larsson et al., 2006).

4.5 STATISTICAL METHODS

The main focus in this thesis has been to increase the understanding of the development of antisocial behavior through behavioral genetic methods. Below is a general introduction to the twin design.

4.5.1 The classical twin design

The classical twin design, which compares the similarity of monozygotic (MZ) and dizygotic (DZ) twins is one of the most powerful methods for estimating the relative contribution of genetic and environmental effects to human traits (Evans et al., 2002).

MZ twins reared together share part of their environment and 100 % of their genes. Resemblance between them is therefore due to genetic and shared environmental effects. The non-shared environmental factors are the extent to which MZ twins do not resemble each other. Resemblance between DZ twins reared together is due to shared environment and to shared genes: DZ twins share on average 50 % of their segregating genes. Consequently, resemblance between them due to genetic effects will be lower for DZ pairs than for MZ pairs. The extent to which DZ twins do not resemble each other is due to non-shared environmental factors and to non-shared genetic effects.

4.5.1.1 Genetic and environmental effects

Genetic effects for a single locus can be defined in terms of an additive genetic value (i.e., alleles at a single locus add up to affect a trait) and a dominant deviation (the deviation from purely additive effects). These effects can be summed across loci. There may also be an interaction between two or more different loci (epistasis). So, the total contribution of genetic influences (G) to a trait is the sum of the additive genetic (A) and dominant effects (D) of alleles at multiple loci, plus variance due to the interaction of alleles at different loci (I):

$$G = A + D + I$$

If all contributing alleles act additively, the correlation of genetic effects in DZ twins will be on average .5. However, if some alleles act in a dominant way, the correlation due to genetic dominance will be .25. The presence of dominant genetic effects decreases the expected phenotypic resemblance in DZ twins relative to MZ twins. Epistasis decreases this similarity even further, the extent depending on number of loci involved and their relative effect on the phenotype (Mather & Jinks, 1982).

Using data from MZ and DZ twins reared together makes it possible to partition the total phenotypic variance ($V_{(p)}$) into components due to additive genetic variance (A), either dominant genetic (D) or shared environmental variance (C), and non-shared environmental variance (E):

$$V_{(p)} = V_{(A)} + V_{(D/C)} + V_{(E)}$$

Dominant genetic and shared environmental influences are negatively confounded, and cannot be estimated simultaneously in a study of MZ and DZ twins reared together.

4.5.1.2 Heritability

Heritability is the proportion of total phenotypic variation in a given characteristic that can be attributed to genetic effects. There are two types of heritability. The broad-sense heritability involves all additive and non-additive genetic variance, whereas the narrow-sense heritability involves only additive genetic variance. It should be stressed that heritability is a population and time specific parameter (Plomin et al., 2001).

4.5.1.3 Twin correlations

A measure of similarity between twins is the intraclass correlations (Neale & Cardon, 1992). Intraclass correlation refers to correlations in defined sub-groups. When calculating intraclass correlations, data is usually double-entered in order to avoid any bias due to birth order (Griffin & Gonzales, 1995).

Twice the difference between the MZ and DZ correlations provides a first estimate of the relative contribution of additive genetic influences (A) to the phenotypic variation in a trait ($A = 2[r_{MZ} - r_{DZ}]$). The contribution of the dominant genetic effect (D) is obtained by subtracting four times the DZ correlation from twice the MZ correlation (D

= $2r_{MZ}$ - 4 r_{DZ}). The proportion of the variance that is due to shared environmental influence (C) is given by subtracting the MZ correlation from twice the DZ correlation (C = 2 r_{DZ} - r_{MZ}). Finally, the contribution of the non-shared environmental influences (E) can be obtained by subtracting the MZ correlation from unit correlation (E = 1- r_{MZ}) (Posthuma et al., 2003).

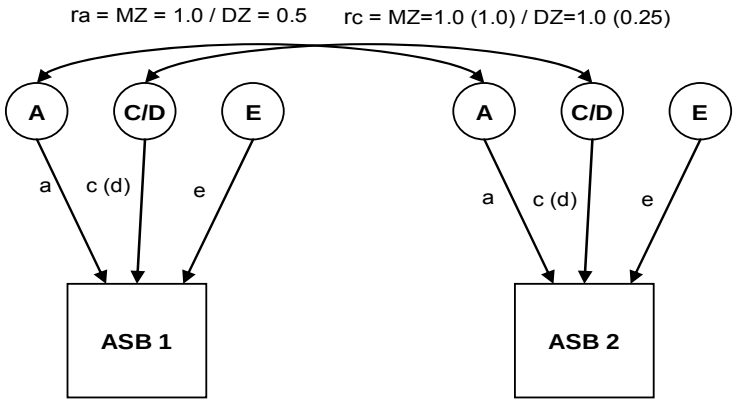
However, this approach is descriptive and does not provide information on how well the estimates describe the observed data, nor does it allow actual testing of different hypothesis.

4.5.1.4 Structural equation modeling

Structural equation modelling is a model-fitting approach used in genetic analyses of twin data. The method estimates parameters (regression coefficients/factor loadings) between latent (unobserved) and observed variables.

Structural equation models are usually presented in path diagrams. In the basic univariate twin model, Figure 2, the latent factors, A, C/D, and E are depicted in circles. The additive genetic correlation (r_a) is set to 1.0 for MZ twins as they are genetically identical, and .5 for DZ as they on average share 50 % of their segregating genes. Since DZ twins share only one fourth of the dominant genetic effect shared by MZ twins, the correlations are 1.0 and .25 for dominance for MZ and DZ twins, respectively. Shared environmental factors refer to non-genetic influences that contribute to similarity within pairs of twins, that is, experiences that twins have in common such as shared familial influences, living in the same neighbourhood. Shared environmental influences are assumed by the model to contribute equally to similarity in MZ and DZ twin pairs, i.e., the equal environment assumption. The shared environmental correlation (r_c) is therefore set to 1 for both groups. Non-shared environmental factors are those experiences that make siblings dissimilar (e.g., head injury). There is no correlation for the unique environment by definition, and this parameter also includes measurement error. The influence of A, C/D, and E on the observed variation for a trait (represented in squares) is given by parameters a, c/d, and e. Squaring the factor loadings i.e., a, c/d, and e, results in the variance explained by each component. Variance explained is usually reported in a standardized form, by dividing the estimated variance for one parameter by the total phenotypic variance.

Figure 2



Univariate model. A: additive genetic; C: shared environment; D: dominance; E: non-shared environment. ASB: antisocial behavior of twin 1 and twin 2. a, c/d, e: factor loadings.

All modelling in this thesis were run in the structural equation modeling software Mx (Neale, 1997), which is specifically tailored for use in behavioral genetic analyses. The models were fitted using a maximum likelihood estimation procedure for raw data. This estimation technique can handle data from samples in which part of the sample might be missing. This raw maximum likelihood approach yields a goodness of fit index called log-likelihood. Twice the difference between the log-likelihood of a full model minus the log-likelihood of a submodel, in which parameters are fixed to zero or constrained to be equal, is χ^2 distributed with the difference in the number of estimated parameters as the degrees of freedom. A significant difference indicates that the model with fewer estimated parameters fits the data worse. The suitability of the models can also determined by comparing the model's Akaike Information Criterion (AIC = χ^2 – 2*df). The AIC represents the balance between model fit and the number of parameters (parsimony), with lower values (i.e., larger negative) of AIC indicating the most suitable model (Neale & Cardon, 1992).

Behavioral genetic modeling makes certain assumptions about the nature of the processes being estimated. The models assume for example that there is random mating operating in the parent generation and equivalent environment for MZ and DZ twins

(Martin et al., 1997, Plomin et al., 2001). In this thesis, a more detailed discussion of these and other assumptions is given in the Discussion section.

4.5.2 Sex-limitation models

In the basic univariate twin model described above, genetic and environmental variance components for a trait can be estimated using data from same-sexed MZ and DZ twins. Apart from estimating genetic and environmental effects on a trait, it may also be interesting to investigate whether sex specific influences are important for a trait. To assess whether the magnitude of genetic and environmental effects differ between males and females (i.e., quantitative differences), only data from like-sexed twin pairs are required. However, to determine whether or not it is the same set of genes or shared environmental experiences that influence a trait in males and females, i.e., qualitatively differences, data from opposite-sexed twin pairs are also needed.

In this thesis, the first aim was to examine the role of sex differences in the genetic and environmental etiology of antisocial behavior. To test for sex differences in antisocial behavior, a series of sex-limitation models were fitted (Study I to IV).

The first model, a *constrained* model**,** assumes no sex-specific effects and constrains genetic and environmental components to be equal in males and females. In the next model, a *scalar sex-limitation* model**,** a difference in the total variance between boys and girls is allowed, but relative contributions of genetic and environmental influences are the same for males and females. In the third model, *a heterogeneity* model**,** the magnitudes of genetic and environmental effects are allowed to differ for males and females, but to be the same for males in same-sexed twin pairs as males in opposite-sexed twin pairs, and similarly for females.

To determine whether or not it is the same set of genes or shared environmental experiences that influences a trait in males and females (i.e., qualitatively differences), again a *heterogeneity* model can be used. If different genetic influences are important for antisocial behavior in males and females, then the opposite-sexed twins will be less genetically similar for the trait than DZ twins, and the genetic correlation will be less than .5. This can be tested by allowing for the genetic correlation for opposite-sexed twin pairs to be estimated in the model, rather than being fixed to .5. Alternatively, if

different environments are more influential in one sex than the other, then one can test whether the correlation between environments shared by twins is less than 1 in opposite-sexed twin pairs. It should be noted that there is barely enough information in the twin design to disentangle genetic and environmental causes of qualitative differences between the sexes (Neale & Cardon, 1992, Neale & Martin, 1989).

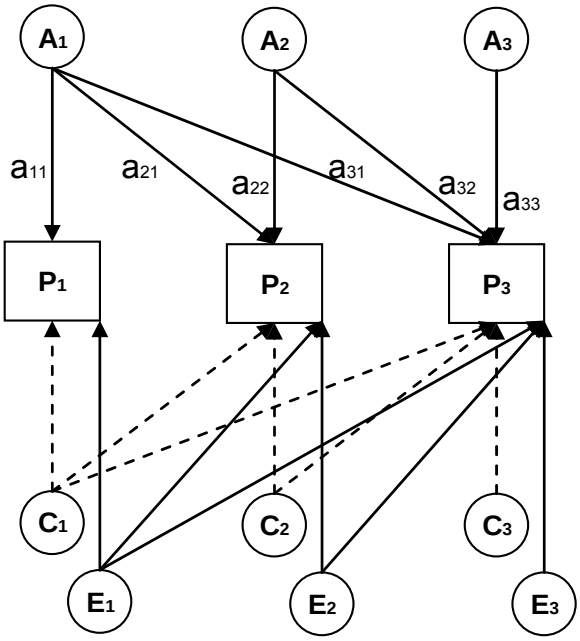
4.5.3 Multivariate models

To investigate the second and third aim of this thesis, that is, to investigate how genetic and environmental factors influence the development of antisocial behavior (study I and II), and to explore the covariation between antisocial behavior and psychopathic personality traits (study III), three different multivariate models were fitted: Cholesky decomposition (Study I, III), independent pathway (Study II, III) and common pathway (Study II, III) (Neale & Cardon, 1992, Plomin et al., 2001).

4.5.3.1 Cholesky decomposition

In a Cholesky decomposition of three measured variables (Figure 3), additive genetic effects (A), shared environmental effects (C), and non-shared environmental effects (E) are portioned into three sets of factors. A1, C1 and E1 influence all three variables, A2, C2 and E2 influence the second and third variables, and A3, C3 and E3 influence the third variable only. This model provides the fullest potential explanation of data.

Figure 3

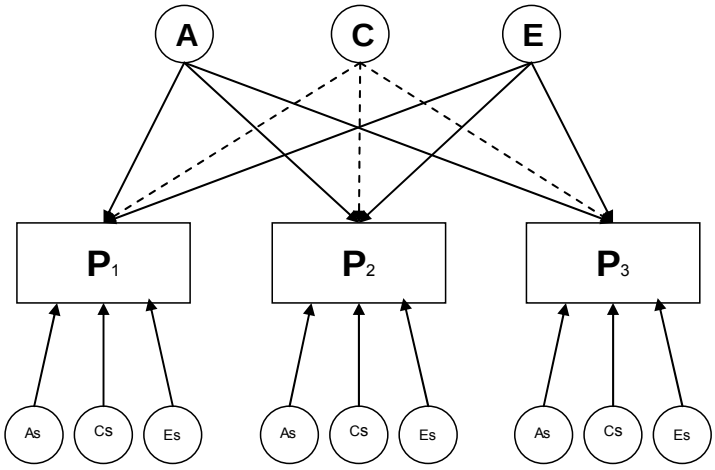


Cholesky decomposition, showing one twin in a pair.

4.5.3.2 Independent pathway model

The independent pathway model (Figure 4) with more than three variables is a submodel of the Cholesky decomposition. In this model, genetic and environmental effects are of two types: general and specific. The model specifies general genetic (A), shared environmental (C), and non-shared environmental (E) effects that load on each measured variable, as well as measurement specific genetic and environmental effects, As, Cs, and Es, where the Es terms include measurement error. It is also possible to combine elements from the Cholesky model and the independent pathway model.

Figure 4

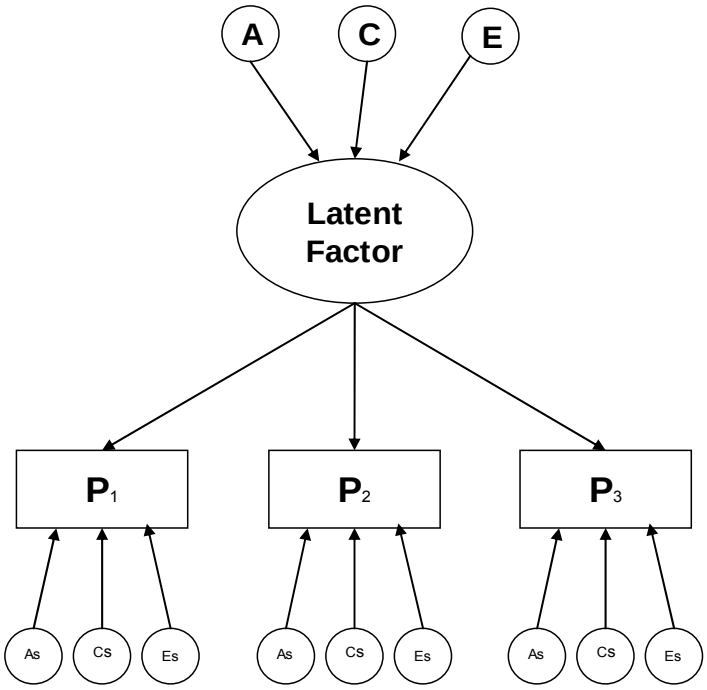


Independent pathway model, showing one twin in a pair.

4.5.3.3 Common pathway model

In the common pathway model (Figure 5), rather than loading directly on the measured variables, the general effects are mediated through a latent underlying factor that represents the variance shared among the measures. The model estimates fewer parameters than the independent pathway model and is therefore more parsimonious. In addition to the genetic and environmental effects of the latent underlying factor; parameters specific to each measure are also estimated (As, Cs, and Es).

Figure 5



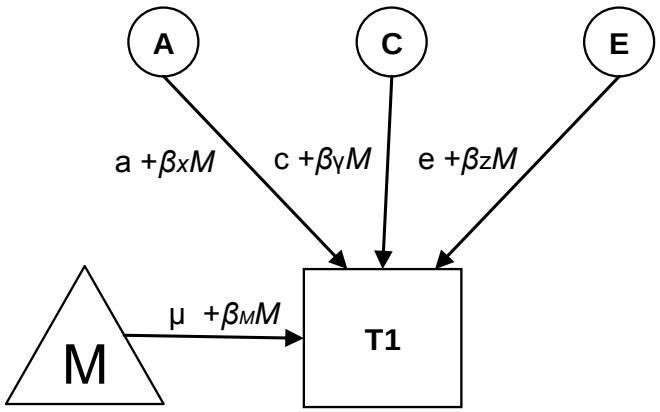
Common pathway model, showing one twin in a pair.

4.5.4 Models testing for gene-environment interaction

Gene-environment interaction (GxE) refers to a genetic susceptibility to environmental risk (Plomin et al., 2001, Boomsma et al., 2002). The fourth aim of this thesis was to investigate whether socioeconomic status moderated the effect of genetic and environmental influences on antisocial behavior (Study IV). To test for gene-environment interaction, two different approaches were used.

In the first approach, a model designed to test GxE of the latent genetic variable with a measured continuous moderator variable was used (Purcell, 2002; see also Wichers et al., 2002, Button et al., 2005). In this model (Figure 6), the phenotypic variance is partitioned into the usual genetic (A), shared environmental (C), and non-shared environmental (E) components, but also interaction effects (parameter: βx, βy and βz) with a continuous measured environmental variable (M = in this case socioeconomic status), as well as a main effect (βm) of socioeconomic status on antisocial behavior. If βx is significant this indicates a gene-environment interaction (analogous for βy and βz).

Figure 6



Path diagram for GxE interaction model: a: additive genetic c: shared environmental, e: non-shared environmental components; *βx*, *β*y and *βz* moderated components of a, c, and e, respectively. M = moderator, *Bm* = main effect of moderator, μ = grand mean.

In the second approach testing for GxE, a binary moderator variable was used. Specifically, the sample was first divided into two groups: less advantaged neighbourhoods (exposed) and more advantaged neighbourhoods (unexposed). Next, genetic, shared environmental and non-shared environmental effects were estimated for the exposed and the unexposed group separately. To test for GxE, parameters were then equated across the two groups, i.e., testing for heterogeneity (Neale & Cardon, 1992, Boomsma et al., 1999, Koopmans et al., 1999, Rose et al., 2001).

5 RESULTS

5.1 SEX DIFFERENCES IN THE GENETIC AND ENVIRONMENTAL ETIOLOGY OF ANTISOCIAL BEHAVIOR

The first aim of this thesis was to investigate the role of sex differences in the genetic and environmental etiology of antisocial behavior. A series of sex-limitation models were fitted to the data. The heterogeneity model, indicating that the same genetic and environmental effects are important in males and females but that their relative magnitude may differ, was found to best describe the data at three measurement occasions (ages 13-14, 16-17, and 19-20). Table 4 presents parameter estimates from the best-fitting model from each of these three time points (study I, II, and IV). At all three time points, heritability was higher in girls, whereas shared environment was more important in boys.

Table 4 Parameter estimates from the best-fitting cross-sectional models with antisocial behavior at ages 13-14, 16-17, and 19-20

	Parameter estimate (95 % CI)					
	Boys			Girls		
	A	C	E	A	C	E
13-14 years	.27 (.04-.52)	.42 (.19-.62)	.31 (.25-.38)	.40 (.14-.56)	.28 (.14-.50)	.32 (.26-.39)
16-17 years	.06 (.00-.33)	.52 (.27-.63)	.42 (.34-.50)	.59 (.38-.79)	.17 (.00-.36)	.24 (.20-.30)
19-20 years	.15 (.00-.52)	.30 (.00-.53)	.55 (.43-.70)	.63 (.41-.70)	.00 (.00-.19)	.37 (.30-.46)

A: additive genetic; C: shared environment; E: non-shared environment

5.2 THE INFLUENCE OF GENES AND ENVIRONMENTS ON THE DEVELOPMENT OF ANTISOCIAL BEHAVIOR

The second aim of this thesis has been to investigate how genetic and environmental factors influence the development of antisocial behavior (study I and II).

In study I, we wanted to examine the influence of genetic and environmental effects on the development of aggressive and nonaggressive behavior in childhood to antisocial behavior in adolescence. In order to do this, a series of bivariate Cholesky decomposition models were fitted (Table 5). For girls we found that the relationship

between aggressive behavior in childhood and early adolescent antisocial behavior was explained by genetic influences ($\Delta\chi^2 = 18.74$; $p < .05$). The correlation between nonaggressive behavior and antisocial behavior was also due to continuity of genetic influences ($\Delta\chi^2 = 37.75$; $p < .05$). For boys, there was no significant mediation between aggressive behavior and antisocial behavior, but there were significant shared environmental effects on the relationship between nonaggressive behavior and antisocial behavior ($\Delta\chi^2 = 4.96$; $p < .05$).

Table 5 Cholesky decomposition models for childhood aggressive and nonaggressive behavior and early adolescent antisocial behavior, by sex

Parameter dropped	Girls		Boys	
	$\Delta\chi^2$	P	$\Delta\chi^2$	P
Test of associations between aggressive behavior and antisocial behavior				
Drop a_{21}	18.74	.00	.20	.65
Drop c_{21}	.00	-	1.03	.31
Drop e_{21}	.00	-	.21	.65
Test of association between nonaggressive behavior and antisocial behavior				
Drop a_{21}	37.75	.00	1.40	.24
Drop c_{21}	.00	-	4.96	.03
Drop e_{21}	.00	-	.58	.44

$a_{21;}$ $c_{21;}$ e_{21}: genetic, shared environmental and non-shared environmental factor loadings between childhood aggressive/nonaggressive behavior and early adolescent antisocial behavior.
$\Delta\chi^2$ = difference in log-likelihoods between the full model and nested models.
All Δdf was 1 (i.e., constrained parameters between the full model and the nested model)

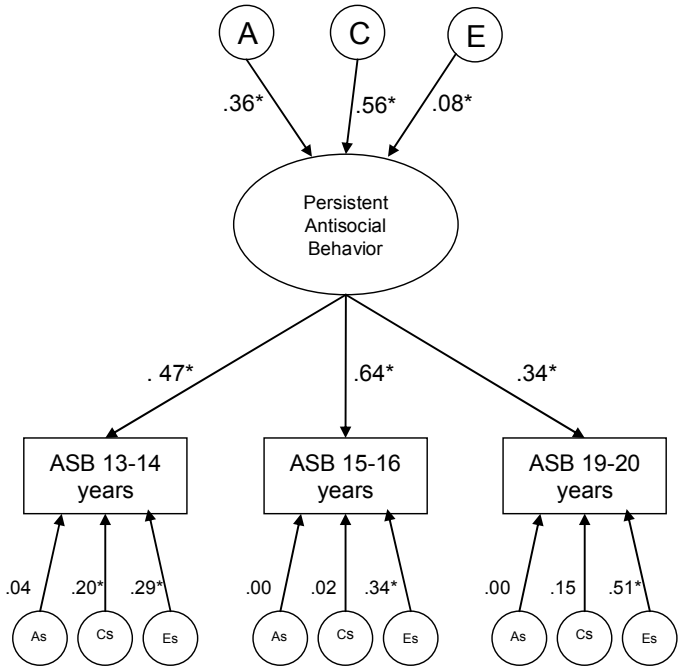
In study II, the aim was to investigate whether a latent persistent factor explains the covariance in antisocial behavior measured at the three time points (13-14 years, 16-17 years, and 19-20 years).

Figure 7a and 7b display squared standardised parameter estimates from the common pathway model for boys and girls, respectively. For boys, variation in the latent factor labelled *persistent antisocial behavior* was influenced for 36 % by a genetic factor, for 56 % by a shared environmental factor, and for 8 % by a non-shared environmental factor. The age-specific genetic and shared environmental effects were non-significant, apart from the shared environment at age 13-14 (20 %). The significant age-specific

non-shared environmental effects, including measurement error, were ranging from 29 to 51 %.

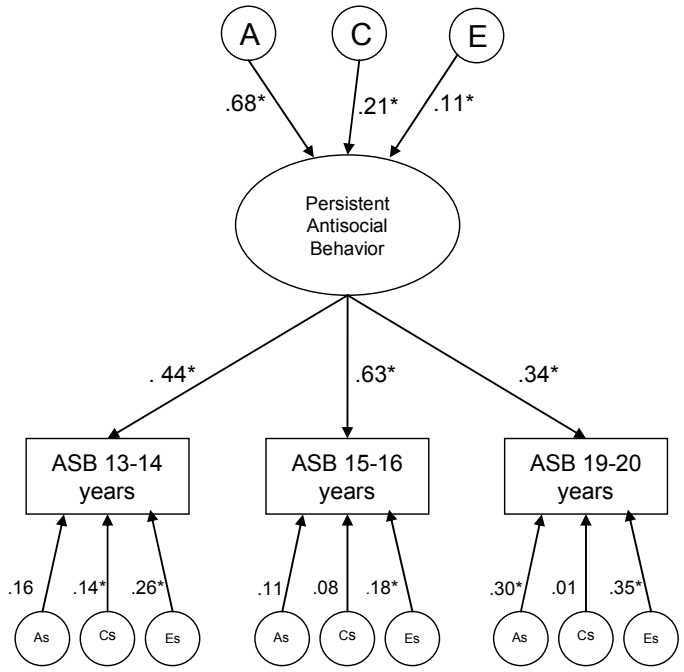
For girls, variation in persistent antisocial behavior was for 68 % due to a genetic factor, for 21 % due to a shared environmental factor, and for 11 % due to a non-shared environmental factor. The age-specific genetic and shared environmental effects were small and non-significant, apart from the shared environment at age 13-14 (14 %), and the genetic effect at age 19-20 (30 %). The significant non-shared environmental effects, ranged from 18 to 35 %.

Figure 7a Boys



Squared standardised parameter estimates from a common pathway model for antisocial behavior in boys. Latent (unobserved) factors are depicted in circle: the A refer to additive genetic factors; the C to shared environmental factors, and the E to non-shared environmental factors. Oval denotes a latent underlying factor (i.e., persistent antisocial behavior). Observed variables are in rectangulars, in this case: antisocial behavior (ASB) at ages 13-14 years, 16-17 years, and 19-20 years. As (additive genetic) is residual variance specific to each time point, likewise for Cs (shared environment), and Es (non-shared environment). Significant estimates are marked with an asterisk.

Figure 7b Girls



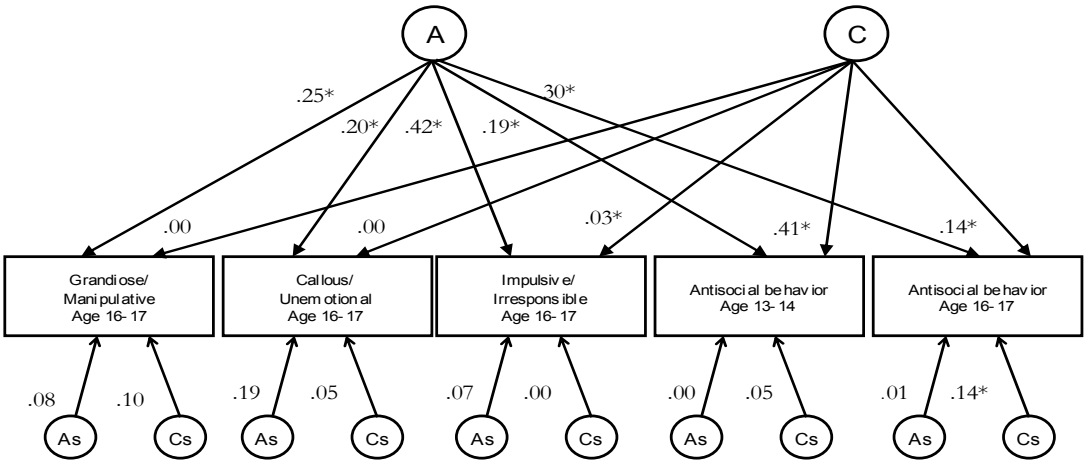
Squared standardised parameter estimates from a common pathway model for antisocial behavior in girls. Significant estimates are marked with an asterisk. Please see above for further explanation.

5.3 COVARIATION

A third aim has been to explore the covariation between antisocial behavior and psychopathic personality traits (study III). In this study an independent pathway model, where the non-shared environmental factors (E) were Cholesky decomposed, was found to best describe the data.

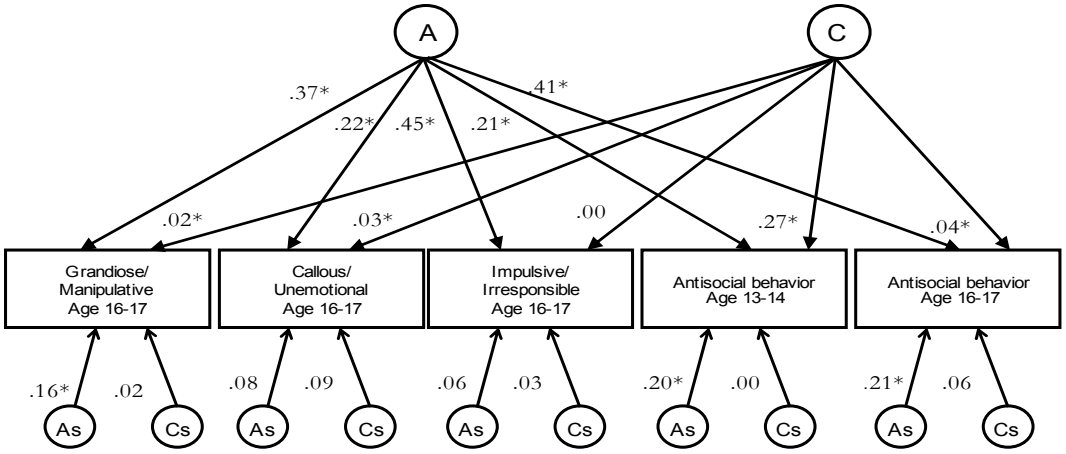
Figures 8a and 8b provide estimates from the best-fitting model for boys and girls, respectively. The common genetic factor (A) explained 20 to 45 % of the variance in the three psychopathic personality dimensions and 19 to 41 % of the variance in antisocial behavior at ages 13-14 and 16-17. The common shared environmental factor (C) loaded significantly on antisocial behavior measures; explaining 14 to 41 % and 4 to 27 % of the variance for boys and girls, respectively. However, the common shared environmental factor only contributed negligible to the three psychopathic personality dimensions. Measurement-specific effects were generally only modest.

Figure 8a Boys



Standardized squared path estimates from the independent pathway model for *psychopathic personality traits* and *antisocial behavior* in boys at ages 13-14 and 16-17. The latent variables A (additive genetic factor) and C (shared environmental factor) are depicted in circles. Measured variables are depicted in rectangles (i.e., grandiose/manipulative age 16-17 to antisocial behavior age 16-17). As (additive genetic) is residual variance specific to each measure and likewise for Cs (shared environment). The non-shared environmental factors were Cholesky decomposed and are not depicted in the figure. Significant estimates are marked with an asterisk.

Figure 8b Girls



Standardized squared path estimates from the independent pathway model for *psychopathic personality traits* and *antisocial behavior* in girls at ages 13-14 and 16-17. Significant estimates are marked with an asterisk. Please see above for further explanation.

5.4 GENE-ENVIRONMENT INTERACTION

The last aim of this thesis was to investigate whether socioeconomic status moderated the effect of genetic and environmental influences on antisocial behavior. To test for gene-environment interaction two different approaches were used (study IV).

In the first approach, a model allowing for the inclusion of a continuous measured environmental variable was used (Purcell, 2002). In case of significant interaction effects, the variance components were plotted as a function of the moderator variable.

Significant interaction effects were found for *occupational status* for boys. Diagram 1a illustrates that the influence of genetic effects on antisocial behavior was higher in families with high occupational status, while the shared environmental effects were lower. Conversely, in families with low occupational status, genetic effects were lower and the shared environmental effects were higher.

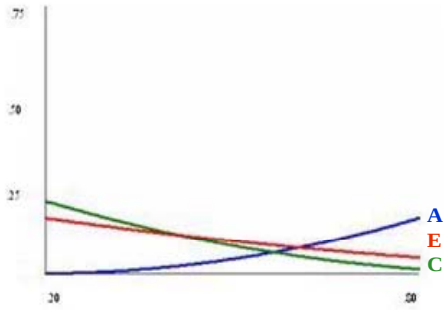
Significant interaction effects were also found for *neighbourhood ethnic diversity* for girls. Plotting the variance component as a function of the moderator variable *neighbourhood ethnic diversity* for girls indicated that the influence of genetic effects on antisocial behavior was higher and the influence of shared environmental effects was lower in areas with low levels of ethnic diversity as compared to areas with high levels of ethnic diversity (Diagram 1b).

We also plotted *neighbourhood ethnic diversity* for boys, as the p-value for the shared environmental interaction (βy) was .08, and a similar pattern emerged for boys as for girls (Diagram 1c).

Finally, we plotted *neighbourhood rate of crime* for girls, as the p-value for the genetic interaction effect (βx) was .09 and .03 for the shared environment interaction (βy). The influence of genetic effects on antisocial behavior was higher in low crime areas, while the shared environment was lower, compared to high crime areas (Diagram 1d).

Diagram 1: Socioeconomic status plotted as a function of variance components

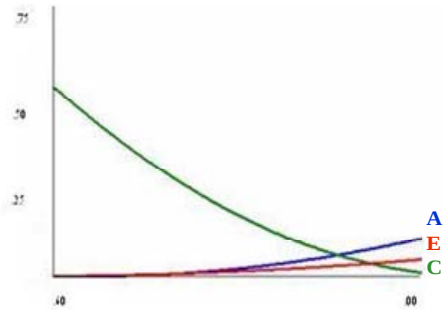
Diagram 1a: Boys
Unstandardised variance components



Low family occupational status

High family occupational status

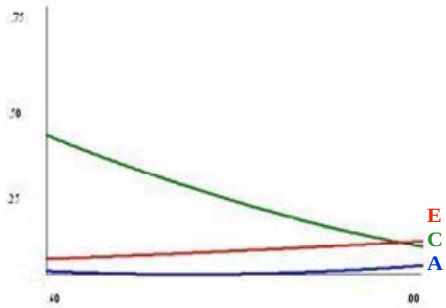
Diagram 1b: Girls
Unstandardised variance components



Areas with high ethnic diversity

Areas with low ethnic diversity

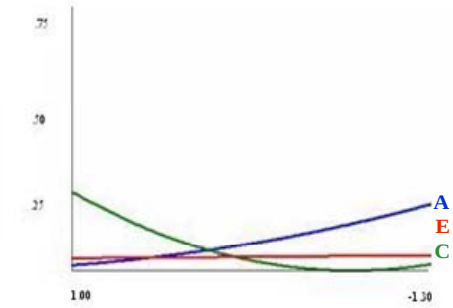
Diagram 1c: Boys
Unstandardised variance components



Areas with high ethnic diversity

Areas with low ethnic diversity

Diagram 1d: Girls
Unstandardised variance components



Areas with high crime rate

Areas with low crime rate

A = addictive genetic component **C** = shared environmental component **E** = non-shared environmental component

In the second approach to test for gene-environment interaction, we investigated whether the estimates for genetic and environmental effects varied between more advantaged and less advantaged neighbourhoods (Table 6). For boys, the full model that estimates different genetic and environmental effects in the two neighbourhood groups fitted the observed data significantly better than the model that constrained estimates to be equal ($\Delta\chi^2 = 12.58$; $\Delta df = 3$; $p = .00$). Consistent with the analyses where socioeconomic status measures were used continuously, heritability was higher

in the more advantaged neighbourhood group (A = 37 %) compared to the less advantaged neighbourhood group (A = 1 %). Conversely, the shared environment was higher in the less advantaged neighbourhood group (C = 69 %) compared to the more advantaged neighbourhood group (C = 13 %). For girls, genetic effects were also higher in more advantaged neighbourhoods (A = 69 %) compared to less advantaged neighbourhoods (A = 61 %), and the shared environment was higher in less advantaged neighbourhoods (C = 16 %) compared to more advantaged neighbourhoods (C = 6 %), but the estimates could be constrained across the two neighbourhood groups ($\Delta\chi^2 = .68$; $\Delta df = 3$; $p = .88$). In both more advantaged and less advantaged neighbourhoods, shared environmental effects were more important for boys than for girls, whereas genetic effects were more pronounced in girls.

Table 6 Genetic and environmental effects for antisocial behavior at age 16-17 in more advantaged and less advantaged neighbourhoods

Type of neighbourhood	Parameter estimate (95 % CI)					
	Boys			Girls		
	A	C	E	A	C	E
More advantaged	.37 (.01-.61)	.13 (.00-.47)	.50 (.39-.63)	.69 (.39-.81)	.06 (.00-.34)	.25 (.18-.33)
Less advantaged	.01 (.00-.19)	.69 (.51-.77)	.30 (.23-.39)	.61 (.39-.78)	.16 (.01-.37)	.23 (.18-.31)

A: additive genetic; C: shared environment; and E: non-shared environment

6 DISCUSSION

The overall aim of this thesis was to investigate the influence of genetic and environmental factors on antisocial behavior from childhood to emerging adulthood. The main findings are discussed below, followed by a section concerning methodological considerations, and last a section including future directions.

6.1 THE NATURE OF ANTISOCIAL BEHAVIOR

6.1.1 The influence of genetic and environmental effects on antisocial behavior

6.1.1.1 Genetic influences on antisocial behavior

There is consensus that genetic factors are important for individual differences in antisocial behavior (DiLalla, 2002, Mason & Frick, 1994, Miles & Carey, 1997, Rhee & Waldman, 2002, Moffitt, 2005a, McGuffin & Thapar, 1992). The results in this thesis are in agreement with this. Genetic effects were found to be important for the development of antisocial behavior in both sexes across the three time points (13-14 years, 16-17 years, and 19-20 years).

How should a genetic effect on antisocial behavior be interpreted? It is unlikely that genes operate on antisocial behavior directly. Rather, genetic influences on multifactorial determined behaviors are thought to operate in a probabilistic fashion. That is, any genetic effect on a behavior increases the likelihood that the behavior will occur, but whether or not it actually happens depends on a range of other factors. Furthermore, genetic effects on antisocial behavior are likely to act indirectly through biological factors such as hormone levels (Popma & Raine, 2006), through temperamental and personality features, such as impulsivity (Lahey et al., 2003), or through cognitive features such as low IQ (Moffitt, 1993b).

It should also be stressed that evidence of genetic influences on antisocial behavior does not implicate that individuals exhibiting antisocial behavior are immune or resistant to interventions. The importance of genetic influences implies that biological processes are involved in the etiology of antisocial behavior. However, it does not

imply that a genetic influence on antisocial behavior requires a biological intervention, but instead it may require an environmental intervention. For example, a genetic liability to antisocial behavior may be best prevented or treated through for example parental and/or teacher training.

6.1.1.2 Shared environmental influences on antisocial behavior

In keeping with previous meta-analyses on antisocial behavior (Miles & Carey, 1997, Rhee & Waldman, 2002), shared environmental effects were found to be important influences on antisocial behavior. Shared environmental effects have been found to be of little importance for personality and psychopathology (Rowe, 1994, Bouchard & Loehlin, 2001, Jang, 2005), the exception is however, antisocial behavior (Kendler et al., 2003, Plomin et al., 1990). Shared environmental effects refer to non-genetic influences that contribute to similarity within pairs of twins. Shared environmental influences on antisocial behavior include for example family factors (e.g., harsh parenting) (Farrington & Loeber, 2000); or contextual factors (e.g., neighbourhood poverty) (Sampson et al., 1997, Brooks-Gunn et al., 1993).

6.1.1.3 Non-shared environmental influences on antisocial behavior

Our results showed that non-shared environmental effects were important influences on antisocial behavior. Non-shared environmental effects may include antisocial experiences unique to the individual and not shared by his or her co-twin, for example belonging to a delinquent peer group. The influence of peers has also been demonstrated in numerous studies (Loeber et al., 1998).

6.1.2 The role of sex differences in the genetic and environmental etiology of antisocial behavior

Our results showed that heritability for antisocial behavior was higher in girls, whereas shared environment was more important in boys at all three measurement occasions (study I to IV). Our finding is in agreement with studies that have shown lower heritability, and stronger shared environmental component on childhood and adolescent antisocial behavior in boys (Jacobson et al., 2002, Lyons et al., 1995, Vierikko et al., 2003), and higher heritability and modest shared environmental influences in girls (Jacobson et al., 2002, Eley et al., 1999, Rose et al., 2004). The

results in this thesis strongly suggest that sex is an important moderator of the magnitude of genetic and environmental factors on antisocial behavior.

6.1.2.1 Stronger genetic effects in girls

A finding in this thesis was that heritability was higher in girls than in boys. An explanation to this have been put forward by Jacobson and colleagues (2002), who have suggested that if genes activated at puberty are an important influence on antisocial behavior, genetic influences on early adolescent antisocial behavior might be stronger for females than for males, given the earlier age of onset for puberty in females (Jacobson et al., 2002). We have previously investigated early pubertal development as a genetic risk factor for antisocial behavior using the TCHAD-study. Pubertal development was measured through a self-report questionnaire (Petersen et al., 1983), when the twins were 13-14 years old. We found a high stability in antisocial behavior from age 13-14 to 16-17, and that this stability was partly due to genetic factors. Of this genetic stability, puberty accounted for 11 % in boys and 8 % in girls (Tuvblad et al., 2005). These results suggest that early pubertal development may not explain the differences in heritablity between boys and girls, but instead explain genetic mediation.

Another possible interpretation of our results of strong genetic influences on antisocial behavior in girls, relates to the hypothesis of a delayed-onset pathway in girls proposed by Silverthorn and Frick (1999). This hypothesis is based on the following: much of the research on antisocial behavior includes boys only. For example, the Dunedin sample includes both boys and girls, but many reports from the data set focused exclusively on the boys because so few girls show the early-starters, severe, persistent pathway (Moffitt & Caspi, 2001; see also Fergusson & Horwood, 2002). Further, girls that experience a delayed-onset of antisocial behavior during adolescence, also experience many of the same problems that are associated with childhood-onset antisocial behavior in boys; namely, high rates of family dysfunction and family psychopathology, poor adult outcomes, and higher rates of cognitive and neuropsychological dysfunction (Marmorstein & Iacono, 2005, Silverthorn & Frick, 1999). Accordingly, Silverthorn and Frick (1999) have suggested that males and females follow different antisocial behavior trajectories, and that the childhood-onset pathway in boys equals a delayed-onset pathway in girls. Following their reasoning, one would expect a high genetic influence on the delayed-onset pathway in girls, given that earlier research has shown

that the childhood-onset, persistent pathway is highly heritable in boys (Taylor et al., 2000a, Jacobson et al., 2001, Slutske et al., 1997c). Although youths with childhood and adolescent onset antisocial behavior were not distinguished, our results for girls indicated that the genetic influences are the most important effect on antisocial behavior, which may be interpreted in support of the delayed-onset pathway. Thus, these results may have implications for developmental models of antisocial behavior.

6.1.2.2 Stronger shared environmental effects in boys

Shared environmental influences were found to be higher in boys than in girls. This finding is in line with the hypothesis that although boys and girls are equally exposed to psychosocial factors or common environmental risk factors (e.g. neighbourhood poverty, family discord, harsh parenting), boys may be more susceptible to certain risk factors (Rutter et al., 1998, Leventhal & Brooks-Gunn, 2000). Consistent with this, Moffitt and colleagues (2001) showed, using the Dunedin study, that although the same family risk factors (e.g., deviant mother-child interaction, inconsistent and harsh discipline, family conflict, low family socioeconomic status) predicted antisocial behavior in both boys and girls, most of the family risks factors had stronger effect on boys' than on girls' antisocial behavior. In other words, boys who experienced family risk factors were at greater risk of developing antisocial behavior than were girls who experienced the same family risk factors. It is however, not clear whether boys react differently than the girls to family conditions, or if boys are treated differently during times of family stress.

Apart from differential susceptibility to risk factors, there may also be differences in exposure to number or severity of risk factors between boys and girls (Rutter et al., 2003). For example, boys tend to receive harsher discipline than do girls (Moffitt et al., 2001). Further, girls are generally more actively discouraged from behaving against societal norms, and this could have a protective effect on girls (Rutter et al., 1998). In contrast, boys' peer groups tend to be bigger and more oriented around activities. They tend to play more outside the home, to be exposed to more peer-group influences and more separate from the world of adults and social control (Kroneman et al., 2004, van Lier et al., 2005).

6.1.2.3 Sex differences

In keeping with previous research, boys were found to have higher mean antisocial behavior values than girls (study II) (Moffitt et al., 2001, Rutter et al., 2003, Ehringer et al., 2006). This indicates that antisocial behavior is a much more deviant behavior among girls. It is therefore interesting that we found a stronger genetic influence for antisocial behavior in girls. The causes for the sex difference in the genetic and environmental etiology of antisocial behavior are clearly an area that requires further research.

Understanding the genetic and environmental causes of antisocial behavior is an important priority for crime prevention. The results in this thesis are consistent with accumulative evidence suggesting that genetic effects on antisocial behavior are more important in girls, whereas shared environmental influences are stronger in boys (Lyons et al., 1995, Vierikko et al., 2003, Jacobson et al., 2002, Eley et al., 1999, Rose et al., 2004). Earlier research on antisocial behavior has tended to focus on boys only, and it may therefore have been easy to ignore the possibility that the genetic and environmental etiology of antisocial behavior may differ in boys and girls. Thus, our results may have implications for research and theory of the etiology of antisocial behavior.

Lastly, our results may also have implications for intervention programs. Males and females may not only differ in their genetic and environmental etiology of antisocial behavior, but they are also likely to have different prognoses and treatment responses. For example, there is an increased likelihood that antisocial girls develop relationships with antisocial males, become pregnant at an early age, and display dysfunctional and, harsh parenting with their own children (Serbin & Karp, 2004). Our results indicate that both boys and girls may benefit from prevention programs on a family or community level, but that girls may also benefit more from individual-based interventions.

6.1.3 The influence of genes and environments on the development of antisocial behavior

The second aim of this thesis was to investigate how genes and environments influence the development of antisocial behavior. This issue was addressed in both study I and II.

6.1.3.1 The development of aggressive and nonaggressive behavior in childhood to antisocial behavior in adolescence

In study I, we investigated the development of aggressive and nonaggressive behavior in childhood to antisocial behavior in early adolescence. For girls we found that the relationship between aggressive behavior and antisocial behavior was explained by genetic influences. The correlation between nonaggressive behavior and antisocial behavior was also due to continuity of genetic influences. For boys, there was no significant mediation between aggressive behavior and antisocial behavior, but there were significant shared environmental effects on the relationship between nonaggressive behavior and antisocial behavior.

With these data we found no differences in developmental trajectories between aggressive and nonaggressive pathways. However, our results suggest that there are sex differences in the development of antisocial behavior. Nonetheless, a main limitation in study I was the lack of power, resulting from the low phenotypic correlations between parent-reported and self-reported data. The small size of the associations, especially in the boys (e.g., there were no significant mediation between childhood aggressive behavior and adolescent antisocial behavior) limits our ability to draw firm conclusions from these data.

6.1.3.2 Genetic and environmental influences on antisocial behavior that persists from early adolescence to emerging adulthood

Very little is known about how genes and environments influence the development of antisocial behavior. The main focus in study II was therefore to examine whether a latent persistent antisocial behavior factor explained the covariance in antisocial behavior measured at three time points, from early adolescence to early adulthood (13-14 years, 16-17 years, and 19-20 years).

We found that strong familial effects (i.e., genetic and shared environmental effects) accounted for approximately 90 % of the total variance in antisocial behavior that persisted from early adolescence to emerging adulthood in both boys and girls, with the remaining 10 % due to a non-shared environmental factor. Our finding of a familial effect that accounts for 90 % of the variation contrasts against the findings from the

meta-analysis by Rhee and Waldman (2002), which found that only 57 % of the variance in antisocial behavior was due to familial effects (genetic factors: 41 %; shared environmental factors: 16 %).

There are a few previous twin studies that also have constructed a latent factor of children's or adolescents' antisocial behavior (Arseneault et al., 2003, Scourfield et al., 2004b). It is important to note that such a latent factor basically is free from measurement error. These studies have reported non-shared environmental factors of 0 % and 18 %, respectively. Our result of a non-shared environmental factor of 10 % in the latent persistent antisocial behavior factor is in agreement with previous research. This indicates that cross-sectional or univariate studies of antisocial behavior are likely to underestimate familial effects and to overestimate non-shared environmental effects, as this parameter also includes measurement error.

Taken together, our results highlight the importance of longitudinal studies for understanding the etiology of serious antisocial behavior. Further, an important question to answer in future research is whether the genetic factor in persistent antisocial behavior also is associated with childhood and/or adulthood psychopathology.

6.2 FACTORS ASSOCIATED WITH ANTISOCIAL BEHAVIOR

6.2.1 Covariation

The third aim of this thesis was to explore the covariation between antisocial behavior and psychopathic personality traits (study III). We found that a common genetic factor contributed substantially to the three psychopathic personality dimensions (grandiose/manipulative dimension; callous/unemotional dimension; impulsive/irresponsible dimension) and to antisocial behavior measured at age 13-14 years and age 16-17 years. Further, a common shared environmental factor was found to influence antisocial behavior, but did not influence psychopathic personality dimensions. There are several possible explanations to this finding.

One interpretation of our results is that antisocial behavior and psychopathic personality share a genetic diathesis. That is, antisocial behavior and psychopathic personality co-

occur because a common genetic liability increases the vulnerability to both dimensions. This common-inherited-liability hypothesis further predicts that a common set of genes would not only influence psychopathic personality traits and antisocial behavior, but also other problem behaviors, reflecting a common genetic vulnerability to disinhibitory behavior or externalizing psychopathology. Recent twin studies have also shown that a behavioral disinhibition or externalizing factor, reflecting the shared variance among various antisocial and problem behaviors, is substantially heritable (> 75 %) (Kendler et al., 2003, Krueger et al., 2002, Young et al., 2000, McGue et al., 2006). This factor is considered to reflect a shared vulnerability underlying various problem behaviors characterized as an inability to resist expressing inappropriate or restricted behavior (Young et al., 2000). In some studies it included adult antisocial behavior, conduct disorder, and substance abuse and dependence (Kendler et al., 2003, McGue et al., 2006), whereas in others studies in addition it also included attention-deficit hyperactivity disorder (ADHD) and personality traits such as novelty seeking (Young et al., 2000) and low constraint (constraint consist of traits that measure responsibility, dependability, and orderliness) (Krueger et al., 2002).

Another interpretation of our results is in line with a hypothesis arguing that the genetic diathesis of a behavior or disorder is partially mediated by temperament and personality (Goldsmith & Gottesman, 1996, Slutske et al., 1997b, Lahey et al., 2003, Kendler et al., 1993a). In our case, genetic influences may act indirectly on antisocial behavior, via psychopathic personality traits. Our results can be interpreted in support of this hypothesis for several reasons. First, in line with previous research (Walters, 2003, Hart & Hare, 1997), antisocial behavior and psychopathic personality were found to be highly correlated. Second, consistent with evidence previously reported across behavioral genetic studies of psychopathic personality traits (Blonigen et al., 2005, Larsson et al., 2006, Taylor et al., 2003), as well as other personality dimensions (Bouchard & Loehlin, 2001) was the common shared environmental factor found to contribute meagerly to the three psychopathic personality dimensions. In contrast, the common shared environmental factor was important for antisocial behavior. This suggests an etiological distinction between psychopathic personality traits and antisocial behavior.

This etiological distinction also corresponds to McCrae and Costa's (1995) model of basic tendencies and characteristic adaptations, basic tendencies being core personality traits and characteristic adaptations being the product of the interaction between basic tendencies and socio-cultural influences (McCrae & Costa, 1995). In other words, psychopathic personality traits might be more of a basic tendency, whereas antisocial behavior could be viewed as characteristic adaptations (Cooke & Michie, 2001).

There are other possible explanations to the observed findings (Rutter, 1997, Middeldorp et al., 2005). For example, the observed covariation could also be due to item overlap. However, there were no overlaps in the items that define psychopathic personality and antisocial behavior, therefore the association is probably not attributed to measurement confounding.

Psychopathic personality traits were measured in study III concurrently and partly after antisocial behavior; consequently it cannot be ruled out that antisocial behavior impairs personality development. Even so, our findings point to the importance of studying sequential patterns of covariation (Angold et al., 1999a) using behavioral genetic methods. An important question for future research is to further investigate whether genetic influences associated with psychopathic personality traits, as well as other personality and temperamental characteristics (Krueger et al., 1994, Cukrowicz et al., 2006) are correlated with the emergence of later antisocial behavior.

6.2.2 Gene-environment interaction

The last aim of this thesis was to investigate whether socioeconomic status moderates the impact of genetic and environmental effects on antisocial behavior (study IV).

Using continuous measures of socioeconomic status we found that in socioeconomically more advantaged environments, the influence of genetic effects on antisocial behavior was higher and the shared environment was lower. In contrast, for adolescents in socioeconomically less advantaged environments, genetic influence on antisocial behavior was lower and the influence of the shared environment was more important. These main results were also supported when arbitrary cut-off between more advantaged and less advantaged neighbourhoods was used.

Our results are in line with the theoretical reasoning put forward by several researchers (Raine, 2002c, Bronfenbrenner & Ceci, 1994). An interpretation of our results in view of the 'social push perspective' (Raine, 2002c) would suggest that the influence of genetic factors are more expressed in a socioeconomically advantaged environment where the environmental risk factors that push or predispose an adolescent to antisocial behavior are not present. In contrast, genetic factors for antisocial behavior will be weaker and the shared environment more important in a socioeconomically less advantaged environment because the environmental risk factors will camouflage the genetic contribution.

Analogous findings were reported in an early Danish twin study, heritability for crime was strikingly greater in those from high socioeconomic backgrounds and those who were rural born (Christiansen, 1977).

From these results we concluded that in addition to intervention directed towards susceptible adolescents in high risk environments, our results suggest that different intervention policies might be efficient in different socioeconomic areas. In more advantaged areas, individually based-interventions might be more effective. In less advantaged areas, intervention policies might be needed on a community level to reduce the influence of common environmental risk factors on adolescents, such as level of crime in the neighbourhood.

6.3 METHODOLOGICAL CONSIDERATIONS

The analyses in this thesis have relied on some basic assumptions underlying all twin studies. Below is a discussion of how these assumptions could have affected the results. Further, possible sampling biases, reliability and validity of the antisocial behavior measure are also discussed.

6.3.1 Assumptions in twin studies

6.3.1.1 The equal environment assumption

In the classical twin design MZ twins who share 100 % of their genes, are compared to DZ twins who on average share 50 % of their segregating genes. If MZ twins are more similar in the trait being studied than are DZ twins, it may be inferred that the difference is caused by genetic effects. To make this inference, it is necessary to rely on

the 'equal environment assumption' (EEA). Explicitly, it has to be assumed that environmentally caused similarity is roughly the same for both types of twins. If this assumption is violated, higher correlations among MZ twins may be due to environmental factors, rather than genetic factors, and heritability estimate will be overestimated (Plomin et al., 2001).

There are different study designs to examine the EEA (Kendler et al., 1993b, Scarr & Carter-Saltzman, 1979). One design compares the impact of twin resemblance of correctly diagnosed zygosity versus perceived zygosity. In other words, twins who view themselves as monozygotic may also be expected to be more alike by others (e.g., parents, teachers, peers). This expectation may influence the similarity of twin's behaviors or traits. Consequently, the self-perception that a twin pair has regarding their zygosity could influence their trait similarity.

Studies that have examined the EEA have generally shown that the assumption is fully justified for numerous phenotypes such as physical activity, eating behavior, psychiatric disorders (e.g., major depression, generalized anxiety disorder, phobia, alcohol dependence, and illicit drug dependence) (Hettema et al., 1995, Kendler et al., 1993b, Klump et al., 2000, Eriksson et al., 2006, Xian et al., 2000), including childhood and adolescent psychopathology such as anxiety disorder, attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder (Jacobson et al., 2002;Cronk et al., 2002) and aggression (Derks et al., 2006). In conclusion, even though we did not test the EEA for antisocial behavior, it is unlikely that the EEA would have severely biased our estimates.

6.3.1.2 Assortative mating

It is generally assumed in twin models that random mating occurs in the parent generation. Assortive mating tends to increase similarity between DZ twins, thereby bias the heritability estimates downward and the shared environmental estimates upward. A significant correlation between spouses for a particular trait is often interpreted as assortative mating (Maes et al., 1998). Assortative mating for antisocial behavior has previously been reported (Taylor et al., 2000b, Krueger et al., 1998). Taylor and colleagues (2000) found that parents of twins were correlated only .3 on delinquency self-report measures, and assumed that assortative mating is modest in

degree. Krueger and colleagues (1998), on the contrary, found a correlation of .54 for assortative mating for self-reported antisocial behavior, and concluded that assortative mating should to be taken into account when modeling antisocial behavior.

At wave 4, mothers and fathers were asked to retrospectively report their antisocial behaviors in their twenties. We found correlation between the parents for antisocial behavior to be .15 ($p < .001$), suggesting that the effect of assortative mating is limited for antisocial behavior in this sample.

6.3.1.3 Gene-environment correlation

It is generally assumed in the twin design that genetic and environmental influences are uncorrelated. Gene-environment correlation (r_{GE}) refers to an individual's unique experiences correlated with his or her personal characteristics. Gene-environment correlation may explain human behavioral development, particularly in parent and child relationships. Gene-environment correlation is generally considered to be of three types (Scarr & McCartney, 1983):

(i) Passive r_{GE} occurs mainly because of gene overlap between parents and their children. For example, children of aggressive parents inherit genetic susceptibility for aggression as well as experience an adverse rearing environment. Neiderhiser and colleagues (2004) recently reported passive-gene-environment correlation for mother's positivity and monitoring toward her child.

(ii) Evocative/reactive r_{GE}: different child characteristics may elicit a particular response from the environment. Previous studies on antisocial behavior have shown that aggressive children tend to elicit more negative environments from their parents (O'Connor et al., 1998a, Ge et al., 1996, Narusyte et al., in press).

(iii) Active r_{GE} is defined as the process whereby an individual actively seeks out environmental situations that are more closely matched to the person's genotype.

We found for girls that the heritability estimates for antisocial behavior increased with age, whereas the influence of the shared environment decreased. This could reflect active r_{GE}, suggesting that as girls grow older they are probably less supervised, and

may select environments e.g., delinquent peers, that are correlated with their phenotypes. Our findings particularly for girls, together with earlier research (O'Connor et al., 1998a, Ge et al., 1996, Narusyte et al., in press, Neiderhiser et al., 2004), suggest that the assumption of no gene-environment correlation may be invalid. Consequently, the heritability estimate for antisocial behavior may include both additive genetic effects and the effects of gene-environment correlation.

6.3.1.4 Sibling interaction and rater bias

Sibling interaction can either be positive or negative. Positive interaction refers to siblings imitating (cooperating) each other's behavior, and negative interaction refers to siblings taking on opposite or competing behavior (Carey, 1986, Carey, 1992). Rater bias occurs when parents either stress the similarities or differences in their children. If there is sibling interaction, either true imitation/contrast effects or rater bias, variance differences between MZ and DZ are expected. A positive sibling interaction effect is confounded with shared environmental effects, and a negative sibling interaction effect is confounded with dominant effects (Vierikko et al., 2004, Vierikko et al., 2003, Rietveld et al., 2003a, Rietveld et al., 2003b).

Twins sometimes engage in antisocial acts together (Rowe, 1983), we therefore tested for possible sibling interaction effects for antisocial behavior at ages 13-14, 16-17, and 19-20. To test for sibling interaction, a reciprocal path was added between the antisocial behavior scores of the twins in a univariate model. However, an ACE model provided a better fit at all three time points, indicating no sibling interaction.

6.3.1.5 Non-additive genetic effects

Twin studies generally assume that only one type of genetic mechanism, usually additive, is operating for a particular trait. Violation of this assumption may lead to underestimation of shared environmental effects. DZ intraclass correlations less than half of the MZ correlations would suggest an effect of non-additive genetic variation. There was no evidence of genetic dominance or epistasis for antisocial behavior at any of the three measurement occasions (ages 13-14, 16-17, and 19-20); hence dominance was not modeled for in any of the studies.

6.3.1.6 Generalisability to singletons

Twins and singletons are generally assumed to be the same with regard to the likelihood that they will show a particular trait such as antisocial behavior. This assumption is probably in general defensible as twins and singletons have been found to experience similar rates of psychiatric disorders (e.g., attention deficit hyperactivity disorder, oppositional defiant disorder, conduct disorder), and behavioral and emotional problems (Moilanen et al., 1999, van den Oord et al., 1995, Gjone & Novik, 1995, Simonoff et al., 1997).

There are however, two ways in which twins differ from singletons: (i) higher rates of obstetric complications, in particular lower birth weight and shorter length of gestation (Plomin et al., 2001), and (ii) delayed language development (Rutter & Redshaw, 1991). It is possible that these differences have a minor effect on traits and behavior later in life (Christensen et al., 2006). It should be mentioned that several studies have shown that children with birth complications are more likely to later develop antisocial behavior (Raine, 2002a). However, it has also been suggested that birth complications may not by themselves predispose crime, but may require the presence of negative environmental risk factors, e.g., poor parenting, maternal rejection (Hodgins et al., 2001, Raine et al., 1997).

6.3.2 Possible sampling biases

Even though the TCHAD-study has a relatively high response rate, there was on average a non-participation rate of 25 %. Attrition further increased the loss, particularly between wave 3 and wave 4. Those missing from the sample are likely to include a disproportionate percentage with psychopathology in either the parents or children. Thus, it is not certain that all of the results are generalizable to individuals with the most extreme externalizing behaviors (Moffitt & E-Risk Study Team, 2002). It should also be mentioned that as with other volunteer twin samples, individuals from lower socioeconomic status groups were more likely not to participate.

Further, this thesis is based on a single Swedish cohort of twins born 1985–1986. Although prevalence rates for antisocial behavior were similar to findings in other samples (Junger-Tas et al., 1994, Ring, 2005), the findings in this thesis need replication in other longitudinal samples in other parts of the world.

6.3.3 Reliability and validity of the antisocial behavior measure

Reliability of a measure is the extent to which it provides consistent information every time we use it. Validity refers to the accuracy with which an instrument assesses what it is supposed to assess.

6.3.3.1 Reliability

Test-retest reliability was measured by comparing the answers from those who responded both to the questionnaire and to the telephone interview (age 16-17; wave 3). There was on average two months between the questionnaire and telephone responses. Correlation between those (n = 72) twins who responded to both the questionnaire and to the telephone interview was good: $r_{(p)} = .73$. These analyses suggest that antisocial behavior is not unduly affected by measurement error associated with differences in occasions.

6.3.3.2 Validity

The items in the antisocial behavior measure were initially derived from an instrument used in the project *Delinquent Behavior among Young People in the Western World* comparing self-reports of antisocial behavior in 14 studies in 13 different countries (Junger-Tas et al., 1994). The validity of the measure was extensively addressed in each of these studies. Overall, the measure was reported to have good psychometric properties and moderate to high validity (see for example Moffitt et al., 1994). The measure was later translated into Swedish, and transformed from interview to questionnaire format (Ring, 1999).

A concurrent validity check for the purpose of this thesis was carried out by comparing the antisocial behavior data in the TCHAD study (age 16-17; wave 3) with results from a school study, which used the same measure of antisocial behavior (Ring, 2005). This study was a population-based sample including 5,600 adolescents age 15-16 years. The prevalence for having committed at least one offence within the last twelve months (excluding 4 items: train/bus fare evasion, truancy, drive car without licence, use false ID) was somewhat higher in the school sample (boys: 67 %; girls: 54 %) compared to the TCHAD study (boys: 53 %; girls: 42 %). The difference in prevalence between these two samples is probably due to differences in data collection. In other words,

there may be a tendency to exaggerate when filling out an anonymous questionnaire at school (Ring, 1999), and a tendency to conceal when filling out an identifiable questionnaire at home. To summarize, the antisocial behavior measure has overall reasonably good validity.

6.3.4 Possible biases related to self-reports

The reliability and validity of the self-report method in antisocial behavior studies has been assessed by several researchers in great detail (Hirschi, 1969, Elliott et al., 1983, Weis, 1986, O'Brien, 1985, Klein, 1989). For example, some studies have used police reports as control to see whether the offences mentioned by respondents also figures in police reports. Such studies have shown that adults tend to underreport offences, while for adolescents the results suggest moderate to high validity (Hindelang et al., 1981). Although the self-report method is accepted as a valid means of measuring antisocial behavior, it has a number of limitations.

Firstly, those individuals for who self-reports are *not* obtained, are likely to be those who are engaged in more serious and frequent antisocial behavior, or those with characteristics associated with an increased risk of antisocial behavior. Self-report studies typically do not include respondents from the most serious antisocial individuals.

Secondly, the extent to which respondents conceal or exaggerate their antisocial acts is very difficult to test fully. Some respondents may intentionally exaggerate their antisocial behavior. While others may be reluctant to reveal their antisocial behavior because they do not feel certain that their responses will be treated confidentially.

Thirdly, self-reports may also be affected by recall bias, i.e., the respondent's answers to a question may be affected not just by the correct answer, but also by the respondent's memory.

Nonetheless, self-report will provide information on unrecorded antisocial acts and antisocial individuals, which is unavailable from any other source.

6.4 FUTURE DIRECTIONS

A next step for behavioral-genetic research into antisocial behavior is to examine endophenotypes. An endophenotype is a heritable trait or characteristic that is not a direct symptom of the condition under investigation but has been shown to be associated with the condition (Gottesman & Gould, 2003). Research into antisocial behavior will find endophenotype studies very useful for explaining how genes increase the probability that people will commit antisocial acts.

Psychophysiological factors are excellent candidates for endophenotypes of antisocial behavior. For example, it has been shown that reduced P300 amplitude (the P300 component is an event-related brain potential (ERP) that is a biological marker for disturbed cognitive processing in psychopathology) can be used to distinguish prisoners with impulsive aggression from other prisoners with non-impulsive forms of aggression (Barratta et al., 1997). More recently, early onset problem behavior with an onset prior to age 15 was reported to be associated with smaller P300 amplitudes (Iacono & McGue, 2006). Moreover, combinations of multiple psychophysiological variables may allow greater differentiation among subtypes of antisocial individuals. This approach has been used successfully, for example, to improve distinction between individuals with and without substance use disorders (Iacono et al., 2000).

Although research points to the possible importance of psychophysiological factors in the development of antisocial behavior (Raine, 1993, Beauchaine, 2001, Bradley et al., 1993), these abnormalities could have developed as a result of, rather than be a cause of, antisocial and violent behavior. Research should therefore be carried out in prospective studies.

Psychophysiological factors associated with antisocial behavior can be examined in behavior genetic research. First, to investigate the genetic and environmental basis of psychophysiological correlates of antisocial behavior, and second, to determine the extent to which genes and environment mediate relationships among these psychophysiological variables and their relationships to children's and adolescent's antisocial behavior.

7 CONCLUSIONS

- Both genetic and environmental factors are important for the development of antisocial behavior. However, the genetic and environmental etiology of antisocial behavior differs between boys and girls. Heritability was found to be higher in girls, whereas the shared environment was more important in boys.

- There are sex differences in the development of antisocial behavior. The association between aggressive behavior in childhood and antisocial behavior in adolescence was genetically mediated in girls. In contrast, the relationship between nonaggressive behavior and antisocial behavior was mainly environmentally dependent in boys.

- Antisocial behavior that persists from early adolescence to emerging adulthood has strong familial effect in both boys and girls.

- There was a strong genetic overlap between psychopathic personality traits and antisocial behavior. This genetic overlap could reflect that psychopathic personality has an important role in mediating genetic effects to antisocial behavior. Alternatively, it may reflect a genetic vulnerability to externalizing psychopathology.

- Socioeconomic status moderates the influence of genetic and environmental factors on antisocial behavior. Genetic influences on antisocial behavior were more important in adolescents in socioeconomically more advantaged environments, whereas the shared environment was higher in adolescents in socioeconomically less advantaged environments.

8 ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to everyone who has contributed to this thesis. First of all, I would like to thank all twins and parents for participating in the Twin Study of CHild and Adolescent Development (TCHAD). Next, I would like to thank:

Paul Lichtenstein, my principal SUPERvisor, for your friendship and invaluable support in so many ways through these years, for being such an incredibly sharp scientist, and for so generously sharing your knowledge with me. Thank you is not enough!

Martin Grann, my co-supervisor for excellent support, brilliant ideas, for your great knowledge and guidance in the field of psychiatric epidemiology. Thank you!

Henrik Larsson, friend and co-author, it was great working with you! You made a real difference at MEB. I agree; we do share a lot of great memories!

Henrik Andershed, Jerzy Sarnecki, Thalia Eley, and Fruhling Rijsdijk for co-authorship.

Nancy Pedersen, head of the Department of Medical Epidemiology and Biostatistics, for help and support, and for being such a great a role model for women in science. I truly admire you!

Margy Gatz and Malcolm Klein for proof reading and encouragement!

Niklas Långström for tremendous support lately, for discussions about everything from research to every day matters.

Gunilla Sonnebring, thank you so much for your outstanding help, from more difficult matters such as endnote to proof reading all my papers and ‘kappa’, and last but not least for friendly chats.

Nils-Magnus Björkman, my former teacher at Stockholm University, for introducing me to Paul Lichtenstein.

All of you who have worked with data collection in the TCHAD-study before me, together with me, and who continued when I started my PhD-studies.

Present and former colleagues at the Department of Medical Epidemiology and Biostatistics for creating a pleasant working atmosphere: Emma Nilsson for great friendship, Jurgita Narusyte, Kenji Kato, Pia Svedberg, Lennart Martinsson, Kerstin Linderholm, Jenny Carlsson, Anastasia Iliadou, Alexandra Ekman, Eva Carlström, Christina Hultman, Camilla Björk, Mats Forsman, Anna Svensson, Anna Hübinette, Christer Henriksson, Erica Spotts, Anna Lindam, Monika Rundgren, Kamila Czene, Maria Hedelin, Lena George, Junmei Miao, Catarina Jansson, Michaela Prochazka, Niklas Bergenvall, Denny Rönngren, Anthony Gunnell, Linda Lindström, Andreas Jacks, Ulrika Eriksson, Pär Sparén, Mårten Kivi, Anna Johansson, Cat Haltur, Sven Cnattingius, Paul Dickman, Måns Flensburg, and many more!!!

Friends, especially Maria Källström, Anna von Werder, Christina Lenndelius, Karin Gunnarsson, Stéphane Tessier, and Mia Sandberg.

My sister-in-law Caroline and my sweet niece Jessica for taking care of Alice when I have been at MEB, and of course Patricia!

My dear sisters Annette and Helene and their families.

Joe, Elizabeth and Alice for making everything worthwhile.

I would also like to acknowledge the funding sources that supported this research.

9 REFERENCES

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 Profile.* Burlington, VT: University of Vermont Department of Psychiatry.

Achenbach, T. M. & Rescorla, L. A.(2000).Manual for ASEBA School-Age Forms & Profiles. Burlington: VT: University of Vermont, Research Center for Children, Youth, & Families.

Ambrosini, P. J. (2000). Historical development and present status of the schedule for affective disorders and schizophrenia for school-age children (K-SADS). *Journal of American Academy of Child and Adolescent Psychiatry, 39*, 49-58.

Andershed, H., Kerr, M., Stattin, H. & Levander, S.(2002).Psychopathic traits in non-referred youths: A new assessment tool. In (Eds, Blaauw, E. & Sheridan, L.), *Psychopaths: Current International Perspectives* (pp. 131-158). The Hague: Elsvier.

Angold, A., Costello, E. J. & Erkanli, A. (1999a). Comorbidity. *Journal of Child Psychology and Psychiatry, 40*, 57-87.

Arseneault, L., Moffitt, T. E., Caspi, A., Taylor, A., Rijsdijk, F. V., Jaffee, S. R., Ablow, J. C. & Measelle, J. R. (2003). Strong genetic effects on cross-situational antisocial behaviour among 5-year-old children according to mothers, teachers, examiner-observers, and twins' self-reports. *Journal of Child Psychol Psychiatry, 44*, 832-848.

Barratta, E. S., Stanford, M. S., Kenta, T. A. & Alana, F. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. *Biological Psychiatry, 41*, 1045-1061.

Bartels, M., Hudziak, J. J., van den Oord, E. J. C. G., van Beijsterveldt, C. E. M., Rietveld, M. J. H. & Boomsma, D. (2003). Co-occurrence of aggressive behavior and rule-breaking behavior at age 12: multi-rater analyses. *Behavior Genetics, 33*, 607-621.

Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13*, 183-214.

Beyers, J. M., Loeber, R., Wikström, P.-O. H. & Stouthamer-Loeber, M. (2001). What predicts adolescent violence in better-off neighbourhoods? *Journal of Abnormal Child Psychology, 29*, 379-381.

Biederman, J., Milberger, S., Faraone, S. V., Kiely, K., Guite, J., Mick, E., Ablon, J. S., Warburton, R., Reed, E. & Davis, S. G. (1995). Impact of adversity on functioning and comorbidity in children with attention-deficit hyperactivity disorder. *Journal of American Academy of Child and Adolescent Psychiatry, 34*, 1495-1503.

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J. & Iacono, W. G. (2005). Psychopathic personality traits: heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological Medicine, 35*, 637-648.

Bohman, M., Cloninger, C. R., Sigvardsson, S. & von Knorring, A.-L. (1982). Predisposition to petty criminality in Swedish adoptees. *Archives of General Psychiatry, 39*, 1233-1241.

Boomsma, D., Busjahn, A. & Peltonen, L. (2002). Classical Twin Studies and Beyond. *Nature, 3*, 872-882.

Boomsma, D., de Geus, E., van Baal, C. M. & Koopmans, J. R. (1999). A religious upbringing reduces the influence of genetic factors on disinhibition: evidence for interaction between genotype and environment on personality. *Twin Research and Human Genetics, 2*, 115-125.

Bouchard, T. J., Jr. & Loehlin, J. C. (2001). Genes, evolution, and personality. *Behavior Genetics, 31*, 243-273.

Bradley, M. M., Cuthbert, B. N. & Lang, P. J. (1993). Pictures as prepulse: attention and emotion in startle modification. *Psychophysiology, 30*, 541-545.

Bronfenbrenner, U. & Ceci, S. J. (1994). Nature-nurture reconceptualized: A bioecological model. *Psychological Review, 101*, 568-586.

Brooks-Gunn, J., Duncan, G., Klebanov, P. K. & Sealand, N. (1993). Do neighbourhoods influence child and adolescent development? *American Journal of Sociology, 99*, 353-395.

Burt, A. S., Krueger, R. F., McGue, M. & Iacono, W. G. (2001). Sources of covariation among attention-deficit/hyperactivity disorder: The importance of shared environment. *Journal of Abnormal Psychology, 110*, 516-525.

Burt, S. A., McGue, M., DeMarte, J. A., Krueger, R. F. & Iacono, W. G. (2006). Timing of menarche and the origins of conduct disorder. *Archives of General Psychiatry, 63*, 890-896.

Button, T. M., Hewitt, J. K., Rhee, S. H., Young, S. E., Corley, R. P. & Stallings, M. C. (2006). Examination of the causes of covariation between conduct disorder symptoms and vulnerability to drug dependence. *Twin Research and Human Genetics, 9*, 38-45.

Button, T. M. M., Scourfield, J., Martin, N. & McGuffin, P. (2004). Do aggressive and non-aggressive antisocial behaviors in adolescents result from the same genetic and environmental effects? *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics), 129 B*, 59-63.

Button, T. M. M., Scourfield, J., Martin, N., Purcell, S. & McGuffin, P. (2005). Family dysfunction interacts with genes in the causation of antisocial symptoms. *Behavior Genetics, 35*, 115-120.

Cadoret, R. J., Cain, C. A. & Crowe, R. R. (1983). Evidence for gene-environment interaction in the development of adolescent antisocial behavior. *Behavior Genetics, 13*, 301-310.

Cadoret, R. J., Yates, W. R., Troughton, E., Woodworth, G. & Stewart, M. A. (1995). Genetic-environmental interaction in the genesis of aggressivity and conduct disorders. *Archives of General Psychiatry, 52*, 916-24.

Carey, G. (1986). Sibling imitation and contrast effects. *Behavior Genetics, 16*, 319-341.

Carey, G. (1992). Twin imitation for antisocial behavior: implications for genetic and family environment research. *Journal of Abnormal Psychology, 101*, 18-25.

Caspi, A., Lynam, D. R., Moffitt, T. E. & Silva, P. A. (1993). Unraveling girl's delinquency: biological, dispositional, and contextual contributions to adolescent misbehavior. *Developmental Psychology, 29*, 19-30.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A. & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*, 851-854.

Christensen, K., Petersen, I., Skytthe, A., Herskind, A. M., McGue, M. & Bingley, P. (2006). Comparison of academic performance of twins and singletons in adolescence: follow-up study. *British Medical Journal, 6*, 1-5.

Christiansen, K. O.(1977).A preliminary study of criminality among twins. In (Eds, Mednick, S. A. &Christiansen, K. O.), *Biosocial bases of criminal behavior* (pp. 89-108). New York: Gardner Press.

Cleckley, H. J. (1941). *The mask of sanity.* St. Louis: MO: Mosby.

Cohen, M. A. (1998). The Monetary Value of Saving a High-Risk Youth. *Journal of Quantitative Criminology, 14*, 5-33.

Cooke, D. J. & Michie, C. (2001). Refining the construct of psychopathy: towards a hierarchical. *Psychological Assessment, 13*, 171-188.

Costello, E. J., Angold, A., Burns, B. J., Stangl, D. K., Tweed, D. L., Erkanli, A. & Worthman, C. M. (1996). The Great Smoky Mountains Study of youths - Goals, design, methods, and the prevalence of DSM-III-R disorders. *Archives of General Psychiatry, 53*, 1129-1136.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G. & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60*, 837-844.

Cottle, C. C., Lee, R. J. & Heilburn, K. (2001). The Prediction of Criminal Recidivism in Juveniles. *Criminal Justice and Behavior, 28*, 367-394.

Cronk, N., Slutske, W. S., Madden, P. A., Bucholz, K. K., Reich, W. & Heath, A. (2002). Emotional and behavioral problems among female twins: an evaluation of the equal environments assumption. *Journal of American Academy of Child and Adolescent Psychiatry, 41*, 829-837.

Crowe, R. R. (1974). An adoption study of antisocial personality. *Archives of General Psychiatry, 31*, 785-791.

Cukrowicz, K. C., Taylor, J., Schatschneider, C. & Iacono, W. G. (2006). Personality differences in children and adolescents with attention-deficit/hyperactivity disorder, conduct disorder, and controls. *Journal of Child Psychology and Psychiatry, 47*, 151-159.

Derks, E. M., Dolan, C. V. & Boomsma, D. I. (2006). A test of the equal environment assumption (EEA) in multivariate twin studies. *Twin Research, 9*, 403-411.

Dick, D. M. & Rose, R. J. (2002). Behavior Genetics: What's New? What's Next? *Current Directions in Psychological Science, 11*, 70-74.

Dick, D. M., Rose, R. J., Viken, R. J., Kaprio, J. & Koskenvuo, M. (2001). Exploring gene-environment interactions: socioregional moderation of alcohol use. *Journal of Abnormal Psychology, 110*, 625-632.

Dick, D. M., Viken, R. J., Kaprio, J., Pulkkinen, L. & Rose, R. J. (2005). Understanding the covariation among childhood externalizing symptoms: genetic and environmental influences on conduct disorder, attention deficit hyperactivity disorder, and oppositional defiant disorder symptoms. *Journal of Abnormal Child Psychology, 33*, 219-229.

DiLalla, L. F. (2002). Behavior genetics of aggression in children: review and future directions. *Developmental Review, 22*, 593-622.

DSM-IV (1994). *Diagnostic and Statistical Manual of Mental disorders.* American Psychiatric Association.

Edelbrock, C., Rende, R., Plomin, R. & Thompson, L. A. (1995). A twin study of competence and problem behavior in childhood and early adolescence. *Journal of Child Psychology and Psychiatry, 36*, 775-785.

Ehringer, M. A., Rhee, S. H., Young, S. E., Corley, R. P. & Hewitt, J. K. (2006). Genetic and environmental contributions to common psychopathologies of childhood and adolescence: A study of twins and their siblings. *Journal of Abnormal Child Psychology, 34*, 1-17.

Eley, T. C., Lichtenstein, P. & Moffitt, T. E. (2003). A longitudinal behavioral genetic analysis of the etiology of aggressive and non-aggressive antisocial behavior. *Development and Psychopathology, 15*, 383-402.

Eley, T. C., Lichtenstein, P. & Stevenson, J. (1999). Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: results from two twin studies. *Child Development, 70*, 155-68.

Elliott, D. S., Ageton, S. S., Huizinga, D., Knowles, B. A. & Canter, R. J. (1983). *The Prevalence and Incidence of Delinquent Behavior: 1976-1980. The National Youth Survey Report NO. 26.* Boulder CO.: Behavioral Research Institute.

Eriksson, M., Rasmussen, F. & Tynelius, P. (2006). Genetic factors in physical activity and the equal environment assumption - the Swedish young male twin study. *Behavior Genetics, 36*, 238-247.

Evans, D. M., Gillespie, N. A. & Martin, N. G. (2002). Biometrical genetics. *Biological Psychology, 61*, 33-51.

Farrington, D. P., Barnes, G. C. & Lambert, S. (1996). The concentration of offending in families. *Legal and Criminological Psychology, 1*, 47-63.

Farrington, D. P. & Loeber, R. (2000). Epidemiology of juvenile violence. *Child and Adolescent Psychiatric Clinics of North America, 9*, 733-748.

Fergusson, D. M. & Horwood, L. J. (2002). Male and female offending trajectories. *Development and Psychopathology, 14*, 159-177.

Foley, D. L., Eaves, L., Wormley, B., Silberg, J., Maes, H., Kuhn, J. & Riley, B. (2004). Childhood adversity, monoamine oxidase a genotype, and risk for conduct disorder. *Archives of General Psychiatry, 61*, 738-744.

Forth, A. E., Kosson, D. S. & Hare, R. D. (2003). *The Psychopathy Checklist: Youth Version. Manual.* North Tonawanda, N.Y.: Multi-Health Systems, Inc.

Frick, P. J., Kimonis, E. R., Dandreaux, D. M. & Farell, J. M. (2003). The 4 year stability of psychopathic traits in non-referred youth. *Behavioral Science and the Law, 21*, 713-36.

Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A. G., Hart, E. A. & Hanson, K. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review, 13*, 319-340.

Ge, X., Conger, R. D., Cadoret, R. J., Neiderhiser, J. M., Yates, W. R., Troughton, E. & Stewart, M. A. (1996). The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology, 32*, 574-589.

Gelhorn, H. L., Stallings, M. C., Young, S. E., Corely, R. P., Rhee, S. H. & Hewitt, J. K. (2005). Genetic and environmental influences on conduct disorder: symptom, domain, and full-scale analyses. *Journal of Child Psychology and Psychiatry, 46*, 580-591.

Gelhorn, H. L., Stallings, M. C., Young, S. E., Corely, R. P., Rhee, S. H., Hopfer, C. & Hewitt, J. K. (2006). Common and specific genetic influences on aggressive and nonaggressive conduct disorder domains. *Journal of American Academy of Child and Adolescent Psychiatry, 45*, 570-577.

Ghodesian-Carpey, J. & Baker, L. A. (1987). Genetic and environmental influences on aggression in 4- to 7-year-old twins. *Aggressive Behavior, 13*, 173-186.

Gjone, H. & Novik, T. S. (1995). parental ratings of behaviour problems: a twin study and general population comparison. *Journal of Child Psychology and Psychiatry, 36*, 1213-1224.

Goldsmith, H. H. & Gottesman, I.(1996).Heritable variability and variable heritability in developmental psychopathology. In (Eds, Lenzenweger, M. &Haugaard, J.), *Frontiers of developmental psychopathology* (pp. 5-43). Oxford: OUP.

Goldstein, R. B., Prescott, C. A. & Kendler, K. S. (2001). Genetic and environmental factors in conduct problems and adult antisocial behavior among adult female twins. *Journal of Nervous and Mental Disease, 189*, 201-209.

Gottesman, I. I. & Gould, T. D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *American Journal of Psychiatry, 1160*, 636-645.

Grann, M., Langstrom, N., Tengstrom, A. & Kullgren, G. (1999). Psychopathy (PCL-R) predicts violent recidivism among criminal offenders with personality disorders in Sweden. *Law and Human Behavior, 23*, 205-17.

Griffin, D. & Gonzales, R. (1995). Correlation analysis of dyad-level data in the exchangeable case. *Psychological Bulletin, 118*, 430-439.

Hare, R. D. (1991). *The Hare Psychopathy Checklist-Revised manual.* Toronto: Multi-Health System.

Hare, R. D. (2003). *The Hare Psychopathy Checklist-Revised (PCL-R): 2nd edition.* Toronto, Ontario, Canada: Multi-Health Systems.

Hare, R. D., Clark, D. A., Grann, M. & Thornton, D. (2000). Psychopathy and the predictive validity of the PCL-R: international perspective. *Behavioral Science and the Law, 18*, 623-645.

Hart, D. J. & Hare, R. D.(1997).Psychopathy: assessment and association with criminal conduct. In (Eds, Stoff, D. M., Maser, J. &Brieling, J.), *Handbook of antisocial behavior* (pp. 22-35). New York: Wiley.

Hawkins, J. D., Herrenkohl, T., Farrington, D. P., Brewer, D., Catalano, R. F. & Harachi, T. W.(1998).A review of predictors of youth violence. In (Eds, Loeber, R. &Farrington, D. P.), *Serious and violent juvenile offenders: Risk factors and successful interventions* (pp. 106-146). CA: Sage: Thousands Oaks.

Heath, A. C., Madden, P. A. & Martin, N. G. (1998). Assessing the effects of cooperation bias and attrition in behavioral genetic research using data-weighting. *Behavior Genetics, 28*, 415-427.

Hemphill, J. F., Hare, R. D. & Wong, S. (1998). Psychopathy and recidivism: A review. *Legal and Criminological Psychology, 3*, 139-170.

Hettema, J. M., Neale, M. C. & Kendler, K. S. (1995). Physical similarity and the equal-environment assumption in twin studies of psychiatric disorders. *Behavior Genetics, 25*, 327-335.
Hicks, B. M., Krueger, R. F., Iacono, W. G., McGue, M. & Patrick, C. J. (2004). Family transmission and heritability of externalizing disorders: a twin-family study. *Archives of General Psychiatry, 61*, 922-928.
Hindelang, M. J., Hirschi, T. & Weis, J. G. (1981). *Measuring delinquency.* Beverly Hills, California: Sage Publishers.
Hinshaw, S. P. (2002). Intervention research, theoretical mechanisms, and causal processes related to externalizing behavior patterns. *Development and Psychopathology, 14*, 789-818.
Hipwell, A. E., Loeber, R., Stouthamer-Loeber, M., Keenan, K., White, H. R. & Kroneman, L. (2002). Characteristics of girls with early onset disruptive and antisocial behaviour. *Criminal Behaviour and Mental Health, 12*, 99-118.
Hirschi, T. (1969). *Causes of Delinquency.* Berkeley: University of California Press.
Hodgins, S., Kratzer, L. & McNeil, T. F. (2001). Obstetric complications. parenting, and risk of criminal behavior. *Archives of General Psychiatry, 58*, 746-752.
Hudziak, J. J., van Beijsterveldt, C. E. M., Bartels, M., Rietveld, M. J. H., Rettew, D. C., Derks, E. M. & Boomsma, D. (2003). Individual differences in aggression: Genetic analyses by age, gender, and informant in 3-, 7-, and 10-year-old Dutch Twins. *Behavior Genetics, 33*, 575-589.
Iacono, W. G., Carlson, S. R. & Malone, S. M. (2000). Identifying a multivariate endophenotype for substance use disorders using psychophysiological measures. *International Journal of Psychophysiology, 38*, 81-96.
Iacono, W. G. & McGue, M. (2006). Association between P3 event-related brain potential amplitude and adolescent problem behavior. *Psychophysiology, 43*, 465-469.
Jacobson, K. C., Neale, M. C., Prescott, C. A. & Kendler, K. (2001). Behavioral genetic confirmation of a life-course perspective on antisocial behavior: can we believe the results? [conference presentation]. *Behavior Genetics, 31*, 456.
Jacobson, K. C., Prescott, C. A. & Kendler, K. (2002). Sex differences in the genetic and environmental influences on the development of antisocial behavior. *Developmental Psychology, 14*, 395-416.
Jacobson, K. C., Prescott, C. A., Neale, M. C. & Kendler, K. (2000). Cohort differences in genetic and environmental influences on retrospective reports of conduct disorder among adult male twins. *Psychological Medicine, 30*, 775-787.
Jang, K. L. (2005). *The behavioral genetics of psychopathology.* Mawah, New Jersey: Lawrence Erlbaum Associates.
Junger-Tas, J., Terlouw, G.-J. & Klein, M. W. (1994). *Delinquent Behavior Among Young People in the Western World.* Amsterdam: Kugler Publications.
Kendler, K. (2001). Twin Studies of Psychiatric Illness. *Archives of General Psychiatry, 58*, 1005-1014.
Kendler, K., Neale, M. C., Kessler, R. C., Heath, A. C. & Eaves, L. (1993a). A longitudinal twin study of personality and major depression in women. *Archives of General Psychiatry, 50*, 853-862.
Kendler, K., Neale, M. C., Kessler, R. C., Heath, A. C. & Eaves, L. J. (1993b). A test of the equal-environment assumption in twin studies of psychiatric illness. *Behavior Genetics, 23*, 21-27.
Kendler, K., Prescott, C. A., Myers, J. & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry, 60*, 929-937.
Kim-Cohen, J., Caspi, A., Moffitt, T. E., Harrington, H., Milne, B. J. & Poulton, R. (2003). Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. *Archives of General Psychiatry, 60*, 709-717.
Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W. & Moffitt, T. E. (2006). MAOA, maltreatment, and gene-environment interaction

predicting children's mental health: new evidence and a meta-analysis. *Molecular Psychiatry*, 1-11.
Klein, M. W. (1989). *Cross-national research in self-reported crime and delinquency.* Dordrecht, The Netherlands: Kluwer.
Klein, M. W. (1995). *The American street gang: its nature, prevalence and control.* New York: Oxford University Press.
Klump, K. L., Holly, A., Iacono, W. G., McGue, M. & Willson, L. E. (2000). Physical similarity and twin resemblance for eating attitudes and behaviors: a test of the equal environments assumption. *Behavior Genetics, 30*, 51-58.
Koopmans, J. R., Slutske, W. S., van Baal, C. M. & Boomsma, D. I. (1999). The influence of religion on alcohol use initiation: Evidence for genotype x environment interaction. *Twin Research and Human Genetics, 29*, 445-453.
Kraemer, H. C. (2003). Current concepts of risk in psychiatric disorders. Services research and outcomes. *Current Opinion in Psychiatry, 16*, 421-430.
Kroneman, L., Loeber, R. & Hipwell, A. E. (2004). Is neighborhood context differently related to externalizing problems and delinquency for girls compared with boys? *Clinical Child and Family Psychology Review, 7*, 109-122.
Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G. & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111*, 411-424.
Krueger, R. F., Moffitt, T. E., Caspi, A., Bleske, A. & Silva, P. A. (1998). Assortative mating for antisocial behavior: Developmental and methodological implications. *Behavior Genetics, 28*, 173-186.
Krueger, R. F., Schmutte, P. S., Caspi, A., Moffitt, T. E., Campbell, K. & Silva, P. A. (1994). Personality traits are linked to crime among men and women: evidence from a birth cohort. *Journal of Abnormal Psychology, 103*, 328-338.
Lahey, B. B., Moffitt, T. E., Caspi, A. (Eds.) (2003). *Causes of conduct disorder and juvenile delinquency.* New York: Guilford Press.
Larsson, H., Andershed, H. & Lichtenstein, P. (2006). A genetic factor explains most of the variation in the psychopathic personality. *Journal of Abnormal Psychology, 115*, 221-30.
Lau, J. Y. F. & Eley, T. C. (2004). Gene-Environments interactions and correlations in psychiatric disorders. *Current Psychiatry Report, 6*, 119-124.
Leventhal, T. & Brooks-Gunn, J. (2000). The neighbourhoods they live in: The effects of neighbourhood residence on child and adolescent outcomes. *Psychological Bulletin, 126*, 309-337.
Lichtenstein, P., Defaire, U., Floderus, B., Svartengren, M., Svedberg, P. & Pedersen, N. L. (2002). The Swedish Twin Registry: a unique resource for clinical, epidemiological and genetic studies. *Journal of Internal Medicine, 252*, 1-22.
Lichtenstein, P., Harris, J. R., Pedersen, N. L. & McClearn, G. E. (1993). Socioeconomic status and physical health, how are they related? An empirical study based on twins reared apart and twins reared together. *Social Science & Medicine, 36*, 441-450.
Lichtenstein, P., Pedersen, N. L. & McClearn, G. E. (1992). The origins of individual differences in occupational status and educational level. *Acta Sociologica, 35*, 13-31.
Lichtenstein, P., Tuvblad, C., Larsson, H. & Carlström, E. (in press). The Swedish Twin study of CHild and Adolescent Development - the TCHAD-study. *Twin Research and Human Genetics*.
Lilly, R. J., Cullen, F. T. & Ball, R. A. (1995). *Criminological Theory. Context and Consequences.* Newbury Park: SAGE.
Lipsey, M. W. & Derzon, J. H.(1998).Predictors of violence or serious delinquency in adolescence and early adulthood. In (Eds, Loeber, R. &Farrington, D. P.), *Serious and violent juvenile offenders* (pp. 86-105). CA: Sage: Thousand Oaks.
Loeber, R., Burke, J. D., Lahey, B. B., Winters, A. & Zera, M. (2000). Oppositional defiant and conduct disorder: a review of the past 10 years, part I. *Journal of the American Academy Child and Adolescent Psychiatry, 39*, 1468-1484.

Loeber, R. & Coie, J.(2001).Continuities and discontinuities of development, with particular emphasis on emotional and cognitive components of disruptive behaviour. In (Eds, Hill, J. &Maughan, B.), *Conduct disorder in childhood and adolescence* (pp. 379-407). Cambridge, UK: Cambridge University Press.

Loeber, R. & Dishion, T. J. (1983). Early predictors of male adolescent delinquency: A review. *Psychological Bulletin, 94*, 68-99.

Loeber, R. & Farrington, D. P. (1998). *Serious and violent juvenile offenders. Risk factors and successful interventions.* Thousands Oaks, California: SAGE Publications, Inc.

Loeber, R. & Farrington, D. P. (2000). Young children who commit crime: Epidemiology, development, origins, risk factors, early interventions, and policy implications. *Developmental Psychology, 12*, 737-762.

Loeber, R., Farrington, D. P. & Waschbusch, A.(1998).Serious and Violent Juvenile offenders. In (Eds, Loeber, R. & Farrington, D. P.), *Serious and violent juvenile offenders. Risk factors and successful interventions.* Thousands Oaks, California: SAGE Publications, Inc.

Loeber, R. & Hay, D. (1997). Key issues in the development of aggression and violence from childhood to early adulthood. *Annual Review of Psychology, 48*, 371-410.

Lynam, D. R. (2000). The Interaction Between Impulsivity and Neighbourhood Context on Offending: The Effects of Impulsivity Are Stronger in Poorer Neighbourhoods. *Journal of Abnormal Psychology, 109*, 563-574.

Lynam, D. R. & Gudonis, L. (2005). The development of psychopathy. *Annual review of clinical psychology, 1*, 381-407.

Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., Eaves, L. J. & Tsuang, M. T. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry, 52*, 906-915.

Maes, H. H. M., Neale, M. C., Kendler, K., Hewitt, J. K., Silberg, J. L., Foley, D. L., Meyer, J. M., Rutter, M., Simonoff. E., Pickles, A. & Eaves, L. J. (1998). Assortative mating for major psychiatric diagnosis in two population-based samples. *Psychological Medicine, 28*, 1389-1401.

Marmorstein, N. R. & Iacono, W. G. (2005). Longitudinal follow-up of adolescents with late-onset antisocial behavior: a pathological yet overlooked group. *Journal of American Academy of Child and Adolescent Psychiatry, 44*, 1284-1291.

Martin, N., Boomsma, D. & Machin, G. (1997). A twin-pronged attack on complex traits. *Nature Genetics, 17*, 387-92.

Mason, D. A. & Frick, P. J. (1994). The heritability of antisocial behavior: A meta-analysis of twin and adoption studies. *Journal of Psychopathology and Behavioral Assessment, 16*, 301-323.

Mather, K. & Jinks, J. L. (1982). *Biometrical Genetics.* New York: NY: Chapman & Hall.

Maughan, B., Rowe, R., Messer, J., Goodman, R. & Meltzer, H. (2004). Conduct Disorder and Oppositional Defiant Disorder in a national sample: developmental epidemiology. *Journal of Child Psychology and Psychiatry, 45*, 609-621.

McBurnett, K., Lahey, B. B., Rathouz, P. J. & Loeber, R. (2000). Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. *Archives of General Psychiatry, 57*, 38-43.

McCrae, R. R. & Costa, P. T. (1995). Trait explanations in personality psychology. *European Journal of personality, 9*, 231-252.

McGue, M., Iacono, W. G. & Krueger, R. (2006). The association of early adolescent problem behavior and adult psychopathology: a multivariate behavioral genetic perspective. *Behavior Genetics, 36*, 591-602.

McGuffin, P. & Thapar, A. (1992). The genetics of personality disorder. *British Journal of Psychiatry, 160*, 12-23.

Mednick, S. A., Gabrielli, W. F. & Hutchings, B. (1984). Genetic influence in criminal convictions: evidence from an adoption cohort. *Science, 224*, 891-894.

Middeldorp, C. M., Cath, D. C., Van Dyck, R. & Boomsma, D. I. (2005). The co-morbidity of anxiety and depression in the perspective of genetic epidemiology. A review of twin and family studies. *Psychological Medicine, 35*, 611-624.

Miles, D. R. & Carey, G. (1997). Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology, 72*, 207-217.

Miles, D. R., Van den Bree, M. & Pickens, R. W. (2002). Sex differences in shared genetic and environmental influences between conduct disorder symptoms and marijuana use in adolescents. *American Journal of Medical Genetics (Neuropsychiatric Genetics), 114*, 159-168.

Moffitt, T. E. (1993a). Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychological Review, 100*, 674-701.

Moffitt, T. E. (1993b). The neuropsychology of conduct disorder. *Developmental Psychology, 5*, 135-151.

Moffitt, T. E. (2005a). Genetic and environmental influences on antisocial behaviors: evidence from behavioral-genetic research. *Advances in Genetics, 55,* 41-99.

Moffitt, T. E. (2005b). The new look of behavioral genetics in developmental psychopathology: gene-environment interplay in antisocial behaviors. *Psychological Bulletin, 131*, 533-554.

Moffitt, T. E. & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescent-limited antisocial pathways among males and females. *Development and Psychopathology, 13*, 355-375.

Moffitt, T. E., Caspi, A., Rutter, M. & Silva, P. (2001). *Sex differences in antisocial behaviour: conduct disorder, delinquency and violence in the Dunedin Longitudinal Study.* Cambridge: Cambridge University Press.

Moffitt, T. E. & E-Risk Study Team. (2002). Teen-aged mothers in contemporary Britain. *Journal of Child Psychology and Psychiatry, 43*, 727-742.

Moffitt, T. E., Silva, P. A., Lynam, D. R. & Henry, B.(1994).Self-Reported Delinquency at age 18: New Zealand's Dunedin Multidisciplinary Health and Development Study. In (Eds, Junger-Tas, J., Terlouw, G.-J. &Klein, M. W.), *Delinquent Behavior Among Young People in the Western World* Amsterdam: Kugler Publications.

Moilanen, I., Linna, S-L., Kumpulainen, K., Tamminen, K., Piha, J. & Almqvist, F. (1999). Are twins' behavioural7emotional problems different from singletons'? *European Child and Adolescent psychiatry, 8*, 62-67.

Moore, T. M., Scarpa, A. & Raine, A. (2002). A meta-analysis of serotonin metabolite 5-HIAA and antisocial behavior. *Aggressive Behavior, 28*, 299-316.

Mustanski, B. S., Viken, R. J., Kaprio, J., Pulkkinen, L. & Rose, R. J. (2004). Genetic and environmental influences on pubertal development: longitudinal data from Finnish twins ages 11 and 14. *Developmental Psychology, 40*, 1188-1198.

Nadder, T. S., Rutter, M., Silberg, J., Maes, H. & Eaves, L. (2002). Genetic effects on the variation and covariation of attention deficit-hyperactivity disorder (ADHD) and oppositional-defiant disorder/conduct disorder (ODD/CD) symptomatologies across informant and occasion of measurement. *Psychological Medicine, 32*, 39-53.

Narusyte, J., Andershed, A.-K., Neiderhiser, J. M. & Lichtenstein, P. (in press). Aggression as a mediator of genetic contributions to the association between negative parent-child relationships and adolescent antisocial behavior. *European Child and Adolescent Psychiatry*

Neale, M. C. (1997). *Mx: Statistical modeling.* Richmond, VA: Department of Psychiatry, Medical College of Virginia.

Neale, M. C. & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families.* Dordrecht, The Netherlands: Kluwer Academic Publications.

Neale, M. C. & Martin, N. G. (1989). The effects of age, sex, and genotype on self-report drunkenness following a challenge dose of alcohol. *Behavior Genetics, 19*, 63-78.

Neiderhiser, J. M., Reiss, D., Pedersen, N. L., Lichtenstein, P., Spotts, E. L., Hansson, K., Cederblad, M. & Elthammar, O. (2004). Genetic and environmental

influences on mothering of adolescents: a comparison of two samples. *Developmental Psychology, 40*, 335-351.
Newman, D. L., Moffitt, T. E., Caspi, A., Magdol, L., Silva, P. A. & Stanton, W. R. (1996). Psychiatric disorder in a birth cohort of young adults: prevalence, comorbidity, clinical significance, and new case incidence from ages 11 to 21. *Journal of Consulting & Clinical Psychology, 64*, 552-562.
Nigg, J. T. & Huang-Pollock, C. L.(2003).An early-onset model of the role of executive functions and intelligence in conduct disorder/delinquency. In (Eds, Lahey, B. B., Moffitt, T. E. &Caspi, A.), *Causes of conduct disorder and juvenile delinquency* New York: The Guilford University Press.
O'Brien, R. M. (1985). *Crime and Victimization Data.* Beverly Hills CA.: Sage.
O'Connor, T. G., Deater-Deckard, K., Fulker, D., Rutter, M. & Plomin, R. (1998a). Genotype-environment correlations in late childhood and early adolescence: antisocial behavioral problems and coercive parenting. *Developmental Psychology, 34*, 970-981.
O'Connor, T. G., McGuire, S., Reiss, D., Hetherington, E. M. & Plomin, R. (1998b). Co-occurrence of depressive symptoms and antisocial behavior in adolescence: a common genetic liability. *Journal of Abnormal Psychology, 98*, 27-37.
Ortiz, J. & Raine, A. (2004). Heart rate level and antisocial behavior in children and adolescents: A meta-analysis. *Journal of American Academy of Child and Adolescent Psychiatry, 43*, 154-162.
Petersen, A. C., Tobin-Richards, M. & Boxer, A. (1983). Puberty: its measurement and its meaning. *Journal of early adolescence, 3*.
Plomin, R., DeFries, J. C., McClearn, G. E. & McGuffin, P. (2001). *Behavioral Genetics.* United States of America: Worth Publisher.
Plomin, R., Nitz, K. & Rowe, D. C.(1990).Behavioral genetics and aggressive behavior in childhood. In (Eds, Lewis, M. &Miller, S. M.), *Handbook of developmental psychopathology* (pp. 119-133). New York: Plenum.
Popma, A. & Raine, A. (2006). Will future forensic assessment be neurobiologic? *Journal of the American Academy Child and Adolescent Psychiatry, 15*, 429-444.
Posthuma, D., Beem, A. L., de Geus, E. J. C., van Baal, C. M., von Hjelmborg, J. B., Iachine, I. & Boomsma, D. I. (2003). Theory and practice in quantitative genetics. *Twin Research and Human Genetics, 6*, 361-376.
Posthuma, D. & Boomsma, D. (2005). Mx script library: structural equation modeling scripts for twin and family data. *Behavior Genetics, 35*, 499-505.
Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research and Human Genetics, 5*, 572-576.
Raine, A. (1993). *The psychopathology of crime, criminal behavior as a clinical disorder.* London: Academic Press Inc.
Raine, A. (2002a). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry, 43*, 417-434.
Raine, A. (2002b). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry, 43*, 417-434.
Raine, A. (2002c). Biosocial Studies of Antisocial and Violent Behavior in Children and Adults: A Review. *Journal of Abnormal Child Psychology, 30*, 311-326.
Raine, A., Brennan, P. A. & Mednick, S. A. (1997). Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: specificity to serious, early-onset violence. *American Journal of Psychiatry, 154*, 1265-1271.
Reynolds, C. R. & Richmond, B. O. (1985). *Revised Children's Manifest Anxiety Scale. RCMAS manual.* Los Angeles: Western Psychological Services.
Rhee, S. H. & Waldman, I. D. (2002). Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies. *Psychological Bulletin, 128*, 490-529.

Rietveld, M. J., Hudziak, J. J., Bartels, M., van Beijsterveldt, C. E. & Boomsma, D. I. (2003a). Heritability of attention problems in children: I. cross-sectional results from a study of twins, age 3-12 years. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics), 117*, 102-113.

Rietveld, M. J. H., Posthuma, D., Dolan, C. V. & Boomsma, D. I. (2003b). ADHD: Sibling interaction or dominance: an evaluation of statistical power. *Behavior Genetics, 33*, 247-255.

Ring, J. (1999). *Hem och skola, kamrater och brott [Home and school, peers and delinquency]*. Edsbruk, Sweden: Akademitryck.

Ring, J. (2005) The Swedish National Council for Crime Prevention [brottsförebyggande rådet, BRÅ], Stockholm.

Robins, L. (1978). Sturdy childhood predictors of adult antisocial behaviour: replications from longitudinal studies. *Psychological Medicine, 8*, 611-22.

Rose, R. J., Dick, D. M., Viken, R. J. & Kaprio, J. (2001). Gene-environment interaction in patterns of adolescent drinking: regional residency moderates longitudinal influences on alcohol use. *Alcoholism: Clinical and Experimental Research, 25*, 637-43.

Rose, R. J., Dick, D. M., Viken, R. J., Pulkkinen, L. & Kaprio, J. (2004). Genetic and environmental effects on conduct disorder and alcohol dependence symptoms and their covariation at age 14. *Alcoholism: Clinical and Experimental Research, 28*, 1541-1548.

Rowe, D. C. (1983). Biometrical genetic models of self-reported delinquent behavior: a twin study. *Behavior Genetics, 13*, 473-89.

Rowe, D. C. (1994). *The limits of family influence: genes, experience, and behavior.* NY: Guilford Press.

Rowe, D. C.(2003).Assessing genotype-environment interactions and correlations in the postgenomic era. In (Eds, Plomin, R., DeFries, J. C., Craig, I. W. &McGuffin, P.), *Behavioral Genetics in the Postgenomic Era* (pp. 71-99). Washington, D.C.: American Psychological Association.

Rowe, D. C., Almeida, D. M. & Jacobson, K. C. (1999). School context and genetic influences on aggression in adolescence. *Psychological Science, 10*, 277-280.

Rutter, M. (1997). Comorbidity: concepts, claims and choices. *Criminal Behaviour and Mental Health, 7*, 265-285.

Rutter, M.(2003).Crucial paths from risk indicator to causal mechanism. In (Eds, Lahey, B. B., Moffitt, T. E. &Caspi, A.), *Causes of conduct disorder and juvenile delinquency* New York: The Guilford University Press.

Rutter, M. (2006). *Genes and behavior. Nature-nurture interplay explained.* Oxford, UK: Blackwell Publishing Ltd.

Rutter, M., Caspi, A. & Moffitt, T. E. (2003). Using sex differences in psychopathology to study causal mechanisms: unifying issues and research strategies. *Journal of Child Psychology and Psychiatry, 44*, 1092-1115.

Rutter, M., Giller, H. & Hagell, A. (1998). *Antisocial Behavior by Young People.* Cambridge, UK: Cambridge University Press.

Rutter, M., Kim-Cohen, J. & Maughan, B. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry, 47*, 276-295.

Rutter, M. & Redshaw, J. (1991). Annotation: growing up as a twin: twin-singleton differences in psychological development. *Journal of Child Psychology and Psychiatry, 32*, 885-895.

Salekin, R. T., Rogers, R. & Sewell, K. W. (1996). A review and meta-analysis of the psychopathy checklist and psychopathy checklist revised: predictive validity of dangerousness. *Clinical Psychology Science and Practice, 3*, 203-215.

Sampson, R. J., Raudenbush, S. W. & Earls, F. (1997). Neighbourhoods and violent crime: A multilevel study of collective efficacy. *Science, 277*, 918-924.

Scarr, S. & Carter-Saltzman, L. (1979). Twin method: defense of a critical assumption. *Behavior Genetics, 9*, 527-541.

Scarr, S. & McCartney, K. (1983). How people make their own environments: A theory of genotype-environment effects. *Child Development, 54*, 425-435.

Scott, S., Knapp, M., Henderson, J. & Maughan, B. (2001). Financial cost of social exclusion: follow up study of antisocial children into adulthood. *British Medical Journal, 323*, 1-5.

Scourfield, J., Martin, N., Eley, T. C. & McGuffin, P. (2004a). The genetic relationship between social cognition and conduct problems. *Behavior Genetics, 34*, 377-383.

Scourfield, J., Van den Bree, M., Martin, N. & McGuffin, P. (2004b). Conduct problems in children and adolescents A twin study. *Archives of General Psychiatry, 61*, 489-496.

Serbin, L. A. & Karp, J. (2004). The intergenerational transfer of psychosocial risk: mediators of vulnerability and resilience. *Annual Review of Psychology, 55*, 333-363.

Silberg, J., Rutter, M., Meyer, J., Maes, H., Hewitt, J. K., Simonoff, E., Pickles, A., Loeber, M. & Eaves, L. (1996). Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *Journal of Child Psychology and Psychiatry, 37*, 803-816.

Silverthorn, P. & Frick, P. J. (1999). Development pathways to antisocial behavior: The delayed-onset pathway in girls. *Development and Psychopathology, 11*, 101-126.

Simonoff, E., Elander, J., Homshaw, J., Pickles, A., Murray, R. & Rutter, M. (2004). Predictors of antisocial personality. *British Journal of Psychiatry, 184*, 118-127.

Simonoff, E., Pickles, A., Meyer, J., Silberg, J., Maes, H., Loeber, M., Rutter, M., Hewitt, J. K. & Eaves, L. (1997). The Virginia twin study of adolescent behavioral development. *Archives of General Psychiatry, 54*, 801-808.

Skeem, J. L. & Cauffman, E. (2003). Views of the downward extension: Comparing the Youth Version of the Psychopathy Checklist with the Youth Psychopathic traits Inventory. *Behavioral Sciences and the Law, 21*, 737-770.

Slutske, W. S. (2001). The genetics of antisocial behavior. *Current Psychiatry Report, 3*, 158-162.

Slutske, W. S., Dinwiddie, S. H., Heath, A., Bucholz, K. K., Dunne, M. P., Statham, D. J. & Martin, N. G. (1997a). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2,682 adult twin pairs. *Journal of Abnormal Psychology, 106*, 266-279.

Slutske, W. S., Eisen, S., Xian, H., True, W. R., Lyons, M. J., Goldberg, J. & Tsuang, M. (2001). A twin study of the association between pathological gambling and antisocial personality disorder. *Journal of Abnormal Psychology, 110*, 297-308.

Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A., Bucholz, K. K., Dunne, M. P., Statham, D. J. & Martin, N. G. (1998). Common genetic risk factors for conduct disorder and alcohol dependence. *Journal of Abnormal Psychology, 107*, 363-374.

Slutske, W. S., Heath, A. C., Madden, P. A., Bucholz, K. K., Statham, D. J. & Martin, N. G. (1997b). Personality and the gentic risk for alcohol dependence. *Journal of Abnormal Psychology, 111*, 124-133.

Slutske, W. S., Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J. & Tsuang, M. (1997c). Testing a developmental taxonomy of antisocial behavior [conference presentation]. *Behavior Genetics, 27*, 606.

Smith, D. A. & Visher, C. A. (1980). Sex and involvement in deviance/crime: A quantitative review of the empirical literature. *American Sociological Review, 45*, 691-701.

Stanger, C., Achenbach, T. M. & Verhulst, F. C. (1997). Accelerated longitudinal comparisons of aggressive versus delinquent syndromes. *Development and Psychopathology, 9*, 43-58.

Statistics Sweden. (1995). *Reports on statistical co-ordination 1982, Swedish socio-economic classification.* Stockholm: Statistic Sweden.

Stattin, H. & Magnusson, D. (1990). *Pubertal maturation in female development.* Hillsdale, New Jersey: Lawrence Erlaum Associates, Inc.

Stouthamer-Loeber, M. & Loeber, M. (2002). Lost opportunities for intervention: undetected markers for the development of serious juvenile delinquency. *Criminal Behaviour and Mental Health, 12*, 69-82.

Stouthamer-Loeber, M., Loeber, R., Wei, E., Farrington, D. P. & Wikstroem, P.-O. H. (2002). Risk and promotive effects in the explanation of persistent serious delinquency in boys. *Journal of Consulting & Clinical Psychology, 70*, 111-123.

Taylor, A., Loney, B. R., Bobadilla, L., Iacono, W. G. & McGue, M. (2003). Genetic and environmental influences on psychopathy trait dimensions in a community sample of male twins. *Journal of Abnormal Child Psychology, 31*, 633-645.

Taylor, J., Iacono, W. G. & McGue, M. (2000a). Evidence for a genetic etiology of early-onset delinquency. *Journal of Abnormal Psychology, 109*, 634-643.

Taylor, J., McGue, M. & Iacono, W. G. (2000b). Sex differences, assortative mating, and cultural transmission effects on adolescent delinquency: a twin family study. *Journal of Child Psychology and Psychiatry, 41*, 433-440.

Thapar, A., Harrington, R. & McGuffin, P. (2001). Examining the comorbidity of ADHD-related behaviours and conduct problems using a twin study design. *British Journal of Psychiatry, 179*, 224-229.

The Swedish National Council for Crime Prevention (2004) (Ed, [brottsförebyggande rådet, B.).

Tolan, P. H. & Gorman-Smith, D.(1998).Development of serious and violent offending careers. In (Eds, Loeber, R. &Farrington, D. P.), *Serious and violent juvenile offenders. Risk factors and successful interventions* (pp. 68-85). SAGE Publications.

Tremblay, R. E., Pihl, R. O., Vitaro, F. & Dobkin, P. L. (1994). Predicting early onset of male antisocial behavior from preschool behavior. *Archives of General Psychiatry, 51*, 732-739.

Trzesniewski, K. H., Moffitt, T. E., Caspi, A., Taylor, A. & Maughan, B. (2006). Revisiting the association between reading achievement and antisocial behavior: new evidence of an environmental explanation from a twin study. *Child Development, 77*, 72-88.

Tuvblad, C., Grann, M. & Lichtenstein, P. (2006). Heritability for adolescent antisocial behavior differs with socioeconomic status: Gene-environment interaction. *Journal of Child Psychology and Psychiatry, 47*, 734-743.

Tuvblad, C., Grann, M., Sarnecki, J. & Lichtenstein, P. (2005). Pubertal timing and antisocial behavior in early and late adolescence. A longitudinal twin study [conference presentation]. *Behavior Genetics, 35*, 823.

Walters, G. D. (2003). Predicting institutional adjustment and recidivism with the psychopathy checklist factor scores: A meta-analysis. *Law and Human Behavior, 27*, 541-558.

van den Oord, E. J. C. G., Koot, H. M., Boomsma, D. I., Verhulst, F. C. & Orlebeke, J. F. (1995). A twin-singleton comparison of problem behaviour in 2-3-year-olds. *Journal of Child Psychology and Psychiatry, 36*, 449-458.

van Lier, P. A., Vitaro, F., Wanner, B., Vuijk, P. & Crijnen, A. A. (2005). Gender differences in developmental links among antisocial behavior, friends' antisocial behavior, and peer rejection in childhood: results from two cultures. *Child Development, 76*, 841-855.

Weis, J. G.(1986).Issues in the measurement in criminal careers. In (Eds, Blumstein, A., Cohen, J., Roth, J. A. &Laub, J. H.), *Criminal careers and 'Career Criminals'* Washington D.C.: National Academy Press.

Vermeiren, R. (2003). Psychopathology and delinquency in adolescents: a descriptive and developmental perspective. *Clinical Psychology Review, 23*, 277-318.

White, H. R., Xie, M., Thompson, W., Loeber, R. & Stouthamer-Loeber, M. (2001). Psychopathology as a predictor of adolescent drug use trajectories. *Psychology of Addictive Behaviors, 15*, 210-218.

WHO (2002). *World Health Organization: World Report on Violence and Health.* Geneva: WHO.

Wichers, M. C., Purcell, S., Danckaerts, M., Derom, C., Derom, R., Vlietinck, R. & Van Os, J. (2002). Prenatal life and post-natal psychopathology: evidence for negative gene-birth weight interaction. *Psychological Medicine, 32*, 1165-1174.

Viding, E., Blair, J. R., Moffitt, T. E. & Plomin, R. (2005). Evidence of substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry, 46*, 592-597.

Vierikko, E., Pulkkinen, L., Kaprio, J. & Rose, R. J. (2004). Genetic and environmental influences on the relationship between aggression and hyperactivity-impulsivity as rated by teachers and parents. *Twin Research and Human Genetics, 7*, 261-274.

Vierikko, E., Pulkkinen, L., Kaprio, J., Viken, R. J. & Rose, R. J. (2003). Sex differences in genetic and environmental effects on aggression. *Aggressive Behavior, 29*, 55-68.

Vitacco, M. J., Rogers, R. & Neumann, C. S. (2003). The antisocial process screening device: an examination of its construct and criterion-related validity. *Assessment, 10*, 143-150.

Xian, H., Scherrer, J. F., Eisen, S. A., True, W. R., Heath, A. C., Goldberg, J., Lyons, M. J. & Tsuang, M. T. (2000). Self-Reported zygosity and the equal-environments assumption for psychiatric disorders in the Vietnam Era Twin Registry. *Behavior Genetics, 30*, 303-310.

Young, S. E., Stallings, M. C., Corley, R. P., Krauter, K. S. & Hewitt, J. K. (2000). Genetic and environmental influences on behavioral disinhibition. *American Journal of Medical Genetics (Neuropsychiatric Genetics), 96*, 684-695.

10 APPENDICES: PAPERS I-IV