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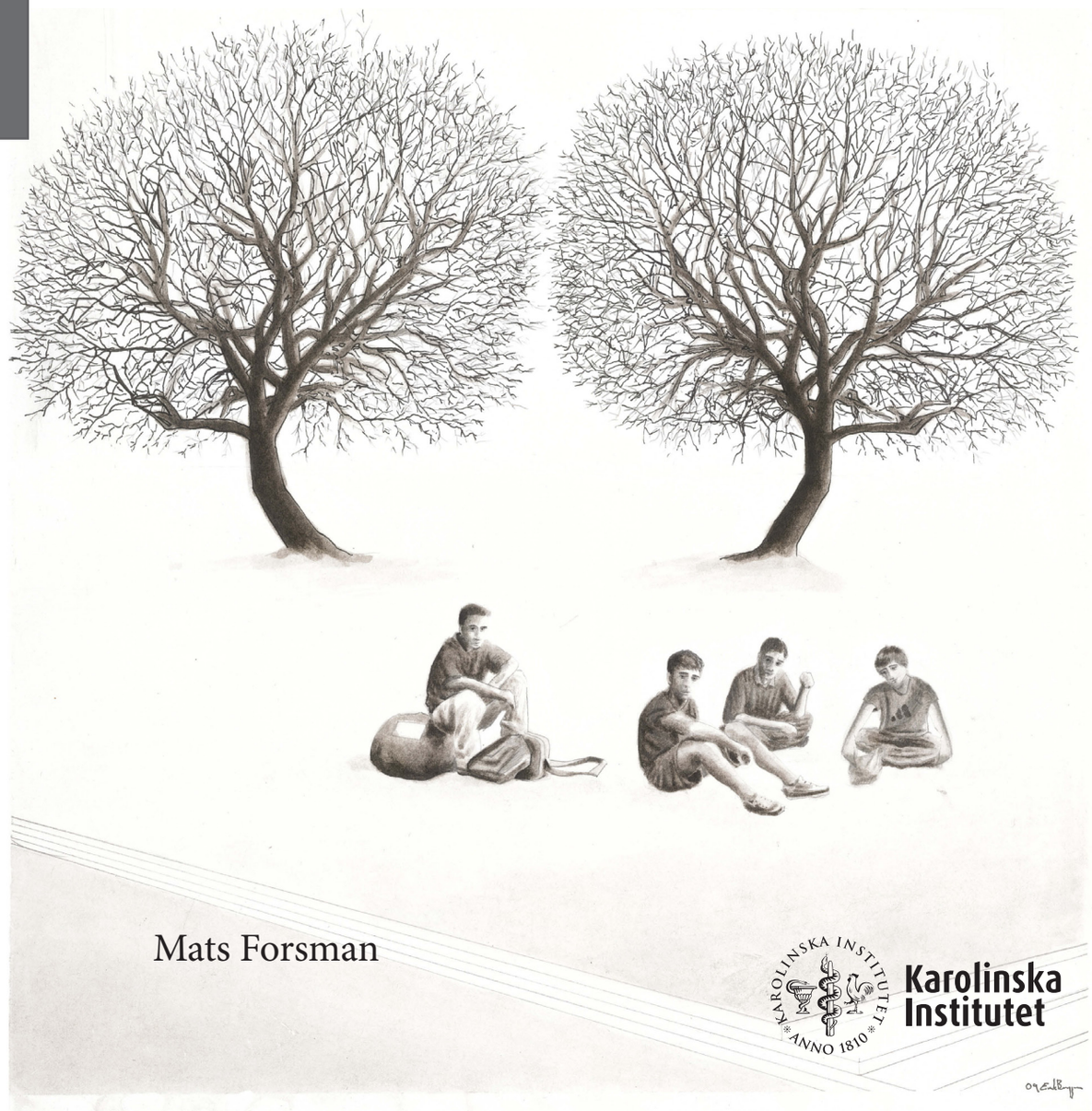

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PSYCHOPATHIC PERSONALITY IN ADOLESCENCE – GENETIC AND ENVIRONMENTAL INFLUENCES

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ABSTRACT

Psychopathy, or psychopathic personality, is a personality disorder characterized by a constellation of deviant interpersonal, affective, and behavioral dimensions. It has consistently been shown that the psychopathic personality can be used to understand the development of antisocial behavior in adolescents. Less research has been devoted to exploring the underlying etiology of psychopathic personality. There has also been a lack of genetically sensitive longitudinal studies that have examined the developmental associations between psychopathic personality and putative psychopathological correlates. This thesis has used longitudinal and multivariate twin data to clarify both the etiology of psychopathic personality and its association with other psychopathological problems.

The data used in this thesis comes from the Twin Study of Child and Adolescent Development, a prospective longitudinal study with data collected when the twins were 8-9, 13-14, 16-17, and 19-20 years old.

Study I in this thesis showed that genetic factors contributed substantially to the stability of the higher-order psychopathic personality factor, whereas environmental factors were of little importance. We also found specific genetic stability in the Callous/unemotional (affective) and Impulsive/irresponsible (behavioral) dimensions. Study II showed that persistent externalizing problems in childhood was associated with both psychopathic personality and antisocial behavior in adolescence. Twin analyses showed that genetic factors explained the association between persistent externalizing problems and psychopathic personality, whereas shared environmental factors explained the association between persistent externalizing problems and antisocial behavior. In Study III it was shown that that psychopathic personality in adolescence predicted antisocial behavior in adulthood via genetic effects, but not the other way around. However, bidirectional effects were found when a measure of persistent antisocial behavior was used. In Study IV, the higher-order psychopathic personality factor was associated with externalizing problems due to genetic factors, whereas the specific variances in the Callous/unemotional and Impulsive/irresponsible dimensions were divergently related to psychopathological problems.

Findings in this thesis highlights the importance of both general and specific etiologic factors in understanding the stability and change of psychopathic personality, as well as for identification of risk and protective factors in the development of externalizing and internalizing problems. Future attempts to identify specific genes could therefore focus on the general, but also the specific variance of the psychopathic personality constellation. Second, this thesis has added to previous research by showing that adolescent psychopathic predicts adult antisocial behavior via genetic effects, but also that persistent antisocial behavior predicts adult psychopathic personality. Researchers are encouraged to include measures of psychopathic personality in younger samples and at several assessment points to clarify the nature of the association between psychopathic personality and antisocial behavior in more detail.

LIST OF PUBLICATIONS

This thesis is based on the following four studies. They will be referred to in the text by their Roman numerals (I-IV).

I. Forsman, M., Lichtenstein, P., Andershed, H., & Larsson, H. (2008). Genetic effects explain the stability of psychopathic personality from mid- to late adolescence. *Journal of Abnormal Psychology, 117*, 606-617.

II. Forsman, M., Larsson, H., Andershed, H., & Lichtenstein, P. (2007). The association between persistent disruptive childhood behavior and the psychopathic personality constellation in adolescence: A twin study. *British Journal of Developmental Psychology, 25,* 383–398.

III. Forsman, M., Lichtenstein, P., Andershed, H., & Larsson, H. (2009). A longitudinal twin study of the direction of effects between psychopathic personality and antisocial behavior. *Journal of Child Psychology and Psychiatry*. [Epub ahead of print].

IV. Forsman, M., Lichtenstein, P., Andershed, H., & Larsson, H. (2009). Psychopathic personality in adolescence: Genetic and environmental overlap with externalizing and internalizing problems. Manuscript submitted for publication.

CONTENTS

LIST OF ABBREVIATIONS

TCHAD	Twin Study of Child and Adolescent Development
MZ	Monozygotic
DZ	Dizygotic
YPI	Youth Psychopathic traits Inventory
G/M	Grandiose/manipulative
C/U	Callous/unemotional
I/I	Impulsive/irresponsible
FD	Fearless/Dominance
IA	Impulsivity/Antisocial
PCL-R	Psychopathy Checklist - Revised
ADHD	Attention Deficit Hyperactivity Disorder
CBCL	Child Behavior Checklist
ABCL	Adult Behavior Checklist
YSR	Youth Self Report
ASR	Adult Self Report

INTRODUCTION

Antisocial behavior (e.g., aggression, violence) in youth not only cause substantial physical and mental harm to victims, but also implies high public expenditure. Antisocial behavior is heterogeneous with respect to age of onset, severity, developmental course, and causes. The need to parse the heterogeneity in the group of youths with antisocial behavior has therefore been appreciated for years. Recently, researchers have used the psychopathic personality constellation in an attempt to understand the development of severe and chronic patterns of antisocial behavior (Lyman & Gudonis, 2006). However, while the association between psychopathic personality and antisocial behavior is relatively well established, knowledge of the underlying etiology of the psychopathic personality constellation is limited. In addition, there has been a lack of genetically sensitive longitudinal studies that have examined the developmental associations between psychopathic personality and putative psychopathological correlates. Information from such studies may provide us with deeper levels of knowledge regarding the developmental origins of psychopathy and antisocial behavior and to advance our understanding of the best methods for prevention and intervention. This thesis used prospective longitudinal twin data and examined how genetic and environmental factors contribute to: (I) the stability and change of psychopathic personality; (II) the association between childhood disruptive behavior and psychopathic personality; (III) the direction of effects between psychopathic personality and antisocial behavior; and (IV) the association between psychopathic personality and externalizing and internalizing psychopathology.

BACKGROUND

PSYCHOPATHIC PERSONALITY IN ADULTHOOD

In contemporary research and clinical practice, psychopathy is most commonly assessed with the Psychopathy Checklist-Revised (PCL-R, Hare, 2003), which is a 20-item clinical rating scale (rated by a psychologist or other professional). The scores are used to predict risk for criminal re-offence and probability of rehabilitation. There is an ongoing debate about how many dimensions that should be included in this personality constellation as measured by the PCL-R (Cooke & Michie, 2006; Hare & Neumann, 2006). Factor analyses have shown that the PCL-R items can be described with three or four dimensions or factors. The three-dimensional model describes psychopathy as a constellation of interpersonal (e.g., grandiosity, egocentricity), affective (e.g., remorselessness, callousness), and behavioral dimensions (e.g., impulsiveness, irresponsibility) (Cooke & Michie, 2006). The four-dimensional model includes an additional dimension measuring antisocial behavior (e.g., revocation of conditional release, criminal versatility) (Hare & Neumann, 2006). Regardless of the exact number of dimensions, research has found support for a hierarchical model, implying a substantial influence of a higher order general factor (see Figure 1) (Cooke & Michie, 2006; Hare & Neumann, 2006; Patrick, Hicks, Nichol, & Krueger, 2007). The higher-order factor (i.e., psychopathy or psychopathic personality) captures the variance that is shared among the psychopathic personality dimensions. However, the hierarchical model also specifies variance that is unique for each dimension and not shared with the higher-order factor (represented by the arrows at the bottom of Figure 1).

Compared with other offenders, adult offenders with a diagnosis of psychopathy generally are more frequently violent (Hare, 2003); are more likely to recidivate (Douglas, Vincent, & Edens, 2006); and response poorly to traditional treatment programs (Harris & Rice, 2006). In addition, adult psychopaths more often exhibit behavioral problems early in life (e.g., Abramowitz, Kosson, & Seidenberg, 2004). Researchers have therefore started to investigate if psychopathic personality can be meaningfully assessed in youths.

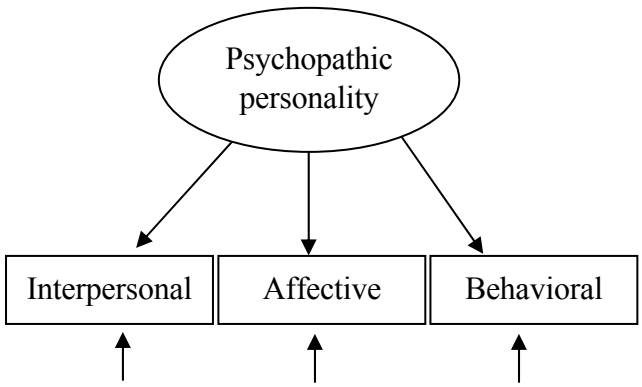

Figure 1. A three-dimensional hierarchical model of psychopathy

PSYCHOPATHIC PERSONALITY IN ADOLESCENCE

All current concepts of personality disorders, including psychopathy, are based on the assumption that the features have their origins in childhood. Thus, it is believed that these features can be measured in adolescence (Rutter, 2005). It is important to study the manifestations of psychopathic personality in youths in order to identify the potential precursors to adult psychopathy and to understand the development of severe antisocial behavior. It may also be that psychopathic personality in adolescence is more malleable than in adulthood allowing for more effective treatment efforts. Studies using both referred and non-referred adolescent samples indicate that the same kinds of features that are measurable in adults can also be assessed in adolescents and that they manifest in similar ways. That is, similar factor-structures have been identified as those typically observed in adult samples (Andershed, Kerr, Stattin, & Levander, 2002; Forth, Kosson, & Hare, 2003; Vitacco, Rogers, & Neumann, 2003) and there is also support for that psychopathic personality can be described with a hierarchical model in adolescence (Larsson, Andershed, Lichtenstein, 2006). In addition, it has consistently been shown that psychopathic personality is associated with antisocial behavior among young offenders as well as in the normal population (Andershed et al., 2002; Lynam & Gudonis, 2005). Thus, a growing body of research suggests that psychopathic personality in adolescence is a reliable, valid and meaningful construct.

GENETIC AND ENVIRONMENTAL INFLUENCES

The focus in this thesis is to gain knowledge of psychopathic personality in youth through behavioral genetic methods. Behavioral genetic methods are used to detect factors that make individuals in a population different from each other in a trait of interest, in this case psychopathic personality traits. Specifically, the twin design relies on studying twins raised in the same family environments. Monozygotic (MZ) twins share all of their genes, while dizygotic (DZ) twins share, on average, only about 50 percent of them. So, if a researcher compares the similarity between sets of MZ twins to the similarity between sets of DZ twins for a particular trait, then any excess similarity between the MZ twins should be due to genes rather than environment. Additionally, comparisons between MZ and DZ twin pairs makes it possible to estimate the importance of environmental influences that are shared between the twins (shared environment) and those environmental influences that are unique for each twin in a pair (non-shared environment). Researchers have used the twin design to try to disentangle the genetic and environmental influences for many different traits and behaviors (Plomin, DeFries, McClearn, & McGuffin, 2008).

Up to date, relatively few studies have studied the etiology of psychopathic personality. These studies have reported that approximately 40% to 70% of the variance in psychopathic personality is due to genetic effects (Larsson et al., 2006; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Viding, Blair, Moffitt, & Plomin, 2005; Waldman & Rhee, 2006). These twin studies have been important in clarifying the genetic and environmental contributions to psychopathic personality. However, as shown in the present thesis, behavior genetic methods can be used to address a number of more refined questions that go far beyond estimating the contribution of genetic and environmental factors for a trait.

First, prospective longitudinal data and recent advances in model fitting techniques allow behavior genetic researchers to ask how the effects of genetic and environmental factors unfold over time. For example, do the same genes and environments affect psychopathic personality over a period of life, or do different genes and environments affect psychopathic personality at different developmental stages? Second, instead of estimating genetic and environmental influence on the variance of one trait at a time, multivariate genetic analysis can be used to understand the etiology of the association between two or more traits. For example, a recent twin-study, using the same data as used in the present thesis, used the three-factor conceptualization of psychopathy and applied a hierarchical model to explore the importance of both general and specific etiological factors for the psychopathic personality construct (Larsson et al., 2006). Results revealed a strong genetic effect behind the higher-order psychopathic personality factor (63%), underpinned by the three psychopathic personality dimensions called the Grandiose/manipulative, the Callous/unemotional, and the Impulsive/irresponsible dimension. Shared environmental effects were of no importance, whereas non-shared environmental effects explained the remaining 37% of the variance. Over and above the effects of the higher-order factor, significant unique genetic influences were found in the Callous/unemotional (22%) and the Impulsive/irresponsible dimension (22%), but not in the Grandiose/manipulative dimension (Figure 2).

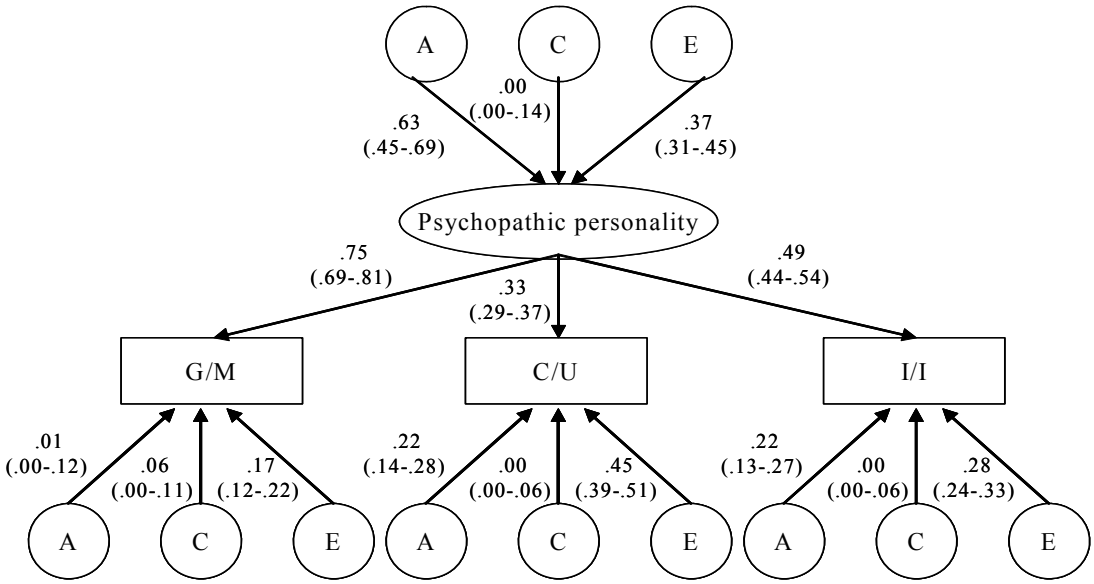

Figure 2. Squared path estimates for the full common pathway model, with 95% Confidence Intervals. G/M = Grandiose/manipulative dimension; C/U = Callous/unemotional dimension; I/I = Impulsive/irresponsible dimension. A = Genetic effects ; C = Shared environmental effects ; E = Non-shared environmental effects.

Likewise, one could examine whether the association between psychopathic personality and antisocial behavior is the result of a common underlying genetic liability or if the association is due to environmental factors? Third, the unique value of longitudinal data for addressing questions of "directionality" between two phenotypes has long been appreciated. For example, if psychopathic personality is a precursor of externalizing problems rather than the other way around, we expect psychopathic personality to

correlate with later externalizing problems more than we expect externalizing problems to correlate with later psychopathic personality. Such cross-lagged longitudinal designs are especially useful in combination with genetically informative data (e.g., longitudinal twin data) which allows one to examine the importance of genetic and environmental effects on the cross-lags.

Specifically, this thesis used longitudinal twin data between ages 8 and 20 years and a multivariate approach to examine the importance of genetic and environmental influences for: the stability and change of psychopathic personality between adolescence and adulthood (Study I); the association between childhood disruptive behavior and psychopathic personality in adolescence (Study II); the direction of effects between psychopathic personality and antisocial behavior between adolescence and adulthood (Study III); and the association between psychopathic personality and externalizing and internalizing psychopathology in adolescence (Study IV).

STABILITY AND CHANGE

The vast majority of research extending the psychopathic personality constellation to youth has been cross-sectional. As a result, there has been limited support for the predictive utility of psychopathic features prior to adulthood. Some researchers have hypothesized that several characteristics of adult psychopathy may be normative and temporary characteristics of adolescence and that, because of this, psychopathic personality may not be a valid or useful construct in this age group (Edens, Skeem, Cruise, & Cauffman, 2001; Seagrave & Grisso, 2002). This concern emphasizes the need for studies exploring the stability of psychopathic personality from adolescence to adulthood. If the psychopathic personality construct is to prove useful for adolescents, evidence is needed that it is at least relatively stable over time, up into adulthood.

In response to this research gap, recent longitudinal studies have examined the stability of psychopathic personality (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2006; Loney, Taylor, Butler, & Iacono, 2007; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007; Lynam, Charnigo, et al., 2007). These studies suggest that psychopathic personality is moderately to highly stable which is in line with normal personality (Roberts & DelVecchio 2000). Based on these findings, it has been suggested that it may be time to move beyond asking *whether* psychopathic personality is stable and to begin asking *why* psychopathic personality is stable (Lynam, Charnigo, et al., 2007).

Only one previous twin study has examined the importance of genetic and environmental contributions to the stability of psychopathic personality (Blonigen et al., 2006). Results showed that genetic effects explained approximately 60% of the stable variance in the Fearless/Dominance (FD) and Impulsivity/Antisocial (IA) factors. However, this study is built upon an uncorrelated model of psychopathy and the two factors were therefore analyzed separately. No previous study has used the hierarchical conceptualization to study stability and change of psychopathic personality between adolescence and adulthood. Thus, there is no knowledge of how genetic and environmental contribute to the temporal stability of the higher-order psychopathic personality factor. Likewise, the stability of the specific effects in the

Callous/unemotional and Impulsive/irresponsible dimensions identified by Larsson et al (2006) is also unknown.

CHILDHOOD RISK FACTORS

Early disruptive behavior such as externalizing problems (i.e., rule-breaking behavior and aggression) and Attention Deficit Hyperactivity Disorder (ADHD) symptoms occur with elevated prevalence not only among youth who grow up to exhibit adult criminality and substance abuse (Vitelli, 1998), but also in those later diagnosed with psychopathy (e.g., Abramowitz et al., 2004). Externalizing problems and ADHD symptoms in childhood may therefore represent independent risk factors for subsequent psychopathic personality. Others have suggested that the joint presence of externalizing problems and ADHD symptoms confers specific vulnerability to psychopathic personality (Lynam, 1996). This reasoning is based on findings showing that children with the presence of both externalizing problems and ADHD symptoms show frequent, severe, and varied forms of antisocial behavior (Loeber, Brinthaupt, & Green, 1990; Lynam, 1998) and show high levels of psychopathic personality traits (Lynam, 1998). However, if the joint presence of the two disruptive disorders confers unique risk for psychopathic personality, then studies should reveal not only main effects for externalizing problems and ADHD symptoms, but also an externalizing problems/ADHD symptoms interaction.

Individual differences in externalizing problems (Rhee & Waldman, 2002), ADHD symptoms (Rutter, Silberg, O'Connor, & Simonoff, 1999), and psychopathic personality (Larsson et al., 2006) are all largely influenced by genetic factors. One possibility is that the observed associations between childhood disruptive behavior problems and psychopathic personality are due to a common genetic vulnerability that is stable over time. However, in lack of genetically informative longitudinal studies, the etiology of these associations is yet poorly understood.

DIRECTION OF EFFECTS

A large number of studies suggest that psychopathy is predictive of future antisocial behavior problems (e.g., recidivism) among adult criminals (e.g., Walters, 2003), and it has been suggested that antisocial behavior should be viewed as a potential consequence of psychopathic personality (Cooke & Michie, 2001). However, few studies have examined this association during the critical period between adolescence and adulthood. In addition, one possibly overlooked alternative is that antisocial behavior also predicts subsequent levels of psychopathic personality. Psychopathic personality may in some individuals develop as a consequence of consistent involvement in antisocial behaviors, such as breaking rules and fighting with others. Some evidence of such an effect comes from studies showing that children who show early starting persistent forms of antisocial behavior are at higher risk for elevated levels of psychopathic personality in adulthood (Moffitt, Caspi, Dickson, Silva, Stanton, 1996; Moffitt, Caspi, Harrington, & Milne, 2002). Thus, development of psychopathic personality due to involvement in antisocial behavior may be especially relevant for early starting persistent forms of antisocial behavior (Moffitt, 2005).

The cross-lagged model (e.g., Burt, McGue, Krueger, & Iacono, 2005) is often used to clarify the phenotypic direction of effects between two phenotypes. This model is advantageous because it constrains all cross-age associations to take the form of phenotypic partial regression coefficients, thereby controlling for the association between the two phenotypes at time-point 1 when examining their effects on each other at time-point 2. With genetically informative data, one can also decompose the cross-lagged coefficients into their genetic and environmental components. No previous study has examined the genetic and environmental contribution to the direction of effects between psychopathic personality and antisocial behavior.

ASSOCIATION TO PSYCHOPATHOLOGY

It has repeatedly been shown that psychopathic personality is positively associated with externalizing problems when composite scores of psychopathic personality has been used (Hare, 2002, 2003; Lynam & Gudonis, 2005). The association between psychopathy and internalizing problems is less clear. Clinical descriptions and theories suggest that psychopathic personality is negatively associated with internalizing problems (Cleckley, 1976), whereas empirical investigations suggest that they are unrelated or even moderately positively related (Hare, 2003; Salekin, Neumann, Leistico, DiCicco, & Duros, 2004; Schmitt & Newman, 1999).

When specific variances have been studied (e.g., with partial correlations), the different dimensions of psychopathic personality show divergent associations to both externalizing and internalizing problems. The specific variance in the behavioral dimension of psychopathy show positive associations with both externalizing and internalizing problems. In contrast, the specific variances in the interpersonal and affective dimensions are unrelated or weakly positively associated with externalizing problems and negatively associated with internalizing problems (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005; Hicks & Patrick, 2004; Patrick, Hicks, Krueger, & Lang, 2005; Verona, Patrick, & Joiner, 2001). Thus, to understand the nature and complexity of the psychopathic personality construct and its relation to psychopathology, it seems reasonable to focus both on the general variance (i.e., covariance among the psychopathic personality dimensions) as well as the variance that is unique for each dimension.

AIMS

The overall aim in this thesis was to gain knowledge about the etiology of psychopathic personality and its association with other psychopathological problems.

Specifically, the following research questions were addressed:

Study I: How do genetic and environmental factors contribute to the stability and change of psychopathic personality between adolescence and adulthood?

Study II: Are persistent externalizing behavior and ADHD symptoms or the combination of the two associated with the psychopathic personality constellation in adolescence? If so, how do genetic and environmental factors contribute to the associations?

Study III: What is the direction of effects between psychopathic personality and antisocial behavior between adolescence and adulthood? How do genetic and environmental factors contribute to this association?

Study IV: How are the general (i.e., the higher-order psychopathic personality factor) and specific variances (i.e., unique variance in each dimension) of psychopathic personality phenotypically and etiologically related to externalizing and internalizing problems in adolescence?

METHODS

PARTICIPANTS

The data used in all of the studies in this thesis comes from the Twin Study of Child and Adolescent Development (TCHAD). TCHAD is a longitudinal study of all 1,480 twin pairs born in Sweden between May 1985 and December 1986. The twins and their parents have been asked to answer mailed questionnaires at childhood (age 8-9, parents only), throughout early (age 13-14) and late adolescence (age 16-17), into early adulthood (age 19-20). The response rate for the parent questionnaire was 75% (n = 1,109) at wave 1, 73% (n = 1,063) at wave 2, 74% (n = 1,067) at wave 3. In wave 4, both parents were approached separately, giving 1158 responses from at least one of the parents (mothers only: n=1061, fathers only: n=795). The response rate for the twin questionnaires was 78% (n = 2,263) at wave 2, 82% (n = 2,369) at wave 3, and 59% (n = 1,705) at wave 4.

REPRESENTAVINESS OF THE SAMPLE

Attrition rate

In the TCHAD study, many efforts have been made to examine bias due to attrition. For example, it has been shown that there are no significant differences in sex ratio, externalizing symptoms, or ADHD symptoms at wave 1 between responders and subjects lost to follow-up at wave 2 (Larsson, Larsson, & Lichtenstein, 2004; Tuvblad, Eley, & Lichtenstein, 2005). Subjects lost to follow up at wave 3 scored higher than the responders in hyperactivity/impulsivity at wave 2 (OR = 1.01, 95% CI 1.01-1.31), but did not significantly differ in antisocial behavior, family socioeconomic status or inattention (Larsson et al., 2006). Finally, it has been shown that subjects lost to follow up at wave 4 scored higher than the responders in psychopathic personality and antisocial behavior at wave 3, but the effect sizes for these differences were relatively small among males (psychopathic personality: $d = .18$; antisocial behavior: $d = .18$) and females (psychopathic personality: $d = .20$; antisocial behavior: $d = .23$) (Study III).

Neighborhood characteristics

At wave 3, participants and nonparticipants were compared in neighborhood characteristics. There was a significant difference for ethnic diversity ($t = 3.63$, $df = 2925$, $p < .001$), indicating that nonparticipating families more often live in neighborhoods characterized by ethnic heterogeneity. However, no significant differences were found for unemployment level, educational level, buying power or neighborhood crime-rate (Tuvblad, Grann, & Lichtenstein, 2006).

ZYGOSITY DETERMINATION

The zygosity determination of the same-sexed twin pairs is based on algorithms derived from discriminant analyses on 106 like-sexed twin pairs participating in a clinical study where zygosity was based on 16 polymorphic DNA-markers. The algorithms only classify pairs that have a 95 % probability of being correctly classified as MZ or DZ

twins. Zygosity was classified by using separate algorithms to parent's response (wave 1, wave 2, and wave 3) and to children's response (wave 2 and wave 3) using four questions dealing with the twins' physical similarity and the frequency with which people confuse them. Zygosity was scored as unknown in cases of contradictions between any of the five zygosity assignments. Zygosity could not be assigned to 86 twin pairs, mainly due to differences between two algorithms (Lichtenstein, Tuvblad, Larsson, & Carlström, 2007)

MEASURES

Psychopathic personality

Psychopathic personality was measured with the Youth Psychopathic traits Inventory (YPI; Andershed et al., 2002) when the twins were 16-17 and 19-20 years old. The YPI is a 50-item self-report instrument for adolescents measuring the three core personality dimensions of psychopathy. The Grandiose/manipulative dimension is composed of four subscales with five items each: dishonest charm, lying, grandiosity, and manipulation; the Callous/unemotional dimension is composed of three subscales with five items each: callousness, unemotionality and remorselessness and the Impulsive/irresponsible dimension is composed of three subscales with five items each: impulsiveness, irresponsibility, and thrill-seeking. Items are scored on a 4-point scale (1 = does not apply at all, 2 = does not apply well, 3 = applies fairly well, 4 = applies very well). Reliability analysis, measured with Cronbach's alpha (α), of the three dimensions showed acceptable internal consistencies at age 16: Grandiose/manipulative (Males: α = .83; Females α = .81); Callous/unemotional (Males: α = .64; Females α = .63); Impulsive/irresponsible (Males: α = .76; Females α = .77) and at age 19: Grandiose/manipulative (Males: α = .83; Females α = .81); Callous/unemotional (Males: α = .71; Females α = .67); Impulsive/irresponsible (Males: α = .70; Females α = .71). We have shown that the three dimensions are associated to a higher-order general factor, but also found etiologic specificity among the dimensions (Larsson et al., 2006). Thus, in this thesis, we have analyzed the three dimensions separately, but also used a composite scale of the measure to capture the common variance among the dimensions. The three dimensions along with corresponding subscales and sample items are presented in Table 1.

ADHD symptoms

Parent-reported ADHD symptoms were measured with a 14-item checklist using the DSM-III-R conceptualizations of the disorder (American Psychiatric Association, 1987). The children were rated on a binary scale (0 = not true; 1 = true) on each item, and then summed up in a composite score. The parents were asked to check symptoms persisting for at least six months. In this thesis (Study I), we used information about the children's level of ADHD symptoms when they were 8-9 and 13-14 years old. Examples of items are "Has difficulty remaining seated when required to do so", "Often shifts from one uncompleted activity to another", and "Often does not seem to listen to what is being said him or her". We did not assess the DSM-criterion "onset before the age of seven".

Table 1. Dimensions, subscales and sample item of the YPI.

Dimension	Subscale	Sample item
Grandiose/manipulative	Dishonest charm	When I need to, I use my smile and charm to use others.
	Grandiosity	I am more important and valuable than other people.
	Lying	Sometimes I lie for no reason, other than because it is fun.
	Manipulation	I can get almost anyone to believe anything.
Callous/unemotional	Callousness	When other people have problems it is often their own fault, therefore one should not help them.
	Unemotionality	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.
Impulsive/irresponsible	Impulsiveness	It often happens that I do things without thinking ahead.
	Thrill-seeking	I like to do things just for the thrill of it.
	Irresponsibility	It happened several times that I have borrowed something and then lost it.

Externalizing and internalizing problems

In this thesis, externalizing and internalizing problems were assessed using both parent- and self-reports. Parents answered the Child Behavior Checklist (CBCL) when the twins were 8-9, 13-14 and 16-17 years old and the Adult Behavior Checklist (ABCL) when the twins were 19-20 years old. The twins answered the Youth Self Report (YSR) when they were 13-14 and 16-17 years and the Adult Self Report (ASR) when they were 19-20 years (Achenbach, 1991; Achenbach & Rescorla, 2001, 2003).

The twins and their parents were instructed to rate externalizing and internalizing problems "now" or "within the past 6 months" using a 3-point scale (0 = not true; 1 = somewhat true; 2 = very true). The externalizing scale in these measures consists of the rule-breaking behavior (sample items: "use alcohol or drugs," "steal at home") and aggression subscales (sample items: "argue," "fights"), whereas the internalizing scale consists of the anxious/depressed (sample items: "cries", "fearful"), withdrawn/depressed ("enjoys little", "lacks energy") and somatic complaints (sample

items: “tired”, “nightmares”) subscales. All items in the externalizing and internalizing scales were summed up in a composite score.

In Study I, when the twins were 16-17 years old, we used information from a self-report delinquency questionnaire (Tuvblad et al., 2005) which consist of items measuring property offences (e.g., shop-lifting, vandalism), drug related offences (e.g., use drugs, sell soft drugs), and violent offences (e.g., arson, beat someone).

TWIN DESIGN

The twin design aids the study of individual differences by highlighting the role of genetic and environmental influences on behavior. The twin design compares the similarity of MZ twin pairs who share 100% of their genes, to that of DZ twin pairs, who share only 50% of their segregating genes. The basic twin model is based on the notion that the total phenotypic variance in a trait is a linear function of additive genetic, shared environmental and non-shared environmental factors. Additive genetic factors refer to the sum effect of genetic alleles at two or more gene loci. Shared environment refers to those environmental factors that contribute to twin similarity, whereas non-shared environmental factors refer to those environments that contribute to twin dissimilarity. Evidence for genetic effects is suggested when MZ twins show greater similarity than DZ twins in measures that are of interest. Consequently, evidence of shared environmental effects is suggested if DZ twin pairs are more than half as similar as MZ twin pairs. Finally, evidence of non-shared environmental effects (which includes measurement error) is suggested if MZ twins are dissimilar to each other (Plomin et al., 2008).

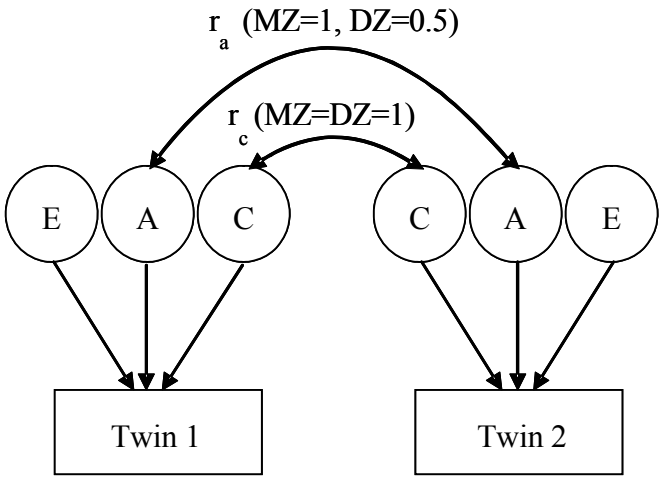

Figure 3. Univariate ACE model. A = Genetic effects, C = Shared environmental effects, E = Non-shared environmental effects, r_a = Genetic correlation, r_c = Shared environmental correlation.

Figure 3 illustrates how the total variance in a phenotype is decomposed into variance due to genetic effects, shared environmental effects and non-shared environmental effects. As MZ twins share both 100% of their genes and shared environment the genetic and the shared environmental correlations are set to 1.0. DZ twins have a

common shared environment, but only share, on average, 50% of their segregating genes. Thus, the genetic correlation between DZ twins is set to 0.5. By definition, there is no correlation between the non-shared environmental factors within a twin pair (Plomin et al., 2008).

Longitudinal and multivariate modeling of twin data

In Study I, III, and IV, a software package called Mx (Neale, Boker, Xie, & Maes, 2006) was used to run different structural equation models. All models in these studies were fitted to raw data by the method of raw maximum-likelihood estimation, which allows for inclusions of singletons and twin pairs with information from only one time-point. The different models are presented below. In Study II, we used the cotwin-control method, which also will be described.

Common pathway model (Study I)

In Study I we focused on three different common-pathway models to explore the correlated nature of the psychopathic personality dimensions (Larsson, et al., 2006). A *1-factor common-pathway model* was used to explore the extent to which one higher-order factor is enough to explain the covariance among the psychopathic personality dimensions within and across age. This model includes common genetic and environmental effects that load onto one higher-order general factor (i.e., psychopathic personality factor) that in turn load onto to the measured phenotypes (i.e., Grandiose/manipulative, Callous/unemotional and Impulsive/irresponsible) at age 16 and 19. The model also specifies unique genetic and environmental factors for each measured phenotype to capture variance not explained by the higher-order general factor.

This model was then extended to a *2-factor common-pathway model* (McArdle & Goldsmith, 1990). In contrast to the 1-factor common-pathway model, this model specifies two age-specific higher-order general factors to account for the covariation within the three psychopathic personality dimensions at mid- and late adolescence. These two higher-order factors are allowed to correlate with each other. As for the 1-factor common-pathway model, this model also specifies unique genetic and environmental factors for each measured psychopathic personality dimension.

Finally, we tested *a 2-factor common-pathway with correlated unique effects*. In contrast to the original 2-factor common-pathway model, this model does not only specify overlap between the two higher-order general factors, but also between the unique genetic and environmental effects for the three psychopathic personality dimensions at mid- and late adolescence. For example, the unique genetic effects on the Grandiose/manipulative dimension in mid-adolescence are allowed to correlate with the corresponding effects in late adolescence.

Cross-lagged twin model (Study III)

In Study III we examined the associations between psychopathic personality and antisocial behavior within and across age by using a cross-lagged twin model (Burt et al., 2005). All phenotypic associations across time-points (i.e., b_{11}, b_{22}, b_{12}, b_{21}, see

Figure 4) are expressed as partial regression coefficients. The cross-age regression coefficients (i.e., b_{11}, b_{22}) estimate the stability of psychopathic personality and antisocial behavior over time, when controlling for the preexisting association between the two phenotypes. The cross-lagged regression coefficients (i.e., b_{12} ,b_{21}) estimate the independent contribution of psychopathic personality at t1 on antisocial behavior at t2 (i.e., b_{12}) and correspondingly the independent contribution of antisocial behavior at t1 on psychopathic personality at t2 (i.e., b_{21}), when controlling for the stability in the two phenotypes. In addition, the covariance between psychopathic personality and antisocial behavior at t1 and between psychopathic personality and antisocial behavior at t2 are decomposed into genetic, shared environmental and non-shared environmental correlations (i.e., r_{a1}, r_{c1}, r_{e1} at t1 and r_{a2}, r_{c2}, r_{e2} at t2).

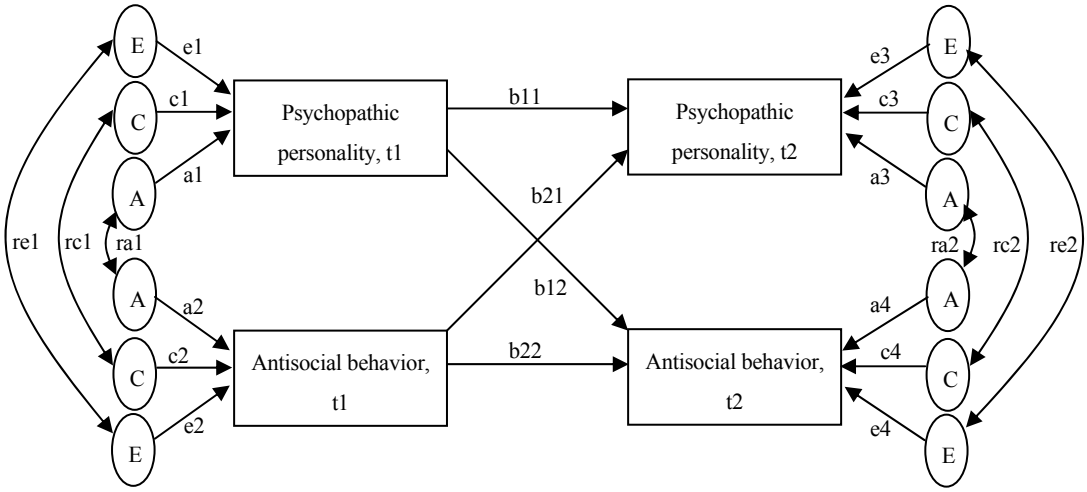

Figure 4. A path diagram of the cross-lagged model. The latent variables A (additive genetic factor), C (shared environmental factor), and E (non-shared environmental factor) are presented in the circles. Measured variables are depicted in rectangles. Standardized path estimates for these factors (i.e., a1,c1,e1 / a2,c2,e2 / a3,c3,e3 / a4,c4,e4), genetic and environmental correlations (i.e., ra1, rc1, re1/ ra2, rc2, re2), cross-age stability paths (i.e., b11, b22) and cross-lagged paths (i.e., b21, b12) are also represented in the diagram. t1 = time-point 1, t2 = time-point 2.

The variances in psychopathic personality and antisocial behavior at t2 were broken down into four different effects: cross-lagged effects, common effects, stability effects and residual effects. That is, externalizing behavior at t2 depends on effects due to the unique contribution of psychopathic personality at t1 (cross-lagged effects), the preexisting association between psychopathic personality and antisocial behavior at t1 (common effects), antisocial behavior at t1 (stability effects), and those specific to antisocial behavior at t2 (residual effects). The same four effects were used to explain psychopathic personality at t2.

Cholesky decomposition (Study IV)

In Study IV a series of bivariate Cholesky models were used to examine the association between different measures of psychopathic personality and externalizing and internalizing problems. The model decomposes the variance in one phenotype into

variance in common with another phenotype, and variance unique for the phenotype. The estimates from Cholesky model can also be used to for calculating how much of the phenotypic correlation that is due to genetic and environmental factors (Neale et al., 2006).

Cotwin-control (Study II)

In Study II we used the cotwin-control method to examine whether genetic, shared or non-shared environmental factors explained the association between the independent variables (i.e., persistent externalizing behavior and persistent ADHD symptoms) and the dependent variables (i.e., psychopathic personality). With twin data, we can modify the traditional case-control design to a cotwin-control design and use data from all twin pairs that are discordant for the variable of interest (e.g., persistent ADHD symptoms) (Plomin et al., 2008).

In the cotwin-control analyses, we first compared the mean levels in the dependent (e.g., psychopathic personality) between the affected and unaffected in the complete sample (i.e., external controls). Next, we selected and compared all discordant DZ and MZ twin pairs where one of the twins was affected and the other not (i.e., used the unaffected cotwin as the control). Three possible interpretations of the results from these comparisons can be made. First, one could find that the mean-difference decreases for the discordant MZ-twins. This suggests that the association is mainly explained by genetic factors. A second possibility is that the difference decrease for both the discordant DZ *and* MZ twin pairs. This would suggest that shared environmental factors are of importance for the association. Finally, one could find that the mean difference between the affected and the unaffected in the whole sample remain at the same level for the discordant DZ and MZ twin pairs. This would suggest that the association is mainly explained by non-shared environmental factors.

Sex-limitation models (Study I, III, IV)

By the inclusion of opposite-sex DZ twins, we were able to run different sex-limitation models to test for qualitative, quantitative, and phenotypic variance differences between the sexes in Mx (Neale et al., 2006). Qualitative genetic sex differences, indicated by a genetic correlation of less than 0.5 between opposite-sex twins, suggest that different genes are responsible for phenotypic variance in the sexes. Qualitative shared environmental sex differences, indicated by a shared environmental correlation of less than 1.0 between opposite-sex twins suggest that different shared environments are responsible for phenotypic variance in the sexes. Quantitative sex differences are suggested by differences in the magnitude of additive genetic, shared environmental and non-shared environmental effects between sexes. We used a scalar model to test for phenotypic variance differences between the sexes. This model allows for sex differences in phenotypic variances, but constrains the A, C and E parameters to be equal between sexes and the genetic correlation between opposite-sex twins to equal the genetic correlation between same-sex DZ twins. Finally, in the constrained model, all variance components were set to be equal between the sexes.

RESULTS

STUDY I

In Study I we examined the stability of psychopathic personality between ages 16-17 and 19-20 years. The age-to-age correlations for the tree psychopathic personality dimensions ranged from r = 0.43 to r = 0.61 and from r = 0.51 to r = 0.58 among males and females, respectively. The Impulsive/irresponsible dimension showed largest stability among males (r = 0.61) and females (r = 0.58). Thus, the psychopathic personality dimensions were moderately to highly stable from mid- to late adolescence.

Next, we examined the contribution of genetic and environmental influences for the stability of psychopathic personality. The model fitting results of multivariate analysis showed that a 2-factor common-pathway model (scalar) with correlated unique effects had the best fit in terms of balance between parsimony and explanatory power. This suggests that there is an overlap between the higher-order general factors at age 16-17 and 19-20, and that the unique effects at age 16-17 overlap with those at age 19-20. Figure 5 provides squared path estimates and confidence intervals for the best fitting model.

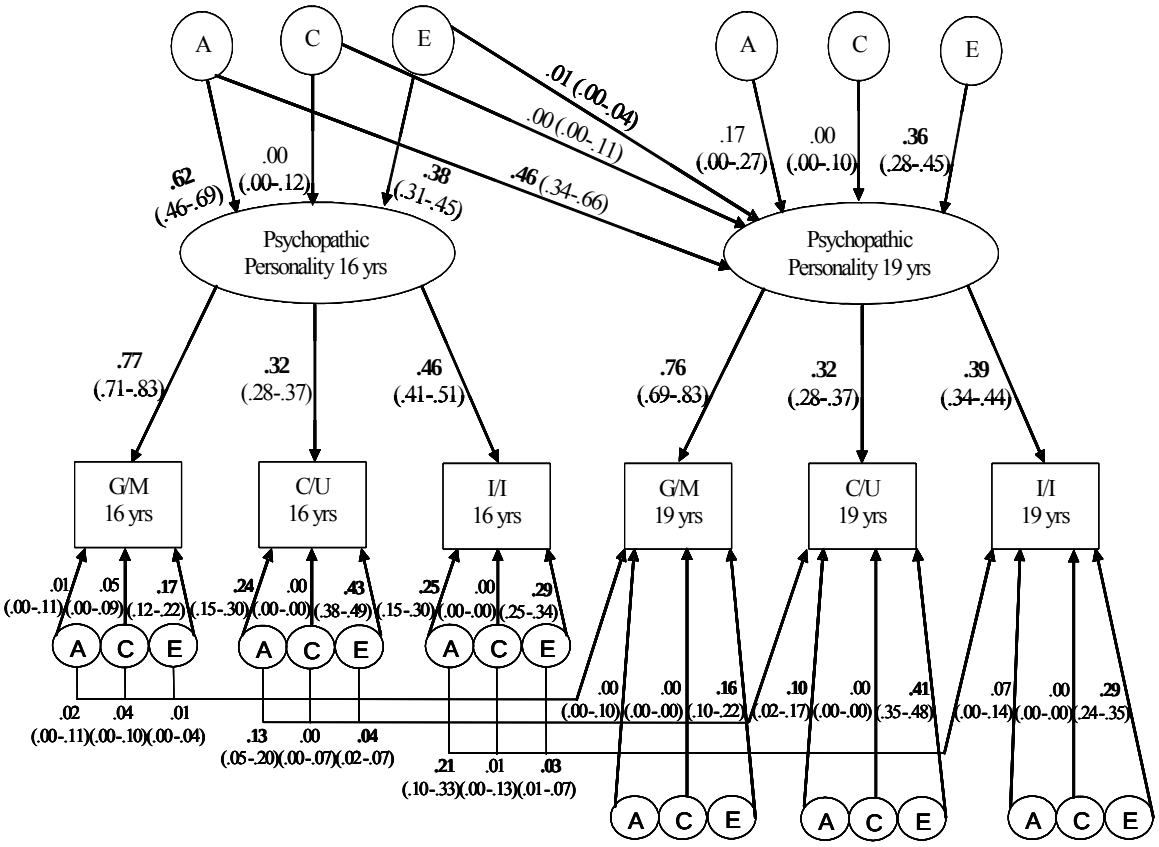

Figure 5. Squared path estimates for the full 2-factor common-pathway model with correlated unique effects. Significant estimates are in bold and confidence intervals (95%) are in parentheses. G/M = Grandiose/manipulative dimension; C/U = Callous/unemotional dimension; I/I = Impulsive/irresponsible dimension.

As shown in Figure 5, additive genetic factors explained 62 and 63% (46%+17%) of the variance in the higher-order psychopathic personality factor at age 16-17 and 19-20, respectively. A substantial part of the genetic effects at age 19-20 was shared with the corresponding effects at age 16-17 (46%), while the unique genetic effects at age 19-20 years was statistically non-significant (17%; CI = 0%- 27%). Non-shared environmental factors explained 38 and 37% of the variance in the in the higher-order psychopathic personality factor at age 16-17 and 19-20, respectively. However, the overlap between the non-shared environmental effects at age 16-17 and 19-20 was negligible and statistically non-significant (1%; CI = 0%- 4%). Shared environmental factors did not significantly contribute to the variance in the psychopathic personality factor at age 16-17 or 19-20.

Further, genetic factors accounted for as much as 90% of the total phenotypic correlation between the higher-order psychopathic personality factors at age 16-17 and 19-20, while non-shared environmental factors accounted for the remaining 10% of the correlation.

Finally, a substantial part of the unique genetic effects in the Callous/unemotional and the Impulsive/irresponsible dimension were shared between the two time-points. For example, more than half of the unique genetic variance in the Callous/unemotional dimension at age 19-20 was shared with the corresponding effects at age 16-17.

In conclusion, genetic factors contributed substantially to the stability of the higher-order psychopathic personality factor, whereas environmental factors were of little importance. However, specific genetic stability was also found in the Callous/unemotional and Impulsive/irresponsible dimensions.

STUDY II

In Study II, we first asked whether persistent externalizing behavior, persistent ADHD symptoms or the combination of the two is associated with the psychopathic personality constellation in adolescence.

We conducted a series of 2 by 2 ANOVAs to examine the effect of persistent externalizing behavior, persistent ADHD symptoms and the interaction of the two on different aspects of the psychopathic personality constellation. For girls, there were no significant associations between our measures of childhood disruptive behavior and psychopathic personality in adolescence. They were therefore excluded from further analyses. For boys, the 2 by 2 ANOVA yielded significant main effects between persistent externalizing behavior ($F = 8.71$, $p < .01$) and persistent ADHD symptoms ($F = 3.79$, $p < .05$) with the composite psychopathic personality scale, while the interaction was non-significant. Further, unlike persistent ADHD symptoms and the interaction, persistent externalizing behavior also showed significant main effects to the Callous/unemotional dimension ($F = 8.17$, $p < .01$), antisocial behavior dimension ($F = 3.77$, $p < .01$) and approached significance to the Grandiose/manipulative dimension ($F = 3.66$, $p=.06$). Finally, both persistent externalizing behavior ($F = 8.34$, $p < .01$) and persistent ADHD symptoms ($F = 5.09$, $p < .05$) showed significant main effects to the Impulsive/irresponsible dimension, while the interaction was non-significant.

Table 2. Cotwin-control analysis of psychopathic personality and antisocial behavior for twins discordant in persistent externalizing behavior among boys.

	n	Mean difference	t	p
At 16-17 years				
Psychopathic personality				
External controls	1008	1.44	4.17	.00
Discordant DZ twins	20	2.85	3.34	.00
Discordant MZ twins	20	-.40	-.47	.64
Grandiose/manipulative				
External controls	1022	.10	2.44	.01
Discordant DZ twins	21	.28	2.13	.05
Discordant MZ twins	20	-.06	-.55	.59
Callous/unemotional				
External controls	1020	.12	3.30	.00
Discordant DZ twins	21	.16	1.91	.07
Discordant MZ twins	20	-.04	-.35	.73
Impulsive/irresponsible				
External controls	1020	.21	4.71	.00
Discordant DZ twins	21	.27	2.12	.05
Discordant MZ twins	20	-.01	-.15	.88
Antisocial behavior				
External controls	1016	1.85	1.94	.05
Discordant DZ twins	20	-.07	-.03	.98
Discordant MZ twins	20	1.1	.33	.74

Note. We used independent t-tests for significance tests of mean differences between the affected and unaffected in the whole sample (i.e., external controls), and paired t-tests for differences between the discordant twin pairs.

Next, we examined whether the observed associations between the independent and dependent variables were explained by genetic, shared or non-shared environmental factors. In the co-twin control analysis we examined mean differences in psychopathic personality between those with and without persistent externalizing behavior in the complete sample and compared it with the mean differences within the discordant twin pairs. The results from these analyses are presented in Table 2.

As shown, among the external controls, boys with persistent externalizing behavior scored significantly higher in psychopathic personality compared to those without persistent externalizing behavior. The difference remained at the same level for the externalizing-discordant DZ twin pairs, but disappeared for the externalizing-discordant MZ twin pairs. For example, the mean difference between those with and without persistent externalizing behavior in the whole sample was 1.44 in the composite psychopathic personality scale. Among the externalizing-discordant DZ twin pairs, the mean difference was 2.85. However, there was no mean difference between the externalizing-discordant MZ twin pairs (-.40). The average mean difference within externalizing-discordant DZ twin pairs was significantly higher than within MZ twin

pairs (t = 2.71, df = 38, p < .05). Similar results were obtained in the analyses with the three psychopathic personality dimensions.

Boys with persistent externalizing behavior also scored significantly higher in the antisocial behavior dimension compared to boys without persistent externalizing problems (Mean difference = 1.85; t = 1.94; p < .05). However, the mean differences dropped and became non-significant both among the externalizing-discordant DZ (Mean difference = -.07; t = -.03; p = 98) and MZ twin pairs (Mean difference = 1.1, t = .33, p = .74).

Finally, among the external controls, boys with persistent ADHD symptoms scored significantly higher in the Impulsive/irresponsible dimension compared to those without persistent ADHD symptoms (Mean difference = 0.20; t = 4.23; p < .01). The mean differences remained approximately at the same level among the ADHD-discordant DZ (Mean difference = 0.16; t = 1.19; p = .25) and MZ twins (Mean difference = 0.24; t = 2.22; p < .05), suggesting that non-shared environmental factors explain the association.

In conclusion, persistent externalizing behavior in childhood was associated with both psychopathic personality and antisocial behavior in adolescence. Genetic factors explained the association between persistent externalizing behavior and psychopathic personality, whereas shared environmental factors explained the association between persistent externalizing problems and antisocial behavior. Persistent ADHD symptoms were mainly associated with the Impulsive/irresponsible dimensions due to non-shared environmental factors.

STUDY III

The aim of Study III was to investigate the direction of effects between psychopathic personality and antisocial behavior, and to investigate the genetic and environmental contribution to this association. To do this, we used a longitudinal cross-lagged twin model.

As shown in Figure 6, the association between psychopathic personality at t1 and antisocial behavior at t2 was significant (b_{12}: $\Delta\chi^2 = 23.20$, $df = 1$, $p<.001$), but the association between antisocial behavior at t1 and psychopathic personality at t2 was not (b_{21}: $\Delta\chi^2 = 2.87$, $df = 1$, $p= 0.09$). Thus, psychopathic personality in adolescence significantly predicted antisocial behavior in adulthood, but the opposite was not true.

Next, by using the parameter estimates from Figure 6, the total phenotypic as well as the total genetic, shared environmental and non-shared environmental variance in antisocial behavior at t2 was broken down into those uniquely contributed by psychopathic personality and those from the preexisting association between antisocial behavior and psychopathic personality at t1 (cross-lagged and common effects). We focused on these two effects in this study although the contribution from antisocial behavior at t1 (stability effects) and those specific to antisocial behavior at t2 (residual effects) also were estimated.

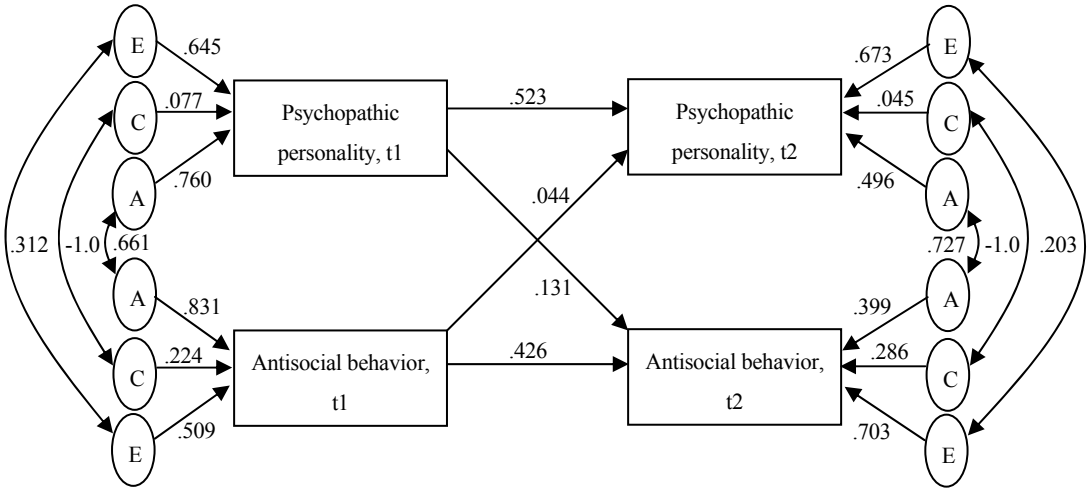

Figure 6. Standardized path estimates for the additive genetic, shared environmental and non-shared environmental factors from the best fitting cross-lagged model.

Table 3. Squared, standardized path coefficients and corresponding percentages of variance accounted for in adult antisocial behavior among males.

	Antisocial behavior at t2			
	Total phenotypic variance	Total A, C, E variances		
		A	C	E
Proportion of variance due to:	1.0	.348	.090	.562
Psychopathic personality at t1	.017 (1.7%)	.010 (2.9%)	.000	.007 (1.2%)
Common effects at t1	.058 (5.8%)	.048 (13.8%)	-.002	.012 (2.1%)
Antisocial behavior at t1	.190 (18.9%)	.131 (37.6%)	.010	.049 (8.7%)
Residual effects at t2	.735 (73.6%)	.159 (45.7%)	.082	.494 (88.0%)

As shown in Table 3, 1.7% ($.131^2 \times .760^2$) + ($.131^2 \times .077^2$) + ($.131^2 \times .645^2$) of the phenotypic variance in antisocial behavior at t2 was due to the unique contribution of psychopathic personality at t1 (i.e., cross-lagged effect) and 5.8% (2 × [.436 × .831 × .661 × .760 × .131]) + (2 × [.436 × .224 × -1.00 × .077 × .131]) + ([2 × [.436 × .509 × .312 × .645 × .131]) of the variance was due to the preexisting association between antisocial behavior and psychopathic personality (common effect) at t1.

Table 3 also shows the contribution of genetic, shared environmental and non-shared environmental effects for antisocial behavior at t2 (A = .348; C =.090 E = .562). The breakdown of effects revealed that unique genetic effects on psychopathic personality at t1 (cross-lagged genetic effects) explained 2.9% ([$.131^2 \times .760^2$] / [.348]) of the genetic variance in antisocial behavior at t2, whereas the effect from the preexisting association between antisocial behavior and psychopathic personality at t1 (common

genetic effects) explained 13.8% ([2 × [.436 × .831 × .661 × .760 × .131]] / [.348]) of the genetic variance. The contribution from the cross-lagged (1.2%) and common effects (2.1%) to the non-shared environmental variance was relatively small. The proportions of the shared environmental variance were incalculable because of the negative shared environmental correlation between psychopathic personality and antisocial behavior at t1.

In our subsidiary aims, we wanted to investigate whether adolescent psychopathic personality predicts adult antisocial behavior over and above concurrent and preexisting antisocial behavior. We also examined whether our measure of persistent antisocial behavior (age 8-9 + 13-14 + 16-17) would predict adult psychopathic personality. Analyses showed that the effect of psychopathic personality at t1 on antisocial behavior at t2 remained statistically significant ($\Delta\chi^2 = 38.86$, $df = 1$, $p < 0.001$) over and above persistent antisocial behavior. In addition, persistent antisocial behavior also predicted psychopathic personality at t2 ($\Delta\chi^2 = 9.67$, $df = 1$, $p < 0.01$). In this model, psychopathic personality explained 2.7% of the total phenotypic variance in antisocial behavior at t2. Unique genetic effects in psychopathic personality at t1 explained 4.3% of the total genetic variance in antisocial behavior at t2. Persistent antisocial behavior explained 0.7% of the total phenotypic variance in psychopathic personality at t2. Unique genetic effects in persistent antisocial behavior explained 1.1% of the total genetic variance in psychopathic personality at t2.

In conclusion, psychopathic personality in adolescence predicted antisocial behavior in adulthood, but not the other way around. However, bidirectional effects were found when a measure of persistent antisocial behavior was used. Genetic factors were of importance in mediating the longitudinal associations between psychopathic personality and antisocial behavior.

STUDY IV

In Study IV we used the hierarchical conceptualization of psychopathy and investigated how the general (i.e., the covariance of the psychopathic personality dimensions) and specific variances (i.e., unique variance in each dimension) of psychopathic personality were phenotypically and etiologically related to externalizing and internalizing problems. Table 4 contains the phenotypic correlations between psychopathic personality and externalizing and internalizing problems. In general, the correlations were similar for boys and girls. The higher-order psychopathic personality factor was substantially associated with externalizing problems, whereas its association with internalizing problems was moderate. The residual Callous/unemotional dimension was negatively correlated with both measures of psychopathology, whereas the residual Impulsive/irresponsible dimension was positively associated externalizing problems, but weakly with internalizing problems. The residual Grandiose/manipulative dimension showed weak, and mostly non-significant, associations with both measures of psychopathology. As a result, the residual Grandiose/manipulative dimension was not included in the following bivariate twin-analyses. Likewise, the association between the residual Impulsive/irresponsible dimension and internalizing problems was excluded from the twin-analyses.

Table 4. Phenotypic correlations between psychopathic personality and externalizing and internalizing problems.

	Higher-order psychopathic personality factor	Residual Grandiose/ manipulative	Residual Callous/ unemotional	Residual Impulsive/ irresponsible
		Boys		
Externalizing	.60***	-.01	-.19***	.23***
Internalizing	.28***	.12***	-.17***	.08**
		Girls		
Externalizing	.60***	-.08**	-.20***	.22***
Internalizing	.30***	.01	-.16***	.11***

Note. * p < .05 ** p < .01 *** p < .001.

Table 5. Genetic and environmental contributions to the overlap between psychopathic personality and externalizing and internalizing problems.

	Higher-order psychopathic personality factor			
		r due to:		
	r	A	C	E
Externalizing	.60	.43 (74%)	.00 (0%)	.17 (26%)
Internalizing	.29	.20 (69%)	.00 (0%)	.09 (31%)
	Residual Callous/unemotional			
		r due to:		
	r	A	C	E
Externalizing	-.20	-.10 (50%)	.00 (0%)	-.10 (50%)
Internalizing	-.17	-.09 (53%)	.00 (0%)	-.08 (47%)
	Residual Impulsive/irresponsible			
		r due to:		
	r	A	C	E
Externalizing	.22	.15 (68%)	.00 (0%)	.07 (32%)

The proportion of the phenotypic correlation that is explained by shared genetic and environmental effects can be obtained by multiplying the square root of the heritability estimate for one phenotype by the square root of the heritability estimate for the other phenotype by the genetic correlation between these two phenotypes. The results from these calculations are shown in Table 5.

The phenotypic correlations between the higher-order psychopathic personality factor and externalizing problems and internalizing problems were mainly due to shared genetic effects. In the combined sample (boys and girls), 74% of the phenotypic correlation between the higher-order psychopathic personality factor and externalizing

problems (r = .60) was due to shared genetic effects and the remaining proportion was due to non-shared environmental effects (26%). Second, shared genetic effects were of importance for the negative phenotypic correlation between the residual Callous/unemotional dimension and externalizing and internalizing problems. For example, in the combined sample, 53% of the phenotypic correlation between the residual Callous/unemotional dimension and internalizing problems (r = -.17) was due to shared genetic effects. Finally, the phenotypic correlation between the residual Impulsive/irresponsible dimension and externalizing problems was also substantially explained by shared genetic effects. In the combined sample, 68% of the phenotypic correlation between the residual Impulsive/irresponsible dimension and externalizing problems (r = .22) was due to shared genetic effects.

In follow-up analyses, the association between the higher-order psychopathic personality factor and externalizing problems remained at the same level when controlling for internalizing problems (r = .54, p <.001). However, the unique association between the higher-order psychopathic personality factor and internalizing problems dropped substantially (r = -.07, p <.01). The residual Callous/unemotional dimension's association to internalizing problems was unaffected (r = -.19, p <.001), whereas its association to externalizing problems dropped, but remained negative and statistically significant (r = -.09, p <.01). Finally, the association between the residual Impulsive/irresponsible dimension and externalizing problems remained at the same level (r = .17, p <.001).

In conclusion, the higher-order psychopathic personality factor was phenotypically associated positively with externalizing problems. The specific variance of the Callous/unemotional dimension was negatively related to both measures, whereas the specific variance of the Impulsive/irresponsible dimension was positively related to externalizing problems. Twin-analyses showed that mainly genetic effects contributed to the phenotypic associations found between psychopathic personality and externalizing and internalizing problems.

DISCUSSION

Collectively, the studies in this thesis highlight the importance of having prospective longitudinal and multivariate twin data to understand the development of psychopathic personality and the association with other psychopathological problems. In summary, findings in this thesis provide evidence that: (I) genetic effects explain the stability of psychopathic personality between adolescence and adulthood, (II) the association between childhood externalizing problems and adolescent psychopathic personality is explained by genetic effects, (III) adolescent psychopathic personality predicts adult antisocial behavior via genetic effects, (IV) the higher-order psychopathic personality factor is strongly related to externalizing problems, whereas specific effects in the Callous/unemotional and Impulsive/irresponsible dimensions show divergent associations to externalizing and internalizing problems. The main findings in each of the studies and future directions are discussed below.

GENETIC EFFECTS EXPLAIN STABILITY OF PSYCHOPATHIC PERSONALITY FROM ADOLESCENCE TO ADULTHOOD

Study I suggest that the temporal stability of psychopathic personality seems to be largely similar as for other personality traits (Roberts & DelVecchio, 2000), and there is little support of the notion that psychopathic traits are temporary characteristics of adolescence (Edens, et al., 2001; Seagrave & Grisso, 2002). In addition, consistent with previous research (Blonigen et al., 2006; Larsson, et al., 2006), the findings in this study provide support for genetic generality (i.e., common genetic effects) and specificity (i.e., unique genetic effects) within the psychopathic personality construct. The high stability and heritability of the psychopathic personality factor suggest that it may be efficient to focus on this general factor rather than on specific dimensions in future molecular genetic research. That is, to maximize chances to identify specific genes that contribute to individual differences in psychopathic personality, one could focus on individuals that show stable levels of overall psychopathic personality. However, given evidence for significant stability of unique genetic effects in both this study and previous research (Blonigen et al., 2006), investigations of unique etiologic factors to specific dimensions of psychopathic personality are also of importance. Therefore, the genetic heterogeneity (i.e., genetic generality and genetic specificity) could be explored further by examining how the general (i.e., higher-order psychopathic personality factor) and the specific variance in psychopathic personality dimensions (e.g., Callous/unemotional dimension) relate, not only to measured genes, but also to emotional and cognitive processes (e.g., Blair, 2003; Hiatt & Newman, 2006; Verona, Patrick, Curtin, Bradley, & Lang, 2004), as well as psychopathological domains of internalizing and externalizing behavior problems (see Study IV).
Similar to previous twin studies of psychopathic personality (Blonigen et al., 2006; Taylor et al., 2003) shared environmental factors were of negligible importance for psychopathic personality. On the other hand, non-shared environmental factors contributed significantly to the higher-order psychopathic personality factor in mid- and late adolescence. However, in contrast to the genetic effects and in line with previous findings (Blonigen et al., 2006), non-shared environmental factors were largely age-specific and thus more associated with change than with stability. This indicates that

the transition from adolescence to early adulthood is an important period to focus on to understand why and how psychopathic personality changes over time.

In summary, the findings in this study give further support for the downward extension of psychopathic personality to adolescents and suggest that the stability of this personality constellation is primarily explained by genetic factors.

THE ASSOCIATION BETWEEN CHILDHOOD EXTERNALIZING PROBLEMS AND ADOLESCENT PSYCHOPATHIC PERSONALITY IS EXPLAINED BY GENETIC EFFECTS

The association between persistent externalizing problems and psychopathic personality was mainly explained by genetic factors, which may suggest that both phenotypes represent different manifestations of a common, genetically influenced phenotype that is stable over the development from childhood to adolescence. However, the association between persistent externalizing problems and psychopathic personality was relatively modest. This highlights the importance of identifying other childhood risk factors that contribute to the development psychopathic personality. Neuropsychological components (e.g., executive functioning) and temperamental/personality traits (e.g., fearlessness, conscience development) in early childhood may be two promising candidates (Frick & Morris, 2004). Thus, future longitudinal twin studies may include measures of these kinds of problems and examine their genetic overlap with psychopathic personality and persistent externalizing problems. In addition, we measured externalizing problems via parent's reports only, which partly may explain the modest associations. Ideally, one would want to combine parent-reports with other sources of information, such as teacher- and self-reports to get a broader picture of the twin's involvement in externalizing problems.

In contrast, we found that shared environmental factors were of importance for the association between persistent externalizing behavior and adolescent antisocial behavior, which provide support for an etiological distinction between psychopathic personality and antisocial behavior. Previous studies have found that both genetic and shared environmental factors are important for the stability of antisocial behavior (Eley, Lichtenstein, & Moffitt, 2003). We did not include measures of specific environmental variables that could explain this association, but critical, hostile, coercive and/or inconsistent-harsh parenting (Hill, 2002), and deviant peer group affiliation might be two examples of shared environmental factors that could be of importance (Rutter, Giller, & Hagell, 1998).

Finally, the association between persistent ADHD symptoms and the Impulsive/irresponsible dimension was explained by non-shared environmental factors. Persistent ADHD symptoms and Impulsive/irresponsible personality are partially conceptually overlapping (e.g., they both include impulsivity), and probably reflect stability of impulsivity-problems over time, which is in line with previous research (e.g., Larsson, Lichtenstein, & Larsson, 2006). Previous studies have reported that stability of ADHD symptoms from childhood to adolescence mainly is due to genetic factors (Larsson et al., 2004; Larsson et al., 2006). However, non-shared environmental

factors are also important for stability in ADHD symptoms. This study has not measured the specific environments that could be responsible for the association, but prenatal environmental factors that affect one but not the other twin might be one example (Linnet et al., 2003; Linnet, et al., 2006).

Persistent disruptive childhood behavior was in this study defined using a dichotomous variable based on the scores from two time-points (i.e., above the 75th percentile at age 8-9 and 13-14). Although this method may capture a particularly problematic subgroup of children, sophisticated trajectory modeling techniques are now available that more accurately identifies subgroups of children based on longitudinal data. Since we defined persistent disruptive childhood behavior as a dichotomous variable we used the cotwin-control method. Although the cotwin-control method can be used to roughly examine the importance genetic and environmental factors for the association between two phenotypes, it does not allow one to parse the shared variance between two phenotypes into components of genetic, shared environmental and non-shared environmental factors.

ADOLESCENT PSYCHOPATHIC PERSONALITY PREDICTS ADULT ANTISOCIAL BEHAVIOR VIA GENETIC EFFECTS

In Study III we showed that psychopathic personality in mid-adolescence predicted levels of antisocial behavior in early adulthood, over and above both concurrent and preexisting levels of antisocial behavior. In contrast, antisocial behavior in mid-adolescence was unrelated to subsequent psychopathic personality. Together with Study I, in which we found a high stability of psychopathic personality, this finding demonstrates the predictive utility of psychopathic personality and adds further support for using the psychopathy construct to identify an important pathway to antisocial behavior. We also showed that the association between adolescent psychopathic personality and adult antisocial behavior mainly was explained by genetic factors. Taken together, these results can be interpreted as a genetically influenced personality-driven process (psychopathic personality → antisocial behavior), where individuals are predisposed to higher risk of involvement in antisocial behavior because of their antisocially prone personality. That is, individuals that have a manipulative interpersonal style, lack empathy and remorse, and lack the ability to consider the consequences of their behavior are at higher risk for future involvement in antisocial behavior.

However, in line with findings in Study 2, we also found evidence for an opposite direction of effects between psychopathic personality and antisocial behavior. When we used a measure of persistent antisocial behavior (measured at age 8-9, 13-14 and 16-17 years) we found evidence of a genetically driven effect from antisocial behavior to psychopathic personality. Thus, over and above the potential existence of a personality-driven process, we also found evidence of behavior-driven processes (antisocial behavior → psychopathic personality). This finding may suggest that engaging in antisocial behavior from childhood to adolescence make individuals emotionally insensitive to the consequences of their behavior on themselves and others, which during development may have an impact on the subsequent levels of psychopathic personality.

The fifth TCHAD data collection, which is planned to be conducted when the twins are in their mid twenties, will increase the knowledge of how psychopathic personality and antisocial behavior are related developmentally as we will have three assessments for both measures. This will make it possible to examine whether antisocial behavior evokes psychopathic personality, which in turn starts having an independent influence on antisocial behavior further on. In addition, we plan to link the TCHAD data with official criminal records, which in combination with self-reports of antisocial behavior at the fifth data collection will deepen our understanding of the association between psychopathic personality and antisocial behavior.

GENETIC EFFECTS EXPLAIN THE ASSOCIATION BETWEEN PSYCHOPATHIC PERSONALITY AND PSYCHOPATHOLOGY IN ADOLESCENCE

In Study I we showed that psychopathic personality can be well described with a hierarchical model in which a higher-order general factor explains the covariance among psychopathic personality dimensions, but also that each dimension contains specific variance. In Study IV we wanted to follow up on these findings and used the hierarchical conceptualization of psychopathy to investigate how both the general (i.e., higher-order psychopathic personality factor) and specific variances of psychopathic personality are related to externalizing and internalizing problems.

Our finding of a large phenotypic and genetic overlap between the higher-order psychopathic personality factor and externalizing problems have several important implications. First, prior research has largely focused on antisocial children with callous/unemotional traits and found that they show particularly severe, stable and genetically influenced forms of antisocial behavior. Our findings suggest that future studies could investigate whether the higher-order psychopathic personality factor can provide useful information regarding this important subgroup in early childhood. Second, previous research has shown that shared genetic factors influence a spectrum of externalizing problems including adolescent antisocial behavior, conduct disorder, alcohol dependence, drug dependence, and behavioral disinhibition (Krueger et al., 2002). The finding of a substantial overlap between the higher-order psychopathic personality and externalizing problems in this study suggest that future attempts to model the externalizing spectrum may benefit from including psychopathic personality traits as well. Third, recent gene identification projects have demonstrated that the strategy of using multivariate externalizing phenotypes can be useful in both linkage and association analyses (Dick et al., 2008). No study has yet tried to identify specific genes involved in psychopathic personality. The results in this study suggest that the higher-order psychopathic personality factor may represent a promising multivariate psychopathy phenotype that efforts of this kind could focus on. Given the high genetic overlap between the higher-order psychopathic personality factor and externalizing problems, we predict that a substantial number of the genes found to be associated with externalizing psychopathology (e.g., Dick et al., 2008) will also have a role in the etiology of psychopathic personality.

The higher-order psychopathic personality was moderately associated with internalizing problems, but in line with previous findings this association was entirely due to co-occurring externalizing problems (Lynam, 1997). Thus, previous research may have overestimated the association between psychopathic personality and internalizing problems by not considering its overlap with externalizing problems.

In line with prior research (Blonigen et al., 2005; Patrick et al., 2005; Salekin et al., 2004), our data suggest that the specific variance in the Callous/unemotional dimension is negatively related to externalizing and internalizing problems, due to genetic and non-shared environmental factors. This might indicate that the specific variance of the Callous/unemotional dimension reflect a component of positive adjustment that act as a protective factor for externalizing and internalizing problems; an interpretation that correspond well with other studies showing that the specific variance in this dimension is positively related to measures of adjustment, such as educational attainment and sociability (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Benning, Patrick, Hicks, Blonigen, & Krueger, 2003). Future studies could focus on youths with high levels of Callous/unemotional traits, but without Grandiose/manipulative and Impulsive/irresponsible traits to identify the factors that explain why some individuals with Callous/unemotional traits do not develop psychopathology.

Finally, the specific variance in the Impulsive/irresponsible dimension was positively related to externalizing problems, mainly due to shared genetic factors (68% and 71% respectively). This specific variance may reflect the hyperactive-impulsive component of Attention Deficit Hyperactivity Disorder (ADHD). This reasoning corresponds well with Study II showing that childhood ADHD predicts the Impulsive/irresponsible, but not the Callous/unemotional and Grandiose/manipulative dimension in adolescence.

METHODOLOGICAL CONSIDERATIONS

The findings in this thesis need to be taken within the context of several basic assumptions underlying the twin method. Measurement issues and potential biases due to attrition are also discussed.

Issues in twin studies

The equal environments assumption

If genetic factors are important for a trait, MZ twins must be more similar than DZ twins. However, when greater similarity of MZ twins is found, it possible that the greater similarity is caused by environmental rather than genetic factors. The equal environments assumption of the twin method assumes that similarity caused by environmental factors is roughly the same for both types twins reared in the same family. This assumption is violated if MZ twins experience more similar environments than DZ twins, which in turn would inflate estimates of genetic effects. The equal environments assumption has not been tested for psychopathic personality, but studies have found it to be valid for anxiety disorder, ADHD, oppositional defiant disorder, and conduct disorder (Cronk et al., 2002).

Assortative mating

The twin method assumes that mating occurs at random in the parent generation. Assortative mating inflates the DZ correlations, thereby lowering estimates of genetic effects and raising estimates of shared environmental effects. In general, shared environmental influences were of little or no importance in the studies of this thesis. In Study II, we found evidence of shared environmental effects for the association between persistent externalizing problems in childhood and antisocial behavior in adolescence. However, at wave 4, mothers and fathers in the TCHAD sample were asked to retrospectively report their antisocial behaviors in their twenties. The correlation was statistically significant but relatively weak ($r = .15$, $p < .001$), suggesting that the potential effect of assortative mating is limited in Study II.

Nonadditive genetic variance

In the basic twin model we often focus on additive genetic effects. Additive genetic effects on a trait occur when alleles at a locus and across loci add up independently of each other. However, the effects of specific alleles can be different in the presence of other alleles. Dominance is a nonadditive effect in which alleles at a specific locus interact rather than add up to influence a trait. Epistasis is a nonadditive effect that occurs when alleles at different loci influence a trait in an interactive manner.

Nonadditive genetic variation for a trait may be present if DZ twin-pair similarity is less than half similar as MZ twins. If nonadditive genetic variance is present but not modeled, then this variance will be estimated as additive genetic variance. The intra-class correlations among DZ twins in Study I may suggest that at least some part of the additive genetic variation in psychopathic personality is due to nonadditive effects.

Generalizability

One criticism of the twin method is that twins differ from other individuals in several important aspects and that results based on twin samples do not generalize to the population as a whole. Two previous reports in the current TCHAD sample have shown that the mean levels in emotional and behavioral measured with the CBCL are at the same level as other Swedish singleton samples (Larsson, Lichtenstein, Fried, El-Sayed, & Rydelius, 2000; Eley et al., 2003). Furthermore, prevalence rates of ADHD in TCHAD are similar to other Swedish reports (Kadesjö & Gillberg, 1998).

Gene-environment interplay

In addition to direct influences on a given phenotype, genetic effects may also play a more indirect role on behavior, being mediated through correlations and interactions with the environment. Gene-environment correlation refer to the finding that that some individuals are more likely to be exposed to a risk environment if they already carry a genetic risk for poor outcome. Gene-environment interaction refers to the possibility that an individual may be genetically sensitive (or insensitive) to certain environmental influences. Both gene-environment correlation and gene-environment interaction has been examined in the TCHAD study for antisocial behavior (Narusyte, 2009; Tuvblad, Grann, & Lichtenstein, 2006), but was not the focus in this thesis. Clearly, future

attempts to explain the heterogeneity of how psychopathic personality develops need to take gene-environment correlation and gene-environment interaction into account.

Measurement issues

The idea of measuring psychopathic personality by asking individuals about themselves may for many readers seem paradoxical. After all, can one expect to identify a condition marked by deceitfulness, lying, and manipulation by asking individuals to respond honestly to questions regarding this condition? The YPI was developed with this specifically in mind (Andershed et al., 2002). The items in the YPI were developed to assess psychopathic personality in an indirect, rather than in straightforward and transparent way. While focusing on the core features of psychopathy, the YPI was specifically developed to avoid a socially desirable response bias by describing feelings and opinions as competences, rather than deficiencies (Andershed et al., 2002). The large correlations between the higher-order psychopathic personality factor and antisocial behavior indicate that the YPI measures important aspects of psychopathic personality.

Similarly, self-reports of antisocial behavior could be misleading in some ways in that psychopathic individuals might lie about their levels of antisocial behavior. However, a recent study showed that self-reported psychopathic personality predicted levels of subsequent offending according to official records (Salekin, 2008). So, it seems that youths that report high levels of psychopathic traits actually are at higher risk for future antisocial behavior, not because they are lying about their antisocial acts.

Attrition

Although the TCHAD study has a relatively high response those missing from the sample are likely to have more psychopathological problems than those that remain. Thus, some results in this thesis may not be generalizable to individuals with the most extreme forms of psychopathological problems. The effects of attrition in the TCHAD sample have to some extent been reported in this thesis and elsewhere (see Study II and III and method section of this thesis). For psychopathic personality and antisocial behavior, the effect size for the difference between participants and participants lost to follow up between waves 3 and 4 was relatively low. Notwithstanding, in relation to wave 1-3, the participation loss at wave 4 was relatively high and it will be of great importance to increase participation in our planned fifth data collection.

CONCLUSIONS

There are two major conclusions in thesis that needs to be emphasized. First, in support for the downward extension of psychopathic personality to adolescents, we could demonstrate that primarily genetic effects explained the large stability of the higher-order psychopathic personality factor, but also that there were significant unique genetic stability in the Callous/unemotional and Impulsive/irresponsible dimensions. The higher-order psychopathic personality factor was substantially related to externalizing problems, whereas the unique variance in the Callous/unemotional and Impulsive/irresponsible dimensions were divergently related to externalizing and internalizing psychopathology. These results highlight the importance of considering both general and specific etiologic factors within the psychopathic personality constellation and may guide future attempts to identify specific genes that are of importance for this personality constellation.

Second, it was shown that genetically influenced psychopathic personality predicted adult antisocial behavior over and above preexisting antisocial behavior. These findings demonstrate the predictive utility of psychopathic personality and add further support for using the psychopathy construct to identify an important pathway to antisocial behavior. Evidence of a genetically mediated link between persistent antisocial behavior and subsequent psychopathic personality was also found. This may indicate that persistent antisocial behavior and psychopathic personality are linked to a genetically influenced phenotype that is stable over time, but may also give evidence of transactional influences in which antisocial behavior evokes psychopathic personality, which in turn start having an independent influence on antisocial behavior further on. Future research would benefit of including measures of psychopathic personality in younger samples and at several assessments points to clarify the nature of this association in more detail.

SVENSK SAMMANFATTNING

Psykopati, eller psykopatisk personlighet är en personlighetsstörning bestående av avvikande interpersonella, affektiva och beteendemässiga dimensioner. Det har påvisats att psykopatiska personlighetsdrag kan användas för att förstå utvecklingen av antisocialitet bland ungdomar. Mindre forskning har tillägnats till att undersöka den bakomliggande etiologin till psykopatiska personlighetsdrag. Det finns även få longitudinella tvillingstudier som har studerat hur psykopatiska personlighetsdrag är relaterat till andra psykopatologiska korrelat. Denna avhandling har använt longitudinell och multivariat tvillingdata för att klargöra etiologin för psykopatiska personlighetsdrag och dess relation till andra psykopatologiska problem.

Data i denna avhandling kommer från Twin Study of Child Adolscent Development, som är en prospektiv longitudinell studie med insamlad data från tvillingar vid 8-9, 13-14 år, 16-17 och 19-20 års ålder.

Studie I i denna avhandling visade att genetiska faktorer var av omfattande betydelse för stabiliteten av den psykopatiska personlighetsfaktorn mellan tonårsåldern och vuxen ålder, medan miljömässiga faktorer var av liten betydelse. Specific genetisk stabilitet observerades även i dimensionerna som mäter Känslokallhet/empatibrist (affektiv) och Impulsivitet/oansvarighet (beteendemässig). Studie II visade att persistenta externaliserade problem är relaterat till både psykopatiska personlighetsdrag och antisocialt beteende i tonåren. Tvillinganalyser visade dock att genetiska faktorer förklarade sambandet mellan persistenta externaliserade problem och psykopatiska personlighetsdrag, medan sambandet mellan persistenta externaliserade problem och antisocialt beteende förklarades av tvillingarnas gemensamma miljö. I Studie III påvisades att psykopatiska personlighetdrag predicerade antisocialt beteende i vuxen ålder via genetiska faktorer. Bidirektionella associationer observerades dock när ett mått på persistent antisocialt beteende användes. I Studie IV visades att psykopatiska personlighetsfaktorn var relaterat till externaliserade problem och att detta samband till stor del kunde förklaras av en gemensam genetisk faktor, medan de specifika varianserna i Känslokallhet/empatibrist (negativt samband) och Impulsivitet/oansvarighet (positivt samband) visade motsatta associationer till psykopatologiska problem.

Denna avhandling understryker vikten av att ta hänsyn till både generella och specifika etiologiska faktorer för att förstå stabilitet och förändring av psykopatiska personalighetsdrag, samt för att identifiera risk och skyddande faktorer i utvecklingen av externaliserade och internaliserade beteendeproblem. Framtida försök att identifiera specifika gener kan därför fokusera på den generella, men också specifika variansen i psykopatisk personlighet. Denna avhandling har också bidragit till forskningen genom att visa att psykopatiska personlighetsdrag i tonåren predicerar antisocialt beteende i vuxen ålder via genetiska effekter, men också att persistent antisocialitet predicerar psykopatiska personlighetsdrag i vuxen ålder. Framtida forskningsprojekt uppmuntras att inkludera psykopatiska personlighetsdrag bland yngre urval och samla in data över flera åldrar, så att relationen mellan psykopatiska personlighetsdrag och antisocialt beteende kan klargöras ännu mer detaljerat.

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