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Aggressive antisocial behavior

Clinical, cognitive, and behavioral covariates of its persistence



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Märta Wallinius



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Gutta cavat lapidem
non vi sed saepe cadendo

Ovidius

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Abstract

Introduction

Aggressive antisocial behavior is a major challenge to society, and studies on the determinants of its persistence are essential to the development of strategies to prevent violence.

Aims & Methods

The overall aim of the thesis was to establish covariates of persistent aggressive antisocial behavior in a population-based cohort and in clinically evaluated, prospectively followed, offender groups. Specific aims were: (1) to quantify the persistence of aggressive antisocial behavior, (2) to identify risk factors for such persistence, (3) to describe the distribution of psychopathic traits (according to the Psychopathy Checklist-Revised; PCL-R) in relation to clinical factors, and (4) to determine thinking styles related to aggressive antisocial behavior.

Results

One percent of the Swedish general population was responsible for 63% of all violent crimes between 1973 and 2004. Risk factors for persistence of aggressive antisocial behavior included its early onset, conduct disorder, antisocial personality disorder, and substance-related disorders. In the PCL-R, the Antisocial facet outperformed all other facets and the total score as a predictor of persistence. The Interpersonal facet showed unique clinical attributes. A distorted thinking style characterized by antisocial features was related to aggressive antisocial behavior.

Conclusions

Aggressive antisocial behaviors and clinical factors including externalizing mental health problems aggregate within individuals. The best predictor of future aggressive antisocial behavior is a history of similar behaviors, especially with an early onset. Scientific and clinical efforts aimed to prevent violence have therefore to focus both on early-onset and persistent aggressive antisocial behavior, and their associated clinical and cognitive characteristics.

Svensk sammanfattning

Introduktion

Aggressivt antisocialt beteende utgör en stor utmaning för dagens samhälle, och undersökningar av vad som ligger bakom ett återkommande sådant beteende behövs för utvecklingen av våldspreventiva insatser.

Syften & Metod

Syftet med avhandlingen var att identifiera faktorer som samvarierar med återkommande aggressivt antisocialt beteende. De specifika målsättningarna var: (1) att beskriva omfattningen av återkommande aggressivt antisocialt beteende, (2) att identifiera riskfaktorer för sådant beteende, (3) att beskriva fördelningen av, och kliniska samband till, psykopatiska personlighetsdrag studerade med Psychopathy Checklist-Revised (PCL-R), och (4) att beskriva samband mellan dysfunktionella tankemönster och aggressivt antisocialt beteende.

Resultat

En procent av den svenska befolkningen stod för 63 % av alla våldsbrottsdomar under åren 1973–2004. Riskfaktorer för återkommande aggressivt antisocialt beteende var tidig debut i sådant beteende, uppförandestörning, antisocial personlighetstörning och missbruksproblem. Bland de psykopatiska personlighetsdragen var tidigare kriminellt beteende den överlägset bästa prediktorn för återkommande aggressivt antisocialt beteende. De grandiosa och manipulativa personlighetsdragen i PCL-R uppvisade unika kliniska karaktäristika. Dysfunktionella tankemönster av en antisocial karaktär var relaterade till aggressivt antisocialt beteende.

Slutsatser

Aggressivt antisocialt beteende och psykisk problematik av en utagerande karaktär ansamlas inom individer. Tidigare aggressivt antisocialt beteende, särskilt med tidig debut, är den enskilt bästa prediktorn av framtida aggressivt antisocialt beteende. Våldspreventiva insatser bör därför fokusera på tidigt debuterande och återkommande aggressivt antisocialt beteende, samt psykisk och social problematik som är associerad med sådant beteende.

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List of papers

This thesis is based on the following papers, which will be referred to in the text by their Roman numerals:

I. Falk Ö, **Wallinius M**, Lundström S, Frisell T, Anckarsäter H, Kerekes N. (submitted). The 1% of the population accountable for 63% of all violent crime convictions.

II. **Wallinius M**, Nilsson T, Billstedt E, Anckarsäter H, Hofvander B. (manuscript). Persistent criminality among violent offenders in emerging adulthood: the importance of early-onset externalizing behaviors.

III. Nilsson T, **Wallinius M**, Gustavson C, Anckarsäter H, Kerekes N. (2011). Violent recidivism: a long-time follow-up study of mentally disordered offenders. *PLoS ONE* 6(10):e25768.

IV. **Wallinius M**, Nilsson T, Hofvander B, Anckarsäter H, Stålenheim G. (2012). Facets of psychopathy among mentally disordered offenders: clinical comorbidity patterns and prediction of violent and criminal behavior. *Psych Res* 198:279–284.

V. **Wallinius M**, Johansson P, Lardén M, Dernevik M. (2011). Self-serving cognitive distortions and antisocial behavior among adults and adolescents. *Crim Justice Behav* 38:286–301.

Papers III to V are reprinted with permission from the publishers.

Abbreviations

AD/HD	Attention Deficit/Hyperactivity Disorder
AMOS	Analysis of MOment Structures
ANOVA	ANalysis Of VAriance
APA	American Psychiatric Association
AUC	Area Under the Curve
CFA	Confirmatory Factor Analysis
COVR	Classification of Violence Risk
DIS-CAT 2.0	Early-onset behavioral DISorders across diagnostic CATegories 2.0
DSM-III-R	The Diagnostic and Statistical Manual of Mental Disorders 3rd edition, Revised
DSM-IV	The Diagnostic and Statistical Manual of Mental Disorders 4th edition
DSM-IV-TR	The Diagnostic and Statistical Manual of Mental Disorders 4th edition, Text Revision
HCR-20	Historical-Clinical-Risk Management-20
HIT	The How I Think questionnaire
ICD-8	The International Classification of Diseases 8th edition
ICD-9	The International Classification of Diseases 9th edition
ICD-10	The International Classification of Diseases 10th edition
KSP	The Karolinska Scales of Personality
LHA	The Life History of Aggression questionnaire
LSI-R	Level of Service Inventory-Revised
NOIR	Nominal, Ordinal, Interval, and Ratio
NPV	Negative Predictive Value
OR	Odds Ratio
PASW	Predictive Analytics SoftWare
PCL-R	The Psychopathy Checklist-Revised

PPV	Positive Predictive Value
RMSEA	Root Mean Square Error of Approximation
ROC	Receiver Operating Characteristics
SAPROF	Structured Assessment of PROtective Factors for violence risk
SAVRY	Structured Assessment of Violence Risk in Youth
SCID-I	The Structured Clinical Interview for Axis I Disorders
SCID-II	The Structured Clinical Interview for Axis II Disorders
SCID-II Screen	The Structured Clinical Interview for DSM-III-R Axis II Disorders, Screening Version
SPSS	Statistical Package for the Social Sciences
START	Short-Term Assessment of Risk and Treatability
SVR-20	Sexual Violence Risk-20
VRAG	Violence Risk Appraisal Guide
VRS	Violence Risk Scale
WHO	World Health Organization

Introduction

Interpersonal violence affects most aspects of modern society and has been declared a global public health priority. Each year approximately half a million people die worldwide due to illegal interpersonal violence[1]. When non-fatal and unreported interpersonal violence is added to this, the economic, social, and human costs reach staggering levels. For instance, non-fatal and fatal injuries from interpersonal violence resulted in total costs of approximately $37 billion in the United States for the one year of 2000[2].

The World Health Organization (WHO) has emphasized the role of the public health sector in the prevention of violence, and stressed the importance of developing national action plans for this purpose[3]. As a first step, data on the magnitude, risk factors, and consequences of violence need to be systematically collected and analyzed. A critical focus must be on the persistence of violent behavior, as the majority of violent crimes in society are committed by recidivists[4].

This thesis aims to identify crucial covariates of persistence in violent criminality using both a general population cohort and specific offender groups. Particular emphasis will be placed on factors that can contribute to the development and improvement of interventions for the prevention of violence.

Violence throughout history

Human violence is certainly not new. Archaeological investigations provide substantial evidence of widespread aggressive and violent behaviors from the earliest prehistoric times[5], suggesting violence is an intrinsic part of human behavior.

Most people behave violently at some point during their lives[6]. Thus, violent acts are committed not only by persistently violent individuals, but also by those who do not normally act violently, but at some point find themselves in a situation where violence is promoted[7]. Most people, however, are never convicted of a violent crime[8].

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[1] Krug et al. 2002
[2] Corso et al. 2007
[3] Butchart et al. 2004
[4] Elonheimo et al. 2009, Krug et al. 2002
[5] DeWall & Anderson 2011, Pinker 2011
[6] Tolan 2007
[7] Farrington 2007
[8] Elonheimo et al. 2009

Throughout human history, violence has been exerted not only by individuals, but also by states, as a means of punishment or entertainment as well as an expression of power or control. Violent games in ancient Rome and public torture and execution in medieval Europe provide many gruesome examples.

Steven Pinker[9] argues there has been a substantial decline of violence from older hunter–gatherer societies to modern society. He demonstrates a fivefold decrease in violent deaths following the transition from hunter–gatherer societies to agricultural societies (the *pacification process*), and a ten- to fiftyfold decline in homicide rates throughout Europe from the late Middle Ages to the 20th century (the *civilizing process*). Norbert Elias[10] attributed this reduction in violence to the centralization of state control, including a monopolization of violence, and the evolution of increasingly mannered social interactions following social, cultural, and psychological changes. In the aftermath of World War II, an increased focus on human rights and reduced acceptance of violence against vulnerable groups in society has led to further reductions in violence[11]. The global homicide rate in the 21st century has been estimated as 8.8 per 100 000 people per year[12].

In Sweden, approximately 90 people (0.9 per 100 000) die from violence each year[13]. This corresponds to a reduction of approximately 25% from the last decades of the 20th century.

Definitions of aggressive antisocial behavior

Several similar, but not identical, terms are used to describe law-breaking and/or harmful interpersonal behavior, and this lack of consensus on language has been criticized as obstructing the coordination and comparison of studies and precluding the development of preventive efforts[14].

The cultural context complicates the development of clear definitions, as behavior that would be recognized as illegal and violent in some cultures would not be considered such in others. Examples include honor-related violence and the physical punishment of children.

Accepting that prevention of aggressive antisocial behavior is the ultimate goal for scientific studies in this area, distinct, quantifiable definitions that can be shared

[9] Pinker 2011
[10] Elias 2000
[11] Pinker 2011
[12] Krug et al. 2002
[13] The Swedish National Council for Crime Prevention 2011
[14] Tolan 2007

across different disciplines and cultures are needed[15]. In the following section, some definitions that have played significant roles in shaping the literature will be discussed, and a heuristic definition for the work included in this thesis proposed. Many of the terms also have other meanings, even in the field of psychology. For example, aggression can refer to an internal drive rather than a type of behavior. The aim of this introduction is to clarify the terminology used in this thesis and not to cover all other possible uses or meanings.

Aggression

In this thesis, *aggression* describes hostile or attacking interpersonal behavior. Aggressive behavior usually expresses the immediate intent to harm[16] or assert dominance over another person or group of persons; however, it may also be directed at animals, objects as proxies for persons, or at the self. Aggression can be expressed in both physical (e.g., kicking, biting, hitting) and non-physical (e.g., threatening or cursing) forms.

Violence

A common core in all definitions of violence is that *violent behavior* is the (threatened or actual) use of physical force or power to physically or psychologically harm another[17]. Violence is regarded as a more extreme and destructive form of aggression[18], even if the meaning of the two concepts may coincide.

The WHO definition of violence[19] (Table 1) has been criticized for being overly inclusive[20], as it includes self-directed violence and violence resulting from an asymmetric power relationship.

[15] Butchart et al. 2004
[16] Anderson & Bushman 2002, DeWall & Anderson 2011
[17] Farrington 2007, Krug et al. 2002, Tolan 2007
[18] Shaver & Mikulincer 2011
[19] Krug et al. 2002
[20] Tolan 2007

Table 1. The WHO definition of violence

The intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation.

Antisocial behavior, criminality, and delinquency

Antisocial behavior is defined as "any action that violates personal or cultural standards for appropriate behavior" (p. 17)[21]. Thus, antisocial behavior is not defined by the violation of any specific law in any specific country, nor does it require that the perpetrator (e.g., a child, adolescent, or person with diminished intellectual capacity) be held legally responsible for the behavior.

Antisocial behavior can include aggression and violent behavior, but aggression or violence need not always be antisocial (e.g., violence as a means of upholding public order and law enforcement). By the same token, antisocial behaviors do not have to involve aggression or violence (e.g., lying, stealing, or illicit drug use).

Don Andrews and James Bonta[22] define *criminality* as "antisocial acts that place the actor at risk of becoming a focus of the attention of criminal justice professionals within the juvenile and/or adult justice systems" (p. 12). *Delinquency* is a specific term referring to criminality committed by juveniles[23].

Externalizing behaviors

Externalizing behaviors are defined as aggressive, acting out, and conduct-disordered behaviors. This concept has been used predominantly in research on emotional and behavioral problems in children[24]. A corresponding term used in the scientific literature is *disruptive behaviors*.

[21] DeWall & Anderson 2011
[22] Andrews & Bonta 2010
[23] Rhee & Waldman 2007
[24] Achenbach & Edelbrock 1978

Aggressive antisocial behavior

In this thesis, *aggressive antisocial behavior*[25] is used to describe all interpersonal behaviors that meet the dual criteria of being aggressive (hostile or attacking) and antisocial (violating personal or cultural standards). Aggressive antisocial behavior has previously been distinguished from nonaggressive antisocial behavior in behavioral genetic studies[26].

Distribution of aggressive antisocial behavior

Aggressive antisocial behavior in society

There is substantial evidence that aggressive antisocial behaviors are not evenly distributed between members of a society. Men commit almost 80% of all homicides worldwide, and the majority of both violent offenders and their victims are found among adolescents and young adults[27]. Previous studies have reported that small groups, generally fewer than 10% of a population, are responsible for the majority of violent crimes in that population[28].

Rates of aggressive antisocial behavior also differ according to economic circumstances, ethnic groups, and urban versus rural communities[29]. Cultural variations in rates of aggressive antisocial behavior are also evident. Cultures that glorify or support violence as a means of problem-solving and that advocate male dominance (e.g., "macho cultures") tend to show higher rates of aggressive antisocial behavior than cultures that emphasize equality between genders[30]. Considerable fluctuations in aggressive antisocial behavior in different societies have also been noted over time.

Aggressive antisocial behavior over the life course

One of the most consistent findings in criminological research is that aggressive antisocial behavior has a curvilinear distribution over the life course[31]. Aggressive

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[25] Hofvander et al. 2009
[26] Burt 2009
[27] Krug et al. 2002, Reza et al. 2001, Tolan 2007
[28] Elonheimo et al. 2009, Loeber et al. 1999, Moffitt et al. 2002, Stattin & Magnusson 1989, Tolan & Gorman-Smith 1999
[29] Krug et al. 2002
[30] Krug et al. 2002, Pinker 2011
[31] Blonigen 2010, Blumstein et al. 1988, Hirschi & Gottfredson 1983

antisocial behavior increases significantly during mid-adolescence, peaks in late adolescence, and decreases rapidly beginning in early adulthood (Figure 1). Many individuals in a population show transient aggressive antisocial behavior during adolescence[32], and several longitudinal studies have found that only a minority (about 10%) of a population do not engage in delinquent behavior at all during adolescence[33]. This increase in aggressive antisocial behavior has been attributed both to biological changes (especially in hormone levels)[34], and to the transition of social influences in adolescence from predominantly familial to peer-dependent[35].

There is also substantial evidence for behavioral continuity in aggressive antisocial behavior over the life course[36]. Childhood aggressive antisocial behavior is a precursor to adult aggressive antisocial behavior, especially serious and persistently offending behavior[37]. Lee Robins[38], however, demonstrated that most children who display disruptive behaviors do not pursue persistent and serious aggressive antisocial behaviors into adulthood.

Figure 1. Distribution of aggressive antisocial behavior over the ages. Reprinted from Moffitt 1993 with permission from the American Psychological Association.



[32] Farrington 2007, Moffitt 2007
[33] Piquero et al. 2005
[34] Archer 1991, Ramirez 2003, van Goozen & Fairchild 2009
[35] Farrington 2007
[36] Farrington 2003, Farrington et al. 2009, Huesmann et al. 2009, Loeber 1982, Moffitt 1993
[37] Loeber et al. 1999, Loeber & Farrington 2000, Pulkkinen et al. 2009, Tolan & Gorman-Smith 1999
[38] Robins 1966, Rutter et al. 2006

Developmental pathways of aggressive antisocial behavior

Based on the distribution of aggressive antisocial behavior in individuals and over the life course, different *developmental pathways* have been suggested. Terrie Moffitt[39] proposed a dual model: *adolescence-limited antisocial behavior* and *life-course-persistent antisocial behavior*, the latter referring to the main focus of this thesis.

Adolescence-limited offenders are common in a population, and follow a transient and almost normative pathway of non-aggressive antisocial behavior that begins in adolescence and declines in young adulthood[40]. These offenders have typically had a normal pre-adolescent development with average to favorable backgrounds, and they usually engage in antisocial behaviors only when they are with their peers[41].

The adolescence-limited pathway is thought to originate in social processes during adolescence (the *maturity gap*), when many individuals are attracted to, and mimic, an antisocial lifestyle as a way to pursue autonomy from their parents and win affiliation with their peers[42]. In early adulthood, adolescence-limited offenders reduce their delinquency as they mature into adult roles and adopt a more conventional way of living. However, previous delinquency can hamper transition into adulthood by creating "snares" such as unfinished education, substance-use or other mental health problems, financial problems, or a criminal record that diminish a young adult's opportunities for pro-social and functional development[43].

Life-course-persistent offenders, in contrast, are only a few pathological individuals in society who display serious aggressive antisocial behavior that persists from early childhood into adulthood[44].

This pathway is thought to result from an interaction between the disruptive and challenging behavior (e.g., difficult temperament, hyperactivity) of a neuro-psychologically impaired child and a high-risk social environment[45]. The risky social environment typically includes susceptibility factors such as inadequate parenting, disrupted family bonds, and poverty[46]. During the child's development, these environmental risks may expand to include poor relations with peers (e.g., peer rejection) and with others outside the family. A severe pathology may develop over the life course, with negative effects on multiple life domains (e.g., mental health,

[39] Moffitt 1993
[40] Moffitt 2007, Moffitt & Caspi 2001
[41] Jeglum Bartusch et al. 1997, Moffitt et al. 2002
[42] Moffitt 1993, Moffitt 2007
[43] Moffitt et al. 2002
[44] Moffitt 1993, Moffitt et al. 2002, Odgers 2009
[45] Moffitt 1993, Moffitt 2007
[46] Farrington et al. 2009, Huesmann et al. 2009, Moffitt 2007, Moffitt & Caspi 2001

partner relationships, parenting, financial circumstances, and adjustment to the labor market)[47].

Several independent, longitudinal studies have found similar groups of individuals characterized by early onset and persistent aggressive antisocial behavior. Examples are *early starters*[48], *chronic offenders*[49], and *high-level chronics*[50]. However, these typologies do not account for all the variation in aggressive antisocial behavior in individuals over time. Thus, other developmental pathways of aggressive antisocial behavior have been distinguished, such as *childhood-limited antisocials*[51], *low-level chronics*[52], and *adult-onset offenders*[53]. There will probably always remain a number of aggressive antisocial individuals who cannot be identified on developmental pathways.

Risk factors for aggressive antisocial behavior

To develop strategies for the prevention of aggressive antisocial behavior, it is necessary to know about the risk factors, i.e., the variables that are associated with an increased probability of aggressive antisocial behavior[54]. The last decades have seen an upsurge in research that has led to an increased understanding of the complex interaction between individual (e.g., genetic, biological, personality) and environmental risk factors for aggressive antisocial behavior.

A history of previous aggressive antisocial acts has repeatedly been shown to be the strongest correlate to, and best predictor of, future aggressive antisocial behavior[55]. Aggression during childhood is associated with, and predicts, aggressive antisocial behavior in adolescence and adulthood[56]. David Farrington[57] has proposed that "the people who are relatively more aggressive at one age also tend to be relatively more aggressive at later ages, even though absolute levels of aggressive behavior and behavioral manifestations of violence are different at different ages" (p. 23).

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[47] Bergman & Andershed 2009, Huesmann et al. 2009, Moffitt et al. 2002, Pulkkinen et al. 2009
[48] Patterson et al. 1992
[49] Chung et al. 2002, Fergusson et al. 2000
[50] Nagin et al. 1995
[51] Moffitt et al. 1996, Raine et al. 2005
[52] Moffitt et al. 2002, Nagin et al. 1995
[53] Eggleston & Laub 2002, Kratzer & Hodgins 1999, Pulkkinen et al. 2009
[54] Heilbrun 2009
[55] Bonta et al. 1998, Grann et al. 2008, Monahan et al. 2001, Walters et al. 2008
[56] Farrington 2007, Loeber et al. 1999, Loeber & Farrington 2000, Pulkkinen et al. 2009, Stattin & Magnusson 1989, Tolan & Gorman-Smith 1999
[57] Farrington 2007

The likelihood of future aggressive antisocial behavior increases steadily with the total number of offenses, regardless of type[58]. A salient finding in many studies is that an early age at onset of aggressive antisocial behavior is especially related to both persistence and severity of aggressive antisocial behavior[59].

The variance in persistent aggressive antisocial behavior has been shown to be under very considerable genetic influence[60], and familial aggregation and cross-generational transmission of aggressive antisocial behavior have been demonstrated[61]. In a recent review, Larry Siever[62] described the complexity of structural and functional neurobiological covariates of aggressive antisocial behavior. Knowledge about the psychosocial covariates (e.g., socioeconomic status and area of residence) of aggressive antisocial behavior have also increased[63]. Yet, we are still far from an integrated scientific model explaining the causation behind aggressive antisocial behavior.

The following section reviews current knowledge of individual clinical risk factors for aggressive antisocial behavior.

Mental disorders

Childhood onset disorders in the form of conduct disorder and attention deficit/hyperactivity disorder (AD/HD), especially hyperactivity, are associated with increased risk of aggressive antisocial behavior during the life course[64]. The DSM-IV-TR[65] diagnostic criteria for conduct disorder (Table 2) refer in several respects to early-onset aggressive antisocial behavior. Some studies support the hypothesis that AD/HD on its own is predictive of aggressive antisocial behavior[66]. However, most studies have found that this risk, especially for more severe and persistent behavior patterns, is mediated through AD/HD being complicated by conduct disorder in childhood[67].

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[58] Farrington 2003, Smith 2007
[59] Farrington 1995, Farrington 2007, Kratzer & Hodgins 1997, Moffitt 1993
[60] Burt 2009, Rhee & Waldman 2002
[61] Frisell 2012
[62] Siever 2008
[63] Farrington 2007, Krug et al. 2002
[64] Barkley et al. 1990, Bergman & Andershed 2009, Copeland et al. 2007, Hill & Nathan 2008, Mordre et al. 2011, Simonoff et al. 2004, Söderström et al. 2004, Sourander et al. 2006
[65] American Psychiatric Association (APA) 2000
[66] Mannuzza et al. 2008, Young & Thome 2011
[67] Lynam 1996, Mordre et al. 2011, von Polier et al. 2012

Table 2. Diagnostic criteria for conduct disorder (DSM-IV-TR)

A. A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as manifested by the presence of three (or more) of the following criteria in the past 12 months, with at least one criterion present in the past 6 months:

Aggression to people and animals
(1) often bullies, threatens, or intimidates others
(2) often initiates physical fights
(3) has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun)
(4) has been physically cruel to people
(5) has been physically cruel to animals
(6) has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)
(7) has forced someone into sexual activity

Destruction of property
(8) has deliberately engaged in fire setting with the intention of causing serious damage
(9) has deliberately destroyed others' property (other than by fire setting)

Deceitfulness or theft
(10) has broken into someone else's house, building, or car
(11) often lies to obtain goods or favors or to avoid obligations (i.e., "cons" others)
(12) has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery)

Serious violations of rules
(13) often stays out at night despite parental prohibitions, beginning before age 13 years
(14) has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lenghty period)
(15) is often truant from school, beginning before age 13 years

B. The disturbance in behavior causes clinically significant impairment in social, academic, or occupational functioning.
C. If the individual is age 18 years or older, criteria are not met for Antisocial Personality Disorder.

Substance-related disorders have consistently been demonstrated as one of the leading clinical risk factors for aggressive antisocial behavior[68]. In Sweden, from 1988 to 2000, the proportion of aggressive antisocial behavior that may be attributed to people with substance-related disorders was 25%[69]. This may be explained by two possible models. First, aggressive antisocial behavior can be seen as a direct effect of substance use as some substances promote aggressive behaviors. Second, aggressive antisocial behavior can be seen as an indirect effect of the lifestyle associated with maintaining a substance abuse (e.g., committing robberies in order to obtain money to buy drugs).

There is substantial evidence that major mental disorders (i.e., psychotic disorders and bipolar disorders) are related to an increased risk of aggressive antisocial behavior[70], especially in co-existence with substance-related disorders[71]. These disorders are, however, also overrepresented among individuals who have had childhood-onset aggressive antisocial behaviors prior to the onset of the major mental disorders[72]. Henry Steadman and colleagues demonstrated that patients with major mental disorders without substance use problems are no more dangerous than other persons living in the same neighborhoods[73]. Furthermore, psychotic disorders were negatively related to recidivism in aggressive antisocial behavior in a meta-analysis by James Bonta and colleagues[74]. Evidence is currently accumulating to suggest that co-morbid substance-related disorders, together with a history of conduct disorder or aggressive antisocial behavior, can explain much of the association between major mental disorders and aggressive antisocial behavior[75].

Personality disorders

Antagonistic, narcissistic, paranoid, negative emotional, sensation-seeking, and impulsive and disinhibited personality traits have been associated with aggressive antisocial behavior[76]. These personality traits are descriptive of the DSM-IV-TR antisocial personality disorder and borderline personality disorder.

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[68] Arseneault et al. 2000, Coid et al. 2006, Elonheimo et al. 2007, Grann et al. 2008, Pulay et al. 2008, Sacks et al. 2009
[69] Grann & Fazel 2004
[70] Arseneault et al. 2000, Brennan et al. 2000, Fazel & Grann 2006, Hodgins et al. 1996
[71] Fazel et al. 2009, Tiihonen et al. 1997
[72] Kim-Cohen et al. 2003
[73] Steadman et al. 1998
[74] Bonta et al. 1998
[75] Arseneault et al. 2000, Elbogen & Johnson 2009, Fazel et al. 2009
[76] Arseneault et al. 2000, Blonigen & Krueger 2007, Farrington 2007, Krueger et al. 1994, Nestor 2002, Skeem et al. 2005

Antisocial personality disorder is the disorder that has most consistently and strongly been associated with an increased risk of aggressive antisocial behavior[77]. Similar to conduct disorder (Table 2), antisocial personality disorder is diagnosed largely on the basis of such behaviors (Table 3).

Table 3. Diagnostic criteria for antisocial personality disorder (DSM-IV-TR)

A. There is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years, as indicated by three (or more) of the following:

(1) failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest
(2) deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure
(3) impulsivity or failure to plan ahead
(4) irritability and aggressiveness, as indicated by repeated physical fights or assaults
(5) reckless disregard for safety of self or others
(6) consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations
(7) lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another

B. The individual is at least age 18 years.
C. There is evidence of Conduct Disorder with onset before age 15 years.
D. The occurrence of antisocial behavior is not exclusively during the course of Schizophrenia or a Manic Episode.

Findings on the association between borderline personality disorder and aggressive antisocial behavior are inconclusive, with some studies supporting a covariation and others not[78]. It has been suggested that axis II co-morbidity can account for an association between borderline personality disorder and aggressive antisocial

[77] Coid et al. 2006, Elonheimo et al. 2007, Yu et al. 2012
[78] Allen & Links 2012, Berman et al. 1998, Coccaro et al. 1997, Johnson et al. 2000, Raine 1993

behavior[79]. Recently, emotional dysregulation was demonstrated as a mediator of increased risk for aggressive antisocial behavior in borderline personality disorder[80].

Other personality disorders that have been specifically associated with aggressive antisocial behavior include paranoid, schizoid, narcissistic, histrionic, and passive-aggressive personality disorders[81]. However, these associations are not as strong or conclusive as that for antisocial personality disorder.

Psychopathy

The psychopathy checklists[82] are commonly used as measures of psychopathic traits and have consistently been associated with increased risk of aggressive antisocial behavior in different settings[83]. In this thesis, psychopathy, or psychopathic traits, refers to traits and behaviors measured by scores on the Psychopathy Checklist-Revised (PCL-R)[84].

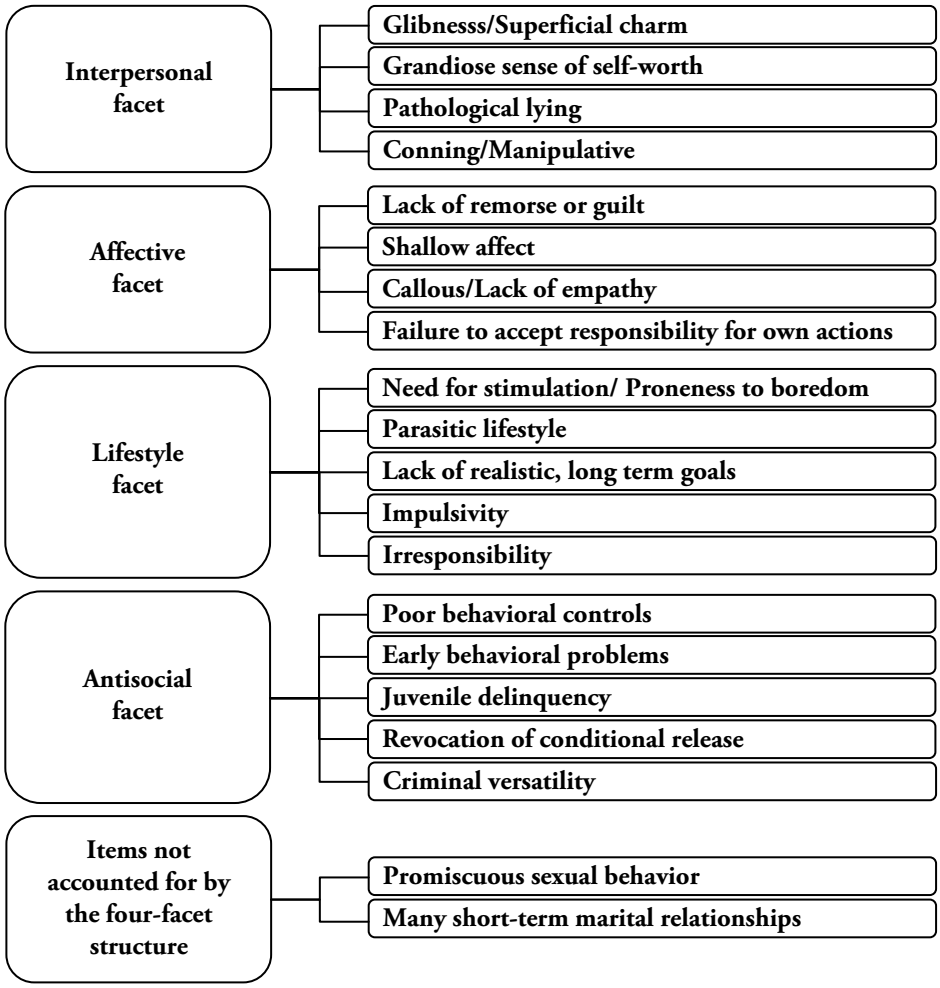
Individuals with highly psychopathic traits have an earlier onset and display more diverse, severe, and persistent patterns of aggressive antisocial behavior than other offenders[85]. Some studies have shown that psychopathy adds incrementally to the prediction of aggressive antisocial behavior when other risk factors (e.g., substance-related disorders, criminal history, personality disorders) are controlled for[86]. Measurements of psychopathy are commonly included in risk assessment guidelines.

Psychopathy is generally defined as a personality disorder comprising interpersonal, affective, lifestyle, and antisocial traits and behaviors[87] (Figure 2). Psychopathy is related, but not equal, to antisocial personality disorder, as its definition places greater emphasis on the interpersonal and affective features first described by Hervey Cleckley[88]. Most offenders with highly psychopathic traits also meet diagnostic criteria for antisocial personality disorder, while most offenders with antisocial personality disorder do not display high levels of psychopathic traits.

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[79] Johnson et al. 2000
[80] Newhill et al. 2012
[81] Berman et al. 1998, Johnson et al. 2000, Pulay et al. 2008
[82] Forth et al. 2003, Hare 1980, Hare 1991, Hare 2003, Hart et al. 1995
[83] Coid & Yang 2011, Doyle et al. 2012, Edens et al. 2007, Guy et al. 2005, Hare et al. 2000, Hemphill et al. 1998, Leistico et al. 2008, Walters 2003
[84] Hare 1991, 2003
[85] Harris et al. 1991, Simourd & Hoge 2000, Tengström et al. 2004
[86] Hemphill et al. 1998, Skeem & Mulvey 2001
[87] Hare & Neumann 2009
[88] Cleckley 1941, Hare & Neumann 2006

Figure 2. Psychopathy as defined by the Psychopathy Checklist-Revised four-facet structure[89].



The modern concept of psychopathy has been operationalized primarily by the work of Robert Hare and colleagues in the development of the psychopathy checklists. However, psychopathy has been recognized for centuries, with the pioneer, Philippe Pinel, introducing the term *manie sans délire* (insanity without delirium) in 1801[90].

[89] Hare 2003
[90] Andrade 2008

Initially, psychopathy as measured by the psychopathy checklists was described by two factors: one reflecting the interpersonal and affective characteristics stressed by Cleckley, and one comprising socially deviant and antisocial behaviors[91]. This structure has since been questioned, and two similar but inherently different models have been proposed: the hierarchical three-factor model[92] and the four-facet model[93].

The hierarchical three-factor model incorporates the interpersonal, affective, and lifestyle features of psychopathy. The proponents of this model argue that the antisocial behaviors not incorporated within this model are a consequence of the core features of psychopathy, and therefore secondary to the construct[94].

The four-facet model (Figure 2) includes the three factors (facets) of the previous model, with the addition of a fourth facet measuring lifetime antisocial behaviors (Antisocial facet). The advocates of this model claim that early-onset and persistent antisocial behaviors are integral and core features of psychopathy[95].

Robert Krueger[96] has suggested that antisocial behavior may be viewed as neither a core feature nor a consequence of psychopathy, but as "an indicator of a different (externalizing) domain that intersects with psychopathy" (p. 196). In this, the externalizing domain refers to a higher order spectrum of mental health problems including aggressive and disinhibitory personality traits and clinical disorders (e.g., oppositional defiant disorder, AD/HD, substance-related disorders, antisocial personality disorder) among children, adolescents, and adults that has been described by Krueger and colleagues as the *externalizing spectrum*[97].

Recently, the predictive ability of psychopathy for aggressive antisocial behavior has been demonstrated to be carried by the Antisocial facet, or social deviance factor, of psychopathy alone[98].

Cognitive distortions

In this thesis, *cognitive distortions* refer to an antisocial thinking style.

How personal and situational susceptibility factors interact to determine aggressive antisocial outcomes may be understood through how individuals process social

[91] Harpur et al. 1989
[92] Cooke & Michie 2001
[93] Hare 2003
[94] Cooke et al. 2004a, Cooke et al. 2006, Skeem & Cooke 2010
[95] Hare & Neumann 2006, Hare & Neumann 2010
[96] Krueger 2006
[97] Krueger 1999, Krueger et al. 2002, Tackett & Krueger 2011
[98] Kennealy et al. 2010, Walters et al. 2008, Walters et al. 2011, Walters & Heilbrun 2010

information[99]. Patterns of social information processes, or cognitive schemas, are established in early childhood[100]. During this period, aggressive behaviors in adult role models (e.g., parents), such as child maltreatment and abuse and domestic violence, form cognitive schemas within the child. These schemas are characterized by the attribution of hostile intentions to others and the perception that self-defensive responses are both effective and desired. With development, these cognitive schemas are consolidated through social interactions with peers (e.g., peer rejection) and adults (e.g., partners).

Antisocial attitudes have been specifically linked to aggressive antisocial behavior[101]. This relation has been described as reciprocal; aggressive antisocial behavior changes the way the individual processes social information about aggressive antisocial behavior, which in turn influences that individual's propensity for aggressive antisocial behavior[102].

Aggression in children has been specifically associated with: a) narrow encoding of environmental cues, b) selective attention to aggressive cues, c) a greater likelihood of attributing hostile intentions to others, d) misinterpreting emotional states of arousal as anger, e) generating fewer possible alternative solutions to problems, f) selecting action-oriented instead of reflective solutions, g) limited interactive skills, and h) an egocentric perspective in solving social problems[103].

Protective factors for aggressive antisocial behavior

Research on protective or compensatory factors against aggressive antisocial behavior is, in contrast to research on risk factors, scarce. Protective factors have been defined as "any characteristic of a person, his/her environment or situation which reduces the risk of future violent behavior" (p. 23)[104].

Protective factors have been suggested to function either by reducing the negative effects of the risk factors or by having an independent, risk-decreasing effect on aggressive antisocial behavior[105]. It is still unclear whether a protective factor should be regarded as equivalent to the absence of a risk factor[106], the opposing end of a risk

[99] Dodge 1980, Huesmann et al. 2011
[100] Dodge 2011
[101] Bandura 1991, Gendreau et al. 1996, Palmer 2007, Sestir & Bartholow 2007, Walters 2002
[102] Dodge 2011, Fontaine et al. 2008, Fontaine & Dodge 2006
[103] McGuire 2009
[104] de Vogel et al. 2007
[105] Rogers 2000
[106] Costa et al. 1999

variable[107], or an independent factor diminishing the risk for aggressive antisocial behavior[108].

Examples of proposed protective factors for aggressive antisocial behavior are self-control, a suitable and stable work situation, and positive attitudes towards authority[109]. The identification of protective factors has been stressed as a major research challenge for the near future[110].

Risk assessments of aggressive antisocial behavior

For several decades, clinicians in psychiatric or forensic settings have been approached with the task of assessing *dangerousness*, i.e., the risk that an individual will harm other persons (or himself/herself). Such assessments have been used for judicial decisions to commit or release individuals to or from the mental health or legal system, as a means of public protection[111]. This practice has been severely criticized for low reliability and validity, and described as equal to "flipping coins in the courtroom"[112].

Two major approaches to assessing the risk of aggressive antisocial behavior are: a) *prediction-only models*, and b) *prediction and risk management models*[113]. Prediction-only models focus on static risk factors such as age, gender, and criminal history. These models are of great value for the prediction of aggressive antisocial behavior, but are unhelpful in risk management interventions. In contrast, prediction and risk management models emphasize dynamic (i.e., changeable) risk factors that can be applied in risk management interventions. Research on dynamic risk factors is emerging, and variables such as substance abuse, antisocial attitudes, impulsiveness, and treatment compliance have been proposed as useful for both the prediction and the prevention of aggressive antisocial behavior[114].

With an increased understanding of aggressive antisocial behavior, methods for more structured approaches to these kinds of assessments have evolved. This development of risk assessment methods (the current term for assessment of dangerousness) are described in terms of three generations: *unstructured clinical judgment*, *actuarial assessment*, and *structured professional judgment*[115].

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[107] Webster et al. 2004
[108] Farrington & Loeber 2000
[109] de Vogel et al. 2007
[110] Farrington 2003
[111] Monahan et al. 2001
[112] Ennis & Litwack 1974, Monahan 1981
[113] Heilbrun 2009
[114] Andrews & Bonta 2010, Douglas & Skeem 2005
[115] Andrade et al. 2009, Mills et al. 2011

Unstructured clinical judgment

For most of the 20th century, risk assessments were performed in mental health settings using unstructured clinical judgment. A clinician (psychiatrist or psychologist) subjectively assessed an individual's risk of aggressive antisocial behavior on the basis of his/her own education, previous experiences, and knowledge on the individual[116].

An early review[117] of the (scarce) research on the accuracy of these assessments revealed discouraging results: "psychiatrists and psychologists are accurate in no more than one out of three predictions of violent behavior" (p. 47). The most common problem with these assessments was the high rate of false positive predictions[118]. Later research, however, demonstrated the predictive ability of the unstructured clinical judgment to be more accurate than chance, although still at a very modest level[119]. Nevertheless, the unstructured clinical judgment approach, although marginally better than chance, can still be criticized for inherent problems with inter-rater and test-retest reliability due to the subjective nature of the assessments[120].

Actuarial assessment

Actuarial risk assessment methods were developed during the 1990s based on research on risk factors for aggressive antisocial behavior. Actuarial assessments are, in contrast to unstructured clinical judgment assessments, based entirely on empirically derived risk factors for aggressive antisocial behavior[121]. In these assessments, the prevalence of (mostly static) risk factors are noted, weighted, and added in an established algorithm, which then provides a probability rate of re-offense. Examples of typical risk factors assessed include age at offense, primary school adjustment, and history of aggressive antisocial behavior. Common actuarial risk assessment guides are the Violence Risk Appraisal Guide (VRAG)[122] and the Classification of Violence Risk (COVR)[123]. The Level of Service Inventory-Revised (LSI-R)[124] and the Violence Risk Scale (VRS)[125] are examples that also emphasize dynamic risk factors.

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[116] Andrade et al. 2009
[117] Monahan 1981
[118] Steadman & Cocozza 1974
[119] Mossman 1994
[120] Hanson & Morton-Bourgon 2009
[121] Heilbrun et al. 2010
[122] Quinsey et al. 1998
[123] Monahan et al. 2005
[124] Andrews & Bonta 1995
[125] Wong & Gordon 1999

Research on actuarial assessment methods has revealed a marginal to modest advantage in predictive accuracy over the unstructured clinical judgment approach[126]. Actuarial assessments have been criticized for being difficult to apply in risk management interventions[127]. Further critiques mention the difficulty of generalizing the specific assessment measure to populations other than those used for the development of the measure.

Structured professional judgment

The structured professional judgment model was developed during the 1990s[128]. In this model, a clinician first assesses the prevalence of empirically derived risk factors for aggressive antisocial behavior according to a guideline. The clinician then makes a final, clinical judgment on risk level (usually: low, moderate, high) by assessing the relevance of the current risk factors. In this model, the clinician can consider individual-specific risk factors that are recognized as relevant for the risk (and prevention) of aggressive antisocial behavior, but that are not included in the guidelines used. Examples of structured professional judgment guidelines are the Historical-Clinical-Risk Management-20 (HCR-20)[129] and the Sexual Violence Risk-20 (SVR-20)[130].

Research has provided support for the predictive validity of structured professional judgment guidelines for aggressive antisocial behavior[131]. However, when this model has been compared with actuarial models, the findings are conflicting[132]. To date, evidence suggests that the predictive accuracies of actuarial and structured professional judgment guidelines are comparable for aggressive antisocial outcome[133].

Using protective factors in risk assessment

Richard Rogers[134] argued that risk assessment of aggressive antisocial behavior without considering protective factors may lead to inaccurate predictions. To date,

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[126] Ægisdóttir et al. 2006, Grove et al. 2000, The Swedish Council on Technology Assessment in Health Care 2005
[127] Heilbrun et al. 2010
[128] Heilbrun et al. 2010
[129] Webster et al. 1997
[130] Boer et al. 1997
[131] de Vogel & de Ruiter 2006, Kropp & Hart 2000, Singh et al. 2011, The Swedish Council on Technology Assessment in Health Care 2005, Welsh et al. 2008
[132] Douglas et al. 2005, Hanson & Morton-Bourgon 2009
[133] Heilbrun et al. 2010
[134] Rogers 2000

some risk assessment guidelines that incorporate protective factors or strengths have been developed. Examples are the Structured Assessment of Violence Risk in Youth (SAVRY)[135] and the Short-Term Assessment of Risk and Treatability (START)[136]. Recently, the Structured Assessment of PROtective Factors for violence risk (SAPROF)[137] was developed with the specific purpose of assessing protective factors in conjunction with risk-focused assessments.

So far, research indicates that assessing protective factors in conjunction with risk assessment leads to a somewhat better predictive validity than using risk-only measures[138]. Aside from providing more balanced risk assessments, an increased focus on protective factors can be beneficial to risk management interventions, because of the emphasis on dynamic factors and a positive, strength-based, and collaborative approach.

Issues in assessing the risk of aggressive antisocial behavior

Several issues need to be considered in the practice of risk assessment. First, the assessor must be aware of the base rate of aggressive antisocial behavior that is applicable in the case at hand. This includes knowledge of a) the population, b) the definition of the behavior assessed, c) outcome measures, and d) the time frame used in establishing the base rate[139]. The assessor needs to remember that the base rate represents an average on the group level, whereas the assessment is made on the individual level. The application of group-averaged risk estimates (as in actuarial risk assessment) on individual level has been criticized for its poor precision, wide confidence intervals, and overlapping categories of risk classification[140]. A recent meta-analysis demonstrated that risk assessment guidelines that were specifically designed for certain populations (e.g., the SAVRY) showed the highest predictive validities[141].

The final clinical judgment, or "clinical override", applied in structured professional judgment guidelines has been subject to controversy. While some studies support an incremental validity of the clinical override over the total score of the risk measure, others show no significant effect on predictive validity[142]. Findings also indicate that

[135] Borum et al. 2006
[136] Webster et al. 2004
[137] de Vogel et al. 2007
[138] de Vogel et al. 2007
[139] Monahan 1981
[140] Hart et al. 2007
[141] Singh et al. 2011
[142] de Vogel & de Ruiter 2006, Douglas et al. 2005, Gore 2008, Kropp & Hart 2000

weighting predictors in actuarial assessments may not be advantageous, as it can result in lowered overall predictive accuracy[143].

The evidence from most research on risk assessment indicates that there might be a "glass-ceiling" effect on the predictive accuracy of empirically derived risk factors[144], with maximum Area Under the Curve (AUC) values at 0.75 to 0.80[145]. Jeremy Coid and colleagues[146] found that most items in common risk assessment guidelines do not independently predict aggressive antisocial behavior. The absolute majority of items that did have an independent, predictive effect were those related to early-onset impulsive and aggressive antisocial behavior. Thus, the assessor needs to be aware of the inherent limitations of risk assessment and be able to differentiate between the predictive and the clinical utility of the existing guidelines.

Finally, it must be noted that statistical association in no way implies causation. To date there exists no evidence-based risk assessment model that can be used to determine risk factors that are causally related to violence.

Comprehensive models of aggressive antisocial behavior

There is obviously a need for a comprehensive model of aggressive antisocial behavior that acknowledges the inherent complexity in a parsimonious way. Several such models have been suggested, and two of them will be mentioned in this thesis: the *WHO ecological model*[147], and the *interactional model* proposed by Kenneth Dodge[148].

In the WHO ecological model, aggressive antisocial behavior is viewed as a result of interaction between individual characteristics, relationships with peers, partners, and family members, the community contexts of the social relationships, and the larger societal circumstances that may increase the likelihood of aggressive antisocial behaviors. The model stresses the necessity of considering multiple causes of aggressive antisocial behavior and the interactions between these causes that may be operating on different levels.

Kenneth Dodge argues that a comprehensive model must account for both environmental and genetic main effects, as well as genetic–environmental interactive effects on aggressive antisocial behavior. Social information processing patterns may

[143] Grann & Långström 2007
[144] Heilbrun et al. 2010
[145] Coid et al. 2011, The Swedish Council on Technology Assessment in Health Care 2005
[146] Coid et al. 2011
[147] Krug et al. 2002
[148] Dodge 2011

mediate these effects in different situations, and thereby lead to a proximal risk of aggressive antisocial behavior.

In comprehensive models of aggressive antisocial behavior, the importance of persistence needs to be considered. Even though we know that persistence in aggressive antisocial behavior is related to adverse outcomes in many respects, knowledge about the determinants of these behaviors is far from complete. Studies on covariates of persistent aggressive antisocial behavior in general population samples and in offender groups are a prerequisite for the advancement of knowledge essential for the prevention of persistent aggressive antisocial behavior.

Aims

General aims

The overall aim of the thesis is to establish covariates of persistent aggressive antisocial behavior in the general population and in offender groups.

Specific aims

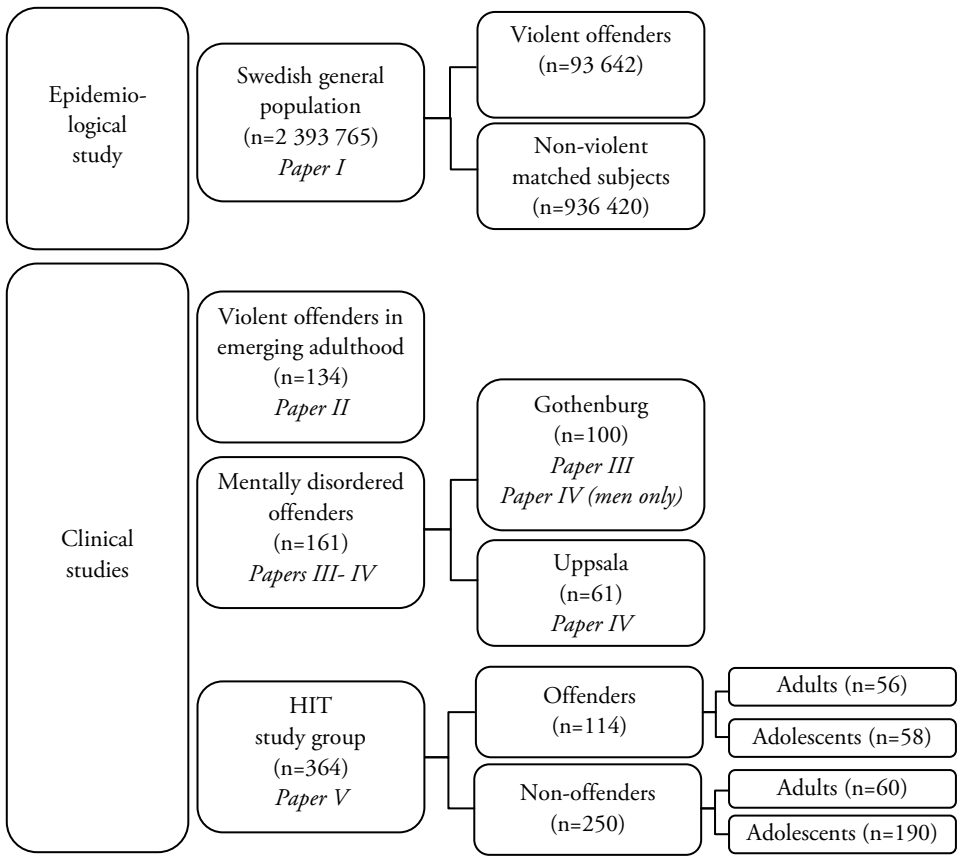
1. Quantify persistence of aggressive antisocial behavior in the general population and offender groups. (*Papers I–IV*)
2. Identify risk factors for persistence of aggressive antisocial behavior. (*Papers I–IV*)
3. Clarify the distribution and clinical covariates of psychopathic traits among offenders. (*Papers II* & *IV*)
4. Determine cognitive covariates of aggressive antisocial behavior. (*Paper V*)

Methods

Subjects

This thesis is based on two types of data: epidemiological data on the Swedish general population (*Paper I*) and clinical data from offender and non-offender groups (*Papers II–V*). The epidemiological data were based on selected birth cohorts from the Swedish general population, while the clinical data were derived from three data sets: violent offenders in emerging adulthood; mentally disordered offenders; and the How I Think (HIT) study group of adult and adolescent offenders and non-offenders (Figure 3).

Figure 3. Structure of the data sets used in the thesis



Swedish general population (Paper I)

A cohort of all individuals born in Sweden between 1958 and 1980, who were living in the country at the age of 15 during the follow-up period of 1973–2004, was identified from the nationwide Multi-generation Register (n=2 393 765). All subjects had to be at least 24 years old at the end of the follow-up period. No first generation immigrants were included in the study to decrease the risk of missing and/or incomplete data.

Subjects convicted of at least one violent offense were identified (n=93 642; 3.9%). For each violent offender, 10 non-violent subjects matched for sex, birth year and month, and having a sibling of the same age and sex as one sibling of the matched offender, were randomly selected from the cohort (n=936 420; 39%). The ratio of men to women was approximately ten to one among the violent offenders and non-violent subjects.

Paper I presents results from the analyses of violent and matched non-violent subjects (n=1 030 062), describing the distribution of violent convictions in the Swedish general population and identifying risk factors for persistence of violent criminality.

Violent offenders in emerging adulthood (Paper II)

Violent offenders in emerging adulthood (n=134) were recruited from an ongoing multicentre study, the DIS-CAT 2.0 study. This study investigates all male offenders aged 18 to 25 years convicted of "hands-on" violent (including sexual) offenses and imprisoned in one out of nine prisons in the western region of the Swedish Prison and Probation Service. Subjects with insufficient knowledge of Swedish or very short stays (<2 weeks) at the prisons were excluded from the study as they could not participate in the clinical examinations. The DIS-CAT 2.0 study started in February 2010 with the aim of studying early-onset behavior and mental disorders in a consecutively recruited cohort of violent offenders in emerging adulthood. The study is set to include a total of 270 offenders.

The response rate, calculated for the total database collected until January 20, 2012 was 72%, as 85 offenders out of 299 offenders that had met inclusion criteria declined participation.

Paper II is based on the first 134 subjects (mean age=22 years) from the DIS-CAT 2.0 study for whom data compilation had been completed in April 2012. *Paper II* investigates the characteristics of violent offenders in emerging adulthood, including covariates of persistent aggressive antisocial behavior. This paper is the first to report on the DIS-CAT 2.0 group, which will also be followed prospectively over several years.

Mentally disordered offenders (Papers III–IV)

Two independent study groups of forensic psychiatric investigees, the Gothenburg group (n=100) and the Uppsala group (n=61) were used for analyses on mentally disordered offenders. All subjects had been found guilty in a court of law of serious crimes, and had sufficient mental health problems to be referred to a forensic psychiatric investigation. All had sufficient fluency in Swedish to participate in the clinical examinations. The subjects might have been found guilty of more than one type of crime (violent, sexual, and non-violent) at the time of the forensic psychiatric investigation, so the possible overlap of crime categories needs to be considered in interpreting the prevalence rates reported below.

In *Paper III*, the Gothenburg group is studied in a long-term follow-up study of violent recidivism. *Paper IV* analyses associations between facets of psychopathy, mental disorders, personality traits, and criminal recidivism in the men from the Gothenburg (n=92) and Uppsala groups (n=61), from now on known as the *combined group of mentally disordered offenders*.

The Gothenburg group

The Gothenburg group comprised 100 consecutively enrolled perpetrators of severe violent (n=77; 77%) and/or sexual crimes (n=26; 26%) referred to a pre-trial forensic psychiatric investigation at the Gothenburg state forensic psychiatry unit between the years 1998 and 2001. The subjects had a mean age of 34 years. The index crimes used as inclusion criteria were: homicide/manslaughter, attempted homicide/manslaughter, aggravated assault, aggravated unlawful threat, robbery, arson, rape, and sexual offenses against minors. No subjects were excluded because of their clinical presentation or for administrative reasons. The response rate was 83% as 21 of 121 eligible subjects declined participation.

The subjects were originally recruited for the Gothenburg Forensic Neuropsychiatry Project, aimed at examining patterns of neuropsychiatric vulnerability factors of relevance to criminal behavior. Data on the group, mainly on neuropsychiatric and biological covariates of violent criminality[149], have been reported previously, including in two theses[150].

[149] Söderström et al. 2003, 2004, 2005
[150] Gustavson 2010, Söderström 2002

The Uppsala group

The Uppsala group comprised 61 consecutively recruited men who during the years 1992 to 1994 were court-referred for a pre-trial forensic psychiatric investigation at the state forensic psychiatry unit in Uppsala. The group included perpetrators of violent (n=44; 72%), sexual (n=12; 20%), and/or nonviolent (n=31; 51%) crimes. Subjects with psychotic disorders, severe somatic disorders, and mental retardation were excluded (n=103). Another 36 individuals were excluded for administrative reasons. As only 8 of a total of 69 eligible subjects refused participation, the response rate was 88%. The mean age of the subjects was 34 years.

Data on the Uppsala group were originally collected for a thesis on biological markers of psychopathy-related personality traits and the influence of psychopathy on mental disorders and related behavioral symptoms[151]. Results from this group have previously been reported in studies on psychopathy, biological markers, and temperamental vulnerability[152].

HIT study group (Paper V)

The HIT study group comprised four groups of Swedish adult and adolescent offenders and non-offenders (n=364). The adult subjects were recruited from male prison inmates (n=56, mean age 37 years) in two low- to medium-security prisons in southern Sweden and male students (n=60, mean age 20 years) from an engineering program at a university in southern Sweden. Among the offenders, the majority (93%) of the inmates approached at the first prison agreed to participate. Thus, only a smaller number was recruited from the other prison, drawn from those who first showed interest. The university students were recruited according to the same principle as the offenders from the second prison—drawn from the first to declare interest in participating. The adult offenders reported a history of mostly drug-related offenses (84%), violent offenses (63%), and theft (61%). Three (5%) of the adult university students reported previous criminality.

The adolescent subjects were adolescents incarcerated under the *Care of Young Persons Act* (SFS 1990:52) in Sweden (n=58, mean age 16 years) and non-incarcerated adolescents from primary and secondary education facilities in Sweden (n=190, mean age 15 years). The ratio between male and female subjects was 50:50 among the adolescent offenders, and 57:43 among the adolescent non-offenders. The adolescent offenders were recruited by ward managers based on their perceived eligibility, while

[151] Stålenheim 1997
[152] Stålenheim 2001, 2004, Stålenheim & von Knorring 1996

the adolescent non-offenders were recruited from whole school classes. All adolescent offenders displayed antisocial behavior.

Select data from individuals in the adolescent groups have previously been reported in a study on moral judgment, empathy, and cognitive distortions[153].

Paper V examines cognitive distortions among the adult and adolescent offenders and non-offenders.

Measures

Three main types of measures (Table 4) were collected and used for the analyses in this thesis:

1. *Retrospective information* covering epidemiological, sociodemographic, and criminal history data (*Papers I–V*)
2. *Clinical measures* supplying information on mental disorders, personality disorders and personality traits, and aggressive antisocial behavior, including risk assessment (*Papers II–V*)
3. *Prospective follow-up data* on criminal recidivism and mortality (*Papers III–IV*)

The majority of the studies had a cross-sectional, retrospective design assessing lifetime occurrence of the characteristics investigated. However, the mentally disordered offenders were also followed prospectively.

Paper I is based solely on epidemiological data. *Papers II* to *IV* used a small group of clinical assessors (psychiatrists/clinical psychologists) to collect data using highly similar methods during clinical interviews and examinations (*Paper II*) or forensic psychiatric investigations (*Papers III* & *IV*). *Papers III* and *IV* also used nationwide official registers for the collection of follow-up data on criminal recidivism. *Paper V* relied solely on self-report measures.

[153] Lardén et al. 2006

Table 4. Measures used in the thesis

Type of measure	Paper I	Paper II	Paper III	Paper IV	Paper V
Retrospective information	Nationwide registers: Multi-generation Register Total Population Register Cause of Death Register Hospital Discharge Register Migration Registers Crime Register Compulsory 9-year Comprehensive School Register	Interviews & files: Sociodemographic data Psychosocial background Criminal history	Forensic psychiatric investigation: Sociodemographic data Psychosocial background Criminal history	Forensic psychiatric investigation: Sociodemographic data Index offense	Self-report: Sociodemographic data Criminal history
Clinical measures	N/A	SCID-I (DSM-IV) SCID-II (DSM-IV) DSM-IV interview protocol PCL-R LHA	SCID-I (DSM-IV) SCID-II (DSM-IV) DSM-IV interview protocol PCL-R LHA HCR-20	SCID-I (DSM-III-R & DSM-IV) SCID-II (DSM-III-R & DSM-IV) KSP PCL-R	SCID-II Screen HIT
Prospective follow-up data	N/A	N/A	Crime Register National Prison and Probation Administration Central Archives of the National Board of Forensic Medicine National Board of Health and Welfare	Crime Register	N/A

Retrospective information

Epidemiological data

For the collection of epidemiological data (*Paper I*), Swedish longitudinal total-population registers were linked through the unique personal identification number that all Swedish citizens are assigned.

The Multi-generation Register was used to identify all individuals and their biological or adoptive parents within the selected birth cohorts. The Cause of Death and Migration Registers provided data on whether the individuals were alive and residing in Sweden during the follow-up period of 1973 to 2004. Records of all convictions in the Swedish lower courts during the follow-up period were obtained from the Crime Register, comprising both custodial and non-custodial sentences. The Total Population Register provided information on sex, birth year, and parents' country of birth. Information on school grades from the final compulsory school year was obtained from the Compulsory 9-year Comprehensive School Register. Finally, the Hospital Discharge Register provided information on mental disorder diagnoses at discharge from hospital during the follow-up period, according to WHOs ICD-8/ICD-9 (codes 290-319) and ICD-10 (codes F00-F99).

Sociodemographic data

In *Paper II*, detailed sociodemographic data covering age, ethnicity, and psychosocial background including adverse childhood circumstances, schooling, institutionalization during childhood/adolescence, and previous contacts with the mental health care system, were collected from interview and file information by the means of a structured protocol, similar to the protocol used in *Paper III*.

For *Papers III* and *IV*, basic sociodemographic data on all subjects were retrieved from the forensic psychiatric investigations. During these investigations, all medical and psychiatric files, criminal and social records, previous forensic and psychological assessments, and police reports on the index crimes, were reviewed.

In *Paper III*, a structured research protocol was used to compile more detailed information on psychosocial background from the forensic psychiatric investigations, including aggravating circumstances during childhood. In *Paper V*, sociodemographic data on the adult subjects were obtained by means of a self-report questionnaire covering age and level of education.

Criminal history data

Detailed information on previous criminality including age at onset, number of previous convictions and prison convictions, and number of previous crimes was gathered using a structured research protocol based on all information available from interviews and files in *Papers II* and *III*. The use of self-report data in combination with file reviews in *Paper II* made it possible to account for criminality prior to age 15, as these offenses would not have been available in official crime registers. Fifteen years of age is defined as the age of criminal responsibility in Sweden.

Number of previous crimes was measured on a 3-point scale (no occasion; single occasion; multiple occasions) in *Paper II*, and on a 4-point scale (no crime; 1 crime; 2–4 crimes; ≥5 crimes) in *Paper III*. In *Paper IV*, information on index offense was retrieved from available files, while *Paper V* collected information on previous convictions for all adult subjects with a self-report questionnaire. The questionnaire had a dichotomous answer format for the non-offenders (yes/no) and more detailed questions about the type and number of previous convictions (1 conviction; 2–3 convictions; 4–7 convictions; ≥8 convictions) for the offenders.

Violent criminality was defined similarly in *Papers I* to *V*, as comprising all harmful interpersonal crimes, including attempted and aggravated forms of homicide, manslaughter, assault, unlawful threat, robbery, threats and violence against an officer, gross violation of a woman's or an individual's integrity, unlawful coercion, kidnapping, illegal confinement, arson, and intimidation. However, in *Papers II* to *V*, sexual offenses of a "hands-on" and violent nature were also included in the definition of violent criminality. In *Paper II*, previous criminality was divided into six categories: 1) violent offenses (homicide/manslaughter, assault, unlawful threat, robbery, sexual offenses, and fire setting/arson), 2) sexual offenses, 3) drug-related offenses, 4) property offenses (theft, breaking and entering, and vandalism), 5) traffic violations (driving under the influence, and driving without a license), and 6) fraud.

Persistence in aggressive antisocial behavior was defined as three or more convictions for violent crimes in *Paper I*, and as "multiple occasions" (≥2) in *Paper II*. In *Papers III* to *IV*, persistence was defined as a reconviction for violent and/or non-violent criminality. Two types of persistence are described in the results section: a) persistence in violent criminality, referring to all types of violent offenses (as defined above), and b) persistence in general criminality, referring to any type of offense.

Clinical measures

Mental disorders

The *Structured Clinical Interview for Axis I disorders (SCID-I)*[154] was used to obtain diagnostic information on mental disorders (*Papers II–IV*). The semi-structured interview covers the symptoms of the major mental disorders item-by-item, and is meant to be performed by a trained clinician or mental health expert. Most disorders covered in the SCID-I interview are evaluated both for current status (meets criteria for the disorder during the last month) and for life-time status, i.e., whether the individual has ever met the criteria for the disorder. In this thesis, only life-time data on categorical diagnoses were used for the analyses. Trained clinicians performed SCID-I interviews for all subjects among the violent offenders in emerging adulthood and the mentally disordered offenders, with the exception of 16 subjects in the Gothenburg group, who due to psychosis were too disorganized to participate in the full interview.

Diagnostic information on mental disorders, usually with onset during childhood and/or adolescence, not covered by the SCID-I, such as AD/HD, autism spectrum disorders, and tic disorders, was retrieved via a structured DSM-IV-based interview using the same procedure as the SCID-I among the violent offenders in emerging adulthood and the Gothenburg group of mentally disordered offenders (*Papers II–III*).

Final, categorical diagnoses of mental disorders, including childhood onset mental disorders, were assigned based on all information available: a) information from the clinical interviews described above (*Papers II–IV*), and b) file information obtained during the forensic psychiatric investigations (*Papers III–IV*) or provided by the Swedish Prison and Probation Service (*Paper II*). All diagnostic assessments of the violent offenders in emerging adulthood and in the Gothenburg group of mentally disordered offenders were performed regardless of diagnostic criteria limiting the possibility of assigning co-morbid diagnoses, and all were reviewed and ensured by a senior clinician.

Personality disorders and personality traits

Information on personality disorders was obtained from semi-structured clinical interviews using the *Structured Clinical Interview for Axis II disorders (SCID-II)*[155] in *Papers II–IV*. The SCID-II interviews were performed according to the procedure described previously for the SCID-I. The final diagnostic assessments of personality

[154] First et al. 1996, Spitzer et al. 1990a
[155] First et al. 1997, Spitzer et al. 1990b

disorders were based on all information available from files and interviews and ensured by a senior clinician.

In *Paper V*, the *Structured Clinical Interview for DSM-III-R Axis II Disorders, Screening Version (SCID-II Screen)*[156] was used for the adult subjects as a self-report measure of aggressive antisocial behavior as defined by conduct disorder and antisocial personality disorder. The SCID-II Screen is a 123-item self-report questionnaire intended to cover the criteria for DSM-III-R personality disorders (yes/no) and to provide an initial screening of individuals in need of more detailed diagnostic assessments[157]. In *Paper V*, only the questions pertaining to conduct disorder during childhood and antisocial personality disorder were administered.

The *Karolinska Scales of Personality (KSP)*[158] was used to obtain self-reported measurements of personality traits related to psychopathy (*Paper IV*). The KSP was originally developed as a tool in research on psychopathy, with the specific aim to find biological correlates to personality traits[159]. The KSP contains 135 items, designed as statements rated on a 4-point Likert scale (from "does not apply at all" to "applies completely"). The items are grouped into 15 subscales: Somatic Anxiety, Psychic Anxiety, Muscular Tension, Social Desirability, Impulsiveness, Monotony Avoidance, Detachment, Psychasthenia, Socialization, Indirect Aggression, Verbal Aggression, Irritability, Suspicion, Guilt, and Inhibition of Aggression.

Three overarching factors were used in the analyses on KSP data: the Psychopathy factor comprising the impulsive and sensation-seeking scales, the Aggression factor based on all aggression-related scales, and the Hostility factor drawn from scales related to suspicion and guilt.

The *Psychopathy Checklist-Revised (PCL-R)*[160] was used for the assessment of psychopathic traits in *Papers II to IV*. The PCL-R is a 20-item rating scale designed to assess psychopathic personality traits and behaviors in clinical, forensic, and research settings. The items are rated on a 3-point scale (0=does not apply, 1=may apply or in some respects applies, 2=does apply) based on information from a semi-structured interview, files, and collateral informants. A PCL-R total score (range 0–40) of 30 points is considered the established cut-off for a high level of psychopathic traits, however, a score of 25 points has been suggested as a more appropriate cut-off in

[156] Spitzer et al. 1991
[157] Spitzer et al. 1989
[158] Schalling & Edman 1993
[159] Gustavsson 1997
[160] Hare 1991, 2003

European contexts[161], as offenders in European settings generally score lower on the PCL-R than their North-American counterparts.

The PCL-R ratings in this thesis were based on all information available from interviews, files, and registers, and performed by raters formally authorized to use the PCL-R. In *Papers II* and *IV* the four-facet structure (Interpersonal, Affective, Lifestyle, and Antisocial; Figure 2) suggested by Hare[162] was used in the analyses. *Paper III* studied only the PCL-R total score.

Cognitive distortions were measured by the *How I Think questionnaire (HIT)*[163] in *Paper V*. The HIT is a 54-item self-report questionnaire that was developed with the aim of measuring cognitive distortions associated with externalizing behaviors[164]. The subjects respond along a 6-point Likert scale (from ''agree strongly'' to ''disagree strongly''). Higher scores reflect higher levels of cognitive distortions.

The questionnaire contains 39 statements pertaining to cognitive distortions, 8 items screening for aberrant or suspect responses, and 7 items with prosocial statements—"positive fillers"— to counterbalance the distortion items. The eight control items are reverse-scored, so that higher scores reflect more pronounced anomalous responding.

The 39 distortion items are divided into four subscales of cognitive distortions; Self-Centered, Blaming Others, Minimizing/Mislabeling, and Assuming the Worst. Each distortion item also refer to one of four behavioral subscales derived from the DSM-IV conduct disorder and oppositional defiant disorder syndromes: Opposition-Defiance, Physical Aggression, Lying, and Stealing, thus providing both a cognitive distortion and an antisocial behavioral dimension in the HIT. The HIT has previously shown promising psychometric characteristics for adolescent groups within various contexts[165].

Aggressive antisocial behavior and risk assessment

The *Life History of Aggression* questionnaire *(LHA)*[166], originally developed for research on neurobiological correlates to aggression, was used for the assessment of lifetime aggressive antisocial behavior in *Papers II* and *III*. The LHA measures the lifetime occurrence of 11 different types of aggressive behaviors and can be rated both as a self-report measure and by clinicians or collateral informants with profound knowledge of the subject. The items are rated on a 5-point scale based on the number

[161] Cooke et al. 2005
[162] Hare 2003
[163] Barriga et al. 2001
[164] Barriga et al. 2000
[165] Barriga & Gibbs 1996, Barriga et al. 2008, Nas et al. 2008, Plante et al. 2012
[166] Brown et al. 1982

of occurrences of the behavior since adolescence (0=no occurrences; 5=more events than can be counted), rendering a total score ranging from 0 to 55[167].

The LHA comprises three subscales; Aggression, Self-directed aggression, and Antisocial behavior. The total score is equal to the sum of the three subscales. The Aggression scale includes items measuring temper tantrums, physical fights, verbal aggression, physical assaults on people or animals, and assaults on property. The Self-directed aggression scale contains items on self-injurious behavior and suicide attempts, while the Antisocial behavior scale describes school disciplinary problems, problems with supervisors at work, and antisocial behavior with or without police involvement. A LHA total score above 15, or an Aggression score above 12, is considered indicative of abnormally high levels of lifetime aggressive antisocial behaviors.

In *Paper II*, the LHA was used as a clinician-rated measure, and the rating was based on all available information from records and interviews. In *Paper III*, the LHA was first administered as a self-report measure, after which information from the forensic psychiatric investigation and files were used in a clinician-rating of the LHA. The LHA total score used in the analyses in *Paper III* is based on the average of the self-reported and the clinician-rated value.

The *HCR-20*[168] was rated as a measure of risk for violent recidivism in *Paper III*. The HCR-20 was designed to structure clinical judgments about the likelihood of future violent behavior and to inform preventive strategies. The scheme contains 20 items divided into three subscales: Historical, covering historical data; Clinical, comprising assessments of the subject's current clinical state; and Risk Management, detailing the individuals adjustment to plausible circumstances considered to be important in assessing the risk of violent recidivism. The items are rated, based on information from interviews, files, and collateral information, on a 3-point scale (from "not present" to "definitely present"). The raters should be experts in conducting mental health assessments and familiar with risk-assessment research.

In *Paper III*, only the 15 historical and clinical items of the HCR-20 were rated by trained assessors because the risk management items could not be rated due to their focus on treatment and risk management plans that had not yet been developed at the time of participation.

[167] Coccaro et al. 1997
[168] Webster et al. 1997

Prospective follow-up data

Data on criminal recidivism used in *Papers III* and *IV* were obtained from the Crime Register. For *Paper III*, data on violent recidivism during ongoing sanction (forensic psychiatric care/prison) were also collected from the National Prison and Probation Administration and the Central Archives of the National Board of Forensic Medicine.

Recidivism (*Papers III–IV*) was defined as at least one reconviction, and violent recidivism counted all reconvictions for violent and aggravated violent crimes (e.g., homicide, manslaughter, assault, robbery, arson, exposing someone to danger, sexual offenses of a "hands-on" nature, and intimate partner violence).

Information about mortality and causes of death was collected from registers provided by the National Board of Health and Welfare (*Paper III*).

The average follow-up periods were 4 to 6 years after forensic psychiatric investigation for the Gothenburg group, and 6 to 8 years for the Uppsala group.

Analytical methods

All data were anonymized, coded, and analyzed with SPSS 15.0, 20.1, or PASW 18.0 software, using two-tailed *p*-values. Confirmatory factor analyses were performed using AMOS[169]. Non-parametric and parametric statistics were applied depending on the distribution of the data. The majority of the data collected in the thesis were on a nominal (e.g., diagnoses of mental disorders) or ordinal (e.g., educational level, PCL-R facet scores) level, with only a few measures on an interval or ratio level (e.g., age) according to the NOIR concept (Nominal, Ordinal, Interval, and Ratio)[170]. This limited the statistical methods available for analysis. However, because many statistical methods, especially non-parametric statistics, are rather stable, the distribution—rather than the NOIR-nature of the data—guided the choice of statistical analyses used in the thesis.

Analysis of between-group differences

Between-group differences on nominal data were analyzed with χ^2-tests in the Swedish general population study (*Paper I*) and Fischer's exact test in the smaller clinical groups (*Papers II–IV*). Both tests compare the observed frequencies that occur in each of the categories with the values that would be expected if there were no

[169] Arbuckle & Wothke 1999
[170] Stevens 1946

association between the variables tested (the *null hypothesis*). Fischer's exact test is preferred for smaller samples[171]. When comparing ordinal, interval, or ratio data between groups, Student's *t*-test was used for comparisons of means when criteria for parametric statistics were fulfilled (*Paper V*), and Mann-Whitney U-tests were used for comparisons of medians in skewed data (*Papers II* & *IV*).

Analysis of variance

In *Paper V*, differences between groups on cognitive distortions were further examined with a three-way analysis of variance (ANOVA) with Tukey-Kramer's post hoc test. ANOVAs are used to compare the variance in means between different groups (in this case three) while controlling for the variability within each of the groups[172]. A statistically significant F-ratio >1 indicates that there is more variability between the groups than within them, thereby rejecting the null hypothesis. Tukey-Kramer's post hoc test can be used to test which between-group differences are statistically significant.

Cronbach's alpha

In *Paper V*, Cronbach's alpha was used to investigate the internal consistency of the HIT questionnaire. Cronbach's alpha measures the correlations between all items in a scale by correlating all possible split halves, thereby examining to what extent the items measure the same underlying construct. Ideally, an alpha should be around 0.9 and not below 0.7[173].

Correlation analysis

Relationships between variables, with at least one variable on the interval level, were examined with Pearson correlations (r) in *Paper V*. The non-parametric alternative, Spearman's rank-order correlations (r_s) was used for analyses of relationships between variables on an ordinal scale or with a skewed distribution (e.g., LHA and PCL-R scores) in *Papers II* to *V*. Correlation analysis is used to quantify the strength and direction (positive or negative) of a linear relationship between two variables by examining the ratio between the covariance between the two variables and the total dispersion across the variables (range –1.00 to 1.00). A correlation coefficient between

[171] Clark-Carter 2004
[172] Pallant 2007
[173] Kline 2000

0.10 and 0.29 is generally considered small, between 0.30 and 0.49 is considered medium, and 0.50 and above is considered large[174]. It is important to note that correlation is not equal to causality.

Regression models

Binary logistic regressions were used to assess the impact of different sets of predictor variables on aggressive antisocial behavior in *Papers I*, *III*, and *IV*. Logistic regression is a statistical technique examining relationships between variables, similar to correlation analysis. However, in logistic regression more sophisticated analyses of the interrelationship between variables can be performed, such as testing the impact of different variables on the dependent variable while controlling for the effects of the other variables, thereby identifying the variables that hold the best predictive power. In using regression models, it is important to be aware of the effects of multi-collinearity (intercorrelations among the predictor variables), as this can corrupt the analysis[175].

In *Papers I*, *III*, and *IV* variables that in previous analyses had shown a relationship with aggressive antisocial behavior were entered into the equations, and Odds Ratios (ORs) were calculated. A statistically significant OR value ≥1 shows that the predictor investigated is associated with an increased likelihood of aggressive antisocial behavior, while an OR value <1 indicates that the variable predicts a decreased likelihood of aggressive antisocial behavior. For instance, an OR value of 4 would mean that a subject with the studied predictor (e.g., antisocial personality disorder) would be four times more likely to show aggressive antisocial behavior than a subject not showing the predictor. In *Paper I*, risk factors differentiating between low-persistence and medium-to-high persistence offenders were included in a multivariate logistic regression.

Receiver operating characteristics analysis

Receiver operating characteristics (ROC) analyses were performed to examine the ability of different variables to predict aggressive antisocial behavior (*Papers II–V*). ROC analysis stems from signal detection theory[176], and presents a graph over all possible cut-off scores of the variables investigated, with the true positive rate (sensitivity) plotted against the false positive rate (1 minus specificity). The total area

[174] Cohen 1988
[175] Pallant 2007
[176] Hanley & McNeil 1982

under the curve in the graph (AUC) can be used as a measure of the overall predictive accuracy of the variables tested, i.e., the probability that a randomly selected aggressive antisocial subject would score higher on the variable measured (e.g., PCL-R score) than a randomly selected non-aggressive subject.

An AUC of 0.50 represents chance prediction, while an AUC of 1.0 indicates perfect prediction. In risk assessment research, AUC values of 0.70 and above are generally considered as moderate to large, and those over 0.75 are considered large[177]. However, some researchers have proposed a more conservative interpretation of the AUC values, in which scores <0.60=low accuracy, 0.60–0.70=marginal accuracy, 0.70–0.80=modest accuracy, 0.80–0.90=moderate accuracy; and >0.90=high accuracy[178]. ROC analyses are insensitivity to base rates, which is an advantage for comparisons of scales or instruments, while it is a disadvantage that it does not assess the predictive value in individual cases.

In *Paper III*, sensitivity (true positives), specificity (true negatives), positive predictive values (PPV), and negative predictive values (NPV) were derived from the logistic regression analyses and the optimal inflection point in the ROC analyses (i.e., the cut-off where the trade-off between sensitivity and specificity is optimal). The PPV equals the percentage of subjects that the model accurately predicts to have the characteristic studied (e.g., aggressive antisocial behavior), while the NPV gives the percentage of subjects that the model accurately predicts not to have the characteristic[179].

Survival analysis

Kaplan-Meier survival analysis is a statistical method that describes the time until a certain event (e.g., violent recidivism) for two or more groups and also accounts for data censored due to follow-up ending before the event has occurred[180]. This results in a plot, the "survival curve," for the groups of interest. To test whether the groups differ in their "survival rate," a non-parametric log-rank test, comparing the estimates of the hazard functions (i.e., the tendency to relapse in aggressive antisocial behavior) of the two groups at each observed event time, can be performed.

In *Paper III*, Kaplan-Meier survival analysis was used to graphically describe patterns of violent recidivism among two sanction groups of mentally disordered offenders. Following this, a log-rank test was used to compare whether the groups differed significantly in their patterns of recidivism.

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[177] Douglas et al. 2007
[178] Sjöstedt & Grann 2002
[179] Pallant 2007
[180] Walters 2009

A disadvantage of using Kaplan-Meier survival analysis in research on aggressive antisocial behavior is that it only counts the time until first relapse; therefore, a pattern of repeated recidivism cannot be analyzed. Furthermore, the Kaplan-Meier technique is sensitive to base rates, resulting in a greater risk of *type II errors* (the failure to reject the null hypothesis when it is, in fact, false; i.e., to believe that the groups do not differ when they actually do), and it cannot be used to study interaction effects.

Factor analysis

Confirmatory factor analyses (CFA) were performed in *Paper V* to test the theoretical structure of the cognitive distortions measured by the HIT questionnaire. CFA is a structured equation modeling technique that examines whether the latent structure of an instrument fits a proposed structure by testing how well the proposed structure captures the item covariance matrices of the instrument[181].

In conducting the CFA, several statistics are calculated to determine the fit of the model to the data. A χ^2-test gives the amount of difference between the expected and the observed covariance matrices, with a χ^2-value close to zero indicating that there is little difference between the expected and the observed covariance matrices. The Root Mean Square Error of Approximation (RMSEA), a measure related to the residual in the model, indicates model fit to data where a smaller RMSEA value (range 0–1) indicates a better fit. In order to claim that the instrument holds an acceptable model fit, the RMSEA needs to be ≤ 0.06[182].

Multiple testing

In the clinical studies, the risk of committing *type I errors* (rejection of the null hypothesis when it is, in fact, true; i.e., to falsely believe that there is a difference when there is none) had to be weighed against the risk of type II errors. Bonferroni corrections were considered, but would have involved too high a risk for type II errors due to their conservative nature[183]. Instead, the level of significance was set to $p \leq 0.01$ in *Papers II* and *IV* to adjust the risk of type I errors while preserving enough power to detect meaningful associations. The effect of multiple testing was considered for all papers during the interpretation of the results.

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[181] Suhr, accessed 2012-02-13
[182] Hu & Bentler 1999
[183] Bland & Altman 1995

Power

For *Paper I*, no a priori power analysis was performed since the study considers selected birth cohorts from the entire Swedish population, and can therefore be considered to access enough subjects to avoid the risk of committing type II errors. As in *Paper I*, no a priori power analyses were performed for *Papers II* to *IV* as they present data from cohorts (*Papers III–IV*), or part of cohorts (*Paper II*), thereby naturally limiting the possible number of participants. For *Paper II*, power analysis was deemed unnecessary as the main aim was descriptive. For *Papers III* and *IV*, previous publications support the notion that these groups hold enough power to detect meaningful relationships[184]. In *Paper V*, a priori power analyses were performed because of the use of CFA, a method requiring a large number of subjects.

When considering statistical power it is also important to take the effect size into consideration as large samples can result in even very small differences reaching statistical significance[185]. For this reason, investigating effect sizes could indicate whether the results obtained are of clinical importance, regardless of their statistical significance. This seems to be especially important to consider in clinical research, where sample sizes often are restricted due to clinical circumstances.

Ethical aspects

The studies were approved by the local Research Ethics Committees at Karolinska Institute (*Paper I*, Dnr 521-2010-2689), Lund University (*Paper II*, Dnr 2009/405), University of Gothenburg (*Papers III* & *IV*, Dnr 724-96), Uppsala University (*Paper IV*, Dnr 310/91), and Linköping University (*Paper V*, Dnr 202/04), and carried out according to the Declaration of Helsinki.

The results presented in this thesis have implications that might affect screening and prevention strategies for aggressive antisocial behavior in society, thereby possibly affecting individuals who display aggressive antisocial behavior. However, in the balancing of risks and benefits from an ethical perspective, the potential benefits to society of a better understanding of aggressive antisocial behavior, that in the longer run might facilitate the development of interventions aimed at preventing these kinds of behaviors, are considered large enough to counterbalance the possible risks to the subjects participating in the studies included in this thesis.

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[184] Söderström et al. 2005, Stålenheim 2004
[185] Clark-Carter 2004

All subjects, except those studied in *Paper I*, who contributed to this thesis were informed about the studies they participated in both orally and in writing, and were given the opportunity to ask questions before providing informed consent for their participation. For the adolescent groups (*Paper V*), parental informed consent was required and collected. All subjects were informed of their ability, at any given time and without being required to give a reason, to discontinue their participation in the studies. In *Papers II* to *V*, subjects institutionalized either in forensic psychiatric care, by the Swedish Prison and Probation Service, or under the Swedish Care of Young Persons Act, were included. As these people were subjected to non-voluntary confinement and thereby vulnerable from the perspective of research ethics, special care was taken with the provision of information and the collection of consent in order to ensure their independent and uncoerced consent. For instance, all confined subjects were given clear information that their participation in or withdrawal from the study would not affect their sentence or their treatment. Furthermore, the subjects in *Papers II* to *IV* were given the opportunity to participate only in the parts of the corresponding study that they chose and to refrain from other parts. In *Paper II*, all subjects were given the opportunity to receive feedback on the preliminary results of their clinical interviews and examinations, and to be given a referral to a medical doctor (psychiatrist, if possible) for continued assessment and treatment of their mental health problems.

In two studies (*Papers II* & *V*), the adult subjects were compensated materially for their participation in the study. The violent offenders in emerging adulthood (*Paper II*) were given approximately €20 after their participation as a compensation for time spent with the clinical assessor. The adult subjects in *Paper V* were given a national lottery ticket (worth approximately €2,5) as a token of our appreciation. These compensations were considered small enough not to compromise the participants' free consent. The mentally disordered offenders (*Papers III* & *IV*) did not receive any compensation for their participation.

To protect the confidentiality and integrity of the subjects contributing to the thesis, all data were anonymized using coded files with the code keys stored separately. Analyses were then performed on computer files with identification numbers that cannot be directly linked to the participating subjects.

Results

Persistence of aggressive antisocial behavior

Swedish general population (Paper I)

In the cohort (all people born in Sweden between 1958 and 1980; n=2 393 765) followed from 1973 to 2004, 1% of the Swedish general population (n=24 342) accounted for 63% of all violent convictions and 59% of all aggravated violent convictions. The base rate for ever being convicted of a violent crime was 3.9% (n=93 642).

Three groups of violent offenders (Table 5) were distinguished based on the distribution of violent convictions: *low-persistence offenders* (1–2 convictions) corresponding to 2.9% of the cohort (n=69 300), *medium-persistence offenders* (3–10 convictions) accounting for 0.9% of the cohort (n=21 530), and *high-persistence offenders* (≥11 convictions) comprising 1‰ of the cohort (n=2812).

The reconviction rate for violent offenses increased with each number of violent convictions. Among those with three violent convictions, 68% were reconvicted, while approximately 80% in the most persistent group of the cohort were reconvicted. If the pattern of violent recidivism could have been stopped at three violent convictions, 53% of all violent convictions during the follow-up period would have been prevented.

The median age at first violent conviction was 21 years in men and 22 years in women.

Violent offenders in emerging adulthood (Paper II)

Eighty-seven percent (n=110) of the violent offenders in emerging adulthood had previously been convicted (any type of crime), while 69% (n=88) manifested persistent violent criminality. Approximately half of the subjects (n=69) reported previous criminality in at least four different crime categories.

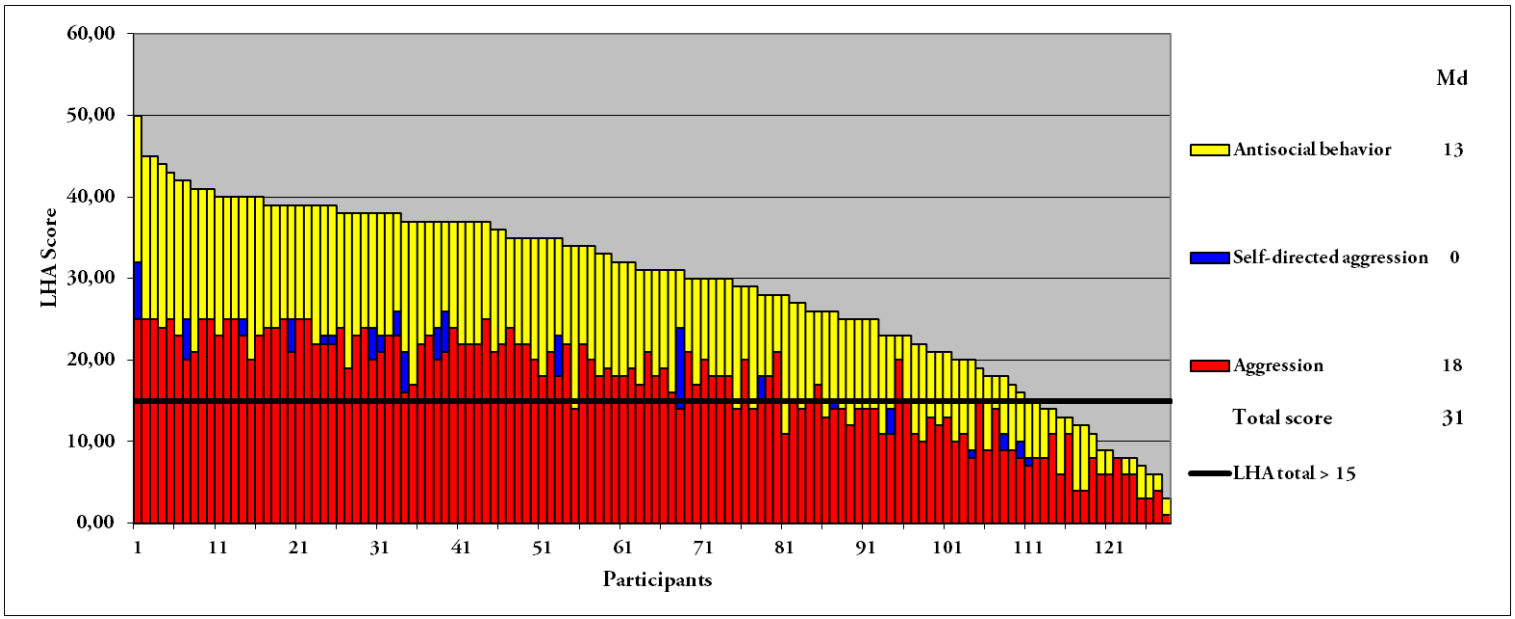
The majority of the subjects, 87% (n=111), displayed abnormally high levels of lifetime aggressive antisocial behaviors as measured by the LHA (Figure 4). Almost two in five (n=49) had presented such seriously aggressive antisocial behavior during childhood and adolescence that they had been institutionalized (previously unpublished data).

The self-reported age at onset of violent criminality varied between 6 and 25 years, with the majority having their onset in late adolescence (M=17 years, SD=3.7 years).

Table 5. Distribution of convictions among violent and non-violent groups in the Swedish general population born 1958–1980; convictions between 1973–2004

	High-persistence offenders (n=2812) n (% within group)	Medium-persistence offenders (n=21 530) n (% within group)	Low-persistence offenders (n=69 300) n (% within group)	Non-violent subjects (n=936 420) n (% within group)
Violent criminality				
Total convictions	46 401	101 767	86 215	N/A
Mean convictions/individual	17	4.7	1.2	N/A
Aggravated violent criminality	2769 (99%)	19 569 (91%)	48 437 (52%)	N/A
Total convictions	23 362	58 335	55 665	N/A
Mean convictions/individual	8.3	2.7	0.8	N/A
Non-violent criminality	2746 (98%)	18 240 (85%)	41 134 (59%)	183 792 (20%)
Theft	2607 (93%)	15 214 (71%)	28 542 (41%)	82 976 (8.9%)
Drug-related offenses	1905 (68%)	7993 (37%)	9724 (14%)	13 839 (1.5%)
Traffic violations	2440 (87%)	14 243 (66%)	28 741 (41%)	129 785 (14%)
Total convictions	142 525	392 211	360 144	517 660
Mean convictions/individual	51	18	5.2	0.6

Figure 4. Distribution of lifetime aggressive antisocial behaviors as measured by the LHA scales among violent offenders in emerging adulthood, plotted in descending order.



Note. Median scores are displayed to the right in the figure. The sum of the three scales equals the LHA total score, with the cut-off for abnormally high scores (LHA total >15) marked in the figure.

Mentally disordered offenders (Papers III–IV)

During follow-up, 22% (n=34) of the combined group of mentally disordered offenders had reoffended violently and another 14% (n=22) were convicted of non-violent criminality (*Paper IV*). In the Gothenburg group (*Paper III*), the majority of the violent relapses occurred after release or discharge. However, almost one third of the relapses into violence (n=6) took place while the offenders were still in custody.

Although offenders sentenced to forensic psychiatric care spent an average of twice as much time at liberty after discharge, they were less likely to reoffend than offenders with a prison sanction ($p<0.05$ in Kaplan-Meier survival analysis with log-rank test).

Risk factors for persistence

Previous aggressive antisocial and/or criminal behavior

In the population-based cohort (*Paper I*), criminal history variables (previous convictions of theft, drug-related offenses, and traffic violations) all increased the risk of persistence in violent criminality with ORs close to 2 in a multivariate logistic regression (Table 6).

Among the violent offenders in emerging adulthood (*Paper II*), persistence in both violent and general criminality were positively associated with a history of drug-related offenses ($p\leq0.001$), while a history of sexual offenses showed a reversed association ($p\leq0.01$) when tested with Fischer's exact test.

Lifetime aggressive antisocial behaviors as measured by the LHA showed modest predictive ability for persistence of violent criminality in ROC analyses and modest to moderate prediction of persistence in general criminality both among violent offenders in emerging adulthood and mentally disordered offenders (*Papers II–III*; Table 7). In a notable exception, self-directed aggression was not related to persistence of aggressive antisocial behavior.

Violence risk as measured by the HCR-20 had a marginal to modest predictive ability for persistence both in violent and general criminality in the mentally disordered offenders (*Paper III;* Table 7).

Age at onset of aggressive antisocial behavior

Young age at onset of violent criminality was associated with increased persistence in violent criminality in the population-based cohort (*Paper I*; Table 8). An age at onset between 15 and 18 years increased the risk of persistence two times (Table 6).

Among the violent offenders in emerging adulthood (*Paper II)*, younger age at onset of violent criminality was the strongest predictor of persistence in violent criminality in ROC analyses (Table 7). Younger age at onset of general criminality, however, only displayed a marginal to modest predictive ability of persistence in violent and general criminality in both violent offenders in emerging adulthood and mentally disordered offenders (*Papers II–III*; Table 7). Yet, in multivariate regression analyses, it was one of the two remaining significant predictors of violent recidivism (OR=0.86, 95% CI=0.76–0.98, $p \leq 0.05$) among the mentally disordered offenders (*Paper III*).

Table 6. Multivariate logistic regression model of risk factors for belonging to the persistent 1% of the total population with 3 or more convictions for violent crime 1973–2004, compared to the low-persistence group

Predictor	B	Odds Ratio	95% CI
Male sex	0.9	2.5**	2.3-2.6
Any missing school grade	0.2	1.3**	1.2-1.4
1st conviction for violence, age 15-18	0.7	2.0**	1.9-2.0
Any conviction for theft	0.7	2.0**	1.9-2.0
Any conviction for a drug-related offense	0.7	1.9**	1.9-2.0
Any conviction for a traffic violation	0.6	1.8**	1.7-1.8
Any diagnosis of a major mental disorder	0.2	1.3**	1.1-1.4
Any diagnosis of a personality disorder	0.8	2.3**	2.1-2.4
Any diagnosis of a substance-related disorder	0.6	1.9**	1.8-2.0
Parent of non-Scandinavian ethnicity	0.1	1.1**	1.0-1.1
Parent with any conviction of a violent crime	0.3	1.3**	1.2-1.4
Parent with any conviction of a nonviolent crime	0.1	1.1**	1.1-1.1
Parent diagnosed with a psychiatric disorder	0.1	1.1**	1.1-1.1
Parent diagnosed with a substance use disorder		-	
Parent died before child's 18th birthday	0.1	1.1*	1.0-1.1

Note. * $p<0.05$, ** $p<0.001$

Psychopathic traits (PCL-R scores)

The Lifestyle and Antisocial facets displayed generally modest to moderate predictive abilities for persistence in both violent and general criminality when tested in ROC analyses (*Papers II* & *IV*; Table 7). The Antisocial facet was consistently the predominant predictor of persistence in both violent and general criminality among the PCL-R facets in ROC analyses (*Papers II* & *IV*; Table 7) and logistic regressions (*Paper IV*).

The Interpersonal facet was only marginally, if at all, better than random in the prediction of persistence in either violent or general criminality when tested in ROC

analyses (*Papers II* & *IV;* Table 7). The Affective facet at best displayed a modest predictive ability (persistence in general criminality among the mentally disordered offenders; *Paper IV*).

When the effect of the Antisocial facet on the prediction of persistence in violent criminality was controlled for in a multi-block logistic regression among the mentally disordered offenders (*Paper IV*), the other three facets no longer showed any significant predictive effect ($\chi 2$=1.8, p=0.61).

Mental disorders and personality disorders

Conduct disorder, substance-related disorders, and antisocial personality disorder were associated with increased persistence in violent criminality across offender groups (*Papers I–III*; some results displayed in Table 8). In the population-based cohort (*Paper I*), a diagnosis of personality disorder increased the risk of persistence 2.3 times and substance related disorders increased the risk by 1.9 times (Table 6). Major mental disorders were associated with increased risk of persistence in violent criminality only in the population-based cohort (Table 6).

Sociodemographic factors

In the population-based cohort (*Paper I*), the absolute majority (approximately 90%) of violent offenders were men. The proportion of women significantly decreased with increasing persistence in violent criminality; 13% (n=8742) of the low-persistence offenders were women compared with 2.1% (n=60) of high-persistence offenders ($p \leq 0.001$ in $\chi 2$-tests). Male sex increased the risk of persistence 2.5 times (Table 6).

School problems, e.g., incomplete school grades and/or truancy, were associated with increased persistence in violent criminality (*Papers I* & *II*). In the population-based cohort (*Paper I*), incomplete school grades increased the risk of persistence in violent criminality 1.3 times (Table 6). Among the persistently violent offenders in emerging adulthood (*Paper II*; previously unpublished data), only 17% (n=15) had finished secondary school at the expected age, and all but two offenders had a history of school truancy. One in two (n=45) reported having bullied others during their school years, and the majority (n=70) had received special support from school.

Poor preconditions during childhood were common in persistently violent offenders (*Papers I–III*). Among the persistently violent offenders in emerging adulthood (*Paper II*; previously unpublished data), approximately two in three (n=56) described being exposed to violence at home, while two in five (n=36) had a parent/care-taker with substance-related problems.

Table 7. Predictors of persistence in violent and general criminality among offender groups, AUC-values with 95% CI

	Persistence in violent criminality		Persistence in general criminality	
	Violent offenders in emerging adulthood	Mentally disordered offenders	Violent offenders in emerging adulthood	Mentally disordered offenders
Young age at onset				
Violent criminality	0.84 (0.75–0.92)**	N/A	0.77 (0.67–0.88)**	N/A
General criminality	0.65 (0.54–0.76)*	0.70 (0.58–0.83)*[a]	0.77 (0.65–0.88)**	0.65 (0.53–0.76)[a, c]
LHA				
Aggression	0.79 (0.71–0.87)**	N/A	0.79 (0.65–0.92)**	N/A
Self-directed aggression	0.48 (0.37–0.59)	N/A	0.50 (0.35–0.64)	N/A
Antisocial behavior	0.72 (0.62–0.82)**	N/A	0.87 (0.77–0.97)**	N/A
Total score	0.77 (0.69–0.86)**	0.74 (0.62–0.86)*[a]	0.81 (0.69–0.94)**	0.68 (0.56–0.81)*[a, c]
PCL-R				
Interpersonal facet	0.65 (0.55–0.75)*	0.61 (0.50–0.72)[b]	0.57 (0.42–0.71)	0.67 (0.57–0.76)**[b]
Affective facet	0.66 (0.56–0.76)*	0.67 (0.57–0.77)*[b]	0.69 (0.57–0.80)	0.73 (0.64–0.81)**[b]
Lifestyle facet	0.75 (0.64–0.85)**	0.73 (0.64–0.82)**[b]	0.89 (0.82–0.96)**	0.75 (0.68–0.83)**[b]
Antisocial facet	0.82 (0.73–0.90)**	0.80 (0.72–0.88)**[b]	0.94 (0.90–0.99)**	0.78 (0.70–0.86)**[b]
Total score	0.81 (0.73–0.90)**	0.73 (0.65–0.82)**[b]	0.90 (0.84–0.96)**	0.78 (0.70–0.85)**[b]
HCR-20				
Total score	N/A	0.71 (0.60–0.83)*[a]	N/A	0.69 (0.57–0.80)*[a, c]

Note. [a] Data from the Gothenburg group of mentally disordered offenders, [b] Data from the combined group of mentally disordered offenders, [c] Previously unpublished data, * $p \leq 0.01$, ** $p \leq 0.001$

Table 8. Characteristics of violent offender groups and non-violent subjects

	Swedish general population				Violent offenders in emerging adulthood
	High-persistence offenders (n=2812) n (% within group)	Medium-persistence offenders (n=21 530) n (% within group)	Low-persistence offenders (n=69 300) n (% within group)	Non-violent subjects (n=936 420) n (% within group)	Persistently violent offenders (n=88) n (% within group)
Age at onset of violent criminality					
<15 years	N/A	N/A	N/A	N/A	23 (26%)[a]
15–18 years	1691 (60%)	9095 (42%)	18 862 (27%)	N/A	52 (59%)[a]
19–23 years	773 (27%)	7065 (33%)	25 341 (37%)	N/A	11 (13%)[a]
≥24 years	348 (12%)	5370 (25%)	25 097 (36%)	N/A	1 (1.1%)[a]
Mental disorders					
Any kind	1778 (63%)	7512 (35%)	10 367 (15%)	21 404 (2.3%)	88 (100%)[a]
Major mental disorders	157 (5.6%)	784 (3.6%)	1438 (2.1%)	4749 (0.5%)	11 (13%)[a]
Substance-related disorders	1679 (60%)	6773 (32%)	8777 (13%)	15 407 (1.6%)	74 (86%)[a]
Personality disorders	624 (22%)	1902 (8.8%)	2228 (3.2%)	3859 (0.4%)	69 (85%)[a]

Note. [a] Previously unpublished data. All differences across the groups in the Swedish general population were significant at $p<0.001$ when analyzed with χ^2-tests

Substance-related problems among primary relatives accounted for a 2.7 times higher risk of violent recidivism (OR 95% CI=1.1–6.9, $p \leq 0.05$) among mentally disordered offenders (*Paper III*), and was, together with age at onset of general criminality the only remaining predictor of violent recidivism in multivariate logistic regressions. In the population-based cohort (*Paper I*), a history of criminal convictions or mental disorders in parents was more common among persistently violent offenders ($p \leq 0.001$ in χ^2-tests), even though the statistical increases in risk were marginal, with ORs close to one (Table 6).

Among the violent offenders in emerging adulthood (*Paper II*), institutionalization during childhood/adolescence was associated with persistence in both violent and general criminality ($p \leq 0.01$).

Psychopathic traits (PCL-R) among offenders

Distribution

Approximately 20% of the violent offenders in emerging adulthood (n=25) and mentally disordered offenders (n=27) scored equal to or above 25 points on the total PCL-R score (*Papers II* & *IV*; some data previously unpublished). The majority of all offenders scored low on the Interpersonal facet (Table 9). However, in contrast to the mentally disordered offenders, the violent offenders in emerging adulthood scored high on the Lifestyle and Antisocial facets. Deficient affective experiences (Affective facet) were somewhat more common among the mentally disordered offenders.

Table 9. Distribution of PCL-R psychopathic traits among offenders, median scores and range (in parentheses)

	Violent offenders in emerging adulthood (n=134)	Mentally disordered offenders, combined group[a] (n=153)
Interpersonal facet	1 (0–8)	1 (0–8)
Affective facet	3 (0–8)	4 (0–8)
Lifestyle facet	7 (0–10)	3 (0–10)
Antisocial facet	7 (0–10)	2 (0–10)
PCL-R total score	19 (2–40)	11 (0–37)

Note. [a] Previously unpublished data

Clinical covariates

Across offender groups, the Interpersonal facet showed unique characteristics (*Papers II* & *IV*). Among the violent offenders in emerging adulthood (*Paper II*), the Interpersonal facet was related only to the presence of paranoid personality disorder ($p \leq 0.01$) and not at all to a history of aggressive antisocial behavior (except for one weak, negative correlation to age at onset of violent criminality ($r_s = -0.26$, $p \leq 0.01$) when tested with Mann-Whitney U-tests and Spearman's correlations. In the mentally disordered offenders (*Paper IV*), the Interpersonal facet was the only facet negatively associated with Cluster A personality disorders ($p < 0.01$) and personality traits tapping psychic anxiety ($r_s = -0.25$, $p < 0.01$). Unlike the other facets, the Interpersonal facet was not related to substance-related disorders or antisocial personality disorder in either of the offender groups.

The Affective facet was related to conduct disorder ($p \leq 0.001$), and antisocial personality disorder/Cluster B personality disorders ($p \leq 0.001$) across offender groups (*Papers II* & *IV;* Mann-Whitney U-tests). However, these associations were weaker than those of the Lifestyle and Antisocial facets.

The Lifestyle and Antisocial facets were alike in their strong associations with conduct disorder, substance-related disorders, and antisocial personality disorder/Cluster B personality disorders (all $p \leq 0.001$ in Mann-Whitney U-tests; *Papers II* & *IV*).

Among the violent offenders in emerging adulthood (*Paper II*), the Lifestyle and Antisocial facets were also related to school truancy ($p \leq 0.01$), AD/HD ($p \leq 0.01$), and institutionalization during childhood/adolescence ($p \leq 0.001$) when tested in Mann-Whitney U-tests. They were also strongly associated with a history of aggressive antisocial behavior as measured by the LHA total score ($0.51 \geq r_s \leq 0.52$, $p \leq 0.001$), and moderately correlated to younger age at onset of violent criminality ($-0.35 \geq r_s \leq -0.41$, $p \leq 0.001$). High scores on these facets were associated with a lower frequency of sexual offenses ($p \leq 0.001$; Mann-Whitney U-tests).

In the mentally disordered offenders (*Paper IV*), the Lifestyle and Antisocial facets were moderately associated with KSP impulsiveness, aggression, and low levels of socialization ($0.34 \geq r_s \leq 0.48$, $p \leq 0.001$; some data previously unpublished).

Cognitive covariates of aggressive antisocial behavior

Cognitive distortions were significantly more common among offenders than non-offenders (*Paper V;* Table 10) and moderately to strongly correlated with aggressive antisocial behavior ($0.45 \geq r_s \leq 0.62$, $p \leq 0.001$). In ROC analysis, cognitive distortions

demonstrated a moderate predictive ability of a high level of aggressive antisocial behavior, AUC=0.81 (95% CI=0.73–0.89, $p \leq 0.001$).

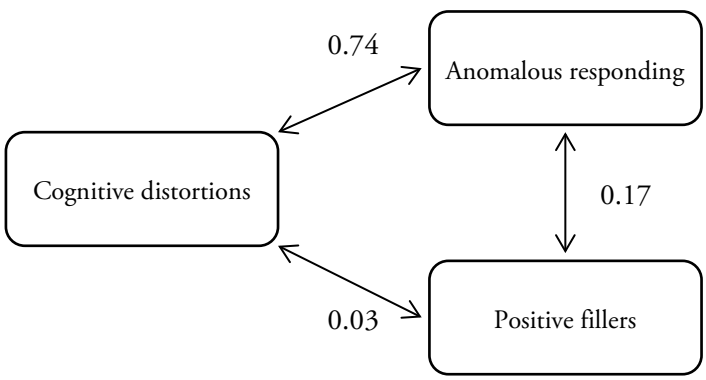
Table 10. Mean and standard deviation (in parentheses) of cognitive distortions among offenders and non-offenders

	Adults		Adolescents	
	NO (n=60)	O (n=56)	NO (n=190)	O (n=58)
HIT total score	1.88 (0.46)	2.72* (0.90)	2.23 (0.79)	3.88* (1.21)
Self-Centered	1.99 (0.61)	2.91* (1.08)	2.32 (0.86)	3.83* (1.36)
Blaming Others	1.83 (0.48)	2.70* (0.91)	2.24 (0.84)	3.88* (1.23)
Minimizing/ Mislabeling	1.99 (0.62)	2.75* (1.01)	2.18 (0.90)	3.74* (1.41)
Assuming the Worst	1.65 (0.44)	2.50* (0.89)	2.11 (0.83)	4.08* (1.18)

Note. NO=Non-offenders, O=Offenders, *$p \leq 0.001$ (two-tailed p-values from t-tests)

When the latent structure of cognitive distortions was investigated using CFA, a three-factor model with one comprehensive cognitive factor (Figure 5) provided the best fit to the data (n=364, $\chi 2$=2115.98, df=1374, $p \leq 0.0001$, [2115.98/1374=1.5]; RMSEA=0.04). This was contrary to the theoretical model proposed for cognitive distortions as measured by the HIT questionnaire.

Figure 5. A three-factor model of cognitive distortions as measured by the HIT questionnaire



Summary of findings

1. One percent of the Swedish general population was responsible for 63% of violent crimes. Increasing persistence in violence was associated with increased offending behavior in general. Among violent offenders in emerging adulthood, 69% manifested persistent aggressive antisocial behavior. (*Papers I* & *II*)
2. A predominant risk factor for persistence of aggressive antisocial behavior was its early onset. Clinical and sociodemographic factors that were relevant to persistence included conduct disorder, antisocial personality disorder, substance-related disorders, and school truancy. (*Papers I–III*)
3. In the PCL-R, the Antisocial facet outperformed all other facets and the total score in the prediction of persistence in aggressive antisocial behavior, while the Interpersonal facet contributed little to nothing. (*Papers II* & *IV*)
4. The four PCL-R facets differed in their associations with clinical characteristics. The Lifestyle and Antisocial facets were similarly associated with impulsivity, conduct disorder, substance-related disorders, and antisocial personality disorder, while the Interpersonal facet exhibited unique attributes. (*Papers II* & *IV*)
5. Cognitive distortions were associated with an increased propensity towards aggressive antisocial behavior, and were best described with a coherent, cognitive factor of aggressive antisocial thinking patterns. (*Paper V*)

Discussion

Comments on the main findings

Persistence of aggressive antisocial behavior

Aggressive antisocial behavior had a much skewed distribution in the general population, in concordance with previous research[186]. Only 1% of the cohort from the Swedish general population was responsible for the majority, 63%, of violent crimes. Each conviction for a violent crime increased the probability of violent reconvictions, confirming arguments from longitudinal studies that "offending causes more offending"[187]. After three violent convictions, the majority of those offenders, 68%, went on to be reconvicted for further violent offenses.

Three in four violent offenders in the general population were described as low-persistence offenders, with one to two convictions of violent crimes during the follow-up period. Some of these offenses were probably committed because the wrong person was in the wrong situation at the wrong time. This suggests that even though a small group is responsible for the great majority of aggressive antisocial behaviors, part of the variance of aggressive antisocial behavior is unlikely to be accounted for by taxonomies.

The violent offenders in emerging adulthood displayed high levels of persistent and diverse aggressive antisocial behavior. As these individuals are probably in the most crime-active period of their lifetime, these findings are not surprising, and support the assertion that offending behavior, especially in youth, is versatile rather than specialized[188].

Persistence, measured as reconvictions for further violent crime, was less frequent among mentally disordered offenders sentenced to forensic psychiatric care. This needs to be further examined before any clear conclusions can be drawn. However, the findings do suggest that the influence of severe mental illness to increase aggressive antisocial behavior is actually rather small[189], and the popular media's presentation of seriously mentally disordered offenders as especially dangerous must be questioned. The results also challenge the use of "time-at-risk" for aggressive

[186] Elonheimo et al. 2009, Loeber et al. 1999, Moffitt et al. 2002
[187] Farrington 2003, Smith 2007
[188] Elonheimo et al. 2011, Farrington 2003, Krug et al. 2002
[189] Fazel & Grann 2006

antisocial behavior as equivalent to time at liberty, since a significant proportion of relapses in aggressive antisocial behavior occur not when the individuals are at liberty, but while they remain in custody.

The onset of violent criminality among both the persistent offenders in the Swedish general population (the 1% group) and the violent offenders in emerging adulthood occurred in the late adolescent years. These findings are concurrent with the well-established peak of criminality in late adolescence[190]. However, one in four among the persistently violent offenders in emerging adulthood had an onset of violent criminality before the age of criminal responsibility (15 years in Sweden). This promotes arguments for the necessity of combining official conviction data with collateral information such as self-reports in research on aggressive antisocial behavior[191]. Otherwise, variations in the age of criminal responsibility between countries may result in biased base rates of aggressive antisocial behavior over the life course.

In summary, it is obvious that there is a small group in society that is characterized by a diverse, persistent, and increasingly severe pattern of aggressive antisocial behavior. This aggregation of aggressive antisocial behavior in individuals corresponds to chronic, or life-course-persistent, offenders[192]. It is likely that at least a substantial proportion of the persistent offenders in the general population (the 1% group) and the violent offenders in emerging adulthood described in this thesis are on a life-course-persistent pathway of aggressive antisocial behavior. Our results support the argument for a phenotype, or behavioral continuity, of aggressive antisocial behavior across the life course[193].

Risk factors for persistence

The most prominent risk factor for persistence across offender groups was an early onset of aggressive antisocial behavior. This implies that, even though a history of such behavior is confirmed as the best predictor of future aggressive antisocial behavior[194], an early onset holds special importance in predicting persistence[195]. Thus, a history of aggressive antisocial behavior including age at onset should always be controlled for in analyses of risk factors for persistence.

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[190] Blumstein et al. 1988, Hirschi & Gottfredsson 1983
[191] Monahan et al. 2001
[192] Moffitt 1993, Tolan & Gorman-Smith 1999
[193] Farrington 2007, Hofvander et al. 2009, Moffitt 1993
[194] Bonta et al. 1998
[195] Farrington 1995, Kratzer & Hodgins 1997, Moffitt 1993

The clinical factors that were most strongly related to persistence in aggressive antisocial behavior were conduct disorder, substance-related disorders, antisocial personality disorder, and the Lifestyle and Antisocial facets of the PCL-R. All of these factors reflect the antisocial and disinhibitory features that characterize the externalizing spectrum of mental health problems[196]. These features also imply underlying neurobiological susceptibilities that can manifest in, for instance, problems with hyperactivity, executive functioning, and social interaction[197]. However, these underlying susceptibility factors are just beginning to be investigated.

Major mental disorders were also associated with an increased risk of persistence, but to a lesser extent. An arrogant and deceitful way of relating to others (PCL-R Interpersonal facet) does not seem to be related to an increased risk of persistent aggressive antisocial behavior, in concordance with previous research[198].

It is obvious that the antecedents to persistently aggressive antisocial behavior are already present during childhood and adolescence[199]. Early-onset aggressive antisocial behavior was manifested by young age at first conviction, conduct disorder, school problems, and institutionalization during childhood and/or adolescence for antisocial behavior. This implies a *homotypic continuity* (i.e., continuity of similar behaviors or phenotypic attributes over time) of aggressive antisocial behavior over the life course[200]. Adult manifestations of this homotypic continuity can be seen in antisocial personality disorder and substance-related disorders. However, there will always be significant individual differences in the stability of aggressive antisocial behavior over the lifetime, especially when followed into the senior years[201].

A familial background of criminality, serious mental health problems, and exposure to violence at home, were noted among persistently aggressive antisocial offenders. This is in line with genetic effects contributing to aggressive antisocial behavior, familial aggregation of behavior, and a pathological interaction between a difficult child and a high-risk environment[202].

The research on risk assessment for recidivistic aggressive antisocial behavior displays a "glass-ceiling" effect[203], with maximum AUC values around 0.75–0.80. It may be possible to achieve higher AUC values (Antisocial facet AUC=0.94 for persistence in general criminality among violent offenders in emerging adulthood). However, the

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[196] Krueger 1999, Krueger et al. 2002, Patrick et al. 2005, Walters et al. 2008
[197] Hofvander et al. 2009
[198] Hall et al. 2004, McGregor et al. 2012
[199] Hodgins 2007, Moffitt 1993
[200] Kim-Cohen et al. 2003
[201] Sampson & Laub 2003, Smith 2007
[202] Burt 2009, Frisell 2012, Moffitt et al. 2002, Rhee & Waldman 2002
[203] Coid et al. 2011

real value of these numbers is questionable as they are due to a circular effect, in which we use a measure of behavior to predict the same type of behavior. Also, the propensity for violence results from an accumulation of risk factors[204].

It seems reasonable to conclude that predictions of human behavior are more accurate than random, but that at the same time human behavior is too complex ever to be more than partly determined.

Psychopathic traits (PCL-R) among offenders

The prevalence of highly psychopathic traits was similar over the offender groups. One in five offenders scored 25 points or more on the PCL-R, which in general meant high scores on the Lifestyle and Antisocial facets. These findings are in line with previous research on the prevalence of psychopathic traits among offenders[205], even though great variation in prevalence (3%–73%) has been noted across studies[206].

The offender groups differed in the distribution of their facet scores, with mentally disordered offenders displaying somewhat more affective deficits. A possible explanation for this disparity could be a higher prevalence of psychotic disorders among the mentally disordered offenders. However, common to all the offender groups was a low score on the Interpersonal facet.

The differential clinical covariations between the facets are in line with suggestions that psychopathy is too complex to be considered as one coherent construct[207]. Eli Robins and Samuel Guze[208] argued early for five strict criteria for a valid and coherent clinical construct: 1) a clinical description including unique characteristics, 2) confirmed biological markers for the construct, 3) delimitation from other disorders, 4) homotypic progression in follow-up studies (stability of the construct over time), and 5) a confirmed familial aggregation compared to unaffected controls. Because psychopathy seems too complex to meet all these criteria, dimensional assessments of psychopathic traits should be preferred to dichotomous diagnoses based on arbitrary cut-off points[209].

The Interpersonal facet displayed unique characteristics in its virtual inability to predict persistent aggressive antisocial behavior and its lack of associations with mental disorders and behaviors commonly associated with psychopathy (e.g.,

[204] Monahan et al. 2001
[205] Assadi et al. 2006, Hare 2003
[206] Coid et al. 2009
[207] Walters et al. 2008
[208] Robins & Guze 1970
[209] Coid & Ullrich 2010

antisocial personality disorder and substance-related disorders). The Lifestyle and Antisocial facets were both associated with disorders, personality traits, and behaviors within the externalizing spectrum. This provides support for a phenotypic continuum, in which highly socially deviant and antisocial psychopathic traits could be viewed as extremes on the end of the continuum[210].

Many researchers and clinicians view the interpersonal and affective traits as core features of psychopathy[211]. Some claim that psychopathic traits need not even express themselves in criminal behavior: "overemphasis on involvement in crime has obscured the nature of psychopathy as a disorder of personality characterised by interpersonally harmful behavior that need not necessarily take criminal form" (p. 142)[212]. Efforts have been made to develop instruments, such as the Comprehensive Assessment of Psychopathic Personality[213], to assess the personality traits that are considered a purer measure of psychopathy . So far, the debate on whether antisocial behavior should be seen as a part or a consequence of a psychopathic personality remains unsolved. In order to provide solid arguments for the debate, large-scale studies on the different facets of psychopathy in relation to aggressive antisocial behavior are needed, both in the general population and in forensic settings.

In summary, the current findings confirm that psychopathy, as currently operationalized in the PCL-R, is heterogeneous[214]. The Antisocial facet, whether or not it is a core feature of the construct, is crucial as a predictor of persistent aggressive antisocial behavior.

Cognitive covariates of aggressive antisocial behavior

Cognitive distortions were associated with an increased propensity towards aggressive antisocial behavior. Previous research has shown that adding measures of criminal thinking to static risk factors (e.g., age, reports of prior incidents) improves the prediction of aggressive antisocial behavior[215]. The AUC (0.81) for cognitive distortions reported in this study was on par with that reported for the Antisocial facet in the prediction of the persistence of violent criminality in *Papers II* (0.82) and *IV* (0.80). This implies that cognitive distortions could be valuable not only for treatment planning, but also as a complement in the assessment and prediction of aggressive antisocial behavior. However, the direction of a relationship between these

[210] Patrick et al. 2005, Walters et al. 2008
[211] Cleckley 1941, Cooke et al. 2004a, Cooke & Michie 2001
[212] Blackburn 2007
[213] Cooke et al. 2004b
[214] Blackburn et al. 2008
[215] Walters 2012

types of cognitive distortions and aggressive antisocial behavior needs to be examined in more depth than is undertaken in the current thesis.

The findings suggest that cognitive distortions associated with aggressive antisocial behavior are best described by a coherent, criminal thinking style. This contrasts with previous findings of a multidimensional character in cognitive distortions[216]. However, it has previously been argued that cognitive distortions over time are incorporated into a holistic criminal mindset[217]. As the current findings were based on both adult and adolescent subjects, the possible effects of socio-moral development on cognitive distortions also need to be considered. To shed more light on this, more research on other offender and non-offender groups is needed.

To summarize, the findings emphasize the importance of considering thinking styles and attitudes as dynamic risk (and protective) factors in the prediction and management of aggressive antisocial behavior[218].

Limitations

Limitations of the studies are discussed in the individual papers, however, some limitations more general to the thesis as a whole need to be addressed in more detail.

General definitions

In this thesis aggressive antisocial behavior was studied as behavior *per se*, and the motives and functions of the behavior, including situational circumstances, consequences, and possible interaction effects, were not considered. An obvious disadvantage of this approach is that many aggressive antisocial acts that are in fact very dissimilar in intentionality or functionality might seem similar when only the visible behaviors are considered. This could result in overly simplistic models of aggressive antisocial behavior[219].

Another limitation was how sexual offenses were handled in the analyses. In *Paper I* sexual offenses were excluded from analyses of violent criminality, while they were included in analyses of violent criminality in *Papers III* and *IV*. In *Paper II*, sexual offenses were studied separately in a few analyses, but as one of a variety of violent offenses in the majority of the analyses. The inclusion of sexual offenses in definitions

[216] Barriga et al. 2001, Simourd & Olver 2002
[217] Samenow 2004
[218] Andrews & Bonta 2010, Douglas & Skeem 2005
[219] Tremblay 2000

of violent criminality has been debated, and it has been proposed that they should be seen as a related but distinct category of interpersonal violence[220]. Sexual offenders are generally more likely to relapse with non-sexual criminality than sexual violence, even though some risk factors for recidivism (e.g., sexual deviancy) seem to be unique to sexual offending[221]. Even if sexual offenses differ in many respects from other forms of interpersonal violence, it would not have been possible to study sexual offenses as a distinct category in this thesis due to the lack of power for such analyses. Nevertheless, this limitation needs to be considered in the interpretation and generalization of the results of this thesis.

We used different definitions of persistence in the analyses in the various papers: three or more violent convictions counted as persistence in *Paper I*, while two or more occasions were sufficient in *Paper II*. In *Papers III* and *IV*, persistence was defined as a relapse of violent and/or general criminality without consideration of any specific number of relapses or reconvictions. These variations stem from variability in the data available for analysis, but they nevertheless restrict generalizability, both between the studies in the thesis, and between these and other studies and contexts. Defining persistence as a certain number of relapses, or reconvictions, will always result in a constructed limit. Because specific studies on persistence seem important, a dimensional approach would be preferable in future research.

Sources of data

Papers II to *V* were largely based on retrospective reporting of subjects' aggressive antisocial behaviors, mental disorders, and psychosocial backgrounds. This may be criticized because recall bias can result in serious under-reporting[222]. However, this risk can be mitigated by using high-quality methods, such as expert-performed, detailed interviews[223]. In *Papers II* to *IV*, we used information from semi-structured, diagnostic interviews in combination with file information, and this might have reduced the risk of recall bias. Nonetheless, prospective longitudinal studies are preferable to retrospective studies to reduce the risk of recall bias, although cross-sectional studies with a longitudinal follow-up, such as those reported here, can combine important retrospective and prospective information.

The sole use of official conviction data in the analyses of persistence of aggressive antisocial behavior (*Papers I* & *IV*) may lead to a substantial underestimation of these

[220] Krug et al. 2002
[221] Hanson & Morton-Bourgon 2005
[222] Moffitt et al. 2010
[223] Hardt & Rutter 2004, Hill & Nathan 2008

kinds of behaviors[224]. Many offenses are never reported to the police, and of those that are reported, an offender is not always identified, prosecuted, and convicted in a court of law. Using only conviction data could also lead to overestimates of the behavioral continuity of aggressive antisocial behavior, which might in fact be more an effect of continuity in police targeting of particular suspects.

In *Paper I*, offense rates may have been underestimated due to the short follow-up of subjects who were still as young as 24 years at the end of the study. Even if aggressive antisocial behavior peaks during late adolescence and decreases in early adulthood, some individuals (approximately 100–150) probably offended after the end of follow-up. However, this would have affected the base rates only minimally as the study included a total of 24 342 persistently violent offenders. The analyses of risk factors were not affected due to the use of age-matched non-offenders.

The base rate of aggressive antisocial behavior, especially persistence, could also have been affected by length of sentence, since individuals with long sentences probably commit fewer offenses in custody than when living in society. This was not controlled for in any of the studies on conviction data (*Papers I, III,* & *IV*). However, the results of *Paper III* indicate that not only length of sentence, but also offenses committed during custody, should be taken into account in future studies. The equation of time-at-risk with time at liberty can obviously lead to underestimations of aggressive antisocial behavior.

Another limitation of the analyses of persistence in aggressive antisocial behavior was that *Papers III* and *IV* considered only the subjects' first occasion of recidivism. Hence, it was not possible to investigate their continued persistence in aggressive antisocial behavior. Furthermore, the groups included in these papers had slightly different follow-up periods, which could have affected the recidivism rates.

Taken together, these limitations might have reduced the base rate of aggressive antisocial behavior, thereby causing a greater risk of false negatives or type II errors in the analyses. This risk needs to be considered especially in *Papers III* and *IV*, in which the sample sizes were restricted. Nonetheless, the findings are concurrent with the prevailing literature in the area and with studies including self-report measures of aggressive antisocial behavior (*Paper II*).

In *Paper I*, we used the Hospital Discharge Register for information on inpatient psychiatric diagnoses, which very likely led to an underestimation of mental disorders, especially substance-related and personality disorders, that might have affected the analyses of risk factors for persistence. However, as the clinical studies in *Papers II* to *IV*, in which psychiatric diagnoses were based on semi-structured clinical interviews

[224] Loeber et al. 1999, Monahan et al. 2001

performed by experienced clinicians, produced similar findings, the results overall are supported. In using official registers, a certain amount of missing information should always be expected.

The LHA was used as a measure of lifetime aggressive antisocial behaviors in *Papers II* and *III*. The majority of violent offenders in emerging adulthood (*Paper II*) scored above the cut-off for abnormally high levels of aggressive antisocial behaviors, thereby providing little variation for continued analyses. Even if the LHA holds face validity for these kinds of analyses of offender groups, the psychometric properties of the instrument should be studied specifically in offender groups in order to provide guidelines for the meaningful interpretation of results in forensic contexts.

Composition of study groups

In *Papers II* to *IV*, we did not use control groups that were representative of the general population. However, since all subjects were consecutively recruited from a well-defined context, it seems reasonable to conclude that they were representative of the offender group from which they were recruited.

In *Paper V*, control groups were used to compare cognitive distortions between offender and non-offender groups. The control groups cannot, however, be claimed as representative of the general population as they were taken from the educational system, and in one case (the adult control group) from a quite homogeneous group of engineering students. The offender groups were not consecutively recruited, which might have led to a selection bias towards more motivated subjects. The subjects were not matched for sociodemographic variables such as age, educational level, and socioeconomic status. This hampered our ability to assess confounder effects in the observed differences in cognitive distortions.

In *Papers II* to *IV*, subjects were recruited from clinical contexts, resulting in rather small sample sizes. This affected the analytical possibilities, especially since multicollinearity between variables could be expected. The results from multivariate analyses, such as logistic regressions, should therefore be interpreted with caution. However, as these samples were recruited from clinical contexts, they may supply important information on groups that are uncommon in society.

Paper IV pooled data from two independent study groups of mentally disordered offenders. The groups differed somewhat in their composition, which can be seen as both a limitation and an advantage, as it provides a broader coverage of mentally disordered offenders in Sweden. Even if the groups differed on some variables, they were recruited from the same type of clinical context and examined with similar methods, thereby providing arguments for the appropriate pooling of the data.

Only three studies (*Papers I, III,* & *V*) included female subjects. In all cases, the numbers of women were too low to make more advanced analyses on the effects of gender. Most research on aggressive antisocial behavior has been performed on men, as they are clearly overrepresented among aggressive antisocial offenders. Because of this, generalizations from this thesis should not be applied to female offenders.

Clinical implications

Based on the current findings, public health agencies should adopt a focus on violence-prevention strategies targeted at young children and adolescents at risk of developing persistent aggressive antisocial behavior. Intervention strategies can be performed at the individual, family, community, and society levels.

Examples of interventions with demonstrated violence-prevention effects are home-visitation during infancy and toddlerhood, pre-school enrichment, and school-based interventions that focus on social development and problem solving skills[225]. Parenting interventions that focus on teaching parents to promote pro-social behaviors, use non-violent and sensitive discipline strategies to curb aggressive antisocial behavior, and engage in their child in positive ways have also been shown to have a violence-prevention effect[226].

One of the main issues in providing these kinds of interventions is ensuring that all families in need of these interventions are given the opportunity to participate[227]. Another challenge is developing intervention models that can be performed collaboratively by different agencies that usually work apart from each other, such as social welfare, the school system, and the health care system. It is also important to consider that the majority of children who display disruptive behaviors do not become persistent aggressive antisocial offenders, as most children cease their aggressive antisocial behavior before adolescence or young adulthood[228]. Nevertheless, the benefits for the children, their families, and society at large of providing early interventions such as those described above on a broad basis to children and families at risk probably outweigh the risks arising from not providing them.

In order to provide effective violence-prevention interventions to children at risk, a first step is to develop effective methods to detect these individuals. One way to do this could be to allocate extra resources at school for this aim, and to use the knowledge that teachers have of their pupils, as it has repeatedly been shown that

[225] Butchart et al. 2004
[226] Dadds & Rhodes 2009
[227] Turner & Sanders 2006
[228] Robins 1966, Rutter et al. 2006

teachers' reports of children's problems are strongly associated with their aggressive antisocial behavior at later stages in life[229].

These findings also stress the importance of providing violence-prevention strategies specifically aimed at the small proportion of the general population that is responsible for the majority of aggressive antisocial behavior in society. This requires well-structured violence-reduction programs that can be applied in both forensic and community settings. Important features in such programs should be the treatment of externalizing disorders such as AD/HD, substance-related disorders, and antisocial personality disorder, and cognitive distortions. However, the main feature should be an emphasis on the reduction of aggressive antisocial behavior. There are structured programs directed at violence reduction, such as the Violence Reduction Programme[230]. Knowledge of the effectiveness of these kinds of programs is still scarce. In work with such programs, it might be useful to consider that "effective interventions need not eliminate all or even most of a person's risk factors. It should be sufficient only to reduce the presence or effect of these factors below the threshold… at which their combined effect is likely to cross the threshold at which violence occurs" (p. 143)[231].

Furthermore, the findings emphasize the importance of providing education and mental health treatment within the correctional system. The methods applied in this thesis, especially those in *Paper II*, show that it is possible for a clinical psychologist to perform state of the art clinical examinations, including neuropsychological assessments, of offenders in prison, and establish diagnoses of mental disorders in approximately two workdays. Lack of resources is often claimed as an argument against providing these examinations, which would create the necessary preconditions for planning individual treatment and violence-prevention interventions. However, the findings in this thesis show that it is possible to do so in both a time- and a cost-effective way.

The findings also have implications for the practice of risk assessment, as they confirm that the best predictor of future behavior is earlier behavior. That is, if risk assessments of violence are used only for predictive purposes, they can probably be simplified and should focus on aggressive antisocial behavior, and particularly on its early onset. Recent research supports this notion, showing that only a small number of the items in common risk assessment instruments carries the predictive power of the instrument[232].

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[229] Sourander et al. 2006, Zara & Farrington 2009
[230] Gordon & Wong 2000
[231] Monahan et al. 2001
[232] Coid et al. 2011

Future research directions

In this thesis I used the term aggressive antisocial behavior to describe the outcome variable. A major challenge in the continued research on aggressive antisocial behavior is the use of a well-defined, quantifiable outcome variable in order to conduct to-the-point research and tailor violence-prevention interventions that can be compared across different settings and cultures.

Even though aggressive antisocial behavior shows relative stability over the life course, we are in need of studies examining these kinds of behaviors as a dynamic phenomenon. In order to do this, longitudinal studies on groups exhibiting these kinds of behaviors are needed. Such studies can contribute to the identification of turning points, i.e., events (positive or negative) that lead to lasting, long-term modifications of the trajectory of the individual[233]. These turning points are often recognized in hindsight as the individual realizes the actual importance of the event[234], and they may be used to discern possible protective factors for aggressive antisocial behavior.

Continued research on protective factors for aggressive antisocial behavior is needed, as it has been shown that the efficiency of predicting aggressive antisocial behavior is improved by including both protective factors and risk factors[235].

Further research on risk factors for aggressive antisocial behavior should focus not only on independent effects of the risk factors, but also on their additive, interactive, and mediating effects in the persistence of aggressive antisocial behavior[236]. A major challenge within this area is to determine which variables are not only associated, but are truly causally associated, with persistence in aggressive antisocial behavior.

Finally, research on the outcome of structured preventive efforts directed at aggressive antisocial behavior is needed in order to provide guidelines for the continued prevention of aggressive antisocial behavior. In this research, the methods should be carefully chosen with attention paid to the limitations of each approach, such as the underestimation of true base rates of aggressive antisocial behavior in official conviction data.

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[233] Rutter 1996, Rönkä et al. 2002
[234] Huesmann et al. 2011
[235] Stouthamer-Loeber et al. 2002
[236] Farrington 2007

References

Achenbach TM, Edelbrock CS. (1978). The classification of child psychopathology: a review and analysis of empirical efforts. *Psychol Bull* 85:1275–1301.

Ægisdóttir S, White MJ, Spengler PM, Maugherman AS, Anderson LA, Cook RS, Nichols CN, Lampropoulos GK, Walker BS, Cohen G, Rush JD. (2006). The meta-analysis of clinical judgment project: fifty-six years of accumulated research on clinical versus statistical prediction. *Couns Psychol* 34:341–382.

Allen A, Links PS. (2012). Aggression in borderline personality disorder: evidence for increased risk and clinical predictors. *Curr Psychiatry Rep* 14:62–69.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders, 4th edn, text revision (DSM-IV-TR).* Washington, DC: American Psychiatric Association.

Anderson CA, Bushman BJ. (2002). Human Aggression. *Annu Rev Psychol* 53:27–51.

Andrade JT. (2008). The inclusion of antisocial behavior in the construct of psychopathy: a review of the research. *Aggress Behav* 13:328–335.

Andrade JT, O'Neill K, Diener RB. (2009). Violence risk assessment and risk management: A historical overview and clinical application. In: JT Andrade (Ed.). *Handbook of Violence Risk Assessment and Treatment: New Approaches for Mental Health Professionals* (pp 3–39). New York: Springer Publishing Company.

Andrews DA, Bonta J. (1995). *Level of Service Inventory-Revised*. Toronto, ON: Multi-Health Systems.

Andrews DA, Bonta J. (2010). *The psychology of criminal conduct* (5th ed.). New Providence, NJ: Matthew Bender & Company.

Arbuckle JL, Wothke W. (1999). *Amos 4.0 user's guide.* Chicago, IL: Smallwaters.

Archer J. (1991). The influence of testosterone on human aggression. *Br J Psychol* 82:1–28.

Arseneault L, Moffitt TE, Caspi A, Taylor PJ, Silva PA. (2000). Mental disorders and violence in a total birth cohort: results from the Dunedin Study. *Arch Gen Psychiatry* 57:979–986.

Assadi SM, Noroozian M, Pakravannejad M, Yahyazadeh O, Aghayan S, Shariat SV, Fazel S. (2006). Psychiatric morbidity among sentenced prisoners: prevalence study in Iran. *Br J Psychiatry* 188:159–164.

Bandura A. (1991). Social cognitive theory of moral thought and action. In WM Kurtines & JL Gewirtz (Eds.). *Handbook of moral behavior and development: Vol. 1. Theory* (pp 45–103). Hillsdale, NJ: Lawrence Erlbaum.

Barkley RA, Fischer M, Edelbrock CS, Smallish L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria: 1. an 8-year prospective follow-up study. *J Am Acad Child Adolesc Psychiatry* 29:546–557.

Barriga AQ, Gibbs JC. (1996). Measuring cognitive distortion in antisocial youth: development and preliminary validation of the "How I Think" questionnaire. *Aggress Behav* 22:333–343.

Barriga AQ, Hawkins MA, Camelia CRT. (2008). Specificity of cognitive distortions to antisocial behaviours. *Crim Behav Ment Health* 18:104–116.

Barriga AQ, Gibbs JC, Potter G, Liau AK. (2001). *How I Think (HIT) questionnaire manual.* Champaign, IL: Research Press.

Barriga AQ, Landau JR, Stinson BL, Liau AK, Gibbs JC. (2000). Cognitive distortion and problem behaviors in adolescents. *Crim Justice Behav* 27:36–56.

Bergman LR, Andershed A-K. (2009). Predictors and outcomes of persistent or age-limited registered criminal behavior: a 30-year longitudinal study of a Swedish urban population. *Aggress Behav* 35:164–178.

Berman ME, Fallon AE, Coccaro EF. (1998). The relationship between personality psychopathology and aggressive behavior in research volunteers. *J Abnorm Psychol* 107:651–658.

Blackburn R. (2007). Personality disorder and antisocial deviance: comments on the debate on the structure of the Psychopathy Checklist-Revised. *J Pers Disord* 21:142–159.

Blackburn R, Logan C, Donnelly JP, Renwick SJD. (2008). Identifying psychopathic subtypes: combining an empirical personality classification of offenders with the Psychopathy Checklist-Revised. *J Pers Disord* 22:604–622.

Bland JM, Altman DG. (1995). Multiple significance tests: the Bonferroni method. *BMJ* 10:170.

Blonigen DM. (2010). Explaining the relationship between age and crime: contributions from the developmental literature on personality. *Clin Psychol Rev* 30:89–100.

Blonigen DM, Krueger RF. (2007). Personality and Violence: The Unifying Role of Structural Models of Personality. In: DJ Flannery, AT Vazsonyi, ID Waldman (Eds.). *The Cambridge Handbook of Violent Behavior and Aggression* (pp 288–305). New York: Cambridge University Press.

Blumstein A, Cohen J, Farrington DP. (1988). Criminal career research: its value for criminology. *Criminology* 26:1–35.

Boer DP, Hart SD, Kropp PR, Webster CD. (1997). *Manual for the Sexual Violence Risk-20: Professional Guidelines for Assessing Risk of Sexual Violence.* Vancouver, BC: British Columbia Institute on Family Violence.

Bonta J, Law M, Hanson K. (1998). The prediction of criminal and violent recidivism among mentally disordered offenders: a meta-analysis. *Psychol Bull* 123:123–142.

Borum R, Bartel P, Forth A. (2006). *Manual for the Structured Assessment for Violence Risk in Youth (SAVRY).* Odessa, FL: Psychological Assessment Resources.

Brennan PA, Mednick SA, Hodgins S. (2000). Major mental disorders and criminal violence in a Danish birth cohort. *Arch Gen Psychiatry* 57:494–500.

Brown GL, Ebert MH, Goyer PF, Jimerson DC, Klein WJ, Bunney WE, Goodwin FK. (1982). Aggression, suicide, and serotonin: relationships to CSF amine metabolites. *Am J Psychiatry* 139:741–746.

Burt SA. (2009). Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. *Clin Psychol Rev* 29:163–178.

Butchart A, Phinney A, Check P, Villaveces A. (2004). *Preventing violence: a guide to implementing the recommendations of the World report on violence and health.* Geneva: Department of Injuries and Violence Prevention, World Health Organization.

Chung I-J, Hill KG, Hawkins D, Gilchrist LD, Nagin DS. (2002). Childhood predictors of offense trajectories. *J Res Crime Delinq* 39:60–90.

Clark-Carter D. (2004). *Quantitative psychological research: A student's handbook.* Hove, UK: Psychology Press.

Cleckley H. (1941). *The Mask of Sanity,* 1st ed. St. Louis, MO: Mosby.

Coccaro EF, Berman ME, Kavoussi R J. (1997). Assessment of life history of aggression: development and psychometric characteristics. *Psych Res* 73:147–157.

Cohen J. (1988). *Statistical power analysis for the behavioral sciences,* 2nd ed. Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Coid J, Ullrich S. (2010). Antisocial personality disorder is on a continuum with psychopathy. *Compr Psychiatry* 51:426–433.

Coid J, Yang M. (2011). The impact of psychopathy on violence among the household population of Great Britain. *Soc Psychiat Epidemiol* 46:473–480.

Coid J, Yang M, Ullrich S, Roberts A, Hare R. (2009). Prevalence and correlates of psychopathic traits in a household population. *Int J Law Psychiatry* 32:65–73.

Coid J, Yang M, Roberts A, Ulrich S, Moran P, Bebbington P, Brugha T, Jenkins R, Farrell M, Lewis G, Singleton N. (2006). Violence and psychiatric morbidity in the national household population of Britain: public health implications. *Br J Psychiatry* 189:12–19.

Coid JW, Yang M, Ullrich S, Zhang T, Sizmur S, Farrington D, Rogers R. (2011). Most items in structured risk assessment instruments do not predict violence. *Journal of Forensic Psychiatry & Psychology* 22:3–21.

Cooke DJ, Michie C. (2001). Refining the construct of psychopathy: towards a hierarchical model. *Psychol Assess* 13:171–188.

Cooke DJ, Michie C, Hart SD. (2006). Facets of Clinical Psychopathy: Toward Clearer Measurement. In: CJ Patrick (Ed.). *Handbook of Psychopathy* (pp 193–202). New York: The Guilford Press.

Cooke DJ, Hart SD, Logan C, Michie C. (2004b). *Comprehensive Assessment of Psychopathic Personality - Institutional Rating Scale (CAPP-IRS).* Unpublished manuscript.

Cooke DJ, Michie C, Hart SD, Clark DA. (2004a). Reconstructing psychopathy: clarifying the significance of antisocial and socially deviant behavior in the diagnosis of psychopathic personality disorder. *J Pers Disord* 18:337–357.

Cooke DJ, Michie C, Hart SD, Clark D. (2005). Assessing psychopathy in the UK: concerns about cross-cultural generalisability. *Br J Psychiatry* 186:335–341.

Copeland WE, Miller-Johnson S, Keeler G, Angold A, Costello EJ. (2007). Childhood psychiatric disorders and young adult crime: a prospective, population-based study. *Am J Psychiatry* 164:1668–1675.

Corso PS, Mercy JA, Simon TR, Finkelstein EA, Miller TR. (2007). Medical costs and productivity losses due to interpersonal and self-directed violence in the United States. *Am J Prev Med* 32:474–482.

Costa FM, Jessor R, Turbin MS. (1999). Transition into adolescent problem drinking: the role of psychosocial risk and protective factors. *J Stud Alcohol* 60:480–490.

Dadds MR, Rhodes T. (2009). Aggression in young children with concurrent callous-unemotional traits: Can neurosciences inform progress and innovation in treatment approaches? In: S Hodgins, E Viding, A Plodowski (Eds.). *The Neurobiological Basis of Violence: Science and Rehabilitation* (pp 85–99). New York: Oxford University Press.

de Vogel V, de Ruiter C. (2006). Structured professional judgment of violence risk in forensic clinical practice: a prospective study into the predictive validity of the Dutch HCR-20. *Psychology, Crime & Law* 12:321–336.

de Vogel V, de Ruiter C, Bouman Y, de Vries Robbé M. (2007). *Guidelines for the assessment of protective factors for violence risk.* Utrecht: Van der Hoeven Stichting.

DeWall NC, Anderson CA. (2011). The General Aggression Model. In: PR Shaver & M Mikulincer (Eds.). *Human Aggression and Violence: Causes, Manifestations, and Consequenses* (pp 15–33). Washington, DC: American Psychological Association.

Dodge KA. (1980). Social cognition and children's aggressive behavior. *Child Dev* 51:162–170.

Dodge KA. (2011). Social Information Processing Patterns as Mediators of the Interaction between Genetic Factors and Life Experiences in the Development of Aggressive Behavior. In PR Shaver & M Mikulincer (Eds.). *Human Aggression and Violence: Causes, Manifestations, and Consequenses* (pp 165–185). Washington, DC: American Psychological Association.

Douglas KS, Skeem JL. (2005). Violence risk assessment: getting specific about being dynamic. *Psychology, Public Policy and Law* 11:347–383.

Douglas KS, Guy LS, Weir J. (2007). HCR-20 violence risk assessment scheme: Overview and annotated bibliography. Retreived August 8, 2012, from *http://kdouglas.files.wordpress.com/2007/10/hcr-20-annotated-biblio-sept-2010.pdf.*

Douglas KS, Yeomans M, Boer DP. (2005). Comparative validity analysis of multiple measures of violence risk in a sample of criminal offenders. *Crim Justice Behav* 32:479–510.

Doyle M, Carter S, Shaw J, Dolan M. (2012). Predicting community violence from patients discharged from acute mental health units in England. *Soc Psychiat Epidemiol* 47:627–637.

Edens JF, Campbell JS, Weir JM. (2007). Youth psychopathy and criminal recidivism: a meta-analysis of the Psychopathy Checklist measures. *Law Hum Behav* 31:53–75.

Eggleston EP, Laub JH. (2002). The onset of adult offending: a neglected dimension of the criminal career. *J Crim Justice* 30:603–622.

Elbogen EB, Johnson SC. (2009). The intricate link between violence and mental disorder: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Arch Gen Psychiatry* 66:152–161.

Elias N. (2000). *The Civilizing Process: Sociogenetic and Psychogenetic Investigations, rev*. ed. Cambridge, Mass.: Blackwell.

Elonheimo H, Sourander A, Niemelä S, Helenius H. (2011). Generic and crime type specific correlates of youth crime: a Finnish population-based study. *Soc Psychiat Epidemiol* 46:903–914.

Elonheimo H, Niemelä S, Parkkola K, Multimäki P, Helenius H, Nuutila AM, Sourander A. (2007). Police-registered offenses and psychiatric disorders among young males: the Finnish "From a boy to a man" birth cohort study. *Soc Psychiat Epidemiol* 42:477–484.

Elonheimo H, Sourander A, Niemelä S, Nuutila AM, Helenius H, Sillanmäki L, Ristkari T, Parkkola K. (2009). Psychosocial correlates of police-registered youth crime: a Finnish population-based study. *Nord J Psychiatry* 63:292–300.

Ennis BJ, Litwack TR. (1974). Psychiatry and the presumption of expertise: flipping coins in the courtroom. *California Law Review* 62:693–752.

Farrington DP. (1995). The development of offending and antisocial behavior from childhood: key findings from the Cambridge Study in Delinquent Development. *J Child Psychol Psychiatry* 36:929–964.

Farrington DP. (2003). Key Results from the First Forty Years of the Cambridge Study in Delinquent Development. In TP Thornberry, MD Krohn. *Taking Stock of Delinquency: An Overview of Findings from Contemporary Longitudinal Studies* (pp 137–183). New York: Kluwer Academic/Plenum Publishers.

Farrington DP. (2007). Origins of Violent Behavior over the Life Span. In: DJ Flannery, AT Vazsonyi, ID Waldman (Eds.). *The Cambridge Handbook of Violent Behavior and Aggression* (pp 19–48). New York: Cambridge University Press.

Farrington DP, Loeber R. (2000). Epidemiology of juvenile violence. *Child Adolesc Psychiatr Clin N Am* 9:733–748.

Farrington DP, Ttofi MM, Coid JW. (2009). Development of adolescence-limited, late-onset, and persistent offenders from age 8 to age 48. *Aggress Behav* 35:150–163.

Fazel S, Grann M. (2006). The population impact of severe mental illness on violent crime. *Am J Psychiatry* 163:1397–1403.

Fazel S, Gulati G, Linsell L, Geddes JR, Grann M. (2009). Schizophrenia and violence: systematic review and meta-analysis. *PLoS Med* 6(8):e1000120.

Fergusson DM, Horwood LJ, Nagin DS. (2000). Offending trajectories in a New Zealand birth cohort. *Criminology* 38:525–551.

First MB, Gibbon M, Spitzer RL, Williams JB. (1996). *User's Guide for the Structured Clinical Interview for DSM-IV Axis I Disorders - Research Version 2.0 (SCID-I).* New York: New York State Psychiatric Institute, Biometrics Research Department.

First M, Gibbon M, Spitzer RL, Williams JB, Smith Benjamin L. (1997). *Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II).* Washington: American Psychiatric Press.

Fontaine RG, Dodge KA. (2006). Real-time decision making and aggressive behavior in youth: a heuristic model of response evaluation and decision (RED). *Aggress Behav* 32:604–624.

Fontaine RG, Yang C, Dodge KA, Bates JE, Pettit GS. (2008). Testing an individual systems model of response evaluation and decision (RED) and antisocial behavior across adolescence. *Child Dev* 79:462–475.

Forth AE, Kosson DS, Hare RD. (2003). *The Psychopathy Checklist: Youth Version manual.* Toronto, ON: Multi-Health Systems.

Frisell T. (2012). *Violent crime: Addressing causation with family-based methods.* Stockholm: Karolinska Institutet.

Gendreau P, Little T, Goggin C. (1996). A meta-analysis of the predictors of adult offender recidivism: what works! *Criminology* 34:575–607.

Gordon A, Wong SCP. (2000). *Violence Reduction Program: Facilitator's Manual.* Saskatoon, Canada: Department of Psychology, University of Saskatchewan. Available at http://www.psynergy.ca

Gore K. (2008). *Adjusted actuarial assessment of sex offenders: The impact of clinical overrides on predictive accuracy.* Dissertation Abstracts International: Section B: The Sciences and Engineering, 68 (7-B), 4824.

Grann M, Fazel S. (2004). Substance misuse and violent crime: Swedish population study. *BMJ*. 328:1233–1234.

Grann M, Långström N. (2007). Actuarial assessment of violence risk: to weigh or not to weigh? *Crim Justice Behav* 34:22–36.

Grann M, Danesh J, Fazel S. (2008). The association between psychiatric diagnosis and violent re-offending in adult offenders in the community. *BMC Psychiatry* 8:92.

Grove WM, Zald DH, Lebow BS, Snitz BE, Nelson C. (2000). Clinical versus mechanical prediction: a meta-analysis. *Psychol Assess* 12:19–30.

Gustavson C. (2010). *Risk and prediction of violent crime in forensic psychiatry.* Lund University, Faculty of Medicine Doctoral Dissertation Series 2010:120. Lund: Lund University.

Gustavsson JP. (1997). *Stability and validity of self-reported personality traits: Contributions to the evaluation of the Karolinska Scales of Personality.* Stockholm: Karolinska Institute.

Guy LS, Edens JF, Anthony C, Douglas KS. (2005). Does psychopathy predict institutional misconduct among adults? A meta-analytic investigation. *J Consult Clin Psychol* 73:1056–1064.

Hall JR, Benning SD, Patrick CJ. (2004). Criterion-related validity of the three-factor model of psychopathy: personality, behavior, and adaptive functioning. *Assessment* 11:4–16.

Hanley JA, McNeil BJ. (1982). The meaning and use of the area under a receiver operating characteristic (ROC) curve. *Radiology* 143:29–36.

Hanson RK, Morton-Bourgon KE. (2005). The characteristics of persistent sexual offenders: a meta-analysis of recidivism studies. *J Consult Clin Psychol* 73:1154–1163.

Hanson RK, Morton-Bourgon KE. (2009). The accuracy of recidivism risk assessment for sexual offenders: a meta-analysis of 118 prediction studies. *Psychol Assess* 21:1–21.

Hardt J, Rutter M. (2004). Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *J Child Psychol Psychiatry* 45:260–273.

Hare RD. (1980). A research scale for the assessment of psychopathy in criminal populations. *Pers Indiv Differ* 1:111–119.

Hare RD. (1991). *The Hare Psychopathy Checklist-Revised*. Toronto, ON: Multi-Health Systems.

Hare RD. (2003). *The Hare Psychopathy Checklist-Revised manual.* Toronto, ON: Multi-Health Systems.

Hare RD, Clark D, Grann M, Thornton D. (2000). Psychopathy and the predictive validity of the PCL-R: an international perspective. *Behav Sci Law* 18:623–645.

Hare RD, Neumann CS. (2006). The PCL-R Assessment of Psychopathy: Development, Structural Properties, and New Directions. In: CJ Patrick (Ed*.). Handbook of Psychopathy* (pp 193–202). New York: The Guilford Press.

Hare RD, Neumann CS. (2009). Psychopathy: assessment and forensic implications. *Can J Psychiatry* 54:791–802.

Hare RD, Neumann CS. (2010). The role of antisociality in the psychopathy construct: comment on Skeem and Cooke (2010). *Psychol Assess* 22:446–454.

Harpur TJ, Hare RD, Hakstian AR. (1989). Two-factor conceptualization of psychopathy: construct validity and assessment implications. *Psychol Assess* 1:6–17.

Harris GT, Rice ME, Cormier CA. (1991). Psychopathy and violent recidivism. *Law Hum Behav* 15:625–637.

Hart SD, Cox DN, Hare RD. (1995). *Manual for the Psychopathy Checklist: Screening Version (PCL:SV).* Toronto, ON: Multi-Health Systems.

Hart SD, Michie C, Cooke DJ. (2007). Precision of actuarial risk assessment instruments: evaluating the 'margins of error' of group v. individual predictions of violence. *Br J Psychiatry* 190:60–65.

Heilbrun K. (2009). *Evaluation for risk of violence in adults.* New York: Oxford University Press.

Heilbrun K, Yasuhara K, Shah S. (2010). Violence Risk Assessment Tools: Overview and Critical Analysis. In: RK Otto, KS Douglas (Eds.). *Handbook of Violence Risk Assessment* (pp 1–17). New York: Routledge.

Hemphill JF, Hare RD, Wong S. (1998). Psychopathy and recidivism: a review. *Leg Crim Psychol* 3:139–170.

Hill J, Nathan R. (2008). Childhood antecedents of serious violence in adult male offenders. *Aggress Behav* 34:329–338.

Hirschi T, Gottfredson M. (1983). Age and the explanation of crime. *Am J Sociol* 89:552–584.

Hodgins S. (2007). Persistent violent offending: what do we know? *Br J Psychiatry* 190:12–14.

Hodgins S, Mednick SA, Brennan PA, Schulsinger F, Engberg M. (1996). Mental disorder and crime: evidence from a Danish birth cohort. *Arch Gen Psychiatry* 53:489–496.

Hofvander B, Ossowski D, Lundström S, Anckarsäter H. (2009). Continuity of aggressive antisocial behavior from childhood to adulthood: the question of phenotype definition. *Int J Law Psychiatry* 32:224–234.

Hu L, Bentler PM. (1999). Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct Equ Modeling* 6:1–55.

Huesmann LR, Dubow EF, Boxer P. (2009). Continuity of aggression from childhood to early adulthood as a predictor of life outcomes: implications for the adolescent-limited and life-course-persistent models. *Aggress Behav* 35:136–149.

Huesmann LR, Dubow EF, Boxer P. (2011). The Transmission of Aggressiveness Across Generations: Biological, Contexual, and Social Learning Processes. In: PR Shaver & M Mikulincer (Eds.). *Human Aggression and Violence: Causes, Manifestations, and Consequenses* (pp 123–142). Washington, DC: American Psychological Association.

Jeglum Bartusch DR, Lynam DR, Moffitt TE, Silva PA. (1997). Is age important? Testing a general versus a developmental theory of antisocial behavior. *Criminology* 35:13–48.

Johnson JG, Cohen P, Smailes E, Kasen S, Oldham JM, Skodol AE, Brook JS. (2000). Adolescent personality disorders associated with violence and criminal behavior during adolescence and early adulthood. *Am J Psychiatry* 157:1406–1412.

Kennealy PJ, Skeem JL, Walters GD, Camp J. (2010). Do core interpersonal and affective traits of PCL-R psychopathy interact with antisocial behavior and disinhibition to predict violence? *Psychol Assess* 22:569–580.

Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R. (2003.) Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. *Arch Gen Psychiatry* 60:709–717.

Kline P. (2000). *The handbook of psychological testing,* 2nd ed. London: Routledge.

Kratzer L, Hodgins S. (1997). Adult outcomes of child conduct problems: a cohort study. *J Abnorm Child Psychol* 25:65–81.

Kratzer L, Hodgins S. (1999). A typology of offenders: a test of Moffitt's theory among males and females from childhood to age 30. *Crim Behav Ment Health* 9:57–73.

Kropp PR, Hart SD. (2000). The Spousal Assault Risk Assessment (SARA) guide: reliability and validity in adult male offenders. *Law Human Behav* 24:101–118.

Krueger RF. (1999). The structure of common mental disorders. *Arch Gen Psychiatry* 56:921–926.

Krueger RF. (2006). Perspectives on the conceptualization of psychopathy. Toward an integration. In: CJ Patrick (Ed*.). Handbook of Psychopathy* (pp 193–202). New York: The Guilford Press.

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. *J Abnorm Psychol* 111:411–424.

Krueger RF, Schmutte PS, Caspi A, Moffitt TE, Campbell K, Silva PA. (1994). Personality traits are linked to crime among men and women: evidence from a birth cohort. *J Abnorm Psychol* 103:328–338.

Krug EG, Dahlberg LL, Mercy JA, Zwi AB, Lozano R. (Eds.). (2002). *World report on violence and health.* Geneva: World Health Organization.

Lardén M, Melin L, Holst U, Långström N. (2006). Moral judgement, cognitive distortions and empathy in incarcerated delinquent and community control adolescents. *Psychology, Crime and Law* 12:453–462.

Leistico A-MR, Salekin RT, DeCoster J, Rogers R. (2008). A large-scale meta-analysis relating the Hare measures of psychopathy to antisocial conduct. *Law Hum Behav* 32:28–45.

Loeber R. (1982). The stability of antisocial and delinquent child behavior: a review. *Child Dev* 53:1431–1446.

Loeber R, Farrington DP. (2000). Young children who commit crime: epidemiology, developmental origins, risk factors, early interventions, and policy implications. *Dev Psychopathol* 12:737–762.

Loeber R, Farrington DP, Waschbusch DA. (1999). Serious and Violent Juvenile Offenders. In: R Loeber & DP Farrington (Eds.). *Serious & Violent Juvenile Offenders: Risk Factors and Successful Interventions* (pp 13–29). Thousand Oaks, CA: Sage Publications.

Lynam DR. (1996). Early identification of chronic offenders: who is the fledgling psychopath? *Psychol Bull* 120:209–234.

Mannuzza S, Klein RG, Moulton JL 3rd. (2008). Lifetime criminality among boys with attention deficit hyperacitivity disorder: a prospective follow-up study into adulthood using official arrest records. *Psych Res* 160:237–246.

McGregor K, Castle D, Dolan M. (2012). Schizophrenia spectrum disorders, substance misuse, and the four-facet model of psychopathy: the relationship to violence. *Schizophr Res* 136:116–121.

McGuire J. (2009). Reducing personal violence: Risk factors and effective interventions. In: S Hodgins, E Viding, A Plodowski (Eds.). *The Neurobiological Basis of Violence: Science and Rehabilitation* (pp 287–327). New York: Oxford University Press.

Mills JF, Kroner DG, Morgan RD. (2011). *Clinician's Guide to Violence Risk Assessment.* New York: The Guilford Press.

Moffitt TE. (1993). Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev* 100:674–701.

Moffitt TE. (2007). A Review of Research on the Taxonomy of Life-Course Persistent Versus Adolescence-Limited Antisocial Behavior. In: DJ Flannery, AT Vazsonyi, ID Waldman (Eds.). *The Cambridge Handbook of Violent Behavior and Aggression* (pp 49–74). New York: Cambridge University Press.

Moffitt TE, Caspi A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Dev Psychopathol* 13:355–375.

Moffitt TE, Caspi A, Harrington H, Milne BJ. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: follow-up at age 26 years. *Dev Psychopathol* 14:179–207.

Moffitt TE, Caspi A, Dickson N, Silva PA, Stanton W. (1996). Childhood-onset versus adolescent-onset antisocial conduct in males: natural history from age 3 to 18. *Dev Psychopathol* 8:399–424.

Moffitt TE, Caspi A, Taylor A, Kokaua J, Milne BJ, Polanczyk G, Poulton R. (2010). How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. *Psychol Med* 40:899–909.

Monahan J. (1981). *The clinical prediction of violent behavior.* Washington, DC: Government Printing Office.

Monahan J, Steadman HJ, Silver E, Appelbaum PS, Robbins PC, Mulvey EP, Roth LH, Grisso T, Banks S. (2001). Rethinking Risk Assessment: the MacArthur study of mental disorder and violence. New York: Oxford University Press.

Monahan J, Steadman HJ, Robbins PC, Appelbaum P, Banks S, Grisso T, Heilbrun K, Mulvey EP, Roth L, Silver E. (2005). An actuarial model of violence risk assessment for persons with mental disorders. *Psychiatr Serv* 56:810–815.

Mordre M, Groholt B, Kjelsberg E, Sandstad B, Myhre AM. (2011). The impact of AD/HD and conduct disorder in childhood on adult delinquency: a 30 years follow-up study using official crime records. *BMC Psychiatry* 11:57.

Mossman D. (1994). Assessing predictions of violence: being accurate about accuracy. *J Consult Clin Psychol* 62:783–792.

Nagin DS, Farrington DP, Moffitt TE. (1995). Life-course trajectories of different types of offenders. *Criminology* 33:111–139.

Nas CN, Brugman D, Koops W. (2008). Measuring self-serving cognitive distortions with the "How I Think" Questionnaire. *Eur J Psychol Assess* 24:181–189.

Nestor PG. (2002). Mental disorder and violence: personality dimensions and clinical features. *Am J Psychiatry* 159:1973–1978.

Newhill CE, Eack SM, Mulvey EP. (2012). A growth curve analysis of emotion dysregulation as a mediator for violence in individuals with and without borderline personality disorder. *J Pers Disord* 26:452–67.

Odgers CL. (2009). The life-course persistent pathway of antisocial behaviour: Risks for violence and poor physical health. In: S Hodgins, E Viding, A Plodowski (Eds.). *The Neurobiological Basis of Violence: Science and Rehabilitation* (pp 23–41). New York: Oxford University Press.

Pallant J. (2007). *SPSS Survival Manual: A Step by Step Guide to Data Analysis using SPSS for Windows,* 3rd ed. Maidenhead, Berkshire: Open University Press.

Palmer EJ. (2007). Criminal thinking. In D Carson, B Milne, F Pakes, K Shalev, A Shawyer (Eds.). *Applying psychology to criminal justice* (pp 147–165). Chichester, UK: Wiley.

Patrick CJ, Hicks BM, Krueger RF, Lang AR. (2005). Relations between psychopathy facets and externalizing in a criminal offender sample. *J Pers Disord* 19:339–356.

Patterson GR, Reid JB, Dishion TJ. (1992). *A social interactional approach: Vol. 4: Antisocial boys.* Eugene: Castalia.

Pinker S. (2011). *The Better Angels of Our Nature: The Decline of Violence In History And Its Causes.* Great Britain: Allen Lane.

Piquero AR, Brezina T, Turner MG. (2005). Testing Moffitt's account of delinquency abstention. *J Res Crime Delinq* 42:27–54.

Plante N, Daigle MS, Gaumont C, Charbonneau L, Gibbs J, Barriga A. (2012). Validation of the 'How I Think Questionnaire' in a population of French-speaking adolescents with externalizing behaviors. *Behav Sci Law* 30:196–210.

Pulay AJ, Dawson DA, Hasin DS, Goldstein RB, Ruan WJ, Pickering RP, Huang B, Chou SP, Grand BF. (2008). Violent behavior and DSM-IV psychiatric disorders: results from the national epidemiologic survey on alcohol and related conditions. *J Clin Psychiatry* 69:12–22.

Pulkkinen L, Lyyra A-L, Kokko K. (2009). Life success of males on nonoffender, adolescence-limited, persistent, and adult-onset antisocial pathways: follow-up from age 8 to 42. *Aggress Behav* 35:117–135.

Quinsey VL, Harris GT, Rice ME, Cormier CA (1998). *Violent offenders: Appraising and managing risk.* Washington, DC: American Psychological Association.

Raine A. (1993). Features of borderline personality and violence. *J Clin Psychol* 49:277–281.

Raine A, Moffitt TE, Caspi A, Loeber R, Stouthamer-Loeber M, Lynam D. (2005). Neurocognitive impairments in boys on the life-course persistent antisocial path. *J Abnorm Psychol* 114:38–49.

Ramirez JM. (2003). Hormones and aggression in childhood and adolescence. *Aggress Violent Behav* 8:621–644.

Reza A, Mercy JA, Krug E. (2001). Epidemiology of violent deaths in the world. *Inj Prev* 7:104–111.

Rhee SH, Waldman ID. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychol Bull* 128:490–52.

Rhee SH, Waldman ID. (2007). Behavior-Genetics of Criminality and Aggression. In: DJ Flannery, AT Vazsonyi, ID Waldman (Eds.). *The Cambridge Handbook of Violent Behavior and Aggression* (pp 77–90). New York: Cambridge University Press.

Robins, LN. (1966). *Deviant children grown up.* Baltimore: Williams & Wilkins.

Robins E, Guze SB. (1970). Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *Am J Psychiatry* 126:983–987.

Rogers R. (2000). The uncritical acceptance of risk assessment in forensic practice. *Law Hum Behav* 24:595–605.

Rutter M. (1996). Transitions and turning points in developmental psychopathology: as applied to the age span between childhood and mid-adulthood. *Int J Behav Dev* 19:603–626.

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

Rönkä A, Oravala S, Pulkkinen L. (2002). "I met this wife of mine and things got on a better track": turning points in risk development. *J Adolesc* 25:47–63.

Sacks S, Cleland CM, Melnick G, Flynn PM, Knight K, Friedmann PD, Prendergast ML, Coen C. (2009). Violent offense associated with co-occurring substance use and mental health problems: evidence from CJDATS. *Behav Sci Law* 27:51–69.

Samenow SE. (2004). *Inside the criminal mind.* New York: Crown Publishers.

Sampson RJ, Laub JH. (2003). Life-course desisters? Trajectories of crime among delinquent boys followed to age 70. *Criminology* 41:555–592.

Schalling D, Edman G. (1993). *The Karolinska Scales of Personality (KSP). An Inventory for Assessing Temperament Dimensions Associated with Vulnerability for Psychosocial Deviance. Manual.* Stockholm: The Department of Psychiatry, Karolinska Institute.

Sestir MA, Bartholow B. (2007). Theoretical explanations of aggression and violence. In T Gannon, T Ward, AR Beech, D Fisher (Eds.). *Aggressive offenders cognition. Theory, research and practice* (pp 157–178). Chichester, UK: Wiley.

Shaver PR, Mikulincer M (Eds.). (2011). *Human Aggression and Violence: Causes, Manifestations, and Consequenses.* Washington, DC: American Psychological Association.

Siever LJ. (2008). Neurobiology of aggression and violence. *Am J Psychiatry* 165:429–42.

Simonoff E, Elander J, Holmshaw J, Pickles A, Murray R, Rutter M. (2004). Predictors of antisocial personality: continuities from childhood to adult life. *Br J Psychiatry* 184:118–127.

Simourd DJ, Hoge RD. (2000). Criminal psychopathy: a risk-and-need perspective. *Crim Justice Behav* 27:256–272.

Simourd DJ, Olver ME. (2002). The future of criminal attitudes research and practice. *Crim Justice Behav* 29:427–446.

Singh JP, Grann M, Fazel S. (2011). A comparative study of violence risk assessment tools: a systematic review and metaregression analysis of 68 studies involving 25,980 participants. *Clin Psychol Rev* 31:499–513.

Sjöstedt G, Grann M. (2002). Risk assessment: what is being predicted by actuarial prediction instruments? *Int J Forensic Ment Health* 1:179–183.

Skeem JL, Cooke DJ. (2010). Is criminal behavior a central component of psychopathy? Conceptual directions for resolving the debate. *Psychol Assess* 22:433–445.

Skeem JL, Mulvey EP. (2001). Psychopathy and community violence among civil psychiatric patients: results from the MacArthur Violence Risk Assessment Study. *J Consult Clin Psychol* 69:358–374.

Skeem JL, Miller JD, Mulvey E, Tiemann J, Monahan J. (2005). Using a five-factor lens to explore the relation between personality traits and violence in psychiatric patients. *J Consult Clin Psychol* 73:454–465.

Smith DJ. (2007). Crime and the life course. In: M Maguire, R Morgan, R Reiner (Eds.). *The Oxford Handbook of Criminology* (pp 641–683). New York: Oxford University Press.

Sourander A, Elonheimo H, Niemelä S, Nuutila A-M, Helenius H, Sillanmäki L, Piha J, Tamminen T, Kumpulainen K, Moilanen I, Almqvist F. (2006). Childhood predictors of male criminality: a prospective population-based follow-up study from age 8 to late adolescence. *J Am Acad Child Adolesc Psychiatry* 45:578–586.

Spitzer RL, Williams JBW, Gibbon M, First MB. (1989). *Instruction manual for the Structured Clinical Interview for DSM-III-R.* New York, NY: Biometrics Research.

Spitzer RL, Williams JBW, Gibbon M, First MB. (1990a). *Structured Clinical Interview for DSM-III-R.* Washington, DC: American Psychiatric Press.

Spitzer RL, Williams JBW, Gibbon M, First MB. (1990b*). Structured Clinical Interview for DSM-III-R Axis II Disorders,(SCID-II).* Washington, DC: American Psychiatric Press.

Spitzer RL, Williams JBW, Gibbon M, First MB. (1991). *Structured Clinical Interview for DSM-III-R Axis II Disorders, screening version.* Stockholm, Sweden: Pilgrim Press.

Stattin H, Magnusson D. (1989). The role of early aggressive behavior in the frequency, seriousness, and types of later crime. *J Consult Clin Psychol* 57:710–718.

Steadman H, Cocozza J. (1974). *Careers of the criminally insane.* Lexington, MA: Lexington Books.

Steadman HJ, Mulvey EP, Monahan J, Robbins PC, Appelbaum PS, Grisso T, Roth LH, Silver E. (1998). Violence by people discharged from acute psychiatric inpatient facilities and by others in the same neighborhoods. *Arch Gen Psychiatry* 55:393–401.

Stevens SS. (1946). On the theory of scales of measurement. *Science* 103:677–680.

Stouthamer-Loeber M, Loeber R, Wei E, Farrington DP, Wikström PO. (2002). Risk and promotive effects in the explanation of persistent serious delinquency in boys. *J Consult Clin Psychol* 70:111–123.

Stålenheim EG. (1997). *Psychopathy and biological markers in a forensic psychiatric population.* Acta Universitatis Upsaliensis. Comprehensive Summaries of Uppsala Dissertations from the Faculty of Medicine 701. Uppsala: Uppsala University.

Stålenheim EG. (2001). Relationships between attempted suicide, temperamental vulnerability, and violent criminality in a Swedish forensic psychiatric population. *Eur Psychiatry* 16:386–394.

Stålenheim EG. (2004). Long-term validity of biological markers of psychopathy and criminal recidivism: follow-up 6–8 years after forensic psychiatric investigation. *Psych Res* 121:281–291.

Stålenheim EG, von Knorring L. (1996). Psychopathy and Axis I and Axis II psychiatric disorders in a forensic psychiatric population in Sweden. *Acta Psychiatr Scand* 94:217–223.

Suhr DD. Exploratory or Confirmatory Factor Analysis? Retreived 2012–02–13 from *http://www2.sas.com/proceedings/sugi31/200–31.pdf*

Söderström H. (2002). *Neuropsychiatric background factors to violent crime*. Göteborg: Göteborg University.

Söderström H, Blennow K, Sjödin A-K, Forsman A. (2003). New evidence for an association between the CSF HVA:5-HIAA ratio and psychopathic traits. *Journal Neurol Neurosurg Psychiatry* 74:918–921.

Söderström H, Sjödin A-K, Carlstedt A, Forsman A. (2004). Adult psychopathic personality with childhood-onset hyperactivity and conduct disorder: a central problem constellation in forensic psychiatry. *Psych Res* 121:271–280.

Söderström H, Nilsson T, Sjödin A–K, Carlstedt A, Forsman A. (2005). The childhood-onset neuropsychiatric background to adulthood psychopatic traits and personality disorders. *Compr Psychiatry* 46:111–116.

Tackett JL, Krueger RF. (2011). Dispositional Influences on Human Aggression. In: PR Shaver & M Mikulincer (Eds.). *Human Aggression and Violence: Causes, Manifestations, and Consequenses* (pp 89–105). Washington, DC: American Psychological Association.

Tengström A, Hodgins S, Grann M, Långström N, Kullgren G. (2004). Schizophrenia and criminal offending: the role of psychopathy and substance use disorders. *Crim Justice Behav* 31:367–391.

The Swedish Council on Technology Assessment in Health Care (SBU). (2005). *Riskbedömningar inom psykiatrin. Kan våld i samhället förutsägas?* Stockholm: SBU.

The Swedish National Council for Crime Prevention (Brottsförebyggande rådet). (2011). *Det dödliga våldets utveckling. Fullbordat och försök till dödligt våld i Sverige på 1990- och 00-talet.* Rapport 2011:5. Västerås: Edita Norstedts.

Tiihonen J, Isohanni M, Räsänen P, Koiranen M, Moring J. (1997). Specific major mental disorders and criminality: a 26-year prospective study of the 1966 northern Finland birth cohort. *Am J Psychiatry* 154:840–845.

Tolan PH. (2007). Understanding Violence. In: DJ Flannery, AT Vazsonyi, ID Waldman (Eds.). *The Cambridge Handbook of Violent Behavior and Aggression* (pp 5–18). New York: Cambridge University Press.

Tolan PH, Gorman-Smith D. (1999). Development of Serious and Violent Offending Careers. In: R Loeber & DP Farrington (Eds.). *Serious & Violent Juvenile Offenders: Risk Factors and Successful Interventions* (pp 68–85). Thousand Oaks, CA: Sage Publications.

Tremblay RE. (2000). The development of aggressive behaviour during childhood: what have we learned in the past century? *Int J Behav Dev* 24:129–141.

Turner KMT, Sanders MR. (2006). Dissemination of evidence-based parenting and family support strategies: learning from the Triple P—Positive Parenting Program system approach. *Aggress Violent Beh* 11:176–193.

van Goozen SHM, Fairchild G. (2009). The neuroendocrinology of antisocial behaviour. In: S Hodgins, E Viding, A Plodowski (Eds.). *The Neurobiological Basis of Violence: Science and Rehabilitation* (pp 201–221). New York: Oxford University Press.

von Polier GG, Vloet TD, Herpertz-Dahlmann B. (2012). ADHD and delinquency - a developmental perspective. *Behav Sci Law* 30:121–139.

Walters GD. (2002). The Psychological Inventory of Criminal Thinking Styles (PICTS). A review and meta-analysis. *Assessment* 9:278–291.

Walters GD. (2003). Predicting institutional adjustment and recidivism with the Psychopathy Checklist factor scores: a meta-analysis. *Law Hum Behav* 27:541–558.

Walters GD. (2012). Substance abuse and criminal thinking: testing the countervailing, mediation, and specificity hypothesis. *Law Hum Behav* Jan 16.

Walters GD, Heilbrun K. (2010). Violence risk assessment and facet 4 of the Psychopathy Checklist: predicting institutional and community aggression in two forensic samples. *Assessment* 17:259–268.

Walters GD, Wilson NJ, Glover AJJ. (2011). Predicting recidivism with the Psychopathy Checklist: are factor score composites really necessary? *Psychol Assess* 23:552–557.

Walters GD, Knight RA, Grann M, Dahle KP. (2008). Incremental validity of the Psychopathy Checklist facet scores: predicting release outcome in six samples. *J Abnorm Psychol* 117:396–405.

Walters SJ. (2009). What is a Cox model? Retrieved 2012-05-22 from *http://www.medicine.ox.ac.uk/bandolier/painres/download/whatis/cox_model.pdf*

Webster CD, Douglas KS, Eaves D, Hart SD. (1997). *HCR-20: Assessing risk for violence.* Vancouver, BC: Mental Health, Law, and Policy Institute.

Webster CD, Martin ML, Brink J, Nicholls TL, Middleton C. (2004). *Manual for the Short-Term Assessment of Risk and Treatability (START), Version 1.0 (consultation ed.).* Port Coquitlam, BC: St. Joseph's Healthcare Hamilton.

Welsh JL, Schmidt F, McKinnon L, Chattha HK, Meyers JR. (2008). A comparative study of adolescent risk assessment instruments: predictive and incremental validity. *Assessment* 15:104–115.

Wong SCP, Gordon A. (1999). *Violence Risk Scale.* Saskatoon, Canada: University of Saskatchewan, Department of Psychology.

Young SJ, Thome J. (2011). ADHD and offenders. *World J Biol Psychiatry* 12:124–128.

Yu R, Geddes JR, Fazel S. (2012). Personality disorders, violence, and antisocial behavior: a systematic review and meta-regression analysis. *J Pers Disord* 26:775–792.

Zara G, Farrington DP. (2009). Childhood and adolescent predictors of late onset criminal careers. *J Youth Adolesc* 38:287–300.