



LUND UNIVERSITY

Risk and prediction of violent crime in forensic psychiatry

Gustavson, Christina

2010

[Link to publication](#)

Citation for published version (APA):

Gustavson, C. (2010). *Risk and prediction of violent crime in forensic psychiatry*. [Doctoral Thesis (compilation)]. Lund University: Faculty of Medicine.

Total number of authors:

1

General rights

Unless other specific re-use rights are stated the following general rights apply:

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

Read more about Creative commons licenses: <https://creativecommons.org/licenses/>

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

LUND UNIVERSITY

PO Box 117
221 00 Lund
+46 46-222 00 00

*Institute of Clinical Sciences, Malmö, Forensic psychiatry, Lund University:
Doctoral thesis*

Risk and prediction of violent crime in forensic psychiatry



LUND UNIVERSITY

Christina Gustavson

Malmö 2010

Cover drawing by AM G-son (Anna Monica Gustavson)

ISSN 1652-8220

ISBN 978-91-86671-36-5

Lund University, Faculty of Medicine Doctoral Dissertation Series 2010:120

Felix qui potuit rerum cognoscere causas.

Vergilius

Posthumously to
Landsfiskal Anders Gustavson, Länsmansgården, Tranemo

Contents

Abstract.....	7
Svensk sammanfattning	8
Acknowledgements.....	9
List of papers.....	11
Abbreviations.....	12
Introduction.....	13
Historical background.....	13
Historical development of forensic psychiatric risk assessments.....	14
“First generation”: dangerousness	14
“Second generation”: risk	15
“Third generation”: management.....	15
Forensic psychiatric risk factors	15
Historical risk factors.....	16
Clinical risk factors.....	17
Combined Instruments	18
PCL-R	18
HCR-20.....	18
Biological risk factors	19
Problematic aspects of forensic psychiatric risk assessments.....	20
Aims of the study.....	22
General aims	22
Specific aims.....	22
Paper I.....	22
Paper II.....	22
Paper III	22
Paper IV	22
Subjects and Methods	23
Baseline study	23
Forensic psychiatric investigations	25
Data collection	25
i. Psychiatric assessments	25
ii. Forensic personality assessments	26
iii. Self-rated personality assessments	26
Neurochemical analyses.....	26
Follow-up study: study population and data collection	27
Independent variables	27
Dependent variables.....	27
Data administration and statistical methods	28
Ethical considerations	28
Detailed methods used for the follow-up study	28
Paper I.....	28
Paper II.....	30
Paper III	30
Results.....	31
Paper I.....	31
Paper II.....	31
Paper III	32

Paper IV	33
Discussion	34
“Risk” in clinical assessments	35
Strengths and limitations.....	36
Clinical implications	37
References.....	38

Abstract

Objective: To test the predictive accuracy for violent recidivism of the age at onset of substance abuse, the platelet MAO-B activity, and various combinations of criminological and clinical risk factors among violent offenders in a prospective Swedish follow-up study.

Subjects: One hundred violent offenders, consecutively admitted for forensic psychiatric investigations between 1998 and 2001 (baseline).

Methods: Psychiatric and psychological data collection at baseline included age at onset of criminal behaviour and substance abuse and measures of platelet MAO-B activity. Known criminological and clinical risk factors were registered as well as ratings with the risk assessment instruments the Psychopathy Checklist-Revised (PCL-R), Historical, Clinical, Risk Management (HCR-20), and Life History of Aggression (LHA). After a mean follow-up time of almost five years, data on violent recidivism was obtained from official crime registers and analysed in relation to the clinical and criminological risk factors and to the results of the risk instruments.

Results: Twenty subjects were reconvicted for violent crimes during follow-up. The age at onset of substance abuse, but not the MAO-B activity (regardless of smoking habits), correlated with risk factors for violence and predicted criminal recidivism. Most criminological and clinical risk factors, such as age at first conviction, number of convictions, history of conduct disorder, substance abuse, and scores on the PCL-R, HCR-20, and LHA, demonstrated modest correlations with violent recidivism and moderate predictive ability with areas under the Receiver Operating Characteristics (ROC) curves between 0.72 and 0.76. Only age at first conviction and a history of substance abuse among primary relatives remained significant predictors in multivariate models. The development and use of forensic psychiatric risk assessments were analysed from a clinical point of view, considering changes over time, ethical dilemmas, and risk for integrity violations and misunderstandings due to divergent expectations and interpretations of terminology.

Conclusions: Early-onset substance abuse and age at first conviction are independent risk factors for recidivistic violence in forensic psychiatry. Simple historical risk factors describing behaviour have as good predictive accuracy as complex clinical risk assessment instruments.

Key words: violence, forensic risk assessment, risk factors, recidivism

Svensk sammanfattning

Syfte: Att undersöka den prediktiva precisionen hos ålder för missbruksdebut, MAO-B aktiviteten i blod, samt olika kombinationer av kriminologiska och kliniska riskfaktorer med avseende på återfall i våldsbrott hos tidigare våldsbrottsdömda i en prospektiv svensk uppföljningsstudie.

Studiepopulation: Hundra våldsbrottslingar, konsekutivt intagna för rättspsykiatrisk undersökning mellan 1998 och 2001 (index), gav sitt samtycke till medverkan i studien.

Metoder: Psykiatriska och psykologiska data insamlades vid index, inklusive debutålder för kriminalitet och för missbruk samt MAO-B aktivitet i trombocyter. Information om kriminologiska och kliniska riskfaktorer insamlades, inklusive resultat av skattningar med bedömningsinstrument (PCL-R, HCR-20, LHA). Uppgifter om nya fällande domar insamlades via officiella register efter närmare fem års uppföljningstid och analyserades i relation till kriminologiska och kliniska riskfaktorer och till resultaten av riskbedömningarna.

Resultat: Tjugo förövare dömdes för nya våldsbrott under observationstiden. Ålder vid missbruksdebuten, men inte MAO-B aktiviteten i trombocyter (oberoende av rökning), korrelerade med riskfaktorer för våld och predicerade återfall i brott. Merparten kriminologiska och kliniska riskfaktorer, som ålder vid första dom, antal tidigare fängelsedomar, uppförandestörning i barndomen, missbruk och poäng på PCL-R, HCR-20 och LHA uppvisade modesta samband med återfall i våldsbrott och moderat prediktiv förmåga med kurvor för arean under Receiver Operating Characteristics (ROC) mellan 0,72 och 0,76. Bara ålder vid den första domen och drogmissbruk bland förstagrads släktingar förblev signifikanta prediktorer i multivariata modeller. Utveckling och användning av riskbedömningsinstrument inom det rättspsykiatriska fältet analyserades utifrån en klinisk utgångspunkt med särskilt avseende på förändringar över tid, etiska dilemman samt risken för integritetskränkningar och felbeslut beroende på motstridiga förväntningar och begreppsförvirring.

Slutsatser: Tidigt debuterande missbruk och fällande domar för tidigare brott är oberoende riskfaktorer för upprepad våldsbrottslighet bland psykiskt störda lagöverträdare. Enkla historiska riskfaktorer har samma prediktiva värde som komplexa riskbedömningsinstrument.

Acknowledgements

This thesis presents results from a follow up study of the Gothenburg Forensic Neuropsychiatry Project, a research project aimed at disclosing neuropsychiatric vulnerability factors in perpetrators of severe violent and sexual crimes. The project was initiated by Professor Anders Forsman and Henrik Anckarsäter in 1995, follow-up studies were started in 2002, and a number of cohorts are still followed continuously.

The project has involved many co-workers who have enhanced the work by their efforts and enthusiasm.

First of all I would like to thank my supervisor, Nóra Kerekes, for her energy, support, and encouragement, which combined with a great sense of humour made it possible to finalize this work.

My gratitude also goes to Henrik Anckarsäter. I am proud and honoured to call him my supervisor because of his brilliant ideas and inventiveness, his ability to see connections, to find new angles, and to approach a problem from unexpected directions.

I am grateful to Thomas Nilsson for his guidance, his great patience in going through registers with me, his ability to remain calm even when I was not, and for his kind support.

I would also like to thank Professor Anders Forsman at the Department of Forensic Psychiatry in Gothenburg for teaching me how to perform the forensic psychiatric investigations which made it possible for me to specialize in this medical field, as well as for his good stories about the unwritten history of forensic activities in Sweden, Ola Ståhlberg, for valuable advice and explanations regarding a variety of topics, ranging from road maps to statistics, Sune Innala, for his supportive kindness in helping me carry out my first international presentation of Paper I in this thesis, and Björn Hofvander, one of the kindest and most polite persons I ever met, for always replying to my questions and always being helpful. Many thanks are due to Anders Yngvesson-Rastenberger and Anita Larsson at the Forensic Psychiatry Department in Malmö, Martin Grann at the Centre for Violence Prevention at the Karolinska Institute in Stockholm, who so kindly shared his knowledge about new instruments and helped me and our research team to find our way through the bureaucracy of official registers, Lars-Erik Ingerloo at the National Board of Forensic Medicine, who shared his own research with us and gave us access to forensic registers built up by him over long time, Martin Rödhölm, Tobias Nordin, and Bengt-Arne Andersson at the Psychiatric Clinic in Borås, Bernard Taylor at SiS, and Birger Gröndahl, my tutor while working in Säter, as well as to colleagues and staff members at the Forensic Psychiatric Clinics in Kristinehamn and Jönköping.

The assistance of Monika Montell and Agneta Brimse at the research group for Forensic Psychiatry in Gothenburg is very much appreciated: Monika who searched for, found, and retrieved investigation reports, court sentences, and other crucial material, and Agneta, as much for her wonderful sense of humour, friendship, and discussions on various topics as for her help with secretarial tasks.

Generous financing and work conditions were provided by the National Board of Forensic Medicine through its Research and Development Committee, from the Head of the

Department of Forensic Psychiatry in Gothenburg at that time, Gunnar Söderström, and from the Göteborg Medical Society.

The follow-up project has also been generously supported by The Psychiatric Clinic and the regional Research and Development (FoU) department in Borås, the Göteborg Medical Society, and the Lindhaga Foundation.

In addition to the colleagues, scientific contacts, and friends acknowledged above, I would like to mention quite another group of persons, who all, in their own way, have given me a great variety of contributions towards the person I am today, as well as to my work and achievements: My families: The Gustavsons, The Carlssons, the Melanders, and particularly my daughters Sarah and Fridah. Furthermore, my thanks go to Birgitta Bergman and Robert B. Paulson.

Finally, I owe my most valuable knowledge in this field to the patients who have shared their time, life histories, and inner worlds with the team during the main study of the project, thus making it possible to realize this follow-up study.

List of papers

I: Gustavson C, Ståhlberg O, Sjödin A-K, Forsman A, Nilsson T, Anckarsäter H: Age at onset as a crucial covariate of psychopathic traits and aggression in adult offenders. *Psychiatry Research* 2007;153:195-198.

II: Gustavson C, Wass C, Mansson J-E, Blennow K, Forsman A, Anckarsäter H, Nilsson T: Platelet monoamine oxidase B did not predict destructive personality traits or violent recidivism: A prospective study in male forensic psychiatric examinees. *Neuropsychobiology* 2010;61:87-96.

III: Nilsson T, Wallinius M, Gustavson C, Anckarsäter H, Kerekes N: Violent recidivism: a long-time follow-up study of systematically assessed mentally disordered perpetrators of severe crimes against others. *Manuscript*.

IV: Nilsson T, Munthe C, Gustavson C, Forsman A, Anckarsäter H: The precarious practice of forensic psychiatric risk assessments. *International Journal of Law and Psychiatry* 2009;32:400-407.

Papers I, II, and IV were printed with permission from the publishers.

Abbreviations

AD/HD	Attention-deficit/hyperactivity disorder
AUC	Area under the curve
CNS	Central nervous system
CSF	Cerebrospinal fluid
DA	Dopamine
DSM-IV	Diagnostic and statistical manual of mental disorders
HCR-20	Historical Clinical Risk management - 20-items”
HVA	Homovanillic acid
LHA	Life History of Aggression
MAO-A	Monoamine-oxidase A
MAO-B	Monoamine-oxidase B
MHPG	3-metoxy, 4-hydroxy phenylglucol
NPV	Negative Predictive Value
PCL-R	Psychopathy checklist – revised
PD	Personality disorder
PPV	Positive Predictive Value
ROC	Receiver Operating Characteristics
SCID-I	Structured Clinical Interview for DSM-IV – Axis I disorders
SCID-II	Structured Clinical Interview for DSM-IV – Axis II disorders
SPECT	Single Photon Emission Computerized Tomography
TCI	Temperament and Character Inventory
5-HIAA	5-hydroxyindolacetic acid
5-HT	5-hydroxy-tryptamine

Introduction

Historical background

The historical tradition of incapacitating violent offenders in order to prevent them from committing new crimes is very old. Penal legislation during antiquity and the middle ages stipulated various forms of punishments and incapacitation depending on the crimes and the perpetrator's social standing. Corporal punishments were far more common than various forms of imprisonment, often in the form of deportations into slave labour or war service for life. Punishments were motivated by the need to undo the damage suffered by society, its government or ruler, and/or God. The unpredictability, inequality, and metaphysical grounds of penal law were questioned by the representatives of the "Age of Enlightenment" [1], the period in western philosophy and intellectual, scientific, and cultural life when reason was advocated as the primary source and legitimacy for authority, i.e. roughly corresponding to the 18th century, and especially the French Revolution (1789-1799). Montesquieu advocated the separation of powers in order to make judicial power independent from the executive power [2]. At the same time, Voltaire demanded that criminal justice should concentrate on the prevention rather than on the punishment of crime [3, 4]. Every citizen was considered to be equal before the law, and criteria were set up for the relationship between the length of the punishment and the severity of the crime [5, 6]. The offender's personal characteristics should have no bearing on the punishment dispensed by the court. Punishments were motivated by ideas of responsibility, in the sense that if an individual who is free to choose how to act makes the choice of committing a criminal act, he is guilty and thus deserves punishment (Immanuel Kant, "The Right to Punish", retrieved from Murphy 1978) [7].

Around the same time, as an almost parallel project to the rethinking of the penal law, modern psychiatry started to develop under the influence of the Age of Enlightenment ideas. This new science studied mechanisms behind human behaviour, focusing not only on persons suffering from various forms of "insanity", but also on those guilty of what was regarded as incomprehensible acts or patterns of behaviour, especially when involving destructive or immoral components. In the late 19th century, Cesare Lombroso rejected the notion that crime was a characteristic trait of human nature and suggested that "anthropological criminality" should be in focus. This was the Darwinian idea that criminals were atavistic individuals with physical and mental characteristics retained from earlier steps in human development, meaning that criminality was inherited (Lombroso C: Sociological Theories of Deviance, retrieved from Rock 2002 [8]). This led to a break with the classic penal law which had required that every crime should be followed by a specific punishment. In the new school, referred to as "positive penal law", punishments should no longer serve as reprisals, but were instead supposed to be the optimal form of "treatment" needed by the individual offender in order not to re-offend, or in the most extreme cases, needed by society as protection against the threat posed by the offender. Forensic psychiatry was developed on the basis of these ideas, which gained increasing influence over legislations across the countries in the western world during the late 19th and early 20th centuries. By this development, courts also became dependent on expertise in psychiatry and psychology in order to grasp the complex psychiatric and psychological factors behind the crime that could be regarded as explanatory or exculpatory. Also predictions of future behaviour ("dangerousness") and proposals for preventive measures started to attract attention. The intention was here also to adapt the punishment to the needs of the perpetrator in order to serve as a relapse-preventive measure. Experts were often persons generally known for their good repute but lacking in documented knowledge about specific issues [9]. Internationally, positive penal law and forensic

psychiatry gained considerable influence during most of the 20th century. This practice came to be questioned, however, especially in the USA, and an intense debate was started in the 1960s, calling for empirical investigation of the accuracy or precision of arbitrary ways of forming judgements [10].

When great numbers of persons who had been assessed as dangerous and placed in institutions in the USA were released after a Supreme Court decision, scientists were for the first time given the opportunity to directly investigate the accuracy and precision of the risk assessments [11, 12]. The fact that psychiatric diagnoses and symptomatology were found to be among the factors with the poorest predictive value regarding “dangerousness” gave rise to further protests against the role of psychiatry in crime prevention, especially when it came to detention and other processes reducing individual liberties [13]. After the ensuing pessimism of the 1970s, research with a focus on “risk” instead of “dangerousness” was launched by groups of experts claiming that psychiatry and psychology indeed had important contributions to make to crime prevention [5, 14]. Research along these lines was first developed in Canada, soon to be followed by many groups of researchers across North America and in Europe.

Historical development of forensic psychiatric risk assessments

In forensic psychiatry, the term “risk” is used as an expression for the probability of violent offending by a certain person during a certain period of time [15]. Forensic psychiatric risk assessments are performed by psychiatrists, psychologists, and other mental health care professionals as ordered by court to provide information of relevance in sentencing and court supervision of compulsory forensic psychiatric treatment. In Sweden today, forensic psychiatric risk assessments are performed by the National Board of Forensic Medicine on behalf of the courts, both as part of pre-trial assessments and in considerations on discontinuance of life-sentences. Forensic psychiatric or psychological risk assessments are also performed in the forensic psychiatric treatment system, in the prison system, in institutional care of adolescents, and in general psychiatry. The clinical objective is to avoid and prevent violent behaviour, not merely to predict the possibility of such acts [16-18]. There is professional consensus today that risk assessments by several different methods and instruments serve better than chance to identify subjects at increased or decreased risk of criminal re-offending. However, considerable controversy of opinions remains about the overall reliability of assessments, the choice of methods, and the possible societal use of these assessments. There is a consensus that in order to be regarded as evidence-based, assessments have to employ methods tested for validity in prospective, controlled trials, and that such validity has to be controlled for effects of gender, ethnicity, age, and type of population. To provide a background to the different methods in use today, let us briefly look at the different phases, referred to as “generations”, of risk assessment methods that have been in use since the renewal of interest in the field during the 1980s.

“First generation”: dangerousness

“Dangerousness” was the classical forensic psychiatric concept used to describe persons thought to be prone to act violently towards others. It was defined as a “potential condition within a certain person in a given environmental situation, which implies a particular risk for a violation of other persons’ legal rights or of the societal organization itself” [19]. Up until the 1970s, risk assessments were clinical assessments of dangerousness provided by psychiatry. This changed, however, when several international studies reported that the vast majority of individuals who had been assessed as “dangerous” by psychiatrists and therefore

deprived of liberty did not relapse in criminality as predicted when released [11, 12, 20]. These findings, which, by the way, have never been disproved in new prospective studies, led to a dramatic reconsideration of the value of such assessments. Psychiatric evaluations of the propensity to commit crimes rapidly fell into disrepute and had minor importance throughout the 1970s and -80s in Sweden as well as in the rest of the western world.

“Second generation”: risk

From the beginning of the 1980s, a strong societal demand for coercive measures against “dangerous” mentally disordered persons re-emerged, leading to a new surge of interest in what was called a new “generation” of violence prediction research. This development was in tune with the emerging dominance of the risk concept in our culture at that time [21]. Instead of categorical assessments of “dangerousness”, the “risk” of violence was measured as the proportion of individuals who relapsed or committed a certain type of crime in a (hypothetical) group sharing similar rating scores on structured or semi-structured rating scales, or “instruments”, such as the Psychopathy Checklist (-Revised) (PCL-R) [22, 23], and the Violence Risk Appraisal Guide [24, 25]. These instruments were translated and adapted to Swedish conditions during the 1990s [26-30].

“Third generation”: management

A third generation of risk research is now proposing “risk management”, a structured clinical assessment, for example by instruments such as the “Historical Clinical Risk management - 20-items” (HCR-20) [31-33]. These instruments focus on identifying risk factors as an aid to prevent violence in the management of individual cases instead of merely performing predictions. Factors such as attitudes, impulsivity, mental state, family and social circumstances, substance use, availability and acceptance of support are taken into consideration when making a structured professional judgment [34]. The obvious argument against this is that since risk factors are not defined as causal factors, it cannot be taken for granted that management of such factors necessarily influences the outcome. A structured professional judgment of management also has to consider the availability and acceptance of support from professionals as well as from family and pro-social friends [35].

Forensic psychiatric risk factors

The first generation of global clinical risk assessments have been developed via actuarial assessments based on statistical methods and led up to today’s combined clinical-actuarial assessments covering both historical and dynamic clinical aspects. The historical variables include criminological, dispositional, and contextual risk factors covering different areas from the life history, while the clinical variables cover current individual characteristics. An overview of the assessment methods and types of variables used in the different risk assessment generations is given in Table 1.

Table 1. The different phases of risk assessment methods

	First generation	Second generation	Third generation
Historical factors	Global clinical assessment based on subjective interpretation of individual history	Actuarial assessment based on, criminological, contextual, and past clinical variables	Actuarial assessment based on, criminological, contextual, and clinical variables
Clinical factors	Global clinical assessment based on subjective interpretation of current traits and symptoms	Actuarial assessment based on items covering current clinical traits and symptoms	Clinical assessment based on items covering current clinical dynamic factors
Focus of assessment	Dangerousness	Risk	Management

Historical risk factors

Actuarial risk assessment instruments are based on historical, concrete, and statistical information. The instruments are predictive or prognostic in nature. They are designed to predict the future rather than intended to include measurements or quantifications of personality traits or psychological dynamics. Other facets of risk, such as severity, duration, or frequency, are ignored. Many studies have shown that a number of these historical risk factors have strong relevance in risk predictions. Prior violent offenses, mainly in the forms of juvenile delinquency (historical risk factors), younger age, and underprivileged social status (dispositional risk factors) recur across studies as the most important predictors of violent recidivism [35-46]. They remain the most important risk factors even when other types of risk factors, such as clinical factors (e.g. major mental disorders), are considered [44, 47]. An early onset of delinquency before the age of 13 years has been shown to increase the risk of subsequent serious, violent, and chronic offending by a factor of 2-3 [48]. Male gender, youth, substance abuse, and low age at onset of antisocial behaviour always turn up as risk factors in research on the probability of relapse in violence [49, 50]. The life history of a person can be analysed and described in a life-time perspective [51, 52] that can be influenced by life factors over time such as working situation and family history as well as short-term incidents like illness, divorce, or parenthood. Early physical maltreatment predicts adolescent psychological and behavioural problems, such as increased levels of aggression, beyond the effects of other factors associated with maltreatment [53].

Group dynamics, in contrast, are generally overlooked in forensic psychiatry, which is strongly focused on the individual. Groups of people act aggressively as a collective entity, not as a collection of aggressive individuals. The psychology of group aggression is based on group dynamics theory [54], which identifies different intensities of group aggression, such as low intensity aggression, including ostracism, hazing, and teasing; mid-intensity, e.g. bullying, harassment; and high intensity aggression, e.g. mobs and gangs. Gang members are known to be among the most serious violent offenders; they more often use weapons in a fight [55] and cause more violent assaults [56].

Clinical risk factors

Clinical risk assessments are based on factors such as psychiatric diagnoses and measures of traits and cognitive functions. It is important to remember that historical factors recur in clinical diagnoses such as conduct disorder or antisocial personality disorder, which makes it difficult to disentangle the overall relevance of clinical factors from that of historical factors.

Clinical risk factors include assessments of violence-related behavioural traits, such as aggression and antisocial personality disorder, but also substance abuse with early onset of poly-drug problems and links to aggression (type II alcoholism [57]). For example, the risk for any type of criminal recidivism was 4.8 times higher and that for violent recidivism 3.7 times higher among subjects with self-reports suggesting a categorical diagnosis of antisocial personality disorder as compared to offenders without [58]. Aggression against self or others is a core component of borderline personality disorder, and the aggressive acts of these patients are largely of the impulsive type [59, 60]. The risk of violent and non-violent offending attributable to substance abuse is also high [61]. Sixteen per cent of all violent crimes in Sweden during 1988-2000 were committed by persons who had hospital discharge diagnoses of alcohol abuse, and more than a tenth of all violent crimes were committed by patients diagnosed with drug abuse. Treatment services aimed at alcohol and drug abusers thus seem to have the potential of reducing violent offending [62].

In the field of mood disorders, research on suicide risk assessments has generally advanced further than that on violence directed at others, and some lessons may undoubtedly be learned from this area. For both suicide – as an introvert aggressive behaviour – and violent acting out – as an extrovert aggressive behaviour – a number of common clinical factors may be used to identify persons at increased risk. One such risk factor is agitated depression [63]. Depression is the most common psychiatric disorder associated with suicide [64]. People who have attempted suicide show a significantly stronger implicit association between death/suicide and self than do psychiatrically distressed individuals who have not attempted suicide [65]. Aggressive behaviours are highly prevalent in depressed youths, with similar types and levels seen in both males and females [66]. Previous suicide attempts are strong risk factors for future death by suicide.

Persons who have developed psychotic (“major”) mental disorders are at increased risk across the lifespan of committing both non-violent and violent crimes [67] as compared to people without such diagnoses [68-70]. The risk increase has been reported to be up to five-fold in several of these studies, and even higher among women [70-74]. This risk has recently been shown to be heavily influenced by concomitant familial effects for crimes and substance abuse, to the extent that when using unaffected sibling controls or controls matched for substance abuse, the increased risk almost disappears [47, 74]. Bivariate analyses have shown the incidence of violence to be higher for people with severe mental illness, but significantly so only for those with co-occurring substance abuse and/or dependence [75]. Multivariate analyses revealed that severe mental illness alone did not predict future violence; which instead was associated with historical (past violence, juvenile detention, physical abuse, parental arrest record), clinical (substance abuse, perceived threats), dispositional (age, sex, income), and contextual (recent divorce, unemployment, victimization) factors. However, most of these factors were endorsed more often by subjects with severe mental illness [75]. In schizophrenia, violence often precedes the clinical symptoms [76], which complicates the use of psychosis as a risk indicator. Studies have reported an increased risk of homicide among people suffering from schizophrenia [77], where the risk of violence may be increased during the first acute phase of the illness (possibly due to confusion) and in the later phases, when

severe violence is more often directed toward close relatives, e.g. parents, than towards strangers [78].

Childhood-onset social interaction and behavioural problems form the most relevant psychiatric symptom cluster in relation to pervasive adult violent behaviour, while late-onset mental disorders are more often associated with single acts of violent or sexual aggression [79]. It has to be remembered that early-onset behaviour disorders have been shown to constitute a core risk factor for all other mental disorders and for adulthood criminality and substance abuse, and therefore may act as a confounder behind all these associations [80] or as an early endophenotype for the later combination of mental disorders and criminality [81].

Attention deficit/hyperactivity disorder (AD/HD) and conduct disorder may interact to give a higher risk for substance abuse than either disorder alone. Different types of abuse have been reported to correlate with oppositional defiant disorder, tics, and separation anxiety disorder [82, 83]. Major depressive disorders in mothers were predictors of substance abuse in their children with AD/HD, while paternal or maternal age, parental level of education, or type of occupation did not affect the risk of abuse [84]. Oppositional behaviour in youths is one of the strongest predictors of a wide range of psychiatric disorders. Compared to girls with AD/HD, boys with AD/HD report higher levels of violent and non-violent delinquency and are described by teachers as having more conduct problems [85].

Combined Instruments

Combined rating instruments, using clinical as well as historical information, have been developed to overcome the limitations of either method alone. Two such instruments that have gained wide acceptance in clinical settings – the PCL-R and the HCR-20 – have been applied in the thesis work and will be briefly presented here.

PCL-R

The concept of psychopathy as reflected in the PCL-R has long been regarded as a core personality indicator of risk across methods and diagnostic categories. Recent research has changed this picture somewhat by showing that the predictive power of the PCL-R is carried by the behavioural factor, i.e. previous criminal behaviour, which by itself outperforms the total score from the instrument. Walters, Raymond, Grann, and Dahle [86] have in a collaborative study analysed six samples from different countries with respect to the four factors of PCL-R and found virtually no support for any incremental validity of factors 1, 2, and 3 (Interpersonal, Affective, and Lifestyle, respectively) above and beyond factor 4 (Antisocial behaviour).

HCR-20

The frequently used inventory, HCR-20 is considered to be a synthesis of the two current methods, the clinical and the historical risk assessments. This instrument collects historical and clinical data as well as information on aspects of risk management. HCR-20 has the advantage of being easy to use, and the predictive accuracy is considered to be approximately 75-80 % [87] when used among persons with previous criminality, but in view of the strong impact of criminal history [88], the usefulness of HCR-20 must be questioned when applied to persons who have not yet committed any crime.

Biological risk factors

Over the past few decades, increasing interest has been directed at the biological basis of aggression and crime and the possibility of using biology to inform us on accountability, causation, and prediction in relation to crime.

Genetic studies, including twin and adoption studies, all conclude that there is a genetic influence to criminal [89] and antisocial behaviours [90, 91]. By such analyses of twin and adoption studies, it has been possible to quantify the overall importance of this susceptibility, which is estimated to explain about 65 % of the inter-individual liability for criminal behaviours and aggressive antisocial behaviours [92], and lead to a four-fold increase in risk for violent crimes in first-degree relatives of a proband sentenced for such a crime [74]. The interaction between biological and social factors is also emphasized as particularly important in many studies [93]. Having stated that biological, constitutional factors are important in the background of violent crimes, it must be added that it has been far more difficult than expected to identify genetic risk factors. Just a decade ago, genetic variants were still debated in the press as “warrior genes” [94-96], but it is now recognized that no genetic variant may explain more than a couple of per cents of the total variation in psychiatric phenomena, including behaviour patterns [97].

The possibilities and problems involved in using biological markers for risk assessments will be exemplified here by two biological measures that are, of course, genetically influenced though first explored before the era of molecular genetics: the monoaminergic neurotransmitters and the activity of the enzyme monoaminoxidase (MAO) B in blood platelets. Among the different neurotransmitters studied, serotonin (5-hydroxytryptamine, 5-HT) has a fundamental role in the regulation of aggressive behaviour. Serotonin facilitates prefrontal cortical regions that are involved in suppressing aggressive manifestations. Studies have shown that selective serotonin reuptake inhibitors (SSRIs) reduce impulsive aggression [98] and neurobiological studies have implicated reduced concentrations of the serotonergic metabolite 5-hydroxyindoleacetic acid (5-HIAA) [99-102] and reduced neuroendocrine responses to serotonergic probes [98, 103-105] in patients with aggressive personality disorder or individuals who have made violent suicide attempts [106, 107]. The catecholamines dopamine and norepinephrine may also increase the probability of aggression. Dopamine is involved in the initiation and performance of aggressive behaviour [108]. Decreased dopamine D1 receptors have been reported in depressed patients with anger attacks [109]. In patients with personality disorders, a blunted serotonergic activity was associated with normal to increased noradrenergic activity [104]. One of the strongest predictive models presented for violent recidivism was described by Virkkunen and co-workers in a prospective study of biochemical and family variables, where violent recidivism was predicted by means of low CSF-concentrations of 5-HIAA (serotonergic metabolite) and 3-methoxy-4-hydroxyphenyl-glycol (MHPG, a norepinephrine metabolite) in combination with early paternal violence, alcoholism, and absence from home as well as presence of brothers in the family [110].

The platelet activity of MAO has long been of interest in psychiatric research as it is easily accessible from peripheral cells (blood) and forms the substrate for a class of very potent psychotropic drugs – the monoamine oxidase inhibitors – and it may reflect biological propensities of relevance for personality traits and psychopathology in the central nervous system (CNS) [111]. There are two subclasses of MAO (A and B) that differ in biochemical properties and functions but are both represented in the brain and in peripheral tissues. MAO in blood platelets is exclusively of the B-type and has the same amino acid sequence as

MAO-B in the brain [112], but no direct correlations have been demonstrated between blood platelets and brain MAO-B [113]. Outside the brain, the MAO-A subtype is mainly detectable in human placenta and fibroblasts. MAO-B is primarily responsible for the metabolism of dopamine and a number of exogenous monoamines, while the A-form has serotonin and noradrenalin as substrates [114]. Since compounds in cigarette smoke inhibit MAO, smoking is associated with low platelet MAO activity [115]. The strongest association with low platelet MAO activity reported has been with substance abuse, especially “type II” alcoholism (with patrilinear inheritance, early-onset substance abuse, and antisocial behaviours [116, 117]), in clinical settings as well as in outpatients. In the context of forensic psychiatric risk assessments, reports on platelet MAO-B as a risk marker have often been extremely enthusiastic (e.g. “a simple blood test predicts dangerous character” [118]). Several studies have reported associations with aggressiveness [119-121] and violence and aggression in crime [122, 123], but not all studies have found such associations [124]. In a study by Alm and colleagues [122], low platelet MAO-B stood out as one of two independent but synergistic predictors for continued criminal activity into adulthood together with psychopathic personality traits according to the PCL-R.

With molecular genetics investigations, a number of candidate genes have been explored in relation to impulsive aggression or disorders characterized by high aggression. For example, 5-HT_{2A} receptor single nucleotide polymorphisms have been associated with childhood-onset aggression [125-127]. An allele for low MAO-A activity has been found in people who displayed high levels of aggression, especially in combination with early life adversities [125, 127, 128], and MAO-A length polymorphisms have been suggested as a useful risk indicator [129, 130]. Serotonin transporter polymorphisms were also associated with aggression in some populations [131, 132]. Moreover, the dopamine receptors D2 and D4 gene variants have been shown to interact in the prediction of adolescent conduct disorder and antisocial behaviour [133, 134].

Other neurobiological systems implicated in research on the neural background of aggression include sex steroids, glutamate/GABA, peptides (such as oxytocin), and opioid receptors.

Problematic aspects of forensic psychiatric risk assessments

The central aspects of forensic psychiatric risk assessments that require particular consideration may be broadly defined into the following categories: (1) accuracy of assessments, (2) ethics, and (3) use and communication of assessments. These will be briefly presented here and further discussed throughout the thesis.

1. To be accurate, an instrument first has to be reliable (i.e. stable over time and across raters), consistent (i.e. measuring a unified construct), and valid for addressing the specific question. Data on validity has to be collected in comparable situations to where the instrument will be used, and gender and ethical groups must be considered specifically.
2. Ethical problems include the role of the rater (e.g. unproblematic for police officers but highly problematic for a doctor, a psychologist, or a social worker, as the methods used by these professionals are based on establishing confidence and trust), the dynamics of risk (amenable to change?), the types of risk factors (the use of factors such as heritability and genes is generally avoided even if they would add to assessments), and confidentiality.

3. Professionals have a responsibility for the consequences of communication with courts and in society. Using psychiatric and/or psychological methods may create the impression that these factors are especially linked to violence and criminal recidivism, and that mentally disordered offenders are more prone to recidivism and dangerousness than other offenders.

Aims of the study

General aims

This thesis aims at presenting the practice of forensic psychiatric risk assessments theoretically, scientifically, and clinically, and the overall purpose was to test the predictive ability of assessment methods for the risk of violent crimes in a follow-up study of a prospective Swedish forensic psychiatric cohort of perpetrators of severe crimes against others.

Specific aims

Paper I

The aims of this study was (i) to examine the influence of the age at onset of substance abuse on other factors of relevance for criminal behaviour, (ii) to investigate if the age at onset of alcohol and drug abuse can be used by itself to predict violent recidivism.

Paper II

Previous reports have described an association between the platelet MAO-B activity and factors of relevance for criminal behaviour such as childhood disruptiveness, adulthood aggression, psychopathic traits and recidivistic violence across adult diagnostic categories. This study was designed to test the accuracy of platelet MAO-B activity as a predictor of criminal recidivism and traits related to violence.

Paper III

In this paper, a broad array of both criminological and clinical risk factors as well as structured assessment instruments were tested for predictive ability, and the relapse rate in violent crimes (reconvictions) was compared between subjects sentenced to compulsory forensic psychiatric treatment and subjects sentenced to prison.

Paper IV

In the final paper, the development and focus of forensic psychiatric risk assessments were discussed from a clinical point of view using the example of Sweden, considering changes over time, ethical dilemmas with risk for integrity violations, and misunderstandings based on divergent expectations and inconsequent use of terminology. The paper includes an overview of the current knowledge of forensic psychiatric risk assessments in Sweden.

Subjects and Methods

The present work is a follow-up study of the initial study of the Gothenburg Forensic Neuropsychiatry Project, here referred to as the “baseline study”. The baseline data was collected between 1998 and 2001, and the collection of follow-up information from official registers was initiated in 2005. A brief description of the subjects and methods of the baseline study is given below.

Baseline study

Recruited for the “baseline study” (1998-2001) of The Gothenburg Forensic Neuropsychiatry Project were violent offenders, arsonists, and sexual offenders consecutively referred by court to a pre-trial forensic psychiatric investigation at the Department of Forensic Psychiatry in Gothenburg. Study subjects were included by criteria defining the type of crimes in the indictment. Violent crimes where the life of the victim had been threatened or taken, arson, and sexual offences (defined as rape or aggravated rape against adults, and sexual crimes against minors corresponding to today’s definitions of rape) were classified as severe crimes qualifying for inclusion in the study. The only exclusion criterion was lack of the basic Swedish education necessary for dealing with the assessment instruments and for availability of registered school and other background data. Out of 121 eligible subjects, 100 agreed to participate, while 21 declined (Table 2). Subjects could participate in some parts of the studies and decline others, and the non-participants registered for each method were separately analysed to control for attrition effects. Characteristics of the study population are given in Tables 2 and 3.

Table 2. Index crimes and legal outcomes in the study population (adapted from Söderström 2002 [135])

	Baseline study (n=100)	Non-consenters (n=21)
Men:Women	92:8	18:3
Age (years)	17-76 (median 30)	17-62 (median 35)
<i>Index crimes</i>		
Violent	61 (61 %)	15 (71 %)
Sexual	25 (25 %)	2 (10 %)
Arson	14 (14 %)	4 (19 %)
Murder/manslaughter	21 (21 %)	1 (5 %)
Attempted murder	17 (17 %)	6 (29 %)
Aggravated assault	17 (17 %)	7 (33 %)
Unlawful threat/robbery	6 (6 %)	1 (5 %)
Rape	3 (3 %)	1 (5 %)
Sexual child abuse	22 (22 %)	1 (5 %)
Arson	14 (14 %)	4 (19 %)
<i>Sentences</i>		
Prison	54 (54 %)	not known
Forensic psychiatric inpatient treatment	46 (46 %)	not known

Table 3. Distribution of psychiatric diagnoses in the study population (adapted from Söderström 2002 [135]). In non-consenters, only major psychiatric diagnoses from the forensic psychiatric investigation reports were available for comparisons.

	Baseline study (n=100)	Non-consenters (n=21)
<u>Axis I diagnoses</u>		
Mood disorders	45 (45 %)	1 (5 %)
Psychotic disorder	20 (20 %)	10 (48 %)
Substance abuse and dependence	56* (56 %)	12 (57 %)
Anxiety disorder	26 (26 %)	1 (5 %)
Autism spectrum disorder	18 (18 %)	not known
Mental retardation	17 (17 %)	not known
Tic disorder	18 (18 %)	not known
AD/HD	39 (39 %)	not known
Conduct disorder	48 (48 %)	not known
<u>Axis II diagnoses</u>		
<i>Cluster A</i>		
Paranoid PD	22 (22 %)	not known
Schizoid PD	22 (22 %)	not known
Schizotypal PD	13 (13 %)	not known
<i>Cluster B</i>		
Histrionic PD	7 (7 %)	not known
Narcissistic PD	16 (16 %)	not known
Borderline PD	31 (31 %)	not known
Antisocial PD	43 (43 %)	not known
<i>Cluster C</i>		
Phobic PD	17 (17 %)	not known
Obsessive/compulsive PD	15 (15 %)	not known
Dependent PD	2 (2 %)	not known
<i>Other PDs</i>		
Personality disorder NOS	5 (5 %)	not known

*Adapted from Söderström 2002, with the addition of 4 cases of substance abuse discovered in the re-analyses presented in Paper I.

The subjects were generally on remand for at least one month before incarceration for up to four weeks at the study department during the forensic psychiatric investigation, but four subjects were assessed as outpatients. All subjects were detoxified when first taken into custody, i.e. before inclusion in the study. The fact that some subjects had a pharmacological treatment that could not be discontinued for study purposes was accounted for in the relevant analyses.

All subjects were convicted of their index crimes, in some cases after a change in the legal definition of the act. For study purposes, the initial legal classification was used as long as the description of the criminal act remained unchanged.

Forensic psychiatric investigations

The majority of persons subjected to a forensic psychiatric investigation in Sweden have committed some type of violent crime [136, 137], possibly under the influence of a mental disorder. Criminal identification is generally not a predominant feature among these subjects, and they do not score especially high on assessment instruments such as the PCL-R [79].

The vast majority (95 %) of perpetrators who have undergone a forensic psychiatric investigation are assigned some type of psychiatric diagnosis, but only about half of all investigated cases prove to have a mental disorder severe enough to exclude a sentence to prison [137, 138]. More than half of those exempted from regular sanctions suffer psychoses, particularly of the type seen in the schizophrenia spectrum. In a smaller proportion of patients, the main diagnosis is severe compulsiveness or severe personality disorder with a tendency to develop psychotic reactions in response to stress and mental strain. Finally, a few cases involve serious neuropsychiatric and developmental conditions characterized by considerable neurocognitive functional impairments.

Data collection

All basic assessments included highly structured psychiatric, psychological, and social evaluations according to a research protocol covering mental health problems, personality aspects, drug abuse, and criminality. In the following, (i) psychiatric assessments; (ii) forensic personality assessment, and (iii) self-rated personality assessments will be detailed. Assessments were based on in-depth personal examinations during the investigation period but also on extensive file reviews, as a forensic psychiatric investigation overrules all secrecy in Swedish health care and makes all types of files and dossiers from birth through child health care and school health care into adulthood available for the investigation team and, in this case, the researchers involved in the study.

i. Psychiatric assessments

Structured clinical interviews were made by trained raters, and all diagnoses were based on the Diagnostic and Statistic Manual of Mental Disorders (DSM-IV); SCID-I [139] was used for Axis I disorders and SCID-II interviews [140] for Axis II diagnoses.

Childhood-onset neuropsychiatric disorders (i.e. autism spectrum disorder, AD/HD, tics, and mental retardation) were diagnosed by additional instruments, such as the Asperger's Syndrome Screening Questionnaire [141], the Asperger's Syndrome Diagnostic Interview [142], and the Wender-Utah Rating Scale [143]. Neuropsychiatric examinations and neurocognitive test assessment included the Wechsler Adult Intelligence Scale [144] evaluated according to the Swedish normative data [145]. A semi-structured collateral interview on childhood development, behaviour, and health was performed with a relative who had known the subject as a child in the 31 cases with such a person available.

Categorical diagnoses were established by the DSM-IV cut-off levels and by clinical consideration of the impact that personality traits had with regard to the psychosocial level of functioning as well as their relationships with the Axis I diagnoses. The numbers of fulfilled Axis I and II criteria were also used as dimensional ratings of dysfunctional/maladaptive personality traits.

ii. Forensic personality assessments

Criminological data was collected in accordance with a structured protocol including basic data on previous and current criminal behaviour. Risk-related assessments were made by the PCL-R [23, 123, 146] and by the HCR-20 [33] check list. Life History of Aggression (LHA) [147] scores (rated on the basis of self-reports, structured interviews, and file information) and previous violent criminality were collected. Furthermore, the protocol was extended to contain a large sociological data set assessed by the psychiatric social worker on the forensic psychiatric investigation team and subsequently checked against the files by a research social worker to ascertain scoring uniformity.

PCL-R assessments of psychopathy included both the original two-factor solution and the three-factor model (i.e: “arrogant and deceitful interpersonal stile” (factor 1); “deficient affective experience (factor 2), and “impulsive and irresponsible behaviour style” (factor 3)) proposed by Cooke and Michie [148]). Regarding HCR-20, only the first 15 items were included in the baseline assessment, as the last 5 items refer to conditions after release from sanctions.

iii. Self-rated personality assessments

In addition to the LHA self-assessment variant, the clinical work-up included the Karolinska Scale of Personality [149] and the Temperament and Character Inventory (TCI) [150] to collect additional information about personality traits, while the completion of the Beck Depression Inventory [151, 152] provided information about the current degree of depression.

Neurochemical analyses

Eighty-one subjects (77 men, 4 women, aged 17-76, median: 30 years among the 100 subjects of the baseline study) consented to venous blood sampling for determination of MAO-B in blood platelets. The female subjects were excluded from the final analyses since a previous publication showed a gender difference in MAO-B activity [153], which was thought to be increased in females [154, 155]. Since compounds in cigarette smoke have been found to inhibit MAO-B [114], we made separate analyses for the 43 smokers and 29 non-smokers (information on smoking was missing in 5 cases).

The control group consisted of 18 healthy blood donors (aged 25-55 years). For the analyses of cerebrospinal fluid (CSF), 36 subjects from the baseline study consented to lumbar puncture.

Platelets were isolated from EDTA-blood as previously described [156]. The protein content of the homogenate was determined by the bicinchoninic acid procedure [157]. The enzyme activity was assayed radiochemically with 5 μ M β -phenylethylamine as substrate [158]. The specific enzyme activity is expressed as μ kat per kilogram cell protein.

Lumbar punctures were performed under standard conditions. The CSF monoamine metabolites homovanillic acid (HVA), 5-HIAA, and MHPG were assessed by high-performance liquid chromatography with electrochemical detection [159]. Monoamine metabolite concentration was expressed as nanomoles per litre (nM/l).

Follow-up study: study population and data collection

The follow-up study, carried out during 2005 and 2006, followed all participants (who gave informed consent for this in connection with the baseline study), with the exception of one individual (n=99) due to problems with the social security number, from their index crime up to January 1, 2005, giving a follow-up time-frame of 44 to 73 months with a mean follow-up period of almost 60 months. The subjects were not investigated in person again during this follow-up study, but the 99 forensic investigations and index sentences were re-read in relation to the extracts from files included in the follow-up (as detailed below) and sentences for relapse crimes, and notes were taken regarding each individual subject's life history and criminal record. Specific attention was given to all information on substance abuse; age at onset, duration, types of drugs, and relation to criminal behaviours. All notes were entered into the database already established from the baseline study. Unless otherwise stated, all findings concerning clinical diagnoses are based on the baseline study [135].

Three national registers (the National Council for Crime Prevention, the National Board of Health and Welfare, and the National Prison and Probation Administration registers) were searched for the following outcomes: (i) incidents during treatment (forensic psychiatric/institutional/prison), (ii) type and number of recidivistic crimes: violent, sexual, and/or other. New inpatient treatment periods and diagnoses assigned were also compared to the baseline data.

In the original group of 100 subjects, 46 subjects were sentenced to forensic psychiatric inpatient treatment and 54 to prison. During the follow up period, a total of 20 of the 99 followed subjects relapsed in violent (n=16) or dangerous (n=4) criminal acts, including 1 murder, 1 arson, 1 rape, 1 case of exposing somebody to danger, 1 duress, 2 aggravated assault, 5 assaults, 3 aggravated unlawful thefts/robberies, and 5 violations of legislation against carrying arms. The number of crimes per subject varied as some subjects committed further recidivistic crimes after the first relapse crime treated as end-point for the follow-up analyses presented here. Three subjects committed suicide during the study period.

Independent variables

From the baseline data, the following independent variables were identified for the analyses in this thesis: (1) age at onset of substance abuse, (2) duration of substance abuse, (3) platelet MAO-B activity, (4) CSF monoamine metabolite concentrations (5-HIAA, HVA, MHPG), (5) childhood-onset behaviour disorders, (6) risk assessments according to HCR-20, (7) personality traits, especially those related to disruptive and aggressive behaviours, (8) LHA scores, (9) psychopathic traits according to the PCL-R, including factor scores, (10) DSM-IV diagnoses of psychosis, substance abuse/dependency, conduct disorder and antisocial personality disorder, (11) criminological risk factors, including age at first conviction, number of convictions for aggravated violence, number of prison convictions, substance abuse/dependency among primary relatives, criminality among primary relatives, unstable and insecure circumstances during childhood, sexual abuse during childhood/adolescence, and (12) sentences to prison or forensic psychiatric care.

Dependent variables

From the baseline study, PCL-R and LHA scores and a history of recidivistic violence already at index (i.e. baseline) were used as dependent variables in some analyses seeking to predict factors of relevance to violent criminal behaviour. The scores were used as dimensional scores or as categories by median splits or proposed cut-off scores reflecting

aggressive behaviour patterns and personality traits. The main outcome measure, however, was recidivism in violence (or dangerousness) during follow-up defined categorically as recidivism vs. non-recidivism based on new convictions during the observation period.

Data administration and statistical methods

All information collected was de-identified and organized in a coded file. The data were analysed by chi-square analyses, non-parametric Mann-Whitney comparisons, Spearman rank correlations, t-tests, ANOVAS, Receiver Operating Characteristics (ROC) Curves, logistic regression analyses, and Kaplan-Meier survival analyses. All statistics were calculated with different versions of the SPSS software (from SPSS version 12.0 in Paper I to PASW version 17.0 in Paper III) and all p-values are two-tailed. Statistical significance was set at the 5 % level, meaning that two-tailed p-values <0.05 are considered significant. Age at onset of substance abuse and a number of clinical assessments had skewed distribution, which motivated the use of non-parametric methods. Due to the exploratory nature of the studies, corrections for multiple comparisons have not generally been applied.

Ethical considerations

This thesis includes follow-up analyses based on a clinical research project known as the Gothenburg Forensic Neuropsychiatry Project [135]. Since the project involved individuals deprived of their liberty, particular emphasis was placed on the voluntary nature of participation. All potential subjects were given oral and written information about the study and opportunities to discuss questions concerning their participation in the study. The subjects had the choice of refraining from participating in any part of the investigation they wished, drop-outs were accepted at any time during the study period, and all subjects were assured that research findings would have no impact on the outcome of the forensic psychiatric investigation. The rate of participation was high though the participants earned no material compensation for their participation. All subjects were informed of the planned register-based follow-up and consented to this part of the study.

All information, both the baseline assessments and the register-based follow-up data, was de-identified and stored in digitalized databases. Thus, coded clinical and laboratory databases were used for the follow-up studies included in this thesis, and de-identified blood samples were stored together with coded clinical information for biochemical investigations.

The project was approved by the Research Ethics Committee at the University of Gothenburg with register number Ö 465-02, approval date 2002-10-21, while aims and methods of this specific thesis work were also tried and accepted by the Research Ethics Committee at the University of Lund with register number 698/2005, approval date 2006-02-16.

Detailed methods used for the follow-up study

Paper I

Dependent variables: LHA, PCL-R-scores, and violent recidivism at baseline, i.e. recidivism defined as having admitted at least one violent crime before the index crime leading to inclusion in the study (as follow-up data was not available at the time of writing this first Paper).

Independent variables: Age at onset of substance abuse, number of childhood hyperactivity symptoms, childhood conduct disorder, abuse of substances other than alcohol, personality disorders.

Retrospective assessments were carried out by scrutinizing the files and records of the subjects of the baseline study. Beside the DSM-IV diagnoses of abuse or dependence and other mental disorders, all data was registered about any substance (alcohol and/or drugs) abuse/dependence, number of years with abuse, and the age when the subject had started his/her alcohol/drug abuse. The number of years with both alcohol and drug abuse was reported by the subjects themselves during interviews in the baseline study, and the self-reported duration of alcohol and/or drug abuse or dependence was compared with information from medical files, records from social authorities, and other available registers (school and military records) to ascertain that the calculated age at onset of substance abuse was as reliable as possible. From the 56 subjects diagnosed with a DSM-IV substance-related disorder, two were excluded, as age at onset of substance abuse could not be reliably determined in those cases. The three-factor model of the PCL-R facets was used in the scoring. Self-rated personality assessments by the TCI were compared to norm data to yield t-scores.

Statistical analyses included chi-square tests, non-parametric Mann–Whitney comparisons, Spearman rank correlations, logistic regressions, and Receiver Operating Characteristics (ROC) curves by the SPSS 12.00 (further references omitted). All p-values are two-tailed. In detail: Spearman correlations were calculated between forensically relevant factors and the age at onset and the duration of substance abuse, respectively. In the next step, ROC curves were plotted using the Area under the Curve (AUC) as an indicator of predictive power across various cut-offs. This method is well suited for solving two kinds of problems in research on risk prediction: (1) the cut-off score that is most effective in distinguishing signal from noise, i.e. relapse from non-relapse, can be calculated from the curve's point of inflection [160], and, (2) the AUC can be used as a measure of the instrument's overall capacity to distinguish between signal and noise at all possible cut-off scores [161, 162]. This value may also be interpreted as the probability of an actual signal having a higher score than the noise in the continuous variable [160]. The AUC is useful in comparing the theoretical accuracy of different test methods at all possible cut-off levels, but provides no information about the accuracy in a single prediction, which requires a predetermined cut-off and knowledge of the prevalence of the studied phenomenon in the actual group of subjects.

The optimal cut off/inflection point was 18 years of age. Subjects with late versus early (before the age of 18) onset of alcohol and/or drug abuse/dependence were subsequently compared for behavioural and neuropsychiatric disorders in childhood, psychopathic traits, personality traits, aggression, global IQ, and criminality in adulthood.

In the final step, to compare the age at onset of substance abuse to other known childhood risk factors and to rule out possible confounding by a higher prevalence of mixed drug abuse in the early-onset group, a multivariate logistic regression analysis was performed with LHA and PCL-R scores over the median split and violent recidivism as dependent variables, and age at onset, number of childhood hyperactivity symptoms, childhood conduct disorder, and abuse of substances other than alcohol as independent variables.

Paper II

Dependent variables: Violent recidivism during the follow-up period, violent recidivism at baseline, personality factors (temperament and character dimensions), LHA and PCL-R scores, and the CSF monoamine metabolites concentrations (HVA as dopamine metabolite, 5-HIAA as serotonin metabolite, and MHPG as noradrenalin metabolite).

Independent variables: Platelet MAO-B activity.

Statistical analyses: Associations between variables were calculated as Spearman rank correlations (to avoid missing correlations due to non-normal distributions among other variables than the MAO-B activity, which itself is normally distributed). The predictive ability of MAO-B activity with regard to criminal recidivism was also calculated in the form of a ROC curve.

In order to investigate any significant difference in MAO-B activity between the different types of offenders, the 45 perpetrators of “classic” violent crimes (murder, manslaughter, aggravated assault, and violent robbery) were compared to the 22 perpetrators of rape, sexual child abuse or the 10 arsonists by an ANOVA across the three offender groups. For a comparison with normal controls, an ANOVA across the three offender groups and the normal contrast group was used. Finally, the confounding effect of smoking was analysed by comparison of smokers and non-smokers (t-test).

Paper III

Dependent variables: DSM-IV diagnoses of psychosis, substance abuse/dependency, conduct disorder, antisocial personality disorder, total scores on LHA, PCL-R, and HCR-20, age at first conviction, number of convictions for aggravated violence, number of prison convictions, substance abuse/dependency among primary relatives, criminality among primary relatives, unstable and insecure circumstances during childhood, and sexual abuse during childhood/adolescence.

Independent variables: Violent recidivism based on reconvictions for violent crimes during follow-up.

Length of follow-up period and basic clinical characteristics was compared between the subjects sentenced to forensic psychiatric care and those sentenced to prison. A Kaplan-Meier survival analysis was also performed, comparing time, in months, until violent relapse for the two groups as well as Spearman correlations between criminological and clinical risk factors and violent recidivism (reconvictions) during the follow-up period. ROC-analyses were performed to examine the predictive ability of the criminological and clinical risk factors (e.g. age at first conviction, number of convictions for aggravated violence, number of convictions, PCL-R total score, LHA total score, and HCR-20 total score on historical and clinical aspects) for criminal recidivism (reconviction). Finally, the predictive power of criminological versus clinical risk factors was analyzed by binary logistic regressions, and these regression models were also used to calculate sensitivity, specificity, and positive predictive value (PPV) as well as negative predictive value (NPV) for both sets of risk factors.

Results

Paper I

Median age at onset was 22 years for alcohol abuse, 14 years for non-alcohol drug abuse, and 16 years for mixed abuse. The age of onset of abuse strongly correlated with several forensic key features, such as aggression, violent recidivism, and all facets of psychopathy, while the number of years with abuse showed no correlations with either forensic assessments or psychiatric disorders in adult- or childhood.

The subjects with early onset of substance abuse differed from those with later onset in demonstrating personality profiles with significantly higher scores on the TCI dimensions Novelty Seeking and lower scores on Reward Dependence, Persistence, and Cooperativeness.

The ROC curve of the inverted age at onset of substance abuse (i.e. 100 – age at onset) predicting a LHA score over the median split had an AUC of 0.88, with an optimal inflection point at 18 years of age or younger, giving a sensitivity of 0.78 and a specificity of 0.92. A similar analysis for predicting PCL-R scores of 20 or more (used as an approximate cut-off for psychopathic traits) had an AUC of 0.88, with 18 years of age yielding a sensitivity of 0.80 and a specificity of 0.71. The ROC for the age at onset as a predictor of violent recidivism had an AUC of 0.76, with the same cut-off as above, i.e. 20 or more (used as an approximate cut-off) yielding a sensitivity of 0.74 and a specificity of 0.70.

Multivariate logistic regression analyses with LHA, PCL-R scores over median splits, and violent recidivism as dependent variables and age at onset, number of childhood hyperactivity symptoms, childhood conduct disorder, and abuse of substances other than alcohol as independent variables, showed that the age at onset was the most significant predictor of violent recidivism ($\beta = -0.079$, S.E. = 0.050, $\exp(\beta) = 0.924$, $p = 0.115$, where the $\exp(\beta)$ expresses an odds ratio below 1 as the risk decreases with increasing age at onset).

In summary, the strong associations of the age at onset of substance abuse with several key forensic features suggested that this is an important factor in the prediction of complex personality disorders and violent recidivism.

Paper II

The study group of violent offenders had a mean platelet MAO-B activity of 5.41 $\mu\text{kat/kg}$ protein ($SD \pm 1.64$), which was not significantly different from the mean platelet MAO-B activity in the control group (mean = 5.21 $\mu\text{kat/kg}$, $SD \pm 1.21$). There was no significant correlation between the MAO-B activity and age. When comparing different categories of offenders (sexual offenders, arsonists, or perpetrators of violent crime), no significant difference in MAO-B activity was found, nor when analyzing the platelet MAO-B activity in women. Furthermore, no correlation was found between the enzyme activity and the age at onset of substance abuse, the presence of poly-substance abuse, or antisocial personality disorder.

Smokers had a mean platelet MAO-B activity of 5.05 $\mu\text{kat/kg}$ protein ($SD \pm 1.38$), while the corresponding measure among non-smokers was 5.70 $\mu\text{kat/kg}$ protein ($SD \pm 1.93$). This

reduction of about 10 % in the enzyme activity among smokers was not statistically significant in this study ($p=0.13$).

Among non-smokers, the MAO-B activity was significantly correlated to the Gillberg and Gillberg criteria for Asperger's syndrome [163], and to the TCI's temperament dimensions Harm Avoidance and Persistence. These correlations, however, would not remain significant after any formal correction for multiple testing.

There were no significant correlations between the platelet MAO-B activity and any of the CSF monoamine metabolite concentrations (HVA, 5-HIAA, and MHPG).

In summary, relationships between the platelet MAO-B activity and behavioural deviations, such as psychopathic traits, aggression, or criminal recidivism, could not be demonstrated in our study population.

Paper III

During a mean follow-up period of 59 ($SD\pm 10.9$) months, 20 individuals (20 %) relapsed in violent criminality according to the defined criteria. Relapses occurred both during ongoing sanction ($n=6$) and after discharge or on parole ($n=14$). Reconvictions for violent criminality were significantly more common among the subjects sentenced to prison ($n=15$, 28 %) as compared to those sentenced to forensic psychiatric care ($n=5$, 11 %), indicating a general rate of violent relapse as low as four violent relapses per 100 patient years among mentally disordered offenders, even if the subjects sentenced to forensic psychiatric care spent significantly more time at liberty (risk) during the follow-up period than those in prison. Time until a first violent relapse (reconviction) was compared between the two sanction groups by a Kaplan-Meier survival analysis, showing not only that the relapses were fewer but also that they occurred earlier in the follow-up period among the subjects sentenced to forensic psychiatric treatment than among those with prison sanctions.

Spearman correlation factors between criminological/clinical risk factors and violent recidivism were overall small to modest.

Among the clinical/historical risk factors, a diagnosis of conduct disorder during childhood fell out as a significant risk factor when cross-tabulated with violent recidivism, and 80 % of those who actually relapsed fulfilled criteria for childhood conduct disorder as compared to 39 % of the non-recidivists. The only other significant risk factor was a diagnosis of substance abuse/dependence. In contrast, none of the dichotomous criminological risk factors showed a statistically significant relation with violent recidivism.

ROC curves were plotted for the three continuous criminological risk factors (age at first conviction, number of convictions for aggravated violence, and number of prison convictions) together with the scores from the assessment instruments (PCL-R total score, LHA total score, and HCR-20 total score) to illustrate their predictive power for violent criminal recidivism. These analyses showed modestly significant predictive abilities for the criminological risk factor age at first conviction and for all three assessment instruments, while two of the criminological risk factors (the number of previous convictions for aggravated violence or of prison convictions) did not predict violent recidivism significantly.

Logistic regression analyses showed that the predictive ability of criminological vs. the combined set of clinical risk factors and scores from the risk assessment instruments was

satisfactory, in both cases arriving at a correct classification of about 80 % of the individuals. Both models were, on the whole, correct in classifying the large group of true negatives, i.e. those who did not relapse, while the accuracy for those who actually relapsed was much lower, identifying 77 % (criminological risk factors) and 50 % (clinical risk factors) correctly.

Paper IV

The progress of risk assessments in Sweden has, on the whole, followed the international development. Categorical assessments of “dangerousness” were successively replaced by assessments of “risk” for violence, which are currently performed as clinical routine, both in the state-run clinical forensic psychiatric investigations and in the hospital system for clinical decisions and as decision-support for administrative and penal courts of law, by means of Swedish versions of instruments such as the HCR-20 and the PCL-R. By these methods, groups with a higher probability than others for violent acting out can in fact be identified, but it is more difficult to assess individuals to arrive at reasonably safe conclusions about the individual risk of relapsing in criminality. Male gender, youth, substance abuse, previous criminality, and low age at onset of antisocial behaviour have always turned up as risk factors in research on the probability of relapse in violence [49, 50].

In Sweden, the level of knowledge and experience could be summarized in the following statements: (1) clinical risk assessments provide results better than chance, (2) at best they correctly classify 70-75 % of cases, and (3) we still lack support for risk assessments of women and ethnic minorities [164] in forensic psychiatry and generally.

Discussion

Historical and contextual risk factors together with clinical assessments and measures have been shown to predict violent recidivism among mentally disordered offenders [37, 38, 40, 42, 43, 45, 46, 61] In this thesis, risk factors from all these different categories have been assessed, for example age at onset of substance abuse as a historical risk factor of clinical origin, MAO-B as a biological factor, and age at first conviction as an historical factor of criminological origin.

Previous psychiatric research on substance abuse and violent crimes has mainly assessed the covariation of the two [62] and the duration of such abuse. Cloninger's model of type II alcoholism characterized by early-onset poly-drug substance abuse and disruptiveness, with a specific personality profile of high Novelty Seeking and low Reward Dependence combined with character immaturity, and a distinct hereditary vulnerability [57], prompted us to specifically address the age at onset of substance abuse in relation to aggression, psychopathy, and violent recidivism (Paper I). The personality and behavioural correlates to the age at onset of substance abuse conformed to the proposed model. Subjects with early onset of substance abuse (i.e. before 18 years of age) had higher scores on behavioural aspects of psychopathy, more AD/HD, conduct disorder, previous aggressive behaviours, violent recidivism, personality disorders, and antisocial temperaments with character immaturity, especially in the form of low Cooperativeness. Even if aggressive impulses are inherent in the forces that help us survive, advance, and assert integrity, the ability to refrain from acting out on impulses and to control behaviour in accordance with superior goals is pivotal in human interaction. The impulse to react with aggression has to be recognized and acted on, or suppressed if destructive, in fractions of a second. Considerations about propriety of behaviour, purpose, and consequences must be processed instantly since there might be little time for deeper reflection. Substance abuse might both increase aggressive impulses and have additional negative effects by impeding responsible, independent judgment and by reducing the ability of impulse control. Age at onset of substance abuse may serve as a marker of risk for violent recidivism and thus also signal substance-related complications that require preventive social, educational, and medical measures. The association between an early age at onset of substance abuse and complications may be genetic but is also understandable from an etiological and pathological view as substance use early in life may hamper personality development and social adjustment more than similar abuse later in life.

In contrast, a similar assessment of platelet MAO-B activity as a predictor of recidivistic violence was negative (Paper II). MAO-B has since long been under investigation as a possible biological marker for various personality factors. It is of special interest due to its accessibility in whole blood, and because it is a more easily obtained sample than CSF. However, we did not find any support for MAO-B being of any reasonable importance for disruptive and antisocial personality traits and violent offending in our study group of violent offenders referred for forensic psychiatric investigations. Nor did the platelet MAO-B activity correlate with any childhood behavioural disorder, abuse-related factor, or psychosocial adversity. There were no associations to crime-related factors such as life history of aggression (LHA), psychopathic personality traits (PCL-R), or recidivistic violent crimes. The analyses in subgroups of smokers/non-smokers did not change the overall negative result, even if there was a correlation to temperament traits among non-smokers. The lack of significant findings could be ascribed to the low power of the present sample size, but the complete lack of associations to relapse in a prospective study design strongly indicates that

MAO-B cannot be used as a marker for violent recidivism with any reasonable validity in a selected offender population, even if correlations have previously been reported in general population samples and group comparisons [114]. Our results illustrate the dangers of extrapolating findings from large-scale cross-sectional studies to predictive models applied among clinical cases. MAO-B may, however, be of interest in research on constitutional factors making individuals vulnerable for substance abuse, especially type II alcoholism [114].

The long-term prospective follow-up study showed that historical risk factors, neither in the form of criminological or clinical data, were strongly related to recidivism in violent crime (Paper III). It also appeared that simple variables, describing, for instance, the age at first conviction, had similar predictive ability as complex clinical risk assessments with instruments such as the HCR-20 and the PCL-R (as judged by ROC analyses yielding similar AUCs). Neither did multivariate models, including all criminological or all clinical variables, lead to any considerable improvement of the prediction of violent recidivism. Each of these two models could correctly predict around 80 % of the subjects, which counts as a modest-good predictive power, but included a large number of true negative predictions, which are self-evident in a group with a low base-rate of re-offending and a relatively large proportion of false positive cases in relation to the true positive proportion, consistent with the evidence from the Backstrom and Dixon era, when a large group of subjects considered to be “dangerous” did not recidivate. The criminological risk factors turned out to be slightly better than the current clinical factors as they could correctly identify three out of four compared to two out of four recidivists, which is in line with the conclusion of the SBU report [164]. It was only in the analysis of the criminological model that we received some significant predictors, namely: (i) age at first conviction, where the risk of relapse increased with lower age and (ii) drug abuse/dependency among primary relatives.

Considering our results, it seems reasonable to suggest that clinicians in their work adopt a humble attitude with regard to what can be said on the basis of risk assessments and clearly state the degree of accuracy achievable by these methods to recipients of “expert opinions” on future re-offending.

“Risk” in clinical assessments

The use of “risk” as a concept in clinical work is doubtful not only from ethical but also from theoretical points of view. Risk factors are seldom causative, but in most cases they are mere markers of underlying traits or propensities that might play a more direct role in the genesis of a behaviour. Using non-causal risk factors also means that we may end up with an array of risk factors that are difficult to handle for ethical reasons. For example, race may easily be demonstrated to be a risk factor for almost anything negative in settings where race is associated with poor socio-economic situations and poverty. As long as risk factors have not been shown to be causal, the concept of risk management seems unfounded and confusing. Here, “risk” is no longer used to designate a statistical probability but rather means “threat” to security. If this is the goal, mental health problems (other than substance abuse and disorders that directly reflect criminal behaviours) have to be put in the larger context of criminality, where they explain but a very small proportion of violence in society.

As we have seen, twin and adoption studies have demonstrated that the variation in criminal propensity is partly due to genetic factors [165-168]. Fortunately, this propensity seems to be extremely polygenetic [166] as found for all other complex psychiatric and societal

conditions, and we do not have to think about the consequences that might have befallen us had some single gene variants with a strong influence on the risk for criminality been demonstrated. But the day when some biological genetic factors have been significantly associated with criminal behaviour might not be that far away, and information about familial aggregation of criminality may most likely add to the statistical prediction of crimes if included in rating schemes. How should we treat these and other biological risk factors? Should we present them to courts or social authorities just as scores from various instruments even if we know that they may lead to undesired effects for our patients, such as prolonged detention?

Another important issue in relation to risk assessments is the question of who should do the assessing. It is obvious that the “best” predictors for violence and recurrence of violence are all reflections of environment [74], behavioural patterns (irresponsible life-style), impulsivity [148, 169, 170], previous convictions [171, 172], basic statistical factors (that also reflect cultural life settings, such as being young and male) [44, 46, 49], and various forms of substance abuse [62, 173], which may play a causal role by reflecting the increased risk for a violent acting-out under the influence of a control-disinhibiting and aggression-enhancing drug such as alcohol [38]. Information on all these factors can be found in registers on previous crimes and other background data and has little to do with psychiatry or psychology. Why then should doctors and psychologists perform assessments that can equally well be performed by the courts or by officials who do not have the same ethical requirements for confidentiality and are not committed to work for the patient’s best interest?

Strengths and limitations

When risk factors of criminal behaviour or of recidivism in criminal behaviours are under investigation, prospective studies are desirable, with large populations that are followed longitudinally and continuously described with the help of validated instruments. The present studies are based on analyses of such a prospective study design though limited by its small size. The study population was also described by careful and quality-controlled clinical assessments performed during a four-week period at a specialized central department for forensic psychiatric investigations by a professional, interdisciplinary, scientific team, including a psychiatrist (specialist in general and forensic psychiatry), a clinical psychologist, a psychiatric social worker, and ward staff, giving the present study a solid clinical fundament. Specialized assessments by instruments as reported here were also performed by a small group of clinical researchers who has received specialized training in the use of state-of-the-art instruments such as the SCID, HCR-20, and the PCL-R.

The three empirical papers in the thesis based their results on the same study population, a distinct subpopulation of perpetrators of severe violent and sexual crimes as they had been court-referred for pre-trial forensic psychiatric investigations. The baseline project included consecutive cases selected by the courts for forensic psychiatric investigation, which facilitates comprehensive and systematic clinical work-ups but reduces the possibility of generalizing the results.

The study population was limited to 100 persons as extensive clinical investigations were carried out in each case. The number of individuals for each collected variable differed, because the study subjects were free to choose participation in some or all assessments, and practical factors sometimes reduced the number included by a specific method. Since violent crimes are rare phenomena in the general population, it is very difficult to collect prospective population-based cohorts with detailed clinical features to study relationships between, for

example, psychiatric disorders, addiction, biological markers, and violent crime. The mental health services in the prison system have not provided the preconditions required for performing a study such as this in previous decades.

Statistical analyses were performed within the subgroups of the study and in comparisons with healthy control groups collected for each laboratory method. Neither approach is scientifically optimal. To recruit truly matched and blindly assessed controls from the general population would, however, be impossible. With this in mind, comparisons between subgroups of study subjects may be advantageous although the impact of the systematic differences between, for example, violent and sexual offenders may still confound the analyses and should be considered in the interpretation of the results.

Clinical implications

Routine questioning about the exact nature of alcohol and other substance use, including developmental information regarding the onset of abuse and familial aggregation, as well as the individual pattern of reactions, interactions, and behaviours while under drug influence, may be useful in explaining factors behind violent events, antisocial behaviour, and psychosocial maladaptation, and may contribute to improved risk assessments, treatment programs, and preventive social, personal, motivational, and medical measures.

The initial enthusiasm about platelet MAO-B as a prognostic test in forensic psychiatry (“a simple blood test predicts dangerousness”) based on what was and remains scant evidence for predictive validity may reflect the demand for new methods and easy solutions in this area. However, the optimal predictive model in forensic psychiatry may be even more simple: just an overall mapping of the age at onset, persistence, and severity of previous substance (ab)use and (violent) crimes together with general facts such as age and sex.

References

1. Fitzpatrick, M., Jones, P., Knellwolf, C., McCalman, I., ed. *The Enlightenment world* 2004, Routledge, London.
2. Gray, L., Burroughs, W., ed. *Constitutional Issues: Separation of Powers. Social Education*. Vol. 5. 1987. 28-30.
3. Gabb, S., ed. *Voltaire: Crusader for justice*. 1990, Libertarian Alliance: UK.
4. Torrey, N., *Voltaire and the Enlightenment: Selections from Voltaire*. 2007, USA: Kessinger Publishing.
5. Monahan, J. *The prediction of violent behavior: toward a second generation of theory and policy*. *Am J Psychiatry*, 1984. 141(1): p. 10-5.
6. Otto, R. *Prediction of dangerous behaviour: A review and analysis of "second-generation" research*. *Forensic Rep*, 1992. 5: p. 103-133.
7. Murphy, J. ed. *Punishment and rehabilitation*. Basic problems in philosophy series. 1978, Wadsworth Pub Co: Belmont, California.
8. Rock, P. ed. *Sociological theories of crime*. The oxford handbook of criminology ed. M. Maguire, Morgan, R., Reiner, R. 2002, Oxford University Press.
9. Faust, D. and J. Ziskin. *The expert witness in psychology and psychiatry*. *Science*, 1988. 241(4861): p. 31-5.
10. Monahan, J., Steadman, H. (Eds), ed. *Violence and mental disorder: Developments in risk assessment*. 1994, University of Chicago Press. : Chicago.
11. Steadman, H.J., Coccozza, J. J. , ed. *Careers of the criminally insane: Excessive social control of deviance*. 1974, Lexington Books: Lexington.
12. Thornberry, T.P., Jacoby, J. E. ed. *The criminally insane. A follow-up of mentally ill offenders*. 1979, University of Chicago Press: Chicago.
13. Ennis, B., Litwack, TR., *Psychiatry and the presumption of expertise: Flipping coins in the courtroom*. *California Law Review*, 1974. 62: p. 693-752.
14. Rice, M., Harris, GT. *Violent recidivism: assessing predictive validity*. *Journal of Consulting and Clinical Psychology* 1995. 63: p. 737-748.
15. Medicine, N.B.o.F. ed. *Riskbedömning vid rättspsykiatrisk undersökning - Riktlinjer och reflexioner. (Risk assessment during forensic psychiatric investigations - General outlines and reflections)* RMV report 2000:1. 2000, National Board of Forensic Medicine: Stockholm.
16. Gunn, J. *Let's get serious about dangerousness*. *Criminal Behaviour and Mental Health*, 1996. 6 (Suppl. 1), : p. 51-64.
17. Heilbrun, K. *Prediction versus management models relevant to risk assessment: The importance of legal decision-making context*. *Law Hum Behav* 1997. 21: p. 347-359.
18. Hart, S.D. *The role of psychopathy in assessing risk for violence. Conceptual and methodological issues*. . *Legal and Criminological Psychology*, 1998. 3: p. 123-140.
19. Kinberg, O. *Kriminologiska grundproblemet*. 1955.
20. Coccozza, J., Steadman, H. *The failure of psychiatric predictions of dangerousness: Clear and convincing evidence*. *Rutgers Law Review*, 1976. 29: p. 1084-1101.
21. Beck, U., ed. *Risk society: Towards a new modernity, theory, culture & society*. 2002, Sage Publications.: London.
22. Hare, R.D. *A research scale for the assessment of psychopathy in criminal populations*. *Journal of Personality and Individual Differences*, 1980. 1: p. 111-119.
23. Hare, R.D. ed. *Manual for the Hare psychopathy checklist-revised*. 1991, Multi-Health Systems: Toronto.

24. Harris, G.T., M.E. Rice, and V.L. Quinsey. *Violent Recidivism of Mentally Disordered Offenders - the Development of a Statistical Prediction Instrument*. Criminal Justice and Behavior, 1993. 20(4): p. 315-335.
25. Quinsey, V.L., Harris, G. T., Rice, M. E., & Cormier, C. ed. *Violent offenders: Appraising and managing risk*. 1998, American Psychological Association.: Washington D.C.
26. Belfrage, H., Fransson, G., Söderberg, E., Vasko, T. *Rättspsykiatriprojekt i Växjö och Sundsvall: Nytt skattningsinstrument ger samstämmiga riskbedömningar* 1998, Läkartidningen, Sweden. p. 2469–2473.
27. Grann, M., Långström, N., Tengström, A., Stålenheim, E. G. *Reliability of filebased retrospective ratings of psychopathy with the PCL-R*. Journal of Personality Assessment 1998. 70: p. 416–426.
28. Grann, M., Belfrage, H., Tengström, A. *Actuarial assessment of risk of violence: predictive validity of the VRAG and the historical part of the HCR-20*. In: M. Grann (thesis), *Personality disorder and violent criminality in Karolinska Institute*. 1998: Stockholm.
29. Grann, M., Långström, N., Tengström, A., Kullgren, G. *Psychopathy (PCL-R) predicts violent recidivism among criminal offenders with personality disorders in Sweden*. . Law and Human Behavior, 1999. 23: p. 205–217.
30. Grann, M., H. Belfrage, and A. Tengstrom. *Actuarial assessment of risk for violence - Predictive validity of the VRAG and the historical part of the HCR-20*. Criminal Justice and Behavior, 2000. 27(1): p. 97-114.
31. Belfrage, H., et al. *Rättspsykiatriprojekt i Växjö och Sundsvall: Nytt skattningsinstrument ger samstämmiga riskbedömningar*. 1998, Läkartidningen Sweden. p. 2469–2473.
32. Webster, C.D., Eaves, D. *The HCR-20 scheme. The assessment of dangerousness and risk British Columbia*. Simon Fraser University and Forensic Psychiatric Services Commission of British Columbia, 1995.
33. Webster, C.D., Douglas, K.S., Eaves, D., Hart, S.D. *HCR-20. Assessing the risk of violence, version 2 Vancouver*. Simon Fraser University, 1997.
34. National Board of Health and Welfare, *Rättspsykiatrisk vård. Utvärdering-omvärdering. (Forensic psychiatric care. Evaluation-revaluation)*. 2002, National Board of Health and Welfare: Stockholm.
35. National Board of Forensic Medicine, ed. *Riskbedömning vid rättspsykiatrisk undersökning - Riktlinjer och reflexioner. (Risk assessment during forensic psychiatric investigations - General outlines and reflections)* RMV report 2000:1;2000, National Board of Forensic Medicine Stockholm.
36. Hanson, R.K. and M.T. Bussiere. *Predicting relapse: a meta-analysis of sexual offender recidivism studies*. J Consult Clin Psychol, 1998. 66(2): p. 348-62.
37. Farrington, D.P. and R. Loeber. *Epidemiology of juvenile violence*. Child Adolesc Psychiatr Clin N Am, 2000. 9(4): p. 733-48.
38. Pillmann, F., Ullrich, S., Sannemuller, U., Marnerso, A. *Acute effects of alcohol and chronic alcoholism as causes of violent crime*. Nervenarzt, 2000. 71(9): p. 715-721.
39. Straus, M.A. and I.L. Ramirez. *Criminal history and assault of dating partners: the role of type of prior crime, age of onset, and gender*. Violence Vict, 2004. 19(4): p. 413-34.
40. Grann, M., J. Danesh, and S. Fazel. *The association between psychiatric diagnosis and violent re-offending in adult offenders in the community*. BMC Psychiatry, 2008. 8: p. 92.

41. Warren, L.J., et al. *Threats to kill: a follow-up study*. Psychol Med, 2008. 38(4): p. 599-605.
42. Farrington, D.P., M.M. Ttofi, and J.W. Coid. *Development of adolescence-limited, late-onset, and persistent offenders from age 8 to age 48*. Aggress Behav, 2009. 35(2): p. 150-63.
43. Kjelsberg, E. and C. Friestad. *Exploring gender issues in the development from conduct disorder in adolescence to criminal behaviour in adulthood*. Int J Law Psychiatry, 2009. 32(1): p. 18-22.
44. Richard-Devantoy, S., et al. [*Homicide and major mental disorder: what are the social, clinical, and forensic differences between murderers with a major mental disorder and murderers without any mental disorder?*]. Encephale, 2009. 35(4): p. 304-14.
45. Mulder, E., et al. *Risk Factors for Overall Recidivism and Severity of Recidivism in Serious Juvenile Offenders*. Int J Offender Ther Comp Criminol, 2010.
46. Wang, R.H., et al., *Risk behaviours among early adolescents: risk and protective factors*. J Adv Nurs, 2010. 66(2): p. 313-323.
47. Fazel, S., et al. *Schizophrenia, substance abuse, and violent crime*. JAMA, 2009. 301(19): p. 2016-23.
48. Loeber, R. and D.P. Farrington. *Young children who commit crime: epidemiology, developmental origins, risk factors, early interventions, and policy implications*. Dev Psychopathol, 2000. 12(4): p. 737-62.
49. Hastings, J.E., Hamberger, L. K. *Sociodemographic predictors of violence*. Psychiatr Clin North Am, 1997. 20(2): p. 323-35.
50. Eriksson, Å. *Schizophrenia and criminal offending: Risk factors and the role of treatment in Department of Clinical Neurosciences, Karolinska Institutet 2008*: Stockholm.
51. Laub, J., Sampson, R.J. ed. *Understanding Desistance from Crime. Crime and Justice*. Vol. 28. 2001, The University of Chicago Press: Chicago. 1-69.
52. Sampson, R., Laub, JH. (2005) *A Life-Course View of the Development of Crime* The ANNALS of the American Academy of Political and Social Science 602, 12-45.
53. Lansford, J.E., Dodge, K. A., Pettit, G. S., Bates, J. E., Crozier, J., Kaplow, J. *A 12-year prospective study of the long-term effects of early child physical maltreatment on psychological, behavioral, and academic problems in adolescence*. Arch Pediatr Adolesc Med, 2002. 156(8): p. 824-30.
54. Goldstein, A. ed. *The Psychology of Group Aggression*. 2010.
55. Beaver, K.M., DeLisi, M., Vaughn, M. G., Barnes, J. C. *Monoamine oxidase A genotype is associated with gang membership and weapon use*. Compr Psychiatry, 2010. 51(2): p. 130-4.
56. Danielsson, A., et al. [*More violent assaults reported to the police and increasing gang violence. A survey of violence-related personal injuries in Umeå*]. Lakartidningen, 2005. 102(12-13): p. 945-8.
57. Cloninger, C.R., M. Bohman, and S. Sigvardsson. *Inheritance of alcohol abuse. Cross-fostering analysis of adopted men*. Arch Gen Psychiatry, 1981. 38(8): p. 861-8.
58. Hiscoke, U.L., et al. *Self-reported personality traits and disorders (DSM-IV) and risk of criminal recidivism: a prospective study*. J Pers Disord, 2003. 17(4): p. 293-305.
59. Latalova, K., *Bipolar disorder and aggression*. Int J Clin Pract, 2009. 63(6): p. 889-99.
60. Latalova, K., Prasko, J. *Aggression in Borderline Personality Disorder*. Psychiatr 2010.

61. Beck, J.C. *Delusions, substance abuse, and serious violence*. J Am Acad Psychiatry Law, 2004. 32(2): p. 169-72.
62. Grann, M. and S. Fazel. *Substance misuse and violent crime: Swedish population study*. BMJ, 2004. 328(7450): p. 1233-4.
63. Kenchadze, V.G., E.D. Chkoniia, and G.G. Sikharulidze. *[Increased risk of suicide in patients with agitated depression]*. Georgian Med News, 2009(177): p. 47-51.
64. Hardy, P. *[Severe depression : morbidity-mortality and suicide]*. Encephale, 2009. 35 Suppl 7: p. S269-71.
65. Nock, M.K., et al. *Measuring the suicidal mind: implicit cognition predicts suicidal behavior*. Psychol Sci, 2010. 21(4): p. 511-7.
66. Knox, M., et al. *Aggressive behavior in clinically depressed adolescents*. J Am Acad Child Adolesc Psychiatry, 2000. 39(5): p. 611-8.
67. Hodgins, S. *Epidemiological investigations of the associations between major mental disorders and crime: methodological limitations and validity of the conclusions*. Soc Psychiatry Psychiatr Epidemiol, 1998. 33 Suppl 1: p. S29-37.
68. Lindqvist, P., Allebeck, P. *Schizophrenia and crime. A longitudinal follow-up of 644 schizophrenics in Stockholm*. . British Journal of Psychiatry 1990. 157: p. 345-350
69. Hodgins, S. *Mental disorder, intellectual deficiency, and crime. Evidence from a birth cohort*. . Archives of General Psychiatry 1992. 49: p. 476-483.
70. Hodgins, S., et al. *Mental disorder and crime. Evidence from a Danish birth cohort*. Arch Gen Psychiatry, 1996. 53(6): p. 489-96.
71. Hodgins, S. *Mental disorder, intellectual deficiency, and crime. Evidence from a birth cohort*. Arch Gen Psychiatry, 1992. 49(6): p. 476-83.
72. Alden, A., et al. *Psychotic disorders and sex offending in a Danish birth cohort*. Arch Gen Psychiatry, 2007. 64(11): p. 1251-8.
73. Lindqvist, P. and P. Allebeck. *Schizophrenia and crime. A longitudinal follow-up of 644 schizophrenics in Stockholm*. Br J Psychiatry, 1990. 157: p. 345-50.
74. Frisell, T., P. Lichtenstein, and N. Langstrom. *Violent crime runs in families: a total population study of 12.5 million individuals*. Psychol Med, 2010: p. 1-9.
75. Elbogen, E.B. and S.C. Johnson. *The intricate link between violence and mental disorder: results from the National Epidemiologic Survey on Alcohol and Related Conditions*. Arch Gen Psychiatry, 2009. 66(2): p. 152-61.
76. Rice, M. *Violent offender research and implications for the criminal justice system*. Am Psychol, 1997. 52(4): p. 414-23.
77. Rasmussen, K. and S. Levander. *[Schizophrenia and violence]*. Tidsskr Nor Laegeforen, 2002. 122(23): p. 2303-5.
78. Nordstrom, A. *Violent offenders with schizophrenia. Quantitative and qualitative studies focusing on the family of origin*, in Department of Clinical Sciences. 2005, Umeå.
79. Soderstrom, H., et al. *Adult psychopathic personality with childhood-onset hyperactivity and conduct disorder: a central problem constellation in forensic psychiatry*. Psychiatry Res, 2004. 121(3): p. 271-80.
80. Kim-Cohen, J., et al. *Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort*. Arch Gen Psychiatry, 2003. 60(7): p. 709-17.
81. Hofvander, B., et al. *Life History of Aggression scores are predicted by childhood hyperactivity, conduct disorder, adult substance abuse, and low cooperativeness in adult psychiatric patients*. Psychiatry Res, 2010.

82. Flory, K. and D.R. Lynam. *The relation between attention deficit hyperactivity disorder and substance abuse: what role does conduct disorder play?* Clin Child Fam Psychol Rev, 2003. 6(1): p. 1-16.
83. Flory, K., et al. *Relation between childhood disruptive behavior disorders and substance use and dependence symptoms in young adulthood: individuals with symptoms of attention-deficit/hyperactivity disorder and conduct disorder are uniquely at risk.* Psychol Addict Behav, 2003. 17(2): p. 151-8.
84. Ghanizadeh, A. and P. Jafari. *Risk factors of abuse of parents by their ADHD children.* Eur Child Adolesc Psychiatry, 2009. 19(1): p. 75-81.
85. Ruchkin, V., et al. *ADHD symptoms and associated psychopathology in a community sample of adolescents from the European north of Russia.* J Atten Disord, 2008. 12(1): p. 54-63.
86. Walters, G.D., Raymond, A.K., Grann, M., Dahle, K.-P. *Incremental validity of the psychopathy Checklist Facet Scores: Predicting release outcome in six samples.* Journal of Abnormal Psychology., 2008. 117: p. 396-405.
87. Douglas, K. ed. *The HCR-20 risk assessment scheme. Overview and annotated bibliography.* Mental Health, Law and Policy Institute. 1998, Simon Fraser University: Burnaby.
88. Waldheter, E., Jones, NT., Johnson, ER., Penn, DL. *Utility of social cognition and insight in the prediction of inpatient violence among individuals with a severe mental illness.* J Nerv ment Dis, 2005. 193(9): p. 609-618.
89. Raine, A. *Features of borderline personality and violence.* J Clin Psychol, 1993. 49(2): p. 277-81.
90. Slutske, W.S., et al. *Modeling genetic and environmental influences in the etiology of conduct disorder: a study of 2,682 adult twin pairs.* J Abnorm Psychol, 1997. 106(2): p. 266-79.
91. Eley, T.C., P. Lichtenstein, and J. Stevenson. *Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: results from two twin studies.* Child Dev, 1999. 70(1): p. 155-68.
92. Burt, S.A. *Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis.* Clin Psychol Rev, 2009. 29(2): p. 163-78.
93. Cloninger, C.R., et al. *Predisposition to petty criminality in Swedish adoptees. II. Cross-fostering analysis of gene-environment interaction.* Arch Gen Psychiatry, 1982. 39(11): p. 1242-7.
94. Crampton, P. and C. Parkin. *Warrior genes and risk-taking science.* N Z Med J, 2007. 120(1250): p. U2439.
95. Alsobrook, J.P., 2nd and D.L. Pauls. *Genetics and violence.* Child Adolesc Psychiatr Clin N Am, 2000. 9(4): p. 765-76.
96. Levitt, M. and N. Manson. *My genes made me do it? The implications of behavioural genetics for responsibility and blame.* Health Care Anal, 2007. 15(1): p. 33-40.
97. Kendler, K.S. *"A gene for...": the nature of gene action in psychiatric disorders.* Am J Psychiatry, 2005. 162(7): p. 1243-52.
98. Coccaro, E.F. and R.J. Kavoussi. *Fluoxetine and impulsive aggressive behavior in personality-disordered subjects.* Arch Gen Psychiatry, 1997. 54(12): p. 1081-8.
99. Soderstrom, H., Blennow, K., Sjodin, A-K., Forsman, A. *New evidence for an association between the CSF HVA/5-HIAA ratio and psychopathic traits.* J Neurol Neurosurg Psychiatry, 2003. 74: p. 918-921.
100. Linnoila, M., et al. *Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior.* Life Sci, 1983. 33(26): p. 2609-14.

101. Lidberg, L., M. Asberg, and U.B. Sundqvist-Stensman. *5-Hydroxyindoleacetic acid levels in attempted suicides who have killed their children*. Lancet, 1984. 2(8408): p. 928.
102. Lidberg, L., et al. *Homicide, suicide and CSF 5-HIAA*. Acta Psychiatr Scand, 1985. 71(3): p. 230-6.
103. Asberg, M., L. Traskman, and P. Thoren. *5-HIAA in the cerebrospinal fluid. A biochemical suicide predictor?* Arch Gen Psychiatry, 1976. 33(10): p. 1193-7.
104. Siever, L. and R.L. Trestman. *The serotonin system and aggressive personality disorder*. Int Clin Psychopharmacol, 1993. 8 Suppl 2: p. 33-9.
105. Coccaro, E.F., et al. *Central serotonin activity and aggression: inverse relationship with prolactin response to d-fenfluramine, but not CSF 5-HIAA concentration, in human subjects*. Am J Psychiatry, 1997. 154(10): p. 1430-5.
106. New, A.S., et al. *Serotonergic function and self-injurious behavior in personality disorder patients*. Psychiatry Res, 1997. 69(1): p. 17-26.
107. Siever, L.J., et al. *d,l-fenfluramine response in impulsive personality disorder assessed with [18F]fluorodeoxyglucose positron emission tomography*. Neuropsychopharmacology, 1999. 20(5): p. 413-23.
108. Ryding, E., M. Lindstrom, and L. Traskman-Bendz. *The role of dopamine and serotonin in suicidal behaviour and aggression*. Prog Brain Res, 2008. 172: p. 307-15.
109. Dougherty, D.D., et al. *Decreased striatal D1 binding as measured using PET and [11C]SCH 23,390 in patients with major depression with anger attacks*. Depress Anxiety, 2006. 23(3): p. 175-7.
110. Virkkunen, M., et al. *A prospective follow-up study of alcoholic violent offenders and fire setters*. Arch Gen Psychiatry, 1996. 53(6): p. 523-9.
111. Ruchkin, V.V., et al. *Platelet MAO-B, personality, and psychopathology*. J Abnorm Psychol, 2005. 114(3): p. 477-82.
112. Chen, K., H.F. Wu, and J.C. Shih. *The deduced amino acid sequences of human platelet and frontal cortex monoamine oxidase B are identical*. J Neurochem, 1993. 61(1): p. 187-90.
113. Zuckerman, M. and D.M. Kuhlman. *Personality and risk-taking: common biosocial factors*. J Pers, 2000. 68(6): p. 999-1029.
114. Orelund, L. *Platelet monoamine oxidase, personality and alcoholism: the rise, fall and resurrection*. Neurotoxicology, 2004. 25(1-2): p. 79-89.
115. Revely, M., Revely, AM., Clifford, CA., Murray, AM. *Genetics of Platelet MAO activity in discordant schizophrenic and normal twins*. Br J Psychiatry, 1983. 142: p. 560-565.
116. Sullivan, J.L., et al. *Platelet MAO in subtypes of alcoholism*. Biol Psychiatry, 1990. 27(8): p. 911-22.
117. von Knorring L, O.L. *Platelet MAO Activity in type 1/type 2 alcoholics*. Alcoholism: Clinical and Experimental Research 1996. 20 (suppl 8): p. 224A-230A.
118. Esel, E., Kose, K., Turan, MT., Basturk, M., Sofuoglu, S., Aslan, SS., Yabanoglu, I., Gonul, AS., Yazici, C. *Monoamine oxidase-B activity in alcohol withdrawal of smokers: is there any relationship with aggressiveness?* Alcohol Alcohol 2002. 37: p. 272-276.
119. von Knorring A-L, B.M., von Knorring L, Orelund L. *Platelet MAO activity as a biological marker in subgroups of alcoholism*. Acta Psychiatr Scand 1985. 72: p. 51-58.
120. Schalling, D., et al. *Markers for vulnerability to psychopathology: temperament traits associated with platelet MAO activity*. Acta Psychiatr Scand, 1987. 76(2): p. 172-82.

121. af Klinteberg, B., Orelund, L., Hallman, J., Wirsén, A., Levander, SE., Schalling, D. *Exploring the connections between platelet monoamine oxidase activity and behaviour: relationships with performance in neuropsychological tasks.* Neuropsychobiology 1990-91. 23: p. 188-196.
122. Alm, P., af Klinteberg, B., Humble, K., Leppert, J., Sorensen, S., Thorell, L-H., Lidberg, L., Orelund, L. *Psychopathy, platelet MAO activity and criminality among former juvenile delinquents.* Acta Psychiatr Scand 1996. 94: p. 105-111.
123. Hare, R. *A research scale for the assessment of psychopathy in criminal populations.* Pers Individ Differ 1980. 1: p. 111-119.
124. Stalenheim, E., von Knorring, L., Orelund, L. *Platelet monoamine oxidase activity as a biological marker in a Swedish forensic psychiatric population.* Psychiatry Res 1997. 69: p. 79-87.
125. Caspi, A., et al. *Role of genotype in the cycle of violence in maltreated children.* Science, 2002. 297(5582): p. 851-4.
126. Mik, H.M., et al. *Serotonin system genes and childhood-onset aggression.* Psychiatr Genet, 2007. 17(1): p. 11.
127. Reif, A., et al. *Nature and nurture predispose to violent behavior: serotonergic genes and adverse childhood environment.* Neuropsychopharmacology, 2007. 32(11): p. 2375-83.
128. Kim-Cohen, J., et al. *MAOA, maltreatment, and gene-environment interaction predicting children's mental health: new evidence and a meta-analysis.* Mol Psychiatry, 2006. 11(10): p. 903-13.
129. Bondy, B., A. Buettner, and P. Zill. *Genetics of suicide.* Mol Psychiatry, 2006. 11(4): p. 336-51.
130. Meyer-Lindenberg, A., et al. *Neural mechanisms of genetic risk for impulsivity and violence in humans.* Proc Natl Acad Sci U S A, 2006. 103(16): p. 6269-74.
131. Patkar, A.A., et al. *Serotonin transporter polymorphisms and measures of impulsivity, aggression, and sensation seeking among African-American cocaine-dependent individuals.* Psychiatry Res, 2002. 110(2): p. 103-15.
132. Davridge, K., Atkinson, L., Douglas, L., Lee, V., Shapiro, S., Kennedy, JL., Beitchman, JH. *Association of the serotonin transporter and 5-HT1Dbeta receptor genes with extreme, persistent and pervasive aggressive behaviour in children.* Psychiatr Genet 2004. 14: p. 143-146.
133. Beaver, K.M., et al., *A gene x gene interaction between DRD2 and DRD4 is associated with conduct disorder and antisocial behavior in males.* Behav Brain Funct, 2007. 3: p. 30.
134. Congdon, E., K.P. Lesch, and T. Canli. *Analysis of DRD4 and DAT polymorphisms and behavioral inhibition in healthy adults: implications for impulsivity.* Am J Med Genet B Neuropsychiatr Genet, 2008. 147B(1): p. 27-32.
135. Soderstrom, H. *Neuropsychiatric Background factors to violent crime*, in *Institute of Clinical Neuroscience, Section of Psychiatry.* 2002, University of Gothenburg: Gothenburg.
136. Holmberg, G., Forsman, A., Grann, M., Ingerloo, L. -E., Skagerberg, S., Somander, L. *Psykiatrisk vård för fängelsedömda. (Psychiatric care for those sentenced to prison).* . Nordisk Tidskrift for Kriminalvidenskap, 1999. 86: p. 206-219.
137. National Board of Forensic Medicine. *Annual statistics.* <http://www.rmv.se/> 2002.
138. Holmberg, G., Kristiansson, M. *Contacts with public services, with special reference to mental health care, preceding a serious crime: A retrospective study of 268 subjects of forensic psychiatric investigations* International Journal of Law and Psychiatry Res, 2006. 29: p. 281-288.

139. First, M., Spitzer, R., Gibbon, M., Williams, J. *User's Guide for the Structured Clinical Interview for DSM-IV Axis I Disorders-Research Version 2.0 (SCID-I)*. New York State Psychiatric Institute, Biometrics Research Department, New York, NY, 1996.
140. First, M., Gibbon, M., Spitzer, R., Williams, J., Smith Benjamin, L. *Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II)*. American Psychiatric Press, Washington, DC., 1997.
141. Ehlers, S., Gillberg, C. *The epidemiology of Asperger syndrome: A total population study*. J Child Psychol Psychiatry 1993. 34(8): p. 1327-1380.
142. Gillberg, C. and E. Billstedt. *Autism and Asperger syndrome: coexistence with other clinical disorders*. Acta Psychiatr Scand, 2000. 102(5): p. 321-30.
143. Ward, M., Wender, PH., Reimherr, FW., *The Wender Utah Rating Scale: An aid in the retrospective diagnosis of childhood attention deficit hyperactivity disorder*. Am J Psychiatry 1993. 150 (6): p. 885-890.
144. Wechsler, D. *Manual for the Wechsler Adult Intelligence Scale-Revised*. The Psychological Corporation, San Antonio, TX, Psychological Corporation 1981.
145. Wechsler, D. *Manual WAIS-R - Swedish Version*. . Stockholm, Psykologiförlaget 1996.
146. Hare, D., Harpur, T.J., Hakstian, A.R., Forth, A.E., Hart, S.D., Newman, J.P. *The revised psychopathy checklist: reliability and factor structure*. Psychol Assess, 1990. 2: p. 338-341S.
147. Brown, G.L., et al. *Aggression, suicide, and serotonin: relationships to CSF amine metabolites*. Am J Psychiatry, 1982. 139(6): p. 741-6.
148. Cooke, D.J. and C. Michie. *Refining the construct of psychopathy: towards a hierarchical model*. Psychol Assess, 2001. 13(2): p. 171-88.
149. Schalling, D., Edman, G. ed. *The Karolinska Scales of Personality (KSP). An inventory for assessing temperament dimensions associated with vulnerability for psychosocial deviance. Manual*. 1993, The Department of Psychiatry, The Karolinska Institute, : Stockholm.
150. Cloninger, C.R., D.M. Svrakic, and T.R. Przybeck. *A psychobiological model of temperament and character*. Arch Gen Psychiatry, 1993. 50(12): p. 975-90.
151. Beck AT, S.R., Brown GK. *Manual for the Beck Depression Inventory-II*. San Antonio, Psychological Corporation, 1996.
152. Beck AT, W.C., Mendelson M. *Beck Depression Inventory (BDI)*. Arch Gen Psychiatry, 1961. 4: p. 561-571.
153. Orelund, L., et al. *Smoking only explains part of the associations between platelet monoamine oxidase activity and personality*. J Neural Transm, 2002. 109(5-6): p. 963-75.
154. Coccini, T., et al. *Platelet monoamine oxidase B activity as a state marker for alcoholism: trend over time during withdrawal and influence of smoking and gender*. Alcohol Alcohol, 2002. 37(6): p. 566-72.
155. Snell, L.D., J. Glanz, and B. Tabakoff. *Relationships between effects of smoking, gender, and alcohol dependence on platelet monoamine oxidase-B: activity, affinity labeling, and protein measurements*. Alcohol Clin Exp Res, 2002. 26(7): p. 1105-13.
156. Svennerholm, L., et al. *Chemical differentiation of the Gaucher subtypes*. Prog Clin Biol Res, 1982. 95: p. 231-52.
157. Smith, P.K., Krohn, R.I., Hermansson, G.T., Mallia, A.K., Gartner, F.H., Provenzano, M.D., Fujimoto, E.K., Goeke, N.M., Klenk, D.C. *Measurement of protein using bicinchoninic acid*. Anal Biochem, 1998. 150: p. 76-85.

158. Fowler, C.J. and K.F. Tipton. *Concentration dependence of the oxidation of tyramine by the two forms of rat liver mitochondrial monoamine oxidase*. *Biochem Pharmacol*, 1981. 30(24): p. 3329-32.
159. Blennow, K., et al. *Cerebrospinal fluid monoamine metabolites in 114 healthy individuals 18-88 years of age*. *Eur Neuropsychopharmacol*, 1993. 3(1): p. 55-61.
160. Rice, M.E., Harris, G.T. *Violent recidivism: Assessing predictive validity*. *Journal of Consulting and Clinical Psychology*, 1995. 63: p. 737-748.
161. Green, D.M., Swets, J. M. ed. *Signal detection theory and psychophysics*. 1966, John Wiley and Sons Inc: New York.
162. Mossman, D. *Assessing predictions of violence: Being accurate about accuracy*. *Journal of Consulting and Clinical Psychology*, 1994. 62: p. 783-792.
163. Gillberg, I.C. and C. Gillberg. *Asperger syndrome--some epidemiological considerations: a research note*. *J Child Psychol Psychiatry*, 1989. 30(4): p. 631-8.
164. SBU. *Riskbedömningar inom psykiatrin - kan våld i samhället förutsägas? (Psychiatric Risk Assessment Methods Are Violent Acts Predictable?)*. 2005, SBU (Statens beredning för medicinsk utvärdering/Swedish Council on Technology Assessment in Health Care): Stockholm.
165. Brennan, P.A. and S.A. Mednick. *Genetic perspectives on crime*. *Acta Psychiatr Scand Suppl*, 1993. 370: p. 19-26.
166. Grigorenko, E.L., et al. *Aggressive behavior, related conduct problems, and variation in genes affecting dopamine turnover*. *Aggress Behav*, 2010. 36(3): p. 158-76.
167. Lagoa, A., et al. *Genetics and criminal behaviour: recent accomplishments*. *Med Sci Law*, 2009. 49(4): p. 274-82.
168. Zuckerman, M. ed. *Psychobiology and personality*. 1991, Cambridge university press: Cambridge.
169. Richard-Devantoy, S., J.P. Olie, and R. Gourevitch. *[Risk of homicide and major mental disorders: a critical review]*. *Encephale*, 2009. 35(6): p. 521-30.
170. Hare, R.D. and C.S. Neumann. *Psychopathy: assessment and forensic implications*. *Can J Psychiatry*, 2009. 54(12): p. 791-802.
171. Amore, M., et al. *Predictors of violent behavior among acute psychiatric patients: clinical study*. *Psychiatry Clin Neurosci*, 2008. 62(3): p. 247-55.
172. Jokinen, J., Forslund, K., Nordström, AL., Lindqvist, P., Nordström, P. *Suicide risk after homicide in Sweden*. *Arch suicide res*, 2009. 13(3): p. 297-301.
173. Palijan, T.Z., L. Muzinic, and S. Radeljak. *Psychiatric comorbidity in forensic psychiatry*. *Psychiatr Danub*, 2009. 21(3): p. 429-36.