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On lifetime violent criminality in a Swedish forensic psychiatric cohort

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DEPARTMENT OF CLINICAL SCIENCES | FACULTY OF MEDICINE | LUND UNIVERSITY



On lifetime violent criminality in a Swedish forensic psychiatric cohort

Hedvig Krona



DOCTORAL DISSERTATION

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Faculty opponent

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On lifetime violent criminality in a Swedish forensic psychiatric cohort		
Abstract		
Introduction		
The pathway from childhood adversity through delinquen	t behaviour in adolescence to an antisocial adult lifestyle has interested for researchers	
for the past decades. Recovery from mental health problem	is and the prevention of future criminal behaviour may be complicated by the	
combination of mental illness and criminality. Identifying	crucial risk factors and enhancing risk prediction is important in reducing criminal	
recidivism and improving mental health.		
Aims and Methods		
The overall aim of this thesis is to explore a total cohort of 125 individuals in the catchment area of the Skåne University Hospital, Malmö, who		
were sentenced to forensic psychiatric in-patient treatment	between 1999 and 2005. Specific aims were: (1) to identify the background and	
clinical characteristics of the cohort; (2) to investigate the relationships between risk assessments, prevalence of adverse events and length of stay in		
forensic psychiatric care; (3) to explore risk factors for criminal recidivism and persistence over the life-span, and (4) to examine whether		
neuroimaging data improves the prediction of criminal recidivism.		
Results		
Two thirds of the cohort (n=84, 67 %) received forensic psychiatric treatment with SCS. The majority suffered from psychotic disorders (n=91,		
73 %). Length of stay was predicted by previous contact with child- and adolescent psychiatry, violent index crime, psychotic disorders, substance		
use and absconding during in-patient treatment. Risk factors for recidivism in crime after in-patient treatment were childhood adversities and		
early-onset criminality. In a lifetime perspective of crime and criminal careers, a sub-group of individuals who were more criminally active than		
the others were characterised by childhood adversities, neurodevelopmental disorders, and substance use disorders. When data from		

neuroimaging investigations were added to already well-known risk factors, accuracy was improved in identifying those who relapsed to crime.

Conclusions

These findings emphasise the importance of identifying early those children and adolescents who experience childhood adversities, are diagnosed with neurodevelopmental disorders, and have an early debut in substance use as these may increase the risk of an antisocial lifestyle and a long criminal career. Violent crime predicts a longer stay in a forensic psychiatric setting regardless of any underlying disorder. Adding neuroimaging data may enhance risk prediction of recidivism in crime.

Key words

Forensic psychiatry, violent crime, lifetime criminality, mental disorders, crime prediction, risk assessment, SPECT Classification system and/or index terms (if any) Supplementary bibliographical information Language English ISBN ISSN and key title 1652-8220 978-91-7619-903-9 Recipient's notes Number of pages Price Security classification

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Hedvig Krona



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Birds flying high, you know how I feel Sun in the sky, you know how I feel Breeze driftin' on by, you know how I feel

> It's a new dawn It's a new day It's a new life For me And I'm feeling good

Feeling Good – Nina Simone

To Hilma and August

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Abstract

Introduction

The pathway from childhood adversity through delinquent behaviour in adolescence to an antisocial adult lifestyle has interested for researchers for the past decades. Recovery from mental health problems and the prevention of future criminal behaviour may be complicated by the combination of mental illness and criminality. Identifying crucial risk factors and enhancing risk prediction is important in reducing criminal recidivism and improving mental health.

Aims and Methods

This thesis has an overall aim to give a description of a naturalistic, total cohort of 125 individuals belonging to the catchment area of the Skåne University Hospital, Malmö, who were sentenced to forensic psychiatric in-patient treatment between the years 1999-2005. Specific aims have been: (1) to identify background and clinical characteristics of the cohort; (2) to investigate the relationship between risk assessments, prevalence of adverse events and length of stay in forensic psychiatric care; (3) to explore risk factors for criminal recidivism and persistence over the life-span, and; (4) to examine if neuroimaging data improves the prediction of criminal recidivism.

Results

Two thirds of the cohort (n = 84, 67 %) received forensic psychiatric treatment with SCS. The majority suffered from psychotic disorders (n = 91, 73 %). Length of stay was predicted by previous contact with child- and adolescent psychiatry, violent index crime, psychotic disorders, substance use and absconding during in-patient treatment. Risk factors for recidivism in crime after in-patient treatment were childhood adversities and early-onset criminality. In a lifetime perspective of crime and criminal careers, a sub-group of individuals who were more criminally active than the others were characterised by childhood adversities, neurodevelopmental disorders, and substance use disorders. When

data from neuroimaging investigations were added to already well-known risk factors, accuracy was improved in identifying those who relapsed to crime.

Conclusions

These findings emphasise the importance of identifying early those children and adolescents who experience childhood adversities, are diagnosed with neurodevelopmental disorders, and have an early debut in substance use as these may increase the risk of an antisocial lifestyle and a long criminal career. Violent crime predicts a longer stay in a forensic psychiatric setting regardless of any underlying disorder. Adding neuroimaging data may enhance risk prediction of recidivism in crime.

Svensk populärvetenskaplig sammanfattning

Introduktion

Våld har alltid funnits närvarande genom mänsklighetens historia, från organiserat militärt våld till interpersonellt våld mellan civila förövare och offer. Om ett barn utsatts för svåra förhållanden och våld under uppväxtåren löper det risk att utveckla psykisk ohälsa, kriminalitet samt våldsamt beteende under tonåren och vuxenlivet. Det är endast en minoritet av alla som är drabbade av psykisk sjukdom som begår brott men om brottet har fängelse i straffskalan kan man dömas till rättspsykiatrisk vård. Vi vet fortfarande förhållandevis litet om vad som kännetecknar denna grupp brottsdömda personer med allvarlig psykisk sjukdom, om det finns meningsfulla undergrupper och vad som predicerar vårdförlopp samt återfall i brott.

Syften och metod

Denna avhandling har haft som övergripande målsättning att undersöka förhållandet mellan individuella karakteristika hos personer dömda till rättspsykiatrisk vård och förekomsten av kriminalitet, i synnerhet våldsbrottslighet, över tid. Specifika målsättningar har varit: (1) att beskriva bakgrunds- och kliniska karakteristika hos kohorten; (2) att undersöka sambandet mellan riskbedömningar, förekomsten av negativa händelser samt längden på slutenvårdstiden inom rättspsykiatrin; (3) att utforska riskfaktorer för återfall i brottslighet liksom för långvarig kriminalitet under livsloppet, samt; (4) att utforska huruvida fynd från röntgenundersökningar förbättrar prediktionen av återfall i brottslighet.

Samtliga delar i avhandlingen utgår från studien Uppföljningsstudier i Rättspsykiatri – Malmökohorten (UPPRÄTT-Malmö) där samtliga individer (n = 125), mantalsskrivna i Skånes Universitetssjukhus (SUS) Malmös upptagningsområde och dömda till rättspsykiatrisk vård under åren 1999–2005, ingår. Detta innebär att alla deltagare i studien led av en allvarlig psykisk störning när de inkluderades i studien och att de

vårdades inom rättspsykiatrin SUS Malmö efter domen. Avhandlingen grundar sig på data från rättspsykiatriska undersökningar, journalgenomgångar samt från svenska officiella register som lagrar data avseende kriminalitet över livsloppet (Brottsförebyggande rådet) och dödsorsaker (Socialstyrelsen).

Resultat

I avhandlingens första och andra artiklar (*Papers I* och *II*) beskrivs bakgrundsfaktorer och kliniska karakteristika. Majoriteten av UPPRÄTT-kohorten är män (n = 101, 81 %) och en tredjedel har haft kontakt med barn- och ungdomspsykiatri (n = 34, 32 %). Den vanligaste diagnosen vid tidpunkten för rättspsykiatrisk undersökning är psykos (n = 91, 73 %) men det är också vanligt med samtidigt missbruk (n = 60, 48 %) samt personlighetsstörningar (n = 34, 27 %). Det var främst fall av våldsbrott som ledde till rättspsykiatrisk vård (n = 105, 84 %).

I avhandlingens första artikel (*Paper I*) analyserades vårdförloppen med fokus på vårdtid och negativa händelser under vården. Artikeln visade stora skillnader i medianvårdtid mellan de som dömts till rättspsykiatrisk vård med (1272 dagar) respektive utan (273 dagar) särskild utskrivningsprövning, trots att båda gruppernas psykiatriska vårdbehov var likartade. I studiegruppen var 71 individer (60 %) inblandade i någon form av negativ händelse, till exempel hot och våld, under vårdtiden. Våldsbrott i indexdomen samt att avvika från vårdinrättningen predicerade längre vårdtider.

I artikel två (*Paper II*) undersöktes återfall i brott efter dom till rättspsykiatrisk vård (i mediantal 6,2 år efter indexdom). Trettiofem individer (28 %) återföll i brott varav 21 individer (17 %) i våldsbrott. Att ha en förstagradssläkting med psykisk ohälsa, att inte ha avslutat grundskolan samt ung ålder vid första dömda brottet var riskfaktorer som predicerade återfall i alla former av brottslighet. Risken för återfall i våldsbrott ökade om man hade en diagnos inom kluster B samt om man hade låga poäng på GAF-skalan vid tidpunkten för den rättspsykiatriska utredningen. Att vårdas enligt LRV med SUP minskade risken för återfall i båda formerna av brottslighet.

Vid studie av kriminalitet under hela livstiden. Med hjälp av klusteranalys grupperades deltagarna i två undergrupper, vilka sedan studerades i förhållande till debutålder och frekvens för olika typer av lagförd kriminalitet men också i förhållande till negativa händelser samt vårdtid. Vi fann en mindre undergrupp som jämfört med övriga personer i kohorten bland annat kännetecknades av lägre utbildningsnivå, mer kontakt med barnoch ungdomspsykiatrin under uppväxtåren samt att man diagnosticerats med såväl neuropsykiatrisk funktionsvariation som missbruk vid tidpunkten för den rättspsykiatriska undersökningen. Denna mindre undergrupp präglades av en mer omfattande kriminalitet under livstiden i form av ett större antal dömda brott samt en lägre ålder vid första brott. Avhandlingens fjärde artikel (*Paper IV*) har haft som fokus att undersöka huruvida man kan förbättra riskbedömningar för återfall i brottslighet med hjälp av neuroradiologiska undersökningar, mer specifikt i form av blodflödesmätningar av hjärnan (SPECT). Jämfört med den traditionella modellens riskfaktorer för recidivism, inkluderande kluster B personlighetssyndrom, ålder vid första dömda brottet samt vid tidpunkten för den rättspsykiatriska undersökningen och missbruk, bidrog information från blodflödesmätningar till en ökad korrekthet i identifieringen av de individer i kohorten som återföll i brottslighet.

Slutsatser

Majoriteten av de rättspsykiatriska patienterna i UPPRÄTT-studien är män som drabbats av psykossjukdom och det är också vanligt med missbruk. Vårdtiden inom rättspsykiatrin varierar kraftigt men inte som en följd av den psykiska ohälsan utan den bedömda risken för återfall och i viss utsträckning av följsamhet till behandlingen. Avhandlingen har visat att riskfaktorer för återfall i kriminalitet efter avslutad vård ofta är kopplade till förhållanden och beteenden som går att spåra till uppväxtåren, såsom sina föräldrars psykiska hälsa, bristfällig skolgång samt utagerande och normbrytande beteende. Brottsaktiviteten över livsloppet varierar kraftigt bland personer dömda till rättspsykiatrisk vård. I linje med tidigare forskning på personer utan allvarlig psykisk sjukdom har vi funnit en mindre grupp mer brottsaktiva individer som uppvisat tidigt debuterande funktionssvårigheter, psykisk ohälsa, normbrytande beteende samt där ett senare missbruk ingår som en del. Dessa individers livslopp präglas av en mer persistent kriminalitet. Studien har visat att traditionella riskbedömningar eventuellt kan förbättras ytterligare, exempelvis genom att nyttja information inhämtade från neuroradiologiska undersökningar. Sammantaget bidrar avhandlingen med en kunskap som kan vägleda kliniker och rättsväsende i bedömning och behandling av rättspsykiatriska patienter.

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List of papers

This thesis is based on the following papers, which are referred to in the text by their roman numerals:

- I. Andreasson H, Nyman M, Krona H, Meyer L, Anckarsäter H, Nilsson T, Hofvander B. (2014). *Predictors of length of stay in forensic psychiatry: The influence of perceived risk of violence*. International Journal of Law and Psychiatry. doi: 10.1016/j.ijlp.2014.02.038.
- II. Krona H, Nyman M, Andreasson H, Vicencio N, Anckarsäter H, Wallinius M, Nilsson T, Hofvander B. (2016) *Mentally disordered offenders in Sweden: Differentiating recidivists from non-recidivists in a 10 year follow-up study.* Nordic Journal of Psychiatry. doi: 10.1080/08039488.2016.1236400
- III. Krona H, Anckarsäter H, Nilsson T, Hofvander B. Patterns of lifetime criminality in mentally disordered offenders – findings from a nationally representative cohort. Manuscript in preparation.
- IV. Delfin C, Krona H, Andiné P, Ryding E, Wallinius M, Hofvander B. Prediction of recidivism in a long-term follow-up of forensic psychiatric patients: Incremental effects of neuroimaging data. PLoS ONE 4(5): e0217127. doi: 10.1371/journal.pone.0217127.

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Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
ASD	Autism Spectrum Disorder
ASPD	Antisocial Personality Disorder
AUC	Area Under the receiver operating characteristic Curve
CD	Conduct Disorder
DSM	Diagnostic and Statistical Manual of mental disorders
FPI	Forensic Psychiatric Investigation
FPSR	Forensic Psychiatric Screening Report
GAF	Global Assessment of Functioning
HCR-20	Historical Clinical Risk Management-20
ICD-10	International Classification of Diseases 10th edition
NDD	Neurodevelopmental Disorder
PCL-R	Psychopathy Checklist-Revised
PCL:SV	Psychopathy Checklist-Screening Version
rCBF	Regional Cerebral Blood Flow
RF	Random Forest classification
SMD	Severe Mental Disorder
SCID-I	Structured Clinical Interview for Axis I Disorders
SCID-II	Structured Clinical Interview for Axis II Disorders
SCS	Special Court Supervision
SPECT	Single-photon emission computed tomography
SUD	Substance Use Disorder
UPPRÄTT	Uppföljning av Rättspsykiatriska patienter [forensic psychiatric follow-up studies] – the Malmö cohort.

Introduction

'Many who live with violence day in and day out assume that it is an intrinsic part of the human condition. But this is not so. Violence can be prevented. Violent cultures can be turned around. [...] Governments, communities and individuals can make a difference.'

Nelson Mandela, 2002 (1)

Violence, "the use of physical force so as to injure, abuse, damage or destroy"(2), occurs in everyday life in many different forms. Although we read and watch news about violence in public scenarios such as wars, terrorist attacks, and mass killings, we also hear about or experience self-directed or interpersonal violence between members of our families or circles of friends. Violence is therefore an experience almost everyone has to relate to in one way or another, and it takes a heavy toll on all societies.

The World Health Organization defines interpersonal violence as violence that occurs between family members, intimate partners, friends, acquaintances, and strangers. It includes child maltreatment, youth violence, intimate partner violence, sexual violence, and elder abuse (3). Self-harming behaviours have also been suggested as part of the concept of violence (4) and approximately 1.3 million people worldwide die each year from some form of violence including self-directed (defined as suicidal behaviour and selfmutilation), interpersonal, and collective violence (violence involving a group of individuals, from spontaneous riots to rebellions, revolutions, terrorism, and war). An estimated 464 000 people were victims of homicide in 2017, with rates differing depending on the socioeconomic status of the reporting countries (5). Added to this number are the tens of thousands who every day become victims of non-fatal violence. In a 2014 World Health Organization report, a quarter of all adults reported having been physically abused as children and one in five women reported having been sexually abused as a child (3). Despite these high numbers, viewed historically global rates of violence are on the decline. Homicides in particular have decreased in recent centuries, due perhaps to such developments as the modern nation-state, judiciary systems, commerce, and feminism (6).

Nevertheless, the consequences of violence are long-lasting and the mechanisms of violence and why it erupts are various and multifaceted. The cost in terms of health care, legal costs, absenteeism from work, and lost productivity is counted in billions of US dollars.

Risk factors for violent crime

From childhood adversity to adult criminality and violence

Economic inequality, inadequate parenting, and drug- and alcohol misuse all increase the likelihood of child maltreatment, youth violence, suicidal behaviour, intimate partner violence, and sexual violence against women (1). Child maltreatment, an umbrella term for several types of abuse, is divided by the World Health Organization into five subtypes: physical abuse; sexual abuse; neglect and negligent treatment; emotional abuse; and exploitation (7) which together with other forms of childhood adversities affect the risk for mental health problems and future violent behaviour.

Pathways to delinquent behaviour, and for some, to an adult antisocial lifestyle has interested researchers for decades (8). Violent behaviour can be observed even in very young children, and to a certain extent may be non-pathological at certain stages. As children become older, they usually internalise socially acceptable behaviours through interactions with their environment (9, 10). Continuing violent behaviour, however, is most often a continuation of problematic aggressive behaviours exhibited in children as young as toddlers. High levels of physical aggression at 17 to 42 months of age may lead to aggressive behaviour later in life and is predicted by the same risk factors as adolescent antisocial behaviour: young age of mother at birth of her first child, poverty, family dysfunction, and coercive parenting (to name a few), and it is especially distinct in males (11). Children who exhibit persistent aggression in elementary school are at high risk to continue both physical violence and non-violent delinquency during and beyond adolescence (12). Aside from harming victims and families, these aggressive children tend to develop conduct problems and later to run a higher risk than others of substance use disorders (SUDs), accidents, mental health problems, spousal abuse, and neglectful parenting (13-16).

The highest incidence and prevalence of offending occurs during adolescence, with a peak at 17 years of age. Although by age 28 almost 85 % of former offenders have abstained from offending (17), 3 % to 7 % of children maintain frequent aggression from childhood into adolescence (18). Childhood maltreatment has been linked to subsequent criminality, with hyperactivity in elementary school, living in a non-intact family, low parental education, child psychiatric history, and conduct problems such as bullying all found to be separate risk factors for criminal offences in late adolescence (19, 20). Duke

and colleagues examined the impact of adverse childhood events in a large sample of students and found that each category of adversities carried an increased risk of violence in male participants and the risk increased with more than one adversity (21). They also concluded that being sexually molested by a family member increased the risk that a boy would later engage in dating violence (threats and/or physical or sexual abuse of a romantic partner) by 45 times.

In a study of male adult offenders, Reavis and colleagues reported a nearly four times higher number of adverse childhood events in the offender group than in a normative adult male population (22). The research team theorised that the link between adverse childhood events and subsequent adult criminal behaviour was a combination of attachment pathology based on intensely negative feelings towards attachment figures following poor treatment paired with neurobiological dysregulation.

These and previous findings (23), emphasise the environmentally progression of violence from generation to generation mediated through childhood adversities (24).

Much research aimed to prevent physical and mental suffering, as well as socioeconomic consequences, has examined the trajectories of childhood aggression and oppositional behaviour through to violent delinquency in adolescence and adulthood.

Biology and violence

Both nature and nurture seem to have a part in violent adult behaviour, which seems to have its roots in early adverse childhood experiences, but also has biological explanations. The most salient emotional processes, including aggression, are thought to happen in subcortical parts of the brain such as the limbic system and the amygdala (25). Lesions to these parts of the brain and the prefrontal cortices have shown impairments in the recognition of expressions of fearful faces (26), cognition, memory, and emotional processes (27) lack of control of aggression, and increased risk for violence (28, 29). Brain injuries or reduced blood flow in these areas can be identified with brain imaging techniques such as single-photon emission computed tomography (SPECT) or functional magnetic resonance imaging (fMRI).

Most serious crimes are perpetrated by males, and research suggests that the male brain may be more biologically vulnerable to adverse environmental experiences, which may disturb the development of the brain and result in epigenetic changes promoting violent behaviour (30). One example is the monoamine oxidase A gene, located on the X chromosome, which encodes the monoamine oxidase A enzyme that metabolises and deactivates several neurotransmitters. Polymorphisms of this gene has been linked to aggressive behaviour in both animal and human models, suggesting that gene–environment effects in childhood may lead to antisocial behaviour later in life (31).

Hormonal changes have also been shown to be linked to aggressive behaviour. Boys who are exposed to higher than normal levels of testosterone have been shown to be less empathetic and more aggressive (30). Studies of the stress-response system have shown that parental warmth is important for the development of correct cortisol response in early childhood and that aberrations may lead to aggressive behaviour (32). It is thus clear that although each individual is born with certain biological pre-conditions, the environment and family nurture are important influences on health and behaviour.

Neurodevelopmental disorders and developmental theories

Many studies (33-37) have linked neurodevelopmental disorders (NDDs) such as attention deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), and learning disabilities to disruptive disorders and later criminality, particularly when combined with childhood adversities. NDDs overlap in many cases (38), persist into adulthood (35, 39, 40) and also carry a risk of other adverse outcomes such as mental disorders (e.g., mood and anxiety disorders) and substance use (41, 42).

Disruptive disorders such as oppositional defiant disorder and conduct disorder (CD) in children and adolescents show a pattern of defiant, uncooperative, negativistic, aggressive, and rule-breaking behaviours. These behaviours are common during development, but not all children and adolescents who show these behaviours will engage in criminal activities as adults. To differentiate between those who continually relapse to antisocial behaviour throughout their lives from those who do not, researchers have proposed different pathways to criminal behaviour.

The developmental taxonomy of antisocial behaviour presented by Moffitt in 1993 (43) outlines pathways for children and adolescents to develop criminal behavioural patterns and differing prognostics for adult criminality. The childhood phenotype predicted to express the rarer persistent criminal pathway was describe as a combination of early-onset neurodevelopmental problems and negative parenting practices. Individuals with these characteristics showed a very stable pattern of conduct problems and antisocial behaviour persisting into adulthood and later forming an antisocial personality disorder. The more common adolescence-limited antisocial behavioural pathway consisted of individuals who prior to adolescence showed no sign of developmental aberrations and much fewer conduct problems yet, during their teens, mimicked the behaviours of antisocial peers in their environment. Moffit's taxonomy suggested that as a social phenomenon the criminal behaviour abated when maturity ensued in the adolescence-limited pathway, whereas it continued into adulthood in the life-course–persistent pathway due to a combination of genetic risk factors and toxic environmental effects (43).

Moffitt's research came to have a major impact as it influenced and helped to combine several disciplines such as criminology, psychopathology, and education (44). However, further research on the taxonomy has suggested a refinement of the two pathways, which have been suggested to be oversimplified (45). For example, the adolescence-limited pathway also contains developmental and other risk factors. In addition, when the Dunedin cohort was followed up at 32 years of age, both the life-course–persistent group

and the adolescence-limited group had high levels of both physical and mental health problems (46).

Other explanations for criminal behaviour have also emerged. Gottfredson and Hirschi proposed that the main factor behind criminal behaviour is the lack of individual self-control (47) Their theory posits that because opportunities to commit crimes are abundant and those who lack self-control will inevitably become involved in criminal behaviour, it is therefore the lack of self-control rather than lack of opportunity that determines an individual's propensity to criminal behaviour. Those individuals with a lack of self-control are also described as impulsive, callous, and in need of immediate satisfaction, factors also associated with risk taking and antisocial behaviour. According to Gottfredson and Hirschi, a child's level of self-control is heavily influenced by the parent's child-rearing practices (47).

Mental disorders and violent crime

The medical field of forensic psychiatry offers treatment for perpetrators of crimes who suffer from major mental disorders (MMDs) and who, in many instances, also have psychosocial problems (48-52). Whether or not an MMD in itself carries an increased risk for criminality is debatable; research has shown an increased risk for criminal recidivism in those with an MMD (53-56), but this may also be explained by a co-morbid SUD (57).

Personality disorders

Ever since the Diagnostic and Statistical Manual of Mental Disorders third Edition (DSM-III (58)) was published in 1980, personality disorders (PDs) have been defined according to the same basic three clusters, all of which entail significant impairments in self and interpersonal functioning and one or more pathological personality traits, all relatively stable across time and consistent across situations (58-60). Studies have shown an increased risk for violent crime amongst adolescents and young adults with PDs in clusters A and B (61), and because the symptoms usually continue to manifest in adulthood, the expected number of individuals with PDs in offender populations is high (62).

PDs are associated with a heightened risk of violence in the adult population. The prevalence of PDs in the general population is between 4-13% (63) but the number rises steeply in offender populations. In a sample of male prisoners, 65 % had a PD diagnosis in any cluster and 47 % had an antisocial personality disorder (ASPD) specifically which makes it 10 times more likely to have ASPD in an offender population compared to the general population (64). In a female offender population, 42 % were reported to have any type of PD and 21 % had ASPD specifically (64).

The diagnosis of ASPD is many times at the adult end of a progression of behavioural disturbances beginning in childhood years with oppositional defiant disorder and later CD. Family factors including familial psychopathology, poor disciplinary practices, maltreatment and neglect and also prenatal risk factors such as in-utero exposure to alcohol and toxins have been studied as potential risk factors for CD (65, 66). Not surprisingly, if a child exhibits symptoms of CD before the age of 15, the risk of committing assault and other aggressive behaviours, such as being convicted for a violent crime later in adulthood, is heightened (67). Furthermore, a larger proportion of individuals with adolescent CD compared with normative adolescents will later in life develop schizophrenia and in a study of adult individuals with schizophrenia, 40 % fulfilled the diagnostic criteria for CD before the age of 15 (42).

Compared with the general population, a PD carries a threefold increase in the odds of a violent outcome regardless of what cluster it belongs to. ASPD has a naturally higher risk with an odds ratio of 12.8 (63). In the same study, offenders with PDs had the same risk of violence as offenders with schizophrenia and they also had a two to three times higher odds of being repeat offenders.

In order to reduce crime, the possibility to offer significantly successful treatment to individuals with PDs and ASPD in particular would have been desirable. Unfortunately, treatment attempts have only shown limited effects and ASPD especially is at best only weakly modifiable and has a poor prognosis (68). In Sweden, PDs are in most cases not assessed as severe enough in themselves to warrant forensic psychiatric treatment but it is common in the forensic psychiatric population to have a PD as an additional diagnose. Offenders with a PD are in most cases sentenced to other sanctions.

Substance use disorders

SUDs are common both in the general population and in offender populations. In a Swedish study from 2004, 16 % of all violent crimes between 1998 to 2000 were committed by individuals diagnosed with alcohol misuse, and out of all violent crimes committed during that timeframe, more than one tenth were committed by individuals with drug misuse (69). A British study concluded that the largest public health impact on serious and repetitive violence was that of hazardous drinking (70).

To some vulnerable individuals, intake of drugs such as cannabis, amphetamines, and cocaine, which have strong psychotomimetic properties, may trigger the emergence of long-lasting psychotic symptoms, as up to 50 % of all individuals suffering from a first episode of psychosis also report a concurrent SUD (71). On an individual level, cannabis use has been found to give a two-fold increased relative risk of later development of schizophrenia, but as not all cannabis users develop schizophrenia and not all individuals who suffer from schizophrenia have a history of cannabis use, the link between cannabis and schizophrenia is not causal but more a component in the explanation of disorder development (72). Nevertheless, continuous use of both nicotine and cannabis during the

first year after a first episode of psychosis negatively affects medication adherence which in turn is a strong predictor of non-remission of psychotic symptoms (73).

Both alcohol and cannabis use elevate the risk for violent offending, explained partially by the disinhibitory effect of alcohol and the possible developmental history of CD among cannabis users, which could lead to their participation in the illicit drug trade (53). Treatment programmes for SUD reduce both psychological symptoms and crime rates, including both acquisitive and drug-related offences (74).

Psychotic disorders

Most mentally disordered offenders in forensic psychiatry suffer from psychotic disorders (75-77). Individuals suffering from psychotic disorders such as schizophrenia are associated with an increased risk of physically aggressive behaviour towards others (57, 78), which may be a direct result of the delusions and heightened levels of perceived threat in the environment that accompanies the disorder. In a meta-analysis by Large and Nielssen, approximately one third of all individuals suffering from a first-time episode of psychosis had exhibited some form of violent behaviour before initial treatment and one in six had committed a more serious violent offence involving assault of another person (79).

Offenders with schizophrenia have in studies been suggested to fall into one of three categories: (1) Type I offenders who early on exhibit symptoms of CD during childhood by displaying a behavioural pattern of aggressiveness which continues into adulthood as an antisocial personality structure and who later come to develop schizophrenia, (2) Type II offenders whose aggressive behaviour begins at the same time as the psychotic symptoms first emerge and (3) Type III offenders who suffer from an MMD for many years until they commit a severe violent act (78). Type I is hypothesised to be influenced by genes and environmental influences linked to both behavioural problems and MMDs, whereas types II and III are linked to neurological changes associated with the emergence of the MMD which includes effects of SUDs and medication (78).

The overlap of individuals displaying both CD and schizophrenia has led researchers to study if there may be a common genetic structure explaining the emergence of both behavioural problems and psychosis. Genetic polymorphisms associated with the development of both antisocial and aggressive behaviour are influenced by childhood adversities such as physical abuse (18, 80, 81), and schizophrenia is associated with a genetic vulnerability that may lead to the development of a manifest psychotic disorder in an individual exposed to environmental risk factors (82). The link between schizophrenia and violent behaviour has also been studied on a genetic level, yet a clear gene × environment explanatory model of the risk of developing both schizophrenia and aggressive behaviour has not yet been found (83).

Other diagnoses and when several disorders interact

Researchers during the past several decades have conducted large epidemiological studies to investigate the association between MMDs and violence and whether the co-morbidity of a SUD mediates a heightened risk of violent offences. In an early large study from 1992 on an unselected Swedish birth cohort, the estimated elevated risk for violence amongst males with a major mental health disorder was reported as four times higher and amongst females nearly three times higher compared to controls (84). Schizophrenia in particular leads to an increased risk of violence but the elevated risk estimates are usually modest and vary greatly between studies (85).

The comorbidity of SUD and MMD contributes to poor impulse control and a lowered adherence to psychological or pharmacological treatment, which in turn heightens the risk for violent events (86). When there is a comorbidity with a SUD, the risk is substantially increased with a four times higher pooled estimate according to Fazel and colleagues' meta-analysis compared to controls without comorbidity (87). This study also concluded that the increased risk of violent behaviour for individuals with schizophrenia and SUD was not different to what it was for individuals with a SUD alone. Instead it seems that the risk factors mediating increased risk for violence are the same for individuals suffering from schizophrenia as for the community at large; young age, a lack of education, prior offending and SUD (79). In a recent study by Sariaslan et al, the elevated risk of a psychotic individual committing a violent offence was found to be due to the same genetic influences which simultaneously elevated the risk to develop the psychiatric morbidity, a SUD and committing violent crime (88). According to this study, the same set of genes may answer for a multitude of symptoms and in extension, behaviours.

Other MMDs, such as affective disorders, psychotic disorders other than schizophrenia and organic brain syndromes resulting from brain injuries, have in epidemiological research been shown to carry a heightened risk for violent offending (89, 90). Conversely, in offender populations the psychiatric problems are prevalent. In a large review of 23000 offenders worldwide by Fazel and colleagues in 2002, prisoners were two to four times more likely to suffer from a psychotic disorder or a major depressive disorder compared to the general population (64).

Having the combination of psychiatric symptoms, violent behaviour and SUD has rendered the forensic psychiatric population to be described as 'the triply troubled' (91) and thereby presenting specific challenges to treatment providers. Yet, when regarding all violent crimes committed in a community in any current timeframe, it is important to remember that 85-90% are not related to MMDs (85) and that research trying to establish causalities between specific disorders and criminal outcome may risk to stigmatise an already vulnerable group of individuals. It is therefore imperative to weigh in all contributory factors when calculating risk and offering treatment (55).

Forensic psychiatry – the assessment and treatment of mentally disordered offenders

As opposed to legal practices in other countries where an offender may plead not guilty or be declared unfit to stand trial by reasons of insanity, the Swedish legislation differs as all offenders are regarded as accountable for their crimes and given a sanction (see the following section for further details on sanctioning). Suffering from an MMD does therefore not exempt from sanctions. The Swedish Penal Code Chapter 30, § 6 states that a person who has committed a crime under the influence of a severe mental disorder (SMD) shall at first hand be sentenced to another sanction than a prison sentence and that the court may only sentence a mentally disordered individual to prison if there are exceptional circumstances (the author's translation) (92). Instead, the recommendation is a sentence to compulsory forensic psychiatric treatment.

The medical field of forensic psychiatry is a sub-speciality to regular psychiatry which in Sweden has two main objectives; first, to offer risk assessments and forensic psychiatric investigations (FPIs) by the order of the courts of law and second, to provide forensic psychiatric treatment. The FPI assesses the presence of SMDs which have overlapping characteristics with MMDs yet with some distinct features.

The Swedish concept of a SMD is defined within a medico-legal discourse The previous theories of 'insanity' and 'equal to insanity' were changed by the implementation of the Forensic Mental Care Act, created in 1991(93) and were replaced by the concept of SMD which refers to the type and degree of a mental disorder. The judicial definition of an SMD is found in the preparatory work of the act, though the descriptions have been subject to slight change due to praxis in the courts, but a clinical diagnosis of an MMD within the regular healthcare framework is not necessarily equivalent of an SMD in a court setting.

SMD is generally and, in most cases, defined as various psychotic states yet with no discernment of the aetiology of the psychosis. The symptoms may be a part of a primary psychotic disorder such as schizophrenia or as a consequence of an affective disorder such as bipolar disorder. All diagnoses included in the definition of an SMD have in common symptoms such as a disturbed perception of reality, thought disturbances, confusion, various forms of delusions, and hallucinations. Severe PDs with compulsive behavioural problems, borderline psychotic symptoms, and uncontrollable impulsivity were also included in the definition of an SMD. The majority of all individuals sentenced to forensic psychiatric care in Sweden have a diagnosis of schizophrenia (75). Co-morbid diagnoses of SUD are very common in the forensic psychiatric population as 50 % of the women and almost 70 % of the males have a documented history of substance abuse (75). A diagnosis of a SUD alone is not enough to warrant a sanction of forensic psychiatric care.

Forensic psychiatric investigations

In order for a court to be able to sentence an offender to forensic psychiatric treatment, two prerequisites have to be fulfilled; he/she has to suffer from an SMD requiring continuous psychiatric care and he/she has to have committed a crime which warrants a sanction more severe than fines. More lenient sentences such as probation, fines, juvenile detention, etc. cannot be exchanged for forensic psychiatric care. Forensic psychiatric patients receive treatment either with or without special court supervision (SCS). Those receiving treatment with SCS are considered high-risk patients for recidivism in crime and cannot be discharged without a court approval.

According to Swedish legislation and The Act on Compulsory Mental Care (94), a courtordered pre-trial FPI is a pre-requisite for a sanction to forensic psychiatric treatment. This requires that the accused has either confessed or is tied to the crime by irrefutable evidence. A request for a forensic psychiatric assessment can be made by either the prosecutor or by the accused, but it is the court that decides whether to proceed with an assessment. The procedure of assessments is regulated in the Act of Forensic Psychiatric Investigation (95).

In most cases, the process begins by the court ordering a "lesser" forensic psychiatric evaluation to be made, which is presented in a forensic psychiatric screening report (FPSR). Approximately 1200 FPSRs are made each year (96). The FPSR is written by a senior forensic psychiatrist after a 1 to 2 hour psychiatric assessment of the offender. Through the FPSR, the forensic psychiatrist gives the court one of three recommendations: sentencing to other sanctions than forensic psychiatric treatment, sentencing to forensic psychiatric treatment without SCS or a suggestion that the prosecuted should be assessed through the more thorough FPI. This is mandatory if the proposed forensic psychiatric treatment is to be given with SCS. Approximately 521 FPIs were done in Sweden in 2018 (97), out of which 291 were individuals assessed to have an SMD.

The FPIs are carried out in two specialised units in Sweden localised in Gothenburg and Stockholm, both run by the state authority of the National Board of Forensic Medicine. Teams consisting of forensic psychiatrists, forensic psychologists, forensic social workers and the staff of the unit ward assess the prosecuted as he or she is in custody for four to six weeks at the unit. The team has access to the social, criminal, and medical histories registered by health, social, prison, and probation services as the FPI overrules otherwise strict privacy regulations between authorities.

In addition to previously known data, the forensic team performs psychometric tests, somatic examinations, interviews, and observations during the assessment. The results are assembled in a forensic psychiatric report that is then delivered to the court. Its main focus is whether or not the accused had or has an SMD at the time of the crime or during the assessment period. The report also recommends whether or not the accused should receive forensic psychiatric treatment, but the final decision is for the court to make.

Treatment in forensic psychiatry

As outlined above, an MMD is by definition a dynamic (i.e., able to change throughout the life course) risk factor. It carries a heightened risk for violence and it is therefore important, in the population of forensic psychiatric patients, to reduce psychiatric symptoms to lessen the risk of recidivism. In most settings, forensic psychiatric treatment rests on three corner-stones: medication, therapy, and rehabilitation. Therapy can be oneon-one or in groups, and it may be directed to drug-relapse prevention, processing of the committed crime or more specified therapies such as dialectical behavioural therapies. Rehabilitation is an umbrella term which includes daily activities and finding an occupation after release.

Most individuals receiving forensic psychiatric treatment are prescribed some form of psychotropic medication. Polypharmacy is common; in many cases the drugs prescribed are from different pharmaceutical groups (e.g., the combination of antipsychotics, antidepressants and mood stabilisers) and at times in high doses. In order to lower the risk for violence in mentally disordered offenders on an individual level, either high dose monotherapy or standard doses of two or more antipsychotics are recommended (98, 99).

In a national pharmaco-epidemiological study of Swedish individuals in which it was hypothesised that psychopharmacological treatment may affect violent behaviour in individuals with MMDs it was found that antipsychotic medication had a protective effect by reducing violent criminality rates and the finding were similar for mood stabilisers prescribed to men with a bipolar disorder. (100). For individuals with schizophrenia, adding a mood stabiliser to an already ongoing treatment with antipsychotics did not reduce violence significantly (100).

Thus, to prevent recidivism in violence amongst forensic psychiatric patients, treatment of an eventual SUD and medication to reduce the risk of relapse in affective disorders and psychotic symptoms seem to be of importance (56). Yet prescribing high doses of antipsychotic medication for a longer period of time may entail detrimental side-effects. Antipsychotic drugs are known to elevate the risk of metabolic syndrome, which leads to increased morbidity and mortality. Individuals with schizophrenia have a 20-25-year shorter life-span compared to the general population (101).

A Swedish five-year nationwide follow-up study of the association between the heightened mortality and exposure to antipsychotic drugs showed an U-shaped curve with a high risk of death amongst individuals with psychosis and no medication but similarly, a high risk amongst individuals with high doses of antipsychotic medication (102). In the latter group the high doses of antipsychotics were associated with higher mortality rates due to natural causes. Olfson et al described in their study that cardiovascular disease and respiratory diseases (e.g. lung cancer and chronic obstructive pulmonary disease) were the leading causes of death amongst individuals with schizophrenia (103). The higher risk of mortality amongst individuals with schizophrenia without antipsychotic treatment was hypothesised in Vermeulen et al.'s study to possibly be linked to a lesser propensity to seek neither psychiatric nor somatic treatment and consequently, running a higher risk of

acquiring somatic illnesses as well as suffering events of violence or suicide (104). In a Swedish study in which forensic psychiatric patients were followed for up to 9.4 years after discharge from forensic psychiatric treatment, the standardised mortality ratio was 10.4%. Approximately half of the deaths were related to somatic illnesses and the second largest cause of death was suicide (49). It is clear that individuals with schizophrenia require special preventive health efforts in order to lower the risk for them both perpetrating and becoming victims of violent crimes as well as suffering worsened mental and somatic health.

Risk factors for offending

Risk factors are in general either historical, describing circumstances which may elevate risk throughout life and thereby static, or dynamic, which implies that changes of the risk factor might alter the assessed risk of future events.

Previous research has suggested that many of the most important risk factors for violent criminality are the same amongst forensic psychiatric patients, offenders in prison and the general population; early-onset criminal behaviour, SUD and PDs (23, 51, 63, 70, 105, 106). SUD being an exception, these risk factors are fairly static in nature and thereby difficult to modify through interventions and treatment.

Repeat offending is common in the prison population; in a Swedish cohort, 43 % of prisoners were reported to be reconvicted for any crime within two years (107) and in a study from the US more than one in three offenders were reconvicted for a violent crime during the same timeframe (108). The relapse rates amongst forensic psychiatric patients is lower, where one in five relapsed in violent crime during an almost five-year long follow-up study (109), yet there is a lack of longer follow-up studies of these individuals.

The best risk factors for predicting recidivism in violent crime are the same for mentally disordered offenders as for non-disordered offenders, namely young age at first crime, male sex and previous conviction of violent crime (53-55, 110). The sub-group of high-risk repeat offenders are distinguished by an early onset and a persistent offending pattern in both the general offender population (43) as well as the forensic psychiatric population (111). In order to correctly predict future violent behaviour it is therefore important to consider the pathway of offending behaviour in risk management plans, regardless of any eventual MMDs such as psychosis being present (112).

The risk factors for violent criminality and violent reoffending are thus overlapping though not completely the same and many of the factors are historical and thereby impossible to alter. In order to improve the prediction of violent events, preferably by including dynamic variables, researchers continuously develop and enhance risk assessment instruments and thus far over 120 are constructed (113). To include dynamic risk factors in the instruments has proven to increase the predictive validity of institutional violence amongst forensic psychiatric populations (114).

Despite the seeming abundance of risk prediction instruments, systematic analyses comparing methods and tools have not been conclusive. Singh and Fazel conducted a metareview in 2010 of 40 reviews consisting of more than 2000 studies which included comparisons of whether actuarial assessments measured risk better than unstructured or structured clinical assessments, what risk assessment tools had the highest rate of predictive validity and whether the tools were applicable for different sample demographics (115). The study found mostly mixed results in the comparisons due to methodological inconsistencies, but also that there was no clear evidence of the risk assessments' validity in psychiatric samples and that the studies reported different previously well-known risk factors as the strongest predictors. In a follow-up study, Singh et al concluded that the instruments which were designed to assess risk in specific populations produced higher rates of predictive validity compared to instruments designed for general populations (113).

Risk assessments

To prevent recidivism in crime, usually defined as reconvictions, clinicians in forensic psychiatry have for many decades had the task to assess the risk for relapse. The estimated risk was then and is still today used for suggestion of judicial decisions such as if the prosecuted should be sanctioned to forensic psychiatric treatment or not, or, if forensic psychiatric treatment already is in progress, if the treatment should continue or if the individual could be released without risking public safety.

Until the later parts of the 20't century, clinicians assessed 'dangerousness' as an entity but the concept was challenged in the 1970s as studies showed that the individuals estimated as dangerous, did not in fact relapse in criminality when released (116). Added to this was critique of the theory of dangerousness as it suggested a dichotomy of either harmlessness or dangerousness where it in fact is a continuous factor.

Up until the 1980's, most risk predictions in mental health settings were performed by a psychiatrist or psychologist using unstructured clinical judgement (i.e., a subjective assessment of the risk of antisocial behaviour based on the clinician's education and previous experience). The unstructured clinical judgement had a predictive ability only slightly better than chance and improved methods were searched for (117).

In order to prevent new violent events in offender populations, risk assessment methods incorporating valid risk factors conferring a high level of predictive ability were on demand. During the 1990's, actuarial risk assessment guides such as Violence Risk Appraisal Guide (VRAG) (118) were developed, which were based mainly on static risk factors, i.e. historical factors which are stable over time. Compared to the unstructured clinical judgement, risk assessments with actuarial instruments showed a modest advantage in predictive accuracy (119, 120), yet they were criticised as it was difficult applying them in a risk moderating situation due to their static nature (121).

Assessing risk by only using a test instrument is thus better than chance but does not allow for individual risk factors to be taken into consideration (122). Therefore, today's most commonly used risk assessment model is the structured professional judgement model, which includes both assessment of the prevalence of statistically and empirically derived risk factors, but also a clinical judgement on risk level including individualspecific risk factors.

The most commonly used risk prediction tools in Swedish forensic psychiatry today are the Psychopathy Checklist-Revised (PCL-R) (123) and the History Clinical Risk-Management-20 (HCR-20, (124)). Psychopathy as a construct was first described by the American psychiatrist Hervey Cleckley in the 1940's (125) and diagnose-wise, psychopathy is conceptually most similar to ASPD in the DSM-IV and DSM-5 (59, 60) and dissocial personality disorder in International Classification of Diseases 10th edition (ICD-10, (126)).

The Canadian psychologist Robert D. Hare further methodologically defined the concept and created the assessment tool PCL-R (123). The PCL-R was designed in a prisoner setting as a diagnostic instrument made to estimate the extent an offender matched a prototypical psychopathic personality, including antisocial items, and by extension offering a risk measurement of future criminal behaviour. The instrument comprises of 20 items grouped into two factors (Interpersonal/Affective and Social Deviant) which in turn are split into four different facets describing the psychopathic personality: Interpersonal, Affective, Lifestyle, and Antisocial (127). Each item is scored on a threepoint scale (0-2), thus making 40 the maximum level of points. The cut-off for psychopathy in the USA is 30 and 25 in the U.K. Individuals with high scores indicating highly psychopathic traits have an earlier onset and a more diverse, severe, and persistent pattern of aggressive antisocial behaviour than other offenders (128, 129).

When examining if the PCL-R is sufficient in assessing risk in forensic psychiatric samples, the Antisocial facet has proven to be the strongest predictor of violent recidivism whereas the first three facets are not predicting violent recidivism (51, 130). The fourth facet describes mainly poor behavioural controls, an early age of onset and previous criminality which, as has previously been described in this thesis, has been identified as known risk factors. Nevertheless, clinicians are encouraged to continue to assess all four facets, in part to further explore and understand certain behaviours connected to psychopathy which facets 1-3 may divulge but also to use the fourth facet as a risk predictor (130).

The HCR-20 is an instrument with 20 items that was constructed to assist in a structured clinical violence risk assessment of a wide variety of offender populations. It consists of three parts where the H stands for historical items mediating a risk for violence later in life, C is for clinical risk items that are rated to affect the risk of violent behaviour in the present time and the R items represent risk management factors that may be influenced to reduce risk both in the present and in the future. By combining both static, historical variables with variables describing clinical state as well as risk management strategies, the

HCR-20 has in some studies been demonstrated as a useful predictor of violent behaviour in some studies (4, 131) yet other studies have shown only a modest prediction of reconviction (109). The mixed results may be due to difficulties in comparing studies, partially due to different study populations.

Finally, neurolaw is an emerging field in risk assessments, as it is an interdisciplinary study which explores how discoveries in neuroscience may have an impact on law and juridical standards. Findings in neuroradiology studies, such as changes in blood flow or cellular metabolism in specified parts of the brain, are hypothesised to be correlated to behaviour and in extension, criminal behaviour. Although connecting changes found in brain imagery to deviant behaviour requires a great leap in conclusions, this new field of science may affect how we perceive risk assessments and also, in some countries, how to interpret the question of criminal liability. In the longer run, this raises the question of whether biology determines all actions or if there is a free will and the freedom of choosing pro- or antisocial actions (132).

Findings of structural differences in the central nervous structures may be used as either mitigating or aggravating in courts of law, and as the linkage between these findings and behavioural changes are still unclear, using brain scans as evidence is enclosed by ethical dilemmas and should be made with great caution (133).
Aims

Main aim

The overall aim of this thesis is to give a detailed description of a total cohort of offenders sentenced to forensic psychiatric treatment in Sweden, to analyse the in-patient treatment time and to report on risk factors and predictors of persistent criminality in a lifetime perspective.

Specific aims

- (1) To identify the background and clinical characteristics of the cohort.
- (2) To investigate the relationship between risk assessments, prevalence of adverse events, and length of stay in forensic psychiatric care.
- (3) To explore risk factors for criminal recidivism and persistence over the life-span.
- (4) To investigate whether neuroimaging data gathered by SPECT gives added accuracy to predictions of recidivism in a long-term follow-up of the cohort.

Methods

Design and procedures

The current thesis consists of four papers which are all based on a longitudinal, follow-up, and follow-back clinical total cohort study called the UPPRÄTT-Malmö study, a Swedish acronym translating to the Forensic psychiatric follow-up studies – the Malmö cohort. This study is unique partially because it is a total cohort, including all individuals sentenced to forensic psychiatric treatment in a specific time frame within a specific region of Sweden, and partially because the registry data are extensive both in width and in the length of time it covers.

Paper I

This paper was conducted as a retrospective file and register study of the UPPRÄTT-Malmö study cohort focusing on the in-patient treatment period after the individuals had been sentenced to involuntary forensic psychiatric treatment. The cohort was followed from the time of the FPIs or FPSRs, through their time of in-patient care (including furlough) until discharge or at the latest until June 30, 2008. This date was chosen due to changes of legislation in Sweden in September 2008 which allowed compulsory outpatient care, and this would probably have effect on the lengths of stays of forensic psychiatric patients and made data interpretation difficult.

Paper I describes adverse events during forensic psychiatric care defined as absconding from staff, noncompliance with conditions for permission to move freely about or leave the hospital area, withdrawal of such permissions, substance use, criminal recidivism, suicide attempts, deaths and violence and threats during the hospital stay.

Paper II

In *Paper II*, the focus was on recidivism during the follow-up time of 0.6 to 9.7 years (1999-01-01 - 2008-12-31). The starting point in this paper is the same as in *Paper I* whereas the ending point is December 31, 2008. The median follow-up time was 6.2 years (range 0.6–9.7 years). Information on dates of new crimes and convictions, dates of legal force of new sentences as well as the following periods of sanctions was provided by the National Council of Crime Prevention and added to baseline data. Recidivism was

defined as crimes warranting a judicial decision and does therefore not include minor crimes warranting lesser sanctions such as fines.

Paper III

The third paper describes the cohort's criminality in a lifetime perspective. The National Council of Crime Prevention provided data of all convictions and all types of crimes from January 1, 1973, when they began keeping registries, until the end of the study's follow-up time, December 31, 2013. All 125 individuals were first included in the study, but seven individuals were omitted as data on lifetime criminality was missing and/or because they were deported from Sweden following the forensic psychiatric in-patient treatment.

The study identifies clusters of individuals in the cohort and compares these clusters on life-time criminality and adverse events during in-patient treatment.

Paper III has a broader definition of committed crimes compared to *Paper II*, as it includes sentences that received a judicial decision as well as milder sanctions such as fines. Data on adverse events during in-patient treatment time as well as length of time of in-patient treatment were the same as used in *Paper I*.

Paper IV

Paper IV investigates the predictive value of neuroimaging data in relation to recidivism in crime. Data from neuroimaging assessments conducted during the FPIs were added to the baseline data. Only individuals who underwent a full FPI were eligible for a neuroimaging assessment due to the longer length of the forensic psychiatric evaluation. Due to local changes in the FPI procedure at the time of the investigations, only 50 individuals in the cohort underwent the SPECT investigations. Out of these, six individuals were omitted from the study as there was a lack of certain background data necessary for the statistical analyses, thus, 44 individuals were included in the study of the fourth paper.

The time at risk was defined as the number of days counted from the individual's first day in in-patient treatment to either reconviction, date of death, date of deportation, or December 31, 2013.

A baseline model of eight traditional risk factors chosen on the basis of previous literature was created. An extended model was then constructed comprising of resting-state regional Cerebral Blood Flow (rCBF) from the neuroimaging measurements added to the baseline model of above-mentioned risk factors.

Data sources and data handling

Baseline data consisted of medical and social histories (including data on somatic and psychiatric health) that were collected retrospectively from the FPIs and the FPSRs. rCBF-data were collected and interpreted with help from the Department of Clinical Neurophysiology, Skåne University Hospital.

Data on sentences was extracted from the written decisions from district courts, courts of appeal, and county administrative courts. The collection was made by several members of the research team.

The National Council of Crime Prevention has provided information of the crimes committed by the cohort from the 1st January 1973 (when the registry was created) until the end-point of the study, December 31, 2013. This includes types of crimes, dates when crimes were committed and sanctions.

Data on times and causes of death were collected from the Causes of Death Register, provided by the National Board of Health and Welfare. Causes of death were coded according to the international version of the ICD-10 (126).

In writing *Paper III*, the same background data as in *Paper II* were used with added information on lifetime criminality gathered from the Swedish National Council for Crime Prevention. The latter data included dates and numbers of the relapses in crime described in *Paper II*, but when comparing the numbers of recidivists between the two publications, differences were found. In order to clarify the impact of these deviations, all follow-up data were scrutinised by the author of this thesis together with her supervisors and an erratum was sent to the publisher of *Paper II* (copy of the errata and correspondence with the Editor-in-Chief is found after *Paper II* in this thesis).

The reasons for the inconsistencies were found to be merging of files and using different codes for treatment episodes as well as individuals when gathering data. For five individuals, reconvictions were not reported, and in two known cases of recidivism, the crime categories were incorrect.

The study population

The UPPRÄTT-Malmö study consists of individuals belonging to the catchment area of the Malmö University Hospital (later Skåne University Hospital), which includes the districts of Malmö, Svedala, Trelleborg, and Vellinge. This region contains urban, smalltown, and countryside areas, which makes the cohort demographically representative. The entire area consisted at the end of 2005 of approximately 361,000 inhabitants.

A total of 127 individuals were sentenced to forensic psychiatric treatment in Malmö during the years of 1999-2005. The cohort includes all individuals sentenced to forensic

psychiatric care during that specific period of time. As no sample selection was made concerning variables such as the sex of the offender, type of crime(s) committed, or type of mental disorder(s), the cohort is considered heterogenic.

Two individuals were excluded from the study due to their legal conditions differing from the rest of the study group; one individual was sentenced to forensic psychiatric care twice during the study period and the other individual was first sentenced to forensic psychiatric care without SCS and shortly thereafter re-sentenced to forensic psychiatric care with SCS. The final cohort consisted thus of 125 individuals, 101 men and 24 women, aged 17 to 79 (median = 38) at the time of the FPI or the FPSR. The majority of the cohort had undergone an FPI (n = 97, 78 %), and a lesser proportion a FPSR (n = 28, 22 %).

Measures

Categorisation of crimes

This thesis reports on the nature and frequency of crimes over the life course, for which mentally disordered offenders in the cohort have been sentenced. In all papers of the thesis, crimes were divided as being either violent or non-violent. The definition of violent crime differs somewhat between the papers. *Papers I* and *II* are both reporting on the nature of the index crime but the definitions of violent crime differ; in *Paper I* it is defined as all forms of crimes against other persons, namely: murder and manslaughter (including attempts thereof), negligent homicide, assault, violence against an officer, violation of a woman's integrity, robbery, arson, and creating a danger to another. It also includes all sex crimes such as rape of adults and children, sexual coercion, sexual exploitation of an individual in dependence, sexual molestation of adults and children, and intercourse with an offspring. The definition of violent crime also includes all aggravated forms of the aforementioned crimes.

The definition of violent crime is widened in *Paper II* to also include unlawful threats against civilians as well as officers and violations against knife- and weapon laws. As the amount and thereby diversity of criminal behaviour multiplied due to lifetime data in *Paper III*, the definition of violent crime came to include the same crimes as in *Paper II* with addition of extortion, kidnapping, illegal restraint, unlawful coercion, violence against an officer, abuse of judicial procedure, violent resistance and rioting.

In *Paper III*, all crimes were categorised into six different categories based on the headings of the Swedish penal code as well as categories used at the National Council of Crime Prevention:

- 1. Violent crimes (see definition above)
- 2. Financial crimes

- 3. Theft or shoplifting
- 4. Traffic offences
- 5. Drug- or alcohol related crimes
- 6. Minor offences (most commonly damage to other people's property).

Clinical assessments

Axis I – Clinical disorders

The diagnostics in the FPIs and FPSRs were set according to the DSM-IV (59). The individuals in the cohort who underwent an FPI were assessed by a forensic psychiatric investigation team, consisting of a forensic psychiatrist, a forensic psychologist, a forensic social worker, and the staff of the treatment ward. The Axis I diagnoses were set by the senior consultant psychiatrist using semi-structured interviews when deemed necessary, generally with the help of the Structured Clinical Interview for Axis I disorders (SCID-I, (134)). The SCID-I is a semi-structured interview meant to be used by a trained clinician or mental health expert and it reviews, item-by-item, the symptoms as described by the DSM-IV. The interview is constructed to review both current illness (present during the past month before the interview) but also occurrence during the lifetime. This thesis reports the current presence of MMDs, but the investigative teams considered any previously known mental health problems when assessing the individuals. Information eligible to the FPI teams were in above all hospital records of previous admissions and contacts with health care prior to the FPI.

The individuals who underwent the FPSR were not assessed by the SCID-I interview. Instead, the investigating senior forensic psychiatrist made a clinical assessment based on previous health records and the symptoms presented during the interview. The diagnoses in the FPSRs were set according to the headings of the DSM-IV rather than in detail with subtypes of each diagnosis as is custom at an FPI. This reflected the limited time available for the investigating psychiatrist during the interview as the setting lacked postulates for fine-tuned diagnostics. The diagnoses in the FPIs were reported according to the numeral structure of DSM-IV. In order to pursue the statistical analyses in this thesis, the subtypes of diagnoses were collapsed into categories according to the headings of the DSM-IV.

Axis II – Personality disorders and intellectual disability

In order to assess the presence of PDs the Structured Clinical Interview for Axis II disorders (SCID-II) (135) was administered by the forensic psychologist during the FPIs. The interview for SCID-II is composed in the same manner as the interview for SCID-I. The final diagnosis of a personality disorder was based on both the SCID-II interview and background information available to the FPI team. The diagnoses were reported by the numeral structure of the DSM-IV.

The forensic psychiatrists conducting the FPSRs did consider the presence of a personality disorder, but a SCID-II interview was never conducted, and a formal specific personality disorder diagnosis was not set. Instead, the FPSRs reported the eventual presence of a personality disorder by grouping it in one of the three diagnostic clusters (A, B, or C) of the DSM-IV. Therefore, the personality disorder diagnoses found in the FPIs were collapsed into the three diagnostic clusters as well, in order to facilitate statistical analyses.

Assessment of cognitive functions

Psychometrical assessments were made on eligible individuals who underwent the FPIs. Due to the severity of the mental disorders the cohort were afflicted of, only a fraction (n = 58, 60 %) were able to participate in these assessments. The cognitive tests used in the assessments were Synonyms, Reasoning and Block tests (SRB) (136), Raven's Progressive Matrices (137) or Wechsler Adult Intelligence Scales-Revised (WAIS-R) (138).

rCBF and neuroimaging assessments

The neuroimaging measurements were carried out using ^{99m}Tc-exametazime (CeretecTM, Nycomed-Amersham/GE Healthcare) and a Ceraspect SPECT camera (Digital Scintigraphics Inc., Waltham, Massachusetts). The individuals were administered 900 MBq of ^{99m}Tc-exametazime in a cubital vein and after 15 minutes, when the ^{99m}Tc-exametazime has passed the blood-brain barrier and reached intracellular space in proportion to the rCBF, images were acquisitioned. The radiation from the ^{99m}Tc-exametazime was recorded in 180^o to allow a 3-dimensional reconstruction of the activity, proportional to the rCBF, after scatter and attenuation corrections, with a resolution of 9 mm full-width at half maximum.

When recorded, the three-dimensional activity was saved into a 128*128*64 voxel matrix and subdivided into 10 slices with 1 cm thickness, parallel to the orbitomeatal line. A region-of-interest set was scaled to fit the outer dimensions of the brain for threedimensional measurement of activity, proportional to the rCBF. The measured value in each region-of-interest was quantified, using Amersham region-of-interest software (GE Healthcare, Buckinghamshire, UK), in percent of the mean ^{99m}Tc-exametazime concentration in the whole brain.

Risk assessments

In this dissertation two types of risk measures were used; the HCR-20 (124) and the PCL Screening Version (PCL:SV, (139)). Out of the total cohort, only the individuals who had undergone a full FPI were rated with the HCR-20 (n = 95, 76 %) and PCL:SV (n = 96, 77 %). In most cases, this had been done at the time of the FPIs by the forensic teams. However, in 25 cases (20 %) the ratings were made retrospectively as they had not been done during the FPIs, alternatively, the ratings could not be found in the file registries. These retrospectively made ratings were based on the interviews and assessments made during the FPIs and on extensive file and register reviews in each case.

Previous studies have shown that it is possible to reliable assess psychopathy (140) and risk factors (141) from file based information.

HCR-20

In the studies of the current thesis, only the ten H- and the five C-items of the HCR-20 (124) were rated and included in the clinical background data. The R items, concerning individual treatment and management plans, had not been developed at the time of the FPIs. The H- and C-ratings were used in statistical calculations both independently as well as a total score. All items were rated on a three-point scale from 0 = not present to 2 = definitely present.

In 14 cases (11 %), when an item had been omitted in the HCR-20 by the forensic teams, the mean score was retrospectively calculated by the thesis author through valid mean substitution as suggested by Raaijmakers (142), by the thesis author and assigned to that item.

PCL:SV

In order to assess psychopathic personality traits, the Hare Psychopathy Checklist-Screening Version (PCL:SV) (139) was used. The instrument measures the interpersonal, emotional and behavioural aspects of the construct of psychopathy. It is a 12-item rating scale which highly correlates to the 20-item full version (123, 143). The items in the PCL:SV are rated from 0 to 2, where 0 = does not apply, 1 = may apply or in some respects applies and 2 = does apply.

In three cases (2 %), when no more than one item of the PCL:SV had been omitted by the forensic teams, a score was imputed according to the PCL:SV manual (139). In one case (1 %) two item ratings of the PCL:SV were missing and it was therefore omitted.

Analytical methods

In this thesis all analyses have been conducted on anonymised data. In *Papers I* to *III* the comparisons of distributions have been analysed using either χ^2 test for dichotomous variables or Mann-Whitney *U* test for continuous data. Analyses of dichotomous variables are reported either by using Pearson χ^2 or Fisher exact χ^2 test (the latter when any cell count was less than five). As for the reason for choosing non-parametric tests, the data distributions were in most variables skewed and therefore required the aforementioned tests. All *p*-values were two-tailed, and the statistically significant level was set to p < .05.

In *Papers II* and *III*, effect sizes were presented as φ scores for the Fisher exact χ^2 test and as r-scores for the Mann Whitney U tests. Following Cohen's model, effect sizes of 0.10 are small, 0.30 a medium effect, and 0.50 a large effect. As for effect sizes of r, Cohen's model states that 0.10 is a small effect, 0.30 a medium effect, and 0.50 a large effect (144).

Data were analysed using SPSS software versions 20.0 and 22.0 for *Papers I* and *II*, version 25.0 for *Paper III*, whereas the analyses in *Paper IV* were made using R statistical programming language (145).

Paper I

In this study, the cohort was divided into two groups: those receiving forensic psychiatric treatment with SCS and those without SCS. Thereafter χ^2 and Mann Whitney *U* tests were used to evaluate differences in these two groups in reference to baseline data and data from the in-patient treatment time.

In order to estimate the median length of stay in hospital and to compare the time to discharge (survival distributions) for the patients treated with or without SCS, survival analyses (The Kaplan-Meier method) and the log-rank tests were used.

To predict length of stay, a Cox proportional hazards regression analysis with timedependent covariates was conducted. In the first step, univariate regression models were established separately for each included covariate in order to identify variables with predictive properties (defined as p <.20). The singled-out variables from the univariate analyses were then entered into a stepwise Cox proportional hazards multivariate regression model. In the final model, measures of relative risk were presented as hazard ratios with p <.05, CI 95 %.

Paper II

Paper II aimed to extract predictive variables of relapse in criminality, especially violent criminality, wherefore statistical group comparisons were made on recidivists versus non-recidivists. All variables that reached a significance level of p < .30 were entered into the Cox regression model, out of which the variable with highest *p*-score was excluded after each analysis until all remaining predictors had a *p*-value of <.05.

In order to test for multicollinearity, a linear regression model was used as to exclude covariates with a VIF > 3.0.

Time at risk was calculated as beginning at the date of intake to the forensic psychiatric hospital until reconviction, date of death, or December 31, 2008, when the follow-up period ended.

Paper III

Ward's method was used to identify the relevant number of clusters in a hierarchical cluster analysis. Dichotomous background variables were entered in to the hierarchical cluster analysis and then one insignificant variable was excluded at a time in a stepwise manner, starting with the one with the highest p-values until all remaining variables had a p < .05.

Preliminary assumption testing was conducted to check for normality, linearity, univariate and multivariate outliers, and homogeneity of variance-covariance matrices. To

test for multicollinearity, a linear regression model was used to exclude covariates with a VIF >3.0.

Since the variables exhibited a non-linear relationship, all continuous data were transformed in accordance with the natural logarithm and as such used in the subsequent analyses. One-way between-group multivariate analyses of variance (MANOVA) were performed to investigate cluster differences in lifetime criminality variables and adverse events during in-patient treatment time. A one-way multivariate analysis of covariance (MANCOVA) was performed as an extension of the MANOVA of adverse events during in-patient treatment, where length of stay was used as the covariate.

Paper IV

In *Paper IV* Barnard's test was used for dichotomous variables as the test is more powerful compared to Fisher's test when sample sizes are small (146). Welch's *t*-test was used for numerical variables instead of Student's *t*-test as it performs better in situations where sample size and variance are unequal between groups (147).

In order to predict recidivism a random forest classification (RF) was used as it is an algorithm with high accuracy, performs well with both small sample sizes and high-dimensional data and it has been previously used successfully to predict recidivism in forensic psychiatric cohorts. In the prediction analysis, each RF model was created using 10 000 trees, using the default of \sqrt{p} predictors at each node with p being the total number of predictors available. In order to prevent class imbalance, the majority class (i.e., non-recidivists) was down-sampled to ensure that each bootstrap sample contained approximately the same number of recidivists as non-recidivists. The RF estimates individual variable importance as well.

To assess predictive performance, several metrics were used. The area under the receiver operating characteristic curve (AUC) was calculated as it represented the probability that a randomly selected recidivist would have been predicted by the model as having a higher probability of recidivism than a randomly selected non-recidivist. The value of the AUC was then corresponded to Cohen's *d*, in order to interpret effect size.

The individual variable importance was estimated through random permutations of each variable and recording how it affected classification accuracy. If a variable was important for the models, a permutation would result in a large decrease in classification accuracy. *Paper IV* reports the scaled mean decrease in accuracy of each variable.

Paper IV also reports accuracy (the proportion of recidivists correctly classified as such), specificity (the proportion of non-recidivists correctly classified as such), positive predictive value (PPV; the correct number of predicted recidivists who are recidivists) and negative predictive value (NPV; the correct number of predicted non-recidivists that are non-recidivists).

Ethical considerations

The studies in this thesis are designed in such a manner that it is not possible to identify any single individual. In order to protect the confidentiality, all data were anonymised using coded files. The key code was kept in a fire proof security locker at the Forensic Psychiatric Hospital in Malmö and later Trelleborg, separate from the study material and database.

The baseline data collection was approved by the regional ethical board in Lund (64/2007). The register-based follow-up was approved both by the aforementioned ethical board as well as one in 2014 (2014/911). As this was a retrospective register study, contacting the study subjects and receiving their informed consent was not considered necessary, and due to the length of time passed since they finished treatment, such contact could have posed a risk to their health and well-being. Upon request by the ethical board we announced our planned registered-based follow-up in the two largest newspapers in the Malmö and Gothenburg area in the beginning of 2015.

Medical research studies on human beings are enclosed by strict ethical frameworks, some of which are stern whereas others are more malleable. The norm is that medical research ethics have to balance the benefits and burdens and that harm and risk to the subjects of research should be avoided at great length (148). To avoid conducting research on forensic psychiatric patients without their consent could be argued to be the most ethical choice, but this has to be weighed against not using the continuously collected registry data to the advantage of the patients.

Forensic psychiatric patients are considered particularly vulnerable as they are placed involuntarily in institutions, dependent on the medical staff and because the mental health problems may have a negative effect on their ability to care for themselves and protect their interests. Research on this particular group requires that the ethical considerations of potential costs and health benefits of scientific research are thoroughly weighted.

The subjects in this particular study were considered vulnerable due to their legal status and the mental disorders they were suffering from, which was considered when the study was designed.

The scientific gain of expanding the knowledge of lifetime violent criminality in a population of forensic psychiatric patients is considered of great importance. By investigating factors important for length of stay, predictors of recidivism in crime as well as identifying background variables of importance for lifelong criminal behaviour, this study may be of help in shortening in-patient treatment time for those who no longer show symptoms of mental disorders and also, to steer resources and efforts to patient groups in greater need. Reducing time in involuntary psychiatric treatment when treatment is no longer necessary is in concordance with the Declaration of Hawaii, which states that psychiatric treatment must not be given in the absence of psychiatric illness (149).

Results

Background and clinical characteristics

In *Papers I* and *II* the background and clinical characteristics of the cohort were described based on information gathered from the FPIs and FPSRs, (Tables 1 and 2).

Approximately half of the cohort (n = 64, 51%) had a migration background, defined as either having one or both parents from another country than Sweden or as having migrated to Sweden themselves. Almost one third of the cohort (n = 36, 29%) had a firstdegree relative with a mental disorder of some kind and 34 individuals (32%) had been in contact with child- and adolescent psychiatric care when young. Twenty-four individuals (23%) had been in custody, foster homes or institutions before the age of 18 and twelve individuals (10%) had not finished primary school (Table 1). At the time of the FPI/FPSR, the majority of the cohort had no occupation (n = 115, 94%), and a little more than half of the cohort had no permanent housing (n = 64, 54%).

A majority of the index crimes (defined as the crimes that had led to the current sentence of forensic psychiatric treatment) were of violent nature (n=105, 84 %) and two thirds of the cohort (n=84, 67 %) received forensic psychiatric treatment with SCS.

As can be seen in Table 2, a majority of the cohort 58 % (n = 72) had more than one Axis I mental disorder and 27 % (n = 34) had a personality disorder diagnosed (most commonly in Cluster B, n = 20, 16 %). The most common Axis I disorder was a psychotic disorder of some kind (n = 91, 73 %), second most common was a SUD (n = 60, 48 %).

By the end of the study (2013-12-31), 27 (22 %) individuals had died. Median age at the time of death was 53 years (range 25–80 years). Sixteen individuals (13 %) died from somatic illness, five (4 %) from substance abuse (intoxications), five (4 %) from suicide, and one (1 %) as a victim of violent crime.

	Total Cohort	Relapse to general crime	ç	Relapse in violent crime	ę
	n = 125	n = 35, 28%	÷	n = 21, 17%	÷
Background characteristics:	n (%)	n (%)		n (%)	
Sentence with SCS	84 (67)	13 (37)***	-0.399	8 (38)**	-0.279
Male sex	101 (81)	28 (80)	-0.013	20 (95) ^a	0.165
Migration background	64 (51)	20 (57)	0.074	12 (57)	0.053
Mental disorder in 1st degree relative	36 (29)	11 (31)	0.036	8 (38)	0.092
Previous contact with Child and Adolescent Psychiatry $(n = 107)$	34 (32)	8 (23)	-0.023	$4 (19)^a$	-0.042
Custody, foster home or institutional care before age of 18 $(n = 105)$	24 (23)	8 (23)	0.108	5 (24)	0.085
Low educational attainment $(n = 117)$	12 (10)	8 (23) ^{a**}	-0.298	5 (24)**	-0.246
No occupation $(n = 123)$	115 (94)	$33 \ (94)^a$	0.089	20 (95) ^a	0.116
Poor financial situation (large debts or no monthly income), $(n = 118)$	27 (23)	13 (37)*	0.233	7 (33)	0.130
No permanent housing $(n = 123)$	64 (52)	19 (54)	0.028	12 (57)	0.046
Marital status single $(n = 121)$	91 (75)	28 (80)	0.071	16 (76)	0.010
Any previous sentences $(n = 120)$	78 (65)	27 (77)*	0.19	15 (71)	0.094
Previous violent criminality $(n = 122)$	41 (34)	16 (46)*	0.192	11 (52)*	0.201

Table 1. Background characteristics of the cohort; comparisons between total cohort and subgroups

^aFisher Exact test. ^bMann-Whitney U test. ^{***} p<.001, ^{**} p<.01, ^{*} p<.05.

Age at FPI/FPSR, median, <i>range</i> $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $38 (17-79)$ $24 (17-79)$ $38 (17-79)$ $24 (17-79)$ </th <th>38 (17-79) 28 (15-78)</th> <th></th> <th>φ</th> <th>n = 21, 17%</th> <th>φ</th>	38 (17-79) 28 (15-78)		φ	n = 21, 17%	φ
Age at first sentenced crime ($n = 112$), median ($range$)28 (15-78)24 (15-78)Diagnosis according to DSM-IV (59), n (%)SUD60 (48)21 (()DSM-IV (59), n (%)Psychotic disorder91 (73)24 (1)DSM-IV (59), n (%)Psychotic disorder15 (12)4 (1)Mood disorder13 (10)5 (1)4 (1)Anxiery disorder or OCD13 (10)5 (1)NDDPersonality disorder, any34 (27)16 (4)Personality disorder Cluster A6 (5)3 (5)Personality disorder Cluster B20 (16)12 (3)Personality disorder Cluster C2 (2)0 (0)Personality disorder NOS9 (7)3 (5)Q ($n = 58$), Median ($nage$)100 ($38-137$)100 (60 GAF at the time of the FPI / FPSR ($n = 124$), median ($nange$)35 ($10-65$)34 (10 PCL:SV (139), Total score ($n = 96$), Median ($nange$)11 ($n-220$ 12 (0 HCR-20 (124), Total score ($n = 95$), Median ($nange$)18 ($n-28$)20 (0	28 (15-78)	38 (22-48) ^b		38 (22-48) ^b	
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$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	60 (48)	21 (60)	0.150	15 (71)*	0.211
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Anxiety disorder or OCD13 (10)5 (1)NDD11 (9)4 (1)NDDPersonality disorder, any34 (27)4 (1)Personality disorder Cluster A6 (5)3 (5)Personality disorder Cluster B20 (16)12 (3)Personality disorder Cluster C2 (2)0 (0)Personality disorder NOS9 (7)3 (5)Personality disorder NOS9 (7)3 (5)IQ $(n = 58)$, Median (nange)100 (38-137)100 (60)GAF at the time of the FPI / FPSR $(n = 124)$, median (nange)35 (10-65)34 (10)PCL:SV (139), Total score $(n = 96)$, Median (nange)11 ($n-22$)12 ($n-28$)HCR-20 (124), Total score $(n = 95)$, Median (nange)18 ($n-28$)20 ($n-28$)	15 (12)	$4 (11)^{a}$	-0.011	$(14)^a$	0.032
NDD11 (9)4 (1)Personality disorder, any $34 (27)$ $16 (4)$ Personality disorder Cluster A $6 (5)$ $3 (2)$ Personality disorder Cluster B $20 (16)$ $12 (3)$ Personality disorder Cluster C $2 (2)$ $0 (0)$ Personality disorder NOS $9 (7)$ $3 (5)$ Personality disorder NOS $100 (38-137)$ $100 (60)$ Personality disorder NOS $100 (38-137)$ $100 (50)$ Personal the time of the FPI / FPSR ($n = 124$), median ($nange$) $35 (10-65)$ $34 (10)$ Personal the time of the PPI / FPSR ($n = 95$), Median ($nange$) $11 (0-22)$ $12 (0)$ Personal to the soure ($n = 95$), Median ($nange$) $18 (0-28)$ $20 (0)$	13 (10)	5 (14)	0.079	$2 (10)^{a}$	-0.013
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Personality disorder Cluster A6 (5)3 (9Personality disorder Cluster B20 (16)12 (3)Personality disorder Cluster C2 (2)0 (0Personality disorder NOS9 (7)3 (5)IQ $(n = 58)$, Median $(narge)$ 100 (38-137)100 (60GAF at the time of the FPI / FPSR $(n = 124)$, median $(narge)$ 35 (10-65)34 (10PCL:SV (139), Total score $(n = 96)$, Median $(narge)$ 11 (0-22)12 (0)HCR-20 (124), Total score $(n = 95)$, Median $(narge)$ 18 (0-28)20 (0)	34 (27)	16 (46)**	0.259	12 (57)**	0.302
Personality disorder Cluster B20 (16)12 (3)Personality disorder Cluster C2 (2)0 (0Personality disorder NOS9 (7)3 (5)IQ $(n = 58)$, Median $(range)$ 100 (38-137)100 (60GAF at the time of the FPI / FPSR $(n = 124)$, median $(range)$ 35 (10-65)34 (10PCL:SV (139), Total score $(n = 96)$, Median $(range)$ 11 (0-22)12 (0)HCR-20 (124), Total score $(n = 95)$, Median $(range)$ 18 (0-28)20 (0)	6 (5)	$(3)^a$	0.110	$3 (14)^{a}$	0.199
Personality disorder Cluster C 2 2) 0 (0 Personality disorder NOS 9 (7) 3 (9 IQ $(n = 58)$, Median (<i>range</i>) 100 (38-137) 100 (60 GAF at the time of the FPI / FPSR ($n = 124$), median (<i>range</i>) 35 (10-65) 34 (10 PCL:SV (139), Total score ($n = 96$), Median (<i>range</i>) 11 (0-22) 12 (0 HCR-20 (124), Total score ($n = 95$), Median (<i>range</i>) 18 (0-28) 20 (0	20 (16)	12 (34)**	0.311	8 (38)**	0.271
Personality disorder NOS 9 (7) 3 (9 IQ $(n = 58)$, Median $(nange)$ 100 $(38-137)$ 100 $(60$ GAF at the time of the FPI / FPSR $(n = 124)$, median $(nange)$ 35 $(10-65)$ 34 (10) PCL:SV (139), Total score $(n = 96)$, Median $(nange)$ 11 $(0-22)$ 12 (0) HCR-20 (124) , Total score $(n = 95)$, Median $(nange)$ 18 $(0-28)$ 20 (0)	2 (2)	$0 (0)^a$	-0.080	$0 (0)^a$	-0.057
IQ $(n = 58)$, Median $(range)$ 100 $(38-137)$ 100 $(60$ GAF at the time of the FPI / FPSR $(n = 124)$, median $(range)$ 35 $(10-65)$ 34 (10) PCL:SV (139), Total score $(n = 96)$, Median $(range)$ 11 $(0-22)$ 12 (0) HCR-20 (124) , Total score $(n = 95)$, Median $(range)$ 18 $(0-28)$ 20 (0)	9 (7)	$(9)^a$	0.033	$3 (14)^a$	0.123
GAF at the time of the FPI / FPSR $(n = 124)$, median $(range)$ $35 (10-65)$ $34 (10$ PCL:SV (139), Total score $(n = 96)$, Median $(range)$ $11 (0-22)$ $12 (0-12)$ HCR-20 (124), Total score $(n = 95)$, Median $(range)$ $18 (0-28)$ $20 (0-2)$	100 (38-137)	100 (<i>60-127</i>) ^b		104 <i>(60-127</i>) ^b	
PCL:SV (139), Total score $(n = 96)$, Median $(range)$ 11 (0-22) 12 (0-12) HCR-20 (124), Total score $(n = 95)$, Median $(range)$ 18 (0-28) 20 (0-12)	nge) 35 (10-65)	34 (10-51) ^b		35 (10-40) ^b	
HCR-20 (124), Total score (n = 95), Median (range) 18 (0-28) 20 (0-	11 (0-22)	12 (<i>0-22</i>) ^b		12 (<i>0-20</i>) ^b	
	18 (0-28)	20 (<i>0-28</i>) ^b		20 $(0-26)^{b}$	
HCR-20, Historical variables $(n = 95)$, Median $(range)$ 12 $(0-19)$ 14 $(0-14)$	12 (0-19)	$14 \; (0-I8)^{\rm b}$		14 (0-17) ^b	
HCR-20, Clinical variables ($n = 96$), Median (<i>range</i>) 7 (0	7 (0-9)	7 (0-10) ^b		7 (0-9) ^b	

Risk assessments, length of stay, and adverse events

In *Paper I* the length of stay of the cohort was investigated with focus on the prevalence of adverse events and the length of stay with comparisons made between individuals receiving care with and without SCS (n = 119, missing data on 6 individuals). At the endpoint of the study, 89 (71 %) individuals had left the clinic, leaving 36 patients (29 %) still in treatment. The median length of stay of the whole cohort was 951 days (2.61 years). Through a Kaplan-Meier analysis, individuals sentenced to forensic psychiatric treatment with SCS were shown to have had a significantly longer length of stay (1272 days or 3.48 years) compared to those receiving treatment without SCS (273 days or 0.75 years; p <.001; see Figure 1).



Figure 1. Length of stay for patients with and without SCS.

Seventy-one individuals (60 %) of the cohort were involved in some form of adverse event and there was a total of 624 adverse events in the cohort during treatment. It was significantly more common with adverse events in general in the group of individuals receiving treatment with SCS compared to those without (p < .05), and the difference was continuing to be significant when the events were further divided in to threats (p < .05) and violence (p < .05). No significant differences were found concerning absconding or

substance use. Men were also more prone to be involved in adverse events compared to women (p < .05). In essence, the SCS group was overrepresented in adverse events compared to the non-SCS group, yet when controlling for the length of stay by dividing the number of adverse events by the number of days in treatment, only the combined number of threats and violent events remained significantly different between the two groups (p < .01; see Table 3).

The individuals who were involved in one or more adverse events had a significantly longer length of stay (median 1206 days or 3.3 years) compared to the 48 individuals (40%) who had not been involved in any such events (median 471 days or 1.29 years) ($p \le .001$).

Variables	Total	With SCS	Without SCS	Group
	<i>n</i> =119	<i>n</i> =82	<i>n</i> =37	comparison
	Mean±SD	Mean±SD	Mean±SD	P
All adverse events	0.483±0.654	0.481±0.576	0.490±0.808	.218
Absconding	0.122±0.230	0.122±0.204	0.122±0.284	.158
Substance abuse	0.253±0.406	0.224±0.349	0.316±0.508	.992
Threats or violent events	0.104±0.219	0.128±0.215	0.049±0.221	.007

A stepwise Cox regression analysis was conducted to predict length of treatment. Variables which reached p <.20 in univariate analyses were entered: female sex, educational level, being married or cohabitant before conviction, being a parent, violent index crime, global assessment of functioning (GAF)-score at the time of the FPI, age at first sentenced crime, number of earlier forensic psychiatric treatment episodes, psychotic disorder, mood disorder, SUD, personality disorder, absconding events, substance use events (both as a dichotomous variable and as a continuous variable), threats or violent events (both as a dichotomous variable and as a continuous variable) and reconviction during treatment. The strongest predictive model showed that the adverse event of absconding (p <.05) and a current conviction of a violent index crime (p <.01) were both significant risk factors that predicted a longer hospital stay where as a diagnosis of a mood disorder (p <.01) emerged as a factor that reduced the in-patient treatment time.

Predictors of recidivism¹

In *paper II* the cohort was followed up from the beginning of forensic psychiatric in-patient treatment time until the end point of this first follow-up, December 31, 2008. A total of 35 individuals (28 %) relapsed in crime during follow-up and a partition of them, 21 individuals (17 %), relapsed in violent crime. The paper presents predictors of both general and violent recidivism by Cox regression analyses (see Table 5).

The background factors of having a first-degree relative with a major mental disorder (p < .05), a low educational attainment (defined as not having finished primary school) (p < .001) and a low age at first criminal offence (p < .001) were all positive predictors of recidivism in any kind of crime. If the forensic psychiatric treatment was combined with SCS, the risk of recidivism in any type of crime was lowered (p < .001).

Recidivism in violent crime was predicted by having a Cluster B personality disorder (p<.05) and having a low GAF (Global Assessment of Functioning)-scale at the time of the FPI (p <.05). As with recidivism in any crime, forensic psychiatric treatment with SCS was negatively predictive of violent recidivism (p <.05).

Continuing the follow-up - recidivism data 2009-2013

When *Paper III* was prepared, data on recidivism following the end point of *Paper II* was gathered and analysed as a part of the description of lifetime criminality. Data were missing for seven individuals. In Table 4 follow-up data on recidivism is presented. Another nine individuals (8 %) relapsed during the added follow-up period 2009-01-01 – 2013-12-31, making it 44 individuals (37 %) in total who relapsed after the index crime. Twenty-one individuals (17 %) relapsed in violent crime during the first follow -up and another six during the second, making it 27 individuals (23 %) who relapsed in violent crime during 15 years following beginning of forensic psychiatric treatment.

Out of those 27 who relapsed in violent crime during 2009 to 2013, six individuals (5 %) relapsed in more serious violent crimes (namely one count of arson, one count of robbery and four counts of sexual crimes against adults and minors).

¹ In 2019, when preparing the manuscript for *Paper III* which included data from *Paper II*, inconsistencies between new follow-up data and those used in *Paper II* were found. The revised results are presented here; for further details the reader is referred to the corrigendum printed after *Paper II* in this thesis.

Table 4. Recidivism in crime during 15 years of follow-up

		Follow-uj	p periods	Total follow-up time
		1999-2008	2009-2013	(1999-2013)
		(n = 35)	(n = 28)	(n = 44)
Number of minut	Violent crimes (<i>range</i>)	63 (0-8)	31 (0-5)	94 (0-8)
Number of crimes	Non-violent crimes (<i>range</i>)	194 (0-22)	161 (<i>0-31</i>)	355 (0-31)
Total number of cri	mes (range)	257 (0-22)	192 (0-31)	449 (0-31)

n = number of individuals who relapsed in crime during specified time period.

		Relapse	: in general crime			Relapse	in violent crime	
	Univariate association		Cox regression		Univariate association		Cox regression	
Variables	þ	В	Hazard ratio (95% CI)	d	þ	В	Hazard ratio (95% CI)	þ
CS	000.	-3.086	0.046 (0.016-0.126)	.000	.002	-1.383	0.250 (0.093-0.670)	.006
Aale sex	.887				.074			
ow educational attainment	.003	-2.022	$0.132\ (0.049-0.360)$.000	.008			
oor financial situation	.012				.157			
rge at first sentenced crime	.014	-0.105	0.900(0.858-0.945)	.000	.115			
Aental disorder in first-degree elative	.686	0.877	2.403 (1.015-5.687)	.046	.302			
revious sentences	.037				.304			
revious violent criminality	.034			-	.027			
UD	.094				.018			
unxiety disorder / OCD	.514				1.000			
ersonality disorder, Cluster B	.001				.002	1.132	3.101 (1.123-8.567)	.029
AF at the time of the FPI/FPSR	.061				.192	-0.081	0.922 (0.862-0.987)	.020
CL:SV, Total score	.113				.115			
HCR-20, Historical variables	.220				.416			

Table 5. Cox regression analysis of predictors of crime in general and violent crim

Psychiatric Survey Report; PCL:SV: Hare Psychopathy Checklist: Screening Version; HCR-20: Historical Clinical Risk Management-20. ^aFisher Exact test. ^bMann-Whitney U test. *** p<.001, ** p<.01, * p<.05.

Patterns of lifetime criminality in mentally disordered offenders

In Paper III, seven individuals were omitted due to missing data (n = 2) or because they were deported from Sweden following the forensic psychiatric in-patient treatment (n = 5). The total number of crimes committed by the UPPRÄTT-cohort between January 1, 1973 until December 31, 2013 was 3381 (median 16.5, range 1-185). Of these, 936 were violent crimes (range 0–45, median 5.5). The median age at first registered committed crime was 20 years (range 15-72 years) and for the first violent crime 27 years (range 15–72 years).

Eight individuals (7 %) had not committed any violent crimes. Another eight individuals had been convicted of some form of lethal violent crime, 14 individuals (12 %) for arson and ten individuals (9 %) for sexual crimes.

Cluster analyses

93 individuals were eligible for cluster analyses and two clusters were identified based on background and clinical data (see Figure 2).

Cluster 1 consisted of slightly more than a quarter of the cohort (n = 26, 28 %); Cluster 2 consisted of 67 individuals (72 %). The individuals in the first cluster were more likely to have experienced childhood adversities (not having finished primary school, having contact with child and adolescent psychiatry, being placed in custody or a youth institution before the age of 18, and having a first-degree relative with an SUD). They were more likely already to have an NDD or later in life to develop an SUD.



*** *p*<.001, ** *p*<.01, * *p*<.05.

Figure 2. Variables in the cluster analyses

Comparisons of lifetime criminality between clusters

There was a statistically significant difference between clusters 1 and 2 on the combined dependent variables, F(7, 85) = 2.42, p = .026; Wilks' Lambda = 0.834; partial eta squared = 0.166, see Figure 3.

When the results of the dependent variables were considered separately, three variables reached statistical significance using an alpha level of 05; lifetime number of financial crimes, F (1, 91) = 12.03, p = .001, partial eta squared = 0.117, number of alcohol- or drug-related crimes, F (1, 91) = 8.82, p = .004, partial eta squared = 0.088, and age at first crime, F (1, 91) = 4.53, p = .036, partial eta squared = 0.047. An inspection of the median scores indicated that individuals of Cluster 1 were more often convicted of financial crimes (median [M] = 3.00, *standard deviation* [SD] = 3.44 vs. M = 0, SD = 1.92), and of alcohol or drug-related crimes (M = 1.50, SD = 7.30 vs. M = 0, SD = 3.44). The individuals in Cluster 1 were also younger at first crime conviction (M = 17.50, SD = 6.69 vs. M = 22, SD = 13.29).

Comparisons between clusters, adverse events, and in-patient treatment time

There was a statistically significant difference between clusters 1 and 2 on the combined dependent variables, F(4, 88) = 2.57, p = .044., Wilks' Lambda = 0.896; partial eta squared = 0.10, see Figure 4. When the results for the dependent variables were considered separately, the only difference to reach statistical significance using an alpha level of .05, was number of events of substance use during in-patient treatment time, F(1, 91) = 7.36, p = .008, partial eta squared 0.075, where individuals in Cluster 1 were more often involved in using drugs or alcohol during in-patient treatment time (M = 3, SD = 5.07 vs. M = 0, SD = 4.50).

When length of stay was added as a covariate, there was no statistically significant difference between the clusters on the combined dependent variables after controlling for the length of in-patient treatment time F(4, 85) = 1.78, p = .106, partial eta squared 0.085.



***p<.001, ** p<.01, * p<.05

Figure 3 Age at first crime and number of crimes in a lifetime, z-scores



*** *p* < .001, ** *p* < .01, * *p* < .05

Figure 4. Number of adverse events during in-patient treatment time, z-scores

Incremental effects of neuroimaging data on prediction of recidivism

rCBF measurements

When studying the variations in cerebral blood flow between recidivists and non-recidivists in *Paper IV*, the individuals in the recidivist group had a statistically significantly lower bilateral parietal lobe and right cerebellar lobe rCBF compared to non-recidivists. There were statistically significant associations between age at FPI and right parietal lobe rCBF (positive association, $\rho = .31$, p = .039) and left frontal lobe rCBF (negative association, $\rho = .35$, p = .02).

As neuroimaging data may be corrupted by psychotropic medication, the statistical analysis of the extended model was conducted with added information of pharmacological data to rule out any potential disturbance effect. This did not, however, give a significant change in the predictive performance; the AUC remained the same at 0.83 and with a slightly increased accuracy from 0.81 to 0.82 (95 % confidence interval [CI]: .67,.92).

The traditional (baseline) prediction model

In the partial dependence plots the risk factors of a cluster B personality disorder, younger age at first crime as well as at the FPI and SUD increased the probability of being classified as a recidivist (see Figure 5).

The predictive performance of the traditional model was modest as the AUC was .69, with a scaled Brier score of .08 and an accuracy of .64 (95% CI: .48,.78), and sensitivity =.63, specificity =.64, PPV =.50, and NPV =.75.

Baseline model



The extended prediction model

When neuroimaging data was added to the traditional prediction model of recidivism in crime to form the extended model, the predictive abilities increased all over. The AUC was .81 with a scaled Brier score of .25 and an accuracy of .82 (95% CI: .67,.92]), and sensitivity =.75, specificity =.86, PPV =.73, and NPV =.86. The variables in the extended model that increased the likelihood of being classified as a recidivist were lower right and left parietal lobe rCBF, Cluster B personality disorder, and age at first crime.

When the extended model was compared with the traditional model it led to an increase of the AUC by 17 %, over 200 % increase in scaled Brier score, and a 28 % increase in accuracy. In numeral terms, the extended model managed to classify two additional recidivists and six non-recidivists compared with the traditional model, in total 12 out of 16 recidivists and 24 out of 28 non-recidivists.

Summary of main findings

- 1. Approximately half of the cohort (n = 64, 51 %) had a migration background, defined as either having one or both parents from another country than Sweden or as having migrated to Sweden themselves and 34 individuals (32 %) had been in contact with child- and adolescent psychiatric care when young. Twenty-four individuals (23 %) had been in custody, foster homes or institutions before the age of 18 and twelve individuals (10 %) had not finished primary school. A majority of the index crimes (defined as the crimes that had led to the current sentence of forensic psychiatric treatment) were of violent nature (n=105, 84 %) and two thirds of the cohort (n = 84, 67 %) received forensic psychiatric treatment with SCS. The majority suffered from psychotic disorders (n = 91, 73 %) (*Papers I* and *II*).
- 2. The length of stay in forensic psychiatric in-patient treatment differed significantly between those sentenced to forensic psychiatric care with SCS and those without SCS. The former group had a median length of time which was almost five times longer compared to the latter, yet the clinical burden of SMDs was the same between the groups. Previous contact with child and adolescence psychiatric services, violent index crime, psychotic disorders, a history of substance use and absconding during treatment predicted longer length of stay. The majority of the cohort (60 %) was involved in some form of adverse events (violence, threats, substance abuse, or absconding) during the in-patient treatment period. Those who received care with SCS were more often involved in violent and threat events (*Paper I*).
- 3. Thirty-five individuals (28 %) relapsed in any type of crime during the first followup period and 21 (17 %) relapsed in violent crime specifically. Relapse in general crime was predicted by low educational attainment, mental disorder in a firstdegree relative, and low age at first sentenced crime. Relapse in violent crime was predicted by Cluster B personality disorder and a low GAF score at the time of the FPI. Being sentenced to forensic psychiatric treatment with SCS was a reducing risk factor of relapse in crime in general as well as violent crime (*Paper II*). The cohort had during their lifetime, that is, the years 1973 to 2013, committed a total of 3381 crimes of which 935 were violent. Two clusters were identified; the smaller Cluster 1 consisted of slightly less than one-third of the cohort and these individuals were more likely to have experienced childhood adversities as well as being diagnosed with a NDD and SUD. Individuals in Cluster 1 were significantly

more often involved in financial and alcohol/drug-related crimes and they were also of a younger age when first sentenced (*Paper III*).

4. Including neuroimaging data to models with previously empirically known risk factors for recidivism, increased the area under the receiver operating characteristic curve (AUC) from .69 to .81, increased the accuracy from .64 to .82 and increased the scaled Brier score from .08 to .25, which all together supports that neuroimaging data may contribute with improved accuracy in recidivism prediction models (Paper IV).

Discussion

Comments on main findings

Comments on aim 1

From an international forensic psychiatric point of view, the UPPRÄTT-Malmö cohort is diagnostically relatively typical as a majority were diagnosed with psychotic disorders and to a lesser extent affective disorders (76, 77). Co-morbidities with PDs and SUD were prevalent. There was a surprisingly low prevalence of neurodevelopmental diagnoses such as ADHD and ASDs (n = 11, 9 %), as previous studies have found these disorders to be overrepresented in forensic populations (150-152). The later years' allocation of financial means towards research and clinical resources assessing and treating ASD have caused an increased prevalence of registered and clinically diagnosed cases of ASD in the community at large, yet this is not to be confused with an actual increase of the prevalence of autism spectrum disorder (153). The increased focus has instead led to a higher rate of identification of the disorder and it is increasingly common that forensic psychiatric patients are assessed and diagnosed as well as an existing NDD requires added regard in forensic psychiatric treatment.

Comments on aim 2

The difference in length of stay between those who received treatment with SCS and those who did not was notable; the former group had an almost five times longer treatment time compared to the latter. As both groups were equally afflicted by severe psychiatric disorders and in need of mental health care, burden of illness and symptom load could not explain the prolonged treatment time: this has been replicated in later studies of forensic patients' length of stay (77). Instead, the results of *Paper I* indicate that the longer stay could be explained as an attempt by the legal system to prevent new criminality, especially as violent index crimes and adverse events of absconding, but no specific diagnoses, were predictive of longer in-patient treatment times.

In international literature, the prolonged time to discharge for individuals sanctioned under a restriction order is in part a result of more severe crimes committed together with a higher degree of complexity and risk involved in their care (154-156). Another possible reason for longer lengths of stay may be a lack of appropriate supported housing. These specialised housings may be necessary in order to securely discharge forensic psychiatric patients into a risk reducing environment.

A prolonged length of time in treatment is troublesome in multiple ways; in-patient treatment time is costly and a prolonged length of stay at a forensic psychiatric hospital will draw funds from other medical treatments and facilities and from other patients seeking care from the council caregivers. Studies have also shown that long-term inpatients with schizophrenia show lower health-related quality of life compared to matched outpatient controls, suggesting that institutionalisation has harmful effects (157). Furthermore, the fact that excess forensic psychiatric treatment is given in order to control crime rather than relieving psychiatric symptoms may be a violation of the Declaration of Hawaii (149), which states that psychiatric care must not be given in the absence of psychiatric illness. The combination of a societal economic burden together with the suspected violation of human rights further emphasises the need for enhancing risk assessments and prevention strategies.

Comments on aim 3

The risk of recidivism in general and in violent crime in particular was heightened if the forensic psychiatric treatment was given without SCS. The level of supervision has in previous reports been shown to be an important factor in modulating risk of recidivism (158), but as can be seen in *Paper I*, treatment with SCS is also correlated with up to five times longer in-patient treatment time, which also affects the probability of criminal behaviour. The findings of *Papers I* and *II* combined show that although the individuals who by court are assessed to have the highest risk of recidivism in crime have both longer in-patient treatment times but also a lesser risk for recidivism in statistical models.

As has been shown in previous studies, this paper confirms the importance of childhood adversities, family and early-onset risk factors in regard to violent recidivism (19, 20, 23, 34, 109). Though, as the study is made of a naturalistic total cohort of forensic psychiatric patients, the reason why only a partition of the previously known risk factors for criminal recidivism were statistically significant may be due to a rather small group size together with a relatively low recidivism rate.

In the cluster analysis in *Paper III*, the smaller of the two identified clusters was characterised by a higher prevalence of substance abuse amongst first-degree relatives, low educational attainment, previous contacts with child and adolescent psychiatric services, out-of-home placements during childhood and adolescence, NDDs, and the development of SUDs later in life. This is in line with previous research findings showing how childhood adversities such as familial psychopathology, maltreatment, and neglect are potential risk factors for conduct disorder (8, 66), and, later on, a long-lasting criminal career (159). NDD diagnoses are theorised to be common in groups of more persistent groups of offenders (43, 160) and are thought to carry a risk of adverse outcomes such as criminality and an antisocial lifestyle in adolescence and adulthood (34-36).

When conducting a MANOVA, the more crime-prone Cluster 1 had a lower age at first crime and had committed a proportionately larger number of financial and drug-related crimes compared to the other cluster. Crime may be a necessity in order to sustain a misuse of drugs and/or alcohol and although violent crime was not a significant variable in the analysis, most individuals in the cohort had committed violent crimes. These findings replicate previous findings of the importance of SUDs for the development and maintenance of criminal behaviour (56, 57, 161).

These findings suggest that there is a high-risk, life-course–persistent phenotype in forensic psychiatric settings defined by NDDs, adverse childhood experiences, early-onset antisocial behaviour, and psychiatric problems including SUDs.

Comments on aim 4

Most individuals in the UPPRÄTT-cohort suffered from psychotic disorders. Psychotic symptoms emerge from a dysfunctional brain and neurobiological findings indicate that for example schizophrenia may be understood as a brain function disorder, yet the molecular biological and genetic findings have not been conclusive of what genes may be responsible for the constellation of symptoms (162). Smaller studies of brain imaging techniques exploring biological underpinnings to aggression in psychotic patients through magnetic resonance imaging have suggested that regions of importance to affective regulation (amygdala, orbito-frontal cortex and the anterior cingulate cortex) and to formation of psychotic symptoms such as hallucinations and delusions (hippocampus and the frontal cortex) are of importance (163).

The SPECT investigations presented in *Paper IV* showed that the recidivists had a lower rCBF in both left and right parietal lobes. This is in accordance with previous studies suggesting a lowered rCBF or reduced glucose metabolism in the same brain areas in violent, impulsive and aggressive individuals (164-167). Reduced parietal blood flow may influence inhibitory controls which in turn might be of importance in criminal behaviour, as both parietal and frontal regions are involved in response inhibition (168, 169). Poor inhibitory control is a consistent marker in externalising psychopathology and involved in SUD (170), ADHD (171), aggression (172), as well as violent (173) and non-violent criminality (174).

The recidivist group had a significantly lower right cerebellar lobe rCBF compared to non-recidivists. In previous studies, the cerebellum has been shown to be involved in cognitive tasks (175) and cerebellar lesions have been shown to be associated with psychopathologies including inhibitory disturbances such as impulsivity and poor attention (176). Yet because the role of the cerebellum in antisocial and criminal behaviour remains unclear and the current study sample was small, this finding is inconclusive and requires further research preferably by using more finely tuned neuroimaging techniques such as functional magnetic resonance imaging. Study results predicting violent behaviour by neuroimaging techniques, particularly in individuals with co-occurring psychotic disorders, are still challenged by small samples. When adding neuroimaging data to conventional risk variables in *Paper IV*, the accuracy of a risk assessment based on traditional risk variables was increased by 28%. The UPPRÄTT-Malmö cohort is small, and the SPECT assessments in today's clinic have been replaced by the more detailed magnetic resonance imaging techniques, so even though the findings add to the growing body of neuroscience, they must be interpreted with caution. Adding neuroimaging data to risk predictions may impact the legal system with regard to length of sentences and choice of treatment (177). It is premature to draw any strong conclusions between anatomical findings and behaviour due to the limited amount of data as well as the lack of knowledge of how changes in brain structure correlates to behaviour, yet if adding results from modern neurocognitive assessments, a connection between behaviour and radiology findings may be strengthened.

Limitations

Each paper in this dissertation includes a detailed discussion about specific limitations adhering to each study, in this section limitations to the dissertation as a whole will be discussed.

Selection of the cohort

All papers in the dissertation are based on studies of the same group of individuals, making up the UPPRÄTT-Malmö study cohort. The choice of presenting a study based on a total cohort could be praised as well as criticised. The UPPRÄTT study group is rather small and as all data on the cohort were not accessible from the Swedish National Council for Crime Prevention due to cohort individuals migrating as well as due to lack of data, the possibility to present robust statistical analyses with high power is therefore limited, in particular when subgroups are analysed. An advantage by including all individuals from a specific area, sentenced to forensic psychiatric in-patient treatment during a set time frame, is that the study becomes representative and naturalistic as it mirrors the clinical everyday practice with this particular group of individuals.

Diagnostics – do we see the full picture?

By definition, the group who underwent the full FPI had a more thorough medical assessment made compared to the group who underwent the FPSR, and thus the diagnoses in the former group would have a more solid ground. The possibility to assess the occurrence of PDs, neuropsychiatric disorders and psychometrical assessments of cognitive function was not feasible in the FPSR-group and it may also have been somewhat limited in the FPI-group as many were severely ill and unmedicated at the time of the investigation, exhibiting a plethora of symptoms which may have shrouded the presence of more discrete symptoms. In addition to that, PDs were not specified in the FPSRs more than to clusters which also lowered the possibility of more detailed analyses.

A higher prevalence of NDDs such as ADHD and ASD would have been expected in the cohort, as the link between NDDs, conduct disorder and criminality is well known (178) and previous studies have shown that these diagnoses are overrepresented in forensic populations (33, 150, 151). When the cohort were undergoing FPI, the Swedish penal system did not include NDDs in the medico-legal definition of SMDs. This resulted in the forensic investigative teams not having had focus on assessing symptoms of NDDs and that the symptoms were attributed to other diagnoses such as PDs.

The complexities of risk assessments

Concerning the risk assessments, only individuals who underwent the full FPI were eligible for HCR-20 (124) and PCL:SV(139) assessments, which reduced the number of individuals available for statistical analyses. It would have been preferable to have access to risk assessments on the total cohort but since the court did not ask for risk of recidivism in the FPSR-group, these assessments were not performed.

The choice of using the shortened version of the risk assessment instruments needs to be addressed as well. The full HRC-20 was not applicable at the time of the FPIs as the study cohort individuals were not receiving in-patient treatment per se at the time of the investigations and no individual treatment nor management plans had been developed. Thus, the last five items regarding assessment of risk in the HCR-20 were omitted.

The research team decided to use the shorter PCL:SV rather than the full version of PCL-R (123), as the forensic investigative teams during the investigations had used the PCL:SV and PCL-R intermittently. The PCL-R is possible to compress into PCL:SV but is not possible to expand the PCL:SV into a full PCL-R. Thus, it was estimated that by using the PCL:SV rather than re-assessing the entire cohort with the PCL-R, the results would be more naturalistic. In previous papers, the high correlation between PCL-R and PCL:SV has been discussed (123, 143).

In 25 cases, the PCL:SV and/or the HCR-20 ratings were not found in the file registries. Therefore, these ratings were made retrospectively by several members of the research team, based on information from the forensic psychiatric reports and on extensive file and register reviews in each case. This approach carries the risk of misinterpreting the written reports of behaviours and symptoms, either under- or overestimating them. As all team members doing the ratings were well acquainted with the instruments, the ratings were assessed as reasonably reliable. The choice of calculating a valid mean substitution in cases when an item had been omitted in the HCR-20, is not a recommended procedure when using the HCR-20 in a clinical practice, and thus the ratings must be seen in light of these limitations due to the setting of data collection.

Interpreting neuroimaging data in a forensic psychiatric context

As the individuals of the cohort varied in age, sex, IQ, diagnoses, and crimes committed, the data from the rCBF must be interpreted cautiously as these variables may affect the results. In *Paper IV* the heterogeneity of the cohort, encompassing the aforementioned variables, was addressed as a limitation as well as the lack of information on the severity of symptoms exhibited by the individuals at the time of the FPIs, which also may have affected the result.

Paper IV did report on data on pharmacological use at the time of the neuroimaging investigations, but another limitation is the uncertainty of adherence to prescribed drugs.

Even if the study cohort individuals were admitted to in-patient treatment at the time of the FPIs, there is always a risk of non-therapeutic levels of prescribed drugs.

Clinical and scientific implications

The overall focus of this thesis is to further improve risk assessments by evaluating risk factors for recidivism in crime amongst forensic psychiatric patients and to suggest loci for further research.

Studies of forensic psychiatric patients with a main focus on those who in the hospital system during a specific timeframe may carry an innate flaw, as patients with a less heavy burden of symptoms and/or less severe criminal histories are released more quickly to society. This leads to a concentration of more severely affected individuals within the hospital system and thereby skewed reports of a more severely ill and affected patient population. The UPPRÄTT study, with its lifetime perspective, incorporates all cases and analyses data from people with heterogeneous backgrounds, health, and criminal behaviours, showing the impact of these factors on the lives of the individuals in this group. As the SCS acted as a risk-reducing factor in this study, further research on recidivism rates after release from involuntary treatment in this group of patients would be interesting.

Because the length of stay differed so much between patients sentenced to treatment with and without SCS despite them being afflicted by the same types of illnesses, finding what factors prolonged the length of stays was of importance. Adverse events were very common and almost two thirds of the cohort were involved in at least one. The finding of this thesis, that adverse events were associated with a longer length of stay, has an important clinical implication as it suggests that the lengthier time within the hospital system was motivated as a crime reducing strategy rather than as a treatment strategy.

Preventing these events are of importance and reducing the risk of them to occur may be done by implementing systematic and evidence-based strategies in daily clinical practice (179, 180). By cooperating and exchanging experiences with other forensic and psychiatric authorities, continuous improvements are possible. Inviting the patients to a continuous dialogue about the self-perceived individual risk factors and need of support may also be successful (181).

What is of far greater importance though, and a challenge for society, is to intervene early in the lives of vulnerable children, in order to reduce the risk for childhood adversities and the aftermath that may come. The short- and long-term consequences of insufficient parental care and burdened childhood environments can be seen in schools and later on in prison and in the forensic psychiatric hospitals.

Both psychiatry and forensic psychiatry needs to notice, assess and treat both chronic clinical conditions and singular trauma-related clinical states. No specific diagnosis has
been proven to predict recidivism in criminality or the length of stay in forensic psychiatric in-patient treatment in this particular cohort. Since the burden of illness is obviously not the reason for the different lengths of treatment, forensic psychiatry has an ethical dilemma. Are extended in-patient times justifiable to prevent a potentially highrisk individual from committing new crimes when there is growing evidence that assessments of mentally disordered offenders tend to overestimate their risk for violence (182)?

Further problems arise when the forensic psychiatric patients are assessed as ready to continue rehabilitation outside of the hospital, as there is a great need of housings with suitable support. Housing is provided by the municipalities but there is a lack of appropriate accommodations and this may prolong the length of stays as the risk of relapse to crime, drug use, and worsened mental health rises steeply if patients are discharged to unstable living conditions. By improved risk predictions, the length of stays could be shortened but it is equally important to continue a supportive environment after discharge as adverse events may arise outside of hospital as well.

Adding information from neuroimaging assessments may be one way to improve risk predictions but today's improved imaging techniques are rarely used in larger risk assessment studies. The link between the theorised atypical neurophysiology of forensic psychiatric patients and the antisocial behaviour is far from clear and requires more research.

Treatment with SCS lowered the risk for recidivism which may be a consequence of the longer in-patient treatment times with staff support and monitored medication. Another hypothesis is that as the patients receiving treatment with SCS were statistically more often involved in adverse events of violence and threat during in-patient treatment, the same kind of behaviour outside a forensic hospital setting might have resulted in prosecution and sentencing. The tolerance of adverse behaviour is to some extent larger amongst forensic psychiatric staff and thus, research in adverse events as equal to criminal behaviour in the community may set the added protective effect of SCS in another light.

This thesis did not find SUDs nor specific mental health disorders to be predictive variables for recidivism in crime which might be due to a high prevalence of SUDs in combination with a low rate of recidivism and the statistical challenges that follows when the measured outcome is relatively rare and the sample is relatively small. Yet treatment and medication of these mental disorders are important pathways to reduce criminal behaviour as they, based on previous research, are known dynamic risk factors (100, 106, 183). Furthermore, there was a very low base-rate of NDDs in the thesis cohort compared to previous studies which have shown strong evidence between these disorders and CD and violent criminality (178). In the wake of changing court praxes and the following tendency to include NDDs (mainly ASDs) in the medico-legal construct of SMD, a clinical implication of this thesis would be to ascertain if NDDs are present and offer appropriate treatment and adjustments.

This thesis contributes to the growing knowledge of childhood adversities as an important predictive risk factor for adult criminal behaviour (20, 34). To prevent offending, public health agencies should put in efforts to try to identify vulnerable families and children at risk of developing antisocial behaviours and to offer intervention strategies at both individual and family levels.

References

- 1. Krug E. World report on violence and health. Geneva: World Health Organization; 2002.
- 2. Merriam-Webster. The Merriam-Webster Online Dictionary 2019 [Available from: https://www.merriam-webster.com/dictionary/violence.
- World Health Organization. Global status report on violence prevention. Geneva: WHO; 2014.
- Gray NS, Hill C, McGleish A, Timmons D, MacCulloch MJ, Snowden RJ. Prediction of violence and self-harm in mentally disordered offenders: a prospective study of the efficacy of HCR-20, PCL-R, and psychiatric symptomatology. Journal of consulting and clinical psychology. 2003;71(3):443-51.
- United Nations Office on Drugs and Crime (UNODC). Global Study on Homicide 2019. In: Affairs DfPAaP, editor. Vienna: United Nations; 2019.
- 6. Pinker S. The Better Angels of Our Nature: Why Violence Has Declined. United States of America: Viking Books; 2011.
- 7. World Health Organization. Child maltreatment 2018 [Available from: http://www.who.int/topics/child_abuse/en/.
- 8. Falshaw L, Browne K. Adverse childhood experiences and violent acts of young people in secure accommodation. Journal of Mental Health. 1997;6(5):443-56.
- 9. Tremblay RE. Developmental origins of disruptive behaviour problems: the 'original sin' hypothesis, epigenetics and their consequences for prevention. Journal of child psychology and psychiatry, and allied disciplines. 2010;51(4):341-67.
- 10. Jambon M, Madigan S, Plamondon A, Jenkins J. Developmental trajectories of physical aggression and prosocial behavior in early childhood: Family antecedents and psychological correlates. Developmental psychology. 2019;55(6):1211-25.
- Tremblay RE, Nagin DS, Seguin JR, Zoccolillo M, Zelazo PD, Boivin M, et al. Physical aggression during early childhood: trajectories and predictors. Pediatrics. 2004;114(1):e43-50.
- 12. Broidy LM, Nagin DS, Tremblay RE, Bates JE, Brame B, Dodge KA, et al. Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: a six-site, cross-national study. Developmental psychology. 2003;39(2):222-45.
- Kokko K, Pulkkinen L. Aggression in childhood and long-term unemployment in adulthood: a cycle of maladaptation and some protective factors. Developmental psychology. 2000;36(4):463-72.

- 14. Fergusson DM, Horwood LJ. Early conduct problems and later life opportunities. Journal of child psychology and psychiatry, and allied disciplines. 1998;39(8):1097-108.
- 15. Stattin H, Magnusson D. The role of early aggressive behavior in the frequency, seriousness, and types of later crime. Journal of consulting and clinical psychology. 1989;57(6):710-8.
- 16. Nagin D, Tremblay RE. Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. Child development. 1999;70(5):1181-96.
- 17. Blumstein A, Cohen J. Characterizing criminal careers. Science. 1987;237(4818):985-91.
- Provencal N, Booij L, Tremblay RE. The developmental origins of chronic physical aggression: biological pathways triggered by early life adversity. J Exp Biol. 2015;218(Pt 1):123-33.
- 19. Sourander A, Elonheimo H, Niemela S, Nuutila AM, Helenius H, Sillanmaki L, et al. Childhood predictors of male criminality: a prospective population-based follow-up study from age 8 to late adolescence. Journal of the American Academy of Child and Adolescent Psychiatry. 2006;45(5):578-86.
- 20. Copeland WE, Miller-Johnson S, Keeler G, Angold A, Costello EJ. Childhood psychiatric disorders and young adult crime: a prospective, population-based study. The American journal of psychiatry. 2007;164(11):1668-75.
- 21. Duke NN, Pettingell SL, McMorris BJ, Borowsky IW. Adolescent violence perpetration: associations with multiple types of adverse childhood experiences. Pediatrics. 2010;125(4):e778-86.
- Reavis JA, Looman J, Franco KA, Rojas B. Adverse childhood experiences and adult criminality: how long must we live before we possess our own lives? Perm J. 2013;17(2):44-8.
- 23. Falk O, Wallinius M, Lundstrom S, Frisell T, Anckarsater H, Kerekes N. The 1 % of the population accountable for 63 % of all violent crime convictions. Soc Psychiatry Psychiatr Epidemiol. 2013.
- 24. Jaffee SR, Caspi A, Moffitt TE, Taylor A. Physical maltreatment victim to antisocial child: evidence of an environmentally mediated process. Journal of abnormal psychology. 2004;113(1):44-55.
- 25. Cardinal RN, Parkinson JA, Hall J, Everitt BJ. Emotion and motivation: the role of the amygdala, ventral striatum, and prefrontal cortex. Neuroscience and biobehavioral reviews. 2002;26(3):321-52.
- Adolphs R, Tranel D, Damasio H, Damasio A. Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. Nature. 1994;372(6507):669-72.
- 27. Damasio AR. The somatic marker hypothesis and the possible functions of the prefrontal cortex. Philos Trans R Soc Lond B Biol Sci. 1996;351(1346):1413-20.
- Brower MC, Price BH. Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: a critical review. Journal of neurology, neurosurgery, and psychiatry. 2001;71(6):720-6.

- 29. Johanson M, Vaurio O, Tiihonen J, Lahteenvuo M. A Systematic Literature Review of Neuroimaging of Psychopathic Traits. Frontiers in psychiatry. 2019;10:1027.
- 30. Golding P, Fitzgerald HE. The early biopsychosocial development of boys and the origins of violence in males. Infant Ment Health J. 2019;40(1):5-22.
- 31. Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, et al. Role of genotype in the cycle of violence in maltreated children. Science. 2002;297(5582):851-4.
- 32. O'Neal CRMB, L; Huang, K-Y; Kiely Gouley, K; Kamboukos, D; Calzada, E. J.; Pine, D.S. Understanding Relations Among Early Family Environment, Cortisol Response, and Child Aggression via a Prevention Experimen. Child development. 2010;81:290-305.
- Billstedt E, Anckarsater H, Wallinius M, Hofvander B. Neurodevelopmental disorders in young violent offenders: Overlap and background characteristics. Psychiatry Res. 2017;252:234-41.
- 34. Pingault JB, Cote SM, Lacourse E, Galera C, Vitaro F, Tremblay RE. Childhood hyperactivity, physical aggression and criminality: a 19-year prospective population-based study. Plos One. 2013;8(5):e62594.
- 35. Howlin P, Savage S, Moss P, Tempier A, Rutter M. Cognitive and language skills in adults with autism: a 40-year follow-up. Journal of child psychology and psychiatry, and allied disciplines. 2014;55(1):49-58.
- 36. Mannuzza S, Klein RG, Moulton JL, 3rd. Lifetime criminality among boys with attention deficit hyperactivity disorder: a prospective follow-up study into adulthood using official arrest records. Psychiatry Res. 2008;160(3):237-46.
- 37. Barkley RA, Fischer M, Smallish L, Fletcher K. Young adult follow-up of hyperactive children: antisocial activities and drug use. Journal of child psychology and psychiatry, and allied disciplines. 2004;45(2):195-211.
- 38. Gillberg C. The ESSENCE in child psychiatry: Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations. Res Dev Disabil. 2010;31(6):1543-51.
- Karam RG, Breda V, Picon FA, Rovaris DL, Victor MM, Salgado CA, et al. Persistence and remission of ADHD during adulthood: a 7-year clinical follow-up study. Psychol Med. 2015;45(10):2045-56.
- 40. Gillberg IC, Helles A, Billstedt E, Gillberg C. Boys with Asperger Syndrome Grow Up: Psychiatric and Neurodevelopmental Disorders 20 Years After Initial Diagnosis. Journal of autism and developmental disorders. 2016;46(1):74-82.
- Biederman J, Monuteaux MC, Mick E, Spencer T, Wilens TE, Silva JM, et al. Young adult outcome of attention deficit hyperactivity disorder: a controlled 10-year follow-up study. Psychol Med. 2006;36(2):167-79.
- 42. Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R. Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. Archives of general psychiatry. 2003;60(7):709-17.
- 43. Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. Psychol Rev. 1993;100(4):674-701.

- 44. Fairchild G, van Goozen SH, Calder AJ, Goodyer IM. Research review: evaluating and reformulating the developmental taxonomic theory of antisocial behaviour. Journal of child psychology and psychiatry, and allied disciplines. 2013;54(9):924-40.
- 45. Carkin DM, Tracy PE. Moffitt Revisited: Delinquent and Criminal Career Paths in the 1958 Philadelphia Birth Cohort. Int J Law Crime Justice. 2015;3(1):14-39.
- 46. Odgers CL, Caspi A, Broadbent JM, Dickson N, Hancox RJ, Harrington H, et al. Prediction of differential adult health burden by conduct problem subtypes in males. Archives of general psychiatry. 2007;64(4):476-84.
- 47. Gottfredson M, Hirschi T. A General Theory of Crime. Palo Alto, United States: Stanford University Press; 1990.
- 48. Hodelet N. Psychosis and offending in British Columbia: characteristics of a secure hospital population. Criminal behaviour and mental health : CBMH. 2001;11(3):163-72.
- Tabita B, de Santi MG, Kjellin L. Criminal recidivism and mortality among patients discharged from a forensic medium secure hospital. Nordic journal of psychiatry. 2012;66(4):283-9.
- 50. Soderstrom H, Sjodin AK, Carlstedt A, Forsman A. Adult psychopathic personality with childhood-onset hyperactivity and conduct disorder: a central problem constellation in forensic psychiatry. Psychiatry Res. 2004;121(3):271-80.
- 51. Wallinius M, Nilsson T, Hofvander B, Anckarsater H, Stalenheim G. Facets of psychopathy among mentally disordered offenders: Clinical comorbidity patterns and prediction of violent and criminal behavior. Psychiatry Res. 2012;198(2):279-84.
- 52. Svennerlind C, Nilsson T, Kerekes N, Andine P, Lagerkvist M, Forsman A, et al. Mentally disordered criminal offenders in the Swedish criminal system. International journal of law and psychiatry. 2010;33(4):220-6.
- Arseneault L, Moffitt TE, Caspi A, Taylor PJ, Silva PA. Mental disorders and violence in a total birth cohort: results from the Dunedin Study. Archives of general psychiatry. 2000;57(10):979-86.
- 54. Fazel S, Grann M. The population impact of severe mental illness on violent crime. The American journal of psychiatry. 2006;163(8):1397-403.
- 55. Walsh E, Buchanan A, Fahy T. Violence and schizophrenia: examining the evidence. The British journal of psychiatry : the journal of mental science. 2002;180:490-5.
- Chang Z, Larsson H, Lichtenstein P, Fazel S. Psychiatric disorders and violent reoffending: a national cohort study of convicted prisoners in Sweden. The lancet Psychiatry. 2015;2(10):891-900.
- Fazel S, Langstrom N, Hjern A, Grann M, Lichtenstein P. Schizophrenia, substance abuse, and violent crime. JAMA : the journal of the American Medical Association. 2009;301(19):2016-23.
- 58. American Psychiatric Association (APA). DSM-III. Washington, DC.: APA; 1980.
- 59. American Psychiatric Association (APA). Diagnostic and statistical manual of mental disorders, DSM-IV. Washington DC: APA Press; 1994.

- 60. American Psychiatric Association (APA). Diagnostic and statistical manual of mental disorders, DSM-5. Arlington, VA: APA Publishing; 2013.
- 61. Johnson JG, Cohen P, Smailes E, Kasen S, Oldham JM, Skodol AE, et al. Adolescent personality disorders associated with violence and criminal behavior during adolescence and early adulthood. The American journal of psychiatry. 2000;157(9):1406-12.
- 62. Coid JW. Personality disorders in prisoners and their motivation for dangerous and disruptive behaviour. Criminal behaviour and mental health : CBMH. 2002;12(3):209-26.
- 63. Yu R, Geddes JR, Fazel S. Personality disorders, violence, and antisocial behavior: a systematic review and meta-regression analysis. Journal of personality disorders. 2012;26(5):775-92.
- 64. Fazel S, Danesh J. Serious mental disorder in 23000 prisoners: a systematic review of 62 surveys. Lancet. 2002;359(9306):545-50.
- 65. Puzzo I, Smaragdi A, Gonzalez K, Martin-Key N, Fairchild G. Neurobiological, Neuroimaging, and Neuropsychological Studies of Children and Adolescents with Disruptive Behavior Disorders. Family Relations. 2016:134-50.
- Pardini D, Frick PJ. Multiple developmental pathways to conduct disorder: current conceptualizations and clinical implications. J Can Acad Child Adolesc Psychiatry. 2013;22(1):20-5.
- 67. Hodgins S, Cree A, Alderton J, Mak T. From conduct disorder to severe mental illness: associations with aggressive behaviour, crime and victimization. Psychol Med. 2008;38(7):975-87.
- Antisocial Personality Disorder. The NICE guideline on treatment, management and prevention. [Internet]. British Psychological Society and The Royal College of Psychiatrists. 2010. Available from: <u>https://www.nice.org.uk/guidance/cg77/evidence/full-guideline-</u> 242104429.
- 69. Grann M, Fazel S. Substance misuse and violent crime: Swedish population study. BMJ. 2004;328(7450):1233-4.
- 70. Coid J, Yang M, Roberts A, Ullrich S, Moran P, Bebbington P, et al. Violence and psychiatric morbidity in a national household population--a report from the British Household Survey. American journal of epidemiology. 2006;164(12):1199-208.
- 71. Addington J, Addington D. Patterns, predictors and impact of substance use in early psychosis: a longitudinal study. Acta psychiatrica Scandinavica. 2007;115(4):304-9.
- 72. Arseneault L, Cannon M, Witton J, Murray RM. Causal association between cannabis and psychosis: examination of the evidence. The British journal of psychiatry : the journal of mental science. 2004;184:110-7.
- 73. Colizzi M, Carra E, Fraietta S, Lally J, Quattrone D, Bonaccorso S, et al. Substance use, medication adherence and outcome one year following a first episode of psychosis. Schizophr Res. 2016;170(2-3):311-7.
- 74. Gossop M, Marsden J, Stewart D, Kidd T. The National Treatment Outcome Research Study (NTORS): 4-5 year follow-up results. Addiction. 2003;98(3):291-303.

- 75. Skåån C, Forslund K. RättspsyK Årsrapport 2017. Litorapid, HisingsKärra: Regionstyrelsen i Västra Götalandsregionen; 2017.
- 76. Coid J, Hickey N, Kahtan N, Zhang T, Yang M. Patients discharged from medium secure forensic psychiatry services: reconvictions and risk factors. The British journal of psychiatry : the journal of mental science. 2007;190:223-9.
- 77. Davoren M, Byrne O, O'Connell P, O'Neill H, O'Reilly K, Kennedy HG. Factors affecting length of stay in forensic hospital setting: need for therapeutic security and course of admission. BMC psychiatry. 2015;15:301.
- 78. Hodgins S, Piatosa MJ, Schiffer B. Violence among people with schizophrenia: phenotypes and neurobiology. Current topics in behavioral neurosciences. 2014;17:329-68.
- 79. Large MM, Nielssen O. Violence in first-episode psychosis: a systematic review and metaanalysis. Schizophr Res. 2011;125(2-3):209-20.
- 80. Byrd AL, Manuck SB. MAOA, childhood maltreatment, and antisocial behavior: metaanalysis of a gene-environment interaction. Biological psychiatry. 2014;75(1):9-17.
- 81. Nilsson KW, Comasco E, Hodgins S, Oreland L, Aslund C. Genotypes Do Not Confer Risk For Delinquency But Rather Alter Susceptibility to Positive and Negative Environmental Factors: Gene-Environment Interactions of BDNF Val66Met, 5-HTTLPR, and MAOAuVNTR. Int J Neuropsychopharmacol. 2015;18(5):1-10.
- 82. van Os J, Linscott RJ, Myin-Germeys I, Delespaul P, Krabbendam L. A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. Psychol Med. 2009;39(2):179-95.
- Hodgins S, Klein S. New Clinically Relevant Findings about Violence by People with Schizophrenia. Canadian journal of psychiatry Revue canadienne de psychiatrie. 2017;62(2):86-93.
- 84. Hodgins S. Mental disorder, intellectual deficiency, and crime. Evidence from a birth cohort. Archives of general psychiatry. 1992;49(6):476-83.
- 85. Joyal C, Dubreucq J-L, Gendron C, Millaud F. Major Mental Disorder and Violence: A Critical Update. Current Psychiatry Review. 2007;3:33-50.
- 86. Witt K, van Dorn R, Fazel S. Risk factors for violence in psychosis: systematic review and meta-regression analysis of 110 studies. Plos One. 2013;8(2):e55942.
- 87. Fazel S, Gulati G, Linsell L, Geddes JR, Grann M. Schizophrenia and violence: systematic review and meta-analysis. PLoS Med. 2009;6(8):e1000120.
- Sariaslan A, Larsson H, Fazel S. Genetic and environmental determinants of violence risk in psychotic disorders: a multivariate quantitative genetic study of 1.8 million Swedish twins and siblings. Molecular psychiatry. 2016;21(9):1251-6.
- Hodgins S, Mednick SA, Brennan PA, Schulsinger F, Engberg M. Mental disorder and crime. Evidence from a Danish birth cohort. Archives of general psychiatry. 1996;53(6):489-96.
- 90. Brennan PA, Mednick SA, Hodgins S. Major mental disorders and criminal violence in a Danish birth cohort. Archives of general psychiatry. 2000;57(5):494-500.

- Lindqvist P. Mental disorder, substance misuse and violent behaviour: the Swedish experience of caring for the triply troubled. Criminal behaviour and mental health : CBMH. 2007;17(4):242-9.
- 92. The Swedish Penal Code. (SFS 1962:700), (2008).
- 93. Act on Forensic Mental Care. (SFS 1991:1129), (1991).
- 94. Act on Compulsory Psychiatric Care. (SFS 1991:1128), (1991).
- 95. Act on Forensic Psychiatric Investigation. (SFS 1991:1137), (1991).
- 96. Skåån C, Forslund K. Årsrapport 2016. Nationellt rättspsykiatriskt kvalitetsregister. Göthenburg; 2017.
- 97. The National Board of Forensic Medicine 2019 [Available from: <u>https://www.rmv.se/om-oss/forskning/aktuell-statistik/</u>.
- 98. Stahl S, Morrissette D. Violence: Neural Circuits, Genetics and Treatment. U.S.A.: Cambridge University Press, New York; 2014.
- 99. Stahl SM. Stahl's Essential Psychopharmacology: neuroscientific basis and practical applications. 3'd ed. U.S.A.: Cambrigde University Press; 2008.
- 100. Fazel S, Zetterqvist J, Larsson H, Langstrom N, Lichtenstein P. Antipsychotics, mood stabilisers, and risk of violent crime. Lancet. 2014;384(9949):1206-14.
- Saha S, Chant D, McGrath J. A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time? Archives of general psychiatry. 2007;64(10):1123-31.
- 102. Torniainen M, Mittendorfer-Rutz E, Tanskanen A, Bjorkenstam C, Suvisaari J, Alexanderson K, et al. Antipsychotic treatment and mortality in schizophrenia. Schizophr Bull. 2015;41(3):656-63.
- 103. Olfson M, Gerhard T, Huang C, Crystal S, Stroup TS. Premature Mortality Among Adults With Schizophrenia in the United States. JAMA psychiatry. 2015;72(12):1172-81.
- 104. Vermeulen J, van Rooijen G, Doedens P, Numminen E, van Tricht M, de Haan L. Antipsychotic medication and long-term mortality risk in patients with schizophrenia; a systematic review and meta-analysis. Psychol Med. 2017;47(13):2217-28.
- 105. Elonheimo H, Niemela S, Parkkola K, Multimaki P, Helenius H, Nuutila AM, et al. Policeregistered offenses and psychiatric disorders among young males : the Finnish "From a boy to a man" birth cohort study. Soc Psychiatry Psychiatr Epidemiol. 2007;42(6):477-84.
- 106. Grann M, Danesh J, Fazel S. The association between psychiatric diagnosis and violent reoffending in adult offenders in the community. BMC psychiatry. 2008;8:92.
- 107. Fazel S, Wolf A. A Systematic Review of Criminal Recidivism Rates Worldwide: Current Difficulties and Recommendations for Best Practice. Plos One. 2015;10(6):e0130390.
- Durose MR, Cooper AD, Snyder HN. Recidivism of Prisoners Released in 30 States in 2005: Patterns from 2005 to 2010. United States of America: Office of Justice Programs; 2014.
- 109. Nilsson T, Wallinius M, Gustavson C, Anckarsater H, Kerekes N. Violent recidivism: a long-time follow-up study of mentally disordered offenders. Plos One. 2011;6(10):e25768.

- 110. Bonta J, Law M, Hanson K. The prediction of criminal and violent recidivism among mentally disordered offenders: a meta-analysis. Psychol Bull. 1998;123(2):123-42.
- 111. Pedersen L, Rasmussen K, Elsass P, Hougaard H. The importance of early anti-social behaviour among men with a schizophrenia spectrum disorder in a specialist forensic psychiatry hospital unit in Denmark. Criminal behaviour and mental health : CBMH. 2010;20(4):295-304.
- 112. Kooyman I, Walsh E, Stevens H, Burns T, Tyrer P, Tattan T, et al. Criminal offending before and after the onset of psychosis: examination of an offender typology. Schizophr Res. 2012;140(1-3):198-203.
- 113. Singh JP, Grann M, Fazel S. A comparative study of violence risk assessment tools: a systematic review and metaregression analysis of 68 studies involving 25,980 participants. Clinical psychology review. 2011;31(3):499-513.
- 114. Wilson CM, Desmarais SL, Nicholls TL, Hart SD, Brink J. Predictive validity of dynamic factors: assessing violence risk in forensic psychiatric inpatients. Law and human behavior. 2013;37(6):377-88.
- 115. Singh J, Fazel S. Forensic risk assessment: A metareview. Criminal justice and behavior. 2010;37.
- Nilsson T, Munthe C, Gustavson C, Forsman A, Anckarsater H. The precarious practice of forensic psychiatric risk assessments. International journal of law and psychiatry. 2009;32(6):400-7.
- 117. Mossman D. Assessing predictions of violence: being accurate about accuracy. Journal of consulting and clinical psychology. 1994;62(4):783-92.
- 118. Quincey V, Harris GT, Rice ME, Cormier CA. Violent Offenders: Appraising and managing risk. Washington, DC.: American Psychological Association; 1998.
- 119. Ægisdóttir S, White MJ, Spengler PM, Maugherman AS, Anderson LA, Cook RS, et al. The meta-analysis of clinical judgement project: fifty-six years of accumulated research on clinical versus statistical prediction. Couns Psychol. 2006;34:341-82.
- 120. Kroner C, Stadtland C, Eidt M, Nedopil N. The validity of the Violence Risk Appraisal Guide (VRAG) in predicting criminal recidivism. Criminal behaviour and mental health : CBMH. 2007;17(2):89-100.
- 121. Sreenivasan S, Kirkish P, Garrick T, Weinberger LE, Phenix A. Actuarial risk assessment models: a review of critical issues related to violence and sex-offender recidivism assessments. The journal of the American Academy of Psychiatry and the Law. 2000;28(4):438-48.
- 122. Dahle KP. Strengths and limitations of actuarial prediction of criminal reoffence in a German prison sample: a comparative study of LSI-R, HCR-20 and PCL-R. International journal of law and psychiatry. 2006;29(5):431-42.
- 123. Hare RD. Hare Psychopathy Checklist-Revised (PCL-R). Toronto, ON: Multi-Health Systems; 1991.
- 124. Webster CD, Douglas KS, Eaves D, Hart SD. HCR-20: Assessing risk for violence. Vancouver, British Columbia, Canada: Mental Health, Law and Policy Institute; 1997.
- 125. Cleckley H. The Mask of Sanity. 5th ed. St Louis, MO: Mosby; 1976.

- 126. World Health Organization. The ICD-10 classification of mental and behavioural disorders: clinical descriptions and diagnostic guidelines. Geneva: World Health Organization; 1992.
- 127. Hare RD. Hare Psychopathy Checklist-Revised (PCL-R): 2nd Edition Technical Manual. North Tonawanda, NY, USA

Toronto, ON, Canada: Multi-Health Systems Inc.; 2003.

- 128. Simourd DJ, Hoge RD. Criminal Psychopathy. A Risk-and-Need Perspective. Criminal justice and behavior. 2000;27(2).
- 129. Harris GT, Rice ME, Cormier CA. Psychopathy and Violent Recidivism. Law and human behavior. 1991;15(6).
- Walters GD, Knight RA, Grann M, Dahle KP. Incremental validity of the Psychopathy Checklist facet scores: predicting release outcome in six samples. Journal of abnormal psychology. 2008;117(2):396-405.
- 131. Grann M, Belfrage H, Tengström A. Actuarial assessment of risk for violence. Predictive validity of the VRAG and the historical part of the HCR-20. Criminal Justice & Behaviour. 2000;27(1):97-114.
- 132. Aharoni E, Funk C, Sinnott-Armstrong W, Gazzaniga M. Can neurological evidence help courts assess criminal responsibility? Lessons from law and neuroscience. Annals of the New York Academy of Sciences. 2008;1124:145-60.
- 133. Martell DA. Neuroscience and the law: philosophical differences and practical constraints. Behavioral sciences & the law. 2009;27(2):123-36.
- 134. First MB, Gibbon M, Spitzer RL, Williams JB. Structured Clinical Interview for DSM-IV Axis I Disorders, Clinician Version (SCID-CV). Washington, D.C.: American Psychiatric Press, Inc.; 1996.
- 135. First MB, Gibbon M, Spitzer RL, Williams JBW, Benjamin LS. Structured Clinical Interview for DSM-IV Axis II Personality Disorders, (SCID-II). Washington, D.C.: American Psychiatric Press, Inc.; 1997.
- 136. Dureman I, Sälde H. SRB Synonymer, Reasning och Block test. 1959.
- 137. Raven J, Raven JC, Court JH. Manual for Raven's Progressive Matrices and Vocabulary Scales. Section 1: General overview. 1998 ed ed. Oxford : Oxford Psychologists, 19981998.
- Wechsler D. WAIS-R Manual. Wechsler Adult Intelligence Scale Revised. San Antonio, TX.: The Psychological Corporation; 1981.
- Hart SD, Cox DN, Hare RD. The Hare Psychopathy Checklist: Screening Version. Toronto, ON: Multi-Health Systems.; 1995.
- Grann M, Langstrom N, Tengstrom A, Stalenheim EG. Reliability of file-based retrospective ratings of psychopathy with the PCL-R. Journal of personality assessment. 1998;70(3):416-26.
- 141. Penney SR, McMaster R, Wilkie T. Multirater reliability of the historical, clinical, and risk management-20. Assessment. 2014;21(1):15-27.

- 142. Raaijmakers Q. Effectiveness of different missing data treatments in surveys with Likert-type data: Introducing the relative mean substitution approach. Educational and Psychological Measurement. 1999;59(5):725-48.
- 143. Cooke DJ, Michie C, Hart SD, Hare RD. Evaluating the Screening Version of the Hare Psychopathy Checklist—Revised (PCL:SV): An Item Response Theory Analysis. Psychological Assessment. 1999;11(1):10.
- 144. Cohen J. Statistical Power Analysis for the Behavioral Sciences. 2'nd ed. Routledge, editor1988.
- 145. R Core Team. R: A language and environment for statistical computing. Vienna, Austria: R Core Team; 2017.
- 146. Lydersen S, Fagerland MW, Laake P. Recommended tests for association in 2 x 2 tables. Stat Med. 2009;28(7):1159-75.
- 147. Delacre M, Lakens D, Leys C. Why Psychologists Should by Default Use Welch's t-test Instead of Student's t-test. International review of social psychology. 2017;30(1):92-101.
- 148. Munthe C, Radovic S, Anckarsater H. Ethical issues in forensic psychiatric reserach on mentally disordered offenders. Bioethics. 2010;24(1):35-44.
- 149. World Psychiatric Association. Declaration of Hawaii. Adopted in 1977 at the 6th World Congress of Psychiatry in Honolulu, Hawaii. Amended at the 7th Congress in Vienna, Italy, in July 1983. World Psychiatric Association; 1977.
- 150. Siponmaa L, Kristiansson M, Jonson C, Nyden A, Gillberg C. Juvenile and young adult mentally disordered offenders: the role of child neuropsychiatric disorders. The journal of the American Academy of Psychiatry and the Law. 2001;29(4):420-6.
- 151. Soderstrom H, Nilsson T, Sjodin AK, Carlstedt A, Forsman A. The childhood-onset neuropsychiatric background to adulthood psychopathic traits and personality disorders. Comprehensive psychiatry. 2005;46(2):111-6.
- 152. Billstedt E, Anckarsater H, Wallinius M, Hofvander B. Neurodevelopmental disorders in young violent offenders: A cross-sectional study of prison inmates. Submitted, not yet in print. 2016.
- 153. Lundstrom S, Reichenberg A, Anckarsater H, Lichtenstein P, Gillberg C. Autism phenotype versus registered diagnosis in Swedish children: prevalence trends over 10 years in general population samples. BMJ. 2015;350:h1961.
- 154. Brown K, Fahy T. Medium secure units: pathways of care and time to discharge over a fouryear period in South London. The Journal of Forensic Psychiatry and Psychology. 2008;20(2):268-77.
- 155. Vollm B, Edworthy R, Holley J, Talbot E, Majid S, Duggan C, et al. A mixed-methods study exploring the characteristics and needs of long-stay patients in high and medium secure settings in England: implications for service organisation. Health Services and Delivery Research. Southampton (UK): Health Services and Delivery Research; 2017.
- 156. Hare Duke L, Furtado V, Guo B, Vollm BA. Long-stay in forensic-psychiatric care in the UK. Soc Psychiatry Psychiatr Epidemiol. 2018;53(3):313-21.

- 157. Kasckow JW, Twamley E, Mulchahey JJ, Carroll B, Sabai M, Strakowski SM, et al. Healthrelated quality of well-being in chronically hospitalized patients with schizophrenia: comparison with matched outpatients. Psychiatry Res. 2001;103(1):69-78.
- Lund C, Forsman A, Anckarsater H, Nilsson T. Early criminal recidivism among mentally disordered offenders. International journal of offender therapy and comparative criminology. 2012;56(5):749-68.
- 159. Simonoff E, Elander J, Holmshaw J, Pickles A, Murray R, Rutter M. Predictors of antisocial personality. Continuities from childhood to adult life. The British journal of psychiatry : the journal of mental science. 2004;184:118-27.
- Raine A, Moffitt TE, Caspi A, Loeber R, Stouthamer-Loeber M, Lynam D. Neurocognitive impairments in boys on the life-course persistent antisocial path. Journal of abnormal psychology. 2005;114(1):38-49.
- 161. Chang Z, Lichtenstein P, Larsson H, Fazel S. Substance use disorders, psychiatric disorders, and mortality after release from prison: a nationwide longitudinal cohort study. The lancet Psychiatry. 2015;2(5):422-30.
- Soyka M. Neurobiology of aggression and violence in schizophrenia. Schizophr Bull. 2011;37(5):913-20.
- 163. Fjellvang M, Groning L, Haukvik UK. Imaging Violence in Schizophrenia: A Systematic Review and Critical Discussion of the MRI Literature. Frontiers in psychiatry. 2018;9:333.
- 164. Raine A, Buchsbaum M, LaCasse L. Brain abnormalities in murderers indicated by positron emission tomography. Biological psychiatry. 1997;42(6):495-508.
- 165. Siever LJ, Buchsbaum MS, New AS, Spiegel-Cohen J, Wei T, Hazlett EA, et al. d,lfenfluramine response in impulsive personality disorder assessed with [18F]fluorodeoxyglucose positron emission tomography. Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology. 1999;20(5):413-23.
- 166. Hirono N, Mega MS, Dinov ID, Mishkin F, Cummings JL. Left frontotemporal hypoperfusion is associated with aggression in patients with dementia. Archives of neurology. 2000;57(6):861-6.
- 167. Soderstrom H, Tullberg M, Wikkelso C, Ekholm S, Forsman A. Reduced regional cerebral blood flow in non-psychotic violent offenders. Psychiatry Research-Neuroimaging. 2000;98(1):29-41.
- 168. Rubia K, Russell T, Overmeyer S, Brammer MJ, Bullmore ET, Sharma T, et al. Mapping motor inhibition: conjunctive brain activations across different versions of go/no-go and stop tasks. NeuroImage. 2001;13(2):250-61.
- 169. Watanabe J, Sugiura M, Sato K, Sato Y, Maeda Y, Matsue Y, et al. The human prefrontal and parietal association cortices are involved in NO-GO performances: an event-related fMRI study. NeuroImage. 2002;17(3):1207-16.
- 170. Aad G, Abajyan T, Abbott B, Abdallah J, Abdel Khalek S, Abdinov O, et al. Jet energy measurement and its systematic uncertainty in proton-proton collisions at [Formula: see text]

TeV with the ATLAS detector. The European physical journal C, Particles and fields. 2015;75:17.

- 171. Sjowall D, Roth L, Lindqvist S, Thorell LB. Multiple deficits in ADHD: executive dysfunction, delay aversion, reaction time variability, and emotional deficits. Journal of child psychology and psychiatry, and allied disciplines. 2013;54(6):619-27.
- 172. Pawliczek CM, Derntl B, Kellermann T, Kohn N, Gur RC, Habel U. Inhibitory control and trait aggression: neural and behavioral insights using the emotional stop signal task. NeuroImage. 2013;79:264-74.
- 173. Hancock M, Tapscott JL, Hoaken PN. Role of executive dysfunction in predicting frequency and severity of violence. Aggressive behavior. 2010;36(5):338-49.
- 174. Meijers J, Harte JM, Jonker FA, Meynen G. Prison brain? Executive dysfunction in prisoners. Front Psychol. 2015;6:43.
- 175. Baillieux H, De Smet HJ, Paquier PF, De Deyn PP, Marien P. Cerebellar neurocognition: insights into the bottom of the brain. Clinical neurology and neurosurgery. 2008;110(8):763-73.
- 176. Gordon N. The cerebellum and cognition. European journal of paediatric neurology : EJPN : official journal of the European Paediatric Neurology Society. 2007;11(4):232-4.
- 177. Poldrack RA, Monahan J, Imrey PB, Reyna V, Raichle ME, Faigman D, et al. Predicting Violent Behavior: What Can Neuroscience Add? Trends Cogn Sci. 2018;22(2):111-23.
- 178. Patterson GR, DeGarmo DS, Knutson N. Hyperactive and antisocial behaviors: comorbid or two points in the same process? Development and psychopathology. 2000;12(1):91-106.
- 179. Hill AK, Lind MA, Tucker D, Nelly P, Daraiseh N. Measurable results: Reducing staff injuries on a specialty psychiatric unit for patients with developmental disabilities. Work. 2015;51(1):99-111.
- 180. Kaunomaki J, Jokela M, Kontio R, Laiho T, Sailas E, Lindberg N. Interventions following a high violence risk assessment score: a naturalistic study on a Finnish psychiatric admission ward. BMC health services research. 2017;17(1):26.
- Olsson H, Audulv A, Strand S, Kristiansen L. Reducing or increasing violence in forensic care: a qualitative study of inpatient experiences. Archives of psychiatric nursing. 2015;29(6):393-400.
- 182. Large MM, Ryan CJ, Singh SP, Paton MB, Nielssen OB. The predictive value of risk categorization in schizophrenia. Harv Rev Psychiatry. 2011;19(1):25-33.
- 183. Patchan K, Vyas G, Hackman AL, Mackowick M, Richardson CM, Love RC, et al. Clozapine in Reducing Aggression and Violence in Forensic Populations. Psychiatr Q. 2018;89(1):157-68.





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