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Beyond Symptoms – Measuring functioning in psychiatric patients and exploring influencing factors

NIKLAS HÖRBERG

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Abstract

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Functioning, the ability to perform important tasks in life, varies greatly among psychiatric patients. Factors such as comorbidity, personality disorders, trauma, personality and attachment style have been linked to functioning, but never studied together. This thesis aims to explore functioning in psychiatric patients by validating clinical measurement tools and identifying key factors influencing it.

Paper I evaluated the psychometric properties of the Sheehan Disability Scale (SDS) in a sample of 160 young psychiatric patients diagnosed with ADHD, Bipolar Disorder, or Borderline Personality Disorder. Exploratory factor analysis confirmed a one-factor structure, and the SDS demonstrated high internal consistency and concurrent validity. The study supports the tool's reliability and utility in clinical settings for assessing functioning.

Paper II evaluated the psychometric properties of the Early Trauma Inventory (ETI) in 243 young psychiatric patients and 56 controls. The factor analysis confirmed the previously seen four-factor model, and showed good to acceptable fit. Cronbach's alpha varied between 0.55 and 0.76, with higher values in clinical samples compared to controls. The test-retest reliability was good, and the test could discriminate between groups with different levels of traumatisation, supporting its discriminant validity. The Swedish translation of the ETI exhibited similar psychometric properties as both the original version and translations.

Paper III examined how childhood trauma and psychiatric comorbidity affect adult functioning - independently, through mediation or moderation. This was done in two samples consisting of 414 psychiatric outpatients and 100 non-clinical participants respectively. The study found that in clinical samples, both childhood trauma and comorbidity affected functioning, with comorbidity partially mediating the effect of trauma. In the non-clinical sample, only comorbidity was associated with functioning. No moderation effects were present in either sample.

Paper IV explored predictors of functioning and their combined predictive power. Temperament and character traits, attachment styles, childhood trauma and psychiatric comorbidity including personality disorders were assessed in 137 psychiatric outpatients. While many variables individually predicted functioning, only the temperament trait Harm Avoidance and the character trait Self-Directedness remained significant predictors in a multiple regression analysis, explaining about one-third of the variance. These findings emphasize the role of personality traits in understanding and predicting the functioning of psychiatric patients.

In summary, the studies in this thesis support that functioning and childhood trauma can be measured with good reliability using the SDS and ETISR-SF, and that personality factors may be important predictors of functioning in psychiatric patients.

Keywords: medicine, psychiatry, functioning, trauma, personality, attachment, comorbidity

Niklas Hörberg, Child and Adolescent Psychiatry, Akademiska sjukhuset, Uppsala University, SE-751 85 Uppsala, Sweden.

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To all the study participants and my patients, who have allowed me to gain some understanding of psychiatry.

The saddest aspect of life right now is that science gathers knowledge faster than society gathers wisdom.

Isaac Asimov

List of Papers

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

I. Hörberg, N., Kouros, I., Ekselius, L., Ramklint, M. (2015) The Swedish version of the Sheehan Disability Scale – a valid and brief measure of functioning. *European Journal of Person Centered Healthcar*e, volume 4, no. 1, p. 208-214 (2016)

II. Hörberg, N., Kouros, I., Ekselius, L., Cunningham, J., Willebrand, M., Ramklint, M. (2016) Early Trauma Inventory Self-Report Short Form (ETISR-SF): validation of the Swedish translation in clinical and non-clinical samples. *Nordic Journal of Psychiatry.* 2019 Feb;73(2):81-89.

III. Hörberg, N., Cunningham, J., Ekselius, L., Ramklint, M. (X) Exploring the role of childhood trauma and psychiatric comorbidity for adult level of functioning. *Manuscript.*

IV. Hörberg, N., Kouros, I., Ekselius, L., Ramklint, M. (2024) Beyond Symptoms - A Cross-sectional Study Exploring Functioning in Psychiatric Outpatients, *Psychiatry,* 2024 Sep 11:1-19.

Contents

Abbreviations

ADHD	Attention Deficit Hyperactivity Disorder
ASQ	Attachment Style Questionnaire
BBA	Borderline-Bipolar-ADHD sample
BP	Bipolar Disorder
BPD	Borderline Personality Disorder
C	Cooperativeness
CON	Confidence
DIS	Discomfort with Closeness
ETISR-SF	Early Trauma Inventory, Self-Rated, Short Form
GAF	Global Assessment of Functioning
HA	Harm Avoidance
K-SADS	Kiddie-Schedule for Affective Disorders and Schizophrenia - Present and Lifetime Version
MINI	Mini International Neuropsychiatric Interview
NEE	Need for Approval
NS	Novelty Seeking
PD	Personality Disorder
PRE	Preoccupation with Relationships
PS	Persistence
PTSD	Post-Traumatic Stress Disorder
QoL	Quality of Life
RD	Reward Dependence
REL	Relationships as Secondary
SCID-I CV	Structured Clinical Interview for DSM-IV-Axis I Disorders, Clinical Version
SCID-II	Structured Clinical Interview for DSM-IV-Axis II Disorders
SD	Self-Directedness (character trait)
SDI	Socio-Demographic Index
SDS	Sheehan Disability Scale
ST	Self-Transcendence
TCI	Temperament and Character Inventory
UPP	Uppsala Psychiatric Patient sample

Introduction

Functioning - what is it and why does it matter?

Functioning refers to how well a person can participate in society and perform important tasks in central areas of life. It relates to many important aspects of life – getting a job, managing everyday life, meeting a spouse and maintaining relationships – in short the person's ability to create the life he or she wants to live.

Functioning is a multifaceted concept, comprising a diverse range of skills and capabilities. A person may exhibit high levels of functioning in certain domains while encountering significant challenges in others (1, 2).

Numerous factors influence an individual's level of functioning, spanning from historical elements such as upbringing and early traumas to more enduring factors such as personality traits and attachment style, as well as dynamic factors such as current mental or physical health status, which will be discussed in detail later in the thesis.

While the concept of functioning as used in this thesis is primarily focused on the abilities and limitations of the individual, it is crucial to recognize the influence of societal and cultural factors, as well as the social support available to the individual. Although these factors may be theoretically distinct from an individual's level of functioning, they significantly influence what a person can realistically achieve. Social norms, cultural expectations, and the availability of support networks all play a role in shaping an individual's opportunities and limitations in life (1).

This thesis centers on young psychiatric patients, with the aim of shedding light on factors that influence their level of functioning, as well as how it can be effectively measured. Gaining a comprehensive understanding of these factors is important - not only for clinicians to better understand their patients, but also to design tailored treatment plans and social interventions, thereby improving opportunities for patients to lead the lives they want to live.

Functioning and Quality of Life

Functioning is, in some ways, related to quality of life (QoL). The specific definition of QoL varies (3), and in some contexts a distinction is made between emotional well-being and life evaluation (4). The World Health Organization (WHO) defines QoL as "*an individual's perception of their position in life in the context of the culture and value systems in which they live and in*

relation to their goals, expectations, standards and concerns" (5). Similar to functioning, QoL encompasses various aspects of life, such as employment, health, relationships, education, work environment, recreation, security, social belonging and freedom. (6). However, while the concept of functioning refers to a person's ability to perform certain tasks in life, QoL pertains to an individual's subjective perception of their life and situation.

It seems reasonable that in general, a lower level of functioning, which restricts a person's ability to live according to their desires, is reflected in a lower QoL. This is in line with research on different age groups and diagnoses, somatic as well as psychiatric (7-10), although it is also important to note that these studies found weak to moderate correlations at a group level, and that individuals with a lower level of functioning and/or disabilities can also live rich and fulfilling lives.

Functioning and psychiatric care

In psychiatry, clinical care and treatment are often based on the patient's current diagnoses and symptoms. Psychiatric diagnoses are primarily defined by sets of symptoms, although impaired functioning is also part of the diagnostic criteria for all psychiatric disorders (11, 12). In clinical practice, clinicians quickly note that, in some cases, symptoms or diagnoses say very little about a patient's level of functioning. This can vary greatly among diagnostically similar patients: one patient with a particular diagnosis may have attained higher education, hold a full-time job and maintain well-functioning relationships, while another with the same diagnosis may never have held a job, or may have constant relationship difficulties. This difference is of course crucial in understanding the patient and his or her situation. Also, the level of improvement in symptoms and functioning may differ, and it is important to note that symptom remission does not always result in full recovery of functioning (13, 14).

Assessments of functioning can be used to estimate the need for care (15), plan treatment or predict treatment results (11, 16), measure change over time, and evaluate treatment effect (16, 17). In many countries, disabilities can qualify individuals for social benefits, and level of functioning can also be a factor in determining an individual's need for communal social support or housing with specialized services.

Measuring functioning

Measuring functioning poses significant challenges: it involves many different areas of life, and functioning does not "occur" in a vacuum; there is also an interaction between the person and his or her environment.

Multiple methods are available for measuring functioning, some aimed at assessing a general, "global" level of functioning, others for testing specific

aspects such as cognitive functioning. Some assessments are self-reported, while others are conducted by healthcare professionals such as psychologists, doctors or occupational therapists, and each method has it's own advantages and disadvantages (18). The question of how to evaluate disability, and who has interpretative precedence, is also important for the understanding and definition of the concept itself (19).

The WHO has developed the International Classification of Functioning, Disability and Health (ICF), describing functioning as an outcome of the interaction between a person's health, and their personal and environmental factors. The level of functioning/impairment can be rated across a multitude of items in the four sub-categories: Bodily functions, Activities and participation, Environmental factors and Body structures (20). While grounded in the ICF, the WHO Disability Assessment Schedule (WHODAS 2.0) (2) has been developed as a more comprehensive and valid (21) tool for assessing health and disability across six domains.

For use in clinical practice it is especially important that measures are relatively simple and time-efficient, and some brief measures of global functioning such as the Global Assessment of Functioning (GAF) (11) and the Sheehan Disability Scale (SDS) (22) will be described in greater detail later in this thesis. The WHODAS 2.0 was not used in the study since there was no Swedish translation available when data collection began.

Theoretical framework

The bio-psycho-social model

The biopsychosocial (BPS) model, proposed by psychiatrist George L. Engel in 1977 (23), is now widely used in the medical community. This model challenged the prevailing mind-body dualism and reductionist views of the psychodynamic and biomedical perspectives of that era. Instead, the BPS model emphasized the significance of social, psychological, and biological factors, the fundamental assumption being that all three dimensions contribute to determining health or illness (see Figure 1 below for a graphical illustration). The model was originally thought to provide a blueprint for research and a framework for teaching as well as a design for action in the real world of health care (23).

According to the BPS model, each individual is born with a certain set of genes that, in turn, influence other factors, such as personality and hormone levels, and which can serve as both vulnerability and protective factors. This innate vulnerability (or framed as its opposite: resilience) interacts with the upbringing environment and events throughout life, and it is this interaction that determines the outcome. For example, traumatic events during childhood may increase the risk of a person later experiencing mental health problems,

just as a secure and stable environment or a certain genetic make-up may mitigate that risk. The gene-environment interactions encompass various theoretical perspectives (24), and the interplay is complex (25, 26). Socioeconomic factors have also been studied and can serve as both general risk and protective factors (27). Additionally, with regard to functioning, genes and social environment have been shown to interact in shaping cognitive ability and physical health (24).

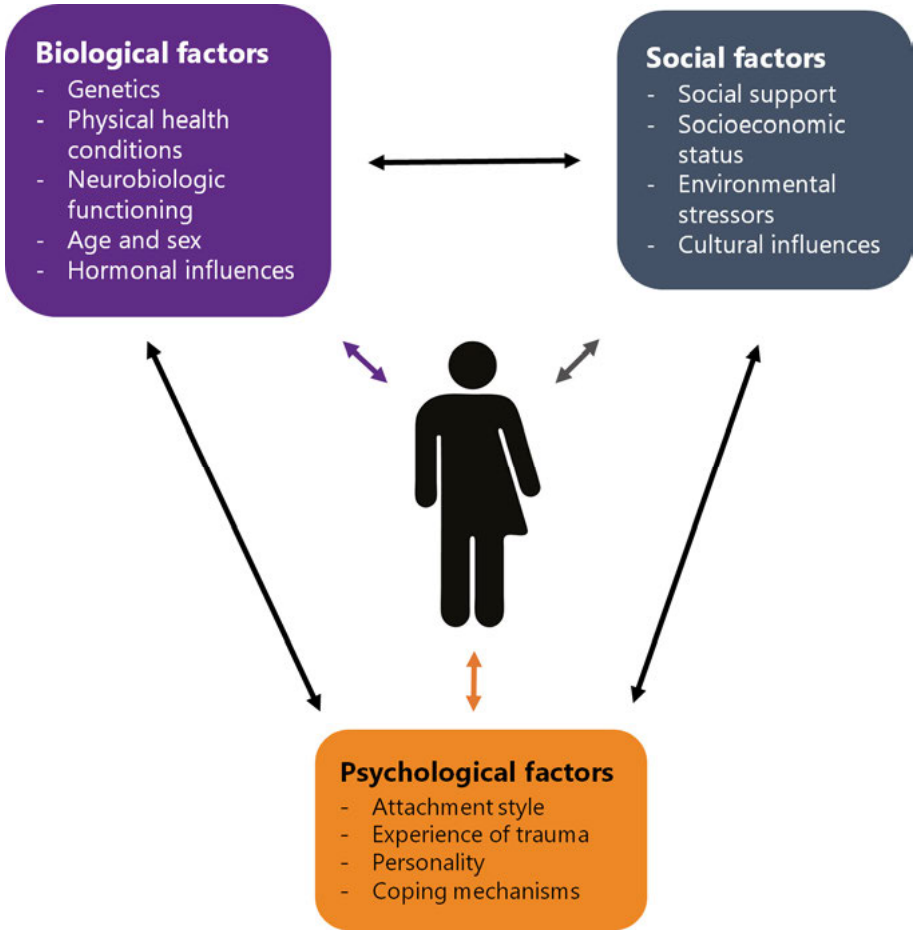


Figure 1. Schematic illustration of biological, psychological, and social factors that interact and together influence the individual.

Over the years, criticism of the model has emerged (28-30), including concerns about further dichotomizing biology and psychology (29), as well as it not being an empirically verifiable theory, and being unsuitable for use as a model in clinical decision-making (30). As in most cases, differences of opinion concerning the value of the BPS model in both science and clinical practice exist, with other researchers proposing ways to implement the model in a

more evidence-based fashion (31). Despite criticism and proposed updates of the model (32, 33), the core concepts remain the same today and the BPS model is considered by most to be relevant (24, 33-35); and it also forms the basis of the ICF (20). Although the model is widely accepted and its evidence base has grown, its implementation into clinical practice has been slower (34, 35).

The vulnerability-stress-model

The vulnerability-stress-model explains the development of psychiatric disorders as a consequence of a predispositional vulnerability interacting with life events and stressful experiences (36). The model is also known as the diathesis-stress-model, derived from the Greek word for predisposition: diathesis (διάθεσις).

According to the model, external stressors interact with a person's disposition, or vulnerability, determining the stress response. The model states that if the combination of stressors and an individual's vulnerability exceed a certain threshold - essentially when the stress surpasses the person's ability to cope - this may lead to the development of a psychiatric disorder, such as depression or anxiety (see Figure 2).

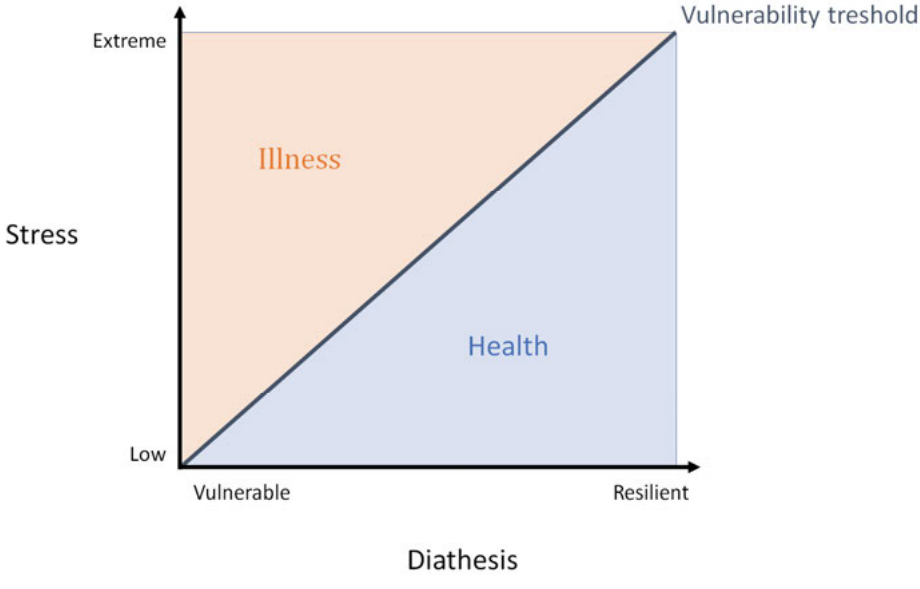


Figure 2. Graphical illustration of the vulnerability-stress model, showing how the combination of a certain level of stress and a certain level of vulnerability/resilience together determines the outcome: health or illness. The X-axis represents the degree of resilience (i.e., the opposite of vulnerability), and the Y-axis corresponds to the level of stress the individual is exposed to.

The model explains why individuals with more vulnerable predispositions are at higher risk of developing mental disorders than others, as they have a lower threshold for tolerating stress without experiencing negative psychiatric consequences.

Congruent with the bio-psycho-social model, the factors determining vulnerability in the vulnerability-stress-model can be social, biological and psychological. For instance, a person with a genetic predisposition for depression, an insecure attachment style or who is living in poor circumstances might have a greater risk of depression when facing new stressful life events.

Initially, only events occurring within the past year were considered stressors in the vulnerability-stress model (36). However, neurobiological research suggests that epigenetic factors and adverse experiences in early childhood can also constitute vulnerability factors that have lifelong effects on physical and psychological functioning (24, 37, 38).

Theoretical models and functioning

The BPS model has also been used to better understand the construct of functioning, shedding light on the various factors that influence it. As we will see later in the thesis, research has linked both biological, social and psychological factors to functioning. As previously mentioned, the ICF (1) is based on a BPS model, which later has been further discussed in terms of a BPS-ICF model (39).

The vulnerability-stress model provides a framework for understanding the interplay between stress and vulnerability leading to psychiatric symptoms. While direct research on the model's impact on functioning is lacking, it is plausible that stress not only contributes to psychiatric conditions but also adversely affects functioning for individuals with high vulnerability.

When combined, the stress-vulnerability model and the BPS model underscore the importance of considering multiple dimensions, and gives a better understanding of the different kind of stressors that are potentially relevant, and which factors might constitute vulnerability factors.

By considering the interplay between vulnerabilities, stressors, and environmental factors, these models may provide insights into how an individual's functioning may be influenced by their unique combination of biological, psychological, and social factors. In the following section, we will look more closely at the individual factors associated with functioning.

Causality, moderation, and mediation

When more than one factor influences the outcome being studied, this can occur in several different ways. The most straightforward explanation is that each factor independently affects the outcome, but this can also occur through other mechanisms such as mediation or moderation (40).

A clarifying example could be the different ways that childhood trauma and psychiatric comorbidity might influence level of functioning. The simplest explanation is that both traumatization and psychiatric comorbidity independently cause impairment (see Figure 3a). Another possibility is mediation: for example, childhood trauma leads to psychiatric morbidity, which in turn causes the impairment. However, partial mediation is more common, where the mediator only explains part of the effect (see Figure 3b). A third possibility is moderation: traumatized individuals with psychiatric disorders are more functionally impaired by the psychiatric comorbidity compared to those without childhood trauma (see Figure 3c). Another potential moderation path is similar: people who have experienced childhood trauma are more functionally affected by, for instance, depression than an equally depressed person without a history of traumatization is.

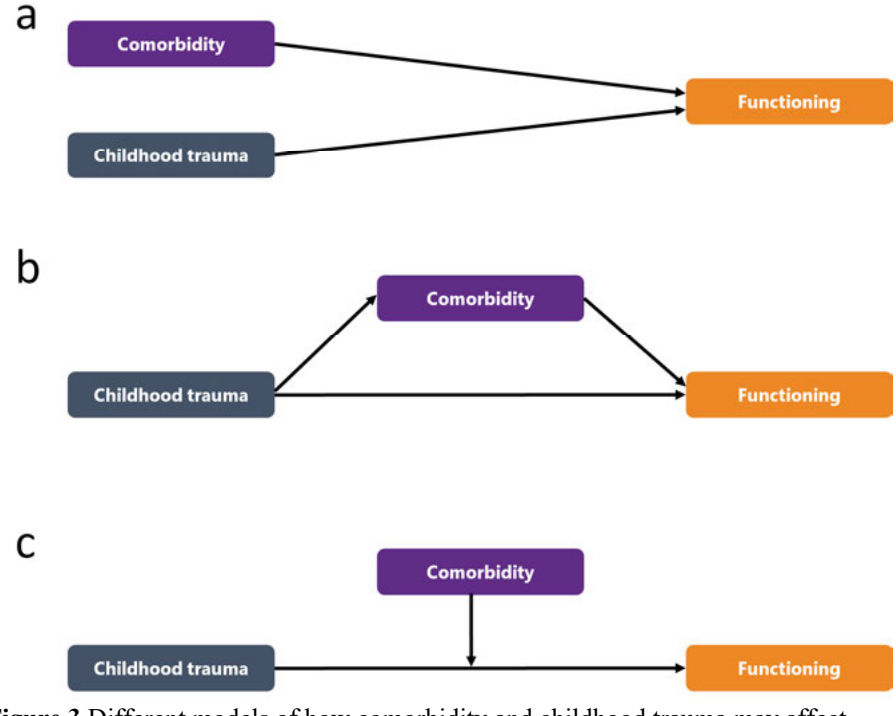


Figure 3 Different models of how comorbidity and childhood trauma may affect functioning: (a) regression model, (b) mediation model and (c) moderation model.

Factors influencing functioning

Psychiatric (co)morbidity

Apart from the psychological pain associated with psychiatric morbidity, patients with psychiatric disorders also suffer from functional impairment. This is perhaps not surprising, since impairment is incorporated in the definitions of psychiatric diagnoses; in both the 4th and 5th editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM), the diagnostic criteria for all disorders require that symptoms lead to clinically significant distress and/or impairment in social, occupational, or other important areas of functioning (11, 12).

Studies conducted in both primary care (41, 42) and specialized psychiatric care (43, 44) have demonstrated a connection between psychiatric morbidity and reduced functioning. Comorbidity, that is, having multiple diagnoses, has been found to be associated with greater functional impairment (41, 44, 45), as has having (at least) one personality disorder (44).

The severity of psychiatric symptoms also appears to affect functioning (42, 43, 46). This seems to apply to most psychiatric diagnoses such as depression, bipolar disorder, substance abuse, OCD and personality disorders (41, 42, 44-45, 47), although some diagnoses, for example depression and personality disorders appear to cause more functional impairment than, for example, anxiety disorders (41, 44, 46). The improvement in functioning that occurs as psychiatric symptoms improve suggests a causal relationship (42), which is in line with the criteria definitions in the DSM.

Measuring psychiatric morbidity – diagnostic procedures

Achieving an agreement on how psychiatric diagnoses should be defined has been a long historical process. In the 1970s, Gruze et al. developed a concept to improve the validity of psychiatric diagnoses, which aimed to make psychiatric diagnoses meaningful constructs that could be distinguished from each other (48). In modern psychiatry, diagnosis is often based on the DSM, published by the American Psychiatric Association. With each new version of the DSM, efforts have been made to increase the validity of diagnoses, and since the third version was published in 1980 (49), diagnoses have been based on sets of diagnostic criteria for each diagnosis. In the diagnostic process, the clinician (or the researcher collecting data) must then assess whether sufficient criteria for a particular diagnosis, and thus the diagnosis itself, are met or not.

Reliability is also discussed in the context of the diagnostic process. Test-retest reliability involves whether the assessor assigns the same diagnosis on different occasions (assuming the symptoms are the same), and inter rater-reliability whether different raters assign the same diagnosis(es) to a particular patient.

There are several diagnostic interviews to assist the clinician in making valid and reliable diagnoses. For most common psychiatric disorders, these

are, among others, the Mini International Neuropsychiatric Interview (MINI) (50) and the Structured Clinical Interview for DSM-IV Axis I Disorders, Clinical Version (SCID-I-CV) (51), and other in-depth interviews for personality disorders, such as the Structured Clinical Interview for DSM-IV-Axis II Disorders (SCID-II) (52). Since completion of the data collection for this thesis, new versions of the diagnostic manual (DSM 5 instead of DSM-IV) and some of the interviews (SCID-5 instead of SCID-I, and SCID-5-PD instead of SCID-II) have been introduced; however, these do not imply any major differences in the content of this thesis.

Diagnostic instruments are also psychometrically examined with regard to sensitivity (i.e., to what extent the true diagnosis is captured by the instrument) and specificity (i.e., to what extent the absence of the diagnosis is correctly excluded by the instrument). The "LEAD concept" (Longitudinal, Expert, All Data available) (53), where, for example, anamnestic data, status, examinations, and the results of diagnostic interviews are considered by experts, is considered the gold standard for psychiatric diagnosis, and is recommended by SBU, among others (54). Other studies have shown that personality disorder diagnoses also can be made with good validity and reliability using interviews developed for this purpose (55).

Trauma

A severe trauma during childhood is a terrible experience in many ways. In addition to the pain of the trauma itself, the psychological impact can remain for years, sometimes throughout life. Beyond psychiatric diagnoses, childhood trauma is another factor that has been shown to affect functioning in various ways, and different characteristics of the trauma seem to influence the extent of its impact.

Descriptions of post-traumatic symptoms: a short history

Trauma has been a part of many people's lives throughout history, and it has been portrayed in writings by early Egyptian physicians and later ancient Greek writers such as Homer (56). Although the type of stressors has changed with time, people's reactions to severe stress has, in many ways, been similarly described, depicting many of the symptoms we today would associate with psychological trauma (56). The first scientific descriptions of post-traumatic symptoms, at the time called Cannonball wind syndrome (or in French: "syndrome du vent du boulet"), are from the Napoleonic war. Many scientific descriptions have been recorded since, often describing a mix of psychiatric and somatic symptoms. The symptoms were believed to be related to heart damage after war experiences, which is evident by the terminology used at the time: "Soldier's Heart", "Irritable Heart Syndrome", "War neurosis" or "Shell shock" (56-58). Sigmund Freud used the term "anxiety neurosis" in the same

context, although with a different view of the condition's psychological pathogenesis (58). Freud believed that it was repressed memories of the trauma that caused diverse somatic symptoms, whereas many others believed that the symptoms originated from a physical damage to the nervous system caused by the trauma. The perhaps most well-known trauma related diagnosis, post-traumatic stress disorder (PTSD), was introduced into the DSM-III in 1980.

Although much research has focused on PTSD and other trauma-related diagnoses, it is important to note that many forms of post-traumatic symptoms do not meet the specific criteria for PTSD, but still warrant clinical and scientific attention. This is due to the significant suffering experienced by those affected, its impact on public health, and, relevant to the theme of this thesis, the increased risk of developing psychiatric disorders and impaired functioning. This is especially pertinent since the impact of trauma can be most profound during childhood.

Childhood trauma

Human brains, like the brains of many other animals, are plastic and continue to develop throughout life through an interaction between genes and the environment. This openness to external influences is often adaptive, and impacts cognitive and emotional development, enabling the brain to adapt and thereby enhancing survival in various environments. Plasticity is most pronounced during the first years in life, but decreases over time; even if it is still present in adulthood it is much less prominent. However, plasticity also poses risks, since early life experiences are particularly influential. Environmental disturbances, especially if they are extreme, can disrupt normal developmental pathways or trigger processes that lead to maladaptive cognitive or emotional behaviours and, in severe cases, mental illnesses. In this context, the consequences of childhood trauma can be seen as the dark side of neuroplasticity. (59)

Childhood is an especially sensitive period with high plasticity, and trauma during this time can affect the brain and body in various, often negative, ways during childhood (59, 60), but also in adulthood (61). As a striking example of the impact of childhood trauma, Silverman et al. found that approximately 80% of young adults who had been victims of childhood physical or sexual abuse met the criteria for at least one psychiatric disorder at age 21 (62).

Although initially not a subject of as much interest as war trauma, the body of research on childhood trauma has grown substantially in the past decades. It has proven to be an important public health problem, since it is associated with a range of adverse psychiatric outcomes as well as somatic discomfort (63).

Childhood trauma is also an important public health concern because it is so common; in the Adverse Childhood Experience (ACE) study, 52% of a large US population had experienced at least one adverse childhood experience (64).

The BPS model has also been thought to be relevant in understanding the impact of childhood trauma (65), and there is a substantial body of evidence backing up the claim that the experience of trauma is indeed linked to both biological, psychological, and social factors. There are several types of studies - from psychophysiological via neuroimaging and neuroendocrinological to genetic and epigenetic - indicating a biological basis for the post-traumatic symptoms (for a review, see Pitman et al. (61)).

Different characteristics of trauma and their impact

In research, traumas are often categorized as either general trauma, sexual abuse, physical abuse, or emotional neglect, even though definitions and categorizations may vary somewhat. However, those who report one type of trauma often report several other types (66) and sometimes the term Adverse Childhood Experience (ACE) (64) is used.

There has been much research, sometimes with conflicting results, on how trauma characteristics such as age at trauma, perpetrator, number of traumas or recency, affects outcomes.

Number of traumas

Generally, the more different types of childhood traumas reported, the more likely the adult respondent is to report engagement in risky health behaviours, such as substance abuse (67). Experience of emotional neglect as well as sexual and physical abuse in childhood has been related to adult suicidal behaviours (68, 69). Both the number of traumatic events, and the number of different trauma types experienced are associated with outcome. A dose-response relationship has been observed between frequency of abuse and several adult psychiatric disorder groups (70, 71).

Age at trauma

Some studies have examined "sensitive periods" when trauma may have more severe consequences, often focusing on PTSD or major depressive disorder (MDD). For instance, trauma at a younger age has been linked to higher PTSD risk (72), though findings are mixed. Maercker et al. (73) found no age-related differences in PTSD risk but noted a higher risk of depression with earlier trauma. In a large prospective study, the age when trauma was experienced was not associated with adult psychiatric history or functional outcomes (74), but in other studies of early trauma up to the age of eight, recency and accumulation seemed to be the most important factors affecting the outcome (75). The results may also vary depending on the outcome variable studied. For example, the risk of developing MDD is greater if the trauma occurs during childhood rather than adolescence, but for PTSD, the results are mixed, with some studies showing a more pronounced risk after 13 years of age (73, 76).

Type of trauma

In a review, different trauma types were seen to have different risks of being associated with certain diagnostic groups (77). The risk of developing PTSD can differ with regard to specific trauma types. A striking example is a study of German women, which found that experiencing rape was 17 times more likely to result in a PTSD diagnosis than experiencing a serious accident (73). In another study, sexual abuse generally led to worse outcomes than physical abuse, which, when controlling for individual, social and family factors, was not a significant predictor of later mental health problems (78). Childhood trauma can also lead to other consequences, such as deliberate self-harm, where emotional abuse appears to be a risk factor, perhaps mediated by self-hatred (79).

Perpetrator

The type of perpetrator can also influence the outcome of trauma. While earlier consensus suggested that a close relationship with the perpetrator resulted in worse outcomes (80), there is also more recent research that indicates, for both sexual and physical trauma, that outcomes can be worse when the perpetrator is a non-caregiver compared to a caregiver (81).

In summary, the literature is in some respects inconsistent, and there has been debate over what best explains psychiatric symptoms following trauma. Factors such as sensitive periods, type of trauma, recency, accumulation and perpetrator all appear to play a role.

Trauma and functioning

Childhood trauma is associated with functional impairment across various contexts (82-86). In several diagnostic groups, there is a dose-response relationship between the level of traumatization and the degree of impairment (70). A review of studies on maltreated children indicated impaired academic performance and social skills (87). In patients with borderline personality disorder (BPD), apart from abuse being common, the severity of sexual abuse and childhood neglect is related to the level of psychosocial impairment (88), and childhood maltreatment is associated with employment disability (84). The association between trauma and impairment has been observed in both prospective studies and population-based materials (74). Furthermore, psychosocial functioning does not seem to improve over time in trauma-affected patients, as it does in non-traumatized patients (82).

How Are Trauma and Functioning Related?

The mechanisms connecting childhood traumatization and functional outcome are not yet fully understood, but it is believed that causes as well as consequences of the trauma may play a role (89). Firstly, there may be some shared risk factors for both childhood traumatization and impairment: some related

to the child's parents (e.g. young parental age, psychiatric morbidity or drug or alcohol abuse, sociodemographic factors, traumatization), others to the child itself (behavioural or mental problems, temperament or personality, and also genetic factors). Secondly, the consequences of the trauma itself may affect functioning. Several mechanisms have been proposed for this: through clinical symptoms of psychiatric disorders, development of insecure attachment styles, increased risk of revictimization, negative effects on brain development and HPA axis (Hypothalamic–pituitary–adrenal axis) and increased risk of neurocognitive or social cognitive deficits. For a review on the subject, see Cotter et al. (89). Other examples of general vulnerability after trauma exposure are the development of insecure attachment styles and insufficient self-regulation strategies (89), especially emotional dysregulation (90).

Comorbidity, trauma and functioning – mediation or moderation?

Trauma and comorbidity

The link between psychiatric morbidity and childhood trauma is well established; the risk of being afflicted with psychiatric disorders is markedly increased when childhood trauma is present (91-93). Early trauma has been linked to various psychiatric conditions, including mood disorders (94-98), attention deficit hyperactivity disorder (ADHD) (99), substance abuse (96) and psychosis (100), in addition to PTSD (98, 101) where trauma is also a mandatory diagnostic criteria (12). Regarding personality disorders, higher levels of childhood trauma have been associated with BPD (102-105), and a causal relationship has been suggested (106), although traumatization is not part of the diagnostic criteria. However it is well known that sexual abuse is common among patients with BPD (107), and certain trauma types are associated with symptom severity (102, 108).

Mediation, moderation or direct effect?

As described earlier in greater detail, it is conceivable that the combination of different factors can affect the outcome in more than one way. This influence may occur through direct causal effect or through mediation or moderation. We have now seen that both psychiatric comorbidity and childhood trauma affect functioning, but these factors are seldom studied together and the mechanism by which they affect functioning is not clear.

A limited number of studies have been conducted on mediation in this context. Mediation effects on functional impairment have been studied in a cohort of gainfully employed persons in the Netherlands, where childhood trauma was shown to influence adult work functioning (measured as absenteeism or presenteeism), partly mediated through depression or through comorbid depression and anxiety (109). One potential mechanism for this mediation could be that childhood trauma leads to insecure attachment styles and insufficient self-regulation strategies (89), which in turn lead to psychopathology (90) affecting functioning. In combat veterans PTSD and depressive symptoms were

found to mediate the relationship between childhood maltreatment and both functioning and health-related QoL (110). In a study using a structural equation model, insecure attachment was a mediator between childhood trauma and symptoms' of eating disorder (111).

As mentioned, it is also possible that trauma and comorbidity, through moderation, together have a stronger negative effect on functioning, i.e., that persons with experience of one are more vulnerable to the detrimental effects of the other. Studies support that previous experience of childhood trauma can aggravate the impact of current psychiatric disorders on functioning in several ways. Firstly, childhood trauma is associated with more severe psychiatric symptoms (88, 89, 112, 113) and may worsen of the course of depressive and anxiety disorders (114). Secondly, it can have negative effects on the developing brain, and is associated with neurocognitive deficits (89), and also affect social cognitive functioning negatively (115). Both these factors might contribute to impairment and make coping with psychiatric symptoms more difficult, a pattern that has been observed in bipolar patients (116). Among BPD patients, social cognitive deficits are thought to be aggravated by childhood trauma (117), and it has been suggested that social cognition moderates the impact of childhood abuse on adult functioning (89).

Another aspect of moderation is that comorbidity can worsen the effects of trauma on functioning. Comorbid depression predicted both symptom severity and impairment from PTSD (118), and comorbid depression has been shown to attenuate executive function deficits (119). Indirect support for comorbidity moderating the effect of trauma on functioning is that pre-existing depression or anxiety is a risk factor for developing PTSD (with associated impairment) following trauma exposure (120), and both depression and anxiety have a negative effect on the remission rate of PTSD (121, 122). It has also been shown that persons with comorbid depression have more posttraumatic symptoms than those without depression (123), although the results are mixed (124).

Summing up, childhood trauma and psychiatric disorders frequently coincide and both impact functioning. Further, there is some evidence indicating potential mediation and moderation effects in this relationship. There are no published studies about how a broader range of psychiatric comorbidities mediates the effect of childhood trauma on functioning. Similarly, there is a lack of mediation studies examining functioning in social and family life or among non-working populations. Moreover, there is a dearth of research investigating the moderation effects of childhood trauma and comorbidity on functioning.

Measuring trauma

Assessing childhood trauma in adults presents challenges, such as the accuracy of recall and the reluctance to report trauma face to face because of associated negative feelings with a risk of false negative results (125), and the validity of recalled trauma memories has been questioned (126-128). The use of

self-report questionnaires is a way to address one of these problems, and in clinical settings it is difficult to see other realistic and more reliable ways to measure childhood trauma other than through the patient's own recalled memories.

A population-based study that compared ACEs prospectively assessed throughout childhood and retrospectively recalled in adulthood showed moderate agreement, even though both were associated with midlife outcomes (129). However, recollections may be influenced by state effects such as depression and stress (130), as well as traits such as neuroticism and agreeableness (129).

To meet the need for valid and reliable tools for the assessment of childhood traumas, several interviewer-administered and self-report instruments have been developed. The majority have acceptable psychometric properties (for reviews see Roy et al. or Pietrini et al.) (131, 132). The Early Trauma Inventory (ETI) (133), which is examined in this thesis, is one such instrument.

Attachment

Attachment is another relevant factor, both in terms of how it can be affected by trauma and how it influences functioning.

Attachment theory

The development of attachment theory

Attachment theory, originally proposed by John Bowlby, describes the way humans relate to important others and the reaction patterns when confronted with separation or loss in these relationships (134). Although Bowlby's original theory was intended to apply throughout life (135), attachment research initially focused mainly on the relationship between the infant child and the primary caregiver. Attachment during infancy is thought to serve an important purpose, which seems logical in an evolutionary perspective: keeping the defenseless infant close to and cared for by an "attachment figure", protecting it from danger and seeing to its primary needs (136).

Attachment theory suggests that one's experiences in close relationships form a "mental depiction" of how relationships work, termed an "internal working model". This model is initially shaped by the relationship with the primary caregiver(s), but over the course of a person's life, other significant relational experiences are thought to be incorporated into the mental model, although the emphasis on childhood experiences in shaping adult attachment has been criticized (137). A history of interactions where the attachment figure is consistently responsive typically serves as a foundation of secure attachment (138). If, on the other hand, the attachment figure is unavailable or unresponsive, secondary attachment strategies develop, characterizing insecure attachment.

Mary Ainsworth, who worked with Bowlby, developed the Strange Situation protocol as a standardized method to determine a child's attachment style. This was conducted through observing and assessing the child's reactions to, and interaction with, the primary caregiver and a stranger in different constellations (138).

Today, attachment theory is not only applied to infant-caregiver relationships, but also aims to explain features of personality and social functioning relevant to close adult relationships (135).

Attachment styles

Attachment can be categorized into different styles, based on the dimensions attachment anxiety and attachment avoidance (139). Low levels of both indicate secure attachment, while high levels of at least one of the dimensions indicate insecure attachment (139). In the general population, secure attachment is thought to be most common, followed by avoidant and anxious (58%, 23% and 19% respectively (140)).

As the theory has evolved, several ways to sub-categorize different insecure attachment styles have emerged. In some instruments, the insecure domain of avoidance can be divided into the sub-domains of Discomfort with Closeness (DIS) and Relationships as Secondary (REL), and the insecure domain of anxiety can be divided into Preoccupation with Relationships (PRE) and Need for Approval (NEE). This is the case in the Attachment Style Questionnaire, which is used in this thesis (141).

Attachment patterns seem to be fairly stable over time, especially in the absence of negative events. Waters et al. present three long-term longitudinal studies, showing that attachment security is significantly stable in two of these, and discontinuity was related to negative life events and circumstances in all three (142, 143). In one retrospective study of college students, adult romantic attachment was best accounted for by childhood attachment, more so than abuse history (144). Attachment also seems to be more stable when measured dimensionally rather than categorically (145).

In summary, a rich body of research on attachment has accumulated over the years. Attachment theory has grown from a framework in which to understand the early establishing of caregiver relationships, via a concept also applied to adult relationships, into a comprehensive framework for understanding important aspects of interpersonal relations, even becoming part of some psychological treatments (146).

Attachment and psychopathology

Unsurprisingly, numerous studies have shown a covariation between insecure attachment and several psychiatric disorders. Insecure attachment seems to increase the risk of developing a cognitive framework predisposing for depression (147), and is correlated with bipolar disorder (148), depression (149)

and eating disorders (150). Correlations can also be seen with personality disorders in general (151). Several studies have been carried out specifically on BPD, consistently finding correlations with different types of insecure attachment (for a review, see Agrawal et al. (152)). At a symptom level, attachment can affect the severity of symptoms. For example, security in peer and parent relationships protects against the negative effects, measured as trauma-related symptoms, of childhood sexual abuse in college females (153).

Functioning in connection with trauma and attachment

In a systematic review, although the results were mixed, attachment was shown to be associated with functioning in patients with severe mental illnesses, although the effect sizes were small (154). Studies linking both childhood trauma and attachment with functioning have mostly emphasized social functioning. One longitudinal study of young adult survivors of childhood sexual abuse found that avoidant attachment correlated with lower satisfaction in romantic relationships (155).

Mediation or moderation?

There is evidence suggesting that mediation and moderation are also present when it comes to attachment and trauma. Regarding mediation, insecure attachment has been shown to mediate the relationship between childhood trauma and psychiatric symptoms (111), academic performance (87), as well as psychological and interpersonal functioning (156), including in partner relationships (157).

Regarding moderation, it has been proposed that secure attachment could serve as a protective factor. Lowell et al. demonstrated that childhood maltreatment and attachment style are significant predictors of later negative emotional and behavioural outcomes. Attachment significantly contributed to the relationship between childhood maltreatment experiences and later outcomes. A possible interpretation is that secure attachment might serve as a protective factor against negative emotional and behavioural outcomes (158). This is supported by other studies showing that security in peer and parent relationships can protect against the negative effects of childhood sexual abuse (153) i.e. through being a moderating factor.

Measurement of attachment

The gold standard for the assessment of attachment style in adults is generally considered to be the Adult Attachment Interview (AAI) (159). However, shorter and more time-efficient self-report measures are also commonly used in research, particularly in larger samples. One example of such a measure is the Attachment Style Questionnaire (ASQ) (141), which assesses attachment style dimensionally.

Personality

What is personality - historical perspectives and modern models

Sometimes, we instinctively know how someone we are familiar with would react in a certain situation, and we are just as certain that another person we know would react completely differently under the same circumstances. But why do people feel and react so differently? How is it that children seem to have preferences and reaction patterns even before they have been shaped by their surroundings, while their siblings can act and react entirely differently? The question of why people are the way they are – their personality – is likely something humanity has pondered for as long as we have existed. It has been documented in writing (as is often the case) at least since the time of the ancient Greeks.

In antiquity, and well into the Middle Ages, it was believed that the balance of bodily fluids determined both health and personality. Regarding temperament, people with an abundance of phlegm were thought to be calm and passive, those with an excess of blood were enthusiastic, active, and social, those with a predominance of yellow bile were aggressive and short-tempered, and those with high levels of black bile were tired and melancholic – the word melancholy itself deriving from the Greek for black bile, μέλαινα χολή (melaina kholé).

Several other more scientifically based models of personality have arisen, employing different paradigms: some are typological and based on personality categories, while others are dimensional and focus on different personality traits. Consequently, there cannot be one definitive definition of personality, since it varies somewhat according to the paradigm. However, the American Psychological Association's definition of personality captures the central aspects of what the concept entails: *"the enduring configuration of characteristics and behavior that comprises an individual's unique adjustment to life, including major traits, interests, drives, values, self-concept, abilities, and emotional patterns."* (160)

Since the 1980s, the most widely accepted model of personality in the scientific community has been the trait-based Five Factor Model (FFM). It was developed based on previous research that utilized a lexical approach. The underlying idea behind the lexical model was that all existing personality traits are described in human language. To reduce the many words describing personality to a reasonable number of traits, they were grouped and then subjected to cluster and factor analyses in several steps. Using this method to the distilled the many different words used to describe personality, the Five Factor Model ended up with five broad personality traits: neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness (161-163). All of these dimensional traits can be more or less pronounced in each individual.

Criticism of the Five Factor Model

Criticism has, however, been directed at the FFM, among others by Robert Cloninger (163) who criticized the FFM on several points. Firstly, he argued that it fails to capture certain personality domains such as individual autonomy, moral values, and other aspects of maturity and self-actualization, some of which are particularly important in the context of personality disorders. Secondly, he claimed that the concept of neuroticism is too heterogeneous, especially in clinical settings, since it includes multiple components such as anxiety, hostility, depression, self-consciousness, impulsiveness, and general emotional vulnerability. This heterogeneity makes it difficult to distinguish between individuals with personality disorders and other patient groups. Thirdly, Cloninger argued that the traits in the FFM do not reflect underlying neurobiological correlates. He pointed out that "*drugs that reduce scores on measures of neuroticism, such as alcohol and benzodiazepines, also consistently reduce scores on measures of introversion, suggesting that these dimensions are not etiologically independent, but rather share biological determinants*" (164).

Temperament and character

Cloninger aimed to create a model of personality that considered both neurobiological factors and social and cognitive development. He proposed what he calls a "psychobiological model" of the structure and development of personality (164) which could better differentiate between individuals with and without personality disorders (165). This model incorporates the domains of temperament and character (166).

Temperament

Temperament is the heritable determinant that manifests early in life. The structure of temperament in Cloninger's model was inferred largely from genetic studies of personality and neurobiological studies of the functional organization of brain networks regulating classic and operant learning responses in rodents. Temperament constitutes a person's preconceptual biases in habit formation and perceptual memory, for example, concerning responses to appetitive or aversive stimuli. According to the model, the phenotype of personality develops as a result of constant interactions between individual and environmental influences. Since personality is shaped by the interaction between the environment and the individual, and how the individual is shaped is influenced by these types of learning processes, these factors (preconceptual biases in habit formation and perceptual memory) are crucial for personality development: "*Therefore, differences between individuals in the adaptive systems involved in the reception, processing, and storing of information about experience define personality in general.*" (166)

Cloninger's model includes four dimensions of temperament: Novelty Seeking (NS), Harm Avoidance (HA), Reward Dependence (RD), and Persistence (PS). Novelty seeking is characterized by exploration in response to novelty, impulsive decision-making, extravagance in approach to cues of reward, quick loss of temper, and active avoidance of frustration. Harm avoidance is defined by pessimistic anticipatory worry, passive avoidant behaviours, such as fear of uncertainty and shyness with strangers, and rapid fatigability. Reward dependence is marked by sentimentality, social attachment, and dependence on the approval of others. Persistence is characterized by perseverance despite frustration and fatigue.

Character

The other domain in Cloninger's model is character, which, as mentioned above, arises from the interaction between temperament and environment. Character development occurs through a process called insight learning or reorganization of self-concepts, defined as "*the development of a new adaptive response as a result of a sudden conceptual reorganization of experience*" (165). This process describes how individuals adapt and reorganize their self-concepts based on new insights, ultimately shaping their character.

There are three character dimensions in Cloninger's model, each related to different aspects of self-concept: Self-directedness (SD), which pertains to the self; Cooperativeness (C), which pertains to the self in relation to others; and Self-transcendence (ST), which pertains to the self in relation to the world as a whole. Self-directedness is associated with agency - the ability to set goals and act consistently and adaptively to achieve them. Cooperativeness refers to social tolerance, empathy, interest in others, and helpfulness. Self-transcendence involves multiple stages, including a reduced focus on the self, transpersonal identification with nature, and spiritual acceptance.

Regarding the spontaneous development of temperament and character traits over longer periods, there is evidence that all traits except for HA change over time, with SD, for example, increasing (167).

Measuring temperament and character

The first instrument developed to measure temperament was the Tridimensional Personality Questionnaire (TPQ) (168), which was soon further developed into the Temperament and Character Inventory (TCI) (166). The TCI measures all the aforementioned temperament and character traits. Each temperament and character domain also has sub-categories, called facets, which describe different aspects of the trait. The TCI has since been revised (TCI-R) (169) and validated in various translations, including French (170, 171), Spanish (172), Italian (173), German and Swedish (174), with good validity being reported.

Criticism of Cloninger's model and the TCI

However, not all research has shown consistency with the seven-factor model in the first version of the TCI, or its underlying theories (175-177). Regarding the TCI-R (with 51 new or revised items and 189 items identical to the first version of the instrument), a validation study has criticized the instrument (178). It suggests that there are shortcomings, and that the hypothesized associations of TCI-R facet scales to domains were not supported by confirmatory or exploratory factor analyses, or the hypothesized designation of items to domain scales. In summary, according to Farmer et al., the TCI-R suffers from the same psychometric weaknesses as the TCI, also casting doubt on the model itself. Additionally, several genetic studies provide a more mixed picture that does not fully align with the theory's division of a genetically determined temperament and a character shaped by environmental factors (179, 180). A more detailed critical discussion of the model can be found in the aforementioned article by Farmer et al. (178), who argue that the model has "significant flaws". However, a response to this criticism has also been presented by Cloninger (181).

In summary, there is a large body of research supporting the validity of the model but also studies that are more critical, making it difficult to ascertain how many of the identified weaknesses lie within the psychobiological model of temperament and character itself, and how many are due to the instruments measuring it.

Cloningers model and the FFM compared

As in all science, it is important to remember that a model is just that – a model, a simplified description of extremely complex relationships. In this case, the psychobiological model represents the intricate interplay between biological, psychological, and social factors that contribute to shaping the complex construct we call personality. Even if the model's clear-cut division between genetically determined temperament and more environmentally influenced temperament was not 100% accurate, the model could still be valid, in the sense of being useful. The fact that it has been successfully used in a large number of studies support this.

Furthermore, since the psychobiological model is probably not alone in having flaws, it becomes interesting to compare its validity with other models for conceptualizing and measuring personality. There has been independent comparative analyses of different models of personality, and the instruments measuring them. To quote two researchers who compared the FFM and Cloninger's psychobiological model:

> Just as different brands of hand soaps differ from one another in their color, smell, and feel, personality inventories differ from one another in a host of ways, including the number and nature of their items and the number and nature of the scales they provide. A latent assumption of many test developers

> seems to be that these differences make comparisons impossible, and every inventory has its unique niche. As with soaps, however, some features of inventories are more crucial than others. Soaps should clean as effectively as possible, while not harming the user's skin. And inventories should predict important life behaviors and outcomes as effectively as possible, for any given amount of testing time. (181)

In the ambitious study to test the validity of 11 different models of personality (which of course is difficult to distinguish from the validity of instruments for measuring personality according to the models), comparisons were made based on several different approaches. The study had three parts: one testing the ability to predict behaviours, another examining agreement with ratings made by significant others, and a third investigating agreement with criteria related to mental health. Among the tested instruments were the NEO-PI (for the FFM model) and the TCI (for Cloninger's psychobiological model).

No specific measure was deemed generally superior. However, the TCI stood out positively in predicting "abnormal tendencies" and "criteria related to mental health." Although there were differences between different criteria regarding this, the TCI was particularly good at certain traits. Overall, the various instruments were fairly equivalent, and all tests except two across-study mean validities fell in the remarkably narrow range of .42 to .45. It should be noted, however, that although most inventories differ very little in terms of average validity across many criteria, the instruments show clearer differences in validity when compared with specific criteria. For example, as mentioned above, the TCI was better than average at predicting sociopathy, magical thinking, and borderline traits (181). The authors state: "*Psychologists often prefer the particular model of personality with which they are most familiar from training and clinical experience. Such preferences are tolerable because there is extensive descriptive overlap among most multidimensional models of personality, and no available model is consistently superior to the others for all purposes*".

In other studies, correlations between various personality traits in both models are found, along with significant differences (182). However, personality traits in both Cloninger's model and FFM demonstrate validity in predicting the clinical presentation of conditions (183), indicating the usefulness of both models. Recent genetic research also supports the existence of a genetic link to temperament (184), and neuroimaging studies show that both temperament and character traits explain differences in the brain's resting-state functional connectivity, although this is not unique to Cloninger's model (185).

The choice of personality model in this thesis

Within the scope of this thesis, the TCI personality assessment instrument was utilized and thereby also the psychobiological model, encompassing its strengths and weaknesses. This was based on the assumption that some kind of innate temperament probably exists, and that personality is shaped through

the interaction between this temperament and the environment. However, it is difficult to determine how well the TCI distinguishes between these two aspects of personality, and we therefore do not differentiate between temperament and character in our analyses. Additionally, the facets, i.e. the subscales of the various temperament and character traits, are not utilized as criticism has been directed towards these based on factor analyses.

Personality in relation to comorbidity and functioning

Since personality influences most aspects of an individual, including their thoughts, feelings, and actions, it is not surprising that there are clear links to both psychiatric morbidity and functioning. In a meta-analysis of studies on the psychobiological model, several personality traits, particularly HA and SD, were found to be associated with psychiatric morbidity (186).

Regarding functioning, similar results were observed, with high HA and low SD being linked to impairment, high persistence with career success, and SD and C with social functioning (187). SD consistently predicted impairment, even after controlling for comorbidity and executive functioning. However, dimensions with a more interpersonal focus, such as RD and C, had little or no effect on functioning in another study (188). Generally, detrimental effects of temperament or character were observed only at the extremes of the dimensions, except for RD, SD, and C, which were consistently beneficial, and HA and ST, which were consistently harmful (187).

These findings do not only apply to the psychobiological model. When personality traits (such as conscientiousness, extraversion, and neuroticism from the FFM) are studied alongside socioeconomic status and cognitive ability, they are at least as predictive as IQ, and more predictive than socioeconomic status regarding divorce, occupational attainment, and mortality (189).

Personality: state, trait and treatment

There are several studies investigating the relationship between psychiatric disorders, treatment, and changes in temperament and character. One of the main challenges in these studies, especially concerning depression, is discerning what constitutes the underlying "true" personality (trait) and what is temporarily affected by e.g. a depression (state). For example, during an ongoing depression, state effects can lead to different responses to personality questions compared to if the same individual were euthymic.

Treatment of depression

Many studies have shown treatment effects in the form of lower HA and/or higher SD. For example, in a study of inpatients with depression treated with maprotiline, treatment was found to increase SD and decrease HA in responders, which could represent state effects (190). Similar results have been observed in other pharmacological treatment studies (191). The consensus seems to be that both state and trait effects are likely present; that is, patients with

depression differ in baseline personality from controls at group level, but the depression itself also influences how individuals rate their personality (192, 193). Another study supporting this hypothesis found that HA decreased with depression treatment but remained higher than in matched controls (194).

Treatment of Anxiety Disorders

In treatment studies of anxiety disorders and eating disorders, described in more detail below, these generally show effects of lower HA and, in the case of cognitive behavioural therapy (CBT), also higher SD. However, in a pharmacological study, SD for non-responders was observed to decrease (195). In a study comparing cognitive therapy with treatment as usual (most often SSRIs) for social phobia, HA was found to decrease regardless of the treatment form, but SD increased only after psychotherapy (196). A randomized clinical trial of CBT for bulimia nervosa showed significant decreases in HA and increases in SD (197). A treatment study of CBT for eating disorders showed that several personality traits, including HA, PS, SD, and ST, were affected by treatment, independent of changes in BMI (198). A study of patients with OCD in which participants received SSRIs, and nearly half also received CBT, showed lower HA after treatment but no difference in SD (199). A pharmacological treatment study found that all patients initially had higher HA than healthy controls, but those who became non-responders also had lower PS, SD, and C at baseline. Both before and after treatment, those who achieved remission had higher HA than those who did not. Among non-responders, SD and C worsened with treatment (195).

Personality disorders

As previously mentioned, there is a connection between personality and various common psychiatric conditions. However, aspects of a person's personality can in themselves constitute a psychiatric disorder. Personality Disorders (PDs) are psychiatric conditions characterized by maladaptive personality traits that are pronounced, enduring, and cause distress and/or impairment for the individual. Although personality and personality traits exist on a continuum, personality disorders in the DSM are primarily defined through categorical diagnoses, although there is an alternative model in DSM-5 that adopts a more dimensional approach (11, 12). Regarding the conventional categorical classification of personality disorders, the DSM describes ten different PDs grouped into three clusters based on similarities between the diagnoses. The eccentric Cluster A includes paranoid, schizoid, and schizotypal PDs; the dramatic Cluster B includes narcissistic, histrionic, borderline, and antisocial PDs; and the fearful Cluster C includes avoidant, dependent, and obsessive-compulsive PDs. PDs are relatively common, with an estimated prevalence of around 6% in the general population according to a WHO study (200), although prevalence varies between studies and is considerably higher in psychiatric populations (201, 202).

The aetiology of personality disorders

Just like personality, personality disorders have a clear hereditary component, with a heritability estimate of 0.6 in a smaller study (203). However, heritability was generally lower in larger samples, where it varies both within and between clusters (204-206). Somewhat surprisingly, the genetics of personality and personality disorders appear to only partially overlap (207).

Apart from genetics, environmental and psychological factors also play a role in the development of PDs. For example, childhood trauma has been linked to PDs, although there are differences between specific PD diagnoses (208), and most research has focused on the well-documented relationship between childhood adversity and BPD (209).

Diagnosing personality disorders

Although there are no fundamental differences when compared to the LEAD diagnostic method described previously, challenges remain in diagnosing PDs. Even when using validated instruments for diagnosis, such as the Structured Clinical Interview for DSM-IV-Axis II Disorders (SCID II) (52) or the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD) (210), assessments often involve challenging differential diagnostic considerations compared to other psychiatric conditions. This is particularly true when distinguishing between temporary state effects and actual traits in the patient's symptoms or behaviours.

Personality disorders, comorbidity and functioning

Individuals with personality disorders often have other psychiatric diagnoses as well (200, 211, 212), and comorbidity in the form of a personality disorder is associated with more anxiety symptoms and poorer functioning (213, 214), as well as worse treatment outcomes (215).

Impairment is often greater when personality disorders are present (44, 216-218), social functioning in particular is more affected (217), and social difficulties can also impact work performance (219), leading to an increased risk of long-term sick leave or early retirement (220, 221).

Aims and hypotheses

General aims

While there is considerable knowledge about the individual factors that impact functioning, there is still a gap in the understanding of how these factors interact and collectively shape an individual's level of functioning. In order to study functioning and the factors related to it, it is essential to have valid measurement tools available in Swedish.

This thesis aims to enhance our understanding of functioning in psychiatric patients by validating clinical measurement tools and identifying key influencing factors. The first objective is to validate two instruments: the SDS for measuring functioning and the Early Trauma Invenory Self-Report Short Form (ETISR-SF) for assessing trauma. The second objective is to examine the mechanisms through which childhood trauma and psychiatric comorbidity influence adult functioning. Lastly, the thesis will explore a broader range of factors that impact functioning in psychiatric outpatients, both separately and in combination.

Study I

Aims

The aim of this study is to further validate the Sheehan Disability Scale (SDS) and for the first time examine the psychometric properties of the Swedish translation.

Hypotheses

Hypothesis to be tested are:

1. The Swedish translation of the SDS will exhibit similar psychometric properties (internal consistency, factor structure) as the original English version and the Spanish translation.

2. There will be a strong correlation between GAF and SDS-scores rated by both patients and clinicians, thereby further confirming the SDS's concurrent validity.

3. The will be a weak correlation between socio-demographic data and the SDS-scores as a measure of external validity.

Study II

Aims

The aims of this study are to examine the psychometric properties of the Swedish translation of the ETISR-SF, and further validate the instrument in two clinical samples of young psychiatric outpatients as well as in one sample of non-clinical controls.

Hypotheses

Hypotheses to be tested are:

1. The Swedish version of the ETISR-SF will exhibit psychometric properties (internal consistency, factor structure) similar to previous translations and to the original English version.

2. Test-retest-reliability of the Swedish version will be comparable with the Spanish, Korean and Portuguese versions.

3. As an indication of discriminant validity, the ETISR-SF will be able to discriminate between non-clinical controls and psychiatric patients, as well as patients with or without two diagnoses (PTSD and BPD) theoretically associated with trauma.

Study III

Aims

The aim is to test different models of the interplay between different levels of childhood trauma and psychiatric comorbidity and their effect on functioning.

Study IV

Aims

The aim of this study is to explore factors beyond symptom severity that explain current levels of functioning in psychiatric outpatients.

Hypotheses

The main hypotheses to be tested are:

1. Functioning is negatively associated with the degree of comorbidity, presence of personality disorders, childhood traumatisation, insecure attachment, personality traits (assessed as temperament and character) and more specifically low levels of C and SD, and high levels of HA.

2. These factors contribute independently in a regression model.

Beyond the main hypotheses, we also explore the following research questions:

A. To what extent do the factors mentioned above account for the variance in the level of functioning?

B. Do the results differ depending on whether a patient-rated or clinician-rated instrument is used?

C. Are different sub-domains of functioning (work/school, social functioning, and family life/home responsibilities) predicted by the same factors?

Participants, materials, and methods

Ethical standards

The studies adhere to the Declaration of Helsinki and were approved by the Regional Ethical Review Board in Uppsala: Dnr 2008/171 for the BBA sample, Dnr 2012/081/01 and 2024/06062/01 for the UPP sample and Dnr 2012/081/03 for the non-clinical sample.

Measures were taken to address the ethical concern that psychiatric patients might feel pressured to participate if asked by their regular psychiatrist/psychologist. In the BBA study, the invitation was sent by mail and it was clarified that participation was voluntary and would not impact their care. In the UPP sample, patients were invited by a research nurse who did not take part in their psychiatric care.

A post-study survey for the UPP sample showed that most participants were satisfied with their involvement (222). Another study within the UPP material investigating why some individuals chose not to participate found that the most common reasons were because of pronounced psychiatric symptoms and/or feeling too tired to take part. Lack of trust in research or the perception that research had little value were less inhibitive factors (223).

Study samples

Participants from three separate samples were used in the studies. Two of the samples were drawn from an outpatient unit for young adults: the Borderline-Bipolar-ADHD sample (BBA-sample) and Uppsala Psychiatric Patient samples (UPP-sample). The third sample consisted of a non-clinical population. The study design and the instruments used across these populations are detailed in Table 1 and further explained in the text below.

Table 1. Overview of the different samples, instruments used, and the studies in which they were used.
Abbreviations: Attention Deficit Hyperactivity Disorder (ADHD), Bipolar Disorder (BP), Borderline Personality Disorder (BPD), Structured Clinical Interview for DSM IV (SCID), Kiddie-Schedule for Affective Disorders and Schizophrenia (K-SADS), Global Assessment of Functioning (GAF), Sheehan Disability Scale (SDS), Mini International Neuropsychiatric Interview (MINI), Early Trauma Inventory Self-Report Short Form (ETISR-SF), Attachment Style Questionnaire (ASQ), Temperament and Character Inventory (TCI), Socio-demographic index (SDI).

	BBA-sample	**UPP-sample**	**Non-clinical sample**
Population	Young adult psychiatric outpatients with at least one of the diagnoses ADHD, BP, BPD	Young adult psychiatric outpatients	Students and university employees
Study design	Clinical, cross-sectional Purposive sample	Clinical, cross-sectional Consecutive sample	Cross-sectional Convenience sample
Interviews and expert-rated instruments	SCID-I SCID-II K-SADS SDS GAF SDI	SCID-I or MINI	MINI
Self-rated instruments	SDS ETISR-SF ASQ TCI	SDS ETISR-SF	SDS ETISR-SF
Used in studies	I, II, IV	II*, III**	II, III

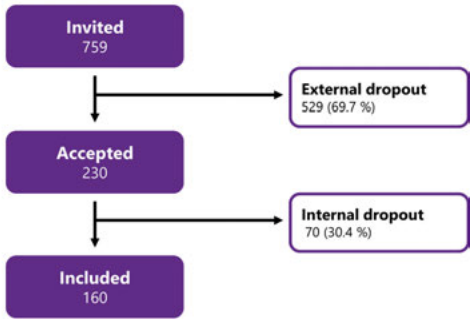
* recruited from the 14th of January to the 2nd of December 2013

** from September 2012 to February 2018

Since participants were recruited consecutively in one of the clinical samples (UPP), the size of this sample varied depending on when the study was conducted. The number of participants and the drop-out rate for each study are presented in Figure 4.

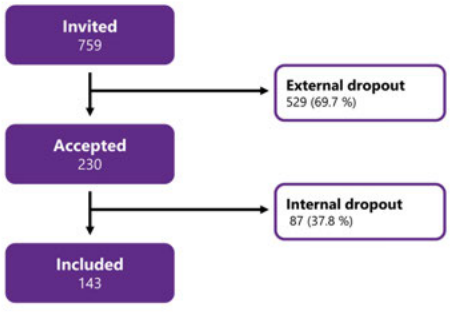
Study I

BBA sample

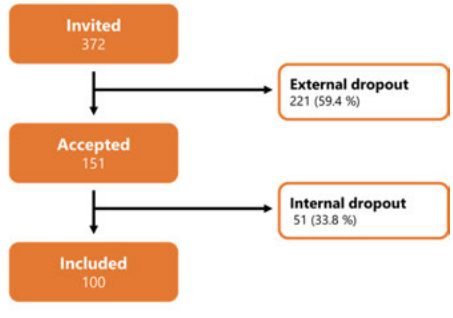


Study II

BBA sample



UPP sample



Non-clinical sample

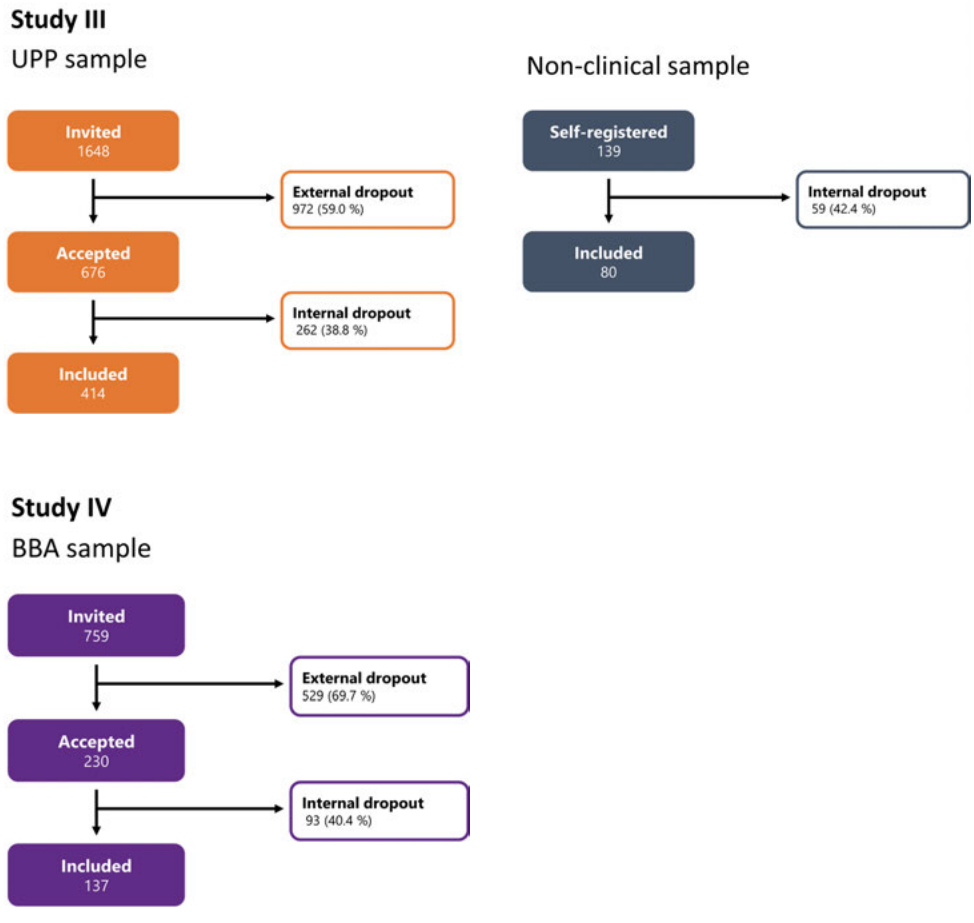


Figure 4. Drop-out rates and number of participants for the samples as used in the different studies.

Drop-out analysis

BBA sample

External drop-out

The external dropout rate was relatively high (69.7%). More women chose to participate in the study (74.3% vs. 55.8%, χ^2=23.35, p<.001). Regarding diagnoses, a higher proportion of participants had BPD (27.0% vs. 16.1%, χ^2=12.17, p<.001) and BP (50.4% vs. 39.9%, χ^2=7.27, p=0.007), while fewer had ADHD (43.9% vs. 55.2%, χ^2=8.18, p=0.004). Participants were also slightly older (mean age 27.9 vs. 26.4, t=-6.87, p<.001).

Internal drop-out

The internal dropout rate varied slightly across the different studies using the BBA sample (30.4%–40.4%), due to differences in the data required for participants to be included in each study. The figures below are based on data from Study I, but the same pattern remains consistent when the BBA sample is used in Studies II and IV.

Participants were slightly older (28.4 vs. 26.9 years, t=-3.71, p<.001), but there was no significant difference in gender distribution (75.0% vs. 72.9% women, χ^2=0.12, p=0.732). A slightly higher proportion of participants had BPD, although this difference was only "borderline significant" falling just short of the threshold for statistical significance (30.6% vs. 18.6%, χ^2=3.59, p=0.058). A significantly higher proportion had BP (55.6% vs. 38.6%, χ^2=5.67, p=0.017). As in the external dropout analysis, fewer participants had ADHD (36.3% vs. 61.4%, χ^2=12.53, p<.001).

UPP-sample

Sine there was no information available on patients declining to participate, only internal drop-out analysis was performed.

In Study 2, there was no difference regarding age (mean age 21.7 vs 21.2, t=1.33, p=0.19) or gender (75.0% vs 75.7% women, χ^2=0.07, p=0.935) between the participants included in the study and those excluded.

In Study 3, there was no difference in gender (76.3% vs 75.6% women, χ^2=0.05, p=0.82) between the participants included in the study and those excluded. Regarding age, the result (mean age 21.3 vs 21.7, t=1.96, p=0.050) fell just below the threshold for significance at the decimal level. (Additionally, the difference in age is so small that any actual difference is unlikely to have relevance.)

Non-clinical sample

Only internal drop-out analysis was performed. In Study 2, there was no difference in age (mean age 26.8 vs 25.5, t=0.62, p=0.54), however there were fewer women (66.1% vs 87.5% women, χ2=3.87, p=0.049) among the participants included in the study compared to those excluded.

In Study III there were no difference in age (mean age 26.0 vs 25.3, t=-0.36, p=0.72) or gender (69.0% vs 59.0% women, χ2=1.26, p=0.26).

Procedure and recruitment of samples

BBA-sample

Recruitment

Patients were recruited from the Unit for Young Adults at the Department of General Psychiatry, University Hospital in Uppsala, Sweden. A total of 759

patients diagnosed between May 1, 2005, and October 31, 2010 with Borderline Personality Disorder (BPD), Bipolar Disorder (BP), Attention Deficit Hyperactivity Disorder (ADHD) (hence the name of the sample, BBA), or any combination of the three were identified from the administrative patient register and all were sent the study invitation by mail. Inclusion criteria were based on a diagnosis of BPD, BP and/or ADHD. The sample was originally intended to examine BPD and disorders with similar symptom profiles, such as emotional instability and impulsivity (224). Twenty-eight patients declined to participate, and 501 never replied. A total of 230 patients (30.3%) agreed to participate and provided written consent. Exclusion criteria included severe psychotic or manic symptoms at the time of the interview. One patient was excluded due to mania. At the time of the study, the participants had previously received treatment as usual at the outpatient clinic, and some still had contact with psychiatric care, while others did not.

Assessment procedure

Each participant in the BBA-sample was interviewed on one or two occasions, depending on the time needed. Prior to the first appointment, the participants completed three self-rated scales, of which only ASQ is used in this thesis. The interviewers included three medical doctors, all of whom had worked at the clinical unit where the patients were recruited. These doctors performed the majority of the interviews, with some (4%) being performed by other clinicians. First, a basic interview collecting anamnestic, social and demographic data, using a checklist, was performed. After the basic interview, complementary semi-structured diagnostic interviews were performed. Finally, the patient's level of functioning was rated, both by the interviewer (GAF and SDS) and the patient (SDS). The interviewers were blind to the patient's SDS ratings when they rated their GAF and SDS.

Assessment training

One psychiatrist and two residents in psychiatry carried out 96% of the diagnostic interviews and performed all professional assessments of functioning. All had previous training in and experience of using GAF. However, none had practiced assessment with the SDS since the scale is designed for self-assessment. Four meetings were therefore held before and during the study where 10 written case-vignettes were rated before each meeting, in total 40 case-vignettes. Both GAF and SDS were assessed, and discussed after inter-rater reliability had been calculated. This was done to measure and maintain inter-rater reliability throughout the study. After the first ten cases had been rated, written examples for the different intervals in the SDS were constructed, similar to those in the GAF-scale, although not as comprehensive. The inter-rater reliability (as measured by intraclass correlation, ICC) between the three doctors varied between 0.83 and 0.92 for the SDS-ratings, and between 0.86 and 0.92 for the GAF ratings. At the end of the study, they reassessed the 40 cases, and the test-retest reliability was 0.89 for both the SDS and GAF. One resident

failed to fill in 10 of the 40 GAF scores both before and after, but had rated all SDS scores.

Regarding diagnostic interviews, the specialist in psychiatry trained the other interviewers in accordance with the SCID manual. Before the start of and during the study, filmed SCID I and II and K-SADS interviews were independently rated and discussed until consensus was reached.

UPP-sample

Recruitment

The UPP sample (UPP is an acronym for Uppsala Psychiatric Patient sample) consisted of young psychiatric outpatients in general psychiatry. Patients were recruited at intake from the Psychiatric Unit for Young Adults at the University Hospital in Uppsala, Sweden. All new patients were consecutively invited to participate. A subsample consisting of patients recruited between January 14 and December 2, 2013 (n=372), was used in Study II, the sample had previously been selected for another study. Those recruited between September 2012 and February 2018 (n=1648) were used in Study III. Written consent was signed by n=151 (40.6%) for Study II and n=676 (41.0%) for Study III.

Assessment procedure

For the UPP samples, the diagnostic interviews (MINI or SCID-I) were conducted as part of regular clinical practice by trained psychiatrists, psychiatry residents, or trained clinical psychologists. All were trained in the use of the diagnostic instruments, but inter-rater reliability data for these raters is not available.

Patients had not received any treatment from the outpatient clinic at the time of assessment, however, some had received treatment in, for example, primary care settings prior to contact.

Patients who provided written consent completed the study's self-report questionnaires on a separate occasion during an appointment with a research nurse, close to the time of the diagnostic evaluation. A subgroup (n=43) was sent another ETISR-SF self-assessment by mail, which was returned on average 11.2 (SD=15.9) weeks after the first had been completed.

Non-clinical sample

Recruitment

The non-clinical sample (n=139) consisted of adults who were recruited from students and employees at Uppsala University. Information about the study was provided during classes at the medical school, teachers' college, psychology courses, and among university staff at the Uppsala Biomedical Center. Since no formal control over the recruitment process was established, this sample is considered a convenience sample.

Assessment procedure

Participants completed questionnaires on-site and structured diagnostic interviews were conducted by telephone by two psychiatrists or a study nurse.

Sample specifics, Study I

BBA sample

Sixty-nine patients were excluded because they did not complete the study measures, or had too much missing data. Finally, 160 participants were eligible. Descriptive data for all sub-samples are presented in Table 2.

Sample specifics, Study II

BBA sample in study II

Eighty-six (37.4%) patients were excluded because they did not complete the necessary study measures, did not participate in the diagnostic interviews or had too much missing data. Finally, 143 patients were eligible.

UPP sample in study II

Of 151 participants, 100 (66.2%) had sufficient diagnostic and ETI-data for this study.

Non-clinical sample in Study II

The non-clinical sample (n=80) was recruited from university employees and students who were asked to participate. Those who completed both MINI and the ETISR-SF were eligible for this study (n=56, 70.0%).

Sample specifics, Study III

UPP sample in Study III

Fifty patients were excluded because more than one month had passed between rating their functioning and their diagnostic interview. Two participants had missing data regarding how much time had passed, but since a large majority (88%) of the participants had <1 month between interview and SDS-rating, they were not excluded. If a single item was missing on the SDS scale, it was calculated as the mean value of the other SDS items. However, if there were missing data for more than one SDS item, the participant in question was excluded from the study. If participants had any missing data on ETISR-SF or concerning diagnosis, they were excluded. In total 212 patients were excluded because of missing data, thus leaving 414 of 676 participants (61.2%) eligible for the study.

Non-clinical sample in Study III

Those who completed all necessary study measures were eligible for this study (n = 100, 71.9%).

Sample specifics, Study IV

To be eligible for the study, participants needed to have available data on functioning, comorbidity, personality disorders, childhood trauma, attachment, and temperament/character. Ninety-two participants were excluded because they did not complete the necessary study measures or had too much missing data. Ultimately, 137 participants met the eligibility criteria.

The subsample used for Global Assessment of Functioning (GAF) calculations (n=130) differed only slightly with a mean age of 23.2 years (SD 2.0) and gender ratios (99 (76.2%) female and 31 (23.8%) male).

Table 2. Characteristics of study sub-samples, including gender distribution, age, and comorbidity of participants.

	BBA sample			UPP sample		Non-clinical sample	
As used in study no.	I n = 160	II n = 143	IV n = 137	II n = 100	III n = 414	II n = 56	III n = 100
Female	120 (75.0%)	108 (75.5%)	103 (75.2%)	75 (75.0%)	316 (76.3)	37 (66.1%)	69 (69.0)
Male	40 (25.0%)	35 (24.5%)	34 (24.8%)	25 (25.0%)	98 (13.7%)	19 (33.9%)	31 (31.0%)
Age, mean (SD)	23.2 (2.7)	23.4 (2.0)	23.3 (2.1)	21.2 (0.4)	21.3 (2.2)	26.8 (0.5)	26.0 (9.3)
Diagnostic groups n (%)							
Any mood disorder	115 (71.9%)	103 (72.0%)	100 (73.0%)	72 (72.0%)	305 (73.7%)	4 (7.1%)	10 (10.0%)
Any anxiety disorder	103 (64.4%)	93 (65.0%)	90 (65.7%)	62 (62.0%)	239 (57.7%)	4 (7.1%)	8 (8.0%)
Any eating disorder	46 (28.8%)	38 (26.6%)	39 (28.5%)	7 (7.0%)	50 (12.1%)	0 (0%)	0 (0%)
Any substance-related disorder	4 (2.5%)	0 (0%) *	3 (2.2%)	12 (12.0%)	44 (10.6%)	4 (7.1%)	7 (7.0%)
Any personality disorder	91 (56.9%)	80 (55.9%)	82 (59.9%)	n/a	n/a	n/a	n/a

* Only four participants were diagnosed with substance related disorders in the BBA-sample, and since none of these subjects completed the ETISR-SF they were excluded from this study.

Instruments

Global Assessment of Functioning (GAF)

GAF is a scale used for the global assessment of functioning and is incorporated in the DSM-IV-TR (225). Level of functioning and level of psychiatric symptoms are designated on a one to 100 scale with ten intervals (1-10, 21-30, 31-40 and so forth) with descriptions to facilitate assessment. Lower scores indicate more severe impairment, whereas zero is assigned when there is insufficient information to make an assessment. Symptoms and functioning can be rated separately, then denoted GAF-S and GAF-F, and the latter is used consistently in this thesis.

The expert-rated GAF has shown very high inter-rater reliability in research settings (43), although lower reliability has been reported in clinical studies with less GAF-training (226) (for a review, see (227)). GAF ratings of patients with severe diagnoses, e.g. schizophrenia, have been shown to be more reliable, even when raters had very little training (228). Although the validity of GAF differs between studies (226, 227), later studies support its meaningfulness (43). There is also a version for self-assessment and comparison of patient and expert ratings has shown good to excellent agreement (44, 229).

Sheehan Disability Scale (SDS)

The brief self-rated Sheehan Disability Scale (SDS) (22) is a combined visual, numerical, and descriptive scale used to assess current (within the last month) functional impairment caused by psychiatric symptoms. A value from zero ("*the symptoms do not affect function at all*") up to 10 ("*symptoms have an extreme effect on functioning*") is ascribed to each of three areas: work/school, social, and family life/home responsibilities. The sum of the three values yields a total SDS score between zero and 30. In addition to the numerical values there are also three intervals where the impairment is categorized as mild (score 1-3), moderate (4-6), or marked (7-9). The instrument is included in the appendix for reference.

The SDS was developed, and is used, as a self-report instrument. In Study I it is used both as a self-report and an expert-rated scale. The Swedish version of the SDS was used with permission from D. V. Sheehan.

The SDS has shown high internal consistency and the same one-factor structure in both the original English version (230-232) and the Spanish translation (233). Previous studies examining the psychometric properties of the SDS have mainly focused on concurrent validity, in addition to reliability. The SDS has been shown to be both reliable and valid when assessing functioning in primary care patients (232), as well as in psychiatric patients with bipolar disorder (230) or social phobia (234), and its validity has been shown to be moderate for patients with panic disorder (235). The SDS has increasingly been used in clinical trials and appears to be sensitive to treatment effects (236) (for a review, see (237)). The sum of the three SDS sub-scales has been shown to be more useful than using the three sub-scales separately (234), and is the scoring method used in the studies included in this thesis. Prior to Study I, there were no psychometric data available for the Swedish translation.

It is not known to what extent SDS scores correlate with socio-demographic data, such as unemployment and sick leave, since this has not been thoroughly studied. One study examined the relation between SDS-scores and socio-demographic data among patients with social phobia, such as problems concerning marital status, living conditions and educational attainment. Somewhat disappointingly, the SDS total and sub-scores did not reflect socio-demographic characteristics except for education, where participants who had

completed college significantly differed in total SDS-score from those who had not (234). Finally, whether self-reported SDS-scores are comparable with clinician-rated SDS-scores, or which best reflects socio-demographic characteristics, have not been examined.

Socio-demographic impairment index

For Study I, a scoring system for the socio-demographic information obtained from the basic interviews was developed and termed the Socio-demographic Impairment Index (SDI). The four authors made individual scoring criteria drafts, and the final scoring system was arrived at after discussion and consensus within the group. Marital status was not judged relevant because of the low age of the participants. The scores for all endorsed items (see Table 3) were summed, resulting in a possible SDI score range of 0 to 15.

Table 3. Items and scoring principles of the socio-demographic index (SDI).

Socio-demographic data		Points
Employment	Unemployed	1
	Part-time sick leave	1
	On sick leave	2
Need of economic assistance	Welfare/social security	1
	Provided for by parents AND age > 20 yrs.	1
Level of education	Not finished elementary school	2
	Not finished high school AND age > 20 yrs.	1
Housing support	Institution or sheltered housing	2
Community support	Student support from university	1
	Support from social services	2
	Support from habilitation services	2
	Social disability support (LSS*)	2
	Other community support	1
Relationships	Having no friends	2
	Having only one friend	1

* LSS, A Swedish law granting support to some disabled groups.

Early Trauma Inventory, Self-Report Short Form (ETISR-SF)

The ETI was originally developed as a comprehensive expert-rated interview, and later a self-report version (ETI-SR) was introduced (133). Psychometric

analyses were conducted to shorten the inventory and remove redundant items, resulting in a briefer self-rated version: the ETISR-SF (238), which is the version used in this thesis. The instrument is included in the appendix for reference.

The ETISR-SF is designed to retrospectively measure trauma before the age of 18, and can be self-administered in about 15 minutes. It comprises 27 specific trauma-items organized in four domains: general trauma (11 items), physical abuse (5 items), emotional abuse (5 items) and sexual abuse (6 items).

For each domain, except for general trauma, there are follow-up questions for each trauma type regarding frequency, type of perpetrator and age of onset (ages 0-5, 6-12 and 13-18). All domains also include Likert scale questions about the extent to which the traumas affect the subject emotionally, at work or school, and in social relationships.

The ETISR-SF has been shown to be a valid instrument for retrospective self-assessment of childhood trauma in diverse populations (238-242), with good test-retest-reliability (240-242). It has been successfully translated and adapted, maintaining its psychometric properties, into several languages and cultural contexts, including Spanish (240), Korean (241), Brazilian Portuguese (242), Dutch (243), and Chinese (244). However, prior to Study II in this thesis, the instrument had not been translated into Swedish or psychometrically tested in this language. The process for translation into Swedish followed recommendations for translation and back-translation. After the first 52 participants in the BBA sample had completed the questionnaire, minor graphical changes were made in order to make the inventory clearer and easier to rate. The first version had similar internal consistency to the second version (Cronbach's alpha = 0.74 for the first version and 0.73 for the second), and are not analysed separately below.

Different methods of scoring ETI have been discussed (133, 238). Simply counting the number of traumatic events has been shown to be as valid as more complex scoring algorithms (238), and this is the method applied in this thesis.

Attachment Style Questionnaire (ASQ)

The Attachment Style Questionnaire (ASQ) (141) is a brief self-report instrument that measures attachment styles dimensionally across five factors. The five factors of the ASQ are entitled Discomfort with Closeness (DIS), Relationships as Secondary (REL), Confidence (CON), Need for Approval (NEE) and Preoccupation with Relationships (PRE). Avoidant attachment is measured by the DIS and REL dimensions, anxious attachment by the NEE and PRE dimensions, and secure attachment by the CON dimension. A two-factor higher order structure was also observed, these factors being avoidance and anxious attachment (245).

The ASQ was originally aimed at assessing attachment in teenagers, and is not specifically designed for assessment of romantic or parent relations but

assesses close relationships in general. It has also been used in other populations, showing satisfactory psychometric properties in adult populations (246), both general and psychiatric, among others used by Fossati et al. (245). It exhibited discriminant validity by yielding different results in the two groups, and the hypothesized five factor structure (described in the introduction) for the 40 items was replicated.

A 29-item short-form version of the ASQ (ASQ-SF) has been developed, showing similar satisfactory internal consistency and an improved factor fit compared to the original ASQ version (247). In the factor analysis the ASQ-SF showed that "less is more", capturing the two broad dimensions as well as the five factors previously hypothesized, showing an even better factor fit than the longer ASQ-version (247). This is the version utilized in this thesis.

The ASQ has been translated into Swedish, psychometrically tested, and satisfactorily validated in both clinical and non-clinical samples (248, 249).

Temperament and Character Inventory (TCI)

The Temperament and Character Inventory (TCI) is a self-report instrument designed to capture the temperament and character traits outlined in Cloninger's psychobiological model, which has been described in detail in the introduction. The questionnaire consists of 238 dichotomous true/false items.

The TCI was developed in 1993 (166), and it has since been revised (169) and validated in various translations, including French (170, 171), Spanish (172), Italian (173), German and Swedish (174), all with reported good validity. The Swedish translation has been tested in clinical samples, achieving an internal consistency of 0.89 (Cronbach's alpha). Norm data is also available for the Swedish translation (250).

SCID-I CV

The Structured Clinical Interview for DSM-IV Axis I Disorders, Clinical Version (SCID-I CV) (51) is a semi-structured interview for axis I disorders. The reliability of SCID-I CV has generally been shown to be good (251-253). SCID-I has demonstrated superior validity over standard clinical interviews (254, 255). For some diagnoses, SCID I-CV only contains screening questions. If the screening questions were positive, the corresponding DSM-IV criteria were assessed. The interviewers had undergone previous training in using the interview.

SCID-II

The Structured Clinical Interview for DSM-IV-Axis II Disorders (SCID-II) was used to diagnose personality disorders (52), including borderline personality disorder. SCID-II has demonstrated good reliability in most studies (55, 256, 257).

K-SADS

The ADHD-module from the K-SADS (Kiddie-Schedule for Affective Disorders and Schizophrenia-Present and Lifetime Version) (258) was used to diagnose ADHD/ADD/HDD. The semi-structured K-SADS interview is designed for children between the age of six and 17 years. K-SADS has good reliability (258). It was chosen since there were no validated diagnostic interviews for the assessment of ADHD in adults translated into Swedish at the time of study start, and the participants were young.

MINI

The Mini International Neuropsychiatric Interview (MINI) (50) is a widely used structured diagnostic interview for several common mental disorders. The validity and reliability have been shown to be good (259, 260). The interviewers had undergone previous training in using the interview.

Statistical analyses

Statistical methods, considerations, and software

Calculations were conducted using IBM SPSS Statistics, with the exception of the factor analysis in Study II, which was performed using MPLUS version 7. SPSS version 21 was used for Studies I and II, while version 28 was used for Studies III and IV. The PROCESS extension was employed for mediation (PROCESS model 4) and moderation (PROCESS model 1) analyses.

For all analyses where significance levels were relevant, the threshold was set a priori at $p < 0.05$.

In all studies, the SDS was treated as a ratio scale, which is standard practice when using this instrument, although it could be argued that it also possesses some characteristics of an ordinal scale.

For drop-out analyses, independent sample t-test were used for continuous data, and chi-squared test for categorical data.

Study I

Differences between groups were calculated using chi-square for dichotomous data and t-test for continuous data. Internal consistency was calculated using Cronbach's alpha (261). To examine if the three SDS items all individually contributed to a single construct, an exploratory factor analysis was conducted. Concurrent validity was gauged by calculating the Pearson correlations between the total SDS scores and the GAF. External validity was estimated by calculating the Spearman correlations between the socio-demographic impairment index and the SDS total scores. Since the SDI scores were heavily skewed towards zero, and nearly 50% of participants scored zero, the group was dichotomized into two categories: those with and those without any SDI points. These groups were then compared in terms of differences in functioning levels. Intraclass Correlation (ICC) (261) was used to calculate inter-rater and test-retest reliability.

Study II

Internal consistency was calculated using Cronbach's alpha (261). A confirmatory factor analysis (CFA) was performed in order to examine if the translated version fits the suggested four-factor model. All three groups were pooled for the analysis, and standardised loadings were used using variance-adjusted weighted least square (WLSMV) estimator. To determine goodness-of-fit, Root mean square error of approximation (RMSEA), Tucker-Lewis Index (TLI) and comparative fit index (CFI) were used. RMSEA-values range from zero to one, lower values indicating a better fit, and values ≤ 0.06 indicating acceptable model fit. TLI- and CFI-values range from 0-1 with higher values indicating better fit. Values larger than 0.9 indicate acceptable fit and >0.95 indicate good fit (262). One item from the general domain ("Seeing someone murdered") was excluded from the factor analysis since no participant endorsed this.

Intraclass Correlation (ICC) was used to calculate test-retest reliability.

Discriminant validity was gauged by calculating the ETISR-SF's ability to distinguish between patients with and without known or probable traumatic history (PTSD- or borderline diagnosis) from the other patients only in the BBA group, since there were too few participants with a PTSD diagnosis in the UPP and non-clinical groups. To further examine discriminant validity, patients and non-clinical participants were also compared. The Mann-Whitney U-test was used, since the ETISR-SF total score (number of traumas endorsed) was not clearly normally distributed, and the size of the compared groups differed considerably.

Study III

The skewness/standard error (skewness ratio) was larger than 1.96: 5.15 and 7.84 for SDS, 5.42 and 6.39 for the ETISR-SF, and 1.18 and 13.31 for comorbidity (clinical and non-clinical samples respectively). Since neither ETISR-SF, comorbidity, nor SDS scores were normally distributed, as determined by skewness ratios, Spearman correlation was used for correlation.

For analyses with dichotomized diagnostic data, the dichotomization was made to create groups as equal in size as possible, since there is no clear theoretical base for dichotomization. Due to large differences in comorbidity between the samples, different dichotomisation cut-offs were used in the clinical and the non-clinical samples (in the clinical sample: having 0-1 vs 2-5 diagnostic groups, in the control sample 0 vs >0 diagnostic groups).

Since the ETISR-SF data was skewed, particularly in the non-clinical sample, and there are no theoretical cut-off points for different degrees of traumatisation, ETISR-SF-data were split into quartiles in some analyses.

Correlations were examined since these are a prerequisite for mediation analyses. Linear regression was used for the regression model.

The two samples were not pooled since the difference in size of the samples would have made the clinical sample dominant in statistical analyses, and there was also a possibility the that there were different mechanisms at play in the two groups.

Study IV

If a single SDS item was missing, it was then calculated as the mean value of the other items. If there were missing data for more than one SDS item, the participant in question was excluded. As for the ETISR-SF, participants with more than three missing items and those with more than one item missing from the ASQ and TCI were excluded. No missing data concerning diagnoses were allowed. Comorbidity data were operationalized as the number of diagnostic groups from which the participant had current diagnoses, based on the SCID-I interview. The groups used are presented in Table 2.

The data on functioning measures were normally distributed based on graphical visualisations and skewness ratios (skewness/standard error-quotient $<\pm 2.5$). Since some variables showed skewness (skewness/std error of no of SCID-I diagnoses: 4.33, C: -3.443, DIS: -3.957), a Generalized Linear Model (GLzM) was constructed to make sure that deviations from the normal distribution did not affect the multiple regression model. Data on the number of PDs were so skewed (skewness/std error = 7.386) that the variable was converted to a dichotomous variable (has/does not have any SCID-II PD). This dichotomous variable and the "original variable" (number of PDs) had approximately the same Spearman correlation as SDS (0.292 and 0.302 respectively).

A GLzM analysis confirmed that the same variables were significant as in the multiple regression model, indicating that skewness did not affect the regression results.

Simple linear regressions for all variables identified those significantly predicting SDS scores (see Table 2). To reduce the number of independent variables in the multiple regression model, only variables which significantly, or close to significantly (sig. < 0.10), predicted functioning individually were included.

Fischer's Z-test was used for comparison of Self-rated and Clinician-rated functioning (263).

Regarding multicollinearity, Spearman correlations between variables (see correlation matrix in the appendix) were mostly below 0.5, with a few between 0.5 and 0.6 (HA & SD, HA & NEE, SD & C, NEE & PRE). VIF values in the multiple regression model were <10, indicating no multicollinearity issues (264).

Results

Study I

Descriptives

Diagnostic characteristics of the study sample are presented in Table 2. Comorbidity was common, and 126 participants (87.7%) met the criteria for diagnoses from more than one diagnostic group, while eight participants (5.0%) had no current diagnosis.

Internal consistency

The SDS showed satisfying internal consistency, with a Cronbach's alpha coefficient of 0.77 for the self-rated, and 0.80 for the clinician-rated data (265). A deletion of any item resulted in a lower alpha coefficient, confirming that all items contributed to a single construct measure.

Factor structure

An exploratory factor analysis was performed which showed that all three SDS-scores loaded on a single factor, with an eigenvalue of 2.06, explaining 68.8 % of the variance for the self-rated SDS data. The corresponding numbers for the clinician-rated data, are 2.15 and 71.6 %.

Validity

The different measures of disability are presented and compared in Table 4. According to the SDS, the impairment was somewhat more pronounced in work-related areas.

Table 4. Patient- and clinician rated levels of functioning, SDI scores (n = 152), and differences in clinician- versus patient-rated SDS ratings.
Abbreviations: SDS, Sheehan Disability Scale; GAF, Global Assessment of Functioning; SDI, socio-demographic index

	Patient ratings Mean (SD)	Clinician ratings Mean (SD)	t	p
SDS Work (0-10)	5.4 (2.9)	5.3 (2.8)	0.281	0.779
SDS Social (0-10)	4.9 (2.7)	4.9 (2.5)	0.070	0.945
SDS Family (0-10)	4.7 (2.8)	4.5 (2.4)	0.692	0.490
SDS Total (0-30)	14.9 (6.9)	14.7 (6.6)	0.548	0.584
GAF (1-100)	-	60.0 (11.8)		
SDI (0-15)	-	1.3 (1.7)		

Differences between those with and without socio-demographic disability, as measured by the SDI, were compared according to their SDS and GAF scores. In those with any socio-demographic impairment (i.e. SDI ≥ 1), GAF was, as expected, lower: 55.7 (SD=11.6) vs. 64.8 (SD=10.0) ($t=5.05$, $p<0.001$). Also, SDS ratings were in line with expectations, as both self-rated and clinician-rated SDS scores were higher in patients with socio-demographic disability. Patient-self-rated SDS scores for those with SDI-score >0 were 16.5 (SD= 6.8) compared to 13.5 (SD=6.9) ($t=2.70$, $p<0.01$) for those with SDI-score zero. Clinician-rated SDS scores were 16.0 (SD= 6.6) vs. 13.1 (SD=6.2) ($t=2.79$, $p<0.01$).

The correlations between different disability measures are presented in Table 5. In general, the SDI and the different measures of functioning correlated with each other (0.40 – 0.76). However, the self-rated SDS score showed a weaker correlation with the SDI (0.28).

Table 5. Correlations between disability measures.
Abbreviations: SDS, Sheehan Disability Scale; GAF, Global Assessment of Functioning; SDI, socio-demographic index.

	SDS patient self-rated	SDS clinician-rated	GAF clinician-rated	SDI
SDS self-rated	1	-	-	-
SDS clinician-rated	0.672	1	-	-
GAF clinician-rated	-0.606	-0.760	1	-
SDI	0.280	0.404	-0.571	1

Study II

Descriptives

Data on gender, age, and comorbidity for all three sub-samples are presented in Table 2. Descriptive ETISR-SF data are presented in Table 6. In general, α if item deleted is lower for general trauma than for the other ETI domains and is significantly lower in the non-clinical sample. The same pattern applies to item-total correlations. In the non-clinical group, there were six trauma types that none of the participants had experienced, compared to one trauma type in the other samples.

Table 6. Frequency of endorsement of ETI items (no., %) and Cronbach's α if item deleted for all samples in Study II. Cronbach α for entire domains is also specified. BBA-sample n=143, UPP-sample n=100, Non-clinical sample n=56.

	BBA sample		UPP -sample		Non-clinical sample	
General trauma						
Natural disaster	4 (2.8%)	0.557	0 (0%)	0.629	0 (0%)	0.144
Serious accident	30 (21.0%)	0.534	21 (21.0%)	0.605	2 (3.6%)	0.123
Serious personal injury	24 (16.8%)	0.535	13 (13.0%)	0.606	2 (3.6%)	0.154
Serious injury/illness of parent	45 (31.5%)	0.531	38 (38.0%)	0.579	16 (28.6%)	0.102
Separation of parents	73 (51.0%)	0.560	39 (39.0%)	0.600	14 (25.0%)	0.024
Serious illness/injury of sibling	37 (25.9%)	0.561	33 (33.0%)	0.629	7 (12.5%)	0.195
Serious injury of friend	43 (30.1%)	0.529	27 (27.0%)	0.599	5 (8.9%)	0.137
Witnessing violence	51 (35.7%)	0.474	44 (44.0%)	0.533	11 (19.6%)	0.126
Family mental illness	66 (46.2%)	0.503	66 (66.0%)	0.532	14 (25.0%)	0.016
Alcoholic parents	33 (23.1%)	0.480	19 (19.0%)	0.616	5 (8.9%)	0.244
Seeing someone murdered	0 (0%)	0.563	0 (0%)	0.629	0 (0%)	0.144
Cronbach's α for entire general trauma domain		0.557		0.623		0.143

Physical abuse						
Slapped in the face	61 (42.7%)	0.674	23 (23.0%)	0.536	8 (14.3%)	0.402
Burned with cigarette	5 (3.5%)	0.710	3 (3.0%)	0.701	0 (0%)	0.523
Punched or kicked	43 (30.1%)	0.583	16 (16.0%)	0.595	8 (14.3%)	0.512
Hit with thrown object	43 (30.1%)	0.598	13 (13.0%)	0.588	5 (8.9%)	0.369
Pushed or shoved	67 (46.9%)	0.634	28 (28.0%)	0.602	12 (21.4%)	0.280
Cronbach's α for entire physical abuse domain		0.696		0.665		0.490
Emotional abuse						
Often put down or ridiculed	53 (37.1%)	0.785	38 (38.0%)	0.669	4 (7.1%)	0.644
Often ignored or made to feel you didn't count	52 (36.8%)	0.794	33 (33.0%)	0.698	6 (10.7%)	0.596
Often told you are no good	42 (29.4%)	0.800	25 (25.0%)	0.758	4 (7.1%)	0.699
Most of the time treated in cold or uncaring way	46 (32.2%)	0.794	24 (24.0%)	0.719	4 (7.1%)	0.644
Parents fail to understand your needs	82 (57.3%)	0.832	57 (57.0%)	0.803	15 (26.8%)	0.605
Cronbach's α for entire emotional abuse domain		0.834		0.774		0.692

Sexual abuse						
Touched intimate parts in uncomfortable way	44 (30.8%)	0.801	21 (21.0%)	0.865	3 (5.4%)	0.341
Someone rubbing genitals against you	19 (13.3%)	0.833	7 (7.0%)	0.805	3 (5.4%)	0.341
Forced to touch intimate parts	22 (15.4%)	0.796	8 (8.0%)	0.806	0 (0%)	0.510
Someone had genital sex against your will	14 (17.5%)	0.825	8 (8.0%)	0.821	2 (3.6%)	0.285
Forced to perform oral sex	17 (11.9%)	0.810	7 (7.0%)	0.797	0 (0%)	0.510
Forced to kiss someone in a sexual way	16 (11.2%)	0.799	4 (4.0%)	0.799	0 (0%)	0.510
Cronbach's α for entire sexual abuse domain		0.838	0.840		0.490	

ETISR-SF domain- and total scores for the different samples are presented in Table 7.

Table 7. ETISR-SF domain and total scores.

	BBA-sample (n=143)	**UPP-sample** (n=100)	**Non-clinical sample** (n=56)
General (m, sd)	2.84 (1.96)	2.80 (1.91)	1.36 (1.21)
Physical (m, sd)	1.53 (1.47)	0.83 (1.19)	0.59 (0.91)
Emotional (m, sd)	1.92 (1.85)	1.77 (1.69)	0.59 (1.06)
Sexual (m, sd)	1.00 (1.64)	0.55 (1.27)	0.14 (0.48)
Total (m, sd)	7.29 (4.68)	5.95 (4.21)	2.68 (2.55)

Internal consistency

Cronbach's alpha, as a measure of internal consistency, varied among the three study groups in Study II, see Table 8. Overall, the non-clinical group showed lower α's, on the four domains as well as on the total ETISR-SF score compared to the clinical groups. Also, the general trauma domain exhibited a lower α in all study groups.

Table 8. Cronbach's alpha for the ETISR-SF domains and total score.

	BBA (n=143)	**UPP (n=100)**	**Non-clinical sample (n=56)**	**All data (n=299)**
General	0.557	0.623	0.143	0.601
Physical	0.696	0.665	0.490	0.692
Emotional	0.834	0.774	0.692	0.819
Sexual	0.838	0.840	0.490	0.838
Total ETI	0.736	0.760	0.552	0.766

Factor analysis

The CFA confirmed that a four-factor model showed adequate model fit, with RMSEA = 0.055, TLI = 0.891, and CFI = 0.92. Overall, the items in the general trauma-domain showed a poorer loading towards the general trauma factor (0.163-0.768 with only one item > 0.7) compared to items in the other domains, where all items showed strong loadings towards their factors - all but one item (0.696) > 0.7 and several > 0.9. See Figure 5 for more details on factor loadings. The covariance between the different factors was < 0.60 (0.361 – 0.598).

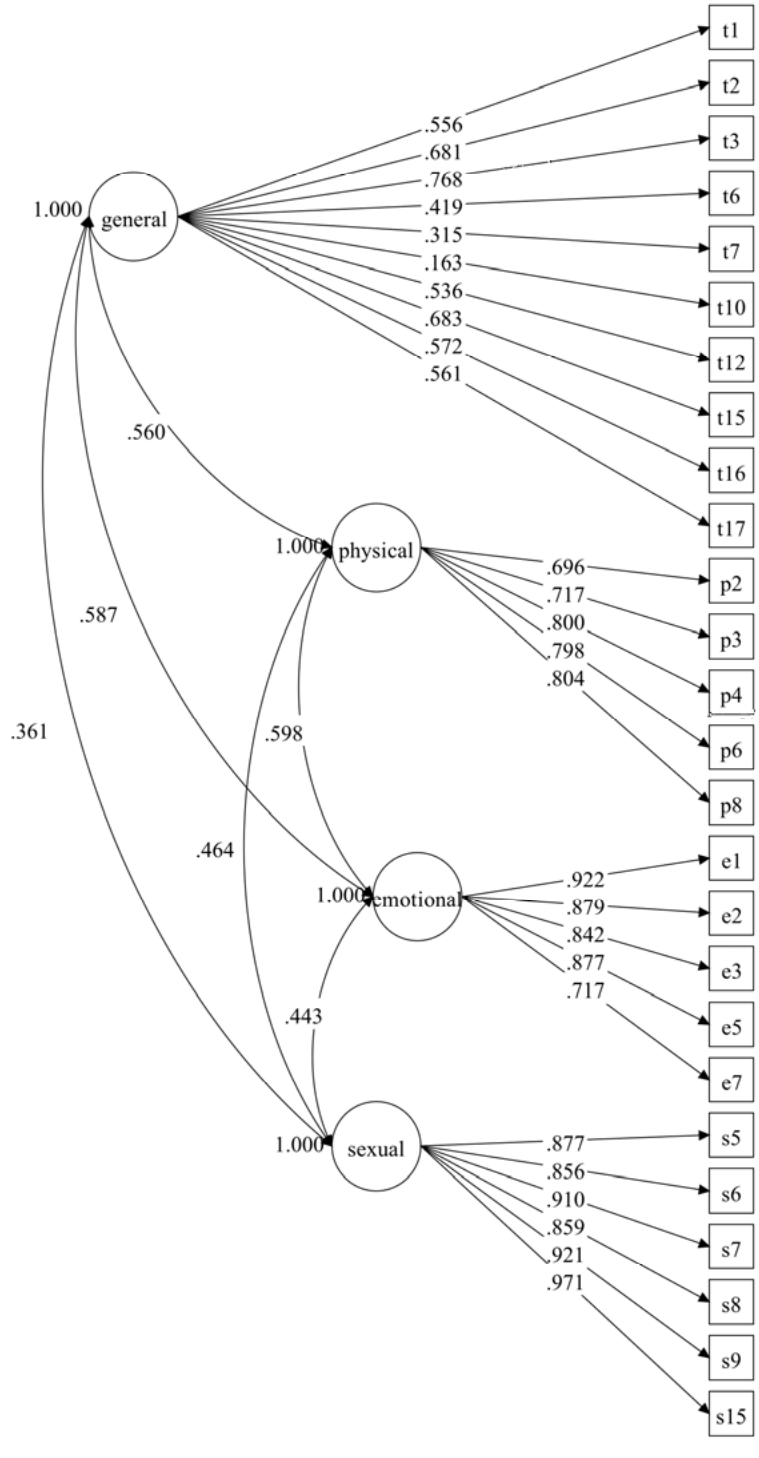


Figure 5. Confirmatory Factor Analysis of the ETISR-SF, four-factor model.

CFA of a second order model, as performed by Osorio et al. (242), was also tested but did not improve the goodness of fit and exhibited similar values (RMSEA = 0.054, TLI = 0.895, CFI = 0.905).

Test-retest reliability

Test-retest reliability was assessed for those participants in the BBA-group who completed the ETISR-SF twice (n = 42). The ICC-values were 0.93 for the global scale, 0.81 for general trauma, 0.86 for physical abuse, 0.92 for emotional abuse, and 0.91 for sexual abuse.

Discriminant validity

According to the Mann-Whitney U-test, the non-clinical sample differed significantly from the two clinical samples with respect to ETISR-SF total scores ($z=-6.796$, $p \leq 0.001$).

When compared within the BBA-sample, the total ETISR-SF scores of patients with a PTSD diagnosis significantly differed from the rest of the subjects in the sample ($z=-3{,}938$, $p \leq 0.001$), and the same was true for those with BPD diagnoses compared to those without ($Z=-3{,}030$, $p < 0.002$).

Study III

Descriptives

Descriptive data concerning functioning (SDS), childhood traumatisation (ETISR-SF) and level of comorbidity for both samples are presented in Table 9. The distribution of SDS scores and comorbidity were more skewed in the non-clinical population.

Table 9. Characteristics of study samples used in Study III. Comorbidity is indicated as the number of diagnostic groups from which the participants have diagnoses.
Abbreviations: Sheehan Disability Scale (SDS), Early Trauma Inventory (ETISR-SF), Standard Deviation (SD).

Level of functioning m (SD)	**Clinical sample (n = 414)**	**Non-clinical sample (n = 100)**
SDS work/school (0-10)	6.3 (2.7)	1.1 (1.8)
SDS relationships (0-10)	6.1 (2.5)	1.2 (2.0)
SDS home (0-10)	5.5 (2.6)	1.0 (1.9)
SDS total score (0-30)	17.9 (6.4)	3.3 (5.4)

Comorbidity		
Mean no. (SD)	1.74 (0.99)	0.26 (0.63)
	n (%)	**n (%)**
0	38 (9.2%)	81 (81.0%)
1	137 (33.1%)	14 (14.0%)
2	148 (35.7%)	4 (4.0%)
3	77 (18.6%)	0 (0.0%)
≥ 4	14 (3.4%)	1 (1.0%)
ETISR-SF score m (SD)		
General trauma (0-11)	2.9 (2.0)	1.4 (1.3)
Physical trauma (0-5)	1.1 (1.3)	0.5 (0.9)
Emotional trauma (0-5)	1.9 (1.8)	0.6 (1.0)
Sexual trauma (0-6)	0.8 (1.5)	0.2 (0.6)
Total score (0-27)	6.7 (4.6)	2.7 (2.6)
Experienced any trauma	397 (95.9%)	84 (84.0%)

Linear regression models on influence of childhood trauma and comorbidity on functioning

In the clinical sample, both childhood trauma and comorbidity were significant predictors for functioning, see Figure 6. In the non-clinical sample, only comorbidity significantly predicted functioning. Using dichotomized comorbidity and ETI-quartiles did not change which associations were significant. Results for all models are illustrated in figure 6.

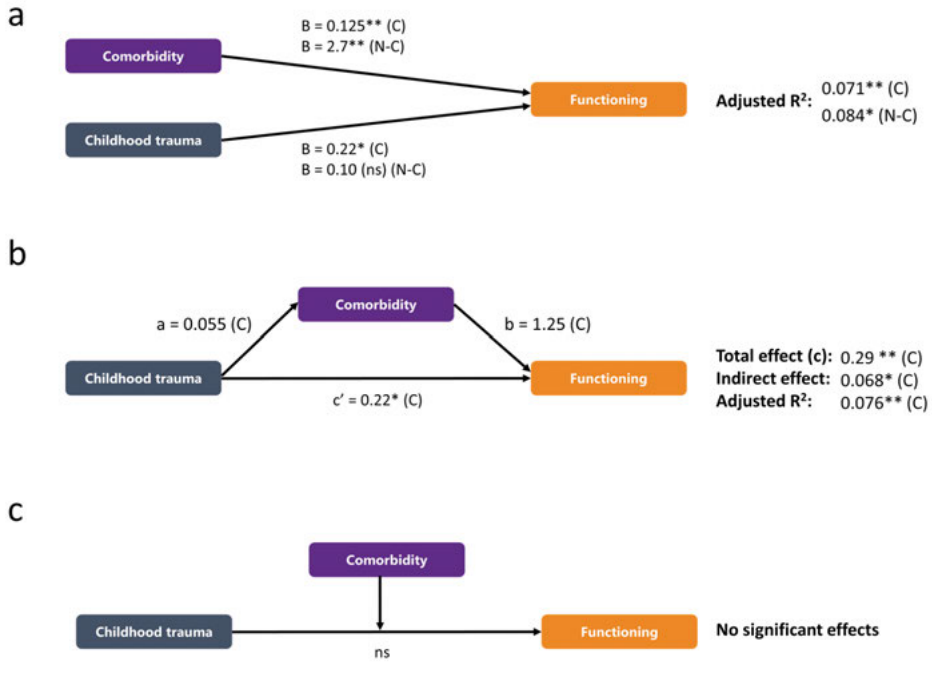


Figure 6. Significant effects of childhood trauma and comorbidity on functioning, all models. C: Clinical sample. N-C: Non-clinical sample.
ns: not significant * $p < 0.05$ ** $p < 0.001$

Mediation models for the relation between childhood trauma on functioning through comorbidity

In the clinical group, all three variables were significantly correlated, which is necessary for a mediation model. There was a partial mediating role of comorbidity on the relationship between childhood trauma and adult functioning, see Figure 6. The indirect effect of childhood trauma on functioning was 0.068 (95% CI 0.030; 0.114, t=3.14, p<0.01), and the direct effect, also significant, of childhood trauma on functioning in the presence of the mediator was 0.22 (95% CI 0.083; 0.355, t=3.17, p<0.01. Thus, the total effect was 0.29 (R^2 = 0.076, $p < 0.001$).

In the non-clinical sample, a mediation model would not fit the data since there was no significant correlation between childhood trauma and functioning ($r = 0.11$, $p = 0.275$).

Moderation models

Two different theoretical moderation models were discussed in the introduction, but since they in fact are identical in a statistical model, they will not be reported separately here. There were no significant interaction effects in the clinical ($p = 0.503$) or the non-clinical sample ($p = 0.281$).

Study IV

Descriptives

Descriptive data on the sample are presented in Table 2 and Table 10. A correlation matrix (Spearman correlations) between all predictors and outcomes is included in the appendix.

Table 10. Descriptive data concerning sample (n = 137) characteristics including functioning, personality disorders, childhood traumatization, attachment, and temperament and character scores.
Abbreviations: SDS: Sheehan Disability Scale, GAF: Global Assessment of Functioning, BPD: Borderline Personality Disorder, PD: Personality Disorder, ASQ: Attachment Style Questionnaire, ETISR-SF: Early Trauma Inventory, TCI: Temperament and Character Inventory.

Functioning	**mean (SD)**
SDS total score	15.04 (6.81)
SDS: Work/school	5.40 (2.96)
SDS: Social	4.96 (2.64)
SDS: Family life	4.67 (2.77)
GAF	59.25 (11.33)

Attachment	
Confidence (CON)	3.39 (1.06)
Discomfort with closeness (DIS)	4.24 (0.86)
Relationships as secondary (REL)	2.80 (0.87)
Preoccupation with relationships (PRE)	3.94 (1.15)
Need for approval (NEE)	4.25 (1.02)

Childhood traumatization	
Experienced any trauma (n, %)	133 (97.1%)
No. of total experienced childhood traumas	7.38 (4.61)

Temperament and Character	
Novelty Seeking (NS)	24.69 (6.17)
Harm Avoidance (HA)	22.58 (7.01)
Reward Dependence (RD)	14.58 (3.48)
Persistence (PS)	4.61 (1.86)
Self-directedness (SD)	21.74 (8.83)
Cooperativeness (C)	29.07 (6.60)
Self-transendence (ST)	12.11 (6.67)

Factors associated with functioning

In the linear regression analyses of the independent variables there were several variables that significantly predicted functioning: having any PD diagnosis, four temperament and character traits (HA, PS, SD, C), and four attachment dimensions (DIS, CON, NEE, PRE); see Table 11 for further details.

Table 11. Simple linear regression analyses predicting functioning (SDS) on all variables individually, R and β-values. Significance level (<0.1) qualifies for use in multiple regression model. N = 137.
Abbreviations. PD: Personality Disorder, ASQ: Attachment Style Questionnaire, ETISR-SF: Early Trauma Inventory, TCI: Temperament and Character Inventory.

	R	***β***	**Sig.**
Comorbidity	.120	0.460	.161
Any PD diagnosis (dichotomous)	.314	4.34	<0.001*
Experienced childhood traumas	.093	0.138	.279
Temperament and Character			
Novelty seeking (NS)	.03	0.033	.732
Harm avoidance (HA)	.497	0.483	<0.01*
Reward dependence (RD)	.095	-0.186	.272
Persistence (PS)	.182	-0.664	.033*
Self-directedness (SD)	.532	-0.410	<0.001*
Cooperativeness (C)	.200	-0.206	.019*
Self-transcendence (ST)	.041	-0.042	.635
Attachment			
Discomfort with closeness (DIS)	.353	2.810	<0.001*
Relationships as secondary (REL)	.022	0.173	.798
Confidence (CON)	.340	-2.176	<0.001*
Need for approval (NEE)	.354	2.357	<0.001*
Preoccupation with relationships (PRE)	.284	1.684	<0.001*
Avoidance	.332	3.251	<0.001*
Anxiety	.383	2.735	<0.001*

Predictors of functioning in a common model

In a multiple regression model, using all the above mentioned significant predictors, only HA and SD remained significant (B = 0.248 *sig*. 0.006 and B = 0.254 and *sig*. 0.003, respectively).

Explained variance in level of functioning

In the multiple regression model, the R-value was 0.620, R^2 0.384 and adjusted R^2 0.340, see Figure 7 for a graphical illustration.

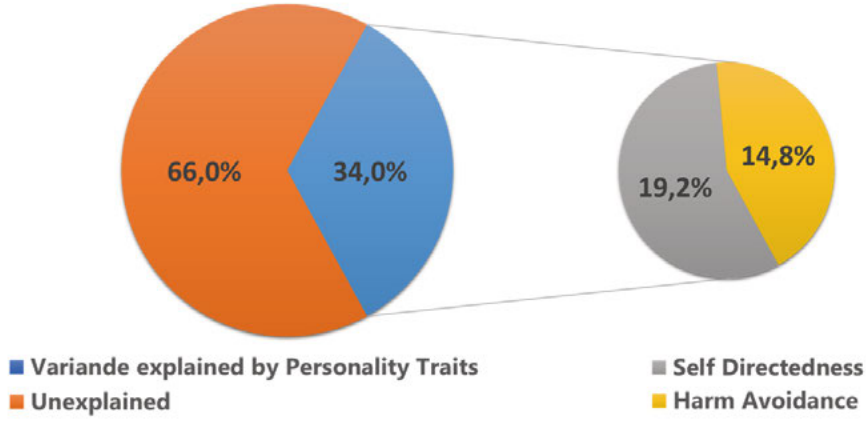


Figure 7. Percentage of variance in functioning explained by personality traits.

Comparison of Self-rated and Clinician-rated functioning

The use of clinician-rated functioning (GAF) did not change the predictors that were significant and the changes in explained variance were minor, see Table 12, and was not significant (Z-value = 0.40).

Specific domains of functioning

There were some differences in significantly predictive variables between the various domains of functioning as measured by the SDS, see Table 10.

Table 12: Multiple regression analyses on different measures of functioning. n = 137 for SDS scores, n = 130 for the GAF-sample. [1] Sig. < 0.05.
Abbreviations: SDS: Sheehan Disability Scale, GAF: Global Assessment of Functioning, HA: Harm Avoidance, SD: Self-directedness, PD: Personality Disorder, C: Cooperativeness.

	SDS total score	SDS work/school	SDS social	SDS family life	GAF
Adj. R^2	.340	.236	.214	.220	.296
Significant[1] predictors	HA, SD	HA, SD	HA	SD	HA, SD

Both HA and SD significantly predicted an SDS score for work/school, while only HA predicted an SDS score for social life and only SD predicted an SDS score for family life/home responsibilities.

Discussion

Key results and discussion

Measuring functioning – Study I

The results support that the psychometric properties of the Swedish translation of the SDS are similar to the original and Spanish versions in terms of factor structure, internal consistency, and concurrent validity. In accordance with previous findings, correlation with socio-demographic data was weak. Exploratory analysis supported a one-factor structure, which is consistent with the SDS literature (230-233, 235). This also confirms the validity of calculating the SDS total score. Patient- and clinician-rated SDS total scores were correlated to the GAF and to the SDI, supporting concurrent and external validity. However, the clinician rated GAF and clinician rated SDS showed higher external validity compared to the patient rated SDS.

A satisfactory internal consistency was expected from a scale that is supposed to measure three somewhat different but related constructs. The alpha coefficients from both the self- and clinician-rated SDS were consistent, albeit somewhat at the low end, compared to most previous studies (230, 232, 233, 235); although Hambrick et al. found a lower alpha coefficient (234).

Used as a clinician-rated instrument, SDS showed excellent inter-rater reliability, although not as good as the GAF. This is probably due to the SDS's more ambiguous anchor points, as has been hypothesized by others (235). The inter-rater reliability was not improved either by the written examples or the ambitious inter-rater training during the study. The strong correlations between self- and clinician-rated SDS scores also support that clinicians and patients rated the same construct.

Patient- and clinician-rated SDS-scores correlated, although both showed weaker correlation with the socio-demographic impairment index. Previous studies have only shown that SDS is sensitive to educational status but not to other socio-demographic factors (234). Therefore, this study indicates that SDS has a stronger external validity than previously shown (234). The clinician-rated measures, both SDS and GAF, showed a stronger correlation to the SDI compared to the self-rated SDS score. A possible explanation is that clinician ratings are probably to a larger extent based on such data, whereas the patients may find it more difficult to discriminate impairment from symptom severity. Another possible explanation is that the socio-demographic index

may reflect functioning over time, while the SDS focuses on current functioning. However, it is open for debate which method best captures the "true" level of functioning: the patient's own opinion, an informed expert's rating, test results in practical tests, or hard sociodemographic facts.

Measuring childhood trauma – Study II

The results of Study II show that the Swedish translation of the ETISR-SF has similar psychometric properties to both the original version and other translations, with similar factor structure and internal consistency. Its ability to distinguish different diagnostic groups, associated with various degrees of trauma, supports its discriminant validity.

The Swedish translation showed high internal consistency for the two clinical samples, comparable to previous research (238-240, 242, 243). As in preceding studies, the Cronbach's α for general trauma was somewhat lower. As previously hypothesised, an explanation for this could be that the general subscale measures a broader range of traumatic events, ranging from natural disasters to mental health problems within the family, and this subscale is therefore more heterogeneous, and does not measure a single construct (240).

The non-clinical sample showed markedly lower internal consistency, particularly in the general and sexual trauma domains. One possible explanation for this is that the non-clinical group generally reported fewer traumatic experiences, which may have contributed to lower internal consistency. Additionally, certain items that were not endorsed by any participants (e.g., witnessing a murder) may have negatively impacted consistency across all subgroups. The non-clinical controls, however, did not endorse several other items (one in the physical abuse subscale and three in the sexual abuse subscale), which together with the smaller sample size, could further explain the relatively lower internal consistency.

The CFA supported the four-factor structure previously suggested. The three different fit indices exhibited good (RMSEA) to acceptable (CFI, TLI) fit. This was comparable to, though slightly weaker than, findings from previous studies (241, 242). The fact that the items in the general trauma-domain exhibited considerably weaker correlations to the latent general trauma factor can be interpreted as the items in that domain not representing one homogenous factor (which is supported by exploratory factor analysis in previous research (238)).

Test-retest reliability for the Swedish translation was good on all subscales as was the total ETISR-SF score, and also comparable with previous translations (240-242). The original English version of the self-rated version has not yet been tested with ICC values, however Bremner et al. showed that the interview version had good test-retest reliability (133). As with internal consistency, the general trauma domain had a somewhat lower ICC-score.

The Swedish version of the ETISR-SF was able to discriminate between groups with different expected levels of traumatisation. This supports the validity of the ETISR-SF, and is also in line with previous research, where ETISR-SF was able to discriminate patients with known associations with trauma from comparison subjects (238).

As measured by the ETISR-SF, the mean value of experienced traumas in a non-clinical Swedish population is 3.0, which can be compared with healthy subjects in a Korean population 2.3 (241) and a US population 3.5 (238). That Swedish subjects have experienced slightly fewer traumas has also been seen in previous research (266).

The interplay between trauma, comorbidity and functioning – Study III

Discussing the results of Study III is more complex, not least because two different samples were analyzed with differing results. In the clinical sample, both childhood trauma and comorbidity significantly predicted functioning in the regression model, and the mediation model showed that the effect of childhood trauma was partially mediated through comorbidity. Neither childhood trauma nor adult comorbidity moderated the effect of the other on functioning. In the non-clinical sample, only comorbidity significantly predicted functioning, and no mediation or moderation between childhood trauma and comorbidity were detected.

In the clinical sample, roughly a quarter of the effect of trauma on functioning was mediated through comorbidity. The present study does not answer the question of how this effect is transmitted, but it could be, for example, by sub-diagnostic post-traumatic symptoms, or negative effects on attachment and emotional regulation, which in turn leads to psychopathology, as elaborated on in the introduction (89, 90).

Explained variance

As is often the case, reality is more complex than simplified statistical models, and neither of the models tested here came even close to providing a complete account of functioning. Traumatization and comorbidity accounted for 7.1% of the variance in functioning in the regression model, and 7.6% in the mediation model, indicating that several factors other than childhood trauma and comorbidity are likely to predict (or affect, if a causal relationship exists) adult levels of functioning. Factors such as social support and psychological variables, for example attachment style, cognitive abilities, personality, or emotional regulation capabilities, could also likely influence the level of functioning, not to mention other health conditions. There are also other childhood variables that could influence to the extent to which childhood trauma leads to impairment later in life, such as good parenting resources and intellectual functioning (267). More indirectly, there are also risk factors connected with

a persons' parents, which affect the likelihood of experiencing trauma in the first place, such as young parental age, parental psychiatric morbidity or substance abuse, and parental traumatization (89).

Differences between samples

There could be several possible explanations for the differences between the samples. To start with, there can be several reasons for the lack of correlation between trauma and functioning in the non-clinical sample, one of which being methodological: the smaller sample size may have prevented a weak correlation from reaching significance. The sample also has a lower variance in functioning and comorbidity, thereby making any actual correlations harder to find statistically. If there is in fact a correlation between trauma and functioning in the non-clinical group (which the present study lacks the power to find), but weaker than in the clinical sample, the difference in strength could be due to the participants in the non-clinical sample having a better ability to cope with the consequences of childhood trauma than the participants in the clinical sample. There is also research showing that resilience or recovery are common trajectories following exposure of potentially traumatic events (268). These studies often focus on PTSD-symptoms rather than functioning, but the same pattern could also be true for functioning. This would mean that at a previous time-point, trauma exposure and functioning would also have been correlated in the non-clinical group, but that these participants moved over time along a trajectory of spontaneous recovery, such that their current functioning is not impacted by the past trauma.

Another possible explanation for a lack of correlation is due to selection bias/ sample composition: since the non-clinical sample consisted of students and university employees – two occupations presumably requiring a high level of functioning – with those having a significant functional impairment being under-represented, thereby excluding persons whose childhood trauma had a pronounced detrimental effect on their adult functioning. The difference in comorbidity between the groups is probably not the reason that childhood trauma only correlated to adult functioning in the clinical sample. If this had been the case, there would have been an interaction effect (i.e. moderation) between traumatization and comorbidity, which there was not.

It is also worth noting that comorbidity might possibly have contributed to a larger degree in a more heterogeneous sample. Despite this, we did not pool the samples in the analysis. The reason for this was twofold: the size difference of the groups would have made the clinical group dominant in statistical analyses, and there was also a possibility that different mechanisms were at play in the two groups (as indicated by the results).

Factors explaining current functioning – Study IV

In the fourth study, several individual variables were found to significantly predict functioning: the majority of temperament and character traits (HA, PS, SD, C) and attachment dimensions (DIS, CON, NEE, PRE), as well as having a personality disorder. More surprisingly, however, neither comorbidity nor childhood trauma did. However, if all variables are taken into account in a multiple regression analysis, only the temperament and character traits Harm avoidance (HA) and Self-directedness (SD) remained significantly predictive. The notion that HA plays a significant role in functioning, especially in a psychiatric population, seems reasonable. Various forms of avoidance behaviours, which align with high HA scores, present clear problems for many patients with for example mood, anxiety, and obsessive-compulsive disorders. The centrality of avoidance in psychiatric disorders is also reflected in the significant focus on reducing avoidance behaviours in CBT. Similarly, the importance of SD for functioning appears logical, given the nature of this character trait - the ability to set goals and act consistently and adaptively to achieve them - seemingly essential for achieving a high level of functioning.

Judging by the high adjusted R^2 in the multiple regression model, personality (although not in the form of personality disorders, but as HA and SD) seems to be an important predictor of functioning in psychiatric outpatients, explaining about one third of the variance, which is very high compared to previous research (187, 189).

There was no difference in the factors that predicted functioning, whether rated by patients (SDS) or clinicians (GAF). Beyond potentially strengthening the validity of both rating measures, this also suggests that possible confounders, such as participants' personality traits, do not substantially impact the reliability of self-rated measures of functioning.

Specific areas of functioning

There were some minor differences when predicting certain areas of functioning. Although the same traits are involved, only HA predicted lower social functioning and only SD predicted higher home/family life functioning, but both HA and SD predicted functioning at work/school. In the specific subtypes of functioning, the variance explained was somewhat lower than for global functioning (adjusted R^2 0.214 – 0.236).

That impairment in the work/school domain of functioning was predicted by both HA and SD may perhaps reflect that such activities require being able to deal with anxiety related to demands and social situations (affected by HA) and the ability to take responsibility for one’s life and commit to one’s goals (SD). Social functioning being predicted solely by HA possibly indicates that anxiety or avoidance is the most common cause of impairment (perhaps in the form of avoidance behaviours from a CBT perspective) in the social domain.

In contrast, only SD predicted functioning in the family life/home responsibilities domain, perhaps reflecting that goal-oriented behaviour is more relevant here than anxiety or avoidance.

Another study focusing on temperament, character, and functioning yielded similar but not identical results (187). For instance, self-directedness was most strongly associated with social functioning, while high persistence was linked to career success. These differences may be attributable to differences in how functioning was conceptualized between the studies, or perhaps the older age of the participants.

Factors *not* predicting functioning

As mentioned, many of the hypothesized factors individually predicted functioning but, somewhat surprisingly, only two temperament and character traits (HA and SD) remained significant in a multiple regression model. It is known that HA and SD are correlated to functioning (187, 188), but it is more surprising that the many other factors in the multiple regression model were not, since previous research has shown an association. A possible explanation is that personality indeed predicts functioning, and the individually significant factors (attachment, temperament and character, and personality disorders) all in some way reflect personality, but that these constructs have a substantial overlap. The most predictive aspects of personality are perhaps HA and SD, thereby making other personality-related constructs (such as personality disorders or attachment, although it can be debated if the latter can be seen as personality-related) non-significant when they are present in the same model. Low SD is known to be predictive of personality disorders (165), but the impairment comes from having "low-SD personality traits" and acting accordingly, not directly from fulfilling the criteria of a specific PD diagnosis.

Similarly, aspects of attachment that are known to affect functioning negatively, attachment anxiety and avoidance (154) (269) (270), are perhaps better explained by the related constructs of HA and SD. Relatively strong correlations between HA and SD and these variables, which has also been seen in previous studies (269), support the view that these constructs are related. With this perspective, attachment can possibly be seen as part of a broader personality construct, though not the component that primarily determines functioning when temperament and character are also taken into account.

However, other studies that also examine attachment and personality together have shown that both constructs contribute to explaining at least the degree of PTSD symptoms (271). Ogle et al. examined the extent to which insecure attachment affects PTSD symptoms, comparing it with other factors such as the personality trait neuroticism (from the Big Five personality model). Regarding PTSD symptoms specifically, this study demonstrates the influence of both personality and attachment, indicating that both factors contribute to the symptom burden in PTSD. Previous research on attachment and the Big Five-model has shown that there are associations between attachment

style and personality trait measures, and that attachment style dimensions are better predictors of relationship quality than measures of the Big Five, but that neither of the measures are redundant (272).

Regardless of any potential overlap between attachment and personality (in terms of temperament and character), the strong explanatory value of HA and SD can be seen as reinforcing the validity of the concepts of temperament and character, as well as the TCI.

It is also noteworthy that comorbidity and childhood trauma, which have been linked to functioning in several other studies (see the Introduction for details), were not significant predictors in this study when personality was taken into account. There could be several possible explanations for this, one being the characteristics of the study sample. The participants had high levels of comorbidity and trauma (>97% having experienced at least one trauma), which lowers the variance, thereby limiting the prospect of finding a significant association. In a more heterogeneous sample also containing participants without diagnoses and with less trauma, e.g., a normal population, comorbidity and trauma might have been predictive (as was the case with comorbidity, but not trauma, in the non-clinical sample in Study III). The relatively small sample size may also have prevented a weak association from reaching significance. A third possible explanation could be that since many participants were or had been actively attending an outpatient clinic, they had received treatment mitigating the effects of trauma and their diagnoses: if a bipolar disorder is treated with a mood stabiliser, or PTSD symptoms are treated with CBT, the impact on functioning is presumably reduced. In addition, in accordance with the treatment protocols at the clinic, axis I disorders (in DSM 5 terminology roughly equivalent to non-personality disorders) are usually treated prior to treatment of personality disorders, which could have resulted in some patients having received treatment for comorbidity but not PDs, with personality-related problems then having a relatively larger impact on functioning than comorbidity-related problems.

General discussion

It can be concluded that, using valid instruments with good reliability, it is possible to assess several factors that are believed to be associated with functioning. Regarding the Swedish translations of the SDS and ETISR-SF, studies I – II within this thesis, have demonstrated that they possess satisfactory psychometric properties, essentially comparable to the original versions and other translations. For the other instruments used, this has been shown in earlier studies.

The purpose of the last two studies was to understand how various underlying factors might predict current functioning, and whether these factors interact with one another.

When examining the factors that predict functioning in psychiatric patients, personality traits - specifically HA and SD - stand out as significant, explaining a large portion of the variance in functioning within a clinical population. However, it is evident that other factors must account for the remaining two-thirds of the variance. Notably, it is surprising that comorbidity and trauma are not among these explanatory factors in the studied material.

Another critical question is *how* these factors actually might influence functioning. While this thesis cannot provide a definitive answer, there are indications that mediation - specifically that the effects of childhood trauma on functioning are partially mediated by comorbidity - may play a role. Although Studies III and IV were conducted in different populations, it is worth reflecting on how the results of Study IV might inform the interpretation of findings in Study III. For instance, is it relevant to examine comorbidity and trauma as explanatory variables for functioning in that material, if these variables are not significant when personality is also considered (as is the case in Study IV)?

It should also be noted that no moderation or mediation analyses were conducted in Study IV. However, this does not preclude the possibility that such effects might exist among the variables investigated.

Causality or not causality – that's the question

Finally, it is important to emphasize that the findings from Study III-IV in this thesis reflect statistical predictions and correlations. This does not necessarily imply that the corresponding causal relationships exist. It does, of course, not rule out the possibility that causal relationships may indeed exist between certain factors either, such as personality traits and functioning, but this cannot be conclusively determined based on the results presented here.

The cross-sectional nature of the studies in this thesis further complicates inferring causality. It could possibly be argued that, since childhood trauma is assessed retrospectively while functioning is evaluated in adulthood, the temporal aspect somewhat strengthens the argument for a causal relationship between these factors. Similarly, one could argue that personality traits, given their relative stability over time, likely precede the current level of functioning. This temporal association might lend some support - though certainly not definitive proof - to the possibility of causality.

It should also be mentioned that some of the underlying theoretical framework (and consequently, some of the hypotheses tested) are built on causal explanatory models. This also applies for some statistical models. For instance, the mediation model used in Study III inherently assumes causal relationships between the variables. Since the studies in this thesis, as mentioned, cannot verify such causal links, the interpretation of the results from the mediation model should instead be understood as follows: *if* corresponding causal relationships do exist, then the mediation model employed in the study might describe (parts of) that relationships.

Ethical considerations

Finally, there are some ethical aspects worth highlighting. From a practical standpoint, participation in the studies, perhaps particularly in the BBA study, could be demanding. For example, revisiting past traumas might trigger negative reactions. One way to address this was to ensure that participants did not have to complete the questionnaire by themselves, and only a handful of participants expressed a need for follow-up support afterwards. A post-study survey for the UPP sample showed that most participants were satisfied with their involvement (273). Another study within the UPP material, which investigated why some individuals chose not to participate found that the most common reasons were because of pronounced psychiatric symptoms and/or feeling too tired to take part. Lack of trust in research or the perception that research had little value were less inhibitive factors (223).

Another perspective concerns stigmatization, that is when a group of people who are considered to deviate from the norm are associated with negative stereotypes. This can lead to worse treatment or care within the healthcare system and, of course, in many other societal contexts. Self-stigma may also arise, where individuals internalize general prejudices, negatively affecting their self-esteem and behaviour, which in itself negatively affects outcomes (274). Could the findings of this thesis contribute to increased stigmatization? That possibility cannot be ruled out. For the sake of discussion, let us temporarily set aside the fact that this thesis does not in any way prove causal relationships, and speculate on the thought that the results could be interpreted in such a manner. The idea that someone might "function less well" due to trauma or mental illness (Study III) or because of certain personality traits (Study IV) could, at first glance, be perceived as negative and stigmatizing.

An often stated counterpoint to this would be that increased knowledge of psychiatric illness and its consequences can, in itself, be a tool to reduce stigmatization. Additionally, it is important to emphasize that psychiatric illness and post-traumatic symptoms are not unchangeable constants but are treatable. To some extent, this reasoning also applies to personality factors, which change over the course of life and can be influenced by interventions - a topic that will be discussed further below. Furthermore, increased awareness of and focus on functional impairment could lead to better treatment of the underlying issues, ultimately improving functioning for psychiatric patients. A greater focus on functioning as an outcome measure could therefore lead to increased empowerment, which in turn reduces self-stigmatization (275). In light of this, the balance between potential negative and positive consequences clearly tips in favor of the positive outcomes.

A more theoretical, moral-philosophical response would be to refer to what is known in moral philosophy as the Is-Ought Problem, or Hume's Law (276, 277). This principle states that one must distinguish between descriptive statements of fact ("is") and prescriptive value judgments or ethical "truths"

("oughts"). Relating this to the results of the thesis: we all possess some more and some less adaptive traits and characteristics, which may, in turn, lead to impairment. However, this says nothing about a person's intrinsic worth or value, as human dignity is in no way tied to such traits or abilities as "functioning".

Methodological considerations, limitations and strengths

Samples

Three samples were used in this thesis, each with its own strengths and weaknesses. A common feature of all samples, which also limits the generalizability, is that all primarily consist of young individuals from a small university town in Sweden, and are generally relatively narrowly selected. Women are also overrepresented in all samples.

A final general consideration is the possibility that individuals who chose not to participate in the study or failed to complete the study measures may have differed from the studied population - for example, through having a lower level of functioning - which could have influenced the results.

The BBA sample

Some limitations specific to the BBA sample include the selection criteria - patients needed to have a diagnosis of BPD, ADHD, and/or BP - which made the sample narrower and less generalizable. Although the sample was diagnostically homogeneous due to the inclusion criteria of diagnoses characterized by emotional instability and impulsivity, there was still a considerable range in other comorbidities. However, the high prevalence of eating disorders (28.5%) and the low prevalence of substance abuse (0 - 2.2%) were somewhat atypical and may further reduced the generalizability of the findings.

Another limitation and potential source of bias was the low response rate and the relatively large proportion of patients excluded due to missing or insufficient data. This was largely because patients participating in the broader BBA study were given the option to engage in different parts of the data collection. Consequently, many were excluded from this study if they had chosen to participate only in certain parts, resulting in insufficient data for the studies in this thesis.

Some strengths, as briefly mentioned earlier, include the extensive amount of information available about the study participants covering comorbidities, personality, attachment, trauma, sociodemographic factors, and multiple measures of functioning - both clinician-rated and self-reported. Additionally, the participants represented a broad range of functioning, from severely im-

paired individuals to those who no longer required psychiatric care. An additional strength is the diagnostic procedure and high inter-rater reliability of clinicians' impairment and diagnostic measures.

The UPP sample

In addition to the previously mentioned weaknesses applicable to all samples, the UPP sample exhibited substantial external and internal drop-out, which may have influenced the study results if the individuals excluded differed from participants in aspects such as lower functioning, higher comorbidity, or other factors. Regarding the diagnostic assessments, these were less homogeneous compared to the BBA study, and there is no information on inter-rater reliability. It may have been lower, since no collective training or inter-rater reliability testing were conducted at the clinic.

The strengths of this sample include a relatively well-diagnosed and diagnostically heterogeneous study population, which is ecologically valid for a young general psychiatric population.

The non-clinical sample

This sample was the least thoroughly examined, with diagnostic assessments based solely on MINI interviews conducted by telephone, likely resulting in lower diagnostic quality, perhaps with an especially heightened risk for over-diagnosing. Additionally, since the control sample was recruited in a university setting, it is more representative of (mostly) younger, high functioning individuals rather than the general population, which limits the generalizability of results outside this group.

The main strength of the non-clinical sample is simply just that it does not consist of psychiatric patients, thereby providing a valuable comparison group to identify differences relative to the patient populations.

Methods and procedure

Study design

Limitations

As previously mentioned a cross-sectional design, while practical to implement offers limited opportunities to draw conclusions about causal relationships. Consequently, caution is advised when interpreting concepts such as mediation and moderation. Measuring childhood trauma retrospectively while assessing *current* functioning and comorbidity, is one way to address this issue within the inherent limitations of the cross-sectional design.

A notable issue in Study II is the absence of a previously validated measure of childhood trauma to validate the ETISR-SF against. Instead, we assessed discriminant validity by comparing participants with and without presumed associations to trauma. However, this approach introduces another challenge, as we cannot confirm whether the reported experiences occurred before the

age of 18 (therefore measured by the ETISR-SF) or later in their lives. The young age of our participants likely reduces this risk, though it does not eliminate it entirely.

Another limitation, particularly relevant to Study IV, is the lack of estimates of symptom severity. Symptom severity may have been a better predictor of functioning than the blunter measure of comorbidity, which is based solely on the number of diagnoses. Additionally, we lacked data on treatment received, which might also have influenced functioning, especially in the clinical groups.

Strengths

In Study I, the incorporation of a socio-demographic index further strengthens its conclusions through validating the SDS not only against another measure of disability but also against hard socio-demographic data.

The strengths of the studies investigating predictors of functioning, especially Study IV, includes the consideration of multiple factors, whereas most previous studies have focused on the effects on functioning by a single variable.

Another strength of these studies is that they include both clinical and non-clinical samples, thereby encompassing a broad range of functioning among participants, from severely dysfunctional to highly functional without impairment. This allowed for the research questions to be examined across markedly different populations.

On the other hand, the inclusion of two "extreme" groups - one highly comorbid, traumatized, and dysfunctional clinical group, and one non-clinical group with very low levels of comorbidity, trauma, and disability – also has its drawbacks. Narrowly defined groups limit generalizability and pose statistical challenges due to reduced variance within each group. An alternative approach could have been to analyse a larger and more heterogeneous sample.

Assessments

All information was gathered from the participants, either through self-report questionnaires or via interviews where assessors interpreted the information provided. Relying solely on a single source of information increases the risk of bias, and additional data from family, friends, or formal tests (e.g. of functioning or personality) could have been valuable.

One weakness of the studies involving the UPP and non-clinical samples is the lack of data on neuropsychiatric or personality disorders, which could influence both comorbidity and functioning.

Additionally, self-rated functioning using the SDS (which was primarily used in all studies) may be a less valid measure of functioning compared to clinician-rated measures, such as the Global Assessment of Functioning (GAF), as suggested by the results of Study I, which may have contributed to weaker correlations in the findings. On the other hand, self-rated instruments

have also been suggested to be superior, providing a better method of capturing an individual's experiences and contextual background, unaffected by the clinicians cultural bias (21).

The method for calculating a "trauma score" based on participants' ETISR-SF responses can theoretically be carried out in multiple ways. The standard way of computing the ETISR-SF total score as described in the methods section, does not account for several trauma-related factors, such as severity, perpetrator, frequency, or age, which could influence later functioning.

A broader issue relevant to the use of the ETISR-SF is recall bias in recollections of childhood trauma. This problem has been discussed in the literature (126-128). However, it is challenging to identify more reliable and feasible methods of measuring childhood trauma than relying on the patient's own recalled memories.

A general issue for many of the self-report measures used and some diagnostic procedures (e.g. personality disorders) is the influence of state on the self-assessment of trait or trait-like variables. For instance, having a current severe depressive episode could lead patients to report a more neurotic personality, more insecure attachment patterns, or lower functioning than would otherwise have been the case without the depressive state influencing their perception.

A strength of both clinical samples is the thoroughly conducted Axis I diagnostics, particularly in the BBA sample, where we were also able to demonstrate good inter-rater reliability.

Finally, while parts of the discussion above assume that personality traits lead to life problems, there is also research suggesting that personality may change in response to life experiences and psychiatric illness.

Conclusions

Study I

- The Swedish translation of the SDS has similar psychometric properties to the original version.
- Exploratory analysis supported a one-factor structure, which confirms the validity of calculating the SDS total score.
- The Swedish translation showed concurrent and external validity.

Study II

- The Swedish translation of the ETISR-SF has similar psychometric properties to both the original version and other translations.
- It has also shown discriminant validity.

Study III

- Comorbidity predicted functioning in both clinical and non-clinical samples.
- Childhood trauma predicted functioning only in the clinical sample.
- The effect of childhood trauma on functioning was partly mediated through comorbidity.
- No moderation effects were detected.
- A relatively small portion of overall functioning was explained by childhood trauma and comorbidity, suggesting that several other factors also influence adult functioning.

Study IV

- The personality traits Harm Avoidance and Self-directedness were strong predictors of functioning.
- When these traits were taken into account, other factors usually associated with impairment, such as attachment, comorbidity or childhood trauma, were no longer significant predictors of functioning in the studied sample.

Clinical implications

Study I

The SDS could be useful in clinical settings due to its satisfactory psychometric properties and ease of administration. However, in practice, changed copyright restrictions and recently introduced high per-use costs have made it practically impossible to use in publicly funded healthcare. This makes validated, free alternatives more appealing to use in practice.

Study II

The satisfying psychometric properties of the ETISR-SF make it a valuable instrument, also for clinical settings. The instrument is probably too detailed for routine use in, for example, primary care, where a simpler screening tool might be preferable. However, in settings where complex psychiatric cases need to be assessed, or in psychiatric clinics specializing in trauma treatment, the ETISR-SF could prove valuable.

Depending on the clinically relevant questions in a given setting, it may also be worth considering an even shorter version of the ETISR-SF. As discussed above, answering detailed questions about trauma can be emotionally taxing, and potentially avoidable if the responses are not essential for the clinical work.

Study III

The results in the clinical group suggest that childhood trauma affects functioning through current comorbidity. This highlights the importance of assessing both post-traumatic symptoms and psychiatric morbidity in psychiatric patient groups, and understanding how different factors can affect each other in the complex picture we often see in specialized psychiatric care.

Study IV

The results highlight the importance of taking personality traits into account in clinical assessments of functioning. If in future research these results are shown to be generalizable, and given there is a causal relationship between HA/SD and functioning, some interesting clinical questions can be raised. Can a person's level of HA/SD be changed through pharmacological or therapeutic interventions, and could treatments directed specifically at these traits be a possible pan-diagnostic method to increase patient functioning? In CBT, for example, techniques aimed at reducing anxiety-driven avoidance behaviours are a central component. Could these techniques or SSRI's be used to reduce a person's HA? Similarly, could techniques from Acceptance Commitment Therapy (ACT) or Good Psychiatric Management (GPM) be applied to increase a person's Self-directedness and thereby level of functioning?

There is some support for treatment, particularly CBT, possibly affecting HA and SD favourably. A difficulty when researching this subject is discerning what constitutes the underlying "true" personality (trait) and to what extent personality is temporarily affected by the depression (state). In the case of depression, several studies on pharmacological treatment have shown effects in the form of lower HA and/or higher SD, although at least some of these effects are believed to be state effects (190, 192-194).

Treatment studies of anxiety disorders and obsessive-compulsive disorder (OCD), which are perhaps less prone to the state vs. trait problem, generally show lower HA and, in the case of CBT, also higher SD after treatment (196, 199). The same has been shown for eating disorders, although results vary regarding if this is connected to state effects such as comorbid depression (197, 198).

Future directions

The SDS and the ETISR-SF

Several validation studies, often with similar results, have been conducted on the instruments in their present form. However, studies comparing the psychometric properties of an instrument with other commonly used tools in the field, such as a comparison between SDS and WHODAS 2.0, could be valuable, especially as the latter can be used free of charge.

There may also be potential for improvements or simplifications of the instruments themselves. Regarding the SDS, potential improvements could be

made in both the wording of the questions and the instructions provided to the person using the instrument. Since there is no timeframe included in the phrasing of the SDS's three questions, at least not in all versions, it is unclear which time period is actually being rated. Adding a time period, such as "this week" or "this month", could increase accuracy. It is also problematic that there have been several similar, but slightly different, versions of the SDS in circulation. Additionally, both "family life" and "home responsibilities" are rated on one item, and the difference and weighting between these two constructs is not clear. This could be clarified (e.g. if the lowest of the two is to be rated if they differ). Further, it is unclear whether impairment due to somatic symptoms is to be included in the rating. This probably did not pose a significant problem in the young and somatically healthy study population used in this thesis, but could be of greater importance in other groups.

Regarding the ETISR-SF, there may be potential to develop an even shorter version of the form, possibly one that is also less emotionally burdensome for participants or patients to complete. From a participant perspective, some had concerns about the length of the instrument, as well as the questions where the trauma categories could be rated as having had a positive impact on emotional wellbeing and functioning, which some participants perceived to be a very unlikely, and almost offensive, option. A solution to both these problems could be to develop an even shorter version of the ETISR-SF, which only measure the number of different traumas, and not any additional information for use in some research and non-specialized clinical settings. The information obtained would not of course be as comprehensive, but since number of traumas seems to be the most widely used ETI-measurement, it would probably be sufficient for at least some situations where the ETISR-SF is used.

Improved understanding of factors affecting functioning

A critical question is which factors and mechanisms should be considered when seeking to understand the determinants of functioning in psychiatric patients. The findings of this thesis highlight personality as a possible key factor, but in light of previous studies, it remains important to also continue examining variables that many other studies have repeatedly shown to be linked to functioning, such as psychiatric comorbidity and trauma.

There are also many other interesting variables that might be associated with functioning, but which were not investigated in this thesis. Examples of these include treatment interventions (both psychological, medical and social) and cognitive capabilities. In other populations, such as older adults, it would also be relevant to consider somatic illnesses and various forms of physical disabilities, in which these are probably more prevalent.

Finally, disentangling an "inherent individual level of functioning" from a person's social context and available support is probably impossible. How-

ever, broadening the perspective to include social factors - such as social support, socioeconomic status, and related societal influences - appears vital to fully understand what impacts functioning.

Another patient-centered approach for future research could involve using qualitative methods to explore patients' own experiences of their functioning and the factors they perceive to have influenced it - an approach that could both enhance understanding of the patient perspective and generate new research ideas.

Regarding mechanisms, it would also be worthwhile to investigate whether mediation and moderation play a role for factors beyond those studied in Study III. For instance, could personality moderate the effects of comorbidity?

A deeper understanding of the factors influencing functioning would not only provide healthcare professionals with better tools to understand their patients and their life situations, but it could also enable healthcare systems and society to tailor interventions that improve functioning in psychiatric patients, which ultimately would help these individuals achieve the life they aspire to lead.

Sammanfattning på svenska

Syftet med denna avhandling är att bidra till förståelsen av vilka faktorer som påverkar funktionsnivån hos unga psykiatriska patienter och hur dessa faktorer kan mätas på ett tillförlitligt sätt. Med funktion avses här en persons förmåga att hantera viktiga livsområden såsom arbete, relationer och vardagslivets krav. Ökad kunskap om vad som påverkar funktionsnivån är viktig – inte bara för att vårdpersonal ska få en bättre förståelse för sina patienter utan också för att kunna erbjuda ett mer individanpassat behandlingsupplägg, vilket i förlängningen ger varje individ bättre förutsättningar att leva det liv som känns mest meningsfullt för dem.

Aktuellt forskningsläge

Tidigare forskning har visat att flera olika faktorer påverkar en individs funktionsnivå. Man vet exempelvis att psykiatriska diagnoser är kopplade till lägre funktionsnivå, särskilt om man har flera diagnoser samtidigt. Även erfarenheter av trauma under barndomen kan vara associerade till sämre funktion som vuxen.

Hur man är som person kan också spela roll. Personlighet kan beskrivas som det bestående mönster av känslor, tankar och beteenden som formar en individs sätt att se på och hantera livet. Tidigare forskning har visat att vissa personlighetsdrag kan påverka funktionsnivån positivt eller negativt. Inom psykiatrin finns det en grupp diagnoser som kallas personlighetssyndrom, vilka innebär att man har vissa maladaptiva personlighetsdrag som orsakar lidande och funktionsförlust för personen i fråga. Dessa diagnoser är tydligt kopplade till lägre funktionsnivå.

Anknytning handlar om hur vi relaterar till viktiga personer i våra liv och hur vi agerar i nära relationer. Man kan antingen ha ett tryggt eller ett otryggt anknytningsmönster, och relationer kan vid otrygg anknytning präglas av undvikande av närhet, bekräftelsesökande eller överdriven oro kring relationer – något som kan vara negativt för funktion i sociala sammanhang.

Studie I och II: utvärdering av bedömningsinstrument

I Studie I undersöktes den svenska översättningen av Sheehan Disability Scale (SDS), ett självskattningsformulär där patienter bedömer sin funktionsför-

måga inom områdena arbete/studier, socialt och ansvar i hemmet. Studien visade att den svenska översättningen har samma faktorstruktur och interna konsistens som originalet, vilket innebär att frågorna hänger ihop på ett meningsfullt sätt. Även instrumentets validitet, dvs om det mäter vad det är avsett att mäta, bedömdes. Resultaten visade att resultatet på SDS var kopplat till olika sociodemografiska faktorer såsom arbete, utbildning och ekonomi, samt att SDS-poängen överensstämmer med ett annat sedan tidigare välbeprövat mått på funktion. Detta talar för god validitet.

I studie II undersöktes egenskaperna hos den svenska översättningen av Early Trauma Inventory (ETISR-SF), ett självskattningsformulär rörande erfarenheter av barndomstrauma inom fyra olika områden. Även här visade resultaten att faktorstruktur och intern konsistens hos översättningen motsvarade originalet, och att instrumentet kunde skilja mellan grupper med olika grad av traumatisering, vilket även stärker dess validitet.

Studie III och IV: faktorer som predicerar funktionsnivån

I Studie III undersöktes tre modeller för hur barndomstrauma och psykiatriska sjukdomar kan samspela för att påverka funktionsnivå som vuxen:

- Faktorerna påverkar funktionsnivån var och en för sig.
- Barndomstrauma ger upphov till psykiatrisk sjuklighet vilket i sin tur påverkar funktionen (mediering).
- Graden av en faktor (tex barndomstrauma) styr hur mycket påverkan en annan faktor (tex psykiatrisk sjuklighet) får på funktionsnivån (moderering).

Resultaten från en studiegrupp bestående av psykiatriska patienter visade att både barndomstrauma och psykiatrisk samsjuklighet var associerade med funktionsförmåga, och förklarade en mindre del av variationen (ca 8%) i funktionsnivå. Ungefär en fjärdedel av den effekten förmedlades via samsjuklighet (dvs mediering). I en kontrollgrupp bestående framförallt av studenter var det endast psykiatrisk sjuklighet som påverkade funktionsförmågan. Ingen moderering kunde påvisas i någon av grupperna.

I Studie IV undersöktes ett större antal faktorer som kan påverka funktion: personlighetsdrag, anknytningsmönster, barndomstrauma, psykiatrisk sjuklighet samt personlighetssyndrom. Syftet var att avgöra vilka faktorer som predicerade funktion, och hur mycket av variationen i funktionsnivå de tillsammans kunde förklara. När alla faktorer analyserandes tillsammans i samma statistiska modell var det enbart två personlighetsdrag som förklarade skillnaderna i funktion. Det första, Harm Avoidance (HA), innebär en benägenhet att känna oro och undvika situationer som framkallar ångest. Det andra, Self-Directedness (SD), handlar om förmågan att sätta upp mål och agera flexibelt för att uppnå dem. Dessa två personlighetsdrag kunde tillsammans förklara ungefär en tredjedel av variationen i funktionsnivå mellan patienterna.

Slutsatser

Sammanfattningsvis stödjer resultaten från avhandlingens delstudier att funktion och barndomstrauma kan mätas på ett tillförlitligt sätt med SDS respektive ETISR-SF samt att bland annat personlighetsfaktorer kan vara viktiga prediktorer av funktionsnivån hos psykiatriska patienter.

Studierna I och II visar att de svenska översättningarna av SDS och ETISR-SF har tillfredsställande psykometriska egenskaper.

Resultaten från Studie III indikerar att psykiatrisk sjuklighet och barndomstrauma skulle kunna ha en koppling till funktionsnivån som vuxen, och att effekten av barndomstrauma till viss del skulle kunna förmedlas (medieras) via psykiatrisk sjuklighet i den psykiatriska population som undersöktes.

Vid en undersökning av ytterligare faktorer som predicerar funktionsnivå hos en annan grupp psykiatriska patienter (Studie IV) framträder två personlighetsdrag, HA och SD, som betydelsefulla, och dessa förklarade en relativt stor del av variationen i funktion. Anmärkningsvärt är att psykiatrisk sjukdom och trauma inte hade något förklaringsvärde.

Slutligen är det viktigt att understryka att resultat från enskilda studier som dessa inte motsvarar någon definitiv ”sanning”, utan snarare bör ses som en indikation på hur det skulle kunna förhålla sig i den typ av material som undersökts. Vidare bör poängteras att resultaten som presenterats i Studie III och IV speglar statistiska prediktioner och korrelationer. Detta innebär inte att motsvarande orsakssamband nödvändigtvis existerar. Samtidigt utesluter det förstås inte möjligheten att det kan finnas kausala samband mellan exempelvis vissa personlighetsdrag och funktionsförmåga, men detta kan inte fastställas utifrån de resultat som presenterats här.

Kliniska implikationer

Även om man bör vara mycket försiktig med att dra alltför långtgående slutsatser från enstaka studier, belyser denna avhandling vikten av att ta hänsyn till personlighet vid kliniska bedömningar av funktionsnivå. Om de resultat som presenterats här kan replikeras i framtida forskning, och förutsatt att det finns ett kausalt samband mellan HA, SD och funktionsförmåga, väcks några intressanta kliniska frågor: Kan en persons nivå av HA och SD förändras genom farmakologiska eller terapeutiska insatser, och skulle behandlingar riktade specifikt mot dessa personlighetsdrag i så fall kunna vara en metod för att förbättra patienters funktionsförmåga, oavsett grunddiagnos?

Det finns i dagsläget viss evidens för att behandlingar, särskilt kognitiv beteendeterapi (KBT), möjligen kan påverka HA och SD på ett gynnsamt sätt. Mer behandlingsforskning där funktionsförmåga används som utfallsmått behövs emellertid för att närmare utvärdera detta och hur dessa behandlingar i så fall kan göras mer framgångsrika.

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References

1. World Health Organisation (WHO). International Classification of Functioning, Disability and Health: ICF. 2001.
2. Üstün TB. Measuring health and disability: Manual for WHO disability assessment schedule WHODAS 2.0: World Health Organization; 2010.
3. Skevington SM. Quality of Life. In: Fink G, editor. Encyclopedia of Stress (Second Edition). New York: Academic Press; 2007. p. 317-9.
4. Kahneman D, Deaton A. High income improves evaluation of life but not emotional well-being. Proceedings of the National Academy of Sciences of the United States of America. 2010;107(38):16489-93.
5. World Health Orgainsation (WHO). QHOQOL: Measuring Quality of Life. https://wwwwhoint/toolkits/whoqol. 2020.
6. Gregory DJ, Ron; Pratt, Geraldine; Watts, Michael; et al. Quality of Life". Dictionary of Human Geography (5th ed.). Oxford: Wiley-Blackwell 2009(ISBN 978-1-4051-3287-9.).
7. Alonso MAM, Barajas MES, Ordóñez JAG, Ávila Alpirez H, Fhon JRS, Duran-Badillo T. Quality of life related to functional dependence, family functioning and social support in older adults. Rev Esc Enferm USP. 2022;56:e20210482.
8. Karakis I, Boualam N, Moura LM, Howard DH. Quality of life and functional limitations in persons with epilepsy. Epilepsy Res. 2023;190:107084.
9. Liao SC, Ma HM, Lin YL, Huang WL. Functioning and quality of life in patients with somatic symptom disorder: The association with comorbid depression. Comprehensive psychiatry. 2019;90:88-94.
10. Nevarez-Flores AG, Sanderson K, Breslin M, Carr VJ, Morgan VA, Neil AL. Systematic review of global functioning and quality of life in people with psychotic disorders. Epidemiol Psychiatr Sci. 2019;28(1):31-44.
11. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th. Ed-RevisEd ed. Washington, DC:: American Psychiatric Association; 2000.
12. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. ed. Washington, DC:: American Psychiatric Association; 2013.
13. Lam RW, Filteau MJ, Milev R. Clinical effectiveness: the importance of psychosocial functioning outcomes. J Affect Disord. 2011;132 Suppl 1:S9-s13.
14. McKnight PE, Kashdan TB. The importance of functional impairment to mental health outcomes: a case for reassessing our goals in depression treatment research. Clin Psychol Rev. 2009;29(3):243-59.
15. Goering P, Lin E, Campbell D, Boyle MH, Offord DR. Psychiatric disability in Ontario. Canadian journal of psychiatry Revue canadienne de psychiatrie. 1996;41(9):564-71.
16. Piersma HL, Boes JL. The GAF and psychiatric outcome: a descriptive report. Community mental health journal. 1997;33(1):35-41.
17. Michelson D, Lydiard RB, Pollack MH, Tamura RN, Hoog SL, Tepner R, et al. Outcome assessment and clinical improvement in panic disorder: evidence from

a randomized controlled trial of fluoxetine and placebo. The Fluoxetine Panic Disorder Study Group. The American journal of psychiatry. 1998;155(11):1570-7.
18. Sen A. Health: perception versus observation. Bmj. 2002;324(7342):860-1.
19. Swain J, French S. Towards an Affirmation Model of Disability. Disability & Society. 2000;15(4):569-82.
20. World Health Organisation (WHO). ICF: International classification of functioning, disability and health. 2002.
21. Federici S, Bracalenti M, Meloni F, Luciano JV. World Health Organization disability assessment schedule 2.0: An international systematic review. Disability and rehabilitation. 2017;39(23):2347-80.
22. Sheehan D. The Anxiety Disease. Scribner. 1983.
23. Engel GL. The need for a new medical model: a challenge for biomedicine. Science (New York, NY). 1977;196(4286):129-36.
24. McCutcheon VV. Toward an Integration of Social and Biological Research. Soc Serv Rev. 2006;80(1):159-78.
25. Assary E, Vincent JP, Keers R, Pluess M. Gene-environment interaction and psychiatric disorders: Review and future directions. Semin Cell Dev Biol. 2018;77:133-43.
26. Rutter M. Developmental psychopathology: a paradigm shift or just a relabeling? Development and psychopathology. 2013;25(4 Pt 2):1201-13.
27. Ekeus C, Lindstrom K, Lindblad F, Rasmussen F, Hjern A. Preterm birth, social disadvantage, and cognitive competence in Swedish 18- to 19-year-old men. Pediatrics. 2010;125(1):e67-73.
28. McLaren N. A critical review of the biopsychosocial model. The Australian and New Zealand journal of psychiatry. 1998;32(1):86-92; discussion 3-6.
29. Tavakoli HR. A closer evaluation of current methods in psychiatric assessments: a challenge for the biopsychosocial model. Psychiatry (Edgmont (Pa : Township)). 2009;6(2):25-30.
30. Alvarez AS, Pagani M, Meucci P. The clinical application of the biopsychosocial model in mental health: a research critique. American journal of physical medicine & rehabilitation. 2012;91(13 Suppl 1):S173-80.
31. Smith RC, Fortin AH, Dwamena F, Frankel RM. An evidence-based patient-centered method makes the biopsychosocial model scientific. Patient education and counseling. 2013;91(3):265-70.
32. Adler RH. Engel's biopsychosocial model is still relevant today. Journal of psychosomatic research. 2009;67(6):607-11.
33. Borrell-Carrio F, Suchman AL, Epstein RM. The biopsychosocial model 25 years later: principles, practice, and scientific inquiry. Annals of family medicine. 2004;2(6):576-82.
34. Wade DT, Halligan PW. The biopsychosocial model of illness: a model whose time has come. Clinical rehabilitation. 2017;31(8):995-1004.
35. Fava GA, Sonino N. The biopsychosocial model thirty years later. Psychotherapy and psychosomatics. 2008;77(1):1-2.
36. Zuckerman M, Riskind JH. Vulnerability to psychopathology: A biosocial model. Springer; 2000.
37. Gutman DA, Nemeroff CB. Persistent central nervous system effects of an adverse early environment: clinical and preclinical studies. Physiol Behav. 2003;79(3):471-8.
38. Heim C, Nemeroff CB. The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. Biol Psychiatry. 2001;49(12):1023-39.

39. Talo SA, Rytökoski UM. BPS-ICF model, a tool to measure biopsychosocial functioning and disability within ICF concepts: theory and practice updated. International journal of rehabilitation research Internationale Zeitschrift fur Rehabilitationsforschung Revue internationale de recherches de readaptation. 2016;39(1):1-10.
40. Wu AD, & Zumbo, B. D. . Understanding and using mediators and moderators. Social Indicators Research. 2008;87(87):367-92.
41. Olfson M, Fireman B, Weissman MM, Leon AC, Sheehan DV, Kathol RG, et al. Mental disorders and disability among patients in a primary care group practice. The American journal of psychiatry. 1997;154(12):1734-40.
42. Ormel J, Von Korff M, Van den Brink W, Katon W, Brilman E, Oldehinkel T. Depression, anxiety, and social disability show synchrony of change in primary care patients. Am J Public Health. 1993;83(3):385-90.
43. Hilsenroth MJ, Ackerman SJ, Blagys MD, Baumann BD, Baity MR, Smith SR, et al. Reliability and validity of DSM-IV axis V. The American journal of psychiatry. 2000;157(11):1858-63.
44. Ramirez A, Ekselius L, Ramklint M. Axis V - Global Assessment of Functioning scale (GAF), further evaluation of the self-report version. Eur Psychiatry. 2008;23(8):575-9.
45. Huppert JD, Simpson HB, Nissenson KJ, Liebowitz MR, Foa EB. Quality of life and functional impairment in obsessive-compulsive disorder: a comparison of patients with and without comorbidity, patients in remission, and healthy controls. Depression and anxiety. 2009;26(1):39-45.
46. Hecht H, von Zerssen D, Wittchen HU. Anxiety and depression in a community sample: the influence of comorbidity on social functioning. J Affect Disord. 1990;18(2):137-44.
47. Farooqui AM, Arya A, Singh A, Dalal PK. Psychiatric Comorbidity, Psychosocial Problems, and Functioning of People Who Inject Opioids: An Observational Study. Addict Health. 2022;14(3):218-23.
48. Robins E, Guze SB. Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. The American journal of psychiatry. 1970;126(7):983-7.
49. American Psychiatric Association. Diagnostic and statistical manual of mental disorders.Third edition (DSM-III). 1980.
50. Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, et al. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J Clin Psychiatry. 1998;59 Suppl 20:22-33;quiz 4-57.
51. First MB, R. L. Spitzer, et al. Structured Clinical Interview for DSM-IV Axis I Disorders, Clinical Version, (SCID-CV). Washington, DC, American Psychiatric Press, Inc. 1996.
52. First MB, R. L. Spitzer, et al. . Structured Clinical Interview for DSM-IV Personality Disorders, (SCID-II). Washington, DC, American Psychiatric Press, Inc. 1997.
53. Spitzer RL. Psychiatric diagnosis: are clinicians still necessary? Comprehensive psychiatry. 1983;24(5):399-411.
54. Statens Beredning för medicinsk och social utvärdering (SBU). Diagnostik och uppföljning av förstämningssyndrom. En systematisk litteraturöversikt. . 2012(SBU-rapport nr 212.).
55. Lobbestael J, Leurgans M, Arntz A. Inter-rater reliability of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID I) and Axis II Disorders (SCID II). Clinical psychology & psychotherapy. 2011;18(1):75-9.

56. Kucmin T, Kucmin A, Nogalski A, Sojczuk S, Jojczuk M. History of trauma and posttraumatic disorders in literature. Psychiatria polska. 2016;50(1):269-81.
57. Loughran T. Shell shock, trauma, and the First World War: the making of a diagnosis and its histories. Journal of the history of medicine and allied sciences. 2012;67(1):94-119.
58. Turnbull GJ. A review of post-traumatic stress disorder. Part I: Historical development and classification. Injury. 1998;29(2):87-91.
59. Opendak M, Sullivan RM. Unique neurobiology during the sensitive period for attachment produces distinctive infant trauma processing. Eur J Psychotraumatol. 2016;7:31276-.
60. Oh DL, Jerman P, Silvério Marques S, Koita K, Purewal Boparai SK, Burke Harris N, et al. Systematic review of pediatric health outcomes associated with childhood adversity. BMC Pediatr. 2018;18(1):83.
61. Pitman RK, Rasmusson AM, Koenen KC, Shin LM, Orr SP, Gilbertson MW, et al. Biological studies of post-traumatic stress disorder. Nature reviews Neuroscience. 2012;13(11):769-87.
62. Silverman AB, Reinherz HZ, Giaconia RM. The long-term sequelae of child and adolescent abuse: a longitudinal community study. Child Abuse Negl. 1996;20(8):709-23.
63. Park S, Hong JP, Bae JN, Cho SJ, Lee DW, Lee JY, et al. Impact of childhood exposure to psychological trauma on the risk of psychiatric disorders and somatic discomfort: single vs. multiple types of psychological trauma. Psychiatry research. 2014;219(3):443-9.
64. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. Am J Prev Med. 1998;14(4):245-58.
65. Marsac ML, Kassam-Adams N, Delahanty DL, Widaman KF, Barakat LP. Posttraumatic stress following acute medical trauma in children: a proposed model of bio-psycho-social processes during the peri-trauma period. Clin Child Fam Psychol Rev. 2014;17(4):399-411.
66. Sesar K, Simic N, Barisic M. Multi-type childhood abuse, strategies of coping, and psychological adaptations in young adults. Croat Med J. 2010;51(5):406-16.
67. Rodgers CS, Lang AJ, Laffaye C, Satz LE, Dresselhaus TR, Stein MB. The impact of individual forms of childhood maltreatment on health behavior. Child Abuse Negl. 2004;28(5):575-86.
68. Marshall BD, Galea S, Wood E, Kerr T. Longitudinal associations between types of childhood trauma and suicidal behavior among substance users: a cohort study. Am J Public Health. 2013;103(9):e69-75.
69. Miller AB, Esposito-Smythers C, Weismoore JT, Renshaw KD. The relation between child maltreatment and adolescent suicidal behavior: a systematic review and critical examination of the literature. Clin Child Fam Psychol Rev. 2013;16(2):146-72.
70. Sugaya L, Hasin DS, Olfson M, Lin KH, Grant BF, Blanco C. Child physical abuse and adult mental health: a national study. Journal of traumatic stress. 2012;25(4):384-92.
71. McKay MT, Kilmartin L, Meagher A, Cannon M, Healy C, Clarke MC. A revised and extended systematic review and meta-analysis of the relationship between childhood adversity and adult psychiatric disorder. J Psychiatr Res. 2022;156:268-83.

72. McCutcheon VV, Sartor CE, Pommer NE, Bucholz KK, Nelson EC, Madden PAF, et al. Age at trauma exposure and PTSD risk in young adult women. Journal of traumatic stress. 2010;23(6):811-4.
73. Maercker A, Michael T, Fehm L, Becker ES, Margraf J. Age of traumatisation as a predictor of post-traumatic stress disorder or major depression in young women. The British journal of psychiatry : the journal of mental science. 2004;184:482-7.
74. Copeland WE, Shanahan L, Hinesley J, Chan RF, Aberg KA, Fairbank JA, et al. Association of Childhood Trauma Exposure With Adult Psychiatric Disorders and Functional Outcomes. JAMA Netw Open. 2018;1(7):e184493-e.
75. Dunn EC, Soare TW, Raffeld MR, Busso DS, Crawford KM, Davis KA, et al. What life course theoretical models best explain the relationship between exposure to childhood adversity and psychopathology symptoms: recency, accumulation, or sensitive periods? Psychological medicine. 2018;48(15):2562-72.
76. Dunn EC, Nishimi K, Powers A, Bradley B. Is developmental timing of trauma exposure associated with depressive and post-traumatic stress disorder symptoms in adulthood? Journal of psychiatric research. 2017;84:119-27.
77. Carr CP, Martins CMS, Stingel AM, Lemgruber VB, Juruena MF. The role of early life stress in adult psychiatric disorders: a systematic review according to childhood trauma subtypes. The Journal of nervous and mental disease. 2013;201(12):1007-20.
78. Fergusson DM, Boden JM, Horwood LJ. Exposure to childhood sexual and physical abuse and adjustment in early adulthood. Child abuse & neglect. 2008;32(6):607-19.
79. Nilsson M, Lundh LG, Westling S. Childhood maltreatment and self-hatred as distinguishing characteristics of psychiatric patients with self-harm: A comparison with clinical and healthy controls. Clinical psychology & psychotherapy. 2022;29(5):1778-89.
80. Edwards VJ, Freyd JJ, Dube SR, Anda RF, Felitti VJ. Health outcomes by closeness of sexual abuse perpetrator: A test of betrayal trauma theory. Journal of Aggression, Maltreatment & Trauma. 2012;21(2):133-48.
81. Kiser LJ, Stover CS, Navalta CP, Dorado J, Vogel JM, Abdul-Adil JK, et al. Effects of the child-perpetrator relationship on mental health outcomes of child abuse: it's (not) all relative. Child abuse & neglect. 2014;38(6):1083-93.
82. Davidson G, Shannon C, Mulholland C, Campbell J. A longitudinal study of the effects of childhood trauma on symptoms and functioning of people with severe mental health problems. Journal of trauma & dissociation : the official journal of the International Society for the Study of Dissociation. 2009;10(1):57-68.
83. Gil A, Gama CS, de Jesus DR, Lobato MI, Zimmer M, Belmonte-de-Abreu P. The association of child abuse and neglect with adult disability in schizophrenia and the prominent role of physical neglect. Child Abuse Negl. 2009;33(9):618-24.
84. Sansone RA, Dakroub H, Pole M, Butler M. Childhood trauma and employment disability. International journal of psychiatry in medicine. 2005;35(4):395-404.
85. Peleikis DE, Mykletun A, Dahl AA. Current mental health in women with childhood sexual abuse who had outpatient psychotherapy. Eur Psychiatry. 2005;20(3):260-7.
86. Gunderson JG, Daversa MT, Grilo CM, McGlashan TH, Zanarini MC, Shea MT, et al. Predictors of 2-year outcome for patients with borderline personality disorder. The American journal of psychiatry. 2006;163(5):822-6.

87. Romano E, Babchishin L, Marquis R, Frechette S. Childhood Maltreatment and Educational Outcomes. Trauma, violence & abuse. 2015;16(4):418-37.
88. Zanarini MC, Yong L, Frankenburg FR, Hennen J, Reich DB, Marino MF, et al. Severity of reported childhood sexual abuse and its relationship to severity of borderline psychopathology and psychosocial impairment among borderline inpatients. The Journal of nervous and mental disease. 2002;190(6):381-7.
89. Cotter J, Kaess M, Yung AR. Childhood trauma and functional disability in psychosis, bipolar disorder and borderline personality disorder: a review of the literature. Irish journal of psychological medicine. 2015;32(1):21-30.
90. Weissman DG, Bitran D, Miller AB, Schaefer JD, Sheridan MA, McLaughlin KA. Difficulties with emotion regulation as a transdiagnostic mechanism linking child maltreatment with the emergence of psychopathology. Development and psychopathology. 2019;31(3):899-915.
91. Kessler RC, McLaughlin KA, Green JG, Gruber MJ, Sampson NA, Zaslavsky AM, et al. Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. The British journal of psychiatry : the journal of mental science. 2010;197(5):378-85.
92. Norman RE, Byambaa M, De R, Butchart A, Scott J, Vos T. The long-term health consequences of child physical abuse, emotional abuse, and neglect: a systematic review and meta-analysis. PLoS Med. 2012;9(11):e1001349.
93. Chen LP, Murad MH, Paras ML, Colbenson KM, Sattler AL, Goranson EN, et al. Sexual abuse and lifetime diagnosis of psychiatric disorders: systematic review and meta-analysis. Mayo Clin Proc. 2010;85(7):618-29.
94. Weiss EL, Longhurst JG, Mazure CM. Childhood sexual abuse as a risk factor for depression in women: psychosocial and neurobiological correlates. The American journal of psychiatry. 1999;156(6):816-28.
95. Widom CS, DuMont K, Czaja SJ. A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. Archives of general psychiatry. 2007;64(1):49-56.
96. McCauley J, Kern DE, Kolodner K, Dill L, Schroeder AF, DeChant HK, et al. Clinical characteristics of women with a history of childhood abuse: unhealed wounds. JAMA : the journal of the American Medical Association. 1997;277(17):1362-8.
97. Mandelli L, Petrelli C, Serretti A. The role of specific early trauma in adult depression: A meta-analysis of published literature. Childhood trauma and adult depression. Eur Psychiatry. 2015.
98. Agorastos A, Pittman JO, Angkaw AC, Nievergelt CM, Hansen CJ, Aversa LH, et al. The cumulative effect of different childhood trauma types on self-reported symptoms of adult male depression and PTSD, substance abuse and health-related quality of life in a large active-duty military cohort. J Psychiatr Res. 2014;58:46-54.
99. Rucklidge JJ, Brown DL, Crawford S, Kaplan BJ. Retrospective reports of childhood trauma in adults with ADHD. J Atten Disord. 2006;9(4):631-41.
100. Read J, van Os J, Morrison AP, Ross CA. Childhood trauma, psychosis and schizophrenia: a literature review with theoretical and clinical implications. Acta psychiatrica Scandinavica. 2005;112(5):330-50.
101. Frissa S, Hatch SL, Gazard B, Fear NT, Hotopf M. Trauma and current symptoms of PTSD in a South East London community. Soc Psychiatry Psychiatr Epidemiol. 2013;48(8):1199-209.
102. Martin-Blanco A, Soler J, Villalta L, Feliu-Soler A, Elices M, Perez V, et al. Exploring the interaction between childhood maltreatment and temperamental

traits on the severity of borderline personality disorder. Comprehensive psychiatry. 2014;55(2):311-8.
103. Widom CS, Czaja SJ, Paris J. A prospective investigation of borderline personality disorder in abused and neglected children followed up into adulthood. Journal of personality disorders. 2009;23(5):433-46.
104. Brown GR, Anderson B. Psychiatric morbidity in adult inpatients with childhood histories of sexual and physical abuse. The American journal of psychiatry. 1991;148(1):55-61.
105. Herman JL, Perry JC, van der Kolk BA. Childhood trauma in borderline personality disorder. The American journal of psychiatry. 1989;146(4):490-5.
106. Ball JS, Links PS. Borderline personality disorder and childhood trauma: evidence for a causal relationship. Curr Psychiatry Rep. 2009;11(1):63-8.
107. Zanarini MC, Williams AA, Lewis RE, Reich RB, Vera SC, Marino MF, et al. Reported pathological childhood experiences associated with the development of borderline personality disorder. The American journal of psychiatry. 1997;154(8):1101-6.
108. Kuo JR, Khoury JE, Metcalfe R, Fitzpatrick S, Goodwill A. An examination of the relationship between childhood emotional abuse and borderline personality disorder features: the role of difficulties with emotion regulation. Child Abuse Negl. 2015;39:147-55.
109. De Venter M, Elzinga BM, Van Den Eede F, Wouters K, Van Hal GF, Veltman DJ, et al. The associations between childhood trauma and work functioning in adult workers with and without depressive and anxiety disorders. Eur Psychiatry. 2020;63(1):e76.
110. Aversa LH, Lemmer J, Nunnink S, McLay RN, Baker DG. Impact of childhood maltreatment on physical health-related quality of life in U.S. active duty military personnel and combat veterans. Child Abuse Negl. 2014;38(8):1382-8.
111. Tasca GA, Ritchie K, Zachariades F, Proulx G, Trinneer A, Balfour L, et al. Attachment insecurity mediates the relationship between childhood trauma and eating disorder psychopathology in a clinical sample: a structural equation model. Child Abuse Negl. 2013;37(11):926-33.
112. Lysaker PH, Beattie NL, Strasburger AM, Davis LW. Reported history of child sexual abuse in schizophrenia: associations with heightened symptom levels and poorer participation over four months in vocational rehabilitation. The Journal of nervous and mental disease. 2005;193(12):790-5.
113. Rijkers C, Schoorl M, van Hoeken D, Hoek HW. Eating disorders and posttraumatic stress disorder. Curr Opin Psychiatry. 2019;32(6):510-7.
114. Hovens JG, Giltay EJ, Wiersma JE, Spinhoven P, Penninx BW, Zitman FG. Impact of childhood life events and trauma on the course of depressive and anxiety disorders. Acta psychiatrica Scandinavica. 2012;126(3):198-207.
115. Rokita KI, Dauvermann MR, Donohoe G. Early life experiences and social cognition in major psychiatric disorders: A systematic review. Eur Psychiatry. 2018;53:123-33.
116. Martinez-Aran A, Vieta E, Torrent C, Sanchez-Moreno J, Goikolea JM, Salamero M, et al. Functional outcome in bipolar disorder: the role of clinical and cognitive factors. Bipolar disorders. 2007;9(1-2):103-13.
117. Roepke S, Vater A, Preißler S, Heekeren HR, Dziobek I. Social cognition in borderline personality disorder. Front Neurosci. 2012;6:195.
118. Bedard-Gilligan M, Duax Jakob JM, Doane LS, Jaeger J, Eftekhari A, Feeny N, et al. An Investigation of Depression, Trauma History, and Symptom Severity in Individuals Enrolled in a Treatment Trial for Chronic PTSD. Journal of clinical psychology. 2015;71(7):725-40.

119. Olff M, Polak AR, Witteveen AB, Denys D. Executive function in posttraumatic stress disorder (PTSD) and the influence of comorbid depression. Neurobiol Learn Mem. 2014;112:114-21.
120. Breslau N, Davis GC, Andreski P, Peterson E. Traumatic events and posttraumatic stress disorder in an urban population of young adults. Archives of general psychiatry. 1991;48(3):216-22.
121. Müller M, Ajdacic-Gross V, Rodgers S, Kleim B, Seifritz E, Vetter S, et al. Predictors of remission from PTSD symptoms after sexual and non-sexual trauma in the community: A mediated survival-analytic approach. Psychiatry research. 2018;260:262-71.
122. Armenta RF, Walter KH, Geronimo-Hara TR, Porter B, Stander VA, LeardMann CA. Longitudinal trajectories of comorbid PTSD and depression symptoms among U.S. service members and veterans. BMC psychiatry. 2019;19(1):396.
123. Adams SW, Bowler RM, Russell K, Brackbill RM, Li J, Cone JE. PTSD and comorbid depression: Social support and self-efficacy in World Trade Center tower survivors 14-15 years after 9/11. Psychol Trauma. 2019;11(2):156-64.
124. Shah R, Nisenbaum R, Classen C, Eynan R, Links PS. Comorbid Depression and Other Predictors of PTSD Severity in Urban Public Transit Employees. Community mental health journal. 2017;53(2):224-32.
125. Hardt J, Rutter M. Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. Journal of child psychology and psychiatry, and allied disciplines. 2004;45(2):260-73.
126. Chu JA, Frey LM, Ganzel BL, Matthews JA. Memories of childhood abuse: dissociation, amnesia, and corroboration. The American journal of psychiatry. 1999;156(5):749-55.
127. Williams LM. Recall of childhood trauma: a prospective study of women's memories of child sexual abuse. J Consult Clin Psychol. 1994;62(6):1167-76.
128. Loftus EF, Garry M, Feldman J. Forgetting sexual trauma: what does it mean when 38% forget? Journal of consulting and clinical psychology. 1994;62(6):1177-81; discussion 82-6.
129. Reuben A, Moffitt TE, Caspi A, Belsky DW, Harrington H, Schroeder F, et al. Lest we forget: comparing retrospective and prospective assessments of adverse childhood experiences in the prediction of adult health. Journal of child psychology and psychiatry, and allied disciplines. 2016;57(10):1103-12.
130. Colman I, Kingsbury M, Garad Y, Zeng Y, Naicker K, Patten S, et al. Consistency in adult reporting of adverse childhood experiences. Psychological medicine. 2016;46(3):543-9.
131. Pietrini F, Lelli L, Verardi A, Silvestri C, Faravelli C. [Retrospective assessment of childhood trauma: review of the instruments]. Riv Psichiatr. 2010;45(1):7-16.
132. Roy CA, Perry JC. Instruments for the assessment of childhood trauma in adults. The Journal of nervous and mental disease. 2004;192(5):343-51.
133. Bremner JD, Vermetten E, Mazure CM. Development and preliminary psychometric properties of an instrument for the measurement of childhood trauma: the Early Trauma Inventory. Depression and anxiety. 2000;12(1):1-12.
134. Bowlby J. Attachment and loss, Vol. 1: Attachment, 2nd ed. Basic Books, New York. 1969.
135. Hazan C, Shaver P. Romantic love conceptualized as an attachment process. Journal of personality and social psychology. 1987;52(3):511-24.
136. Bowlby J. Attachment and Loss, Vol. 1: Attachment. 2 ed. New York: Basic Books; 1969. p. 436.
137. Mikulincer M, Shaver PR. The Attachment Behavioral System in Adulthood: Activation, Psychodynamics, and Interpersonal Processes. Advances in

experimental social psychology, Vol 35. San Diego, CA, US: Elsevier Academic Press; 2003. p. 53-152.
138. Ainsworth MS, Blehar MC, Waters E, Wall S. Patterns of Attachment: A Psychological Study of the Strange Situation. Hillsdale, NJ: Lawrence Erlbaum; 1978.
139. Brennan KA, Clark CL, Shaver PR. Self-report measurement of adult attachment: An integrative overview. In: Rholes JASWS, editor. Attachment theory and close relationships. New York, NY, US: Guilford Press; 1998. p. 46-76.
140. Bakermans-Kranenburg MJ, van IMH. The first 10,000 Adult Attachment Interviews: distributions of adult attachment representations in clinical and non-clinical groups. Attachment & human development. 2009;11(3):223-63.
141. Feeney JA, Noller P, Hanrahan M. Assessing adult attachment. Attachment in adults: Clinical and developmental perspectives. New York, NY, US: Guilford Press; 1994. p. 128-52.
142. Waters E, Hamilton CE, Weinfield NS. The stability of attachment security from infancy to adolescence and early adulthood: general introduction. Child development. 2000;71(3):678-83.
143. Waters E, Weinfield NS, Hamilton CE. The stability of attachment security from infancy to adolescence and early adulthood: general discussion. Child development. 2000;71(3):703-6.
144. Styron T, Janoff-Bulman R. Childhood attachment and abuse: long-term effects on adult attachment, depression, and conflict resolution. Child Abuse Negl. 1997;21(10):1015-23.
145. Sibley CG, Liu JH. Short-term temporal stability and factor structure of the revised experiences in close relationships (ECR-R) measure of adult attachment. Personality and Individual Differences. 2004;36(4):969-75.
146. Zilberstein K. The use and limitations of attachment theory in child psychotherapy. Psychotherapy (Chicago, Ill). 2014;51(1):93-103.
147. Morley TE, Moran G. The origins of cognitive vulnerability in early childhood: Mechanisms linking early attachment to later depression. Clinical Psychology Review. 2011;31(7):1071-82.
148. Morriss RK, van der Gucht E, Lancaster G, Bentall RP. Adult attachment in bipolar 1 disorder. Psychology and psychotherapy. 2009;82(Pt 3):267-77.
149. Strodl E, Noller P. The relationship of adult attachment dimensions to depression and agoraphobia. Personal Relationships. 2003;10(2):171-85.
150. O'Shaughnessy R, Dallos R. Attachment research and eating disorders: a review of the literature. Clinical child psychology and psychiatry. 2009;14(4):559-74.
151. Fossati A, Feeney JA, Donati D, Donini M, Novella L, Bagnato M, et al. Personality disorders and adult attachment dimensions in a mixed psychiatric sample: a multivariate study. The Journal of nervous and mental disease. 2003;191(1):30-7.
152. Agrawal HR, Gunderson J, Holmes BM, Lyons-Ruth K. Attachment studies with borderline patients: a review. Harvard review of psychiatry. 2004;12(2):94-104.
153. Aspelmeier JE, Elliott AN, Smith CH. Childhood sexual abuse, attachment, and trauma symptoms in college females: the moderating role of attachment. Child Abuse Negl. 2007;31(5):549-66.
154. Pearse E, Bucci S, Raphael J, Berry K. The relationship between attachment and functioning for people with serious mental illness: a systematic review. Nordic journal of psychiatry. 2020;74(8):545-57.

155. Lassri D, Luyten P, Fonagy P, Shahar G. Undetected Scars? Self-Criticism, Attachment, and Romantic Relationships Among Otherwise Well-Functioning Childhood Sexual Abuse Survivors. Psychol Trauma. 2017.
156. Shapiro DL, Levendosky AA. Adolescent survivors of childhood sexual abuse: the mediating role of attachment style and coping in psychological and interpersonal functioning. Child Abuse Negl. 1999;23(11):1175-91.
157. McCarthy G, Taylor A. Avoidant/ambivalent attachment style as a mediator between abusive childhood experiences and adult relationship difficulties. Journal of child psychology and psychiatry, and allied disciplines. 1999;40(3):465-77.
158. Lowell A, Renk K, Adgate AH. The role of attachment in the relationship between child maltreatment and later emotional and behavioral functioning. Child Abuse Negl. 2014;38(9):1436-49.
159. Main M. Introduction to the special section on attachment and psychopathology: 2. Overview of the field of attachment. J Consult Clin Psychol. 1996;64(2):237-43.
160. Association AP. APA Dictionary of Psychology. 2024.
161. Digman JM. Personality structure: Emergence of the Five-Factor Model. Annual Review of Psychology. 1990;41.
162. Cattell RB. The description of personality: basic traits resolved into clusters. The Journal of Abnormal and Social Psychology. 1943;38(4):476-506.
163. McCrae RR, John OP. An introduction to the five-factor model and its applications. Journal of personality. 1992;60(2):175-215.
164. Cloninger CR, Svrakic DM, Przybeck TR. A psychobiological model of temperament and character. Archives of general psychiatry. 1993;50(12):975-90.
165. Svrakic DM, Whitehead C, Przybeck TR, Cloninger CR. Differential diagnosis of personality disorders by the seven-factor model of temperament and character. Archives of general psychiatry. 1993;50(12):991-9.
166. Cloninger CR, Svrakic DM, Przybeck TR. A psychobiological model of temperament and character. Archives of general psychiatry. 1993;50(12):975-90.
167. Josefsson K, Jokela M, Cloninger CR, Hintsanen M, Salo J, Hintsa T, et al. Maturity and change in personality: developmental trends of temperament and character in adulthood. Development and psychopathology. 2013;25(3):713-27.
168. Cloninger CR, Przybeck TR, Svrakic DM. The Tridimensional Personality Questionnaire: U.S. normative data. Psychol Rep. 1991;69(3 Pt 1):1047-57.
169. Cloninger CR. The temperament and character inventory-revised. St Louis, MO: Center for Psychobiology of Personality, Washington University. 1999.
170. Hansenne M, Delhez M, Cloninger CR. Psychometric properties of the temperament and character inventory-revised (TCI-R) in a Belgian sample. J Pers Assess. 2005;85(1):40-9.
171. Pelissolo A, Mallet L, Baleyte JM, Michel G, Cloninger CR, Allilaire JF, et al. The Temperament and Character Inventory-Revised (TCI-R): psychometric characteristics of the French version. Acta psychiatrica Scandinavica. 2005;112(2):126-33.
172. Gutiérrez-Zotes JA, Bayón C, Montserrat C, Valero J, Labad A, Cloninger CR, et al. [Temperament and Character Inventory Revised (TCI-R). Standardization and normative data in a general population sample]. Actas espanolas de psiquiatria. 2004;32(1):8-15.
173. Fossati A, Cloninger CR, Villa D, Borroni S, Grazioli F, Giarolli L, et al. Reliability and validity of the Italian version of the Temperament and Character Inventory-Revised in an outpatient sample. Comprehensive psychiatry. 2007;48(4):380-7.

174. Brändström S, Richter J, Nylander PO. Further development of the Temperament and Character Inventory. Psychol Rep. 2003;93(3 Pt 2):995-1002.
175. Herbst JH, Zonderman AB, McCrae RR, Costa PT, Jr. Do the dimensions of the temperament and character inventory map a simple genetic architecture? Evidence from molecular genetics and factor analysis. The American journal of psychiatry. 2000;157(8):1285-90.
176. Ball SA, Tennen H, Kranzler HR. Factor replicability and validity of the Temperament and Character Inventory in substance-dependent patients. Psychol Assess. 1999;11(4):514.
177. Gana K, Trouillet R. Structure invariance of the Temperament and Character Inventory (TCI). Personality and Individual Differences. 2003;35(7):1483-95.
178. Farmer RF, Goldberg LR. A psychometric evaluation of the revised Temperament and Character Inventory (TCI-R) and the TCI-140. Psychol Assess. 2008;20(3):281-91.
179. Ando J, Ono Y, Yoshimura K, Onoda N, Shinohara M, Kanba S, et al. The genetic structure of Cloninger's seven-factor model of temperament and character in a Japanese sample. Journal of personality. 2002;70(5):583-609.
180. Ando J, Suzuki A, Yamagata S, Kijima N, Maekawa H, Ono Y, et al. Genetic and environmental structure of Cloninger's temperament and character dimensions. Journal of personality disorders. 2004;18(4):379-93.
181. Grucza RA, Goldberg LR. The comparative validity of 11 modern personality inventories: predictions of behavioral acts, informant reports, and clinical indicators. J Pers Assess. 2007;89(2):167-87.
182. Capanna C, Struglia F, Riccardi I, Daneluzzo E, Stratta P, Rossi A. Temperament and Character Inventory-R (TCI-R) and Big Five Questionnaire (BFQ): convergence and divergence. Psychol Rep. 2012;110(3):1002-6.
183. Scholte-Stalenhoef AN, Pijnenborg GHM, Hasson-Ohayon I, Boyette LL. Personality traits in psychotic illness and their clinical correlates: A systematic review. Schizophrenia research. 2023;252:348-406.
184. Zwir I, Arnedo J, Del-Val C, Pulkki-Råback L, Konte B, Yang SS, et al. Uncovering the complex genetics of human temperament. Mol Psychiatry. 2020;25(10):2275-94.
185. Nostro AD, Müller VI, Varikuti DP, Pläschke RN, Hoffstaedter F, Langner R, et al. Predicting personality from network-based resting-state functional connectivity. Brain Struct Funct. 2018;223(6):2699-719.
186. Komasi S, Rezaei F, Hemmati A, Rahmani K, Amianto F, Miettunen J. Comprehensive meta-analysis of associations between temperament and character traits in Cloninger's psychobiological theory and mental disorders. J Int Med Res. 2022;50(1):3000605211070766.
187. Gutiérrez F, Gárriz M, Peri JM, Vall G, Torrubia R. How temperament and character affect our career, relationships, and mental health. Comprehensive psychiatry. 2016;70:181-9.
188. He JA, Antshel KM, Biederman J, Faraone SV. Do Personality Traits Predict Functional Impairment and Quality of Life in Adult ADHD? A Controlled Study. Journal of attention disorders. 2019;23(1):12-21.
189. Roberts BW, Kuncel NR, Shiner R, Caspi A, Goldberg LR. The Power of Personality: The Comparative Validity of Personality Traits, Socioeconomic Status, and Cognitive Ability for Predicting Important Life Outcomes. Perspectives on Psychological Science. 2007;2(4):313-45.
190. Hirano S, Sato T, Narita T, Kusunoki K, Ozaki N, Kimura S, et al. Evaluating the state dependency of the Temperament and Character Inventory dimensions in

patients with major depression: a methodological contribution. J Affect Disord. 2002;69(1-3):31-8.
191. Corruble E, Duret C, Pelissolo A, Falissard B, Guelfi JD. Early and delayed personality changes associated with depression recovery? A one-year follow-up study. Psychiatry research. 2002;109(1):17-25.
192. Abrams KY, Yune SK, Kim SJ, Jeon HJ, Han SJ, Hwang J, et al. Trait and state aspects of harm avoidance and its implication for treatment in major depressive disorder, dysthymic disorder, and depressive personality disorder. Psychiatry Clin Neurosci. 2004;58(3):240-8.
193. Boz C, Gazioglu S, Altunayoglu V, Hocaoglu C. Effect of serotonergic antidepressant therapy on temperament and character scales in patients with chronic tension-type headache. Psychiatry Clin Neurosci. 2007;61(5):534-42.
194. Richter J, Eisemann M, Richter G. Temperament and character during the course of unipolar depression among inpatients. Eur Arch Psychiatry Clin Neurosci. 2000;250(1):40-7.
195. Marchesi C, De Panfilis C, Cantoni A, Giannelli MR, Maggini C. Effect of pharmacological treatment on temperament and character in panic disorder. Psychiatry research. 2008;158(2):147-54.
196. Mörtberg E, Bejerot S, Aberg Wistedt A. Temperament and character dimensions in patients with social phobia: patterns of change following treatments? Psychiatry research. 2007;152(1):81-90.
197. Anderson CB, Joyce PR, Carter FA, McIntosh VV, Bulik CM. The effect of cognitive-behavioral therapy for bulimia nervosa on temperament and character as measured by the temperament and character inventory. Comprehensive psychiatry. 2002;43(3):182-8.
198. Dalle Grave R, Calugi S, Brambilla F, Abbate-Daga G, Fassino S, Marchesini G. The effect of inpatient cognitive-behavioral therapy for eating disorders on temperament and character. Behaviour research and therapy. 2007;45(6):1335-44.
199. Lyoo IK, Yoon T, Kang DH, Kwon JS. Patterns of changes in temperament and character inventory scales in subjects with obsessive-compulsive disorder following a 4-month treatment. Acta psychiatrica Scandinavica. 2003;107(4):298-304.
200. Huang Y, Kotov R, de Girolamo G, Preti A, Angermeyer M, Benjet C, et al. DSM-IV personality disorders in the WHO World Mental Health Surveys. The British journal of psychiatry : the journal of mental science. 2009;195(1):46-53.
201. Beckwith H, Moran PF, Reilly J. Personality disorder prevalence in psychiatric outpatients: a systematic literature review. Personal Ment Health. 2014;8(2):91-101.
202. Zimmerman M, Rothschild L, Chelminski I. The prevalence of DSM-IV personality disorders in psychiatric outpatients. The American journal of psychiatry. 2005;162(10):1911-8.
203. Torgersen S, Lygren S, Oien PA, Skre I, Onstad S, Edvardsen J, et al. A twin study of personality disorders. Comprehensive psychiatry. 2000;41(6):416-25.
204. Kendler KS, Czajkowski N, Tambs K, Torgersen S, Aggen SH, Neale MC, et al. Dimensional representations of DSM-IV cluster A personality disorders in a population-based sample of Norwegian twins: a multivariate study. Psychological medicine. 2006;36(11):1583-91.
205. Torgersen S, Czajkowski N, Jacobson K, Reichborn-Kjennerud T, Røysamb E, Neale MC, et al. Dimensional representations of DSM-IV cluster B personality disorders in a population-based sample of Norwegian twins: a multivariate study. Psychological medicine. 2008;38(11):1617-25.

206. Reichborn-Kjennerud T, Czajkowski N, Neale MC, Ørstavik RE, Torgersen S, Tambs K, et al. Genetic and environmental influences on dimensional representations of DSM-IV cluster C personality disorders: a population-based multivariate twin study. Psychological medicine. 2007;37(5):645-53.
207. Czajkowski N, Aggen SH, Krueger RF, Kendler KS, Neale MC, Knudsen GP, et al. A Twin Study of Normative Personality and DSM-IV Personality Disorder Criterion Counts: Evidence for Separate Genetic Influences. The American journal of psychiatry. 2018;175(7):649-56.
208. Bierer LM, Yehuda R, Schmeidler J, Mitropoulou V, New AS, Silverman JM, et al. Abuse and neglect in childhood: relationship to personality disorder diagnoses. CNS spectrums. 2003;8(10):737-54.
209. Porter C, Palmier-Claus J, Branitsky A, Mansell W, Warwick H, Varese F. Childhood adversity and borderline personality disorder: a meta-analysis. Acta psychiatrica Scandinavica. 2020;141(1):6-20.
210. First MB WJ, Benjamin LS, Spitzer RL. User's Guide for the SCID-5-PD (Structured Clinical Interview for DSM-5 Personality Disorder. American Psychiatric Organisation. 2015.
211. McGlashan TH, Grilo CM, Skodol AE, Gunderson JG, Shea MT, Morey LC, et al. The Collaborative Longitudinal Personality Disorders Study: baseline Axis I/II and II/II diagnostic co-occurrence. Acta psychiatrica Scandinavica. 2000;102(4):256-64.
212. Newton-Howes G, Tyrer P, Anagnostakis K, Cooper S, Bowden-Jones O, Weaver T. The prevalence of personality disorder, its comorbidity with mental state disorders, and its clinical significance in community mental health teams. Soc Psychiatry Psychiatr Epidemiol. 2010;45(4):453-60.
213. Skodol AE, Grilo CM, Pagano ME, Bender DS, Gunderson JG, Shea MT, et al. Effects of personality disorders on functioning and well-being in major depressive disorder. Journal of psychiatric practice. 2005;11(6):363-8.
214. Tyrer P, Tyrer H, Yang M, Guo B. Long-term impact of temporary and persistent personality disorder on anxiety and depressive disorders. Personal Ment Health. 2016;10(2):76-83.
215. Newton-Howes G, Tyrer P, Johnson T, Mulder R, Kool S, Dekker J, et al. Influence of personality on the outcome of treatment in depression: systematic review and meta-analysis. Journal of personality disorders. 2014;28(4):577-93.
216. Skodol AE, Gunderson JG, McGlashan TH, Dyck IR, Stout RL, Bender DS, et al. Functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder. The American journal of psychiatry. 2002;159(2):276-83.
217. Newton-Howes G, Tyrer P, Weaver T. Social functioning of patients with personality disorder in secondary care. Psychiatric services. 2008;59(9):1033-7.
218. Johnson JG, Rabkin JG, Williams JB, Remien RH, Gorman JM. Difficulties in interpersonal relationships associated with personality disorders and axis I disorders: a community-based longitudinal investigation. Journal of personality disorders. 2000;14(1):42-56.
219. Ettner SL, Maclean JC, French MT. DOES HAVING A DYSFUNCTIONAL PERSONALITY HURT YOUR CAREER? AXIS II PERSONALITY DISORDERS AND LABOR MARKET OUTCOMES. Ind Relat (Berkeley). 2011;50(1):149-73.
220. Gjerde LC, Røysamb E, Czajkowski N, Knudsen GP, Ostby K, Tambs K, et al. Personality disorders and long-term sick leave: a population-based study of young adult Norwegian twins. Twin research and human genetics : the official journal of the International Society for Twin Studies. 2014;17(1):1-9.

221. Østby KA, Czajkowski N, Knudsen GP, Ystrom E, Gjerde LC, Kendler KS, et al. Personality disorders are important risk factors for disability pensioning. Soc Psychiatry Psychiatr Epidemiol. 2014;49(12):2003-11.
222. Cunningham JL, Zanzi M, Willebrand M, Ekselius L, Ramklint M. No regrets: Young adult patients in psychiatry report positive reactions to biobank participation. BMC psychiatry. 2017;17(1):21.
223. Bixo L, Cunningham JL, Ekselius L, Öster C, Ramklint M. 'Sick and tired': Patients reported reasons for not participating in clinical psychiatric research. Health Expect. 2021;24 Suppl 1(Suppl 1):20-9.
224. Kouros I, Holmberg H, Ekselius L, Ramklint M. Temperament, but not childhood trauma, distinguishes borderline personality disorder from bipolar disorder and ADHD. Nordic journal of psychiatry. 2024;78(1):79-86.
225. Zanarini MC, Frankenburg FR, Hennen J, Reich DB, Silk KR. Psychosocial functioning of borderline patients and axis II comparison subjects followed prospectively for six years. Journal of personality disorders. 2005;19(1):19-29.
226. Jones SH, Thornicroft G, Coffey M, Dunn G. A brief mental health outcome scale-reliability and validity of the Global Assessment of Functioning (GAF). The British journal of psychiatry : the journal of mental science. 1995;166(5):654-9.
227. Goldman HH, Skodol AE, Lave TR. Revising axis V for DSM-IV: a review of measures of social functioning. The American journal of psychiatry. 1992;149(9):1148-56.
228. Startup M, Jackson MC, Bendix S. The concurrent validity of the Global Assessment of Functioning (GAF). The British journal of clinical psychology / the British Psychological Society. 2002;41(Pt 4):417-22.
229. Bodlund O, Kullgren G, Ekselius L, Lindström E, von Knorring L. Axis V--Global Assessment of Functioning Scale. Evaluation of a self-report version. Acta psychiatrica Scandinavica. 1994;90(5):342-7.
230. Arbuckle R, Frye MA, Brecher M, Paulsson B, Rajagopalan K, Palmer S, et al. The psychometric validation of the Sheehan Disability Scale (SDS) in patients with bipolar disorder. Psychiatry research. 2009;165(1-2):163-74.
231. Hodgins DC. Reliability and validity of the Sheehan Disability Scale modified for pathological gambling. BMC psychiatry. 2013;13:177.
232. Leon AC, Olfson M, Portera L, Farber L, Sheehan DV. Assessing psychiatric impairment in primary care with the Sheehan Disability Scale. International journal of psychiatry in medicine. 1997;27(2):93-105.
233. Luciano JV, Bertsch J, Salvador-Carulla L, Tomas JM, Fernandez A, Pinto-Meza A, et al. Factor structure, internal consistency and construct validity of the Sheehan Disability Scale in a Spanish primary care sample. Journal of evaluation in clinical practice. 2010;16(5):895-901.
234. Hambrick JP, Turk CL, Heimberg RG, Schneier FR, Liebowitz MR. Psychometric properties of disability measures among patients with social anxiety disorder. Journal of anxiety disorders. 2004;18(6):825-39.
235. Leon AC, Shear MK, Portera L, Klerman GL. Assessing impairment in patients with panic disorder: the Sheehan Disability Scale. Soc Psychiatry Psychiatr Epidemiol. 1992;27(2):78-82.
236. Sheehan KH, Sheehan DV. Assessing treatment effects in clinical trials with the discan metric of the Sheehan Disability Scale. International clinical psychopharmacology. 2008;23(2):70-83.
237. Sheehan DV, Harnett-Sheehan K, Raj BA. The measurement of disability. International clinical psychopharmacology. 1996;11 Suppl 3:89-95.

238. Bremner JD, Bolus R, Mayer EA. Psychometric properties of the Early Trauma Inventory-Self Report. The Journal of nervous and mental disease. 2007;195(3):211-8.
239. Hyman SM, Garcia M, Kemp K, Mazure CM, Sinha R. A gender specific psychometric analysis of the early trauma inventory short form in cocaine dependent adults. Addict Behav. 2005;30(4):847-52.
240. Plaza A, Torres A, Martin-Santos R, Gelabert E, Imaz ML, Navarro P, et al. Validation and test-retest reliability of Early Trauma Inventory in Spanish post-partum women. The Journal of nervous and mental disease. 2011;199(4):280-5.
241. Jeon JR, Lee EH, Lee SW, Jeong EG, Kim JH, Lee D, et al. The early trauma inventory self report-short form: psychometric properties of the korean version. Psychiatry Investig. 2012;9(3):229-35.
242. Osorio FL, Salum GA, Donadon MF, Forni-Dos-Santos L, Loureiro SR, Crippa JA. Psychometrics properties of early trauma inventory self report - short form (ETISR-SR) for the Brazilian context. PLoS One. 2013;8(10):e76337.
243. Rademaker AR, Vermetten E, Geuze E, Muilwijk A, Kleber RJ. Self-reported early trauma as a predictor of adult personality: a study in a military sample. Journal of clinical psychology. 2008;64(7):863-75.
244. Wang Z, Du J, Sun H, Wu H, Xiao Z, Zhao M. Patterns of childhood trauma and psychological distress among injecting heroin users in China. PLoS One. 2010;5(12):e15882.
245. Fossati A, Feeney JA, Donati D, Donini M, Novella L, Bagnato M, et al. On the Dimensionality of the Attachment Style Questionnaire in Italian Clinical and Nonclinical Participants. Journal of Social and Personal Relationships. 2003;20(1):55-79.
246. Ravitz P, Maunder R, Hunter J, Sthankiya B, Lancee W. Adult attachment measures: a 25-year review. Journal of psychosomatic research. 2010;69(4):419-32.
247. Karantzas GC, Feeney JA, Wilkinson R. Is less more? Confirmatory factor analysis of the Attachment Style Questionnaires. Journal of Social and Personal Relationships. 2010;27(6):749-80.
248. Axfors C, Sylvén S, Skalkidou A, Ramklint M. Psychometric properties of the attachment style questionnaire in Swedish pregnant women: short and full versions. J Reprod Infant Psychol. 2017;35(5):450-61.
249. Håkanson T. Attachment Style Questionnaire, Översättning till svenska samt inledande utprovning. 1996.
250. Brändström S, Richter J, Przybeck T. Distributions by age and sex of the dimensions of temperament and character inventory in a cross-cultural perspective among Sweden, Germany, and the USA. Psychol Rep. 2001;89(3):747-58.
251. Skre I, Onstad S, Torgersen S, Kringlen E. High interrater reliability for the Structured Clinical Interview for DSM-III-R Axis I (SCID-I). Acta psychiatrica Scandinavica. 1991;84(2):167-73.
252. Zanarini MC, Frankenburg FR. Attainment and maintenance of reliability of axis I and II disorders over the course of a longitudinal study. Comprehensive psychiatry. 2001;42(5):369-74.
253. Zanarini MC, Skodol AE, Bender D, Dolan R, Sanislow C, Schaefer E, et al. The Collaborative Longitudinal Personality Disorders Study: reliability of axis I and II diagnoses. Journal of personality disorders. 2000;14(4):291-9.
254. Ramirez Basco M, Bostic JQ, Davies D, Rush AJ, Witte B, Hendrickse W, et al. Methods to improve diagnostic accuracy in a community mental health setting. The American journal of psychiatry. 2000;157(10):1599-605.

255. Kranzler HR, Kadden RM, Babor TF, Tennen H, Rounsaville BJ. Validity of the SCID in substance abuse patients. Addiction (Abingdon, England). 1996;91(6):859-68.
256. Arntz A, van Beijsterveldt B, Hoekstra R, Hofman A, Eussen M, Sallaerts S. The interrater reliability of a Dutch version of the Structured Clinical Interview for DSM-III-R Personality Disorders. Acta psychiatrica Scandinavica. 1992;85(5):394-400.
257. Maffei C, Fossati A, Agostoni I, Barraco A, Bagnato M, Deborah D, et al. Interrater reliability and internal consistency of the structured clinical interview for DSM-IV axis II personality disorders (SCID-II), version 2.0. Journal of personality disorders. 1997;11(3):279-84.
258. Ambrosini PJ. Historical development and present status of the schedule for affective disorders and schizophrenia for school-age children (K-SADS). Journal of the American Academy of Child and Adolescent Psychiatry. 2000;39(1):49-58.
259. Amorim P, Lecrubier Y, Weiller E, Hergueta T, Sheehan D. DSM-IH-R Psychotic Disorders: procedural validity of the Mini International Neuropsychiatric Interview (MINI). Concordance and causes for discordance with the CIDI. European psychiatry : the journal of the Association of European Psychiatrists. 1998;13(1):26-34.
260. DV Sheehan YL, K Harnett Sheehan, J Janavs, E Weiller, A Keskiner, J Schinka, E Knapp, MF Sheehan, GC Dunbar. The validity of the Mini International Neuropsychiatric Interview (MINI) according to the SCID-P and its reliability. Eur Psychiatry. 1997(12):232-41.
261. Cronbach L. Coefficient alpha and the internal structure of tests. Psychometrika. 1951(16):297–334.
262. Hu LB, P.M. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. Structural Equation Modeling. 1999;6(6):1-55.
263. Lenhard W, Lenhard A. Hypothesis tests for comparing correlations. 2014.
264. Hair JF, Black, W. C., Babin, B. J., & Anderson, R. E. . Multivariate data analysis. 7th ed. Harlow, England: Pearson; 2014.
265. Nunnally JC. Psychometric Theory. New York: McGraw-Hill. 1978.
266. Gerdner A, Allgulander C. Psychometric properties of the Swedish version of the Childhood Trauma Questionnaire-Short Form (CTQ-SF). Nordic journal of psychiatry. 2009;63(2):160-70.
267. Masten AS, Hubbard JJ, Gest SD, Tellegen A, Garmezy N, Ramirez M. Competence in the context of adversity: pathways to resilience and maladaptation from childhood to late adolescence. Development and psychopathology. 1999;11(1):143-69.
268. Galatzer-Levy IR, Huang SH, Bonanno GA. Trajectories of resilience and dysfunction following potential trauma: A review and statistical evaluation. Clin Psychol Rev. 2018;63:41-55.
269. Chotai J, Jonasson M, Hägglöf B, Adolfsson R. Adolescent attachment styles and their relation to the temperament and character traits of personality in a general population. Eur Psychiatry. 2005;20(3):251-9.
270. MacDonald K, Berlow R, Thomas ML. Attachment, affective temperament, and personality disorders: a study of their relationships in psychiatric outpatients. J Affect Disord. 2013;151(3):932-41.
271. Ogle CM, Rubin DC, Siegler IC. The relation between insecure attachment and posttraumatic stress: Early life versus adulthood traumas. Psychol Trauma. 2015;7(4):324-32.

272. Noftle EE, Shaver PR. Attachment dimensions and the big five personality traits: Associations and comparative ability to predict relationship quality. Journal of Research in Personality. 2006;40(2):179-208.
273. Cunningham JL, Zanzi M, Willebrand M, Ekselius L, Ramklint M. No regrets: Young adult patients in psychiatry report positive reactions to biobank participation. BMC psychiatry. 2017;17(1):21-.
274. Oexle N, Müller M, Kawohl W, Xu Z, Viering S, Wyss C, et al. Self-stigma as a barrier to recovery: a longitudinal study. Eur Arch Psychiatry Clin Neurosci. 2018;268(2):209-12.
275. Corrigan PW, Rao D. On the self-stigma of mental illness: stages, disclosure, and strategies for change. Canadian journal of psychiatry Revue canadienne de psychiatrie. 2012;57(8):464-9.
276. Cohon R. Hume's Moral Philosophy. The Stanford Encyclopedia of Philosophy. 2018.
277. Hume D. Treatise of Human Nature. 2nd ed ed. Selby-Bigge LA, editor. London, England: Oxford University Press; 1978.

Patient:_ _ _ _ _____ **Datum:** ___/___/___

FUNKTIONSSKALOR

Instruktioner – Var god markera EN siffra för varje skala.

Arbete/Skola

Symptomen har stört Ditt arbete/Ditt skolarbete:

Inte alls	Lite grann			I viss utsträckning			Markant			Oerhört mycket
0	1	2	3	4	5	6	7	8	9	10

Socialt liv

Symptomen har stört Ditt sociala liv:

Inte alls	Lite grann			I viss utsträckning			Markant			Oerhört mycket
0	1	2	3	4	5	6	7	8	9	10

Familjeliv/Ansvar i hemmet

Symptomen har stört Ditt familjeliv/Dina uppgifter i hemmet:

Inte alls	Lite grann			I viss utsträckning			Markant			Oerhört mycket
0	1	2	3	4	5	6	7	8	9	10

FÖRLORADE DAGAR

Hur många dagar under den <u>senaste månaden</u> gjorde symptomen att du missade skolan eller arbetet eller att du inte kunde utföra dina normala dagliga aktiviteter?

☐☐ **dagar**

UNDERPRODUKTIVA DAGAR

Hur många dagar under den <u>senaste månaden</u> kände du dig så nedsatt av dina symptom att din produktivitet minskade, trots att du gick till skolan eller arbetet?

☐☐ **dagar**

ETI-SR-SF

förkortad version

Early Trauma Inventory©

JD Bremner & EA Mayer (J Nerv Ment Dis 2007; 195; 211-218)

ett frågeformulär om svåra upplevelser under uppväxten

Svensk översättning efter tillåtelse av upphovsmännen;
N Hörberg, M Ramklint, M Willebrand, D Fraser, L Ekselius
Institutionen för neurovetenskap, Enheten för Psykiatri, Uppsala Universitet, 2008.

Det händer att människor är med om händelser som kan ses som mycket påfrestande. Upplevde Du något av följande innan Du fyllde 18 år? Ringa in den siffra som bäst motsvarar det antal gånger Du upplevde händelsen före det Du blev 18 år.

1. Var Du med om någon livshotande naturkatastrof? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

2. Var Du med om någon allvarlig olycka? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

3. Blev Du själv någonsin allvarligt skadad? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

4. Var Du med om att någon av dina föräldrar eller den som tog hand om dig blev allvarligt sjuk eller skadad? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

5. Var Du med om att dina föräldrar separerade eller skilde sig? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

6. Var Du med om att något av dina syskon blev allvarligt sjuk eller skadad? Om ja, hur många gånger var Du med om det?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

7. Var Du med om att någon vän blev allvarligt skadad? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

8. Blev Du någonsin vittne till våld mot någon annan, inkluderande familjemedlemmar? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

9. Var någon i Din familj psykiskt sjuk, eller hade någon form av ’nervöst sammanbrott’? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

10. Hade Dina föräldrar, eller den som tog hand om dig, problem med alkohol? Om ja, hur länge?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 år*	*2-3 år*	*4-5 år*	*6-10 år*	*Fler än 10 år*

11. Blev Du någon gång vittne till ett mord eller såg någon som var mördad? Om ja, hur många gånger?

0	**1**	**2**	**3**	**4**	**5**
Aldrig	*1 gång*	*2-3 gånger*	*4-5 gånger*	*6-10 gånger*	*Fler än 10 gånger*

Om Du svarat att Du varit med om något av det som beskrivs i frågorna ovan, fundera på hur stor effekt händelsen eller händelserna kan ha på dig idag.

1. Tror Du att händelsen eller händelserna påverkar dig känslomässigt nuförtiden?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

2. Tror Du att händelsen eller händelserna påverkar din nuvarande funktionsförmåga på jobbet eller i skolan?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

3. Tror Du att händelsen eller händelserna påverkar dina nuvarande familje- och vänskapsrelationer?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

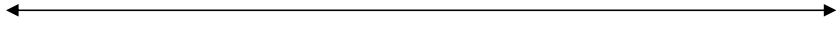
Man kan under uppväxten bli mycket strängt uppfostrad, få mycket smisk eller bestraffas fysiskt eller på något annat sätt. Fundera på om du före 18 års ålder var med om något av följande. För varje händelse du varit med om när du var yngre än 18 år, kryssa i "Ja".

1. Fick Du någon gång en örfil?	❑ **JA** ❑ **NEJ**
2. Blev Du någon gång bränd med varmt vatten, en cigarett eller något annat?	❑ **JA** ❑ **NEJ**
3. Blev Du någon gång sparkad eller slagen?	❑ **JA** ❑ **NEJ**
4. Blev Du någon gång träffad av något som avsiktligt kastades på dig?	❑ **JA** ❑ **NEJ**
5. Blev Du någon gång knuffad?	❑ **JA** ❑ **NEJ**

Om du svarat "JA" på någon av frågorna som handlat om att på olika sätt bli slagen eller fysiskt bestraffad ovan, fundera på hur stor effekt händelsen eller händelserna kan ha på dig idag.

1. Tror Du att händelsen eller händelserna påverkar dig känslomässigt nuförtiden?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**



0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

2. Tror Du att händelsen eller händelserna påverkar din nuvarande funktionsförmåga på jobbet eller i skolan?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

3. Tror Du att händelsen eller händelserna påverkar dina nuvarande familje- och vänskapsrelationer?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

Ibland kan människor under uppväxten känna det som att de inte kan göra någonting rätt i sina föräldrars ögon – deras föräldrar trycker ner dem, skriker alltid åt dem, säger att de inte duger. Kommer Du ihåg om något sådant hände dig när Du var under 18 år? Fundera på om Du upplevt något av följande innan Du fyllde 18. För varje händelse Du varit med om när Du var yngre än 18 år, kryssa i "Ja".

1. Blev Du ofta nedtryckt eller förlöjligad?	❑ **JA** ❑ **NEJ**
2. Blev Du ofta ignorerad, eller fick andra Dig att uppleva att Du inte räknades med?	❑ **JA** ❑ **NEJ**
3. Fick Du ofta höra att Du inte dög?	❑ **JA** ❑ **NEJ**
4. Blev Du oftast behandlad på ett kallt eller icke kärleksfullt sätt?	❑ **JA** ❑ **NEJ**
5. Kände Du ofta att dina föräldrar (eller de du bodde hos) inte förstod sig på dig eller dina behov?	❑ **JA** ❑ **NEJ**

Om du svarat "JA" på någon av frågorna ovan som handlat om att på olika sätt bli känslomässigt försummad eller illa behandlad, fundera på hur stor effekt händelsen eller händelserna kan ha på dig idag.

1. Tror Du att händelsen eller händelserna påverkar dig känslomässigt nuförtiden?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

2. Tror Du att händelsen eller händelserna påverkar din nuvarande funktionsförmåga på jobbet eller i skolan?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

3. Tror Du att händelsen eller händelserna påverkar dina nuvarande familje- och vänskapsrelationer?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**

0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

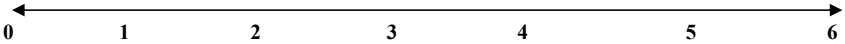
Under uppväxten kan människor göra sexuella erfarenheter som de inte velat göra, eller som fått dem att känna sig obehagliga eller illa till mods. Ibland kan dessa erfarenheter vara med någon de känner och ibland med främmande människor. Minns Du om någonting sådant hände dig när du var under 18 år? Fundera på om Du upplevt något av följande innan Du fyllde 18. För varje händelse Du varit med om när du var yngre än 18 år, kryssa i "Ja".

1. Blev Du någonsin berörd på en intim eller privat del av kroppen (t.ex. bröst, lår, könsorgan) på ett sätt som förvånade dig eller fick dig att känna obehag?	❑ **JA** ❑ **NEJ**
2. Var Du någonsin med om att någon gnuggade sitt könsorgan mot dig?	❑ **JA** ❑ **NEJ**
3. Blev Du någonsin tvingad till att röra vid någon annans intima eller privata kroppsdelar?	❑ **JA** ❑ **NEJ**
4. Hade någon samlag med dig mot din vilja?	❑ **JA** ❑ **NEJ**
5. Blev Du någonsin tvingad att utföra oralsex mot din vilja?	❑ **JA** ❑ **NEJ**
6. Blev du någonsin tvingad att kyssa någon på ett erotiskt snarare än tillgivet sätt?	❑ **JA** ❑ **NEJ**

Om du svarat "JA" på någon av frågorna ovan kring olika sexuella aktiviteter, fundera på hur stor effekt händelsen eller händelserna kan ha på dig idag.

1. Tror Du att händelsen eller händelserna påverkar dig känslomässigt nuförtiden?

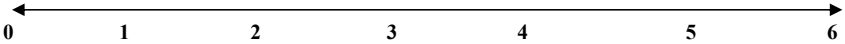
❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**



0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

2. Tror Du att händelsen eller händelserna påverkar din nuvarande funktionsförmåga på jobbet eller i skolan?

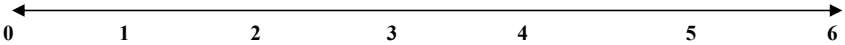
❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**



0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

3. Tror Du att händelsen eller händelserna påverkar dina nuvarande familje- och vänskapsrelationer?

❑ **JA** På vilket sätt? Ringa in ditt svar ❑ **NEJ**



0	**1**	**2**	**3**	**4**	**5**	**6**
extremt negativt	*tämligen negativt*	*något negativt*	*ingen påverkan*	*något positivt*	*tämligen positivt*	*extremt positivt*

Appendix (3)

Spearman correlation matrix between all predictors in Study IV). 2-tailed significances. N = 137.
** Sig < 0.05 level*
*** Sig. < 0.01 level*

		SDS score	Co-morbidity	Any PD diagnosis	No. of childhood traumas	Novelty seeking (NS)	Harm avoidance (HA)	Reward dependence (RD)	Persist-ence (PS)	Self-directed-ness (SD)	Cooperativ e-ness (C)	Self-trans-cendence (ST)	Dis-comfort with closeness (DIS)	Relation-ships as secondary (REL)	Cofidence (CON)	Need for approval (NEE)	Pre-occupation with relations (PRE)
SDS score	**Correlation Coefficient**	1.000	0.108	.292**	0.079	0.017	.452**	-0.083	-.200*	-.537**	-.190*	-0.042	.314**	0.101	-.317**	.326**	.243**
	Sig.		0.209	0.001	0.358	0.842	0.000	0.338	0.019	0.000	0.026	0.624	0.000	0.242	0.000	0.000	0.004
Co-morbidity	**Correlation Coefficient**		1.000	.211*	0.073	0.043	.282**	-0.023	0.055	-.179*	-0.087	0.147	.383**	0.035	-.282**	.195*	.189*
	Sig.			0.013	0.399	0.617	0.001	0.789	0.523	0.037	0.314	0.086	0.000	0.688	0.001	0.022	0.027
Any PD diagnosis	**Correlation Coefficient**			1.000	.251**	0.049	.256**	-0.061	-0.062	-.396**	-.299**	0.086	.244**	.214*	-.234**	.297**	.171*
	Sig.				0.003	0.571	0.003	0.479	0.473	0.000	0.000	0.319	0.004	0.012	0.006	0.000	0.045
No. of childhood traumas	**Correlation Coefficient**				1.000	0.144	0.092	0.033	0.072	-.276**	-.204*	.329**	.220**	0.148	0.002	0.122	.186*
	Sig.					0.093	0.286	0.699	0.402	0.001	0.017	0.000	0.010	0.084	0.978	0.155	0.030
Novelty seeking (NS)	**Correlation Coefficient**					1.000	-0.080	0.122	-.224**	-0.129	-0.059	0.095	0.019	0.004	0.155	0.078	0.066
	Sig.						0.354	0.156	0.009	0.134	0.497	0.268	0.828	0.964	0.071	0.363	0.445

Harm avoidance (HA)	**Correlation Coefficient**						1.000	0.063	-.182*	-.521**	-.188*	0.074	.350**	0.102	-.465**	.514**	.417**
	Sig.							0.469	0.033	0.000	0.028	0.390	0.000	0.237	0.000	0.000	0.000
Reward depen-dence (RD)	**Correlation Coefficient**							1.000	-0.083	0.127	.383**	0.104	-.348**	-.350**	.260**	.240**	.244**
	Sig.								0.337	0.140	0.000	0.228	0.000	0.000	0.002	0.005	0.004
Persist-ence (PS)	**Correlation Coefficient**								1.000	.224**	.183*	0.104	0.130	0.080	-0.006	0.018	-0.012
	Sig.									0.008	0.032	0.226	0.130	0.353	0.940	0.834	0.891
Self-directed-ness (SD)	**Correlation Coefficient**									1.000	.500**	-0.152	-.422**	-.306**	.462**	-.498**	-.456**
	Sig.										0.000	0.075	0.000	0.000	0.000	0.000	0.000
Cooperativ eness (C)	**Correlation Coefficient**										1.000	-0.023	-.186*	-.283**	.169*	-0.004	-0.154
	Sig.											0.788	0.029	0.001	0.048	0.961	0.073
Self-trans-cendence (ST)	**Correlation Coefficient**											1.000	0.142	-0.043	0.065	-0.007	.200*
	Sig.												0.097	0.620	0.451	0.937	0.019
Discomfort with closeness (DIS)	**Correlation Coefficient**												1.000	.296**	-.492**	.342**	.222**
	Sig.													0.000	0.000	0.000	0.009

Relation-ships as secondary (REL)	**Correlation Coefficient**													1.000	-.290**	.191*	0.086
	Sig.														0.001	0.025	0.315
Con-fidence (CON)	**Correlation Coefficient**														1.000	-.453**	-.302**
	Sig.															0.000	0.000
Need for approval (NEE)	**Correlation Coefficient**															1.000	.572**
	Sig.																0.000
Preoccupation with relation-ships (PRE)	**Correlation Coefficient**																1.000
	Sig.																

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