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INTEGRATED STUDY MASTER'S FINAL THESIS

What Does It Mean Recovery From Mental Disorder?

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Abbreviations

ADHD – Attention-Deficit Hyperactivity Disorder

AMY – Amygdala

ASPD – Antisocial Personality Disorder

BPD – Borderline Personality Disorder

CBT – Cognitive Behavioural Therapy

CDC – Centres for Disease Control and Prevention

CHIME – Connectedness, Hope, Identity, Meaning, Empowerment

CSTC – Cortico-Striato-Thalamo-Cortical Circuit

DBT – Dialectical Behaviour Therapy

DSM-5 – Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition

ERP – Exposure and Response Prevention

HPA – Hypothalamic-Pituitary-Adrenal (axis)

ICD-11 – International Classification of Diseases, 11th Revision

MBT – Mentalisation-Based Therapy

MTA – Multimodal Treatment Study of ADHD

NICE – National Institute for Health and Care Excellence

OCD – Obsessive-Compulsive Disorder

OCPD – Obsessive-Compulsive Personality Disorder

PTSD – Post-Traumatic Stress Disorder

SSRI – Selective Serotonin Reuptake Inhibitor

1 Summary

Mental disorders are a major cause of disability and may impair emotional regulation, cognition, interpersonal relationships, occupational functioning and social participation. Although psychiatric treatment has traditionally focused on symptom reduction, contemporary mental health literature increasingly understands recovery as a broader and more individualised process. Recovery may include not only clinical improvement, but also functional reintegration, personal empowerment, identity reconstruction and renewed participation in social life. Therefore, examining recovery through a multidimensional biopsychosocial framework is crucial for understanding psychiatric outcomes beyond remission alone.

The aim of this thesis was to examine the concept of recovery from mental disorders within a multidimensional biopsychosocial framework and to explore how recovery processes manifest across selected psychiatric conditions. The objectives were to review conceptual definitions of recovery in mental health literature, examine recovery mechanisms across selected psychiatric disorders, identify common adaptive processes involved in recovery trajectories, and synthesise a multidimensional framework for understanding recovery.

This thesis was conducted as a narrative literature review. Scientific literature was obtained mainly from PubMed, supplemented by reference lists of selected publications and established psychiatric textbooks when needed. The review focused on attention-deficit hyperactivity disorder, borderline personality disorder, post-traumatic stress disorder, antisocial personality disorder and obsessive-compulsive disorder. Peer-reviewed English-language publications addressing psychological, neurobiological, social and therapeutic aspects of recovery were included. The selected literature was analysed using thematic narrative synthesis.

The results showed that recovery cannot be defined solely as the disappearance of symptoms. Across the disorders examined, recovery involved several overlapping mechanisms, including emotional regulation, cognitive restructuring, behavioural adaptation, development of compensatory strategies, identity reconstruction and social reintegration. The findings also indicated that recovery is diagnosis-sensitive: in some disorders it may involve reduction of compulsive or avoidance behaviours, while in others it may involve long-term executive compensation, improved relational functioning or reduced harmful behaviour. Social support, access to treatment and reduction of stigma were also identified as important factors influencing recovery.

In conclusion, recovery from mental disorder is best understood as a dynamic, individualised and multidimensional process shaped by biological, psychological and social factors. It often reflects adaptive restructuring and improved functioning rather than complete symptom elimination. This perspective supports more integrative and person-centred approaches to psychiatric care.

Santrauka

Psichikos sutrikimai yra viena iš pagrindinių negalios priežasčių ir gali sutrikdyti emocijų reguliaciją, kognityvines funkcijas, tarpasmeninius santykius, profesinį funkcionavimą ir dalyvavimą socialiniame gyvenime. Nors psichiatrinis gydymas tradiciškai buvo orientuotas į simptomų mažinimą, šiuolaikinėje psichikos sveikatos literatūroje sveikimas vis dažniau suprantamas kaip platesnis ir labiau individualizuotas procesas. Sveikimas gali apimti klinikinį pagerėjimą, tačiau taip pat ir funkcinę reintegraciją, asmeninį įgalinimą, tapatybės atkūrimą bei atnaujintą dalyvavimą socialiniame gyvenime. Todėl sveikimo nagrinėjimas taikant daugiamatę biopsichosocialinę sistemą yra svarbus siekiant suprasti psichiatrinius rezultatus neapsiribojant vien remisija.

Šio baigiamojo darbo tikslas buvo išnagrinėti sveikimo nuo psichikos sutrikimų sampratą taikant daugiamatę biopsichosocialinę sistemą ir ištirti, kaip sveikimo procesai pasireiškia pasirinktų psichiatrinių būklių kontekste. Darbo uždaviniai buvo apžvelgti konceptualias sveikimo apibrėžtis psichikos sveikatos literatūroje, išnagrinėti sveikimo mechanizmus pasirinktų psichikos sutrikimų atvejais, nustatyti bendrus adaptyvius procesus, dalyvaujančius sveikimo trajektorijose, ir susisteminti daugiamatę sveikimo supratimo sistemą.

Šis baigiamasis darbas atliktas kaip naratyvinė literatūros apžvalga. Mokslinė literatūra daugiausia buvo renkama iš PubMed duomenų bazės, prireikus papildant ją pasirinktų publikacijų literatūros sąrašais ir pripažintais psichiatrijos vadovėliais. Apžvalgoje daugiausia dėmesio skirta dėmesio trūkumo ir hiperaktyvumo sutrikimui, ribiniam asmenybės sutrikimui, potrauminio streso sutrikimui, antisocialiam asmenybės sutrikimui ir obsesiniam-kompulsiniam sutrikimui. Įtrauktos recenzuojamos publikacijos anglų kalba, nagrinėjančios psichologinius, neurobiologinius, socialinius ir terapinius sveikimo aspektus. Atrinkta literatūra buvo analizuojama taikant teminę naratyvinę sintezę.

Rezultatai parodė, kad sveikimas negali būti apibrėžiamas vien kaip simptomų išnykimas. Visuose nagrinėtuose sutrikimuose sveikimas apėmė kelis persidengiančius mechanizmus, įskaitant emocijų reguliaciją, kognityvinį restruktūrizavimą, elgesio adaptaciją, kompensacinių strategijų vystymą, tapatybės atkūrimą ir socialinę reintegraciją. Gauti duomenys taip pat parodė, kad sveikimas priklauso nuo diagnozės: kai kurių sutrikimų atvejais jis gali apimti kompulsinio ar vengiančio elgesio mažėjimą,

o kitų atvejais – ilgalaikę vykdomųjų funkcijų kompensaciją, geresnį santykių funkcionavimą arba žalingo elgesio sumažėjimą. Socialinė parama, gydymo prieinamumas ir stigmos mažinimas taip pat buvo nustatyti kaip svarbūs sveikimą veikiantys veiksniai.

Apibendrinant galima teigti, kad sveikimas nuo psichikos sutrikimo geriausiai suprantamas kaip dinamiškas, individualizuotas ir daugiamatis procesas, kurį formuoja biologiniai, psichologiniai ir socialiniai veiksniai. Jis dažnai atspindi adaptyvų persitvarkymą ir pagerėjusį funkcionavimą, o ne visišką simptomų išnykimą. Toks požiūris pagrindžia labiau integruotus ir į asmenį orientuotus psichiatrinės pagalbos metodus.

2 Keywords

Mental health recovery, psychiatric recovery, biopsychosocial recovery, psychotherapy mechanisms, emotional regulation in psychiatric disorders, multidimensional framework of recovery.

3 Introduction

In psychiatry, the meaning of improvement has changed considerably over time. Traditionally, treatment success was often understood mainly through symptom reduction, diagnostic remission or prevention of relapse. These outcomes remain clinically important, especially when symptoms cause distress, risk or functional impairment. However, they do not fully describe what recovery means in the life of a person affected by a mental disorder.

Many individuals continue to experience certain symptoms or vulnerabilities while gradually rebuilding stable daily functioning, relationships, autonomy and a sense of identity. Others may no longer fulfil diagnostic criteria but still struggle with occupational participation, social reintegration, emotional regulation or self-confidence. This distinction shows that recovery cannot be reduced to the absence of symptoms alone. It must also include the way individuals adapt psychologically, regain functioning and reconstruct meaningful participation in life.

This broader understanding is relevant for both clinical practice and society. Mental disorders affect not only internal emotional and cognitive processes, but also education, work, relationships, family life and social inclusion. Therefore, recovery-oriented psychiatry requires attention to biological, psychological and social factors that influence long-term outcomes. Such a perspective is especially important because different psychiatric disorders may have different recovery trajectories, and complete symptom elimination is not always realistic or necessary for meaningful improvement.

The present thesis focuses on recovery across selected psychiatric conditions: attention-deficit hyperactivity disorder, borderline personality disorder, post-traumatic stress disorder, antisocial personality disorder and obsessive-compulsive disorder. These disorders differ in their diagnostic features, mechanisms and treatment approaches. Nevertheless, they also demonstrate shared recovery-related processes, including improved emotional regulation, cognitive restructuring, behavioural adaptation, compensatory strategies, identity reconstruction and social reintegration.

The relevance and novelty of this thesis lie in its attempt to integrate disorder-specific and transdiagnostic perspectives on recovery across several psychiatric conditions. Instead of viewing recovery only as a clinical endpoint or symptom remission, this thesis approaches recovery as a dynamic biopsychosocial process involving several interrelated domains: clinical recovery, functional recovery, personal recovery and social recovery. Clinical recovery refers to symptom reduction and relapse prevention; functional recovery concerns restoration of academic, occupational and interpersonal functioning; personal recovery involves empowerment, identity reconstruction, hope and meaning; and social recovery includes social reintegration, connectedness, reduction of stigma and restoration of supportive relationships. This multidimensional approach allows recovery to be understood not only as a medical outcome, but also as a developmental, psychological and social process.

3.1 Aim of the Thesis

The aim of this thesis is to evaluate the concept of recovery from mental disorders within a multidimensional biopsychosocial framework and to explore how recovery processes manifest across selected psychiatric conditions.

3.2 The Objectives

1. To evaluate the conceptual definitions of recovery in mental health literature
2. To find common denominators of recovery mechanisms across selected psychiatric disorders
3. To establish common adaptive and maladaptive processes involved in recovery trajectories
4. To synthesise a multidimensional framework for understanding recovery

4 Methodology

4.1 Literature Search Strategy and Methodology

This thesis was conducted as a narrative literature review focusing on the theoretical frameworks, neurobiological mechanisms and clinical recovery processes in attention deficit hyperactivity disorder,

borderline personality disorder, post-traumatic stress disorder, antisocial personality disorder, and obsessive-compulsive disorder.

4.2 Data Sources

Scientific literature was obtained primarily from PubMed, which served as the main database for identifying relevant peer-reviewed articles. Additional sources including reference lists of selected publications and established psychiatric textbooks were used when it was necessary to clarify theoretical concepts.

4.3 Search Strategy

Literature searches were conducted using a combination of keywords related to mental health recovery and specific disorders examined in this thesis: Mental health recovery, biopsychosocial recovery, attention deficit hyperactivity disorder, borderline personality disorder, post-traumatic stress disorder, antisocial personality disorder, obsessive compulsive disorder, psychotherapy mechanisms, emotional regulation in psychiatric disorders.

Search filters were applied as appropriate to find peer reviewed articles and several well-known English-language publications on the subject. Preference was given to recent literature published between 2016-2026, while seminal earlier publications were included when necessary to provide theoretical foundations.

4.4 Inclusion and Exclusion Criteria

Inclusion Criteria:

1. Peer reviewed scientific articles
2. Studies addressing recovery in psychiatric disorders
3. Research discussing psychological, neurobiological or social mechanisms of recovery
4. Studies focusing on recovery from ADHD, BPD, PTSD, ASPD or CPD

Exclusion Criteria:

1. Articles not available in English
2. Publications lacking scientific or clinical relevance to psychiatric recovery
3. Data studies focused exclusively on disorders outside the scope of this thesis
4. Non-scientific sources without academic review

4.5 Data Selection and Organisation

Relevant articles were identified through database searches and screening based on titles and abstracts to assess their relevance to the research topic. Publications that appeared relevant were then reviewed in full text. Following thorough screening, 61 publications were selected from the initial 657 articles considered. These sources were deemed most relevant because they directly addressed the topic of the thesis.

The selected literature was organised according to major thematic domains, including the conceptual definition of recovery, neurobiological mechanisms, psychological processes, therapeutic interventions and disorder-specific recovery trajectories.

4.6 Type of Studies Included

The literature reviewed in this thesis included a range of studies such as:

1. Systemic reviews and narrative reviews
2. Observational and longitudinal studies
3. Neuroimaging studies
4. Clinical outcome studies
5. Theoretical and conceptual psychiatric literature

Including multiple study types allowed for a comprehensive understanding of recovery processes across different methodological approaches in psychiatric research. Also, it prioritized evidence levels, starting from systematic reviews and meta-analyses through longitudinal and observational studies, and finally analysing clinical outcome, neuroimaging studies and theoretical reviews (56).

4.7 Data Analysis

The selected literature was analysed using a thematic narrative synthesis approach. Articles were reviewed and key concepts related to recovery mechanisms, therapeutic processes and functional outcomes were extracted. Notes were taken during the review process to identify reoccurring themes in conceptual patterns across different psychiatric disorder.

The findings were then organised into thematic sections that examine both shared mechanisms of recovery across disorders and disorder specific recovery trajectories. This approach allowed the literature to be interpreted within a broader biopsychosocial framework of mental health recovery.

5 Literature Review

5.1 Understanding Mental Health

5.1.1 Definition of Mental Health

Mental health can be understood as a multidimensional state of psychological well-being that enables individuals to regulate emotions, think coherently, maintain relationships, cope with stress and function effectively in daily life. Within the psychiatric framework, disturbances in core mental functions such as thinking, emotion, attention, cognition, perception and memory may give rise to persistent and clinically significant patterns of thought, emotional experiences and behaviours that fulfil the diagnostic criteria for a mental disorder (1,28).

Mental health therefore represents more than the absence of psychiatric symptoms. It also includes the capacity to cope with stress, maintain social participation and fulfil a functional role within society. Difficulties in emotional regulation, cognitive processing or interpersonal functioning may contribute to the development of psychiatric conditions such as mood disorders, anxiety disorders, personality disorders and neurodevelopmental disorders (1,10)

Understanding mental health as a multidimensional concept is important because psychiatric disorders often affect several domains of functioning simultaneously, including emotional stability, cognitive performance, relationships and social participation (1,10)

5.1.2 Global Burden of Mental Disorders.

Mental disorders contribute substantially to the global burden of disease, particularly through years lived with disability (YLD), highlighting their major public health impact (10). Despite the availability of effective treatments, many individuals with mental disorders do not receive adequate care. Structural barriers such as limited access to services, financial constraints, shortage of mental health professionals and stigma contribute to a large treatment gap across many countries (10).

These disparities emphasise the importance of early identification, accessible treatment and integrated mental health systems that address both clinical symptoms and social determinants of mental health (10).

5.1.3 Developmental and Attachment Foundations

Psychological development begins early on in life, strongly influenced by caregiver relationships and environmental stability. Attachment theory, developed by John Bowlby, proposes that the early

interactions with caregivers shape internal working models of the self and others, influencing emotional regulation, interpersonal expectation and stress response throughout the lifespan (9).

These developmental experiences therefore play an important role in shaping psychological resilience and vulnerability to later mental health disturbances, particularly when early relational stress occurs during sensitive periods of development (9,59).

5.1.4 Trauma and Psychological Vulnerabilities

Exposure to trauma and chronic stress can significantly increase vulnerability to psychiatric disorders. Traumatic experiences may disrupt emotional regulation systems and alter cognitive appraisal processes, contributing to persistent fear responses, avoidance behaviour and maladaptive coping strategies (16,17).

Neuroscientific research suggests severe or repeated stress can affect neural circuits involved in threat detection, emotional regulation and memory processing, including in the amygdala, hippocampus and prefrontal cortex (16).

These neurobiological and psychological changes may contribute to the development of disorders such as post-traumatic stress disorders and other trauma related conditions (16,17).
Understanding the impact of trauma provides important insight into the developmental pathways through which mental health disturbances may emerge.

5.1.5 Trauma and Biological Vulnerability

Pre-existing biological factors may shape how traumatic experiences are processed and help explain why some individuals develop persistent psychiatric symptoms after trauma while others may not. Individual differences in stress response systems, emotional reactivity and neural regulation may influence threat detection, memory processing and emotional control following trauma. Variation in the functioning of the amygdala, hippocampus, prefrontal cortex and HPA axis may increase vulnerability to trauma-related disorders (16,17).

Thus, the effects of trauma are not determined by the external event alone, but also by the biological characteristics of the individual exposed to it.

5.1.6 Trauma and Social Vulnerability

Trauma is also shaped by social and environmental contexts. Interpersonal violence, neglect, family instability, stigma, social isolation and lack of supportive relationships may intensify the psychological

impact of traumatic experiences and reduce opportunities for adaptive recovery (9,10,17). On the other hand, safe relationships, social support and access to appropriate care may buffer the long-term effects of trauma and promote emotional stabilisation, resilience and functional reintegration (3,10,17).

5.1.7 Evidence-Based Therapeutic Interventions

Several evidence-based psychotherapeutic interventions have demonstrated effectiveness in treating psychiatric disorders. Cognitive behavioural therapy (CBT) is among the most extensively studied psychological treatments and has demonstrated strong efficacy across mood disorders, anxiety disorders and OCD (5,6). CBT focuses on identifying maladaptive cognitive patterns and modifying behavioural responses that maintain psychological distress.

Neuroimaging research indicates that successful cognitive behavioural interventions are associated with increased activation in prefrontal regulatory regions and improved modulation of limbic emotional circuits involved in emotional processing (4,8).

Dialectical behaviour therapy (DBT) represents another evidence-based intervention, particularly for individuals with BPD and severe emotional dysregulation. Clinical studies demonstrate significant reduction in self-harm behaviour, impulsivity and emotional instability following structured DBT treatment (7,8,35). Trauma-focused therapies have also demonstrated strong efficacy in individuals with PTSD by facilitating emotional and structured processing of traumatic memories (16,17).

Social interventions are essential components of psychiatric treatment. These include psychoeducation, family-based intervention, community mental support and strategies aimed at reducing stigma and improving access to care. Such interventions may strengthen adherence to treatment, improve social functioning, enhance support systems and facilitate long-term recovery as well as reintegration into daily life (3,10,61).

In addition to psychological and social interventions, biological treatments play an important role in psychiatric care. Pharmacological treatment may support symptom stabilisation in specific disorders, for example stimulant treatment in ADHD and serotonergic treatment approaches in OCD, by influencing neurochemical systems involved in attention, inhibitory control and symptom regulation (20,21,22,23).

5.1.8 Biological Factors in Mental Health

Biological factors contribute significantly to mental health and include dysregulation of neurotransmitter systems, altered neural circuitry and neurodevelopmental vulnerabilities. Serotonin,

dopamine and norepinephrine are involved in emotional regulation, reward processing, executive functioning and behavioural control, which represent core domains affected across several of the psychiatric disorders examined in this thesis, particularly ADHD and OCD (13,20,21,22,23).

Pharmacological treatments act in part through modulation of these neurochemical systems and may improve symptom regulation by influencing both synaptic transmission and broader brain network activity (20,21,22,23). However, contemporary evidence suggests that recovery-related biological change is not limited to acute neurochemical effects. Therapeutic intervention may also be associated with broader neural adaptive processes including changes in functional connectivity within prefrontal-limbic circuits involved in emotional regulation and cognitive control (4,8).

From this perspective, biological factors in mental health are not restricted to static abnormalities but include dynamic regulatory systems that may be modified through treatment and adaptation. This view supports a transdiagnostic model in which recovery is associated with improved regulation of shared neural systems, rather than disorder-specific mechanisms exclusively (49,50,54).

5.1.9 Conceptual Transition Toward Recovery

While clinical symptom reduction remains an important treatment goal, contemporary psychiatric models increasingly recognise that improvement in mental health extends beyond clinical remission.

Functional recovery involves restoration of social participation, occupational functioning and relational stability, while personal recovery emphasises rebuilding identity, hope and psychological agency following illness (3,40,61).

Pharmacological treatments may contribute to symptom stabilisation and functional improvement in specific psychiatric disorders by modulating neurochemical systems and neural circuits involved in emotional regulation, executive functioning and behavioural control. In the disorders examined in this thesis, this is particularly relevant to ADHD, where catecholaminergic systems are involved in attention and executive control, and to OCD, where serotonergic modulation and cortico-striato-thalamo-cortical circuit dysfunction are implicated in symptom regulation (20,21,22,23). These disorder-specific biological mechanisms also support the broader transdiagnostic view that recovery may involve improved regulation of shared neural and behavioural systems rather than isolated symptom suppression alone (49,50,54).

These broader recovery-oriented perspectives emphasised that effective mental health treatments should address biological, psychological and social dimensions of functioning rather than focusing exclusively on symptom reduction.

The following sections examine recovery as a multidimensional process that unfolds across clinical, functional, personal and social domains.

5.2 Understanding Recovery from Mental Disorder.

5.2.1 From Symptom Reduction to Recovery as a Process

Historically, improvement in mental health treatments was defined primarily in terms of symptom reduction. Within traditional psychiatric models, recovery was often equated with remission, that is, the reduction of symptoms below the diagnostic threshold as defined in the DSM-5 (1). This definition remains clinically important, especially in disorders in which symptom severity directly impairs safety or daily functioning. However, clinical remission alone does not necessarily indicate full recovery. An individual may no longer meet the diagnostic criteria while still experiencing impaired social functioning, unstable identity or difficulty with emotional regulation. Longitudinal outcome studies support this distinction, showing that some individuals achieved symptom reduction without regaining full occupational or interpersonal functioning, whereas others continued to experience mild residual symptoms while functioning effectively in daily life (3,40,61).

Recovery-oriented and longitudinal literature suggests that recovery may follow a nonlinear course, characterised by periods of improvement, stabilisation, partial relapse, recurrence or renewed adaptation under stress (3,33,60,61). In some cases, apparent remission may partially reflect masking or compensatory functioning rather than full resolution of the underlying vulnerability. Because many psychiatric conditions have a chronic or reoccurring dimension, stressful life events may trigger renewed symptom expression even after a period of stabilisation. Although psychotherapy can provide individuals with tools to manage these difficulties more effectively, the acquisition and consistent application of such strategies often take time, reinforcing the view that recovery is a gradual and long-term adaptive process rather than a fixed endpoint (3,60,61).

These findings suggest that recovery cannot be reduced to symptom elimination alone, instead, it should be understood as a broader adaptive process involving clinical stabilisation, functional integration and psychological restructuring (3,40,61).

5.2.2 Dimensions of Recovery

Modern recovery frameworks recognise that improvement in mental health occurs across multiple domains**.** Within this thesis, the multidimensional understanding of recovery can be explained in part by the CHIME framework, proposed by Professor Mike Slade et al., particularly in relation to personal and social recovery while also extending beyond it to include clinical and functional dimensions (3).

5.2.2.1 The CHIME Framework of Personal Recovery

A major conceptual contribution to recovery literature, the CHIME framework identifies 5 key processes of personal recovery: connectedness, hope, identity, meaning and empowerment (3). This model shifted attention away from symptom reduction alone and towards the lived experience of recovery as a personal, relational and socially embedded process. Within this framework, recovery is understood not merely as the absence of psychiatric symptoms, but as the rebuilding of a meaningful life despite ongoing vulnerability (3).

In the present thesis, the CHIME framework is used primarily to help explain the personal and social dimensions of recovery. Hope, identity, meaning and empowerment are closely related to personal recovery, while connectedness is especially relevant to social recovery. At the same time, the broader framework adopted in this thesis also includes clinical and functional recovery, extending beyond the CHIME model alone (3,40).

5.2.2.2 Clinical Recovery

Clinical recovery refers to measurable reduction in symptom severity and improvement in clinically observable manifestations of mental disorder. It is typically evaluated through standardized diagnostic criteria, structured clinical assessment and symptom rating scales (1). In long-term recovery, reduced relapse frequency may also reflect improved clinical stability and relapse-prevention capacity (60). Clinical recovery remains an important component of psychiatric treatment, particularly in disorders where symptom severity directly compromises safety or daily functioning. However, clinical improvement alone does not necessarily indicate full recovery, as symptom remission may occur without restoration of interpersonal stability, identity coherence, or functional independence (3,33,40).

5.2.2.3 Functional Recovery

Functional recovery refers to the restoration of the individual's ability to participate effectively in everyday life, including academic, occupational, social and interpersonal domains, as well as the capacity to manage responsibilities, maintain routines and sustain a degree of independent living.

Unlike clinical recovery, which focuses primarily on symptom change, functional recovery is concerned with how the individual performs in real-life contexts. This distinction is important because recovery-oriented literature emphasises that meaningful improvement may involve restored functioning, autonomy and participation even when some vulnerability or residual symptoms remain (3,40,61).

5.2.2.4 Personal Recovery

Personal recovery refers to the internal psychological transformation that occurs during the healing process. It involves rebuilding self-confidence, restoring hope, restructuring identity and developing a coherent narrative that integrates the experience of illness without allowing it to define the self entirely.

Personal recovery is closely linked to empowerment, self-awareness and the ability to tolerate vulnerability without psychological collapse. It represents the shift from being controlled by symptoms to actively managing them (3).

The CHIME Framework (connectedness, hope, identity, meaning, empowerment) provides a structured model for understanding this dimension (3). Recovery in this sense does not require complete absence of symptoms but reflects increased agency and resilience.
Recovery focuses on personal empowerment, hope, and autonomy rather than just symptom elimination.

Together, these dimensions highlight that recovery operates simultaneously at the biological psychological and social levels.

5.2.2.5 Social Recovery

Social recovery refers to the process of reintegration into society including restoration of social roles, reduction of stigma, rebuilding of trust, and participation in community life. While functional recovery focuses on individual performance in work or education, social recovery emphasises social inclusion and the rebuilding of relational networks (3).

In relation to the CHIME framework, this dimension corresponds most closely to connectedness, which refers to having a supportive relationship, gaining a sense of belonging and participating in meaningful social life (3).

Mental disorders frequently result in social withdrawal, interpersonal rupture and structural exclusion. Therefore, recovery cannot be considered complete without addressing this social context in which the

individual lives. Recovery includes rebuilding supportive relationships, reducing both self-stigma and public stigma, accessing community resources and restoring participation in the community. (3,10).

Thus, recovery is not solely an internal psychological process but also a relational and structural one.

5.2.3 Recovery is an Adaptive Developmental Process

Mental disorders frequently disrupt core regulatory systems, including emotional modulation, cognitive appraisal, behavioural flexibility and relational security. Recovery, therefore, can be conceptualised as the gradual restructuring of these disrupted systems (3,5,9,49,50,54).

Across diagnostic categories, several adaptive processes consistently emerge: strengthening emotional regulation, increasing cognitive flexibility, reducing avoidance-based behaviour, rebuilding attachment security and restoring identity coherence. Although the expression of these processes may differ between disorders and individuals, the underlying adaptive mechanisms share similarities (3,5,9,49,50,54).

For example, exposure-based interventions reduce conditioned fear and avoidance responses, trauma-focused therapies support emotional processing and integration of traumatic memories, and executive-functioning strategies in neurodevelopmental disorders promote structured behavioural control. In each case, recovery reflects improved regulation and adaptive functioning rather than simple symptom disappearance (6,16,17,21,23).

These processes are supported by therapeutic intervention and neuroplastic adaptation. Research demonstrates that effective psychotherapy is associated with measurable functional changes in neural circuits involved in executive control and emotional modulation (4,8).

This perspective allows recovery to be understood as a learning process. Just as developmental growth requires repeated exposure, error correction and environmental support, psychological recovery involves adaptation shaped by therapeutic intervention and social context (3,59,61).

5.2.4 Cognitive and Behavioural Mechanisms of Recovery

The cognitive behavioural model proposes that psychiatric disorders are maintained by maladaptive beliefs, distorted threat appraisal and reinforced avoidance behaviour (5). Recovery within these frameworks involves restructuring functional cognitive schemas and modifying maladaptive behavioural responses.

Cognitive behavioural therapy (CBT) has consistently demonstrated efficacy in reducing symptoms across anxiety, depression and OCD by targeting distorted thinking and avoidance-maintained fear cycles (6).

Neuroimaging research further supports this model demonstrating that successful cognitive behavioural interventions are associated with increased activation in prefrontal regulatory regions and decreased limbic hyperactivity (4).

Therefore, cognitive restructuring and behavioural re-engagement represent foundational mechanisms of recovery across multiple psychiatric conditions.

5.2.5 Emotional Regulation and Attachment in the Recovery Process

Emotional dysregulation is a core feature across multiple psychiatric conditions and may function as a transdiagnostic mechanism linking symptoms across diagnostic categories (50,51). Individuals who struggle to tolerate distress or modulate intense affect often develop compensatory behaviours such as avoidance, compulsivity, impulsivity or substance use (52,53).

Recovery frequently requires strengthening emotional regulation capacities. Therapeutic approaches such as dialectical behaviour therapy (DBT) emphasise stress tolerance, emotional labelling and impulsivity control. DBT has shown effectiveness in improving emotional regulation and reducing maladaptive behaviour, particularly in BPD (7). Neurobiological studies suggest such intervention enhanced top-down emotional control mechanisms (8).

Attachment theory further expands the understanding of recovery. Early caregiver relationship experiences shape internal working models of self and other (9). Insecure or disorganised attachment patterns may predispose individuals to difficulties in emotional regulation and interpersonal trust. Recovery, therefore, may involve the development of relational security and corrective interpersonal experiences within therapeutic and social relationships (3,9).

In this sense, recovery is not solely intrapsychic but relational. A safe interpersonal environment supports neural and psychological restructuring.

5.2.6 Developmental and Contextual Determinants of Recovery

5.2.6.1 Developmental Timing

Developmental timing may influence recovery because the effects of stress, vulnerability and intervention differ across the lifespan. Developmental neuroscience suggests that sensitive periods of

neural development interact with exposure to stress, shaping later emotional regulation, threat processing, resilience and neuroplasticity (59). Early life stress may therefore have a stronger impact when it occurs during periods of heightened developmental plasticity, particularly in childhood and adolescence. At the same time, these developmental windows may also create opportunities for early intervention, because adaptive support during periods of increased plasticity may promote more favourable recovery trajectories (59).

Importantly, developmental timing may also influence the formation of maladaptive mechanisms. When stress, trauma, invalidation or chronic instability occur during sensitive developmental periods, the individual may develop coping patterns that are initially protective or functional in the original environment but later become maladaptive. Examples include avoidance, emotional suppression, hypervigilance, impulsive reactions, compulsive control or mistrust of others. These patterns may reduce distress in the short term, but over time they can interfere with emotional regulation, relational security and flexible functioning. Therefore, recovery may require not only symptom reduction, but also the modification of maladaptive developmental adaptations into more flexible and functional coping strategies (59).

When psychiatric symptoms emerge early in life, recovery may involve not only symptom reduction, but also the development of adaptive coping strategies, self-understanding and functional compensation. This is particularly relevant in neurodevelopmental disorders such as ADHD, where symptoms may persist into adulthood while functioning may improve through compensatory strategies and environmental support (13,23,24). In contrast, later-onset disorders may destabilise previously established patterns of functioning, but recovery may be supported by pre-existing coping resources, identity structure and social roles. Therefore, developmental timing influences both vulnerability to mental disorder and the pathways through which recovery may occur.

5.2.6.2 Environmental and Social Context

Recovery does not occur in isolation. Social support, socio-economic stability, stigma exposure, educational opportunities and access to evidence-based treatment significantly influence outcome trajectories (10).

Family accommodation patterns, cultural beliefs about mental illness and structural barriers to care may either reinforce maladaptive behaviour or support adaptive restructuring, directly affecting the recovery process (10,15,61). Early intervention, psychoeducation, community-based support and recovery-

oriented services may improve treatment engagement, social functioning, functional participation and the likelihood of sustaining recovery-oriented gains (10,61).

5.2.6.3 Recovery Trajectories and Relapse

Recovery from a mental disorder rarely follows a straight line. Recovery-oriented and longitudinal literature suggests that many psychiatric conditions may involve fluctuating trajectories characterised by periods of improvement, stabilisation, partial relapse and renewed adaptation (3,33,60,61). Relapse should therefore not automatically be understood as failure, but rather as a possible part of a long-term recovery process.

Relapse prevention literature emphasises that relapse is often gradual and may begin before the return of full clinical symptoms. Early warning signs, emotional dysregulation, avoidance, reduced self-care and weakening of coping strategies may precede clinical deterioration (60). From this perspective, recovery involves learning to recognise early warning signs, understand triggers, strengthen coping strategies and seek support before symptoms become more severe (60).

This perspective aligns with a chronic-illness model of recovery, in which stabilisation, adaptation and relapse prevention are prioritised over the expectation of absolute cure. What changes over time is not only symptom intensity, but also the individual's relationship to symptoms: the ability to identify risk states earlier, regulate emotional responses more effectively and re-engage with treatment or support when necessary (3,60,61).

5.2.6.4 Chronic Conditions and Adaptive Compensation

In certain psychiatric conditions, particularly neurodevelopmental disorders and some personality disorders, complete symptom elimination may not be realistic or necessary as the only marker of recovery. Instead, recovery may involve adaptive compensation, restructuring and improved functional management of persistent vulnerabilities (3,13,33,34,61).

Long-term ADHD research suggests that although symptom intensity often declines across development, impairments and vulnerabilities may persist into adolescence and adulthood. Many individuals develop adaptive compensatory strategies such as self-awareness, structured routines, organisational systems, environmental modification and behavioural strategies that can improve functional outcomes (13,23,24).

Similarly, in personality disorders, especially borderline personality disorder, recovery may reflect improvement in emotional regulation, relational stability, identity integration and reduced impulsive

behaviour rather than complete disappearance of temperamental or developmental vulnerabilities (33,34,37). In this case, recovery reflects increased autonomy and functional integration. The person becomes more capable of managing vulnerabilities rather than being controlled by them.

At the same time, compensation is not always adaptive. In some cases, chronic illness may also be associated with maladaptive forms of adjustment, such as rigid identification with the sick role or defensive reliance on grandiose or compensatory self-structures (3,45,46). Although such processes may reduce distress temporarily or preserve psychological stability, they do not necessarily reflect genuine recovery because they may limit flexibility, responsibility and deeper psychological integration.

This reinforces the idea that recovery does not always equal cure. Sometimes it equals adaptation, although the quality of that adaptation may differ significantly.

5.2.6.5 Environmental Scaffolding and Social Reintegration

The social environment plays a significant role in shaping recovery outcomes. Family accommodation patterns, stigma, access to therapy, financial stability and cultural attitudes towards mental illness may either reinforce maladaptive behaviour or facilitate adaptive change (10,15,61). In disorders such as OCD, family members who repeatedly provide reassurance or adjust routines around rituals may unintentionally maintain symptoms (15). Conversely, families who receive psychoeducation and establish supportive boundaries may enhance treatment engagement and facilitate long-term recovery (15,61).

Access to early and evidence-based interventions, community-based support and recovery-oriented services is associated with improved social reintegration, treatment engagement, functional stability and recovery-oriented outcomes (10,61).

Scaffolding in mental health recovery can be conceptualised as a structured, collaborative and time-limited support process that enables the individual to progressively develop skills, enhance stability and move towards greater autonomy. Like physical scaffolding in construction, this approach provides tailored and adaptive support that facilitates emotional regulation, engagement in daily life activities and restoration of meaningful social roles before internal regulation becomes stronger. Over time, coping mechanisms and emotional regulation may improve, and the individual may become less dependent on external scaffolding (3,61).

Recovery can therefore be understood as both an individual and socially embedded process shaped by family relationships, access to care, community support and broader structural conditions (3,10,61).

5.2.6.6 Identity Reconstruction and Meaning

One of the most profound aspects of recovery involves the reconstruction of identity. Mental disorders disrupt self-concept, produce shame, guilt, perceived weakness, and internalisation of stigma (3,10).

Personal recovery models emphasise the integration of distressing experiences into coherent life narratives (3). In trauma-related disorders, narrative restructuring allows individuals to regain agency and reinterpret past events without being dominated by them (17).

Identity reconstruction involves shifting from a passive illness-centred identity to an adaptive self-concept characterised by agency, resilience and self-efficacy. This transformation does not eliminate vulnerability but allows each individual to relate to it differently (3,61)

5.2.7 Integrative Definition of Recovery in this Thesis

Based on the theoretical and empirical literature reviewed, this thesis adopts an integrative biopsychosocial definition of recovery. Recovery is conceptualised as a dynamic and individualised process involving symptom reduction or stabilisation, restoration of functional capacity, cognitive and emotional restructuring, social reintegration and reconstruction of a coherent and empowered identity. This definition recognises that recovery is not limited to clinical remission, but also includes the person's ability to regain agency, adapt to vulnerability, improve quality of life and participate meaningfully in daily life (1,3,40,61).

Within this thesis, recovery is understood across four interrelated domains: clinical, functional, personal and social. Clinical recovery refers to the reduction or stabilisation of symptoms; functional recovery refers to restored participation in academic, occupational and interpersonal roles; personal recovery involves hope, meaning, empowerment, self-awareness and identity reconstruction; and social recovery includes connectedness, reduction of stigma, supportive relationships and reintegration into community life (3,10,40,61). Recovery-oriented services are therefore important because they address not only symptom control, but also social functioning, autonomy, community participation and quality of life among people living with severe mental illness (61).

This integrative definition allows recovery to be understood as both diagnosis-sensitive and transdiagnostic. Different disorders may require different recovery pathways, but the reviewed literature suggests that several adaptive processes recur across psychiatric conditions, including

improved emotional regulation, cognitive flexibility, behavioural re-engagement, relational security, self-awareness and social participation (49,50,54). Therefore, recovery in this thesis is understood not as complete elimination of all vulnerability, but as the gradual strengthening of adaptive biological, psychological and social functioning.

5.2.8 Concept and Measurement Limitation of Recovery Models

Despite the growing emphasis on recovery-oriented care, defining and measuring recovery remains challenging. Clinical remission can be measured using structured tools and diagnostic criteria. But elements such as hope, identity reconstruction, empowerment and resilience are more subjective and difficult to quantify (3). Different studies use different operational definitions which makes comparison between the findings complex.

Furthermore, neuroimaging studies suggest that psychotherapy may be associated with changes in brain function within circuits involved in emotional regulation, including prefrontal-limbic systems (4,8). These findings support the possibility of adaptive neural change, but they should be interpreted cautiously because causality between neural changes and subjective recovery cannot yet be definitively established.

Cultural context also shapes how recovery is understood. Concepts of autonomy, productivity and social integration may be different across societies, influencing both treatment goals and their perceived outcomes (10).

Recognising this limitation does not invalidate recovery models. Instead, it emphasises the need for flexible multi-dimensional recovery models that integrate clinical, psychological and social dimensions (3,61).

5.2.9 Methodological Implications

The conceptualisation of recovery outlined above directly shaped the methodological design of this thesis. Because recovery was understood as a multidimensional process extending beyond symptom remission, the literature review was structured to examine emotional regulation, cognitive restructuring, identity development, relational functioning and social reintegration across psychiatric disorders.

The selected publications were analysed to identify both disorder specific recovery patterns and transdiagnostic mechanisms that contribute to adaptive functioning. This thematic synthesis allowed the preservation of diagnostic differences while highlighting shared structural processes involved in recovery trajectory.

By aligning the literature analysis with a multidimensional recovery framework, the study ensures coherence between the theoretical foundation of recovery, the research objectives and the interpretation of the reviewed evidence.

5.3 Selected Disorders and Diagnostic-Specific Recovery

5.3.1 Attention Deficit Hyperactivity Disorder (ADHD)

5.3.1.1 Definition and Epidemiology

ADHD is one of the most common neurodevelopmental disorders of childhood and adolescence. According to epidemiological data from the CDC roughly 1 in 9 US children have ever received an ADHD diagnosis (11.4% or 7.1 million children) and 10.5% (6.5 million) had current ADHD showing an increase vs. previous years (18).

ADHD is not simply a behavioural problem. It is a neurodevelopmental condition with a strong genetic contribution and measurable neurobiological correlates. Twin and family studies demonstrate significant heritability (20). Neuroimaging research constantly identifies differences in frontostriatal circuits, including reduced activation and structural variations in the prefrontal cortex, caudate nucleus and cerebellum. These areas are responsible for executive control, attention regulation and impulse inhibition (13,20). Dysregulation of catecholaminergic pathways, particularly the dopamine and norepinephrine systems, further contributes to impaired executive function and reward processing (13,20).

However, neurobiology alone does not explain the full clinical picture. Environmental factors such as prenatal or early-life exposure to toxicants, low birth weight, prematurity, psychosocial stress and early instability may interact with genetic vulnerability and influence symptom severity and long-term outcome (13).

5.3.1.2 Clinical Presentation and Functional Impact

According to DSM-5 criteria, ADHD is defined by persistent patterns of inattention and/or hyperactivity and impulsivity, lasting at least six months, causing clinically significant impairment in social academic or occupational functioning (1).

Inattention in ADHD includes difficulty sustaining attention, forgetfulness in daily activity, poor organisational skill, and distractibility, while hyperactivity and impulsivity include excessive motor activity, difficulty remaining seated, causing social interruption and impulsive decision making.

Importantly these behaviours are not explained by defiance, lack of intellect, or unwillingness to cooperate. They reflect impaired executive regulation (23).

It is important to note that ADHD does not present uniformly across sex or developmental contexts. Predominantly inattentive presentations may be less behaviourally disruptive and may include features such as daydreaming, “spacing out,” internal distractibility, passivity or academic inconsistency. Increased awareness of attention-regulation symptoms has contributed to greater recognition of ADHD in girls, adolescents and adults, while boys remain more likely to receive a diagnosis, possibly because overt hyperactive behaviours are more easily observed (18,19,27). This variability highlights the necessity of nuanced clinical assessment beyond overt hyperactivity, and it reinforces the fact that ADHD is fundamentally a disorder of executive regulation rather than wilful misconduct.

ADHD is classified into three clinical presentations:

1. Predominantly inattentive presentation
2. Predominantly hyperactive/impulsive presentation
3. Combined presentation

These presentations are not fixed categories; they may shift during different development stages depending on environmental demands and maturation (13,23).

5.3.1.3 Developmental Course and Lifespan Perspective

Longitudinal research shows that ADHD symptoms often change rather than disappear with age (13). Although symptom levels and impairment may improve over time, long-term follow-up data show that normalization is often not achieved, and individuals with childhood ADHD may continue to show poorer academic, behavioural and social functioning than comparison peers (24). In the MTA 8-year follow-up, only 30% of participants fulfilled DSM-IV criteria for ADHD, although this may underestimate persistence because child-based symptom thresholds were used for adolescents and adults (24).

The developmental variability reinforces that ADHD is not a static childhood disorder, but a condition interacting continuously with environmental expectations and cognitive maturation.

5.3.1.4 Differential Diagnosis

ADHD must be differentiated from several psychiatric and medical conditions that may present with similar symptoms of inattention, impulsivity or behaviour dysregulation. One important distinction is between ADHD and mood disorders, particularly major depressive disorder and bipolar disorder. In

depressive episodes, difficulties with concentration and motivation may resemble inattentive symptoms, however, these impairments typically occur in the context of persistent low mood, anhedonia, negative thoughts, guilt ruminative thinking and pessimistic cognition. In ADHD, attention difficulties are more chronic and often begin during childhood (1).

Anxiety disorder may also present with difficulty in concentrating, particularly when excessive worry interferes with sustaining attention. But unlike ADHD, attention disruption in anxiety disorders is usually secondary to intrusive anxious thoughts rather than a primary deficit in attention regulation. Learning disorders and intellectual developmental disorders must be considered when evaluating academic difficulties, as these conditions may affect education performance independently of attentional capacity (1).

Substance use disorders may mimic symptoms of impulsivity and impaired concentration, particularly in adolescents and adults. In such cases, careful clinical assessment is required to determine whether attention difficulties precede substance abuse or emerge as a consequence of intoxication or withdrawal. Accurate differential diagnosis is essential because treatment approaches differ substantially between these conditions (1).

5.3.1.5 ADHD Through a Recovery-Oriented Lens.

Through the recovery framework outlined earlier, ADHD presents a distinct but coherent model. Recovering from ADHD rarely means complete elimination of core attentional vulnerability. Instead, it involves adaptive restructuring and executive compensation.

Pharmacological treatment, particularly stimulant medication targeting dopamine and norepinephrine systems, has demonstrated significant efficacy in reducing symptom severity and improving executive functioning (23). The Multimodal Treatment Study of ADHD (MTA), originally designed as a large collaborative trial comparing treatment strategies for children with ADHD (14), later showed in long-term follow-up that although treatment-related improvements may be maintained over time, the initial advantages of children with more intensive treatment diminish, and many children continue to show functional impairments compared with non-ADHD peers (24).

However, symptom reduction alone does not equal recovery. True functional recovery in ADHD includes development of a structured routine, executive skill training, psychoeducation, family support, environmental modification and improved self-awareness.

Children who grew up in a supportive environment where the caregivers understood the neurobiological nature of ADHD are more likely to develop adaptive coping strategies and a healthier sense of self. Longitudinal studies demonstrated parental psychoeducation and consistent behavioural structure significantly improve functional outcomes (19) In contrast, chronic criticism, punishment and misunderstanding of symptoms are associated with increased shame, decreased self-esteem and reinforced oppositional behaviour (13).

Therefore, attachment and environmental context play indirect but powerful roles in ADHD outcomes.

5.3.1.6 Emotional Regulation and Identity

Although ADHD is primarily defined by inattention, hyperactivity and impulsivity, emotional dysregulation is increasingly recognised as an important clinical component. Many individuals with ADHD struggle with low frustration tolerance, irritability and rapid emotional shifts, often described as emotional lability. These emotional responses are not simply secondary personality traits but are closely linked to difficulties in emotion regulation and may involve dysfunction within striato-amygdalo-medial prefrontal networks (25).

Over time, repeated difficulties, peer rejection or negative feedback from authority figures may influence social development. Longitudinal research suggests that children with untreated ADHD are at increased risk of low self-esteem, internalisation of symptoms and social difficulties during adolescence and adulthood (13,58). When behaviours are misunderstood as laziness or lack of effort, individuals may internalise these labels which can persist even when symptoms improve later.

Recovering from ADHD, therefore, extends beyond symptom control. It also involves identity restructuring. When individuals understand their neurodevelopmental profile and their compensatory tools, self-precision may shift from self-blame to informed self-management. This cognitive reframing is consistent with broader recovery models discussed earlier in this thesis, where agency replaces helplessness and vulnerability becomes manageable rather than defining.

5.3.1.7 Adaptive Compensation in Recovery

Unlike episodic psychiatric disorders in which remission is the primary goal, recovery in ADHD often reflects adaptive compensation rather than complete resolution of core traits. Longitudinal studies demonstrate that although symptom intensity frequently declines with age, executive vulnerabilities may persist into adulthood (13,23).

Long-term follow-up from the MTA study suggests that although symptoms and impairments may improve over time, many individuals continue to experience functional difficulties, highlighting the need to focus recovery-oriented care not only on symptom reduction but also on impairment, daily functioning and sustainable support strategies (24).

However, not all compensatory patterns are adaptive. In some children and adolescents with ADHD, especially when hyperactivity, impulsivity and peer rejection are prominent, attempts to gain attention, status or belonging may develop into maladaptive forms of compensation. These may include aggression, repeated physical conflicts, disruptive peer leadership, association with antisocial peer groups or conduct-related behaviours (57,58). Rather than supporting recovery, such patterns may reinforce impulsivity, social exclusion and conflict with authority, increasing the risk of later delinquency or criminal justice involvement. Longitudinal evidence shows that childhood ADHD, particularly when associated with conduct disorder, is linked to increased risk of adult delinquency and criminal outcomes (57). Peer rejection and impaired friendship quality in children with ADHD have also been associated with poorer long-term psychosocial outcomes (58).

Therefore, recovery-oriented care in ADHD should not only strengthen adaptive compensation, but also identify and redirect maladaptive compensatory behaviours before they become entrenched. This pattern illustrates the central argument of this thesis: recovery does not always mean elimination of vulnerability. In ADHD, it often means learning to regulate, compensate and reorganise around persistent vulnerabilities in ways that improve functioning rather than reinforce maladaptive developmental pathways.

5.3.1.8 Conceptual Integration Within the Recovery Framework

ADHD demonstrated recovery is diagnosis sensitive. In this disorder, clinical recovery refers to reduction of symptom severity, often supported by stimulant or non-stimulant pharmacotherapy (23). Functional recovery reflects executive compensation and sustained participation in the academic, occupational and social domains. Personal recovery involves improved self-understanding and identity stabilisation (3).

Neurobiological vulnerabilities, developmental timing, family response and environmental support interact dynamically to shape long-term ADHD trajectories (13,19,25). When properly supported, individuals with ADHD can achieve stable functioning and meaningful social integration despite persistent attention deficits.

ADHD does not simply disappear over time. It is reorganised within the individual developmental context.

5.3.2 Borderline Personality Disorder (BPD)

5.3.2.1 Definition and Diagnostic Criteria

BPD is characterised by pervasive instability in emotional regulation, interpersonal relationships, impulse control and identity coherence. According to the DSM 5 TR, diagnosis requires a persistent pattern of five or more of these characteristic features: emotional dysregulation, intense fear of abandonment, unstable and rapidly shifting relationships, identity disturbances, impulsive behaviour, reoccurring suicidal behaviour or self-injury, chronic feeling of emptiness, inappropriate anger, transient stress-related paranoia and dissociation (1).

Unlike episodic psychiatric disorders such as major depressive disorder or panic disorder, BPD reflects enduring patterns of emotional and relational functioning. Symptoms typically begin in adolescence or early adulthood and are expressed across contexts (1,34). The ICD 11 conceptualises personality disorders dimensionally, emphasising severity of impairment in self-functioning and interpersonal functioning, which aligns closely with the clinical presentation of BPD (28).

Importantly, BPD is not simply emotional instability, but rather a disorder that impacts the regulation of affect, impulse, identity and attachment.

5.3.2.2 Etiology, Biological Sensitivity and Environmental Invalidation

BPD does not emerge from a single traumatic event nor from genetics alone. Rather, it develops through interactions between biological vulnerabilities and environmental experience.

Studies on twins suggest moderate heritability, indicating that emotional sensitivity and impulsivity temperaments may have genetic underpinnings (29). Neurobiological research suggests abnormalities in frontolimbic circuits involved in emotional processing and regulation in BPD. Some fMRI studies report exaggerated amygdala responses and impaired habituation to emotional stimuli, while meta-analytic findings indicate broader dysfunction across limbic and prefrontal regulatory regions rather than a simple pattern of consistent amygdala hyperactivity (8,30). This dysregulation may contribute to rapid emotional escalation and difficulty returning to baseline once activated.

However, biological sensitivity alone does not produce BPD. Developmental research highlights the role of chronic invalidation, attachment disruption, emotional neglect and exposure to trauma in childhood (31,32). Linehan's biosocial model integrates this idea by proposing that BPD develops

through transactions between biological emotional vulnerability and an invalidating developmental environment, in which emotional expression is persistently dismissed, punished or inadequately responded to (32).

In such conditions, a child does not learn regulation strategies. Emotional experiences become overwhelming, poorly understood and externally managed through impulsive behaviour or relational instability. Thus, BPD pathology reflects arrested development of emotional regulation within a relational context.

5.3.2.3 Clinical Manifestation and Functional Consequences

Clinically, BPD is characterised by extreme fluctuation in moods that may shift within hours. Emotional responses are intense and often triggered by perceived rejection or imagined abandonment. Relationships are unstable and swing between idealisation and devaluation (1,34). Fear of abandonment plays a central role in the disorder. Even a minor separation may be experienced as catastrophic (1,34).

Self-injuring BPD often functions as a maladaptive regulation strategy rather than a primary suicidal act. Many patients describe self-harm as a way to reduce unbearable emotional tension or dissociation. Longitudinal studies demonstrate that self-harm behaviour is most frequent in early adulthood and tends to decrease with age in many individuals (29,33).

Functionally, BPD significantly impairs occupational stability, relational continuity and social integration. Comorbidities such as depression, PTSD, eating disorders and substance abuse disorder are common in BPD patients. Hospitalisation is frequent during early stages of the disorder (34).

5.3.2.4 Treatment and Mechanisms of Change

According to NICE and contemporary review evidence, psychotherapy is the cornerstone of treatment for BPD (37,34). DBT was the first treatment specifically developed for BPD and is one of the most empirically supported interventions (32). Randomised control trials demonstrate significant reduction in suicidal behaviour, self-harm and psychiatric hospitalisation among individual's receiving DBT (35).

Mentalisation based therapy (MBT) focuses on improving the individual's capacity to understand both their own and others' mental state, thinking and feelings, thereby stabilising interpersonal relationships and reducing emotional reactivity (36). Similarly, Schema therapy addresses maladaptive core beliefs and attachment patterns, and demonstrates efficacy in randomised clinical trials (39).

Pharmacotherapy may be used together with psychotherapy to target mood instability, impulsivity or comorbid conditions. However, no medication has been shown to treat BPD as a whole (34).

These findings carry a practical therapeutic message: even though borderline personality disorder is characterised by longstanding emotional instability, its symptoms can be progressively reshaped through targeted skill development and relational restructuring.

5.3.2.5 Longitudinal Course and Prognosis

BDP was historically considered a chronic condition with a poor prognosis. Fortunately, longitudinal research has significantly revised this view. Follow up studies indicated that a substantial portion of patients achieve symptomatic remission over 10 to 15 years. However, remission does not necessarily imply full functional recovery. Many individuals continue to experience interpersonal instability or occupational impairment despite a reduction in acute symptoms (33,38).

Self-harming and suicidal behaviour tend to decline with age, while identity disturbances and interpersonal sensitivity may persist (33). Emotional intensity often becomes less extreme over time, particularly with structured therapeutic intervention (38,34).

Distinction between remission and recovery is particularly relevant within the theoretical framework.

5.3.2.6 Differential Diagnosis

BPD mood shifts are rapid, reactive to interpersonal stressors and typically last hours to days. In contrast, bipolar disorder involves episodic mood changes lasting weeks and occurring independently of immediate relational triggers (1).

Post-traumatic stress disorder may share emotional reactivity and dissociation with BPD, but PTSD symptoms are characteristically linked to re-experiencing trauma, avoiding trauma related stimuli and persistent threat-related arousal (16,17).

ADHD disorder may overlap with BPD in impulsivity, emotional dysregulation and interpersonal difficulties. However, ADHD is a neurodevelopmental disorder with onset in childhood and is primarily characterised by persistent inattention, hyperactivity and executive dysfunction, whereas BPD is defined by instability in identity, affect regulation, interpersonal relationships and fear of abandonment (1,19,25). Although both conditions may involve affective lability and behavioural impulsivity, the interpersonal sensitivity and disturbance of self-image seen in BPD patients are not core features of ADHD (25,34).

Accurate diagnosis requires structured clinical assessment and evaluation of long-standing interpersonal patterns, identity disturbances, developmental history and the context in which the symptoms emerge, rather than reliance on isolated symptom clusters alone.

5.3.2.7 Recovery in Borderline Personality

Within the multidimensional recovery framework adopted in this thesis, recovery in borderline personality disorder extends beyond symptom reduction and reflects a progressive restructuring of emotional, relational and identity systems (3,34). While clinical remission may involve reduction in self-harm, impulsivity and affective instability (1), full recovery cannot be defined solely by behavioural stabilisation.

The CHIME framework provides a structured model for understanding personal recovery. In individuals with BPD, connectedness refers to the gradual development of stable and secure relationship bonds that are no longer dominated by fear of abandonment. Hope emerges when a patient begins to experience emotional states as tolerable rather than catastrophic. Identity reconstruction is particularly central in BPD. Given the diagnostic criterion of an unstable self-image, recovery involves the formation of a coherent and conscious sense of self. Meaning develops as individuals integrate traumatic or invalidating developmental experiences into a coherent life narrative, rather than repeatedly re-experiencing past events in fragmented forms. Empowerment develops as emotional regulation skills replace impulsive reactions, allowing the individual to exercise greater agency over behaviour rather than being dominated by affective intensity (3).

Recovery in BPD may be conceptualised simultaneously from the functional and developmental perspectives. Functionally, it is reflected in the individual's increasing ability to sustain occupational roles and maintain relatively stable, reciprocal relationships despite ongoing emotional sensitivity, indicating improved regulation of affect rather than its elimination. Developmentally, recovery represents progressive strengthening of top-down regulatory processes and greater tolerance of interpersonal vulnerability, such that affective arousal is more often processed reflectively rather than acted upon impulsively. Evidence from DBT research suggests that structured psychotherapy can improve emotional regulation and reduce BPD symptom features, while neuroimaging findings show decreased amygdala activation after DBT and improved amygdala habituation associated with better emotional regulation (7,8). These findings support the understanding of recovery in BPD as an adaptive process involving improved emotional regulation rather than simple symptom suppression.

5.3.3 Post Traumatic Stress Disorder (PTSD)

5.3.3.1 Definition and Diagnostic Criteria

PTSD is a trauma and stressor related psychiatric disorder that develops following the exposure to death or the threat of death, serious injury, sexual assault or violence. According to the DSM-5 criteria,

exposure may occur through direct experience, witnessing the event, learning that it occurred to a close family member or a friend, or repeated exposure to distressing details or traumatic events (1).

PTSD is characterised by 4 core symptom clusters:

1. Intrusive re-experiencing (flashbacks, nightmares, intrusive memories)
2. Avoidance of trauma related thoughts, emotional or external reminders
3. Negative alteration in cognition and mood
4. Persistent hyperarousal and exaggerated stress response

Symptoms must persist for more than one month and result in clinically significant distress or functional impairment (1).

Unlike transient stress reaction, PTSD reflects a persistent dysregulation of adaptive stress-regulation mechanisms in which trauma-related cues continue to trigger threat perception, hyperarousal and intrusive symptoms even when the individual is no longer in danger (1,16).

5.3.3.2 Neurobiological and Stress Regulation Mechanisms

From a neurological perspective, PTSD is associated with dysregulation within the neural circuits responsible for fear processing, emotional regulation and memory integration.

Functional imaging studies generally demonstrate increased amygdala activation in PTSD in response to threat-related stimuli, trauma reminders and fearful cues (16). Clinically, this exaggerated amygdala response corresponds to hypervigilance, exaggerated startle response, and intense physiological arousal during reminders of trauma. At the same time, reduced activity of the medial prefrontal cortex weakens top-down inhibitory control over fear responses (16). As a result, emotional reactions become rapid, poorly regulated and difficult to calm, contributing to irritability, intrusive memories and persistent anxiety (16).

The hippocampus, which is responsible for contextualising memory and distinguishing past from present, also shows functional alteration in PTSD (16). Impaired hippocampal processing disrupts the integration of traumatic memories into coherent autobiographical narratives. Clinically, this dysfunction may manifest as flashbacks and dissociation re-experiencing, in which the traumatic event is relieved as if it were occurring in the present moment. Together, hyperactive threat detection, impaired contextual memory and weakened regulatory control result in defective fear extinction, allowing trauma-associated cues to continue triggering survival responses long after the danger has passed.

In addition, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis may alter stress-hormone signalling and contribute to persistent physiological arousal in PTSD (16). Sleep disturbance, concentration impairment and persistent somatic tension reflect this sustained physiological arousal (1,16). Importantly, these alterations represent maladaptive but modifiable stress learning that can be unlearned rather than seen as irreversible damage (17,16).

Trauma-focused psychotherapy, particularly exposure-based approaches, may promote corrective learning by reducing avoidance and allowing trauma-related cues to be experienced in a safe context (17). This is consistent with fear-extinction models in which medial prefrontal mechanisms help regulate amygdala-mediated fear responses (16). In this sense, recovery involves restoration of regulatory balance within fear circuitry rather than erasure of traumatic memory.

5.3.3.3 Developmental Vulnerabilities and Attachment.

Exposure to trauma alone does not inevitably result in PTSD, and developmental, relational and individual vulnerability factors influence whether post-traumatic symptoms persist (16). Attachment theory provides an explanatory framework for understanding how early relational security may support the development of stable affect regulation and the capacity to process overwhelming emotional experiences (9). When regulatory and relational resources are limited, traumatic memories may remain poorly integrated, fragmented or difficult to contextualise, contributing to persistent re-experiencing and impaired recovery (16,17).

5.3.3.4 Clinical Manifestation and Functional Consequences

Clinically, PTSD is characterised by recurrent intrusive memories, nightmares, flashbacks, emotional numbness, avoidance behaviour, hypervigilance, irritability, sleep disturbance and impaired concentration (16).

Avoidance is particularly central to the maintenance of PTSD. Individuals attempt to suppress trauma-related thoughts or avoid situations that trigger emotional distress. While avoidance provides temporary relief, it prevents the natural extinction of conditioned fear responses and reinforces the perception that trauma reminders remain dangerous (16,17).

The mechanism parallels processes observed in OCD, where avoidance and ritualistic behaviour temporarily reduce anxiety but strengthen maladaptive fear association over time (21). In both disorders, the absence of corrective exposure maintains pathological fear networks.

Functionally, PTSD often leads to occupational instability, social withdrawal, emotional detachment and impaired relational trust. Without intervention, symptoms may fluctuate, intensify under stress or persist chronically (16). In addition to neurobiological and psychological mechanisms, social factors significantly influence recovery trajectories in PTSD. Shame, guilt, social rejection, lack of recognition and limited social support may delay help-seeking, reinforce avoidance and intensify self-blame. Trauma-focused treatment and recovery-oriented care therefore often require psychoeducation, meaning-making, adaptive reappraisal of trauma-related beliefs, restoration of self-efficacy and strengthening of social support (3,17). Within the multidimensional framework, reduction of stigma and restoration of social validation represent important components of personal and social recovery (3,10,17).

5.3.3.5 Differential Diagnosis

PTSD shares several clinical features with other trauma-related and anxiety disorders, making differential diagnosis an important aspect of clinical assessment. Acute stress disorder may present with symptoms like PTSD, including intrusive memories, hyperarousal and avoidance behaviours. However, acute stress disorders occur within the first month following a traumatic event, whereas PTSD is diagnosed when symptoms persist for longer than a month (1).

Generalised anxiety disorder and panic disorder may also involve persistent anxiety and psychological hyperarousal. Unlike PTSD, these disorders are not necessarily linked to a specific traumatic event and typically lack the characteristic re-experiencing symptoms such as intrusive memories or trauma related flashbacks (1).

Depressive disorders may overlap with PTSD through symptoms such as sleep disturbance, concentration difficulty and emotional numbing. However, PTSD is distinguished by trauma-related intrusive recollections, avoidance of trauma reminders and persistent threat-related arousal (1). Dissociative disorder may also resemble PTSD when symptoms include depersonalisation, derealisation or disruption in memory and identity. Although dissociative disorders are classically distinguished by these features rather than by overt fear-based symptoms alone, this distinction is not absolute. Trauma-related dissociation may itself represent a defensive response to overwhelming threat, including freezing or shutdown states. The dissociative subtype of PTSD demonstrates that dissociation and trauma-related symptoms can coexist within the same conditions (1,54).

Therefore, dissociative disorders and PTSD remain diagnostically distinct, but may overlap in both clinical presentation and underlying trauma-related mechanisms (1,54).

5.3.3.6 Treatment and Mechanisms of Change

Trauma-focused psychotherapies are among the main evidence-based treatments for PTSD and commonly include psychoeducation, emotional regulation skills, exposure to traumatic memories, cognitive restructuring, meaning making and reorganisation of trauma-related memory processes (17). Prolonged exposure therapy and trauma focused cognitive behavioural therapy aim to reduce avoidance and facilitate controlled engagement with trauma memories. Through repeated, structured exposure within a safe therapeutic setting, the patient gradually experiences corrective learning, so that trauma reminders are no longer experienced as inherently dangerous.

This process is conceptually similar to exposure and response prevention in OCD, where ritualistic behaviour temporarily reduces anxiety but reinforces maladaptive fear-related responses over time (21). In PTSD treatment, exposure-based work aims not to erase the traumatic memory, but to reduce avoidance, change threatening meanings attached to trauma memories and help the patient discriminate between past trauma and present safety (17). This is consistent with fear extinction models in which prefrontal regulatory mechanisms help modulate amygdala-mediated fear responses (16). By confronting rather than avoiding traumatic memories, the individual rebuilds tolerance to distress and restores adaptive emotional regulation.

Trauma-focused psychotherapy directly targets maladaptive cognitive and behavioural processes by reducing avoidance, changing problematic appraisals, accessing and integrating trauma memories, and supporting behavioural re-engagement (17). Therefore, recovery in PTSD depends not only on symptom reduction, but also on behavioural re-engagement, cognitive integration and restoration of adaptive emotional regulation (17).

5.3.3.7 Recovery in PTSD - an Adaptive Integration Process

Within the multidimensional recovery framework adopted in this thesis, recovery in PTSD extends beyond symptom reduction. Clinical recovery may involve decreased flashbacks, improved sleep and reduced avoidance (1,16).

Functional recovery is reflected in reengagement in occupational and academic roles, restoration of interpersonal relationships and increased participation in social environments previously avoided.

Personal recovery involves integration of the traumatic experience into a coherent life narrative rather than persistent fragmentation. The individual no longer relives the trauma as a present threat but recognises it as a past event (3,17).

From a developmental perspective, recovery represents improved emotional regulation, increased tolerance of distress, reduced avoidance-based coping and greater ability to distinguish past trauma from present safety (16,17). When trauma has affected interpersonal trust or attachment security, recovery may also involve rebuilding relational safety through supportive therapeutic and social relationships (9,17).

Neurobiological models of PTSD suggest that recovery involves improved regulation of fear circuitry, including stronger prefrontal modulation of amygdala-mediated threat responses (16). Trauma-focused psychotherapies support this process clinically by reducing avoidance, changing threatening appraisals, integrating trauma memories and helping the patient discriminate between past trauma and present safety (17). Therefore, recovery is better understood as adaptive regulation and integration rather than simple symptom disappearance.

Importantly, recovery in PTSD does not require forgetting the trauma; it requires transforming how the individual relates to it. The memory remains, but its emotional intensity, psychological charge and behavioural control gradually diminish as the individual develops new meanings, reduces avoidance and learns to distinguish past trauma from present safety (17). The individual gradually regains agency over behavioural and emotional responses, which reflects the broader recovery principle that improvement involves adaptive regulation, meaning integration and functional participation in life rather than complete elimination of vulnerability (3,17).

5.3.4 Antisocial Personality Disorder (ASPD)

5.3.4.1 Definition and Diagnostic Framework

ASPD is a chronic and pervasive personality disorder characterised by a sustained pattern of disregard and violation of the rights of others, beginning in childhood or early adolescence and persistent into adulthood (1). According to the DSM-5 TR criteria, diagnosis requires evidence of conduct disorder before the age of fifteen, alongside enduring adult features such as deceitfulness, impulsivity, irritability, aggression, reckless disregard for safety, consistent irresponsibility and lack of remorse (1). ASPD reflects maladaptive personality traits and persistent interpersonal dysfunction, although the overt behavioural expression of this trait may fluctuate across developmental stages and environmental contexts.

The ICD 11 conceptualise this condition as a dissocial personality disorder, emphasising severe disturbances in interpersonal functioning, including insensitivity, hostility and disregard for social

obligations (28). This dimensional approach highlights that the disorder involves not only observable behaviour but also impairment in affect regulation, empathy and moral internalisation.

5.3.4.2 Epidemiology and Social Burden

Epidemiological studies estimate the prevalence of ASPD at approximately 1–4% in the general population, with substantially higher rates in forensic and incarcerated populations (41,48). The disorder shows strong associations with substance use disorders, ADHD, mood disorders and other externalising or personality-related pathologies (1,48). Social consequences are considerable and include criminal involvement, occupational instability, disrupted relationships, increased incarceration rates and substance misuse.

Overt antisocial behaviour such as physical violence, reckless driving and substance misuse may change across development and may decline in frequency with age, although interpersonal dysfunction, limited empathy and maladaptive personality traits may persist into adulthood (1,48). This pattern suggests that a reduction in observable antisocial behaviour does not necessarily indicate full functional recovery but may instead reflect partial behavioural attenuation without deeper relational change.

5.3.4.3 Developmental and Attachment-Based Models

From a developmental perspective, attachment theory provides a coherent framework for understanding the emergence of antisocial patterns. Early caregiving disruption such as neglect, inconsistent discipline, coercive parenting or exposure to violence may interfere with the formation of secure attachments, impair emotional regulation and hinder internalisation of moral standards (9,48). When early relational environments fail to provide stability and attuned responsiveness, adaptive survival mechanisms such as defensive detachment, mistrust, hostility and diminished sensitivity to others' needs may develop (48).

Social-cognitive and mentalisation-based perspectives further refine this developmental framework by emphasising difficulties in reflective functioning, social cognition and the capacity to recognise one's own and others' mental states (42,48). In antisocial and callous-unemotional developmental pathways, reduced sensitivity to others' distress, impaired perspective-taking and deficits in decision-making or reinforcement learning may contribute to difficulties anticipating interpersonal consequences, recognising emotional impact and developing guilt in a socially integrated manner (43,48). Within this model, antisocial behaviour is conceptualised not simply as deliberate misconduct, but as reflecting disrupted integration between affective processing, social cognition and higher-order cognitive regulation.

5.3.4.4 Neurobiological Mechanism

Neurobiological research complements developmental models by identifying alterations in the fronto-limbic circuit among individuals with antisocial and psychopathic traits (43,44). Structural and functional imaging studies have reported reduced amygdala reactivity associated with impaired fear conditioning and decreased responsiveness to distress cues in others. This diminished sensitivity may contribute to reduced empathy and limited behavioural adaptation following punishment (43,44).

Dysfunction within frontal brain regions, particularly the orbitofrontal and ventromedial prefrontal cortex has been linked to impaired impulse control, poor decision making, reduced autonomic reactivity to socially meaningful stimuli and psychopathic like behaviour (44). These alterations may help explain persistent risk-taking, aggression and limited behavioural inhibition (43,44).

Neuroimaging and computational accounts further suggest abnormalities in reward and punishment learning, particularly involving striatal and ventromedial prefrontal systems that process prediction error and expected value information (43,48). Individuals with antisocial traits may demonstrate reduced behavioural adjustment following loss or punishment, reflecting altered integration of reward and aversive feedback (48). This imbalance in reward and loss processing may contribute to continued risk-seeking and reinforcement-driven behaviour despite adverse consequences (43,48).

Collectively, these findings indicate that anti-social traits are associated not only with impaired fear conditioning but also with dysregulated reward processing. However, these neurobiological patterns represent increased vulnerability rather than deterministic causes.

5.3.4.5 Cognitive Behavioural and Schema Perspectives

Cognitive behavioural formulation identifies enduring maladaptive schemes such as entitlement, mistrust, hostility and beliefs that interpersonal relationships are inherently exploitative (45). These schemas are conceptualised as emerging in early adverse environments and become reinforced through repeated interpersonal conflict and maladaptive behavioural patterns (45,48). Over time, these cognitive patterns may contribute to persistent distrust of others, antagonistic interpersonal expectations and a tendency to interpret social interactions as competitive or threatening rather than cooperative.

Schema therapy suggests that rigid cognitive and interpersonal patterns may be partially modified through structured, long-term therapeutic work focused on emotional awareness, responsibility and the restructuring of distorted beliefs and interpersonal expectations (46). In ASPD and antisocial behaviour, such approaches should be understood within a broader, structured and multimodal management

framework, because treatment engagement and sustained behavioural change remain clinically challenging (47,48).

Current management guidelines emphasise structured behavioural interventions, substance misuse treatment, anger regulation strategies and environmental stabilisation rather than attempts at comprehensive personality transformation (47). Pharmacological intervention has limited evidence for modifying core antisocial traits and primarily targets comorbid conditions such as substance use disorder, mood instability or impulsive aggression (47).

5.3.4.6 Differential Diagnosis in Antisocial Personality Disorder

Antisocial personality disorder must be distinguished from other psychiatric conditions that involve impulsivity, behavioural dysregulation or difficulties with social norms. There is one important distinction between ASPD and BPD. While both disorders may involve impulsive behaviours and interpersonal conflict, BPD is characterised primarily by emotional instability, fear of abandonment, and identity disturbance, whereas ASPD is defined by persistent disregard for the rights of others and the violation of social norms (1,34).

Substance use disorders may also produce behaviours resembling antisocial traits, including impulsivity, aggression and legal problems. In such cases, clinicians must determine whether antisocial behaviour occurs exclusively during periods of intoxication or represents a stable personality pattern independent of substance use (1).

Conduct disorder in childhood is closely related to antisocial personality disorder and often precedes the development of the adult disorder. However, conduct disorder is diagnosed during childhood or adolescence, while ASPD requires evidence of persistent behavioural patterns continuing into adulthood. Distinguishing APD from narcissistic personality disorder may also be necessary, as both conditions may involve exploitative interpersonal behaviour. However, narcissistic personality disorder is primarily characterised by grandiosity and the need for admiration rather than the pervasive violation of social rules (1,48).

5.3.4.7 Recovery in Antisocial Personality Disorder

Recovery in ASPD requires a functional and pragmatic perspective. Unlike episodic psychiatric disorders, in which symptom remission is central, ASPD is defined by enduring lifelong personality traits and entrenched behavioural reinforcement patterns (1,28). Accordingly, recovery is best

conceptualised not as a complete personality transformation, but as a sustained reduction in harmful behaviour, improved regulatory capacity and functional social participation (47,61).

Within the multidimensional framework, recovery in ASPD can be understood across several domains. Clinical recovery refers to a reduction in aggressive, exploitative and criminal behaviour. Functional recovery reflects improved occupational stability, reduced legal involvement and sustained engagement in structured social roles. Personal recovery may involve increased reflective capacity, recognition of interpersonal consequences and strengthening of impulse control. Social recovery includes decreased reliance on coercive interpersonal strategies and improved participation in prosocial networks.

From a cognitive behavioural perspective, antisocial behaviour is maintained through the reinforcement of maladaptive schemas such as entitlement and dominance (43,45,48). Recovery involves the gradual disruption of theses reinforcement cycles, identification of triggers and development of alternative behavioural responses, reflecting a shift from reward-driven reactivity towards more regulated decision making (43,45,48).

Attachment-informed and mentalisation-based approaches conceptualise recovery as strengthening reflective functioning, accountability and awareness of interpersonal consequences (9,42,48). Improving reflective capacity and perspective-taking may support better impulse control and more regulated social decision-making, although these goals may be harder to achieve in individuals with pronounced callous-unemotional or psychopathic traits (43,48).

Longitudinal evidence suggests that overt antisocial behaviour may attenuate with age due to neurodevelopmental maturation and environmental contingencies (1,48). However, meaningful recovery extends beyond the reduction of criminality and is defined by sustained behavioural responsibility and social reintegration (47).

In addition, societal and institutional stigma surrounding antisocial behaviour may further complicate recovery trajectories by reinforcing external labelling, limiting treatment engagement and reducing opportunities for structured rehabilitation (10,48,61).

In this framework, recovery in ASPD reflects adaptive restructuring within enduring personality vulnerability rather than complete transformation of temperament. It involves gradually strengthening regulatory and reflective capacities sufficient to support stable social functioning.

5.3.5 Obsessive-Compulsive Disorder (OCD)

5.3.5.1 Definition and Diagnostic Framework

OCD is a chronic psychiatric disorder characterised by the presence of obsessions, compulsions or both (1). Obsessions are recurrent and persistent intrusive thoughts, urges or images that are experienced as unwanted and cause marked anxiety or distress. Compulsions are repetitive behaviours or mental acts performed in response to obsessions or according to rigid rules, aimed at reducing distress or preventing feared outcomes (1).

Although compulsions temporarily reduce anxiety, they reinforce the perceived threat and maintain the obsessive-compulsive cycle (21). The disorder is time consuming and often significantly impairs occupational, academic and interpersonal functioning (2)

5.3.5.2 Epidemiology Functional Impact

The lifetime prevalence of OCD is commonly estimated at around 2% of the general population, with some sources reporting a broader range of approximately 1–4% (15,21). OCD age of onset ranges from very early childhood into adulthood, and childhood onset is common, with approximately 30–50% of individuals having onset in childhood (21). OCD commonly co-occurs with other psychiatric conditions, including depressive, anxiety-related and tic-related disorders (21).

Functional consequences include reduced quality of life, social withdrawal, impaired concentration and occupational disruption (2,15). Family members may become involved in accommodating compulsive rituals, unintentionally reinforcing the disorder and increasing the overall burden (15).

5.3.5.3 Neurobiological Mechanism

In neurobiology, OCD is associated with dysfunction in the cortico-striato-thalamo-cortical (CSTC) circuit (21,22). Hyperactivity within the orbitofrontal cortex, anterior cingulate cortex and caudate nucleus has been implicated in OCD and may contribute to exaggerated threat monitoring, error detection and persistent attention to perceived danger (21,22). Clinically, hyperactivation of these circuits may correspond to intrusive doubts, persistent threat monitoring and a sense that something is “not right” even in the absence of objective danger (21, 22). Heightened responsibility beliefs and catastrophic interpretations of intrusive thoughts are better understood within the cognitive behavioural model of OCD (5). The caudate nucleus, part of the cortico-striato-thalamo-cortical (CSTC) circuit, helps regulate the flow of information through the basal ganglia, and acts as a gate involved in inhibiting inappropriate actions. It is crucial for habit formation. An impaired gating mechanism

prevents the proper suppression of signals from the orbitofrontal cortex (OFC), resulting in failure to filter or gate behavioural responses. In OCD, this may be experienced as difficulty terminating repetitive actions such as checking or cleaning, which leads to the rigid, repetitive and compulsive ritual characteristics of the disorder (21).

Serotonergic neurotransmission has been implicated in the modulation of the CSTC circuits involved in inhibitory control and threat monitoring. Evidence of the association is supported by the clinical efficacy of selective serotonin reuptake inhibitors (SSRIs), although treatment response is often partial and likely reflects broader neuroadaptive processes rather than a single neurotransmitter mechanism (21,22).

The delayed clinical response to SSRIs, the partial nature of treatment response in many patients and the difficulty of defining treatment-resistant OCD suggest that OCD pathology cannot be explained by serotonergic signalling alone. Instead, it likely involves interactions between serotonergic, dopaminergic, glutamatergic and GABAergic systems, CSTC circuit dynamics and learned behavioural reinforcement patterns (11,21).

Therefore, OCD can be understood as a disorder of maladaptive threat monitoring and impaired inhibitory control in which fear signals are amplified and corrective learning is disrupted (21,22).

5.3.5.4 Cognitive Behavioural Model

The cognitive behavioural model conceptualises OCD as a cycle of intrusive thoughts, catastrophic misinterpretations, anxiety escalation and compulsive neutralising behaviours (5,21). Intrusive thoughts are universal. In OCD, however, they are interpreted as personally meaningful or morally significant, leading to exaggerated responsibility and threat appraisal (5).

Compulsion temporarily reduces anxiety, reinforcing the belief that the ritual prevented harm (5,21). This short-term relief creates a paradoxical learning process in which the behaviour becomes negatively reinforced. That is, because anxiety decreases immediately after the ritual, the individual learns that the compulsion is necessary to prevent the threat. Over time, the mechanism strengthens the obsession-compulsion cycle. Although compulsions provide temporary emotional relief, they simultaneously prevent the corrective learning that the feared outcome would not occur, thereby maintaining the disorder (11). Avoidance behaviours further interfere with natural extinction of fear responses.

In this sense, compulsive rituals function as maladaptive coping strategies that reduce immediate distress while reinforcing the underlying fear network.

Exposure and response prevention (ERP), the “gold standard” of psychotherapeutic intervention, directly targets this cycle by encouraging systematic exposure to feared stimuli while preventing compulsive rituals (11). Through repeated exposure without neutralisation, extinction learning occurs and anxiety diminishes over time (11).

5.3.5.5 Differential Diagnosis in OCD

Obsessive compulsive disorder must be differentiated from several psychiatric conditions that involve repetitive thoughts or behaviours. One of the most important distinctions is between OCD and obsessive-compulsive personality disorder (OCPD). While both conditions may involve rigid thinking patterns and perfectionism, OCPD is characterised by enduring personality traits related to control and orderliness rather than intrusive thoughts and anxiety-driven compulsive rituals (1).

Anxiety disorders may also present with intrusive worry and repetitive behaviours intended to reduce distress. However, in generalised anxiety disorder these worries are typically related to real life concerns such as health, finances or relationships whereas, obsessions in OCD are often irrational, intrusive and difficult for individuals to control (1).

Psychotic disorders may occasionally resemble OCD when intrusive thoughts appear bizarre or irrational (1). The crucial distinction lies in the degree of insight. Meaning, that individuals with OCD usually recognise that their obsessions are excessive or unreasonable, whereas delusional beliefs in psychotic disorders are typically held with strong conviction. Tic disorders and certain neurodevelopmental conditions may also present with repetitive behaviours; however, these behaviours are generally not driven by intrusive obsessive thoughts (1,2).

5.3.5.6 Treatment Approaches

First-line treatment for OCD includes cognitive behavioural therapy with exposure and response prevention, along with selective serotonin reuptake inhibitors (SSRIs) as pharmacotherapy (21). ERP facilitates corrective learning by exposing the individual to feared stimuli while preventing compulsive neutralisation, thereby weakening maladaptive threat associations and reducing the reinforcement cycle that maintains obsessions and compulsions (21).

Neuroimaging studies suggest that successful CBT may be associated with changes in hyperactive cortical-striatal activity, supporting the possibility of treatment-related modulation of neural circuits

involved in symptom regulation (4,22). However, symptom reduction may be gradual and residual vulnerability often persists (12).

Family involvement in treatment is important, particularly in reducing accommodating behaviours that maintain the disorder (15).

5.3.5.7 Recovery in Obsessive Compulsive Disorder

OCD recovery requires a multidimensional perspective. Unlike acute anxiety episodes, OCD often follows a chronic and fluctuating course (12). Therefore, recovery is not defined solely by the complete absence of intrusive thoughts, but by a sustained reduction in compulsive behaviours, improved distress tolerance and functional reintegration (11,12).

Within the multidimensional framework adopted in this thesis, recovery in OCD can be understood across several interconnected domains. Clinical recovery refers to the reduction in the frequency and intensity of obsessions and compulsions (11,12). Functional recovery involves restoration of occupational performance, social participation and the significant reduction in time consumed by rituals that interfere with daily activities (2,11). Personal recovery refers to the individual's ability to experience intrusive thoughts without feeling compelled to act on them, developing greater tolerance of uncertainty and increased confidence in managing anxiety provoking situations (3,11,21). Social recovery may involve reduced reliance on family members to accommodate compulsive behaviours, improved openness about symptoms, and greater participation in interpersonal and social environments that were previously avoided (15).

Importantly, intrusive thoughts may not disappear entirely. Many individuals with OCD continue to experience occasional intrusive thoughts but they gradually learn that these thoughts do not require a behavioural response. Through exposure-based learning, patients repeatedly encounter feared situations without performing compulsions, allowing them to observe that the feared consequences do not occur. Over time, this process weakens catastrophic interpretations of intrusive thoughts and strengthens the ability to tolerate uncertainty (21).

Longitudinal studies indicate that, while full remission occurs in some individuals, many experience partial remission with periods of improvement and occasional symptom recurrence, particularly during times of stress (11,12). In this sense, recovery in OCD often involves learning to manage symptoms effectively and maintaining functional stability rather than eliminating all intrusive thoughts.

Stigma can also influence recovery. Individuals with OCD may hide their symptoms because of shame or fear of being misunderstood, especially when obsessions involve taboo, aggressive, sexual, religious or socially sensitive themes (11,21). This concealment may delay treatment and increase social isolation. Reducing stigma and helping patients understand intrusive thoughts within an OCD framework can support both personal recovery and social reintegration (3,11).

Overall, recovery in OCD can be understood as a gradual process in which maladaptive fear responses weaken, inhibitory control improves and individuals regain the ability to participate in daily life without being dominated by compulsive rituals (11,21).

5.4 Transdiagnostic Mechanism of Recovery Across Psychiatric Disorders

Although, psychiatric disorders are traditionally described as distinct diagnostic categories, the literature reviewed in this thesis suggests that several recovery mechanisms operate across multiple conditions. This thesis examines ADHD, BPD, PTSD, OCD, and ASPD, which demonstrate different symptom profiles, yet share underlying psychological and behavioural processes that influence recovery trajectories (49).

One recurring mechanism across disorders is the strengthening of emotional regulation capacities (50). Difficulties in regulating affect are evident in several conditions examined in this thesis. In BPD, emotional dysregulation contributes to impulsive behaviour and interpersonal instability, while in PTSD dysregulated fear responses and hyperarousal maintain avoidance and intrusive symptoms (16,34). Similarly, individuals with ADHD often demonstrate heightened emotional reactivity and difficulty modulating frustration (25,26). Across these disorders, recovery frequently involves the development of strategies that increase tolerance of emotional states and improve regulatory control over behavioural responses (17,50).

A second shared process involves cognitive restructuring and modulation of maladaptive beliefs. Cognitive models suggest that psychiatric disorders are maintained by distorted interpretations, maladaptive schemas and rigid threat appraisal patterns. In OCD, catastrophic misinterpretation of intrusive thoughts reinforces compulsive rituals, while in PTSD maladaptive trauma-related beliefs sustain avoidance and hypervigilance (5,17,21). Psychotherapeutic interventions such as cognitive behavioural therapy aim to modify these cognitive patterns and promote more flexible interpretations of internal experiences and environmental cues (6).

Another important dimension of recovery is behavioural adaptation and executive compensation. In neurodevelopmental conditions such as ADHD, recovery rarely involves the complete disappearance of

attention vulnerabilities. Instead, individuals frequently develop compensatory strategies that support organisation, planning and sustaining goal-directed behaviour. Similarly, behavioural regulation strategies are central to treatment approaches in BPD and PTSD, where individuals learn to replace impulsive or avoidance-based responses with more adaptive coping behaviours (7,17,34).

Finally, social and interpersonal functioning emerge as shared components of recovery across disorders. Difficulties in relational stability and trust are particularly prominent in BPD, PTSD and ASPD. Recovery, therefore, often includes an improved capacity for maintaining relationships, increased social participation and reduced reliance on maladaptive interpersonal strategies. These improvements reflect not only symptom reduction but also broader psychological adaptation (3,17,34,47,48,61).

Transdiagnostic research increasingly demonstrates that shared psychological mechanisms underlie diverse psychiatric conditions. Core processes such as emotional regulation, cognitive flexibility, and executive functioning deficits appear consistently across disorders including ADHD, BPD, PTSD, OCD and ASPD, supporting a unified model of recovery grounded in adaptive regulatory processes rather than disorder-specific symptom reduction (49,50,54).

6 Discussion

The purpose of this thesis was to examine the concept of recovery in mental disorders within a multidimensional biopsychosocial framework and to explore how recovery processes manifest across selected psychiatric conditions. The literature review demonstrates that recovery in psychiatry cannot be understood solely in terms of symptom remission. Instead, recovery represents a complex and individualised process that involves psychological adaptation, functional reintegration and social participation.

One of the most consistent findings across the disorders examined in this thesis is the multidimensional nature of recovery. Traditional clinical models of psychiatric improvement have primarily emphasised symptom reduction as the primary indicator of treatment success. However, the literature increasingly highlights that clinical remission alone does not necessarily correspond to full recovery. Individuals may experience significant improvement in daily functioning, social relationships and personal well-being even when residual symptoms remain. Conversely, symptom stabilisation may occur without a full restoration of social or occupational functioning. These observations support contemporary recovery-oriented frameworks that conceptualise recovery across multiple domains, including clinical stabilisation, functional capacity, personal identity restructuring and social reintegration.

Despite the heterogeneity of the psychiatric disorders examined in this thesis, several common recovery mechanisms appear across diagnostic categories. Emotional regulation emerges as a central factor influencing recovery trajectories in many conditions. Disorders such as BPD, PTSD and ADHD involved varying forms of dysregulated emotional responses, including affective instability, impulsivity, hyperarousal and anxiety-driven behavioural patterns. Therapeutic interventions frequently target these regulatory processes through the development of coping strategies, cognitive restructuring and behavioural regulation skills. Treatments such as cognitive behavioural therapy, dialectical behavioural therapy and trauma-focused therapy demonstrate that strengthening emotional regulation capacities may facilitate broader improvement in functioning and psychological stability across different psychiatric conditions.

Another theme emerging from the literature is that recovery often reflects adaptive compensation rather than complete symptom elimination. Many psychiatric conditions involve enduring vulnerabilities related to neurodevelopmental factors, personality traits or trauma-related adaptation. As a result, recovery frequently involves the development of strategies that allow individuals to manage ongoing difficulties while maintaining stable functioning in daily life. For example, individuals with attention deficit hyperactivity disorder may develop organisational and behavioural strategies that compensate for executive functioning deficits, while individuals with obsessive compulsive disorder may learn to tolerate intrusive thoughts without engaging in compulsive behaviour. Similarly, individuals with personality disorders may gradually strengthen emotional regulation and interpersonal functioning through structured psychotherapy. This pattern suggests that recovery in psychiatry often involves adaptive restructuring rather than the complete disappearance of symptoms.

Neurobiological research further supports the concept of recovery as a dynamic adaptive process. Studies examining the effects of psychotherapy and behavioural interventions indicate that recovery may be associated with the changes in neural circuits involved in emotional regulation, executive functioning and threat processing. Increased regulatory activity within prefrontal cortical regions and improved modulation of limbic structures have been observed following successful therapeutic interventions. These findings suggest that the psychological treatment may facilitate neuroplastic changes that support improved emotional regulation and behavioural control. Such neurobiological adaptations reinforce the view that recovery involves gradual restructuring of psychological and neural systems rather than simply representing the reversal of a pathological state.

The role of social and environmental factors also emerges as a significant component of the recovery process. Recovery does not occur solely within the individual, but it is also shaped by interpersonal relationships, social support networks and broader societal conditions. Access to effective treatment, a stable social environment and supportive relationships may facilitate recovery trajectories, while stigma, social isolation and structural barriers to care may hinder recovery. These findings underscore the importance of integrating social and environmental considerations into recovery-oriented psychiatric care.

Although this thesis provides an overview of recovery mechanisms across several psychiatric disorders, it is important to recognise the limitations inherent in the scope and methodology of the review. The analysis focused on a selected group of disorders, including ADHD, BPD, PTSD, ASPD and OCD. Other major psychiatric conditions, such as schizophrenia, bipolar disorder and major depressive disorder, were not included within the scope of this thesis. Therefore, the findings cannot be generalised to all mental disorders.

Another limitation is that this thesis was conducted as a narrative literature review and did not include original empirical data collection. Consequently, the conclusions are based on interpretation and synthesis of existing literature rather than direct measurement of patient recovery outcomes. In addition, recovery is defined differently across studies, which makes direct comparison between disorders and treatment outcomes more complex.

Despite these limitations, the literature reviewed in this thesis supports the view that recovery from mental disorder is best understood as a dynamic, multidimensional and adaptive process influenced by biological, psychological and social factors. Recovery may involve symptom stabilisation, functional improvement, psychological adaptation and social reintegration, rather than complete symptom elimination alone. Recognising recovery as a dynamic and individualised process could lead to the development of more comprehensive and person-centred approaches to psychiatric treatment.

7 Conclusions

This thesis examined the concept of recovery in mental disorders within a multidimensional biopsychosocial framework and explored how recovery processes manifest across selected psychiatric conditions. The literature reviewed demonstrates that recovery in psychiatry cannot be defined solely as the absence of symptoms. Instead, recovery represents a dynamic and individualised process that involves psychological adaptation, functional improvement and reintegration into social and occupational roles.

Across the disorders examined in this thesis, including attention deficit hyperactivity disorder, borderline personality disorder, post-traumatic stress disorder, antisocial personality disorder and obsessive-compulsive disorder, several common recovery mechanisms were identified. Emotional regulation, cognitive restructuring, the development of adaptive coping strategies and supportive social environments appear to play a central role in facilitating recovery. These mechanisms suggest that despite the differences in diagnostic categories, many psychiatric conditions share underlying processes that influence recovery trajectories.

The findings of the thesis also indicate that recovery frequently involves adaptive compensation rather than complete symptom elimination. Individuals may continue to experience certain vulnerabilities while developing strategies that enable stable functioning and improved quality of life. This perspective reflects a shift from traditional symptom-centred models of psychiatric treatment towards more holistic and person-centred approaches that emphasise functional and social well-being.

Overall, the literature supports the view that recovery and mental health is best understood as a multidimensional process shaped by biological, psychological and social factors. Recognising the complexity of recovery may contribute to the development of more integrative treatment approaches that address not only symptom reduction but also the broader aspect of psychological functioning and social participation.

Future research should explore recovery trajectories across additional psychiatric conditions and investigate how transdiagnostic mechanisms could lead to more personalised treatment approaches.

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