Childhood Trauma and Psychopathology

Investigating the Role of Social Functioning, Emotional Processing, and Sleep in Post-Traumatic Stress Disorder and Borderline Personality Disorder

Inaugural-Dissertation

zur Erlangung des Doktorgrades der Philosophie

an der Ludwig-Maximilians-Universität München

Fakultät für Psychologie und Pädagogik

Lehrstuhl für Klinische Psychologie und Psychotherapie

vorgelegt von

Isabelle Paulina Göhre

aus Wiesbaden, Deutschland

München, 2026

Erstgutachterin: Prof. Dr. Katja Bertsch

Zweitgutachter: Prof. Dr. Thomas Ehring

Tag der mündlichen Prüfung: 06. Februar 2026

Childhood Trauma and Psychopathology

Investigating the Role of Social Functioning, Emotional Processing, and Sleep in Post-Traumatic Stress Disorder and Borderline Personality Disorder

Danksagung

Ich möchte mich von tiefstem Herzen bei allen bedanken, die mich während meiner Promotion unterstützt haben und ohne die diese Arbeit nicht möglich gewesen wäre.

Zunächst gilt mein größter Dank meiner Doktormutter, Prof. Dr. Katja Bertsch, die mich vom ersten Tag an durch ihre hervorragende Betreuung unterstützt hat. Danke, dass du mich stets ermutigt hast, gedanklich einen Schritt weiterzugehen und mir hohe Ziele zu setzen. Du hast mir immer den Rücken gestärkt, hattest immer ein offenes Ohr und Zeit, um Ideen zu durchzusprechen, und mir während der Promotion sehr viele Freiheiten ermöglicht. Als Wissenschaftlerin warst du mir durch dein kritisches und logisches Denken, deine Neugier und Leidenschaft für die Forschung sowie deine Geduld und unermüdliche Ausdauer ein sehr großes Vorbild. Zugleich hattest du viel Empathie und Verständnis für die Herausforderungen während der Promotion und hast mich durch deine Expertise und klinische Erfahrungen betreut. Dadurch hast du mich sowohl in meinem beruflichen Werdegang als auch in meiner persönlichen Weiterentwicklung maßgeblich geprägt.

Ich möchte mich auch aufrichtig bei Prof. Dr. Thomas Ehring für seine Ko-Betreuung, seine wertvollen Ideen und sein großes Engagement bedanken. Ebenso danke ich Prof. Dr. Ellen Greimel für die Übernahme der Begutachtung und für die aufgewendete Zeit.

Ich möchte mich auch herzlich bei meinen Kolleginnen und Kollegen des Lehrstuhls für Klinische Psychologie und Psychotherapie der LMU München bedanken. Vielen Dank für die stets unterstützende und angenehme Arbeitsatmosphäre, den offenen Austausch und die zahlreichen wertvollen Anregungen. Ein besonderer Dank gilt Mia, Milena, Sarah und Aleya, die mit ihrer positiven Art und viel Humor so oft Leichtigkeit in herausfordernde Tage gebracht haben.

Ich möchte mich auch ganz besonders bei allen Patientinnen und Studienteilnehmerinnen bedanken, die sehr bedeutende Einblicke in ihre persönlichen

Erfahrungen gegeben haben. Vielen Dank für Ihre Offenheit und Ihr großes Vertrauen in die Forschung.

Ein herzliches Dankeschön gilt meinen Freundinnen und Freunden, die mich auf diesem Weg immer wieder motiviert und aufgebaut haben. Ein besonderer Dank geht an Caro und Alessandro für eure unglaubliche Unterstützung und euren Rückhalt in jeder Phase dieser Zeit. Ebenso danke ich allen, die mich während des Studiums, der Promotion und der Ausbildung zur Psychotherapeutin begleitet haben. Vielen Dank an Kathi, Rike, Mia, Meggy, Anna, Marie, Laurie, Lars und Claire für eure Freundschaft, eure Aufmunterung und all die gemeinsamen Momente, die diesen Weg so viel leichter und schöner gemacht haben.

Ein ganz besonderer Dank gilt meiner geliebten Familie, die in allen Höhen und Tiefen der vergangenen Jahre stets an meiner Seite war. Ihr hattet immer ein offenes Ohr, habt mich mit liebevollen Worten gestärkt und mir Halt gegeben. Keine Worte können meine tiefe Dankbarkeit dafür ausdrücken. Meinem Bruder Marcel danke ich von Herzen. Du bist für mich seit jeher ein großes Vorbild durch deine Geradlinigkeit und grenzenlose innere Stärke und du hast mir geholfen, über mich hinauszuwachsen. Meiner Mutter danke ich für ihre unerschütterliche Zuversicht, die mir während der Promotion viel Mut und Kraft gegeben hat. Mein besonderer Dank gilt meinem Vater. Du hast mir durch deine bedingungslose Warmherzigkeit, Geduld und Empathie das Selbstvertrauen gegeben, um zu promovieren.

List of Scientific Publications and Author Contributions

Study I

Göhre, I., Vogel, L., Brückl, T., Hupe, H., Janina Wollinger, J., Wolkenstein, L., Aleksic, M., Merscher, A., Semm, A., Claus, N., Goerigk, S., Kopf-Beck, J., Binder, E., Padberg, F., Reinhard, M.A., Ehring, T., Bertsch, K. (2025). Old Scars Don't Hurt? A Systematic Review on the Association between Child Maltreatment and Adult Social Functioning.

Accepted for publication on 20 March 2026 in the Journal of Trauma, Violence, & Abuse. This chapter is a pre-copyedited version of a submitted article.

Author contributions: F.P., T.E., and K.B. developed the overall research question and review design. Title- and abstract screening was performed by KB, TB, TE and IG. Full-text screening, data extraction and a narrative synthesis were performed by all authors. I.G. and K.B. drafted the manuscript. All authors provided critical revisions and approved the final version of the manuscript for submission.

Study II

Göhre, I., Back, S., Schütz-Bosbach, S., Ren, Q., Wolkenstein, L., Rupp, A., & Bertsch, K. (2025). Psychological and Neurophysiological Measures of Emotion Dysregulation in Borderline Personality Disorder and Posttraumatic Stress Disorder. *Borderline Personality Disorder and Emotion Dysregulation*, *12*(1), 37.

This chapter is based on the post-peer-review, pre-copyedited version of an article published in Borderline Personality Disorder and Emotion Dysregulation. The final version of the publication can be accessed at https://doi.org/10.1186/s40479-025-00313-3

Author contributions: IG and KB developed the current research question. However, the overall study concept and design were developed by SB and KB. Data collection was performed by IG and SB. The data analysis and interpretation were performed by IG, KB, QR, SB and LW. IG drafted the manuscript. All authors provided critical revisions and approved the final version of the manuscript for submission.

Study III

Göhre, I., Back, S., Weiß, M., Ehring, T., Herpertz, S.C., Schmahl, C., Wolkenstein, L., Bertsch, K. (2025). Fear of the Night: Links Between Emotion Dysregulation, Sleep Disturbances and Childhood Trauma Across Trauma-Related Disorders.

Submitted for publication in the Journal of Aggression, Maltreatment & Trauma. This chapter is a pre-copyedited version of a submitted article.

Author contributions: IG and KB developed the current research question. However, the overall study concept and design were developed by SB and KB. Data collection was performed by SB and IG. The data analysis and interpretation were performed by MW, KB and IG. IG drafted the manuscript. All authors provided critical revisions and approved the final version of the manuscript for submission.

General Abstract

Traditional categorical systems provide useful diagnostic distinctions between mental disorders. However, they struggle to capture their high comorbidity, heterogeneity, and overlapping etiological pathways (Dalgleish et al., 2020). Consequently, a paradigm shift has been promoted toward dimensional and transdiagnostic frameworks that conceptualize psychopathology along a continuum of shared risk factors and mechanisms (Harvey et al., 2004). Within this perspective, *traumatic childhood experiences (TCE)* represent a key transdiagnostic risk factor for the development of various forms of psychopathology (Jaffee, 2017; Lewis et al., 2021; Walsh et al., 2017). TCE include emotional, physical, and sexual abuse and/or emotional and physical neglect (Bernstein et al., 2003).

Two trauma-related conditions that share a high prevalence of TCE are post-traumatic stress disorder (PTSD) and borderline personality disorder (BPD). PTSD and BPD share overlapping symptoms (e.g., emotion dysregulation, interpersonal dysfunction) and show disorder-specific profiles (e.g., sleep disturbances) (Hailes et al., 2019; Jowett et al., 2020; Krause-Utz, 2021). TCE might therefore operate as a *distal risk factor* for PTSD and BPD and might contribute to both shared symptoms and disorder-specific patterns by influencing more *proximal* mechanisms (Nolen-Hoeksema & Watkins, 2011). Research exploring the impact of TCE on shared biopsychosocial mechanisms in PTSD and BPD is scarce yet essential for advancing prevention and intervention strategies that address overarching TCE-related impairments. Therefore, in this thesis, the links between TCE and three possible mechanisms, namely, disturbances in *social functioning*, *emotion processing*, and *sleep*, were investigated in individuals with PTSD, BPD, as well as healthy controls in three complementary studies.

Study 1 investigated the transdiagnostic relationship between TCE and social functioning, and the mechanisms underlying this link, across adults with and without psychopathology through a systematic literature review. TCE were associated with various

impairments in social functioning, with mostly small to moderate effect sizes. The most robust links emerged with intimate partner violence (IPV), aggression, social connectedness, attachment, and early maladaptive schemas. Evidence for associations with sexuality, intimate partner relationships, and social-cognitive processes was less consistent. Moreover, the review identified psychopathology (including PTSD and BPD features), insecure attachment, early maladaptive schemas and emotion dysregulation as key mechanisms between TCE and social dysfunction. This motivated the subsequent examination of emotional processes as a further mechanism.

Study 2 examined the link between TCE and *emotional reactivity and regulation* cross-sectionally in women with PTSD, BPD, and healthy controls (HC). A multimethod experimental design was used that integrated self-report, behavioral, and neurophysiological measures (event-related potentials, ERPs). Across participants, higher TCE were correlated with emotional hypo-reactivity to positive (r = -.35, p < .001) and neutral (r = -.22, p = .011) stimuli, consistent with findings of emotional numbing after chronic trauma (Clarke et al., 2024; Sill et al., 2020; Wu et al., 2023). However, this association disappeared once group differences (PTSD, BPD, HC) and medication use were controlled for. Moreover, TCE were not correlated with neurophysiological variables of emotion processing or the effects of instructed emotion regulation (i.e., cognitive reappraisal). In contrast, TCE remained associated with self-reported emotion dysregulation (β = .24, p = .004) even after adjusting for group and medication use. These findings suggest that TCE might confer a more trait-like vulnerability to emotion dysregulation expressed across both disorders but might not strongly shape state-level reactivity to stimuli or the effectiveness of an instructed regulation strategy. Both overlapping and disorder-specific alterations emerged as individuals with PTSD showed greater emotional hypo-reactivity, whereas adults with BPD exhibited heightened global dysregulation and impulsivity.

Study 3 explored the association between TCE and past-month and past-night *sleep disturbances* across three complementary samples. These included treatment-seeking PTSD outpatients and two case-control samples of individuals with PTSD, BPD, and healthy controls. TCE were correlated with poor sleep quality, (nightmare distress), fear of sleep, reduced restorative sleep, pre-sleep psychological balance, increased exhaustion, and psychosomatic symptoms at bedtime (r = .26 to .56, p < .05). However, when emotion dysregulation was taken into account, the direct effect of TCE on sleep became non-significant. These findings might suggest that TCE may serve as a distal risk factor, influencing sleep indirectly through more proximal affective mechanisms. Emotion dysregulation was associated with poorer past-month sleep quality, (nightmare distress), fear of sleep and past-night sleep disturbances above and beyond hyperarousal, group and TCE (β = .21 to .37; p < .046). Individuals with PTSD and BPD overlapped on sleep disturbances. However, fear of sleep remained specifically elevated in PTSD, while emotion dysregulation appeared to largely account for sleep disturbances in BPD.

In conclusion, this thesis aimed to address the lack of research on the link between TCE and proximal mechanisms in trauma-related disorders such as PTSD and BPD. Findings across three studies indicated that TCE might act as a broad vulnerability factor, associated with disturbances in social functioning (Study 1), emotional processing (Study 2), and sleep (Study 3). Emotion dysregulation emerged as a central mechanism linking TCE to social impairment and sleep disturbance. At the same time, disorder-specific alterations after TCE were evident (e.g. fear of sleep in PTSD; impulsivity in BPD) that were in line with Ehlers & Clark's (2000) cognitive model of PTSD and Linehan's (1993) biosocial model of BPD. These patterns support integrating dimensional and categorical perspectives to develop comprehensive models of trauma-related psychopathology.

The results support mechanism-focused assessments in clinical practice and suggest that early stabilization of emotion regulation may help to mitigate downstream interpersonal and sleep disturbances. Depending on individual profiles, transdiagnostic treatments may be used next to disorder-specific interventions to target shared mechanisms (Bohus & Vonderlin, 2024; Cloitre et al., 2002). Limited by the cross-sectional design and focus on two disorders, future research should employ longitudinal, multimodal, and ecologically valid assessments and incorporate data-driven approaches (Goerigk et al., 2023; Huang et al., 2025). Moreover, future research should map mechanisms between TCE and psychopathology across multiple disorders. Specifically, features of complex PTSD (cPTSD) and levels of personality functioning might represent higher-order pathways through which TCE confer transdiagnostic vulnerability while shaping disorder-specific outcomes (Ford & Courtois, 2021; Kampling et al., 2022).

Table of Contents

1 General Introduction

1.1 Transdiagnostic Models of Psychopathology

Since the mid-19th century, different forms of mental health difficulties have been classified into diagnoses in a categorical approach based on distinct clusters of symptoms (Dalgleish et al., 2020). Up until today, this classification is exemplified by diagnostic systems like the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 2013) and the International Statistical Classification of Diseases and Related Health Problems (ICD; World Health Organization, 2019). By using a *lingua franca*, the DSM and ICD provide a framework to guide clinical research and facilitate diagnostic and treatment decisions worldwide.

However, these psychiatric nosologies have been widely criticised for failing to account for the complexity, comorbidity, heterogeneity and temporal instability of diagnoses inherent in psychopathology (Dalgleish et al., 2020). A large body of evidence suggests that, instead of arbitrary boundaries between "healthy" and "pathological", mental health processes unfold along a continuous spectrum (Haslam et al., 2012; Haslam et al., 2020). Consequently, a paradigm shift has emerged in favour of a transdiagnostic perspective on psychopathology (Harvey et al., 2004). In response, dimensional clinical models such as the Hierarchical Taxonomy of Psychopathology (HiToP; Kotov et al., 2018) and research approaches like the Research Domain Criteria (RDoC; Insel et al., 2010) have appeared. These models account for comorbidity as they facilitate the identification of core risk factors and mechanisms across biological, psychological, and social domains that underlie various forms of psychopathology (Harvey et al., 2004). A key advantage of these models is their potential to pinpoint transdiagnostic factors which may lead to a more effective prevention, assessment and treatment of a broad range of mental health conditions (Nolen-Hoeksema & Watkins, 2011).

Although the weight of empirical evidence favours dimensional frameworks of psychopathology (Haslam et al., 2020), categorical models remain useful for administration in

clinical practice and transitioning to dimensional approaches presents significant challenges. Moreover, categorical nosologies benefit from disorder-specific, theory-driven models that can be directly translated into treatment interventions, whereas transdiagnostic approaches lack metatheoretical models (Dalgleish et al., 2020). Accordingly, contemporary research increasingly seeks integrative frameworks that can explain shared and divergent illness trajectories.

To develop such an integrative account, research must identify (1) *risk factors* that cut across diagnostic boundaries and (2) *mechanisms* that explain both the overlap between disorders and disorder-specific alterations. This examination of relevant biopsychosocial risk factors and mechanisms should therefore be conducted both across and within diagnostic categories, as undertaken in this thesis. This dual approach enables the identification of disorder-specific features that warrant targeted interventions, while simultaneously revealing shared mechanisms that may benefit from transdiagnostic interventions (Hofmann & Hayes, 2019). The following section introduces *traumatic childhood experiences (TCE)* as a distal transdiagnostic risk factor for a broad spectrum of psychiatric outcomes, thereby operationalising the integrative agenda sketched above.

1.2 Childhood Trauma as Transdiagnostic Risk Factor for Psychopathology

Multifinality is a developmental concept describing how the same environmental factor can lead to different types of psychopathologies (Cicchetti & Rogosch, 1996). *Traumatic childhood experiences (TCE)* exemplify this principle, as they represent a significant risk factor for the development of various forms of psychopathology (Jaffee, 2017; Lewis et al., 2021; Walsh et al., 2017). Although multiple terms, such as interpersonal childhood trauma, adverse childhood experiences (ACEs), early life adversity (ELA) and childhood maltreatment (CM), are often used interchangeably, this thesis uses TCE to refer consistently to the same concept,

as defined below. TCE are defined as adverse interpersonal experiences of maltreatment occurring before the age of 18, which may involve physical, sexual, and/or emotional abuse, as well as physical and/or emotional neglect (Bernstein et al., 2003). Based on this definition, around 31% of adults in Germany report at least one type of TCE, with higher prevalences for physical and emotional neglect than abuse (Witt et al., 2017). As TCE are not only associated with mental but also with physical health conditions (e.g., cancer, diabetes, chronic pain), the annual economic burden linked to their long-term effects is estimated to be $428 billion in the U.S. (Klinger-König et al., 2024; Peterson et al., 2018). In light of this societal and individual burden and given that TCE are among the most influential yet preventable contributors to psychopathology, transdiagnostic prevention programs with early intervention have emerged as a crucial and effective strategy (Johnson-Motoyama & Davis, 2020).

TCE are a particularly potent risk factor for the onset, severity, as well as co-morbidity of disorders such as Post-Traumatic Stress Disorder (PTSD) and Borderline Personality Disorder (BPD) (Hailes et al., 2019; Jowett et al., 2020; Krause-Utz, 2021). Individuals with PTSD are approximately 1.8 times more likely to report TCE than individuals without a mental disorder (McLaughlin et al., 2017). Notably, more than one in four children exposed to interpersonal trauma are estimated to develop PTSD (Alisic et al., 2014). All types of TCE increase the risk of PTSD, with specifically strong effects of sexual, physical, and emotional abuse compared to neglect (Messman-Moore & Bhuptani, 2017; Widom, 1999). Overall, cumulative exposure to different types of TCE significantly heightens the risk of developing lifetime PTSD following subsequent traumas (e.g., accidents), with each additional TCE type associated with a 28% increase in risk (da Silva et al., 2024).

Similarly to PTSD, individuals with BPD are approximately 13 times more likely to have experienced one type of TCE than individuals without a mental disorder and 3 times more likely than adults with other mental disorders (Porter et al., 2020). Specifically, experiences of

emotional neglect and abuse are highly prevalent among individuals with BPD, next to high prevalences (62%) of childhood sexual abuse (Temes et al., 2020). Emotional maltreatment may play a critical role in the development of BPD (Kuo et al., 2015; Porter et al., 2020).

Building on these shared trauma pathways, individuals with comorbid PTSD-BPD report more chronic TCE, a higher likelihood and severity of childhood sexual abuse (e.g., penetration, intrafamilial), physical abuse and neglect, as well as poly-victimisation and earlier abuse onset (Jowett et al., 2020; Pagura et al., 2010). Kühner et al. (2025) found that emotional neglect (80%), emotional abuse (72%), and sexual abuse (59%) were highly prevalent in PTSD patients with comorbid personality disorder symptoms and linked to greater BPD symptom severity. The elevated risk for the emergence of PTSD and BPD after TCE persists throughout adulthood, also increasing the risk of re-victimisation (e.g. sexual abuse) across life (da Silva et al., 2024; Walker & Wamser-Nanney, 2023).

Taken together, previous research provides strong evidence that TCE are a transdiagnostic risk factor for mental disorders (Goerigk et al., 2023; Huang et al., 2025). This can be exemplified in PTSD and BPD, two disorders in which trauma plays a strong etiological role. But how do TCE shape the clinical presentations of PTSD and BPD? Despite shared etiological factors and overlapping symptoms, these disorders are categorically distinct in contemporary nosologies. The following section compares their diagnostic criteria, shared and unique symptoms and implications for transdiagnostic models of trauma-related psychopathology.

1.3 Categorical and Dimensional Perspectives on PTSD and BPD

From a *categorical perspective*, the DSM-5 classifies PTSD and BPD as distinct entities, each defined by specific criteria and associated with tailored, evidence-based treatments (American Psychiatric Association, 2013). PTSD is uniquely characterised by the requirement of exposure to a traumatic event (Criterion A), which may involve (actual/threatened) death,

(sexual) abuse, violence or injury which was either directly experienced, witnessed in person or experienced indirectly (e.g. through family/work) (American Psychiatric Association, 2013; Bisson, 2007). It is estimated that 1.9% (Europe) to 6.8% (US) of individuals develop PTSD after trauma exposure (Alonso et al., 2004; Kessler et al., 2005).

PTSD is characterised by four symptom clusters, namely: (B) intrusive re-experiencing of the trauma, (C) avoidance of trauma-related stimuli, (D) negative alterations in cognitions and mood and (E) alterations in arousal and reactivity (American Psychiatric Association, 2013). Of note, in the DSM-5, PTSD is more broadly conceptualised, whereas the authors of the ICD-11 distinguish between PTSD and complex PTSD (cPTSD) (World Health Organization, 2019). The latter refers to reactions following prolonged or repeated "complex" interpersonal trauma, such as chronic child abuse, genocide, sex trafficking and/or war-related trauma (Cloitre, 2020). The diagnosis of cPTSD according to ICD-11 requires symptoms from three PTSD clusters (B, C, E) and three clusters of disturbances of self-organisation. These are emotional numbing and dysregulation, self-perceptions as a failure or worthless and emotional detachment in relationships (World Health Organization, 2019). Throughout this thesis, all references to PTSD will adhere to the DSM-5 definition.

Three of the leading frameworks to explain PTSD (Brewin & Holmes, 2003) are the cognitive theory by Ehlers and Clark (2000), the dual representation theory by Brewin et al. (1996) and the emotional processing theory by Foa et al. (1989). These models align with first-line trauma-focused psychotherapies according to the APA and German S3 guidelines, such as Cognitive Behavioural Therapy (TF-CBT), Cognitive Processing Therapy (TF-CPT) or Prolonged Exposure (PE) (APA, 2025; Schäfer et al., 2019).

In *Ehlers and Clark's (2000) cognitive model*, a persistent threat perception is maintained by (a) negative appraisals of the trauma and its consequences (e.g. "*I am permanently damaged*", "*no one can be trusted*") and (b) fragmented, poorly contextualised trauma

memories weakly integrated into autobiographical memory. (c) Maladaptive behavioural strategies such as avoidance, suppression, rumination and safety behaviours perpetuate these processes and prevent disconfirmation of beliefs.

In contrast to PTSD, BPD does not require a traumatic event for diagnosis. Instead, it involves a pervasive, inflexible pattern of instability in self-image, relationships, and affect, present across contexts (Bradley et al., 2007). As a personality disorder, it typically emerges in adolescence or early adulthood and affects approximately 1.7% of the population (Gunderson et al., 2018). According to DSM-5, BPD is diagnosed if five out of the following nine criteria are met: abandonment reaction, unstable and intense relationships, identity disturbance, impulsivity, recurrent suicidal behaviour or self-harm, affective instability, chronic feelings of emptiness, inappropriate or intense anger, and paranoid ideation or dissociation (American Psychiatric Association, 2013). Key theoretical frameworks of BPD are the biosocial model of Linehan (1993), the mentalization theory by Fonagy and Bateman (2008) and the schema modes of Young et al. (2003). These models directly align with first-line treatments according to the APA and the German S3 guidelines, such as Dialectical Behaviour Therapy (DBT), Mentalization-Based Treatment (MBT) and Schema Therapy (DGPPN, 2022; Keepers et al., 2024; Linehan, 2014).

Linehan's (1993) biosocial model posits that BPD arises from the interaction between genetic predispositions to emotion sensitivity and exposure to TCE, particularly the invalidation of the child's emotional responses, as key drivers of BPD. A conflicting emotional bonding, with caregivers as a simultaneous source of threat and security, can lead to a disorganised attachment, in which a child varies between an impulse to seek closeness and a reflex to avoid it (i.e., approach-avoidance dilemma) (Holmes, 2004). This interaction between genetic vulnerabilities and TCE fosters emotion dysregulation, the disorder's core feature, and

impairs self- and interpersonal functioning (Carpenter & Trull, 2013; Daros & Williams, 2019; Linehan, 1993).

While the categorical approach highlights apparent differences in diagnostic thresholds, symptom structure, theoretical models and treatment modalities between PTSD and BPD, a *dimensional perspective* emphasises their substantial overlap (Knefel et al., 2016). First, co-occurrence rates range from 30% to 70% and are associated with increased clinical severity, including heightened suicidality, greater functional impairment, more complex comorbidities and reduced treatment compliance (Frías & Palma, 2014; Heffernan & Cloitre, 2000; Pagura et al., 2010; Scheiderer et al., 2015). Second, the disorders share substantial overlap in their clinical phenomenology, such as alterations in social functioning and emotional processing as portrayed in Table 1 (Ford & Courtois, 2021; Knefel et al., 2016; Río-Casanova et al., 2016). Given the high heterogeneity of both disorders, their symptom-level overlap is not surprising (Bradley et al., 2007; Bryant et al., 2023). Third, evidence that treatments of BPD can also alleviate PTSD symptoms and vice versa has spurred the development of transdiagnostic interventions, which aim to address both conditions simultaneously by targeting shared underlying mechanisms (Bohus et al., 2013; Harned et al., 2012). From a biological perspective, BPD and PTSD share genetic vulnerabilities, as FKBP5 gene variants, which regulate stress hormones (glucocorticoid receptor complex), are implicated in both disorders, particularly through gene-environment interactions with TCE (Martín-Blanco et al., 2016; Wang et al., 2018). These findings correspond with the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis in PTSD and BPD, alongside shared fronto-limbic network abnormalities, marked by increased limbic activity and reduced frontal engagement (Amad et al., 2019; Drews et al., 2019; Schumacher et al., 2019).

Taken together, PTSD and BPD share TCE as a risk factor, high comorbidity, symptom overlap, shared treatment efficacy and neurobiological similarities, yet they also differ in core

diagnostic features and therapeutic approaches. PTSD requires exposure to a traumatic event and is primarily treated with trauma-focused therapies, whereas BPD involves pervasive instability in self, affect, and relationships and is primarily treated with structured psychotherapies such as DBT or MBT. These insights underscore the need to integrate categorical and dimensional frameworks in the study of PTSD and BPD, which remains a limited approach to date (Ford & Courtois, 2021; Hood et al., 2024).

Moreover, these commonalities and distinctions point to the operation of *transdiagnostic mechanisms* in the aftermath of TCE. This pattern fits the transdiagnostic framework proposed by Nolen-Hoeksema and Watkins (2011). TCE can be conceptualised as a *distal* risk factor that shapes *proximal* biopsychosocial processes. These proximal mechanisms, in turn, funnel individuals toward specific disorders, accounting for both shared symptomatology and comorbidity between disorders (*multifinality*) as well as the development of specific disorders (*divergent trajectories*) (Nolen-Hoeksema & Watkins, 2011). Accordingly, delineating these proximal mechanisms is essential for advancing the understanding of pathways linking TCE to PTSD and BPD. Against this backdrop, three mechanisms that show symptomatic overlap and divergence between PTSD and BPD are alterations in social functioning, emotion processing and sleep. Table 1 maps core DSM-5 criteria onto these domains to visualize shared features and disorder-specific signatures.

Table 1

Overlap and Distinctions in PTSD and BPD: DSM-5 Symptoms Across Social Functioning, Emotion Processing and Sleep

	Post-Traumatic Stress Disorder (PTSD)	Borderline Personality Disorder (BPD)
Social functioning	– Detachment/ estrangement from others (D6) – Distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others (D2) – *Avoidance of trauma reminders, including e.g. people and conversations (C2)*	– Pattern of unstable, intense relationships characterized by alternating between extremes of idealization and devaluation (2) – Transient, stress-related paranoid ideation (9) – *Frantic efforts to avoid real or imagined abandonment (1)*
Emotion processing	– *Irritable behaviour & angry outbursts, typically with verbal or physical aggression toward people or objects (E1)* – Persistent negative emotional state (D3) – Intense/ prolonged psychological distress at exposure to trauma cues (B4) – Avoidance of feelings associated with traumatic events (C1) – Hypervigilance (E4) and exaggerated startle response (E5) – Persistent inability to feel positive emotions (D7) – Reckless or self-destructive behaviour (E.2)	– *Inappropriate, intense anger / difficulty controlling anger (8)* – Affective instability due to marked reactivity of mood e.g., intense episodic dysphoria, anxiety, or irritability (6) – Chronic feelings of emptiness (7) – Impulsivity in potentially self-damaging areas (4) – Recurrent suicidal behaviour, gestures, or threats, or self-mutilating behaviour (5)
Sleep	– Sleep disturbance e.g., difficulty falling / staying asleep or restless sleep (E6) – Recurrent distressing dreams of the trauma (B2)	--

Note. Diagnostic criteria are based on DSM-5 (APA, 2013). Italicized sections represent an overlap between alterations in social functioning and emotional processing

1.4 Transdiagnostic Mechanisms Linking Childhood Trauma with PTSD and BPD

A meaningful investigation of proximal transdiagnostic mechanisms should begin with an understanding of how the profound stress of TCE can alter biological, psychological, and social processes across the lifespan. Exposure to TCE occurs during a period of heightened brain plasticity, when the developing brain is being calibrated to the environment (Kolb & Gibb, 2014). Consequently, adverse experiences can profoundly alter neurobiological development, such as epigenetic processes, brain structure and function and the stress response systems (Cross et al., 2017). In childhood, persistent fear and anticipation of abuse can trigger a premature activation of stress-response pathways, such as cortico-limbic circuits and the HPA axis (McCrory et al., 2011; Nemeroff, 2004). As a result, the HPA axis has been found to exhibit an upregulated stress response after TCE, characterised by altered cortisol secretion and increased stress sensitivity and reactivity (Cattane et al., 2017; Herringa, 2017). Consequently, a heightened limbic reactivity and a reduced functional coupling of the medial prefrontal cortex (mPFC) and the amygdala have been reported in trauma-exposed children compared to those without exposure (Marusak et al., 2015; Wolf & Herringa, 2016).

These biological alterations may be advantageous in threatening environments by enabling the fast detection of threats through hypervigilance and by protecting the child's safety through the avoidance of danger. However, when the threatening or traumatic situation is no longer present, they can become maladaptive in the sense that they do not fit the context, are out of proportion and lack flexibility (McLaughlin & Lambert, 2017). Given the critical role of these brain structures in stress, emotion, and social processing, these alterations can profoundly affect core regulatory and relational processes in individuals with TCE.

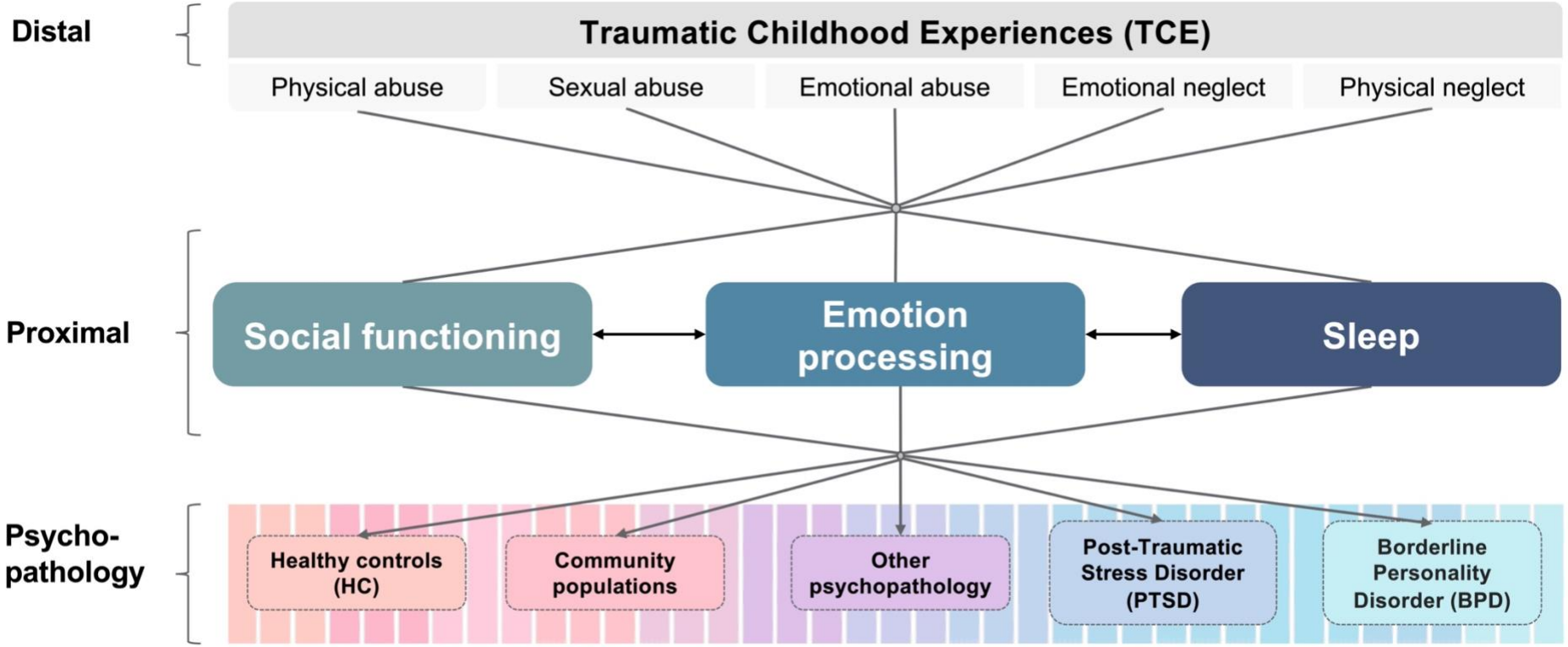
Given the multidimensional biopsychosocial impact of TCE, this thesis adopts a multimethod approach, integrating self-report, experimental and neurophysiological data to elucidate transdiagnostic processes. Proximal mechanisms will be defined as processes (a)

influenced by TCE, (b) altered in individuals with psychopathology relative to healthy controls, and (c) showing both shared and disorder-specific alterations (Harvey et al., 2004; McLaughlin et al., 2020). In three consecutive studies, this thesis concentrates on disturbances in *social functioning*, *emotion processing,* and *sleep*. These three mechanisms were selected based on the following converging considerations. First, they align with the RDoC framework, which guides transdiagnostic research by outlining domains relevant across disorders, including Social Processes, Negative/Positive Valence Systems, and Arousal/Regulatory Systems (Insel et al., 2010). Second, transdiagnostic models of TCE emphasise disruptions in social interaction, emotional processing, and sleep as key mechanisms of psychopathology (Harvey et al., 2011; Jaffee, 2017; Kajeepeta et al., 2015; McLaughlin et al., 2020; Pfaltz et al., 2022). Third, social dysfunction and emotion dysregulation represent areas of substantial symptomatic overlap between PTSD and BPD (see Table 1).

This thesis synthesises multimethod evidence on these three clinically relevant mechanisms through which TCE might shape overlapping and disorder-specific pathways in PTSD and BPD. PTSD and BPD serve as exemplary case conditions, both marked by high TCE prevalence and pronounced disturbances in these mechanisms and are therefore examined in focused detail within a broader transdiagnostic framework. Figure 1 illustrates the proposed relationships based on the current knowledge discussed in the following sections.

Figure 1

Theoretical Framework of Mechanisms Linking Childhood Trauma to PTSD and BPD



Note.

1.4.1 Childhood Trauma and Social Functioning

Extensive research indicates that impairments in *social functioning* serve as a key set of transdiagnostic mechanisms linking TCE to psychopathology (McLaughlin et al., 2020; Pfaltz et al., 2022). In this thesis, social functioning is defined as the capacities required to form and maintain mutually satisfying relationships (e.g., intimacy, empathy) and to fulfil social roles across family, peer, romantic, sexual, and vocational domains (Weissman, 1975; World Health Organization, 2019). TCE may disrupt social functioning via intertwined biological, psychological, and social changes observed across various clinical populations. TCE is associated with social-information biases, attachment insecurity and reduced social support, which undermines the development of enduring relationships (Cross et al., 2017; Cyr et al., 2010; McLaughlin et al., 2020; Pfaltz et al., 2022; Wang et al., 2021). Across diagnoses, these alterations may lead to deficits in *global social functioning* (e.g., aggression, loneliness) and *social cognition* (e.g., theory of mind, empathy) (Carr et al., 2020; Fares-Otero, De Prisco, et al., 2023; Haslam & Taylor, 2022; Pfaltz et al., 2022).

As presented in Table 1, social dysfunction is integral to the diagnostic criteria of PTSD and BPD, with significant overlap as well as partially distinct profiles (American Psychiatric Association, 2013; World Health Organization, 2019). In PTSD, social difficulties frequently centre on emotional detachment/ estrangement from others and trauma-related avoidance. Increased threat biases, insecure attachment (avoidance and anxiety) and lower social support have been reported after TCE in PTSD (Crow et al., 2021; Guay et al., 2006; Huang et al., 2020). Meta-analytic evidence indicates impairments across social roles (e.g., caregiving/parenting) and social cognition (i.e., theory of mind, affective empathy) as well as broader indices of social functioning (Couette et al., 2020; Nietlisbach et al., 2010; Scoglio et al., 2022).

In BPD, social dysfunction plays a central role in the aetiology and symptomatology of the disorder as described in the biosocial model of BPD (Lazarus et al., 2014; Linehan, 1993). Social functioning is broadly and chronically disrupted due to a disorganised/insecure attachment manifesting in unstable and intense relationships, heightened rejection sensitivity, and rapid shifts between idealisation and devaluation, often accompanied by impulsive responses to perceived abandonment (Brumariu et al., 2020; Schulze et al., 2022). Converging evidence suggests threat-focused biases, lower trust, and a tendency to attribute malevolent intent, alongside difficulties in social problem-solving, mentalizing and cognitive empathy (Lazarus et al., 2014). Unsurprisingly, global deficits in social functioning have been reported across various types of relationships in BPD, which affect the ability to adhere to social norms and roles and to access social support (Carlson et al., 2020; Schulze et al., 2022). Thus, whereas PTSD often presents with trauma-specific withdrawal, detachment and avoidance, BPD might be characterised by relationship instability and volatility spanning broader contexts.

Despite these robust links between TCE and social impairment in PTSD and BPD, several research gaps appear. Firstly, the specific relationships between TCE types (e.g., emotional neglect, physical abuse) and particular social deficits (e.g., relationship quality, social cognition) remain insufficiently understood (Pfaltz et al., 2022). Secondly, the transdiagnostic mechanisms (e.g., attachment insecurity, social support) that underlie the relationship between TCE and social functioning need to be investigated further (Fares-Otero et al., 2023; McNeil & Rehman, 2024; Shahab et al., 2021). Importantly, social dysfunctions are tightly interwoven with the ways TCE reshape *emotional experience,* as an accurate reading of others' emotions and intentions and adequate responding are essential for adaptive interpersonal functioning (Gross & John, 2003; Kim & Cicchetti, 2010; McNeil & Rehman, 2024). To trace this deeper layer of the cascade, the next section examines how TCE influence

emotion processing across transdiagnostic psychopathology and specifically in PTSD and BPD.

1.4.2 Childhood Trauma and Emotion Processing

Based on a wealth of research, alterations in *emotion processing* constitute a second set of transdiagnostic mechanisms that link TCE to psychopathology (Dvir et al., 2014; McLaughlin et al., 2020; Weissman et al., 2019). TCE may shape emotion processing via biopsychosocial alterations that affect two core processes: (i) *emotional reactivity* (response to negative cues/ stressors) and (ii) *emotion regulation* (flexibility in modulating experience, expression, and arousal to meet situational demands) (McLaughlin et al., 2020; Miu et al., 2022; Thompson, 1991). Emotion regulation is a transdiagnostic, multifaceted capacity that can be conceptualised through *global abilities* (e.g., emotional awareness, clarity, acceptance, impulse control, goal-directed behaviour) and the use of *strategies* at distinct stages of the emotion-generative process along Gross's Process Model (Cludius et al., 2020; Gratz & Roemer, 2004; Gross, 1998b, 2015). Emotion regulation strategies are typically categorised as *adaptive* (e.g., reappraisal) when they target the root of distress or yield durable relief and *maladaptive* (e.g., self-harm) when they are ineffective or carry long-term costs (Aldao et al., 2010; Cludius et al., 2020; Gross, 1998a; Martínez-Priego et al., 2024).

On a neurobiological level, TCE have been associated with amygdala hyperreactivity and reduced prefrontal control, which may contribute to increased emotional reactivity and impaired regulation (Dvir et al., 2014; McLaughlin et al., 2015). At the psychosocial level, invalidation of emotions and maladaptive modelling by caregivers may impair emotional awareness, differentiation and regulation (Assed et al., 2020; Bérubé et al., 2023; Flechsenhar et al., 2024; Milojevich et al., 2020; Miu et al., 2022). Consequently, TCE have been linked to a *heightened threat vigilance* (e.g., increased reactivity to angry faces) and a *hostile attribution*

bias (e.g., assuming greater hostile intentions of others) as mediators to psychopathology (Glaser et al., 2006; Hein & Monk, 2017; Heleniak et al., 2016; McLaughlin et al., 2015; Weissman et al., 2019; Weltz et al., 2016). Moreover, longitudinal studies and meta-analyses indicate that both global emotion dysregulation and overreliance on maladaptive instead of adaptive emotion regulation strategies (e.g., suppression, rumination and deficits in reappraisal) mediate the relationship between TCE and psychopathology (Heleniak et al., 2016; Kim & Cicchetti, 2010; Miu et al., 2022; Weissman et al., 2019). The next section compares PTSD and BPD on their heightened emotion reactivity and dysregulation after TCE, highlighting shared and disorder-specific alterations as displayed in Table 1.

Individuals with PTSD tend to experience heightened negative and reduced positive reactivity and an increased *threat sensitivity,* specifically to trauma-related cues. Moreover, elevated anger and aggression have been linked to an increased hostile attribution bias (Briggs-Gowan et al., 2016; Hébert et al., 2021; Javanbakht et al., 2011; Machlin et al., 2024; Masten et al., 2008; Zhu et al., 2020; Zukerman et al., 2023). Moreover, PTSD symptom severity is linked to global emotion dysregulation and greater reliance on maladaptive strategies (Christ et al., 2021; Lee et al., 2015; O'Brien et al., 2023; Seligowski et al., 2015). Converging evidence points to two profiles found in PTSD: a reexperiencing/ hyperarousal subtype characterised by *under-regulation* (e.g., anger, self-destruction; reduced PFC inhibition of limbic regions) and a dissociative subtype with *over-regulation* (e.g., avoidance, numbing; heightened PFC inhibition) (Lanius et al., 2010; Litz et al., 2002; Steuwe et al., 2012).

In BPD, emotion dysregulation is the disorder's core feature (Linehan, 1993). Heightened threat sensitivity and hostile attribution biases (e.g., misclassifying neutral/sad faces as angry) might foster views of others as malevolent/untrustworthy and rejection- or anger-related interpretations of ambiguous situations (Arntz et al., 2011; Bertsch et al., 2018; Fertuck et al., 2013; Lobbestael & McNally, 2016). Consequently, the rapid and intense

increases of negative affect might be context-dependent for trauma-related cues such as social rejection, abandonment or childhood abuse (Gratz et al., 2013; Limberg et al., 2011; Lobbestael & Arntz, 2015; Socci et al., 2024). Moreover, BPD is characterised by a marked *under-regulation* of emotions due to deficits in adaptive (e.g. reappraisal, acceptance) and a surplus of maladaptive strategies (e.g. impulsivity, aggression, suicidality, self-harm) (Carpenter & Trull, 2013; Daros et al., 2018; Daros & Williams, 2019; Sorgi-Wilson & McCloskey, 2022). The reduced emotion identification in BPD might yield polarised affect (i.e., good/ bad), while low distress tolerance prompts a more rapid use of maladaptive strategies according to the emotional-cascade model (Carpenter & Trull, 2013; Selby et al., 2009).

To summarise, although PTSD and BPD show heightened emotional reactivity and greater reliance on maladaptive strategies, disorder-specific profiles might diverge (Jowett et al., 2020; Messman-Moore & Bhuptani, 2017; Tull et al., 2020). In PTSD, hyperarousal-driven reactivity might be cue-bound to trauma reminders (e.g., physical threats) with alternations between over- and under-regulation (Ford & Courtois, 2021). In contrast, BPD might show interpersonal hyperreactivity to a wider range of ambiguous or socially threatening stimuli, more chronic under-regulation and a stronger reliance on maladaptive strategies such as self-harm (Jowett et al., 2020). Neurobiologically, both display amygdala hyperreactivity, but BPD tends to recruit prefrontal control less than PTSD, consistent with more persistent regulation difficulties (Ford & Courtois, 2021; Schulze et al., 2019).

To conclude, substantial research indicates that emotion dysregulation is an important transdiagnostic mechanism that significantly increases the risk of PTSD and BPD as well as the severity of symptoms following TCE (Burns et al., 2010; Messman-Moore & Bhuptani, 2017). However, evidence directly contrasting PTSD and BPD on emotion reactivity and regulation is limited (Dixon-Gordon et al., 2013; Kockler et al., 2017). Because self-report and laboratory results often diverge, comprehensive multimethod designs are required to determine

how TCE shapes common and disorder-specific emotion processes (Bortolla et al., 2020; Koenigsberg et al., 2009; Krause-Utz et al., 2019; Marissen et al., 2010; McRae, 2013; Popkirov et al., 2018; Woodward et al., 2015). Moreover, emotion processing does not fade when the lights go out; rather, it might be bidirectionally intertwined with nocturnal sleep disturbances after TCE (Palmer & Alfano, 2017). As the third transdiagnostic mechanism of this dissertation, the next section examines how TCE influences sleep disturbances and emotion processing across transdiagnostic psychopathology as well as in PTSD and BPD.

1.4.3 Childhood Trauma and Sleep

A growing body of evidence links TCE with subjective and objective sleep disturbances up to 50 years post-exposure (Chapman et al., 2013; Chapman et al., 2011; Karatzoglou et al., 2024; Lind et al., 2016; Sullivan et al., 2019). As chronic insomnia is highly prevalent (10%) and frequently co-occurs with the majority of psychiatric disorders, it is increasingly recognised as a causal, transdiagnostic driver of psychopathology (Fairholme et al., 2013; Harvey et al., 2011; NIH., 2005). Across the lifespan, sleep disturbances and emotion processing have been found to mediate the link between TCE and psychopathology via intertwined biopsychosocial pathways (Bader et al., 2007; Laskemoen et al., 2021; Liu et al., 2023; Schäfer & Bader, 2013; Spilsbury, 2009).

On a neurobiological dimension, TCE can upregulate stress systems as seen in HPA-dysregulation, cortisol rhythm changes and elevated arousal alongside limbic-prefrontal alterations (Agorastos & Olff, 2020; Cattane et al., 2017; Herringa, 2017). During sleep, these neural systems process emotional memories made during the day to support fear extinction and affective homeostasis. Firstly, the affective charge of daytime experiences is reduced, secondly the sensitivity of emotional brain networks is recalibrated for adaptive next-day reactivity (Gieselmann et al., 2019; Goldstein & Walker, 2014; Nielsen & Levin, 2007; Pace-Schott et

al., 2015). According to the hyperarousal theory of insomnia, overactive emotion circuits during the day, keep the brain in a state of hyperarousal and vigilance, which impairs the onset and continuity of sleep (Riemann et al., 2010; Semsar et al., 2021; Sinha, 2016). These sleep disruptions and sustained hyperarousal can, in turn, impair adaptive emotion regulation on the next day (Mauss et al., 2013; Palmer & Alfano, 2017).

On a psychological dimension, this elevated hyperarousal, which manifests along neurological, physiological, cognitive and emotional dimensions, is linked with rumination and difficulties regulating negative affect before sleep (Andrews & Hanna, 2020; Gupta & Sheridan, 2018; Levin & Nielsen, 2009; Palagini et al., 2017; Palmer & Alfano, 2017; van Trigt et al., 2025; Werner et al., 2020). This is evident in increased maladaptive sleep-related cognitions and emotions, such as *fear of sleep* and feelings of entrapment or hopelessness (Werner et al., 2021). *Fear of sleep* is defined as the fear of being dangerously vulnerable during sleep, including a fear of recurrent nightmares and loss of control (Werner et al., 2020). As fear of sleep can lead to poor sleep habits (e.g., avoiding bedtime) and safety behaviours (e.g., sleeping with lights on), it is thought to maintain poor *sleep quality* and *nightmares* over time (Short et al., 2018; Werner et al., 2021). Lastly, sleep can impact socio-emotional functioning by impairing the ability to recognise and express emotions and has been associated with increased social withdrawal and interpersonal conflicts (Ben Simon et al., 2020; Gordon & Chen, 2014). Consequently, the same biological perturbations that impair emotion processing after TCE may also erode sleep architecture, thus creating a shared pathway to later psychopathology. The next section examines sleep disturbances after TCE within PTSD and BPD by exploring shared versus disorder-specific features and the bidirectional links with emotion dysregulation.

Sleep disturbances are now considered hallmark features of PTSD as they are among the most prevalent symptoms (80–90% experience insomnia, 50–70% nightmares) and play a

role in role in the development and maintenance of the disorder (Sinha, 2016; Spoormaker & Montgomery, 2008). Alterations have been found on objective measures of sleep duration, efficiency and architecture and subjective measures of *sleep quality*, *nightmares* and *fear of sleep* (Harvey et al., 2003; Leskin et al., 2002; Zhang et al., 2019). While hyperarousal remains a central explanatory mechanism, converging work highlights emotion dysregulation as a coupled process that is bidirectionally linked with sleep disturbance in trauma-exposed and PTSD samples (Levin & Nielsen, 2009; Palmer & Alfano, 2017; Riemann et al., 2010; Semsar et al., 2021; Sinha, 2016).

In contrast to PTSD, sleep disturbances are not diagnostic criteria of BPD and have received little attention in clinical practice and research despite their high prevalence of 63% suffering from insomnia and 49% from nightmares (Hafizi, 2013; Selby et al., 2013; van Trigt et al., 2025; Vanek et al., 2021). Alterations have been found on objective measures of sleep continuity and architecture as well as subjective measures of *sleep quality*, *nightmares and fear of sleep,* independent of comorbid PTSD or depression (Harty et al., 2010; Schredl et al., 2012; Semiz et al., 2008; Winsper et al., 2017; Wood et al., 2015). In BPD, the main focus has been on the reciprocal relationship between sleep disturbances and core BPD symptoms of emotion dysregulation (e.g., affective instability, self-harm, impulsivity), which may reflect difficulties in downregulating hyperarousal and hyperreactivity (Grove et al., 2016; Jenkins et al., 2022; Selby, 2013; Socci et al., 2024; van Trigt et al., 2025). Within the biosocial model, TCE may precipitate sleep problems that interact bidirectionally with emotion dysregulation to potentiate BPD risk alongside environmental and neurobiological vulnerabilities (Morales-Muñoz et al., 2021).

To summarise, both PTSD and BPD show high rates of poor *sleep quality, nightmares and fear of sleep,* which appear to be bidirectionally coupled with emotion dysregulation via biopsychosocial pathways, including hyperarousal (Dolan et al., 2023; Messman et al., 2023;

Morales-Muñoz et al., 2021). However, sleep disturbances are only a central diagnostic criterion for PTSD and might reflect broader emotion dysregulation rather than trauma-specific processes in BPD (van Trigt et al., 2025). Although substantial evidence implicates sleep disturbance and emotion dysregulation as mechanisms linking TCE to BPD and PTSD, mechanistic research and direct comparisons remain scarce. Studies are needed to clarify shared versus disorder-specific relationships (Harvey et al., 2011; Karatzoglou et al., 2024).

1.5 Summary and Aims

Categorical systems (e.g., DSM and ICD) provide distinct diagnoses but struggle to capture the complexity, comorbidity and heterogeneity of mental disorders. This has prompted a paradigm shift to transdiagnostic frameworks, which conceptualise psychopathology along a continuum with shared risk factors and mechanisms. TCE are a key transdiagnostic risk factor for mental disorders, such as PTSD and BPD, two disorders with high TCE prevalence, with both shared features and distinct symptom patterns. Consequently, TCE might act as a *distal risk factor*, shaping *proximal mechanisms* that drive high comorbidity and shared symptomatology (*multifinality*) as well as disorder-specific outcomes (*divergent trajectories*). Research exploring the impact of TCE on shared biopsychosocial mechanisms in PTSD and BPD is scarce, yet essential for advancing prevention and intervention strategies that address overarching TCE-related impairments. Therefore, this thesis examines how TCE are linked with *social functioning*, *emotion processing*, and *sleep* as three potential mechanisms within and across PTSD and BPD.

To date, converging evidence indicates that TCE disrupt *social functioning* across mental disorders through processes, such as insecure attachment, reduced social support and social information biases. In PTSD, social deficits appear to be more trauma-specific, involving avoidance and detachment, while BPD may exhibit broader interpersonal instability. However,

specific relationships between TCE subtypes (e.g., emotional neglect, physical abuse) and particular social deficits (e.g., relationship quality, social cognition), as well as their underlying mechanisms, remain unclear.

Moreover, social dysfunctions are tightly interwoven with the ways TCE reshape *emotional processing*. Following TCE, both PTSD and BPD report heightened emotional reactivity and dysregulation, including greater reliance on maladaptive emotion regulation strategies. Preliminary evidence, across self-report, behavioural, and neurophysiological measures, indicates both overlap and disorder-specific alterations. Individuals with PTSD tend to show a hyperreactivity to trauma-related cues (e.g. physical threats) along with alternations between under- and over-regulation. Individuals with BPD might show broader hyperreactivity to interpersonal threats (e.g., rejection) and more chronic under-regulation of emotions. However, multimethod comparisons of how TCE relate to emotion reactivity and regulation in PTSD versus BPD remain scarce and are urgently needed.

Following TCE, disruptions in emotion processing might be bidirectionally linked to *sleep disturbances* such as insomnia, nightmares and fear of sleep. Sleep disturbances are a hallmark of PTSD and prevalent in BPD yet are less systematically studied in the latter. Furthermore, there is a lack of research exploring differences in sleep disturbances between PTSD and BPD, as well as their associations with emotion dysregulation and TCE.

Despite evidence linking TCE to disruptions in social functioning, emotion processing, and sleep, multimethod studies examining these mechanisms and their interplay in PTSD and BPD are lacking. This thesis addresses these gaps through three complementary studies with the following goals.

(1) The first study aimed to explore the transdiagnostic association between TCE (overall and subtypes) and distinct domains of adult social functioning and identify mechanisms (e.g., attachment) within this link. To achieve this aim, a systematic review was

conducted that included cross-sectional, longitudinal and case-control studies in adults with and without psychopathology, synthesising transdiagnostic associations as well as disorder-specific findings (e.g., PTSD and BPD).

(2) The second study used a cross-sectional experimental design to compare emotional reactivity and regulation in PTSD, BPD, and healthy controls (HC) to distinguish shared from diagnosis-specific patterns. The study aimed to investigate the explanatory value of TCE beyond traditional diagnostic boundaries to explain alterations in emotion processing. The multimethod design integrated self-report measures with a laboratory emotion-regulation task with subjective affect ratings and neurophysiological markers (i.e., event-related potentials) recorded during passive viewing and cognitive reappraisal of emotional stimuli.

(3) Finally, the third study combined three cross-sectional studies to examine whether TCE are associated with sleep disturbances (sleep quality, fear of sleep, nightmares) beyond emotion dysregulation and whether alterations in sleep and the emotion regulation–sleep link can be found across PTSD and BPD. This study used self-report measures of sleep (past-month and night), emotion dysregulation, hyperarousal and TCE in multi-group samples (sample 1: PTSD outpatients, sample 2 and 3: PTSD, BPD, HC).

While the three studies are predominantly cross-sectional and therefore preclude causal conclusions, their findings may offer insights to guide the generation of hypotheses. They may help to identify potential mechanisms for longitudinal evaluation.

2 Cumulative Studies of this Thesis

Study I:

Old Scars Don’t Hurt? A Systematic Review on the Association between Child Maltreatment and Adult Social Functioning

Göhre, I., Vogel, L., Brückl, T., Hupe, H., Janina Wollinger, J., Wolkenstein, L., Aleksic, M., Merscher, A., Semm, A., Claus, N., Goerigk, S., Kopf-Beck, J., Binder, E., Padberg, F., Reinhard, M.A., Ehring, T., Bertsch, K. (2025). Old Scars Don’t Hurt? A Systematic Review on the Association between Child Maltreatment and Adult Social Functioning.

Accepted for publication on 20 March 2026 in the Journal of Trauma, Violence, & Abuse. This chapter is a pre-copyedited version of a submitted article.

Old Scars Don't Hurt? A Systematic Review on the Association between Child Maltreatment and Adult Social Functioning

Isabelle Göhre*[1,2], Lea Vogel[1,2], Tanja Brückl[1,3], Kerstin Hupe[1,3], Janina Wollinger[1,3], Larissa Wolkenstein[1,2], Milena Aleksic[2], Alma-Sophia Merscher[4], Anamaria Semm[2], Nathalie Claus[2,5], Stephan Goerigk[1,6,7], Johannes Kopf-Beck[1,2], Elisabeth Binder[1,3], Frank Padberg[1,6], Matthias A. Reinhard[1,6], Thomas Ehring[1,2], Katja Bertsch[1,4]

[1]German Center for Mental Health (DZPG), Partner Site Munich Augsburg

[2]Department of Psychology, Ludwig-Maximilians-Universität München, Munich, Germany

[3]Max Planck Institute of Psychiatry, Munich, Germany

[4]Julius-Maximilians-Universität, Würzburg, Germany

[5]Institute of Psychology, University of Bremen, Bremen, Germany

[6]Department of Psychiatry and Psychotherapy, LMU University Hospital, Ludwig-Maximilians-Universität München, Munich, Germany

[7]Department of Psychology, Charlotte Fresenius Hochschule, Munich, Germany

* Corresponding Author: Isabelle Göhre, M.Sc., Dept of Psychology, LMU Munich, Germany, Leopoldstr. 13, 80802 Munich, Tel: 089-21805199, E-Mail: isabelle.goehre@psy.lmu.de

Acknowledgements: Not applicable

Author contributions: F.P., T.E., and K.B. developed the overall research question and review design. Title- and abstract screening was performed by KB, TB, TE and IG. Full-text

screening, data extraction, and a narrative synthesis were performed by all authors. I.G. and K.B. drafted the manuscript. All authors provided critical revisions and approved the final version of the manuscript for submission.

Statements and Declarations: Not applicable

Declaration of conflicting interest: We have no conflict of interest to disclose.

Funding: I.G., L.V., T.E., L.W, J.K, K.H., J.W., S.G., M.R., F.P, E.B., T.B and K.B. are supported by the German Center for Mental Health (DZPG), funded by the Federal Ministry of Education and Research (Bundesministerium für Bildung und Forschung [BMBF]) and the Ministry of Bavaria (01EE2303A) and the current review was part of the DZPG project MUC-1 Early Trauma.

Data availability: The extracted data supporting this review's findings are available on request from the corresponding author.

Abstract

Social impairments are commonly reported by individuals with a history of child maltreatment (CM). However, a systematic review on the relationship between different types of CM and impairments across various domains of social functioning in adults with and without mental disorders that also addresses mediating and moderating factors is still missing. Therefore, we systematically searched the PsycINFO and Web of Science databases for peer-reviewed studies published from 2003 to February 2025 that examined the association between CM before the age of 18 and various clinically relevant impairments in social functioning in adulthood (≥18 years). A total of 281 articles were included and clustered in seven domains of social functioning. Most studies revealed negative associations between CM and social functioning, with small to moderate effect sizes. The strongest evidence was found for intimate partner violence (IPV), aggression, social connectedness, attachment, and early maladaptive schemas, while findings for other domains - such as sexuality, intimate partner relationships, and social-cognitive processes - were less consistent. Psychopathology, insecure attachment, and emotion regulation were the most frequently identified mediating or moderating factors. Taken together, CM and social dysfunctions need to be assessed and considered more systematically in psychotherapeutic treatment across disorders. Furthermore, longitudinal studies with standardized measures of social functioning are needed to identify transdiagnostic as well as disorder-specific targets for mechanism-based preventions and interventions for individuals with CM experiences.

Keywords: child maltreatment, abuse, neglect, interpersonal functioning, social functioning

Introduction

Social functioning is a central determinant of well-being across the general population. Impairments in social functioning can have debilitating effects on multiple domains of life and are common in many mental disorders. The significance of impaired social functioning is acknowledged in the ICD-11, where they are regarded as one of the two key domains in the assessment of the severity of personality disorders (World Health Organization, 2019). Similarly, impairments in social functioning are part of to the diagnostic criteria of Post-Traumatic Stress Disorder (PTSD) and complex PTSD (American Psychiatric Association, 2013; World Health Organization, 2019), but also prominent in other mental disorders, such as affective, anxiety, or psychotic disorders (Keltner & Kring, 1998). Identifying risk factors, as well as potential mediators and moderators that may explain which social impairments are experienced in whom and why, is therefore of high clinical relevance.

Being the most significant single risk factor for mental disorders and physical illness later in life (Jaffee et al., 2017; Klinger-König et al., 2024), childhood maltreatment (CM) has also been proposed to contribute to impairments in social functioning. According to Bernstein et al. (2003), CM is defined as the experience of emotional, physical, or sexual abuse or emotional or physical neglect before the age of 18 (McLaughlin et al., 2020).

In fact, there is an expanding body of research indicating diverse social deficits in individuals with a history of CM, both in non-clinical (Pfaltz et al., 2022) and clinical populations, such as borderline personality (BPD), PTSD, affective or psychotic disorders (Fares-Otero et al., Aversa et al., 2014; Cotter et al., 2015; 2023). These social impairments not only hinder the use or availability of social support, a key factor in resilience following trauma (Carr et al., 2020; McLaughlin et al., 2020), but also diminish the effectiveness of evidence-based psychological interventions contributing to reduced treatment success, prolonged treatment duration, and higher dropout rates (Dinger et al., 2013; Quilty et al., 2013;

Tibi et al., 2019). The growing body of evidence highlighting social impairments associated with CM stands in stark contrast to the limited attention such dysfunctions have received in etiological models and psychotherapeutic approaches to CM-related disorders to date.

A central aim of our project is to improve the knowledge about social functioning of individuals exposed to CM. Both CM and social functioning represent broad and multidimensional constructs, encompassing a wide range of experiences and processes. As a first step, we are therefore interested to identify which specific types of CM are linked with which specific social deficits as well as to examine possible moderators and mediators of these associations in adulthood. Importantly, our focus spans the full spectrum of mental health functioning, including both clinical and non-clinical populations. Given the conceptual breadth of this topic and the heterogeneity of studies, a systematic review of the literature appeared the most promising way allowing a balance between systematic synthesis and clinical knowledge in a broad and widespread field of research (Pfaltz et al., 2022). Following the ICD-11 definition of impairments in social functioning in personality disorders, we define social functioning as comprising: (1) the core skills needed to initiate and sustain mutually fulfilling interpersonal relationships, encompassing intimacy, empathy, and conflict resolution (World Health Organization, 2019) and (2) the ability to apply these skills to effectively fulfill one's roles in various relationships, such as those in work, friendships, partnerships, and family (Weissman, 1975). This broad definition was deliberately adopted to map the existing literature across social functioning domains and to generate hypotheses and identify priorities for future research.

Three earlier reviews and meta-analyses have addressed partly overlapping topics (Carr et al., 2020; Fares-Otero, De Prisco, et al., 2023). However, all of them had a much narrower scope by focusing on specific age groups (adolescents), particular mental disorders (depression), or psychosocial adjustment defined very broadly without focusing on specific

deficits. All three reviews suggest that CM is related to social impairments, highlighting the relevance of the topic addressed in the current article. However, our review extends these earlier works by (a) adopting a transdiagnostic perspective, (b) assessing subtypes of CM and (c) diverse clinically relevant social deficits as well as (d) focusing on mediating and moderating factors of the CM-social deficit-association. Therefore, in the current pre-registered systematic review, we addressed two key questions:

1. Which associations between CM and social functioning have been observed? We focused on social functioning in adult clinical and non-clinical samples and explored the relationship between different domains of social functioning and CM in general as well as its subtypes.

2. Which general and specific factors moderate or mediate the association between CM and social functioning?

Methods

Search Strategy

We followed the guidelines set by the Preferred Reporting Items for Systematic Reviews and Meta- Analyses (PRISMA). A protocol for the review was preregistered on the International Prospective Register of Systematic Reviews (PROSPERO) on January 30, 2024, with the registration number CRD42024504187. The protocol is available at: https://www.crd.york.ac.uk/prospero/display_record.php?ID=CRD42024504187

On December 27, 2023, a systematic search was conducted in the databases PsychINFO and Web of Science for peer-reviewed studies published in English after 2003, which explored the relationship between CM before the age of 18 and impairments in social functioning in adulthood (≥18 years). A supplementary literature search was performed to ensure the inclusion of studies published up to February 10, 2025. The 2003 cut-off was selected based on the publication year of Bernstein et al.'s definition of CM that was also used in the current study.

According to Bernstein et al. (2003), CM was defined as experiences of emotional, physical, or sexual abuse or of emotional or physical neglect before the age of 18 years. Social functioning was conceptualized according to multiple frameworks and perspectives, including the ICD-11 personality disorder framework (World Health Organization, 2019) of interpersonal dysfunction which encompasses the desire for relatedness and perspective-taking, mutual relationship quality and conflict management. This was further complemented by aspects of impaired social functioning emphasized in diverse clinical models (e.g., detachment, loneliness, mistrust, rejection sensitivity), such as the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2018), Schema Therapy (Young et al., 2003) and the Cognitive Behavioral Analysis System of Psychotherapy (CBASP; McCullough, 2006). Additionally, the selection process was guided by clinical observations of impaired interpersonal symptoms linked to early trauma, as synthesized by independent clinical scientist-practitioners. Although search terms were intentionally broad, inclusion decisions were guided by whether outcome measures captured an explicit interpersonal component based on our definition in the introduction. The search strategy can be found in the supplementary material.

Eligibility Criteria

Studies were eligible for inclusion if they met the following criteria: (1) a quantitative analysis of longitudinal, experimental, cross-sectional, or clinical trial data; (2) inclusion of a validated psychometric measure of emotional abuse, emotional neglect, physical abuse, physical neglect, and/or sexual abuse; (3) inclusion of a validated psychometric measure of social impairment; (4) participation of individuals with and/or without mental disorders from both community and specialized settings; (5) participants aged 18 years or older at the time of the outcome measurement, with exposure to CM occurring before the age of 18 (including retrospective reports); (6) ethical approval and documentation of written informed consent as

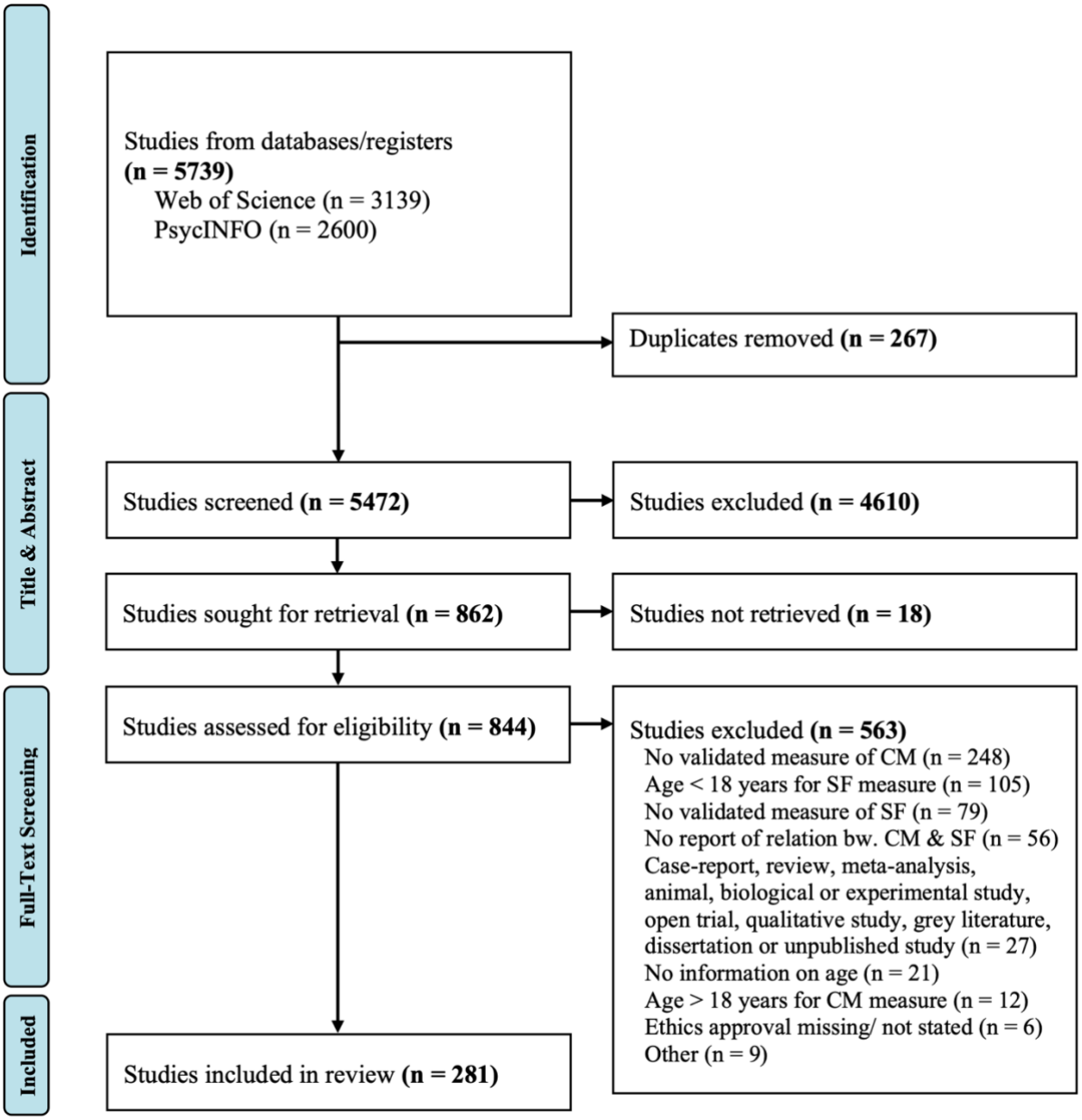
stated in the published article. Excluded were case reports, reviews, meta-analyses and animal studies, open trials, qualitative studies, grey literature, dissertations, and unpublished studies.

Data Extraction and Synthesis

As displayed in the flowchart (Figure 1), a total of 281 publications were included in the review. The web-based collaboration software platform Covidence (Veritas Health Innovation, 2024) was used for duplicate removal, title and abstract screening (conducted by KB, TB, TE and IG), as well as full-text screening and data extraction (by all authors). Due to the extensive scope of the review, dual screening and data extraction by multiple reviewers were not feasible. The following information was extracted from the articles: authors, publication year, country, type of population, study aim, study design, descriptives (age, gender, diagnoses), validated measures of CM and social functioning, reported statistics (e.g., correlation/ regression coefficients, odds ratios) and identified moderators or mediators. When studies reported effect sizes, this interpretation was extracted as presented. If no interpretation was provided and correlation coefficients were reported, effect sizes were classified using conventional thresholds by Cohen (1992) ($r > .10$ = small/weak; $r > .30$ = medium/moderate; $r > .50$ = large/strong) to allow for consistent reporting across studies. Given the heterogeneity of social functioning outcomes, we categorized them into seven domains, in line with previous reviews (Fares-Otero et al., Carr et al., 2020; 2023; Haslam & Taylor, 2022): (1) Social connectedness and social-cognitive processes; (2) attachment and early maladaptive schemas; (3) sexuality; (4) aggression, violence, and offending; (5) intimate relationships; (6) intimate partner violence (IPV) and revictimization; and (7) parenting behavior. For each domain, the study findings were summarized in a narrative synthesis based on the statistical data.

Figure 1

PRISMA Flowchart of the Systematic Review Procedure



Study Characteristics

The 281 included publications presented data from a total of 263,016 participants. Sample sizes varied, ranging from 36 to 25,778 participants. However, it is important to note that the exact total sample size cannot be reliably determined, as data from the same cohorts (i.e., MIDUS, RYDS, MTurk) or studies was likely used across sixteen publications (Abajobir

et al., 2017; Fitzgerald & Gallus, 2020; Fitzgerald & Morgan, 2022; Gallagher et al., 2023; Kisely et al., 2024; Lassri & Gewirtz-Meydan, 2025; Lassri et al., 2023; Smith et al., 2005; Smith et al., 2013; Widom, 2024; Widom et al., 2014; Widom et al., 2008; Widom et al., 2006; Wong et al., 2019; Zamir & Lavee, 2014; Zamir & Lavee, 2015). The studies were conducted across a diverse range of countries, which can be grouped by continent as follows: *America* [United States (n=151), Canada (n=14), Peru (n=1)]; *Europe* [Germany (n=15), the United Kingdom (n=7), Italy (n=6), Spain (n=6), Norway (n=5), the Netherlands (n=3), Portugal (n=2), Switzerland (n=2); Finland (n=1), Belgium (n=1), Hungary (n=1)], *Middle East* [Israel (n=14), Iran (n=4), Lebanon (n=1)]; *Africa* [South Africa (n=1), Cameroon (n=1), Kenya (n=1), Ethiopia (n=1), Botswana (n=1), Uganda (n=1)]; *Asia* [China (n=12), South Korea (n=6), Turkey (n=8), Taiwan (n=2), the Philippines (n=1), Japan (n=1), Bangladesh (n=1), Indonesia (n=1)] and *Oceania* [Australia (n=9)]. The age of participants in these studies ranged from 18 to 89 years, with a weighted mean age of 28.90 years (SD=9.62), based on the studies that reported age data. The studies included community populations (n=128 studies), psychiatric patients (n=44), healthy controls (n=11) as well as forensic (n=25) or other populations (n=98). Across all studies, 57.04% of participants were female, 42.83% were male and 0.13% identified as diverse.

The association between CM and social functioning was predominantly examined using correlational models, with 62 studies (22.06%) relying exclusively on correlations. Other statistical approaches included regression analyses (34.88%), t-tests, ANCOVAs, or MANCOVAs (12.10%), odds ratios (OR), risk ratios (RR), or hazard ratios (HR) (11.74%), chi-squared tests (3.56%), and multilevel modeling (MLM) (1.07%). Mediation or moderation analyses were conducted in 94 studies, predominantly based on cross-sectional data (84.04%), with only fifteen studies utilizing longitudinal data. CM was most frequently assessed

retrospectively using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003) in 47.26% of studies.

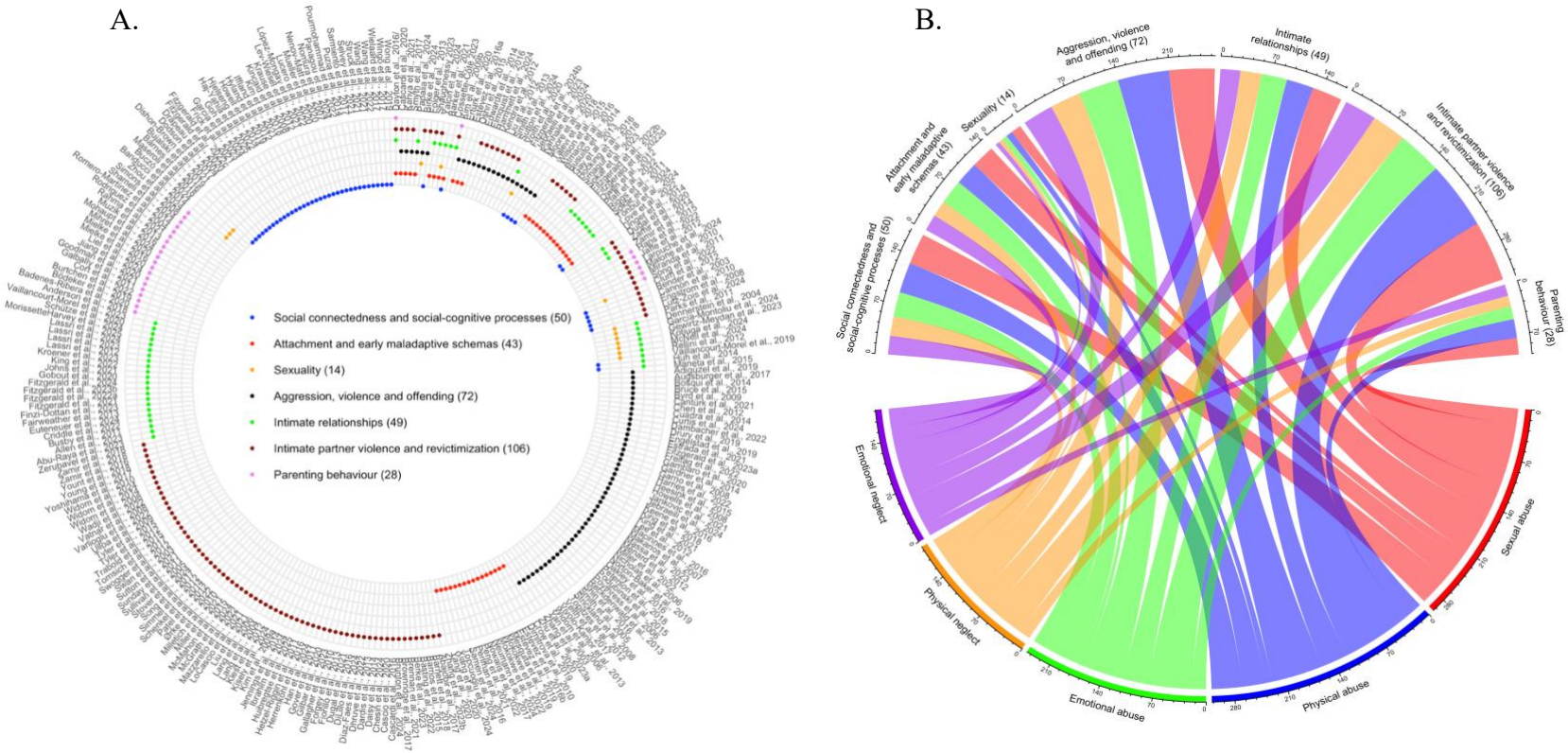
Results

As outlined above, we organized the results into seven categories, which align with classifications used in previous reviews and meta-analyses (Fares-Otero et al., Carr et al., 2020; 2023). However, these categories remained broad, encompassing studies with conceptually diverse constructs and measures. We summarized the associations of CM with (1) social connectedness and social-cognitive processes, (2) attachment and early maladaptive schemas, (3) sexuality, (4) aggression, (5) intimate partner relationships, (6) intimate partner violence (IPV) and revictimization, and (7) parenting behavior.

Comprehensive details of all included studies are available in Table S.1 on the Open Science Framework (OSF), accessible via the following link (under the 'Files' tab): https://osf.io/6qcun/?view_only=e753d74927c44cb09db231ee4747f480. Figure 2A shows the distribution of social functioning domains examined across studies, with IPV and revictimization being the most frequently studied domain of social functioning (n=106), while sexuality emerged as the least explored domain (n=14). Figure 2B visualizes the associations explored between different types of CM and various domains of social functioning in the included studies. Note that in most studies, CM was assessed as an overall measure that combined multiple types, meaning most associations do not correspond to specific forms of CM. Physical abuse (87.54%) and sexual abuse (83.63%) were the most commonly examined forms of CM, whereas emotional abuse (67.62%), emotional neglect (52.67%), and physical neglect (53.02%) were assessed less frequently.

Figure 2

Domains of Social Functioning Explored in each Study (A) and Explored Associations Between Types of CM and Domains of Social Functioning (B)



Note. The graph starts clockwise with studies that explored multiple domains of social functioning (A). The thickness of the lines represents the number of studies investigating each association (B).

Social connectedness and social-cognitive processes

Fifty studies investigated the relationship between CM and social connectedness as well as social-cognitive processes.

Social Connectedness

Social connectedness refers to an individual's sense of belonging within a social network, encompassing feelings of closeness, mutual support, and meaningful relationships (Lee & Robbins, 1995). It was most commonly measured using questionnaires that assess social support (e.g., Perceived Social Support Scale; Procidano & Heller, 1983), the size of social networks (e.g., Lubben Social Network Scale; Lubben, 1988), or loneliness (e.g., UCLA Loneliness Scale; Russell, 1996).

Social Network and Social Support. Thirteen cross-sectional studies and four longitudinal studies consistently demonstrated a small to moderate negative correlation between CM and social support, as well as smaller social networks. The majority of these studies was conducted among North American college students, although similar findings were also reported in clinical and non-clinical populations and forensic samples. Individuals with CM reported lower perceived support from both family and friends. Whereas different types of CM were not systematically compared, emotional neglect, emotional abuse, and physical neglect showed higher correlations with low social support than sexual or physical abuse. Among these, emotional neglect demonstrated a particularly strong negative association with social connectedness to family members.

Loneliness and Lack of Belongingness. Moderate positive associations have been identified between both loneliness as well as a lack of belongingness and CM in six cross-sectional and one longitudinal study. These links were observed in both community samples and clinical populations, including individuals with persistent depressive disorder (PDD) or BPD. Among all CM types, emotional abuse and neglect demonstrated the strongest

associations with loneliness and thwarted belongingness in both clinical and non-clinical samples. Physical abuse showed a small positive association with thwarted belongingness, while sexual abuse was not significantly related in a healthy sample. Survivors of childhood sexual abuse reported less mattering to parents but showed no difference in their sense of mattering to friends than participants without the experience of abuse.

Social-Cognitive Processes

This paragraph includes studies on various social competencies and social-cognitive and -emotional processes, such as empathy and theory of mind, which were assessed using a range of different methods.

Social Competences. Overall, the findings of eleven cross-sectional and one longitudinal study revealed a significant association between CM and reduced social competences, which encompassed aspects such as decreased social adjustment and prosocial activities, as well as increased withdrawal, emotional sensitivity, need for approval, a lack of sociability as well as more domineering and intrusive behaviors. This association was observed across diverse populations, including university students, adults with and without low birth weight, and patients with schizophrenia, schizoaffective disorder, bipolar disorder, personality, mood and anxiety disorders but not in healthy controls and veterans with PTSD and/or depression.

Empathy and Theory of Mind. physical abuse was associated with cognitive, but not affective, empathy deficits in both community and forensic samples. However, a study with a mixed sample (i.e., clinical and non-clinical) reported the opposite effect, while no significant correlation between CM and empathy was found in a community sample. In adult patients with PDD compared to those with episodic depression or healthy controls, general CM was associated with heightened empathic distress and interpersonal problems, with medium to large effect sizes. In sum, the nine cross-sectional studies exploring the relationship between CM

and empathic concern or perspective-taking (Theory-of-Mind) have yielded highly inconsistent results.

Social Expectations and Interactions. Evidence from five cross-sectional and two longitudinal studies suggests that individuals with experiences of CM may carry negative expectations and mistrust into social interactions, which can shape both their perceptions of others and the way they interact with them. For instance, individuals with CM history may have a heightened desire to feel comfortable in social settings yet simultaneously expect to receive less social support. They may also experience more social embarrassment and social anxiety. However, one study found no link between CM and fear of evaluation. In social interactions, individuals with CM may encounter more critical reactions from others and face higher levels of social rejection from peers.

Attachment and Early Maladaptive Schemas

Anxious and Avoidant Attachment

According to attachment theory, a secure attachment with an attachment figure (e.g., caregiver/ partner) is marked by a comfort with emotional closeness and intimacy. In contrast, insecure attachment styles manifests as either (a) anxiety, with fear of abandonment and preoccupation with intimacy, or (b) avoidance, marked by emotional distance and a discomfort with closeness (Bowlby, 1982). A total of thirty-three cross-sectional studies and two longitudinal studies examined the relationship between CM and attachment, with the Experiences in Close Relationships Scale (ECR) (ECR; Wei et al., 2007) being the most commonly used measure.

The studies revealed significant positive associations, ranging from weak to strong, between CM and both anxious and avoidant attachment styles. Notably, the associations were generally stronger with emotional abuse than with physical or sexual abuse, especially in the case of anxious attachment. The strength of these associations appears to be influenced by

factors such as the abused individual's gender, with one study noting a slightly stronger relationship between emotional abuse and attachment in women than in men. Moreover, stronger associations were found between CM and attachment to caregivers or romantic partners compared to friends. No significant correlations were found between CM and secure attachment or close and dependent attachment.

Early Maladaptive Schemas (EMS)

According to Schema therapy (Young et al., 2003), early maladaptive schemas (EMS) are pervasive patterns of thoughts, emotions, and behaviors that develop in early childhood as a result of unmet core emotional needs. The schemas can be characterized into the five domains: Disconnection/Rejection, Impaired Autonomy, Impaired Limits, Other-Directedness, Overvigilance and Inhibition. In total, nine cross-sectional studies investigated the relationship between CM and EMS, with Young Schema Questionnaire (YSQ; Young & Brown, 1990) being the most commonly used assessment tool. These studies consistently found significant associations, with the most pronounced associations observed for emotional abuse/neglect. The "Disconnection/Rejection" schema domain, encompassing EMS such as social isolation, emotional deprivation, abuse and defectiveness, showed one of the strongest links to CM. Additionally, schemas related to failure and vulnerability to harm, both part of the "Impaired Autonomy" domain, also displayed notably strong associations with CM. In contrast, evidence concerning other domains, such as "Other-Directedness" was less conclusive.

Sexuality

Fourteen studies explored the relationship between CM and sexuality, with the majority focusing exclusively on female participants. Each study employed a different measurement approach, and six of these studies specifically examined the relationship between sexual and/or physical abuse and sexual outcomes.

Sexual Functioning and Well-being

The nine cross-sectional and two longitudinal studies examining the relationship between CM and adult sexual functioning found weak associations. Women with a history of CM reported lower levels of sex drive, sexual activities, sexual motivation, sexual communication as well as reduced arousal, fewer orgasms, and higher levels of sexual shame and sexual problems. Sexual satisfaction was negatively associated with CM in three studies, with weak effect sizes observed in both women and men.

Sexual Risk-Taking

In three cross-sectional and one longitudinal study, all forms of CM, with the exception of emotional neglect, were significantly associated with risky sexual behavior, such as unprotected sex with casual partners, compulsive sexual behavior (hypersexuality), partner concurrency, exchanging sex for drugs, and behaviors linked to HIV risk. The associations ranged from weak to moderate in strength, with the strongest effect sizes observed for sexual abuse.

Aggression, Violence and Offending

A total of seventy-two studies were identified, most of which utilized the Buss-Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992) to measure aggression and the Conflict Tactics Scale (CTS; Straus, 1979) to assess violence, or relied on data from official police records.

Aggression

The forty cross-sectional and five longitudinal studies, primarily involving community, psychiatric, or forensic samples, reported a positive association of weak to moderate strength between CM and general aggression. Although types of CM were not statistically compared in these studies, associations appeared strongest for emotional and physical neglect and abuse. Furthermore, the gender of both the abusive and abused individual seemed to influence the strength of the association. For example, emotional or sexual abuse by the father was linked to

higher aggression in women, while less maternal but higher paternal physical abuse was associated with increased aggression in men. Stronger correlations were found in populations exposed to additional stressors, such as veterans, refugees, and psychiatric patients, compared to community samples.

When examining distinct dimensions of aggression - such as physical aggression, verbal aggression, anger, and hostility - associations with CM appear to be most pronounced between physical abuse and physical aggression and the links tend to be stronger for anger and hostility than for verbal aggression. Furthermore, positive associations were observed across a wide range of outcome measures, including trait hostility, hostile cognitions, hostile attribution bias, hostile-dominant interpersonal style/problems, hostility following military deployment, trait anger, outward anger-related behaviors, trait physical and verbal aggression, appetitive and reactive aggression, impulsive/ premeditated aggression, psychological aggression, and even sexual aggression.

Violence and Offending

The association between CM and violent behavior, including violent sexual offending has been addressed in twenty cross-sectional and seven longitudinal studies. Significant positive correlations of weak to moderate strength have been reported between CM and various outcomes, including a lifetime propensity for violence, a history of violent (assaultive) behavior, violence risk or tendency, criminal thinking, the frequency of violent behavior and violent arrests, child-to-parent violence, elder abuse, sexual violence perpetration, and stalking. The heightened risk for violence after CM was corroborated by six large longitudinal studies.

Certain aspects of the abuse, such as its type, the cumulative number of adversities, or the re-experience of violence during adulthood, have been found to increase the risk for subsequent violence. Specifically, stronger associations with violence were found for physical, emotional, or sexual abuse compared to physical and emotional neglect. Violence and CM were

particularly correlated in individuals with mental disorders, such as schizophrenia, within forensic populations. The only six studies, however, in which no significant association between CM and aggression or violence was found, were carried out in forensic or psychiatric settings, suggesting the influence of additional risk factors for aggression and violence in these populations. The results regarding gender effects remain inconclusive, though a pattern has emerged linking the type of CM to the type of offending. Physical abuse showed a comparatively higher correlation with non-sexual violence, while correlations were higher between sexual abuse and sexual violence.

Intimate Relationships

A total of forty-nine studies were identified. The majority of the studies (thirty-one cross-sectional and six longitudinal) explored the relationship between CM and the quality or satisfaction with adult intimate relationships, typically using the Couple Satisfaction Index (CSI; Funk & Rogge, 2007) or the Dyadic Adjustment Scale (Sabourin et al., 2005). Overall, most of the studies were cross-sectional (n = 42) and included community samples (n = 29).

Relationship Satisfaction and Quality

Overall, twenty-six studies, mostly cross-sectional, found a negative correlation between CM and relationship satisfaction in adulthood. However, the effects were generally weak when considering general CM experiences. The strongest negative correlations were observed for emotional abuse, while results remained inconclusive for other forms of CM, such as physical abuse and sexual abuse, with two studies finding no significant correlation. Additionally, the results suggest a stronger association between CM and relationship satisfaction in women compared to men. The results of twelve studies indicate a negative association between experiences of CM and relationship quality (including subdimensions such as affection and love) in adulthood. Emotional abuse emerged as a predominant subtype of CM, while findings for other subtypes remained inconclusive or under-researched.

Problematic Behaviors in Intimate Relationships

Eleven cross-sectional and two longitudinal studies, involving community, undergraduate, forensic and other samples, reported weak to moderate associations between CM and various types of problematic behaviors in intimate partnerships. CM was linked to increased levels of openly expressed anger and aggression within the family, marital strain, directiveness, conflicts, hostility, as well as verbal aggression toward partners. Additionally, CM was related to a tendency to conceal distressing or negative information about oneself. CM was associated with reduced responsiveness, more negative communication patterns, lower commitment, lower marital support and more detachment and at the same time it was linked with higher dependency and rejection sensitivity in intimate relationships. Three studies also provided preliminary evidence suggesting that CM shows a weak to moderate association with perceiving one's partner more negatively and making negative attributions about their behavior.

Intimate Partner Violence and Revictimization

Intimate Partner Violence

Ninety-seven studies (seventy-five cross-sectional and twenty-two longitudinal) investigated the association between CM and intimate partner violence (IPV). Approximately three-quarters of these studies used the CTS to assess IPV with a primary focus on various domains of violence, such as physical, sexual, and emotional or psychological violence. In twenty-three studies, IPV was examined bidirectionally, while forty-eight studies focused on IPV *victimization* (violence from partner toward the study participant) and twenty-six studies on IPV *perpetration* (violence from study participant toward the partner). Thirty-nine studies focused exclusively on female participants while primarily examining IPV victimization, and eleven studies focused solely on male participants, mostly investigating IPV perpetration.

With the exception of four studies, all studies consistently reported at least a weak to moderate association between some form of CM and IPV. CM was found to be related to both

IPV victimization and perpetration in women and men. Notably, ten out of eleven prospective studies, that followed children and adolescents with CM experiences into adulthood, reported elevated rates of physical and/or psychological IPV victimization and/or perpetration. An epidemiological study examining specific effects of different types of CM on IPV in a nationally representative sample of over 25.000 U.S. adults confirmed that CM is related to more reciprocal intimate violence. So far, there is limited evidence for specific associations between CM types and the types of adult IPV (sexual, emotional, or physical) across studies. However, childhood sexual abuse appears to have an additional specific link with general IPV victimization in both women and men. There is some evidence that the associations between CM and IPV vary between men and women depending on the type of CM. Childhood sexual abuse and physical neglect appear to be more consistently associated with both victimization and perpetration in women, whereas emotional abuse may be a more relevant factor in men. The association between CM and IPV appears to be particularly high in populations exposed to multiple cumulative risks, such as veterans, parental mental and substance use problems, inter-parental violence, family conflicts, poverty, and socially disadvantaged neighborhoods.

Revictimization

Seven cross-sectional and three longitudinal studies consistently reported a significant positive association between CM and revictimization in adulthood. The findings were similar for male and female participants and appeared to be stronger in specific groups facing additional stressors, such as individuals who were deaf or hard of hearing or those living in communities with higher poverty rates. Some evidence suggests that childhood sexual abuse may be a stronger predictor of sexual and physical violence in adulthood compared to physical abuse. Certain characteristics of childhood sexual abuse, such as use of physical force or the age at which the abuse began, emerged as significant predictors of sexual revictimization.

However, exposure to any form of CM may have a stronger association with revictimization than the specific type of CM experienced.

Parenting Behavior

A total of twenty-eight studies examined the relationship between parental experiences of CM and subsequent parenting behavior. Most of the studies focused on one or two specific types of CM and utilized a variety of measures to assess parenting behavior, such as the Emotional Availability Scales (EAS; Pipp-Siegel & Biringen, 1998). Notably, only eight studies also included male participants (fathers).

Parental Sensitivity and Parental-Child-Bonding

Eleven cross-sectional and four longitudinal studies investigated the following links. The association between CM and parental sensitivity/ competence and/or parental-child-bonding was investigated in ten studies, parental reflective functioning in two studies and parental self-efficacy and maternal representation of the child were each investigated in one study. Results regarding CM and paternal sensitivity were inconsistent. Three studies reported no significant association, while four studies found a weak negative association between CM and maternal sensitivity, although this was not observed for emotional availability. Furthermore, CM was negatively related to parental reflective functioning/ meta-parenting and parenting self-efficacy. CM was also negatively associated with mother-infant-bonding and child-mother relationship quality. Additionally, pregnant women with experiences of physical neglect were more likely to exhibit distorted prenatal representations of the child characterized by inconsistency and unrealistic expectation. The studies did not compare different types of CM.

Risk for Child Maltreatment

The risk for CM has been examined in twelve cross-sectional studies and one longitudinal study. Despite variations in measurements and methodology, all but one study

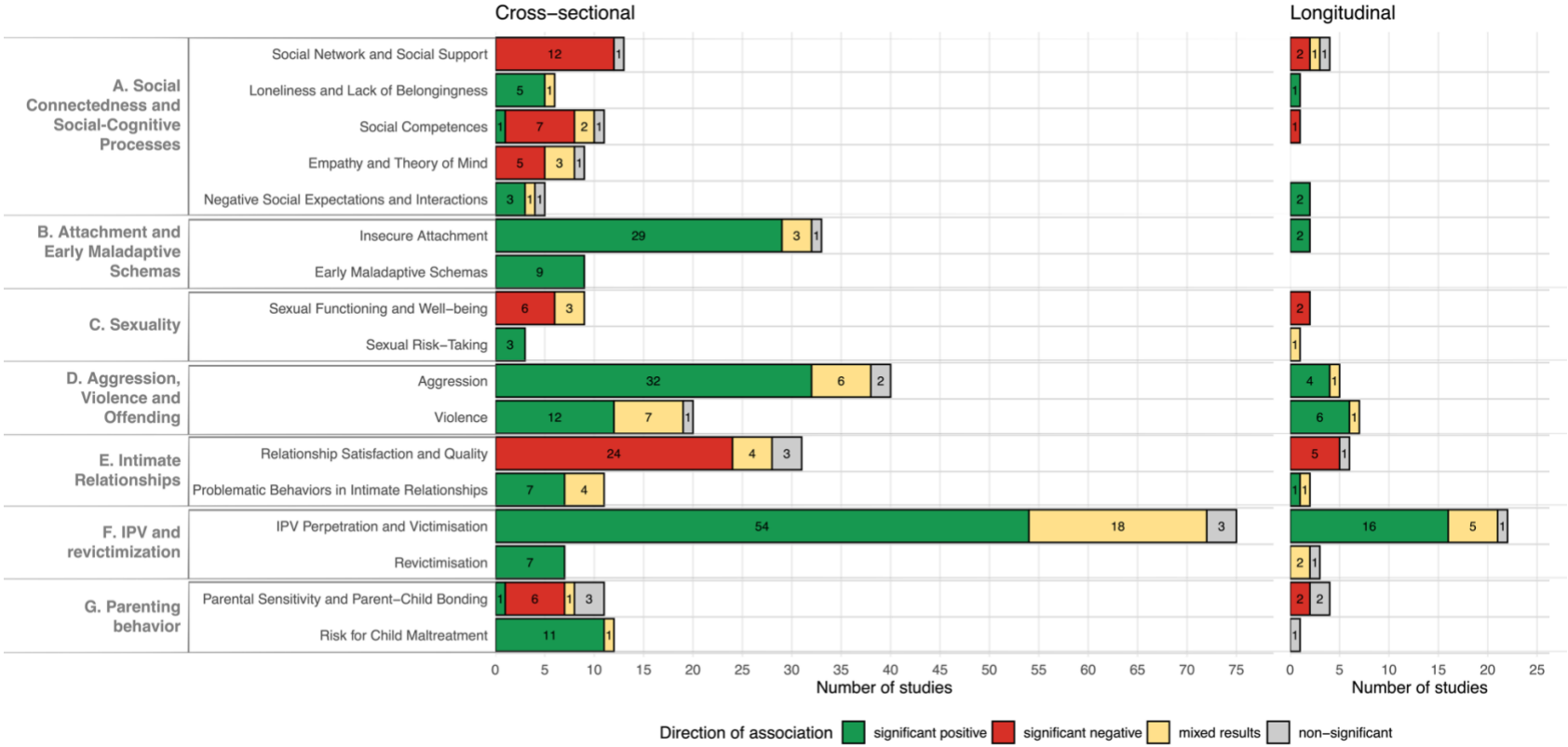
revealed a significant association between parental experiences of CM and an expected heightened risk of maltreatment in their offspring and maladaptive parenting practices and attitudes. Five studies found weak to moderate correlations between childhood sexual abuse and parental maltreatment behavior. Moreover, parents who had experienced physical abuse and/or emotional victimization were more likely to report positive attitudes toward physical discipline and an increased child abuse potential. Finally in a forensic sample, individuals convicted of child sexual offending were more likely to have experienced sexual abuse during childhood.

Summary of Main Findings

Overall, the majority of studies suggest a predominantly positive association between CM and impairments in multiple domains of social functioning. Using a harvest plot, Figure 3 displays the distribution of findings and direction of associations across the domains and study designs. The associations were generally of small to moderate strength and varied depending on both the type of CM and the social functioning outcome. As displayed in Figure 3, the strongest body of evidence emerged for the domains of IPV, aggression, social connectedness, attachment, early maladaptive schemas, and risk of CM. In contrast, the domains of sexuality, intimate partner relationships, parental sensitivity, and social-cognitive processes (i.e., empathy and theory of mind) have been less extensively studied and show less consistent findings.

Figure 3

Harvest Plot of Associations Between Childhood Maltreatment and Social Functioning Outcomes Across Study Designs



Note. Bars represent the number of studies reporting significant positive, significant negative, mixed, or non-significant associations between childhood maltreatment (CM) and social functioning (SF) outcomes. Mixed results refer to studies in which associations were observed only for specific subgroups (e.g., patients vs. healthy controls), specific outcome components (e.g., cognitive but not emotional empathy), or specific CM subtypes (e.g., childhood sexual abuse but not childhood physical abuse)

Mediators and Moderators of the Link Between CM and Social Functioning

In the following, we will summarize mediators and moderators of the relationship between CM and specific domains of social functioning identified in research to date. Table 1 provides a structured overview of all identified mediators and moderators, clustered into three overarching categories: psychological processes, interpersonal patterns, and contextual factors. Moreover, we report the number of cross-sectional and longitudinal studies for each SF domain, given the limitations of cross-sectional data for mediation and moderation modelling. The most frequently identified mechanisms are presented in Figure 4. Further details regarding all mediators and moderators are provided on the OSF in Table S.1 (see link).

Table 1

Mediators and Moderators Linking Childhood Maltreatment and Social Functioning Domains

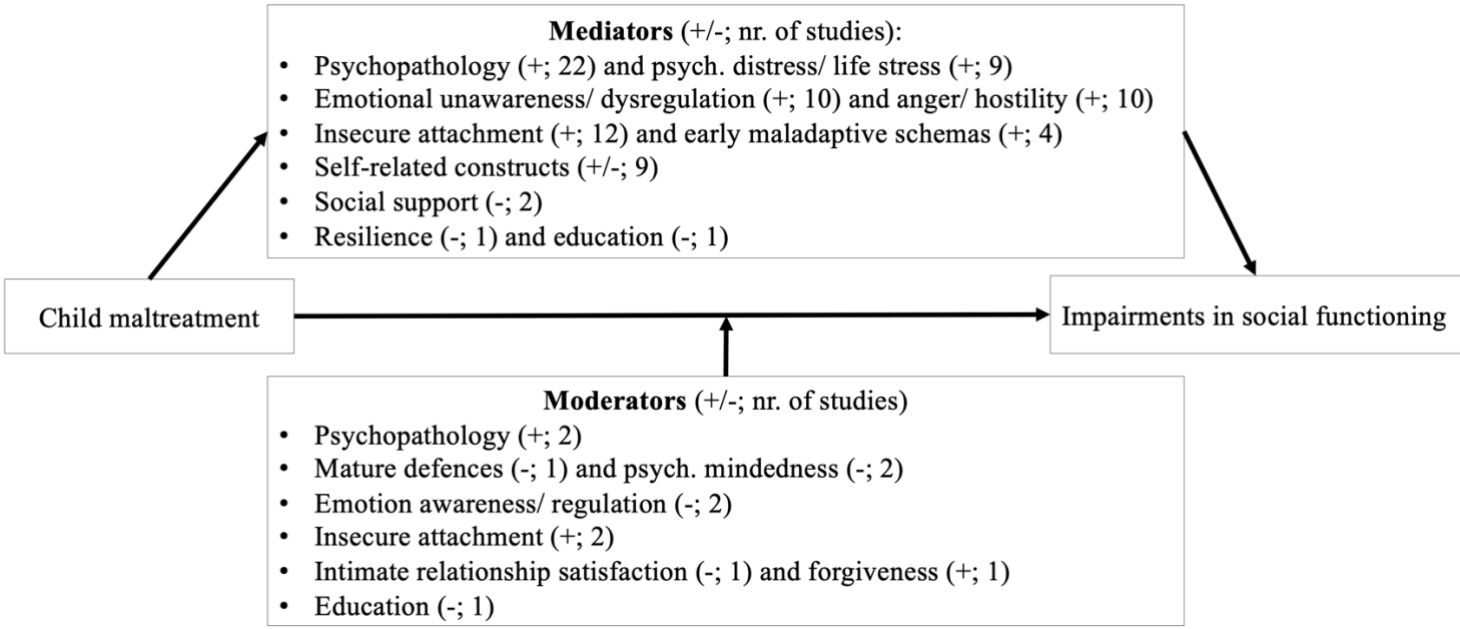
Domains of SF	MED/ MOD	L	C-S	Psychological processes	Interpersonal patterns	Contextual factors
A.Social connectedness	MED	1	5	- Personal distress - Depression - Difficulties in emotion regulation - Self-concept clarity	- Emotional support - Rejection sensitivity	
B. EMS	MED	-	1		- Problematic relationship with the father	
C. Sexuality	MED	2	2	- Emotion recognition - Fear of intense emotions - Self-compassion	- Partner responsiveness - Basic need satisfaction in relationships	
	MOD	-	1		- Intimate relationship satisfaction	
D. Aggression or violence	MED	4	20	- Early aggression - Childhood externalizing behaviors - Maladaptive personality traits - PTSD symptoms - Problematic alcohol use - Criminal thinking - Conduct disorder - Anxiety - Difficulties in emotion regulation - Difficulties in affect integration - Anger rumination - Low resilience - Reduced self-compassion	- Adolescent peer social skills - Attachment anxiety - Impulsivity - Interpersonal hostile-dominance/hostility - Insecure attachment - Maladaptive schemas	- Environmental instability - School protective index
E. Intimate relationships	MED	2	22	- Psychological well-being/ distress - PTSD/ PTSS symptoms - Trait anxiety - Emotion recognition - Fear of intense emotions - Experiential avoidance - Psych. mindfulness - Impaired mentalizing - Self-qualities (e.g. compassion, criticism)	- Satisfaction/ quality - Insecure attachment - Dyadic coping - Empathic accuracy - Interpersonal sensitivity - Perception of partner responsiveness - Partner attributions - Sexual shame	

	MOD	-	4	- Sexual-related PTSS - Robust mentalizing - Mature defenses	- Forgiveness	
F. IPV & revictimization						
IPV victimization & re-victimization	MED	2	11	- Life stress / psychological distress - PTSD symptoms - Drinking problems - Dissociation - Difficulties in emotion regulation - Experiential avoidance - Hostility	- Low social support - Schemata: Disconnection/ rejection, emotional deprivation, vulnerability to harm/ illness - Rejection sensitivity - Fearful dating experiences - Mistrust	
	MOD	1	3	- Emotional awareness - Emotion regulation - Psychological mindedness	- Attachment insecurity	
IPV Perpetation	MED	3	11	- PTSD symptoms - Maladaptive personality traits - Substance use - Problematic alcohol use - Depressive symptoms - Emotion dysregulation - Problematic anger, negative urgency - Hostility and hostile masculinity - Aggressive sexual fantasies - IPV victimization - Executive functioning - Self-esteem	- Disconnection/ rejection schema domains - Maladaptive communication patterns - Dominance and shame - Male peer support for violence	- Low Grade Point Average (GPA)
	MOD	1	1	- Impulsive and irresponsible traits of psychopathy	-	- GPA
G. Parenting or maltreating behavior	MED	-	8	- Parental psychopathology (e.g., IPV-related PTSD) - Perceived (parenting) (dis)stress - Meta-parenting - IPV	- Shame	- Family functioning

Note. MED, Mediators; MOD, Moderators; L, number of longitudinal studies; C-S, number of cross-sectional studies; EMD, Early maladaptive schemas.

Figure 4

Mediators and Moderators Between Childhood Maltreatment and Impairments in Social Functioning



Note. This figure displays only the most frequently identified mediators and moderators across studies. Self-related constructs include self-criticism, self-concealment, self-compassion, self-concept clarity and self-qualities. Parentheses indicate the number of studies and the effect of the mechanism, with the symbols representing '−' = decreasing and '+' = increasing the association between CM and impairments in social functioning.

Discussion

Impairments in social functioning might be associated with CM experiences across the population and are prominent in many mental disorders. Following our goal to develop interventions for improving social functioning in individuals with CM, we were interested which social deficits are related to which types of CM and which mediator and moderators need to be considered. Differentiating between domains of social functioning and linking social dysfunction to CM as a potential correlate or risk factor can support the development of transdiagnostic models of social dysfunctions and mechanism-based interventions (Goerigk et al., 2023).

Relationship between CM and Social Functioning

We found an association between CM and fundamental social competencies and their application in specific domains of social functioning confirming previous reports (Carr et al., 2020; Haslam et al., 2020). Interestingly, the strength of the associations ranged from small to moderate and varied based on the type of CM and the specific social function. The strongest evidence emerged for IPV, aggression, social connectedness, attachment, and early maladaptive schemas, whereas findings for other domains, including sexuality, intimate partner relationships, and social-cognitive processes, were more inconsistent. The negative association between CM and social connectedness was reflected in lower social support, increased loneliness, interpersonal difficulties, diminished relationship satisfaction, and negative social expectations, while findings on empathic concern and perspective-taking were mixed. Weak to strong associations emerged between CM, particularly emotional abuse, and insecure attachment, as well as early maladaptive schemas. In terms of sexual health, studies found weak associations between CM, particularly sexual abuse, and reduced sexual functioning, satisfaction, and increased risky sexual behaviors. CM also correlated weakly to moderately with physical and verbal aggression, hostility, anger, violence and offending, with stronger

associations for physical, emotional, or sexual abuse. Most studies consistently revealed at least weak to moderate associations between some form of CM and IPV, both as victimization and perpetration, as well as to revictimization. Research has documented associations often described in the context of intergenerational patterns of CM, with parental CM history being correlated with child maltreatment risk and maladaptive parenting, though findings on parental sensitivity were inconsistent.

Potential Mechanisms in the Link between CM and Social Functioning

A secondary objective was to identify potential mechanisms in the sense of mediating and moderating factors within the association between CM and impaired social functioning. Across studies and domains, the most frequently observed factors included psychopathology, insecure attachment, and emotion dysregulation which is consistent to previous reports (Fares-Otero et al., 2023; McNeil & Rehman, 2024; Shahab et al., 2021).

First, the most commonly identified mediator and moderator were psychopathology and psychological distress, including a range of conditions, such as PTSD symptoms, personality traits (including antisocial, borderline, and narcissistic tendencies), maladaptive personality features, traits of psychopathy, depression, social anxiety, dissociation, problematic substance use, and conduct disorder. This pattern may suggest that impaired social functioning co-occurs with or follows CM-related psychopathology. However, cross-sectional designs limit inferences regarding temporal causality. It remains unclear, whether the strength of the CM-social functioning association differs between adults with and without psychopathology and a deeper understanding of the complex interactions among the types of CM, psychopathology, and social functioning is needed.

Second, attachment insecurity appeared not only as an outcome but was also investigated and reported as a significant mediator and moderator in the relationship between CM and other domains of social functioning, such as IPV victimization and revictimization,

antisocial behavior and criminal thinking, as well as various relationship characteristics, including quality, satisfaction, respect, attributions, and dyadic adjustment. This aligns with models proposing that attachment security - alongside early schemas, and socio-cognitive competencies, e.g., social perception, sharing, and mentalizing - are fundamental elements that develop continuously from a very young age (Flechsenhar et al., 2022). These elements are theorized to be influenced by early experiences, including CM, and are thought to underlie more complex social processes and domains of social functioning. These include the ability to build and maintain satisfying interpersonal relationships, resolve conflicts, navigate aversive social situations, and care for children. In line with this, (Pfaltz et al., 2022) suggested that it is crucial to identify attachment-related changes that contribute to broader social functioning difficulties, such as a lack of perceived social support.

Lastly, emotional regulation and awareness was the third most often reported mediating or moderating factor in seven studies investigating the association between CM and the availability of social support, trait anger, IPV perpetration and victimization. McNeil and Rehman (2024) emphasize the importance of recognizing internal factors, such as emotional awareness and regulation, which can enhance communication skills and, in turn, improve external factors like the social network size.

Table 2

Critical Findings of the Review

• CM is negatively associated with social functioning in adulthood, with the strongest evidence for IPV, aggression, social connectedness, attachment insecurity, and early maladaptive schemas. Findings for other domains are more inconsistent and need to be examined more thoroughly in high-quality studies (e.g., sexuality, intimate partner relationships, and social-cognitive processes).

- The strength of associations varies from small to moderate depending on the CM type and specific social outcome.
- Psychopathology, insecure attachment, and emotion dysregulation were the most frequently identified mediating or moderating factors of the relationship.
- There is a lack of longitudinal and transdiagnostic studies, leaving gaps in understanding causal directions and in developing transdiagnostic models and mechanism-based interventions for CM-related social dysfunctions across mental health conditions.

Implications for Clinical Practice and Future Research

This systematic review offers valuable insights into the relationship between different types of CM and various domains of social dysfunction. Firstly, one of the most important findings is the non-specificity of CM sequelae in the sense of a rather widespread association of different types of CM with very different domains of social functioning, ranging from fundamental social competencies (e.g., empathy) to specific domains (e.g., sexuality). Therefore, clinicians and researchers across clinical, community, and preventive contexts should assess CM types and domains of social functioning comprehensively in individuals with CM experiences. Interestingly, emotional abuse and neglect showed the strongest and broadest associations across multiple domains of social functioning such as social connectedness, attachment, early maladaptive schemas, aggression, as well as relationship satisfaction and quality (Fares-Otero et al., 2023; Haslam & Taylor, 2022). Yet these forms of CM were among the least studied in existing literature and are often underrecognized in clinical settings and therefore require more attention. In contrast, childhood sexual abuse appeared to be more consistently associated with outcomes related to sexuality and interpersonal victimization, whereas childhood physical abuse might be more specifically linked to aggressive outcomes. However, a key limitation of the present review is that effect sizes were not systematically

comparable across childhood maltreatment subtypes or social functioning outcomes, precluding formal comparisons. Future research should therefore conduct focused, domain-specific and subtype-specific meta-analyses to quantify and compare the relative strength of associations across different forms of childhood maltreatment.

Secondly, the role of psychopathology as the most frequent mediator suggests that social dysfunction may be associated with other psychopathological symptoms and could improve through targeting them. An alternative hypothesis, however, would be that both may reinforce each other in a bidirectional cycle of chronicity. Consequently, social dysfunction should be considered as therapeutic target in clinical practice, with future research directly examining its impact on other psychopathological symptoms to help break these possible reinforcing cycles. Thirdly, insecure attachment, maladaptive schemas, and emotion dysregulation are key processes linking CM to social dysfunction. Therapeutic approaches that address these underlying mechanisms (e.g., DBT, MBT, CBASP) may improve interpersonal outcomes across diagnostic categories.

Finally, although many of the reported associations were characterized by weak to moderate statistical effect sizes, the lack of established clinical benchmarks for most social functioning outcomes limits direct inferences regarding clinical significance. Consequently, the review emphasizes the need for a more consistent definition of social functioning and a more comprehensive, nuanced assessment of its various domains and establishment of clinically meaningful thresholds. Futurre research would benefit from more high-quality mixed method designs with real-life longitudinal assessments of social functioning to better understand causal relationships and temporal dynamics, particularly in underexplored domains (e.g., sexuality and parenting behavior). This would help improve comparisons across studies. Prospective longitudinal studies are also necessary to more closely study the relationship between social dysfunction and other symptoms of psychopathology in CM survivors.

Additionally, a deeper exploration of underlying mechanisms is essential for developing personalized transdiagnostic treatments that address specific social impairments in individuals with CM. The review suggests that insecure attachment, early maladaptive schemas, and deficits in emotion regulation may be more general outcomes of CM. However, it remains unclear whether specific types of CM are linked to distinct patterns within these domains and whether these patterns, in turn, contribute to more complex social dysfunctions, such as challenges with social connectedness, parenting, or aggression.

Table 3

Implications for Research, Practice and Policy

- Clinicians and researchers should assess multiple CM types, including emotional abuse and neglect, as well as a broad range of social functioning abilities (e.g. empathy) and domains (e.g., sexuality) within multiple types of relationships, as CM effects are widespread.
- Promoting emotion regulation and fostering secure attachment in clinical practice may significantly improve social functioning in survivors of CM. Researchers should explore whether targeting key mediators (e.g., through DBT, MBT, CBASP) improves social outcomes and prioritize developing mechanism-based, transdiagnostic interventions.
- Researchers should focus on standardizing measures and establishing consistent definitions of social functioning given the significant variability in constructs and measures used across domains.
- Future research should employ ecologically valid, high-quality longitudinal methods to clarify causal pathways and explore the complex links between types of CM, mechanisms and specific social processes.

Strengths and Limitations

While the current review boasts several strengths, including a thorough examination of the association of childhood maltreatment (CM) with various domains of social dysfunction and a focused synthesis of key trends and gaps in the literature, it is important to acknowledge some notable limitations. First, we did not calculate general effect sizes or meta-analytic relationships across studies, limiting our ability to provide quantitative summaries or pooled estimates of the outcomes. Although the number of included studies might suggest the feasibility of a meta-analysis, the considerable heterogeneity across studies in terms of conceptualization, measurement, and reporting of social functioning as well as populations, exposure types, and analytic strategies precluded meaningful quantitative synthesis. Conducting a meta-analysis would have risked producing misleading or non-generalizable conclusions. Instead, our goal was to provide a comprehensive synthesis to map the domains of social functioning affected by childhood trauma, identify gaps in the literature, and inform future research directions and clinical practice.

Second, an important limitation of this review is the absence of double screening and double data extraction. Given the large volume of articles screened (n = 5,739) and included (n = 281), titles, abstracts, or full text screening were conducted by a single reviewer and data extraction was not independently verified by a second reviewer. This approach deviates from best practice for systematic reviews and may increase the risk of selection bias, missed studies, and extraction errors. To mitigate these risks, we followed predefined eligibility criteria, piloted the screening and extraction forms, and documented all decisions. However, these steps cannot fully replace independent verification and this limitation should be considered when interpreting the findings. Third, we did not distinguish between different methods of CM assessment. In most studies, CM was assessed through retrospective recall and self-report, with the CTQ being one of the most commonly used instruments.

Additionally, the studies included in this systematic review present several important limitations that should be considered when interpreting the findings. First, the majority relied on cross-sectional data (83.3%), limiting causal inference. Second, the evidence base is dominated by WEIRD (White, Educated, Industrialized, Rich, Democratic) samples, which constraints the cultural validity of the findings and likely reflects culturally specific norms regarding social relationships. Consequently, the relevance of these results for non-WEIRD populations remains uncertain. Importantly, culture may interact with gender, as both CM and SF are shaped by sociocultural context. A gender bias might have confounded the literature as 57% of the participants across studies were female and research of several SF domains, particularly IPV, sexuality, and paternal behavior, focused predominantly on one gender and specific types of CM (e.g., the impact of sexual abuse on sexual revictimization in women). Such gender-specific approaches often align with prevailing stereotypes and potentially obscure gender-specific mechanisms. This focus limits the generalizability of findings across gender-diverse perspectives and all forms of CM. As such, future research should prioritize culturally diverse samples, adopt gender-inclusive designs and systematically explore the effects of various types of CM and their outcomes (e.g., victimization and perpetration) across diverse populations, ensuring balanced representation and consideration of all relevant factors.

Conclusion

CM, both in its general form and across distinct subtypes, appears to be linked with dysfunction in various domains of social and interpersonal functioning, with mostly small to moderate associations. These findings highlight the importance of comprehensively assessing CM and its effects on social functioning in both clinical practice and research. This includes evaluating general abilities (such as empathy and attachment), specific domains (like sexuality), and a range of relationships (including work, friendships, partnerships, and family). Such an approach can help facilitate the early identification of trauma histories and contribute to the

development of more targeted and effective interventions for interpersonal dysfunctions. Potential mechanisms mediating the relationship between CM and social dysfunction, such as psychopathological symptoms, insecure attachment, and emotion dysregulation, have already been identified and may serve as transdiagnostic targets for interventions. However, most evidence to date is correlational and based on cross-sectional designs. Future longitudinal research is crucial for further clarifying the pathophysiological pathways linking CM to psychosocial functioning and for refining diagnostic and therapeutic models that address interpersonal dysfunctions.

References

Abajobir, A. A., Kisely, S., Williams, G. M., Clavarino, A. M., & Najman, J. M. (2017). Substantiated childhood maltreatment and intimate partner violence victimization in young adulthood: A birth cohort study. *Journal of Youth and Adolescence*, *46*(1), 165-179. https://doi.org/10.1007/s10964-016-0558-3

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. American Psychiatric Publishing. https://doi.org/10.1176/appi.books.9780890425596

Aversa, L. H., Lemmer, J., Nunnink, S., McLay, R. N., & Baker, D. G. (2014). Impact of childhood maltreatment on physical health-related quality of life in U.S. active duty military personnel and combat veterans. *Child Abuse & Neglect*, *38*(8), 1382-1388. https://doi.org/10.1016/j.chiabu.2014.03.004

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T.,…Zule, W. (2003). Development and validation of a brief screening version of the childhood trauma questionnaire. *Child Abuse & Neglect*, *27*(2), 169-190. https://doi.org/10.1016/s0145-2134(02)00541-0

Bowlby, J. (1982). Attachment and loss: Retrospect and prospect. *American Journal of Orthopsychiatry*, *52*(4), 664-678. https://doi.org/10.1111/j.1939-0025.1982.tb01456.x

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, *63*(3), 452-459. https://doi.org/10.1037/0022-3514.63.3.452

Carr, A., Duff, H., & Craddock, F. (2020). A systematic review of reviews of the outcome of noninstitutional child maltreatment. *Trauma Violence Abuse*, *21*(4), 828-843. https://doi.org/10.1177/1524838018801334

Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*(1), 155-159. https://doi.org/10.1037/0033-2909.112.1.155

Cotter, J., Kaess, M., & Yung, A. R. (2015). Childhood trauma and functional disability in psychosis, bipolar disorder and borderline personality disorder: A review of the literature. *Irish Journal of Psychological Medicine*, *32*(1), 21-30. https://doi.org/10.1017/ipm.2014.74

Dinger, U., Zilcha-Mano, S., McCarthy, K. S., Barrett, M. S., & Barber, J. P. (2013). Interpersonal problems as predictors of alliance, symptomatic improvement and premature termination in treatment of depression. *Journal of Affective Disorders.*, *151*(2), 800-803. https://doi.org/10.1016/j.jad.2013.07.003

Fares-Otero, N. E., Alameda, L., Pfaltz, M. C., Martinez-Aran, A., Schäfer, I., & Vieta, E. (2023). Examining associations, moderators and mediators between childhood maltreatment, social functioning, and social cognition in psychotic disorders: A systematic review and meta-analysis. *Psychological Medicine*, *53*(13), 5909-5932. https://doi.org/10.1017/S0033291723001678

Fares-Otero, N. E., De Prisco, M., Oliva, V., Radua, J., Halligan, S. L., Vieta, E., & Martínez-Arán, A. (2023). Association between childhood maltreatment and social functioning in individuals with affective disorders: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, *148*(2), 142-164. https://doi.org/10.1111/acps.13557

Fitzgerald, M., & Gallus, K. (2020). Emotional support as a mechanism linking childhood maltreatment and adult's depressive and social anxiety symptoms. *Child Abuse & Neglect*, *108*, 104645. https://doi.org/10.1016/j.chiabu.2020.104645

Fitzgerald, M., & Morgan, A. A. (2022). Childhood maltreatment and provision of support and strain to family relationships in adulthood: The role of social anxious and depressive symptoms. *Journal of Social and Personal Relationships.*, *40*(6), 1987-2007. https://doi.org/10.1177/02654075221137320

Flechsenhar, A., Kanske, P., Krach, S., Korn, C., & Bertsch, K. (2022). The (un)learning of social functions and its significance for mental health. *Clinical Psychology Review*, *98*, 102204. https://doi.org/10.1016/j.cpr.2022.102204

Funk, J. L., & Rogge, R. D. (2007). Testing the ruler with item response theory: Increasing precision of measurement for relationship satisfaction with the Couples Satisfaction Index. *Journal of Family Psychology*, *21*(4), 572-583. https://doi.org/10.1037/0893-3200.21.4.572

Gallagher, A. R., Moreland, M. L., Watters, K. N., & Yalch, M. M. (2023). Relative effects of childhood trauma, intimate partner violence, and other traumatic life events on complex posttraumatic stress disorder symptoms. *Traumatology*, *29*(1), 57-64. https://doi.org/10.1037/trm0000379

Goerigk, S., Reinhard, M. A., Barton, B. B., Burkhardt, G., Ehring, T., Bertsch, K.,…Padberg, F. (2023). Transdiagnostic analysis of adverse childhood experiences and their association with psychopathology-A TRANS-D conform study. *Psychiatry Research*, *329*, 115545. https://doi.org/10.1016/j.psychres.2023.115545

Haslam, N., McGrath, M. J., Viechtbauer, W., & Kuppens, P. (2020). Dimensions over categories: a meta-analysis of taxometric research. *Psychological Medicine*, *50*(9), 1418-1432. https://doi.org/10.1017/S003329172000183X

Haslam, Z., & Taylor, E. P. (2022). The relationship between child neglect and adolescent interpersonal functioning: A systematic review. *Child Abuse & Neglect*, *125*, 105510. https://doi.org/10.1016/j.chiabu.2022.105510

Jaffee, S. R., Takizawa, R., & Arseneault, L. (2017). Buffering effects of safe, supportive, and nurturing relationships among women with childhood histories of maltreatment [Article]. *Psychological Medicine*, *47*(15), 2628-2639. https://doi.org/10.1017/s0033291717001027

Keltner, D., & Kring, A. M. (1998). Emotion, social function, and psychopathology. *Review of General Psychology*, *2*(3), 320-342. https://doi.org/10.1037/1089-2680.2.3.320

Kisely, S., Najman, J. M., & Strathearn, L. (2024). Childhood maltreatment and intimate partner violence: 30-year follow-up of a birth cohort study. *Journal of Aggression Maltreatment & Trauma*, *33*(7), 844-863. https://doi.org/10.1080/10926771.2024.2343941

Klinger-König, J., Erhardt, A., Streit, F., Völker, M. P., Schulze, M. B., Keil, T.,…Grabe, H. J. (2024). Childhood trauma and somatic and mental illness in adulthood. *Deutsches Ärzteblatt International*, *121*(1), 1-8. https://doi.org/10.3238/arztebl.m2023.0225

Kotov, R., Krueger, R. F., & Watson, D. (2018). A paradigm shift in psychiatric classification: the hierarchical taxonomy of psychopathology (HiTOP). *World Psychiatry*, *17*(1), 24-25. https://doi.org/10.1002/wps.20478

Lassri, D., & Gewirtz-Meydan, A. (2025). Breaking the cycle: Mentalizing moderates and mediates the link between childhood sexual abuse and satisfaction with adult romantic relationships. *Psychological Trauma: Theory, Research, Practice, and Policy*, *17*(1), 10-19. https://doi.org/10.1037/tra0001692

Lassri, D., Gewirtz-Meydan, A., & Zamir, O. (2023). The mediating role of self-compassion between childhood maltreatment and satisfaction with intimate relationships. *MINDFULNESS*, *14*(4), 970-978. https://doi.org/10.1007/s12671-023-02107-6

Lee, R. M., & Robbins, S. B. (1995). Measuring belongingness: The social connectedness and the social assurance scales. *Journal of Counseling Psychology*, *42*(2), 232-241. https://doi.org/10.1037/0022-0167.42.2.232

Lubben, J. E. (1988). Assessing social networks among elderly populations. *Family & Community Health*, *11*(3), 42-52. https://doi.org/10.1097/00003727-198811000-00008

McCullough, J. P. (2006). *Treating chronic depression with disciplined personal involvement: cognitive behavioral analysis system of psychotherapy (CBASP)*. Springer.

McLaughlin, K. A., Colich, N. L., Rodman, A. M., & Weissman, D. G. (2020). Mechanisms linking childhood trauma exposure and psychopathology: A transdiagnostic model of risk and resilience. *BMC Medicine*, *18*(1), 96. https://doi.org/10.1186/s12916-020-01561-6

McNeil, J., & Rehman, U. S. (2024). The role of emotional processes in explaining the link between severity of childhood maltreatment and relationship difficulties. *Journal of Interpersonal Violence*, *39*(9-10), 2165-2188. https://doi.org/10.1177/08862605231216695

Pfaltz, M. C., Halligan, S. L., Haim-Nachum, S., Sopp, M. R., Åhs, F., Bachem, R.,…Seedat, S. (2022). Social functioning in individuals affected by childhood maltreatment: Establishing a research agenda to inform interventions. *Psychotherapy and Psychosomatics*, *91*(4), 238-251. https://doi.org/10.1159/000523667

Pipp-Siegel, S., & Biringen, Z. (1998). Assessing the quality of relationships between parents and children: The emotional availability scales. *The Volta Review*, *100*(5), 237-249.

Procidano, M. E., & Heller, K. (1983). Measures of perceived social support from friends and from family: Three validation studies. *American Journal of Community Psychology*, *11*(1), 1-24. https://doi.org/10.1007/bf00898416

Quilty, L. C., Mainland, B. J., McBride, C., & Bagby, R. M. (2013). Interpersonal problems and impacts: further evidence for the role of interpersonal functioning in treatment outcome in major depressive disorder. *Journal of Affective Disorders*, *150*(2), 393-400. https://doi.org/10.1016/j.jad.2013.04.030

Russell, D. W. (1996). UCLA loneliness scale (version 3): Reliability, validity, and factor structure. *Journal of Personality Assessment*, *66*(1), 20-40. https://doi.org/10.1207/s15327752jpa6601_2

Sabourin, S., Valois, P., & Lussier, Y. (2005). Development and validation of a brief version of the dyadic adjustment scale with a nonparametric item analysis model. *Psychological Assessment*, *17*(1), 15-27. https://doi.org/10.1037/1040-3590.17.1.15

Shahab, M. K., de Ridder, J. A., Spinhoven, P., Penninx, B., Mook-Kanamori, D. O., & Elzinga, B. M. (2021). A tangled start: The link between childhood maltreatment, psychopathology, and relationships in adulthood. *Child Abuse & Neglect*, *121*, 105228. https://doi.org/10.1016/j.chiabu.2021.105228

Smith, C. A., Ireland, T. O., & Thornberry, T. P. (2005). Adolescent maltreatment and its impact on young adult antisocial behavior [Article]. *Child Abuse & Neglect*, *29*(10), 1099-1119. https://doi.org/10.1016/j.chiabu.2005.02.011

Smith, C. A., Park, A., Ireland, T. O., Elwyn, L., & Thornberry, T. P. (2013). Long-term outcomes of young adults exposed to maltreatment: The role of educational experiences in promoting resilience to crime and violence in early adulthood. *Journal of Interpersonal Violence*, *28*(1), 121-156. https://doi.org/10.1177/0886260512448845

Straus, M. A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales. *Journal of Marriage and the Family*, *41*(1), 75-88. https://doi.org/10.2307/351733

Tibi, L., van Oppen, P., van Balkom, A., Eikelenboom, M., Emmelkamp, P. M. G., & Anholt, G. E. (2019). Predictors of treatment outcome in OCD: An interpersonal perspective. *Journal of Anxiety Disorders*, *68*, 102153. https://doi.org/10.1016/j.janxdis.2019.102153

Veritas Health Innovation. (2024). *Covidence systematic review software*. In www.covidence.org

Wei, M., Russell, D. W., Mallinckrodt, B., & Vogel, D. L. (2007). The experiences in close relationship scale (ECR)-short form: Reliability, validity, and factor structure. *Journal of Personality Assessment*, *88*(2), 187-204. https://doi.org/10.1080/00223890701268041

Weissman, M. M. (1975). The assessment of social adjustment: A review of techniques. *Archives of General Psychiatry*, *32*(3), 357-365. https://doi.org/10.1001/archpsyc.1975.01760210091006

Widom, C. S. (2024). Childhood maltreatment, revictimization, and partner violence victimization through midlife: A prospective longitudinal investigation. *Journal of Interpersonal Violence*, *39*(19/20), 4087-4112. https://doi.org/10.1177/08862605241264524

Widom, C. S., Czaja, S., & Dutton, M. A. (2014). Child abuse and neglect and intimate partner violence victimization and perpetration: A prospective investigation. *Child Abuse & Neglect*, *38*(4), 650-663. https://doi.org/10.1016/j.chiabu.2013.11.004

Widom, C. S., Czaja, S. J., & Dutton, M. A. (2008). Childhood victimization and lifetime revictimization. *Child Abuse & Neglect*, *32*(8), 785-796. https://doi.org/10.1016/j.chiabu.2007.12.006

Widom, C. S., Schuck, A. M., & White, H. R. (2006). An examination of pathways from childhood victimization to violence: The role of early aggression and problematic alcohol use. *Violence and Victims*, *21*(6), 675-690. https://doi.org/10.1891/vv-v21i6a001

Wong, A. E., Dirghangi, S. R., & Hart, S. R. (2019). Self-concept clarity mediates the effects of adverse childhood experiences on adult suicide behavior, depression, loneliness,

perceived stress, and life distress. *Self and Identity*, *18*(3), 247-266. https://doi.org/10.1080/15298868.2018.1439096

World Health Organization. (2019). International statistical classification of diseases and related health problems (11th ed.). https://icd.who.int/

Young, J. E., & Brown, G. (1990). Young Schema Questionnaire. *New York: Cognitive Therapy Center*.

Young, J. E., Klosko, J. S., & Weishaar, M. E. (2003). *Schema therapy: A practitioner's guide*. Guilford Press.

Zamir, O., & Lavee, Y. (2014). Psychological mindedness as a protective factor against revictimization in intimate relationships. *Journal of Clinical Psychology*, *70*(9), 847-859. https://doi.org/10.1002/jclp.22061

Zamir, O., & Lavee, Y. (2015). Emotional awareness and breaking the cycle of revictimization. *Journal of Family Violence*, *30*(6), 675-684. https://doi.org/10.1007/s10896-015-9711-0

Study II:

Psychological and Neurophysiological Measures of Emotion Dysregulation in Borderline Personality Disorder and Posttraumatic Stress Disorder

Göhre, I., Back, S., Schütz-Bosbach, S., Ren, Q., Wolkenstein, L., Rupp, A., & Bertsch, K. (2025). Psychological and neurophysiological measures of emotion dysregulation in borderline personality disorder and posttraumatic stress disorder. *Borderline Personality Disorder and Emotion Dysregulation, 12*(1), 37. https://doi.org/10.1186/s40479-025-00313-3

This chapter is based on the post-peer-review, pre-copyedited version of an article published in Borderline Personality Disorder and Emotion Dysregulation.

The final version of the publication can be accessed at https://doi.org/10.1186/s40479-025-00313-3

Psychological and Neurophysiological Measures of Emotion Dysregulation in Borderline Personality Disorder and Posttraumatic Stress Disorder

Isabelle Göhre[1,2*], Sarah Back[1,2], Simone Schütz-Bosbach[1], Qiaoyue Ren[1], Larissa Wolkenstein[1,2], André Rupp[3], Katja Bertsch[2,4]

[1] Department of Psychology, Ludwig-Maximilians-Universität München, Germany

[2] German Center for Mental Health (DZPG), partner site Munich, Germany

[3] University Hospital Heidelberg, Germany

[4] Department of Psychology, Julius-Maximilians-Universität Würzburg, Germany

* Corresponding Author: Isabelle Göhre, M.Sc., Dept of Psychology, LMU Munich, Germany, Leopoldstr. 13, 80802 Munich, Tel: 089-21805199, E-Mail: isabelle.goehre@psy.lmu.de

Abstract

Background: Emotion dysregulation is a central feature in trauma-associated disorders such as posttraumatic stress disorder (PTSD) and borderline personality disorder (BPD). However, it remains unclear whether emotion dysregulation is a transdiagnostic phenomenon closely linked to childhood trauma, or if disorder-specific alterations in emotion processing exist. Following a multimethodological approach, we aimed to assess and compare the reactivity to and regulation of emotions between patients with BPD and PTSD, as well as healthy controls, and identify associations with childhood trauma.

Methods: A total of 135 women, 43 healthy controls, 43 with BPD and 49 with PTSD, took part in a multimethodological assessment of emotional reactivity and regulation. Self-report measures were used to assess childhood trauma and emotion dysregulation. Additionally, participants performed a classic emotion regulation (ER) paradigm. Subjective emotional valence ratings and neurophysiological responses (P3 and late positive potential, LPP) were measured in response to negative, positive, and neutral pictures (emotional reactivity) and during active regulation vs. passive viewing of negative pictures (ER).

Results: Regarding emotional reactivity, during the experimental paradigm both patient groups reported lower emotional valence after viewing positive or neutral pictures compared to healthy controls. Furthermore, P3 amplitudes in response to neutral pictures were reduced in both patient groups and in response to negative pictures, specifically in patients with PTSD. Regarding ER, while both patient groups self-reported significant disturbances in ER, neither valence ratings nor neurophysiological responses assessed during the ER task (P3, LPP) differed from healthy controls. Across groups, childhood trauma was related to decreased emotional valence ratings on neutral and positive pictures and higher self-reported emotion dysregulation.

Conclusions: Patients with BPD and PTSD exhibited a reduced emotional reactivity in response to positive and neutral information. Specifically, patients with PTSD demonstrated hypo-reactivity to neutral and trauma-unrelated negative stimuli, which might be due to altered attentional resource allocation following trauma. Although patients reported using adaptive ER strategies less frequently in daily life, they effectively implemented them when instructed to, highlighting important clinical and theoretical implications.

Keywords: trauma-related disorders, emotion regulation, emotional reactivity, childhood trauma, event-related potentials.

Introduction

Emotion dysregulation is a key feature of various trauma-associated disorders, including borderline personality disorder (BPD) and post-traumatic stress disorder (PTSD) [1, 2]. Especially traumatic experiences in childhood can result in psychosocial and neurobiological alterations in emotion processing [3-5]. Two key components influenced at different stages of emotional processing are: *emotional reactivity* and *emotion regulation (ER)* [6]. Emotional reactivity refers to the implicit response to emotionally salient stimuli and encompasses changes in subjective experience, behaviour and physiology [2]. According to Gross's process model, ER describes the complex processes by which individuals influence which emotions they experience, when they experience them, and how they express and respond to them [7, 8]. Therefore, ER strategies can be applied at different stages of the emotion-generative process, either before (antecedent-focused, e.g., cognitive reappraisal) or after (response-focused, e.g., emotion supression) an emotional reactivity has occurred. Clinical frameworks, such as Linehan's biosocial model of BPD [5], further support that deficits can occur at different stages, from heightened emotional sensitivity/ reactivity to deficits in the use of adaptive regulation strategies such as antecedent-focused, cognitive reappraisal [2].

While both BPD and PTSD are marked by alterations in emotion processing, their primary diagnostic features suggest that they differ in their emotional reactivity and regulation. Individuals with BPD often display pronounced emotional *hyper-reactivity* (i.e., emotional instability) alongside an *under-regulation* of emotional reactions with deficit in adaptive strategies and a surplus of maladaptive strategies such as self-harm [2, 9]. Individuals with PTSD may also exhibit an emotional *hyper-reactivity* (i.e., hyperarousal) to emotional threats but often attempt to *over-regulate* distress stemming from traumatic memories through emotional avoidance or numbing [10]. Despite these distinctions, direct comparisons of

emotional reactivity and regulation in BPD and PTSD remain scarce [11, 12]. This represents a critical gap, since both disorders share significant overlap in their etiological underpinnings - such as traumatic experiences in childhood - and in their symptomatic manifestations of emotion dysregulation [3-5]. Therefore, it remains unclear whether emotion dysregulation reflects a transdiagnostic phenomenon closely linked to childhood trauma, or if disorder-specific alterations in emotion processing exist. Clarifying this distinction is crucial for improving the clinical differentiation and informing the development of transdiagnostic models and interventions targeting emotion dysregulation.

Another limitation of existing research is its reliance on single-method approaches, such as self-reports or experimental tasks, which fails to capture the multidimensional nature of emotional reactivity and regulation across psychological, behavioral, and neurophysiological domains. A multimethodological approach - integrating self-reports, experimental paradigms, and neurophysiological data - provides a more comprehensive understanding of these processes. Revealing discrepancies between subjective and objective measures can guide accurate assessments and improve clinical interventions that aim to target a specific ER process.

In summary, there is a pressing need for research that systematically compares emotional reactivity and regulation among individuals with BPD and PTSD using a multimethodological approach. In the following sections, we will first outline different methods for assessing emotional reactivity and regulation, including self-reports, experimental paradigms, and neurophysiological data. Next, we will provide an overview of the current knowledge on emotion processing in BPD and PTSD patients based on these methods. We will then explore knowledge on the transdiagnostic link between childhood trauma and emotion processing in BPD and PTSD and conclude by outlining our research aims based on these insights.

Methods for assessing emotion processing

Self-report questionnaires, like the Difficulties in Emotion Regulation Scale (DERS) [13], assess dysfunctions in emotion processing, including the awareness, clarity, and acceptance of emotional reactions as well as the use of adaptive ER strategies. As proposed by McRae [14], questionnaires aim to measure the *frequency* of using ER strategies flexibly in everyday life over the *long-term*. In contrast, experiments measure the *short-term* emotional reactivity to stimuli (e.g., positive, negative and neutral pictures) and the *success* of employing an instructed ER strategy in the laboratory. For example, one experimentally measurable ER strategy is *cognitive reappraisal*, which e.g., involves reinterpreting the meaning of a negative picture to reduce its emotional impact [15]. The emotional reactivity to pictures and the effect of the ER strategy can be measured on multiple levels, including subjective ratings of emotional valence and objective neurophysiological responses. Other modalities, such as behavioral or physiological measures (e.g., heart rate, pupil dilation), can also provide valuable insights.

The neural activity in response to emotional pictures can be assessed through event-related brain potentials (ERPs) [16]. Among other ERP components, the P3 and the Late Positive Potential (LPP) are particularly recognized as reflecting cortical emotion processing [17]. The centroparietal P3, a positive deflection around 300 to 500ms following picture presentation, exhibits enhanced amplitude in healthy individuals in response to emotionally salient (i.e., negative and positive) compared to neutral pictures [16]. The P3 is thought to represent conscious attentional processing of emotional information and resembles the early phase of the LPP. The LPP, a positive wave extending up to 1500ms post-stimulus, is associated with more conscious processing of emotionally significant pictures with intrinsic motivational relevance [18]. As ER processes typically require more time to develop, they might primarily impact later components, such as the LPP [19]. In healthy individuals,

cognitive reappraisal of negative pictures was associated with decreased LPP amplitudes compared to passive viewing [19-22].

Emotion processing in BPD and PTSD

Emotional reactivity

Self-report studies indicate heightened negative emotional reactivity in individuals with BPD [23-25] and PTSD [26-28]. However, experimental findings on valence ratings in response to emotional pictures remain inconsistent. Studies have found that individuals with BPD [29-31] and PTSD [32] report lower positive valence (hypo-reactivity) after exposure to (positive and) neutral pictures compared to healthy volunteers. However, findings on valence ratings after negative pictures have yielded mixed results. Studies using *trauma-unrelated negative pictures* often find no discernible differences from healthy controls in PTSD patients [33, 34] and BPD patients [29]. However, studies using *trauma-related negative pictures* report heightened negative valence ratings in individuals with PTSD [32, 35, 36] and BPD [37]. This might suggest that negative emotional hyperreactivity in BPD and PTSD is not generalized but context-dependent, particularly for pictures depicting trauma-related themes, such as childhood abuse, abandonment or social rejection [29, 38-40].

Research on P3 and LPP amplitudes in response to emotional pictures in BPD and PTSD is scarce, with existing findings remaining highly inconsistent and difficult to compare due to methodological and analytical differences [41-43]. One study has found elevated LPP amplitudes in response to negative compared to neutral pictures in patients with BPD [30]. However, studies that compared the amplitude to negative, positive or neutral pictures separately, found no difference compared to healthy controls [29, 44]. Moreover, studies using pictures of emotional faces have found reduced P3 and LPP responses compared to HC [45], particulalry for positive (i.e., happy) faces [46, 47]. A similarly inconsistent pattern emerges

for PTSD, with some studies indicating a reduced P3 amplitude in response to trauma-unrelated negative or neutral pictures compared to HC [34, 36, 41], while others find an increased P3 amplitude to trauma-related pictures [35]. In contrast, several studies report no significant differences to HCs in the P3 amplitude [33, 48] or LPP amplitude [33, 48-50].

Emotion regulation

Reviews and meta-analyses of self-report studies suggest that individuals with BPD exhibit deficiencies in the *frequency* of attenuating intense emotions in every-day life through adaptive ER strategies such as cognitive reappraisal [24, 51]. Individuals with PTSD often self-report general emotion dysregulation, characterized by difficulties in reducing emotional reactivity and less frequent use of adaptive ER strategies [1, 52].

In contrast, findings from experimental studies have not reliably demonstrated difficulties in ER in both BPD [30, 44, 53] and PTSD [50]. Several studies report no significant difference between patients and healthy controls in ER tasks. When instructed to, individuals with BPD [29, 51, 54] and PTSD [50] might be able to employ cognitive reappraisal to down-regulate negative valence ratings. Moreover studies in BPD [30] and PTSD patients [49, 50] have found no difference to HCs in the effect of cognitive reappraisal on LPP amplitudes.

Childhood trauma and emotion dysregulation

Explanatory models of emotion dysregulation in trauma-associated disorders, such as BPD and PTSD, propose childhood trauma as a shared etiological mechanism [3-5]. Childhood trauma - encompassing sexual, physical, and emotional abuse as well as physical and emotional neglect before the age of 18 years - can impact emotion processing on both psychosocial and neurophysiological levels in BPD and PTSD [3, 31, 55]

On a psychosocial level, abuse and neglect can result in insecure or disorganized attachments due to a lack of secure emotional bonding [56, 57]. Due to the caregivers invalidating and abusive reactions to emotional needs, a child might cope with a hyperreactivity

to threat-related socio-emotional cues [58] and maladaptive ER strategies (e.g., avoidance) [59]. On a neurobiological level, childhood trauma might be associated with increased threat sensitivity, driven by limbic hyperactivation (e.g., amygdala, hypothalamus-pituitary-adrenal axis) alongside impaired regulation by fronto-cortical circuits [55, 60, 61].

Despite these well-documented effects of childhood trauma on emotion processing, its influence on experimental measures of emotional reactivity and regulatios, such as valence ratings and neurophysiological responses, remains underexplored in BPD and PTSD. In BPD, more severe childhood trauma experiences and lower parental attachment has been linked to greater emotional reactivity to positive pictures in valence ratings [31]. Moreover, a study in BPD patients found that childhood trauma was correlated with ERP amplitudes in response to pictures depicting neutral, physical painful, and psychological painful interactions during a "Social Interaction Empathy Task" [62]. In contrast, studies in healthy adults [63] and university students [64] have reported a link between childhood trauma and reduced ERP amplitudes in response to unpleasant pictures, with stronger effects in individuals with lower top-down impulse control. Similarly, in trauma-exposed adolescents, a higher number of traumatic events correlated with a reduced ERP difference between positive and neutral IAPS pictures [65]. The authors interpreted this blunted response as reduced affective discrimination or desensitization to intense emotions after childhood trauma. Importantly, an intervention study that targeted emotional coping strategies in adolescents with childhood trauma highlighted the potential to improve affective discrimination in form of increased LPP responses [66].

These findings underscore the need to examine the transdiagnostic role of childhood trauma in psychosocial and neurophysiological emotion dysregulation in BPD and PTSD to inform the development of clinical interventions. In this regard, it is crucial to determine whether childhood trauma serves as one of the primary transdiagnostic factors driving emotion

dysregulation in BPD and PTSD that provides explanatory value beyond traditional diagnostic boundaries.

The present study

Although emotion dysregulation is a hallmark feature of BPD and PTSD, it remains unclear whether it represents a transdiagnostic consequence of childhood trauma or involves disorder-specific alterations, such as differences in the intensity of emotional reactivity or the capacity for emotion regulation unique to each disorder. Existing research lacks direct comparisons of the disorders and a multimethod approach. To address this, we assessed emotion processing using self-reports as well as emotional valence ratings and neurophysiological responses (ERPs) in an experimental task in female patients with BPD, female patients with PTSD, and healthy women. Emotional reactivity was measured through responses to positive, neutral, and negative images, while ER was assessed by comparing affective responses during cognitive reappraisal versus passive viewing of negative images.

Based on existing literature, we hypothesized (1) elevated emotional reactivity in the experimental task, operationalized as lower valence ratings and increased P3 and LPP amplitudes in response to negative emotional stimuli in individuals with BPD and PTSD compared to healthy controls. (2) The instruction to down-regulate negative emotions (reappraisal vs. view condition) was hypothesized to result in a smaller increase in valence ratings and a lower decrease in late LPP amplitudes in individuals with BPD and PTSD compared to healthy controls, indicating less effective downregulation. (3) We expected significantly elevated self-reported emotion dysregulation, as measured by the total score and subscales of the DERS, in patients with BPD and PTSD compared to healthy controls. (4) We expected childhood trauma, as measured by the total score of the Childhood Trauma Questionnaire (CTQ), to be associated with alterations in emotional reactivity and regulation

across diagnostic categories, suggesting a potential transdiagnostic relevance. (5) Due to a lack of previous studies, no a priori hypotheses could be formulated concerning the differences between BPD and PTSD and therefore exploratory comparisons were conducted. Since emotion dysregulation is the core symptom of BPD [5], one may expect that individuals with BPD would show more pronounced alterations in emotional reactivity and regulation than those with PTSD.

Methods

Participants

Forty-three women with BPD, fourty-nine women with PTSD and forty-three healthy women were included in this study. Exclusion criteria comprised age <18 or >50 years; a body-mass-index <17 or >30; insufficient proficiency in the German language; chronic medical illness, including cardiovascular abnormalities; substance abuse or dependency in the past six months; and a lifetime diagnosis of schizophrenia, schizoaffective or bipolar disorder. HC had never received a psychiatric diagnosis or undergone a psychotherapeutic/psychiatric treatment. Patients with BPD had to have a primary DSM-5 diagnosis of BPD (≥5 diagnostic criteria); patients with PTSD had a primary DSM-5 diagnosis of PTSD but did not fulfil ≥3 diagnostic criteria of BPD.

Parts of the study were assessed within a larger preregistered project (preregistration at www.drks.de: DRKS00019945), which originally included the recruitment of HC and individuals with BPD and specified analyses focusing on interoception as possible mediator between childhood trauma and ERP-based emotion regulation. The current analyses, which include the recruitment of a PTSD group as well as group comparisons of multiple ER measures (self-report and experimental variables), and the hypothesis regarding transdiagnostic effects of childhood trauma, were however not part of the preregistration. Participants were recruited

by referral of practitioners and hospitals, self-help organizations, and advertisements (e.g., in newspapers and social media). The study was performed in accordance with the principles of the Declaration of Helsinki and approved by the ethics committee of the Department of Psychology, LMU Munich. Participants provided written informed consent and were financially reimbursed.

Experimental protocol

For all participants, the diagnostic process comprised an extensive telephone screening for inclusion and exclusion criteria (approx. 45min) followed by an onsite diagnostic appointment (approx. 3h; see below) and the experimental session (approx. 2h). Before the experiment, participants underwent a urine toxicology screening to exclude acute substance abuse. They filled out several state questionnaires and were prepared for the EEG measurement which took place in a sound-proofed and electrically shielded laboratory. Self-report questionnaires were filled out digitally either at home or in the laboratory prior to the experiment.

Diagnostic and self-report measures

Onsite diagnostic appointments consisted of three interviews performed by experienced diagnosticians with at least a M.Sc. in psychology and clinical training. The International Personality Disorder Examination - Borderline Section [IPDE-BOR, 67] is a semi-structured interview that allows both a categorical BPD diagnosis and a dimensional assessment of symptom severity according to DSM-IV criteria. A diagnosis was made if five or more of the nine criteria were met, based on 15 items rated as 0 = “absent/normal”, (1) “exaggerated/accentuated” or (2) criterion level/pathological, yielding a total score range of 0 - 30. The IPDE-BOR has demonstrated a high interrater reliability compared to other diagnostic tools [68]. The Clinician-Administered PTSD Scale for DSM-5 [CAPS-5, 69] was used as a

structured interview to diagnose PTSD and assess symptom severity. The clinician-assigned scores range from 0 = "absent" to 4 = "extremely", based on a combination of the frequency and intensity of the 30 DSM-5 symptoms, with a total score range of 0 - 120. The German version of the CAPS has shown a high internal consistency and inter-rater reliability in a trauma-exposed sample [70]. Lastly, the Structured Clinical Interview for DSM-5 Disorders – Clinicial Version [SCID-CV, 71] was included as a semi-structured diagnostic tool for the assessment of comorbid Axis-I disorders according to DSM-5. The SCID-CV has demonstrated a high clinical validity and inter-rater reliability [72].

For the assessment of childhood trauma and emotion dysregulation the following self-report questionnaires were used:

The German version of the **Childhood Trauma Questionnaire** (CTQ) [73] was used to retrospectively assess childhood trauma before the age of 18 years. Its 28 items (a five-point Likert scale ranging from 1 = "*never true"* to 5 = "*very often true*") can be summarized to the five subscales "emotional abuse", "sexual abuse", "physical abuse", "emotional neglect", and "physical neglect" and a total sum score (range from 25 to 125 by exluding 3 items on minimization/denial), with higher scores indicating more traumatization. Previous studies have shown robust psychometric properties of the CTQ with acceptable to high internal consistencies of the subscales (except for physical neglect) in clinical [74] and healthy[75] as well as community [73] samples. In our sample, a Cronbach's alpha of 0.89 indicated good internal consistency of the total scale.

The German version of the **Difficulties in Emotion Regulation Scale** (DERS) [13] assesses emotion dysregulation. Its 36 items (a Likert scale from 1 = "*almost never*" to 5 = "*almost always*") can be summarised in six subscales reflecting distinct facets of emotion dysregulation ("nonacceptance of emotional responses", "difficulties engaging in goal-directed behaviour", "impulse control difficulties", "lack of emotional awareness", "limited access to

ER strategies", and "lack of emotional clarity") and a total score ranging from 36 to 180, with higher scores indicating higher emotion dysregulation. The DERS has demonstrated a high internal consisity for all subscales except for "lack of emotional awareness" in a clinical sample [76] and adequate reliability of all subscales in student samples [13, 77] which was confirmed by a Cronbach's alpha coefficient of 0.97 in the current study.

The German version of the Revised **Beck Depression Inventory-II** (BDI-II) [78] was administered as a self-report measure to evaluate depressive symptoms. The questionnaire consists of 21 items, with participants rating their symptoms over the past two weeks on a Likert scale (0–3 points). The total score is derived by summing all item responses. The BDI-II has demonstrated high internal consistency in psychiatric and non-clinical samples [79] similarly to the current sample (α=.95).

Experimental task

The ER paradigm was an adapted version from Schönfelder et al. [20], which is based on earlier protocols by Kanske et al. [80] for use in ERP research. Similar versions of the task have been found to be effective in modulating ERPs in healthy controls [20, 80, 81], BPD patients [30] and PTSD patients [49], supporting its sensitivity to emotion regulation processes. Participants were exposed to 40 negative, 20 neutral, and 20 positive pictures in two blocks with a brief break of 2 minutes between blocks and a total duration of approximately 23 minutes. Each trial started with a fixation cross for 500 ms followed by a one-word instruction (2000 ms) (VIEW or DECREASE). Afterwards an IAPS picture was displayed for 5000 ms after which the participants had to provide a valence rating (4000ms). A time-varying intertrial interval of 3500 to 5500 ms was displayed before the next trial sarted (see Fig. 1 for trial structure and timing).

At the beginning of the experiment, participants were instructued to either attentively look at the content of the picture (VIEW) or to actively reduce emotional reactions (DECREASE). The latter condition (DECREASE) was always followed by pictures with negative content (i.e., 20 negative pictures in the DECREASE and the VIEW condition, respectively). To achieve this, they were asked to emotionally distance themselves from the picture by trying to become an uninvolved observer and assuming that the depicted situation was only a re-enactment. Each trial ended with a rating of the current emotional valence ("*How are you feeling right now?*") using the Self-Assessment Manikin (SAM) Scale for Valence [82], a non-verbal, pictorial 9-point scale ranging from 1= "*unpleasant*" via 5="*neutral*" to 9="*pleasant*" that is displayed in Fig. 1. Pictures were presented in a pseudorandomized order with no more than three pictures of the same valence and no more than six times the same instruction in a row. They were drawn from the International Affective Picture System (IAPS) [83] based on the valence ratings of the female normative IAPS sample. The 40 negative pictures displayed accidents, mutilation, loss, pain, or illness (low normative valence ratings of M=1.97, SD=1.30). The same set of negative pictures was presented to all participants. These images were not explicitly matched to participants' trauma histories, and were therefore considered trauma-unrelated. However, we cannot rule out that some content (e.g., scenes of accidents) may have resembled aspects of individual trauma experiences. The 20 neutral pictures depicted individuals engaged in everyday activities (e.g., working) as well as streets with pedestrians or offices (moderate valence ratings of M=5.27, SD=1.40) and the 20 positive pictures showed romantic couples, scenes with happy families or children, successful athletes, and activities (high normative valence ratings of M=7.71, SD=1.52). All three valence categories included pictures of humans among others. In our total sample, the mean valence ratings of negative pictures were slightly higher (M=3.17, SD=0.94) and the ratings of positive pictures were slightly lower (M=6.33 SD=1.20) compared to the normative female IAPS

samples. Neutral pictures were rated similarly (*M*=5.55, *SD*=0.73). For detailed information about the pictures used in this study, including IAPS identification numbers and normative ratings of valence, please refer to our supplementary material.

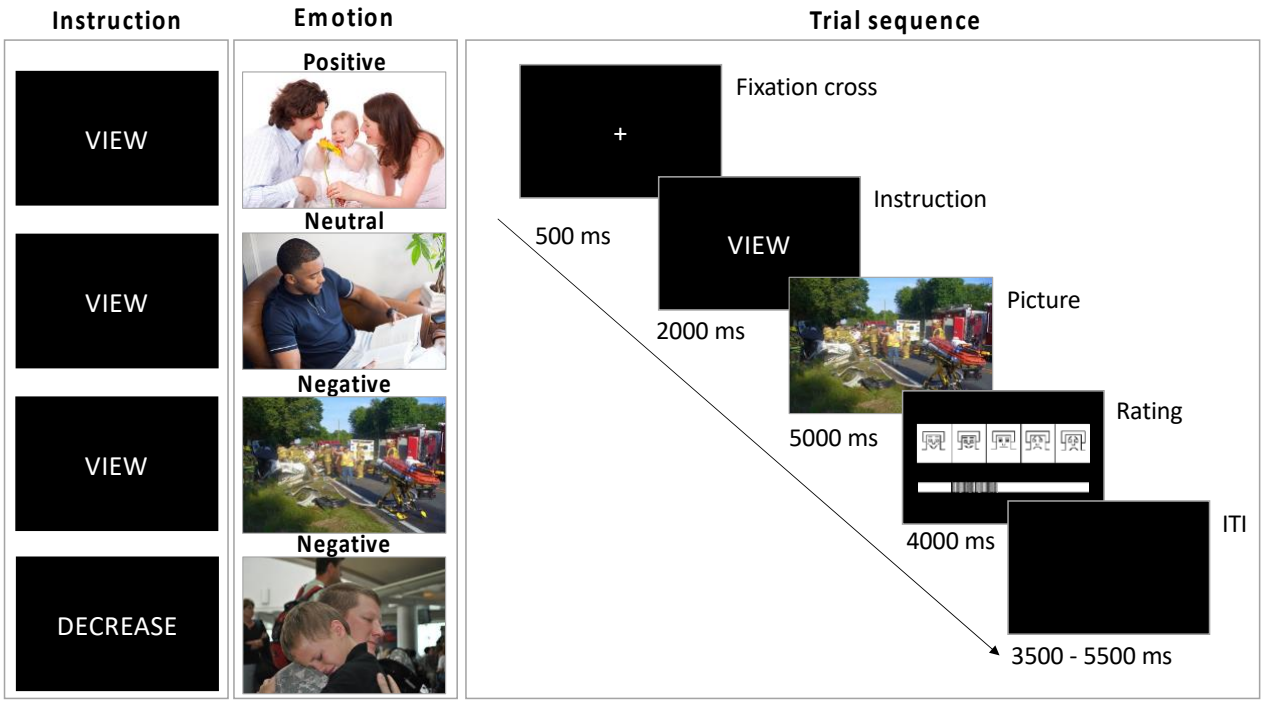


Fig. 1 Schematic task overview. Images are resembling the IAPS pictures used. ITI, Inter-Trial Interval.

Data Acquisition

Stimulus presentation and acquisition of ratings of emotional valence were implemented with Presentation Software (Version 23.0, Neurobehavioral Systems). EEG data were recorded with 64 Ag/AgCI electrodes mounted in an EEG headcap with an equidistant 10-10 electrode system, average reference, and impedance <10 kΩ (TMSi, Oldenzaal, The Netherlands). In addition, a vertical electrooculography (VEOG) with infra- and supraorbital electrodes was recorded.

Data Processing

The EEG data was processed in Brain Vision Analyzer 2.2 (Brain Products GmbH, Munich, Germany). First, we removed the peripheral electrodes AF8, FT7, FT8, FT9, FT10, T7, T8, PO9, and PO10 based on post hoc visual inspection due to excessive noise from muscular artefacts and subsequently recalculated the average reference. Next, data were down-sampled to 250 Hz and filtered with a 0.1 to 30-Hz (24 dB/oct) bandpass filter and a 50 Hz Notch filter. After a manual visual inspection for the exclusion of singular artefacts, an Independent Component Analysis (ICA) was used to correct artefacts, such as vertical, and horizontal eye-artefacts, or noise. The signal was then segmented into ERP segments from -200ms to 5000ms relative to the picture onset and a baseline correction was applied using the -200ms pre-stimulus period. Hereafter, a semiautomatic artefact rejection was used to remove epochs with artefacts based on the following criteria of Schönfelder et al. [20]: a maximum allowed voltage step of 50µV/ms, an absolute difference of ≤ 300µV within a trial (i.e., max. peak to min. peak difference) and a lowest allowed activity of 0.50µV within 100ms. Finally, trials were averaged within the VIEW condition for each emotion category (negative, neutral, positive) separately and in the DECREASE condition for negative pictures. All grand averages of stimulus-locked ERPs were calculated for patients with BPD, patients with PTSD, and healthy controls separately.

Relevant topographies of the P3 and LPP were determined with a nonparametric cluster-based permutation *t*-test (cluster-defining threshold p=.05; two-tailed; iterations = 5000) using the FieldTrip toolbox (Oostenveld et al., 2011) and aligned to prior research [16]. Permutation analysis allows for statistical tests over whole time series and electrodes, while still controlling for multiple comparisons [84]. The following electrodes and time windows were used for analysis: Fz, F1, F2, FCz, FC1, FC2, FC3, FC4, Cz, C1, C2, C4, C5, C6, CPz, CP1, CP2, CP4, CP5, CP6, Pz, P1, P2, P3, P4, P5, P5, POz, PO3, PO4, Oz, O1, O2; time

windows: P3 (300–500ms) and LPP (600–1500ms). For the main effect of valence (negative vs. neutral vs. positive), a significant centroparietal cluster (CPC) was found over the following electrodes: Cz, C1, C2, CPz, CP1, CP2, CP3, CP4, Pz, P1, P2. For the main effect of ER (reappraisal vs. view negative), a fronto-central cluster (FCC) was found with the following electrodes: Fz, F1, F2, FCz, FC1, FC2, Cz, C1, C2, CPz, CP1, CP2. Finally, the mean amplitudes over the CPC and FCC clusters were extracted for the two time windows and used in subsequent statistical analysis.

Statistical Analysis

To analyze differences between the patient groups and healthy controls in self-report measures of childhood trauma and emotion dysregulation, we used Kruskal-Wallis rank sum tests due to the non-normal distribution of the data. Group differences and group-by-condition interactions in emotional valence ratings during the experimental task were analyzed with two repeated-measures analyses of variance (rmANOVAs). The first rmANOVA was analyzed with the between-subject factor group (BPD, PTSD, HC) and the within-subject factor emotion category on "view" trials (negative, neutral, positive) to assess *emotional reactivity*. The second rmANOVA was computed with the factors group (BPD, PTSD, HC) and ER condition (decrease vs. view) to assess ER. Please note that the latter analysis only included trials with negative pictures. Similar rmANOVAs were run to examine emotional reactivity (3x3 rmANOVAs) in centro-parietal P3 and LPP amplitudes as well as to assess ER (3x2 rmANOVAs) in fronto-central P3 and LPP amplitudes. Dunn Multiple Comparisons with Bonferroni correction for multiple testing were used as post-hoc tests. Finally, Pearson correlations and multiple regression analyses were computed to analyze whether childhood trauma is significantly associated with self-reported and neurophysiological data on emotional reactivity and regulation, while controlling for BPD and PTSD diagnosis and medication. A medication index was computed based on the Antidepressant Treatment History Form (ATHF)

by Sackeim [85], which is a widely validated tool for the systematic assessment of antidepressant treatment trials and treatment resistance. In this study, it was used to quantify the current psychotropic medication use by assigning each medication a score from "1" to "4" based on the prescribed daily dosage. For participants taking multiple medications, individual scores were summed to create a medication index, reflecting overall medication load. This index was used in multiple regression analyses as continuous predictor. Of note, only individuals with BPD without an acute comorbid PTSD diagnosis (n = 34) were included in the multiple regression analysis to avoid confounding of the diagnoses. To account for potential confounding effects of comorbid PTSD within the BPD group, we also repeated all main analyses after excluding the 9 BPD participants with comorbid PTSD. Specifically, we re-ran the rmANOVAs on valence ratings and ERP amplitudes, as well as the Kruskal-Wallis tests. The exclusion of these cases did not significantly alter the pattern of the results. Detailed outputs of these control analyses are provided in the supplementary material (Table S1, S2, S3).

Statistical tests were run in IBM SPSS version 28. Statistical significance was set at p=.05, effect sizes are reported as proportions of explained variances (η^2) and Hynh-Feldt sphericity corrections were applied in case of significant Mauchly-test (Hynh & Feld, 1976). Of note, Bonferroni correction was applied for all primary rmANOVAs and Kruskal-Wallis tests with post-hoc comparisons. Correlational and regression analyses were considered exploratory and are reported without correction. Sensitivity analyses (G*Power; $\alpha = .05$, power = .80) indicated the study could detect effects of $\eta^2 \approx 0.018$ (2×3 rmANOVA), $\eta^2 \approx 0.015$ (3×3 rmANOVA), $\eta^2 \approx 0.068$ (Kruskal-Wallis), and $R^2 \approx 0.084$ (multiple regression), reflecting adequate power to detect small-to-medium effects across analyses.

Results

Participants

A total of N = 135 female participants took part in the study: 43 healthy women (HC; M_{age}=26.56, SD=4.70, range: 20-45 years), 43 women with BPD (M_{age}=26.37, SD=6.53, range: 18-46 years) and 49 women with PTSD (M_{age}=26.35, SD=6.92, range: 19-50 years). Descriptive statistics and group comparisons are presented in Table 1. Chi-square tests revealed significant associations between group and both education ($\chi^2(4)=17.23$, p=.002) and occupational status ($\chi^2(4)=13.84$, p=.008). BPD participants were overrepresented among those with low education and those out of the workforce (all |adj. res.| ≥ 2.0), while HC participants were more often employed and less often out of the workforce; no significant differences were found for the PTSD group. Group comparisons of the CTQ total score indicated that HCs experienced significantly less childhood trauma in comparison to both patients with BPD (p<.001) and patients with PTSD (p<.001). There was no significant difference between patients with BPD and PTSD on the CTQ total score (p=.447). However, patients with PTSD showed significantly more experiences of physical abuse than those with BPD (p=.026).

Table 1 Sample characteristics (N = 135)

Characteristics **M (SD) or N (%)**	**HC** **(n = 43)**	**BPD** **(n = 43)**	**PTSD** **(n = 49)**	**HC vs. BPD vs. PTSD**	**BPD vs. PTSD**
Age (years) [a]	26.56 (4.70)	26.37 (6.53)	26.35 (6.92)	p = .480	-
Education [a]				**p = .002**	**p > .05**
Low	2 (4.7%)	13 (30.2%)	5 (10.4%)		
Medium	23 (53.5%)	22 (51.2%)	33 (68.8%)		
High	18 (41.9%)	8 (18.6%)	10 (20.8%)		
Occupation				**p = .008**	**p > .05**
Not in workforce	1 (2.3%)	9 (20.9%)	3 (6.1%)		
In education	23 (53.5%)	24 (55.8%)	34 (69.4%)		
Employed	19 (44.2%)	10 (23.3%)	12 (24.5%)		
Current comorbid disorders (n, %)[b]					

PTSD	-	9 (21.4%)	49 (100%)	-	**p < .001**
Depression	-	18 (42.9%)	26 (54.2%)	-	p = .284
Sleeping disorders	-	12 (28.6%)	12 (25.0%)	-	p = .702
Anxiety disorders	-	12 (28.6%)	7 (14.6%)	-	p = .105
OCD	-	1 (2.4%)	3 (6.1%)	-	p = .374
ADHD	-	7 (16.7%)	3 (6.3%)	-	p = .117
Eating disorder	-	6 (14.3%)	5 (10.4%)	-	p = .576
Current psychotherapy	-	27 (62.8%)	30 (62.5%)	-	p = .977
Medication index	-	1.37 (2.09)	1.0 (1.31)	-	p = .898
BPD and PTSD Symptomatology					
IPDE score	0.22 (0.57)	20.95 (4.37)	6.02 (4.13)	**p < .001**	**p < .001**
CAPS total	-	-	38.06 (9.52)	-	-
CTQ[c]					
Emotional abuse	6.98 (3.74)	16.52 (5.09)	17.61 (5.66)	**p < .001**	p = 1.00
Physical abuse	5.21 (1.09)	7.67 (3.87)	9.76 (5.01)	**p < .001**	**p = .026**
Sexual abuse	5.17 (0.66)	8.76 (4.23)	11.55 (5.82)	**p < .001**	p = .154
Emotional neglect	8.02 (4.41)	15.95 (4.88)	16.80 (4.86)	**p < .001**	p = 1.00
Physical neglect	6.17 (1.94)	10.60 (4.23)	10.49 (3.71)	**p < .001**	p = 1.00
Total	31.55 (9.56)	59.50 (16.05)	66.20 (17.19)	**p < .001**	p = .447
BDI-II Total	3.16 (3.16)	27.95 (11.19)	23.79 (10.57)	**p < .001**	p = .668

Note. M, Mean; SD, Standard Deviation; HC; Healthy Controls; BPD; Borderline Personality Disorder; PTSD, Post Traumatic Stress Disorder; IPDE, International Personality Disorder Examination; CAPS-5, Clinician-Administered PTSD Scale for DSM-5; CTQ, Childhood Trauma Questionnaire; BDI-II, Beck Depression Inventory II; OCD, Obsessive Compulsive Disorder; ADHD, Attention Deficit Hyperactivity Disorder; Comorbidity and medication: counts do not add up due to multiple diagnoses and polypharmacy, respectively. Education and occupation were recoded into three levels. Education: low = no degree or middle school, medium = high school or vocational training, high = university degree. Occupation: Not in workforce = incapacity for work/ pension/ unemployed, in education = high school/ apprentice/ trainee/ university student, employed = part- & fulltime. [a] PTSD (n = 48); [b] BPD (n = 42), PTSD (n = 48); [c] HC (n = 42), BPD (n = 42), PTSD (n = 49). [d] HC (n = 41), BPD (n = 39), PTSD (n = 47). All p-values are Bonferroni-corrected for multiple comparisons.

Experimental measures of emotional reactivity and regulation

Emotional reactivity

A successful emotion induction was confirmed by a significant main effect of emotion category on *emotional valence ratings*, $F(2,262)=535.31$, $p<.001$, $\eta^2_p=.80$) (see Fig. 2A). Positive pictures induced higher emotional valence ($M=6.33$, $SD=1.20$, $p<.001$) than neutral pictures ($M=5.55$ $SD=0.73$) and negative pictures ($M=3.17$, $SD=0.94$, $p<.001$).

A significant group effect with a large effect size ($\eta^2_p=.19$) manifested wherein patients with BPD and PTSD exhibited decreased valence ratings compared to the control group ($F(2,131)=15.25$, $p<.001$). Moreover, a significant group by emotion category interaction with a moderate effect size ($\eta^2_p=.15$) was found ($F(4,262)=11.53$, $p<.001$). According to posthoc tests (Bonferroni-corrected), patients with BPD and patients with PTSD expressed significantly lower valence ratings in response to positive (BPD < HC: $d=-1.38$, $p<.01$; PTSD < HC: $d=-1.36$, $p<.01$) as well as to neutral pictures (BPD < HC: $d=-0.51$, $p<.05$; PTSD < HC: $d=-0.58$, $p<.01$) compared to HC. There were no significant group differences in valence ratings in response to negative pictures ($p>.05$).

Visual inspection of the *neurophysiological data* (see Table 2 and Fig. 2B and 2C) indicated larger P3 and LPP amplitudes in response to negative and positive vs. neutral pictures with the most pronounced emotional effects in patients with PTSD. Confirming this, the rmANOVA revealed a significant main effect of emotion category for the centro-parietal P3 ($F(2,264)=39.40$, $p<.001$, $\eta^2_p=.23$) and LPP ($F(2,264)=42.30$, $p<.001$, $\eta^2_p=.24$) amplitudes. For P3, this effect was qualified by a significant group by emotion category interaction with a medium effect size ($F(4,264)=4.71$, $p=.001$, $\eta^2_p=.07$). Interestingly, group differences were particularly prominent in response to neutral pictures, where P3 amplitudes were lowest for patients with PTSD and largest for HC (PTSD < BPD: $d=0.54\mu V$, $p<.01$; PTSD < HC: $d=0.96\mu V$, $p<.01$; BPD < HC: $d=0.96\mu V$, $p<.01$). Patients with PTSD also showed lower P3 amplitudes to negative pictures than HC ($d=0.51\mu V$, $p<.01$). For the LPP, the group by emotion category interaction did not reach statistical significance ($F(4,264)=1.51$, $p=.201$, $\eta^2_p=.02$). No

significant group effects were found in the P3 ($F(2,132)=1.65$, $p=.196$, $\eta^2_p=.02$) or LPP ($F(2,132)=2.43$, $p=.092$, $\eta^2_p=.04$).

To summarize, both patient groups showed reduced valence ratings for positive and neutral pictures compared with healthy controls. Regarding electrophysiological measures, both patient groups exhibited significantly lower P3 amplitudes to neutral pictures, and patients with PTSD additionally showed reduced P3 amplitudes to negative pictures. No group differences were observed in LPP amplitudes.

Table 2 P3 and LPP amplitudes in different conditions

		HC	**BPD**	**PTSD**
		M (SD)	***M (SD)***	***M (SD)***
P3 (CPC)	Neutral pictures*	1.03 (1.25)	0.62 (1.66)	0.07 (1.58)
	Negative pictures*	1.48 (1.37)	1.14 (1.72)	0.97 (1.71)
	Positive pictures	1.33 (1.40)	1.01 (1.69)	1.11 (1.66)
LPP (CPC)	Neutral pictures	0.42 (0.93)	0.11 (1.07)	0.36 (1.19)
	Negative pictures	1.16 (1.31)	0.81 (1.25)	1.36 (1.34)
	Positive pictures	0.89 (1.27)	0.72 (1.08)	1.35 (1.34)
P3 (FCC)	View negative pictures	-1.63 (1.68)	-2.02 (1.99)	-2.49 (1.94)
	Regulate negative pictures	-1.33 (1.57)	-1.67 (2.15)	-2.15 (2.14)
LPP (FCC)	View negative pictures	0.46 (1.31)	0.30 (1.38)	0.35 (1.22)
	Regulate negative pictures	1.04 (1.39)	0.51 (1.46)	1.11 (1.57)

Note. Amplitude in μV; *M*, Mean; *SD*, Standard Deviation; HC; Healthy Controls; BPD; Borderline Personality Disorder; PTSD, Post Traumatic Stress Disorder; CPC, Centro-Parietal Cluster; FCC, Frontro-Central Cluster. *significant interaction effect between group and picture category (for details see text).

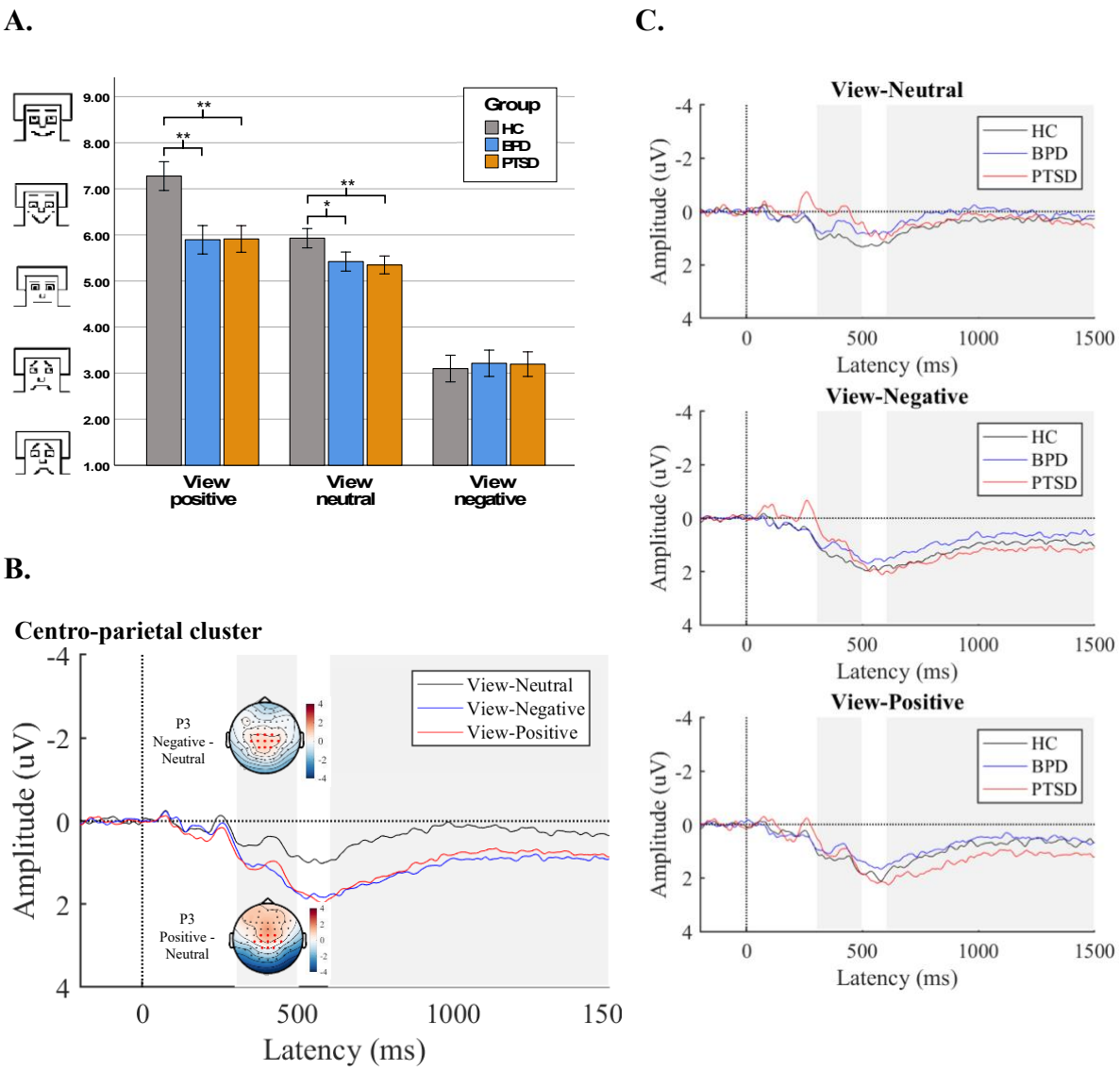


Fig. 2 Emotional valence ratings and ERP responses for emotional reactivity. (A) Emotional valence ratings; (B) ERP waveforms (μV) for viewing trials with neutral, negative, and positive pictures. Topographies show the difference in activity at the P3 (300 – 500 ms) for the negative vs. neutral and positive vs. neutral trials over the cluster of centro-parietal electrodes (marked in red). (C) ERP waveforms per condition.

Instructed emotion regulation

As displayed in Fig. 3A, participants of all groups showed increased *ratings of emotional valence* after cognitively reappraising (M=3.89, SD=0.91) vs. viewing negative pictures (M=3.17, SD=0.94) confirmed by a significant main effect of ER (F(1,131)=173.10, p<.001) with a large effect size (η^2_p=.57). Not consistent with our a priori hypothesis, the group by ER condition interaction did not reach statistical significance (F(2,131)=2.71, p=.07, η^2_p=.04). There was no significant effect of group (F(2,131)=0.21, p=.811, η^2_p=.003).

Visual inspection of *neurophysiological data* (see Table 2 and Fig. 3B and 3C) suggested more positive fronto-central P3 and LPP amplitudes in the reappraisal (decrease) vs. view condition across groups. Only the main effect of ER condition reached statistical significance (P3: F(1,132)=20.72, p<.001, η^2_p=.14; LPP: F(1,132)=26.87, p<.001, η^2_p=.17), while the group ER condition interaction did not reach statistical significance (P3: F(2,132)=0.04, p=.959, η^2_p=.001; LPP: F(2,132)=2.83, p=.063, η^2_p=.04). No significant group effects were found in the P3 (F(2,132)=2.31, p=.103, η^2_p=.03) or LPP (F(2,132)=1.04, p=.356, η^2_p=.02).

Thus, across groups, instructed cognitive reappraisal of negative pictures resulted in higher emotional valence ratings and increased fronto-central ERP amplitudes (P3 and LPP). However, no significant differences were observed between the patient groups and HC in the effect of reappraisal on ratings and ERPs.

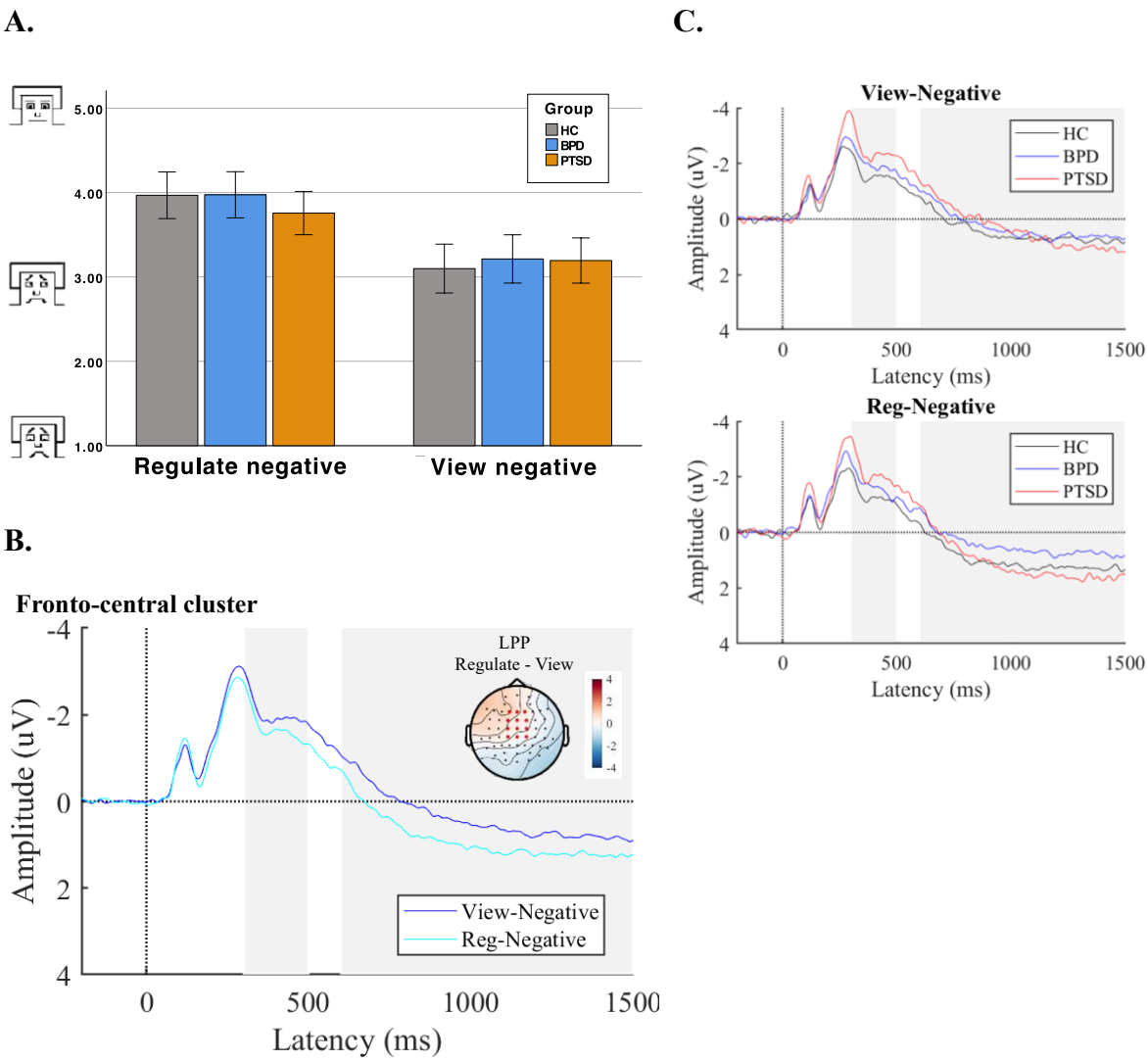


Fig. 3 Emotional valence ratings and ERP responses for instructed ER. (A) Emotional valence ratings; (B) ERP waveforms (μV) on ER trials. Topographies show the difference in activity at the LPP (600 – 1500 ms) for the view negative vs. regulate negative trials over the cluster of fronto-central electrodes (marked in red). (C) ERP waveforms per condition.

Self-reported measures of emotion regulation

Patients with BPD and PTSD reported significantly higher emotion dysregulation than HC (see Table 3 for significant group differences for total score and all subscales, all *ps*<.001). Effect sizes were large for all comparisons, with η^2 values ranging from .34 to .59. Bonferroni-corrected pairwise comparisons between patients with BPD and PTSD indicated that individuals with BPD reported significantly higher total DERS scores (p=.013, r≈0.26) and greater difficulties with impulse control (p<.001, r≈0.35). Across all post-hoc comparisons, effect sizes (r) ranged from .10 to .35, indicating small to moderate differences.

Table 3 Group comparisons of self-reported emotion dysregulation (DERS)

Characteristics M (SD)	**HC (n = 38)**	**BPD (n = 40)**	**PTSD (n = 45)**	**HC vs. BPD vs. PTSD** p, η^2	**PTSD vs. BPD** p (adj), r
Difficulties in Emotion Regulation Scale, DERS [a]					
Non-acceptance	10.84 (4.33)	22.15 (5.18)	19.36 (6.32)	**p<.001**, $\eta^2 \approx 0.45$	p=.225, r ≈ 0.16
Emotional awareness	13.08 (3.09)	20.59 (3.87)	18.49 (5.67)	**p< .001**, $\eta^2 \approx 0.34$	p=.120, r ≈ 0.19
Impulse control	8.11 (2.32)	20.21 (6.03)	13.71 (5.93)	**p<.001**, $\eta^2 \approx 0.52$	**p<.001**, r ≈ 0.35
Emotional clarity	8.26 (3.24)	17.33 (4.08)	14.51 (4.87)	**p<.001**, $\eta^2 \approx 0.44$	p=.051, r ≈ 0.22
ER strategies	12.79 (5.27)	27.63 (6.99)	23.89 (6.78)	**p<.001**, $\eta^2 \approx 0.49$	p=.216, r ≈ 0.17
Goal-directed behaviour	11.21 (4.05)	20.03 (4.43)	18.58 (5.08)	**p<.001**, $\eta^2 \approx 0.39$	p=.785, r ≈ 0.10
Total	64.29 (16.09)	127.95 (20.52)	108.53 (24.74)	**p<.001**, $\eta^2 \approx .59$	**p=.013**, r ≈ 0.26

Note. Kruskal-Wallis test statistics are reported for group comparisons. Effect sizes are reported as η^2 for overall tests and as r for pairwise post-hoc comparisons. Interpretation thresholds: $\eta^2 \geq .01$

(small), ≥ .06 (medium), ≥ .14 (large); r ≥ .10 (small), ≥ .30 (medium), ≥ .50 (large). Significant p-values are highlighted in bold. All p-values are Bonferroni-corrected for multiple comparisons.

Exploratory analyses of associations between childhood trauma, emotional reactivity, and emotion regulation

We first conducted exploratory correlation analyses to examine associations between childhood trauma (CTQ total score) and emotional valence ratings, ERP amplitudes and self-reported emotion dysregulation (DERS total score) (see Table 4 for details). Across groups, childhood trauma was positively associated with lower valence ratings in response to positive (r=-.35, p<.001) and neutral (r=-.22, p=.011) pictures in the experimental task and self-reported emotion dysregulation (r=.61, p<.001) but not with any of the ERP amplitudes (all rs≤-.15, ps≥.078).

To further explore these patterns, we conducted multiple regression analyses to examine whether childhood trauma was associated with valence ratings and self-reported emotion dysregulation, while accounting for BPD and PTSD diagnosis and medication use. When covariates were included in the model, childhood trauma was no longer significantly associated with valence ratings for positive (β=.07, 95% CI [−.17, .27], p=.548) or neutral pictures (β=.05, 95% CI [−.16, .31], p=.674). However, childhood trauma remained significantly associated with self-reported emotion dysregulation (β=.24, 95% CI [.08, .40], p=.004), alongside BPD diagnosis (β=.78, 95% CI [.61, .94], p<.001), PTSD diagnosis (β=.52, 95% CI [.33, .71], p<.001) and medication use (β=−.16, 95% CI [-.28, -.04], p=.010). BPD diagnosis uniquely explained 27.5% of the variance in DERS scores, with PTSD accounting for 9.2%, and CTQ and medication for 2.8% and 2.2%, respectively.

Finally, motivated by the divergence between self-report and neurophysiological findings, we conducted additional exploratory correlations between self-report (DERS total), valence ratings and ERP measures (see Table 4). In brief, these analyses showed that negative

correlations between a higher self-reported emotion dysregulation with lower valence ratings after positive (r=-.50, p<.001) and neutral pictures (r=-.34, p<.001) and lower valence after cognitive reappraisal (r=-.20, p=.026) in the experimental task. Of note, none of the questionnaires or valence ratings were significantly correlated with neurophysiological measures (all rs≤-.16, ps≥.066).

Table 4 Correlations between questionnaires, emotional valence ratings and ERP amplitudes in all participants (N = 135)

		1	2	3	4	5	6
1.	CTQ total	--					
2.	DERS Total	.607**	--				
3.	Valence ratings: negative pictures	.033	-.034	--			
4.	Valence ratings: neutral pictures	-.220*	-.338**	.218*	--		
5.	Valence ratings: positive pictures	-.348**	-.498**	-.192*	.611**	--	
6.	Valence ratings: cognitive reappraisal	-.052	-.201*	.754**	.460**	.057	--
7.	P3 CPC view negative	-.085	.045	.121	.060	-.010	.098
8.	P3 CPC view neutral	-.153	-.019	.079	.108	.078	.092
9.	P3 CPC view positive	-.015	.104	.045	-.024	-.052	.018
10.	LPP FCC view negative	.008	-.061	.129	.159	.030	.146
11.	LPP FCC regulate negative	-.046	-.028	.066	.149	.086	.013

Note. Pearson correlation coefficients, * p<0.05 ** p<0.01. CTQ, Childhood Trauma Questionnaire; DERS, Difficulties in Emotion Regulation Scale, CPC, Centro-Parietal Cluster; FCC, Frontro-Central Cluster. P-values are not corrected for multiple comparisons.

Discussion

The present study is the first to assess and compare emotional reactivity and regulation between patients with BPD and PTSD and healthy volunteers using a multimethodological

approach. We found decreased valence ratings to positive and neutral stimuli, alongside reduced P3 amplitudes in response to neutral stimuli in both patient groups, and in response to negative stimuli in patients with PTSD suggesting an hypo-reactivity. With regard to ER, there was a striking discrepancy between self-report and experimental data: While both patient groups reported significant emotion dysregulation, they did not differ in valence ratings nor neurophysiological data (P3, LPP) in the experimental ER task. Across participants, lower emotional valence ratings for neutral and positive pictures and greater self-reported emotion dysregulation were related to childhood trauma.

Emotional reactivity

In the experimental paradigm, participants displayed hypo-reactivity to (positive) and neutral images, as evidenced by reduced emotional valence ratings and P3 responses with large effect sizes. The groups did not differ in valence ratings in response to negative images and patients with PTSD additionally demonstrated decreased P3 amplitudes in response to negative images. These findings contradict our a priori hypothesis and previous research [30, 86]. A potential explanation could be the use of trauma-unrelated negative pictures. It has been shown that trauma-related negative stimuli (e.g., combat-related pictures) can elicit stronger P300 amplitude and lower valence ratings in PTSD patients vs. controls [32, 35, 36] and that patients with BPD and comorbid PTSD showed an emotional hypereactivity in valence ratings towards pictures with trauma-related content (i.e., sexual abuse) compared to HC [37]. This hyperreactivity towards trauma-related stimuli could be pronounced when compared to a reduced reactivity to neutral or trauma-unrelated stimuli [29, 34, 36, 41, 42, 87, 88]. Of note, since there is a lack of studies that systematically investigate this possible pattern of hyper- and hypo-reactivity in BPD and PTSD patients using ERP and valence ratings, the cited references include studies that assessed different emotional response systems, including self-reports,

ERPs, heart rate, and skin conductance. In the present study, a hypo-reactivity could be confirmed, where both patient groups showed lower valence ratings for positive and neutral pictures as well as reduced P3 responses to neutral pictures.

According to Javanbakht et al. [36], this phenomenon might be a consequence of a shift in attentional resource allocation following trauma exposure. Specifically, enhanced hypervigilance towards trauma-related stimuli might come at the expense of a hypo-reactivity towards neutral, unrelated stimuli [89]. Moreover, Adenauer et al. [90] have described this dampening of emotional reactions and reduced emotional discrimination as a "vigilance-avoidance" pattern, positing it as a defence mechanism aimed at avoiding the processing of stimuli unrelated to trauma that may be overwhelming. Neurocircuitry models have supported both an under-modulation of the prefrontal cortex (PFC) resulting in a hyperreactivity of the amygdala to trauma cues as well as an over-modulation of the PFC as dissociative shutdown of trauma-unrelated emotion processing in PTSD [91]. It is also proposed that this phenomenon primarily impacts early automatic bottom-up reactivity [36], potentially explaining why group differences were observed only in the P3 component and not in the LPP, which involves more conscious processing.

Emotion regulation

Consistent with our second hypothesis, both patient groups self-reported greater deficits in ER, which is consistent with previous literature [24, 51]. General emotion dyregulation of patients with BPD was significantly higher than of patients with PTSD. This difference between diagnostic groups was most pronounced with regard to emotional impulse control, consistent with previous research suggesting impulsivity as a core feature of BPD [92].

However, contrary to our third hypothesis there was neither a difference in valence ratings nor in neurophysiological data between the groups with regard to the effects of instructed cognitive reappraisal in the experiment. Nevertheless, these findings are in line with

previous studies showing a similar discrepancy between self-report and experimental measures of emotion (dys)regulation [30, 49, 54, 93]. This divergence may stem from the assessment of different processes and timescales of ER, as supported by the weak or absent associations between these measures in the current study. McRae [14] proposed that questionnaires measure the *long-term frequency* of using ER strategies in everyday life, while experiments evaluate the *short-term success* of employing a specific strategy as instructed in a laboratory.

This brings up the clinically and theoretically relevant question if patients with BPD and PTSD exhibit deficits in their short-term ability to regulate emotions or in their long-term habitual use of adaptive strategies. Some evidence suggests that emotion dysregulation in these patient groups is not characterized by an unsuccessful, but rather an infrequent use of adaptive ER strategies [24, 93]. Consistent with our findings, patients may retain the ability to consciously regulate emotions when instructed to do so but struggle in daily life to flexibly select and apply adaptive strategies, and may instead rigidly resort to maladaptive ones [9]. This latter aspect bears clinical significance, since effective ER entails the ability to flexibly choose regulation strategies tailored to the situational context to achieve a particular goal [94]. These findings underscore the importance of combining multimethod approaches of self-report and experimental measures to capture distinct aspects of emotion processing such as dispositional tendencies and in-the-moment regulatory performance.

Childhood trauma and emotional reactivity and regulation

In exploratory analyses, childhood trauma was associated with lower valence ratings for positive and neutral pictures, which, however, did not remain significant after controlling for diagnoses and medication. Moreover, no significant associations emerged between childhood trauma and ERP responses. These findings are partly in contrast with previous studies in healthy adults [63] and university students [64], which linked childhood trauma to dampened ERP responses to unpleasant vs. neutral pictures and to diminished ERP differences

between positive and neutral pictures in trauma-exposed adolescents [65]. This effect was interpreted as a desensitization to emotions or reduced affective discrimination following childhood trauma, which aligns partially with our observed (uncorrected) correlation between childhood trauma and lower valence ratings. However, given the exploratory nature of our analyses and the scarcity and inconsistencies of comparable research, no firm conclusions can be drawn from our study regarding the relationship between childhood trauma and valence ratings or neurophysiological ERP components in controls and patients with BPD and PTSD.

Lastly our fourth hypothesis was confirmed, as childhood trauma remained significantly associated with self-reported emotion dysregulation after controlling for diagnoses and medication use. While this result also stems from exploratory analyses, it offers preliminary support for the transdiagnostic explanatory value of childhood trauma beyond traditional diagnostic boundaries [3, 58, 95]. Future research should further examine the biopsychosocial mechanisms connecting childhood trauma and emotion dysregulation, considering both risk and resilience factors, to improve transdiagnostic interventions [96].

Limitations and future directions

Our study included a relatively large sample of patients with BPD and PTSD as well as healthy controls, with sensitivity analyses confirming adequate power to detect small-to-medium effects across key comparisons, lending strength to our multimethod investigation of emotional reactivity and regulation. Nevertheless, some limitations that may impact the generalizability and validity of the results warrant consideration. (1) Only female participants were included in this study, reducing the generalization of findings as gender differences have been found in ER [97]. (2) Comorbid disorders such as depression, which are highly frequent in the included patient samples, were not controlled for although they can profoundly influence emotion processing [98]. The reason for this is that negative affectivity is a key feature of BPD which is very difficult to disentangle from depressive mood. (3) Some patients with BPD had

a comorbid diagnosis of PTSD, which can impact the subjective and physiological emotional reactivity [54]. However, we repeated all main analyses after excluding BPD participants with comorbid PTSD and this did not significantly change our results (see supplementary material Table S1, S2, S3). (4) Our study used DSM-5-based diagnostic tools (e.g., CAPS-5, SCID) and did not assess complex PTSD (CPTSD), so no firm conclusions can be drawn about this diagnosis. However, given the high rate of severe childhood trauma in our sample, some participants may have met ICD-11 criteria for CPTSD. As emotion dysregulation is central to CPTSD (Ford & Courtois, 2021), future research should clearly distinguish between BPD, PTSD, and CPTSD to better understand shared and distinct mechanisms underlying these disorders. (5) While our findings support a dampened reactivity to trauma-unrelated stimuli, they do not clarify whether this hypo-reactivity is linked to a hyper-reactivity toward trauma-related stimuli.

Besides addressing these limitations, future research should explore emotion reactivity and regulation abilities with multimethodological designs on multiple time scales and with multiple measures [14]. Beyond retrospective questionnaires, emotional processes should be assessed in daily life using ecological momentary assessment [99, 100]. This approach enables the sampling of emotional reactivity and regulation in everyday situations, allowing to measure the flexibility in choosing adaptive vs. maladaptive strategies, effectiveness, and temporal interactions with other time-varying factors (e.g., dissociation or symptomatology). These more ecologically valid indices of emotion processing are highly relevant to the theoretical understanding of emotional dysfunction and to the improvement of treatment for BPD and PTSD.

Clinical implications

First, the dampened emotional reactivity to positive and neutral information in BPD and PTSD patients as well as negative trauma-unrelated information in PTSD patients,

underscores the potential benefit of interventions targeting emotional discrimination [66]. Attentional training could be used to enhance the recognition of safety signals, helping to counterbalance the allocation of attentional resources to threat-related socio-emotional cues [23, 58]. Secondly, if patients use adaptive ER strategies less frequently, therapists should support patients in flexibly choosing adaptive strategies based on the situational context [5]. Encouraging the generalization and practice of these skills in personalized and everyday contexts is paramount. Lastly, in the clinical assessment of patients' biographical histories, clinicians should remain mindful of the etiological significance of childhood trauma, which may contribute to changes in emotional reactivity and regulation [101].

Conclusions

This study suggests that patients with BPD and PTSD demonstrate weaker emotional valence and neurophysiological reactions (i.e., P3) towards (positive) and neutral information than healthy individuals in an experimental paradigm. Moreover, although both patient groups self-reported difficulties in ER, neither exhibited impaired cognitive reappraisal skills in experimental assessments. This was evidenced by similar emotional valence ratings and ERPs (i.e. P3/LPP amplitudes) compared to controls. These discrepancies underscore the importance of using multimethodological study designs and integrating more ecologically valid measures in future research, to further elucidate emotional dynamics, develop integrative models and enhance transdiagnostic therapeutic interventions.

Abbreviations

BDI-II	Beck Depression Inventory II
BPD	Borderline Personality Disorder
CTQ	Childhood Trauma Questionnaire
CPC	Centro-Parietal Cluster
DERS	Difficulties in Emotion Regulation
ERP	Event-Related Potentials
FCC	Frontro-Central Cluster
HC	Healthy Controls
PTSD	Post-Traumatic Stress Disorder
LPP	Late Positive Potential
rmANOVAs	Repeated-Measures Analyses of Variance

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki and received ethical approval from the ethics committee of the Department of Psychology at LMU Munich.

Consent for publication

Not applicable.

Availability of data and material

The final dataset will be made openly available on OSF upon completion of all preregistered analyses associated with the broader study (DRKS00019945). The data supporting this study's findings are available on request from the corresponding author.

Competing interests

We have no conflict of interest to disclose.

Funding

This work was funded by grants from the German Research Foundation (Deutsche Forschungsgemeinschaft, DFG), awarded to Katja Bertsch for her work on interoceptive processing in Borderline Personality Disorder (BE5292/ 3-2 and BE5292/5-1). I.G., K.B. and S.B. are supported by the German Center for Mental Health (DZPG) funded by the Federal Ministry of Education and Research (Bundesministerium für Bildung und Forschung [BMBF]) and the Ministry of Bavaria (01EE2303A).

Authors' contributions

IG and KB developed the current research question. However, the overall study concept and design were developed by SB and KB. Data collection was performed by IG and SB. The data analysis and interpretation were performed by IG, KB, QR, SB and LW. IG drafted the manuscript. All authors provided critical revisions and approved the final version of the manuscript for submission.

Acknowledgements

Not applicable.

References

1. Seligowski AV, Lee DJ, Bardeen JR, Orcutt HK. Emotion regulation and posttraumatic stress symptoms: a meta-analysis. Cogn Behav Ther. 2015;44(2):87-102. doi: 10.1080/16506073.2014.980753.
2. Carpenter RW, Trull TJ. Components of emotion dysregulation in borderline personality disorder: a review. Curr Psychiatry Rep. 2013;15(1):335. doi: 10.1007/s11920-012-0335-2.
3. Messman-Moore TL, Bhuptani PH. A review of the long-term impact of child maltreatment on posttraumatic stress disorder and its comorbidities: An emotion dysregulation perspective. Clinical Psychology: Science and Practice. 2017;24(2):154-69. doi: 10.1111/cpsp.12193.
4. Kuo JR, Khoury JE, Metcalfe R, Fitzpatrick S, Goodwill A. An examination of the relationship between childhood emotional abuse and borderline personality disorder features: The role of difficulties with emotion regulation. Child Abuse Negl. 2015;39:147-55. doi: https://doi.org/10.1016/j.chiabu.2014.08.008.
5. Linehan MM. Cognitive-behavioral treatment of borderline personality disorder. Guilford Press; 1993.
6. Río-Casanova Ld, González A, Páramo M, Dijke AV, Brenlla J. Emotion regulation strategies in trauma-related disorders: pathways linking neurobiology and clinical manifestations. Reviews in the Neurosciences. 2016;27(4):385-95. doi: doi:10.1515/revneuro-2015-0045.
7. Gross JJ. The emerging field of emotion regulation: An integrative review. Review of General Psychology. 1998;2(3):271-99. doi: 10.1037/1089-2680.2.3.271.
8. Gross JJ. Emotion regulation: Current status and future prospects. Psychological Inquiry. 2015;26(1):1-26. doi: 10.1080/1047840X.2014.940781.

9. Daros AR, Guevara MA, Uliaszek AA, McMain SF, Ruocco AC. Cognitive Emotion Regulation Strategies in Borderline Personality Disorder: Diagnostic Comparisons and Associations with Potentially Harmful Behaviors. Psychopathology. 2018;51(2):83-95. doi: 10.1159/000487008.
10. Ehring T, Quack D. Emotion Regulation Difficulties in Trauma Survivors: The Role of Trauma Type and PTSD Symptom Severity. Behavior Therapy. 2010;41(4):587-98. doi: https://doi.org/10.1016/j.beth.2010.04.004.
11. Dixon-Gordon KL, Gratz KL, Tull MT. Multimodal assessment of emotional reactivity in borderline personality pathology: the moderating role of posttraumatic stress disorder symptoms. Compr Psychiatry. 2013;54(6):639-48. doi: 10.1016/j.comppsych.2012.12.019.
12. Kockler TD, Tschacher W, Santangelo PS, Limberger MF, Ebner-Priemer UW. Specificity of emotion sequences in borderline personality disorder compared to posttraumatic stress disorder, bulimia nervosa, and healthy controls: an e-diary study. Borderline Personal Disord Emot Dysregul. 2017;4:26. doi: 10.1186/s40479-017-0077-1.
13. Gratz KL, Roemer L. Multidimensional Assessment of Emotion Regulation and Dysregulation: Development, Factor Structure, and Initial Validation of the Difficulties in Emotion Regulation Scale. Journal of Psychopathology and Behavioral Assessment. 2004;26(1):41-54. doi: 10.1023/B:JOBA.0000007455.08539.94.
14. McRae K. Emotion Regulation Frequency and Success: Separating Constructs from Methods and Time Scale. Social and Personality Psychology Compass. 2013;7(5):289-302. doi: https://doi.org/10.1111/spc3.12027.
15. Cludius B, Mennin D, Ehring T. Emotion regulation as a transdiagnostic process. Emotion. 2020;20(1):37-42. doi: 10.1037/emo0000646.

16. MacNamara A, Joyner K, Klawohn J. Event-related potential studies of emotion regulation: A review of recent progress and future directions. International Journal of Psychophysiology. 2022;176:73-88. doi: https://doi.org/10.1016/j.ijpsycho.2022.03.008.
17. Hajcak G, MacNamara A, Olvet DM. Event-related potentials, emotion, and emotion regulation: an integrative review. Dev Neuropsychol. 2010;35(2):129-55. doi: 10.1080/87565640903526504.
18. Olofsson JK, Nordin S, Sequeira H, Polich J. Affective picture processing: An integrative review of ERP findings. Biological Psychology. 2008;77(3):247-65. doi: https://doi.org/10.1016/j.biopsycho.2007.11.006.
19. Thiruchselvam R, Blechert J, Sheppes G, Rydstrom A, Gross JJ. The temporal dynamics of emotion regulation: an EEG study of distraction and reappraisal. Biol Psychol. 2011;87(1):84-92. doi: 10.1016/j.biopsycho.2011.02.009.
20. Schönfelder S, Kanske P, Heissler J, Wessa M. Time course of emotion-related responding during distraction and reappraisal. Soc Cogn Affect Neurosci. 2014;9(9):1310-9. doi: 10.1093/scan/nst116.
21. Füstös J, Gramann K, Herbert BM, Pollatos O. On the embodiment of emotion regulation: interoceptive awareness facilitates reappraisal. Social Cognitive and Affective Neuroscience. 2012;8(8):911-7. doi: 10.1093/scan/nss089.
22. Gan S, Yang J, Chen X, Yang Y. The electrocortical modulation effects of different emotion regulation strategies. Cogn Neurodyn. 2015;9(4):399-410. doi: 10.1007/s11571-015-9339-z.
23. Bertsch K, Hillmann K, Herpertz Sabine C. Behavioral and Neurobiological Correlates of Disturbed Emotion Processing in Borderline Personality Disorder. Psychopathology. 2018;51(2):76-82. doi: 10.1159/000487363.

24. Daros AR, Williams GE. A Meta-analysis and Systematic Review of Emotion-Regulation Strategies in Borderline Personality Disorder. Harv Rev Psychiatry. 2019;27(4):217-32. doi: 10.1097/hrp.0000000000000212.
25. Qaisar SB. Emotional Responsiveness in Borderline Personality Disorder: A Review of Self-Report and Physiological Measures. UTSC's Journal of Natural Sciences. 2021.
26. Litz BT, Litz BT, Gray MJ. Emotional Numbing in Posttraumatic Stress Disorder: Current and Future Research Directions. Australian & New Zealand Journal of Psychiatry. 2002;36(2):198-204. doi: 10.1046/j.1440-1614.2002.01002.x.
27. Nietlisbach G, Maercker A, Rösler W, Haker H. Are Empathic Abilities Impaired in Posttraumatic Stress Disorder? Psychological Reports. 2010;106(3):832-44. doi: 10.2466/pr0.106.3.832-844.
28. Clinchard C, Harp NR, Lorenz T, Neta M. Proposing a model whereby negative valence bias increases the risk for more severe dysphoric posttraumatic stress disorder and depression symptomology. Emotion. 2024. doi: 10.1037/emo0001335.
29. Bortolla R, Cavicchioli M, Fossati A, Maffei C. Emotional Reactivity in Borderline Personality Disorder: Theoretical Considerations Based on a Meta-Analytic Review of Laboratory Studies. J Pers Disord. 2020;34(1):64-87. doi: 10.1521/pedi_2018_32_382.
30. Marissen MA, Meuleman L, Franken IH. Altered emotional information processing in borderline personality disorder: an electrophysiological study. Psychiatry Res. 2010;181(3):226-32. doi: 10.1016/j.pscychresns.2009.10.006.
31. Lopez-Villatoro JM, Diaz-Marsa M, Garcia-Ramos A, Galvez-Merlin A, Carrasco JL. Altered emotional response pattern related to complex trauma in patients with borderline personality disorder. Clinical Psychology & Psychotherapy. 2023;30(6):1256-63. doi: https://doi.org/10.1002/cpp.2897.

32. Wolf EJ, Miller MW, McKinney AE. Emotional processing in PTSD: heightened negative emotionality to unpleasant photographic stimuli. J Nerv Ment Dis. 2009;197(6):419-26. doi: 10.1097/NMD.0b013e3181a61c68.

33. Wessa M, Karl A, Flor H. Central and peripheral psychophysiological responses to trauma-related cues in subclinical posttraumatic stress disorder: a pilot study. Exp Brain Res. 2005;167(1):56-65. doi: 10.1007/s00221-005-0007-0.

34. Burgmer M, Rehbein MA, Wrenger M, Kandil J, Heuft G, Steinberg C, et al. Early affective processing in patients with acute posttraumatic stress disorder: magnetoencephalographic correlates. PLoS One. 2013;8(8):e71289. doi: 10.1371/journal.pone.0071289.

35. Bleich AV, Attias J, Furman V. Effect of repeated visual traumatic stimuli on the event related P3 brain potential in post-traumatic stress disorder. Int J Neurosci. 1996;85(1-2):45-55. doi: 10.3109/00207459608986350.

36. Javanbakht A, Liberzon I, Amirsadri A, Gjini K, Boutros NN. Event-related potential studies of post-traumatic stress disorder: a critical review and synthesis. Biol Mood Anxiety Disord. 2011;1(1):5. doi: 10.1186/2045-5380-1-5.

37. Sauer C, Arens EA, Stopsack M, Spitzer C, Barnow S. Emotional hyper-reactivity in borderline personality disorder is related to trauma and interpersonal themes. Psychiatry Res. 2014;220(1-2):468-76. doi: 10.1016/j.psychres.2014.06.041.

38. Matzke B, Herpertz SC, Berger C, Fleischer M, Domes G. Facial reactions during emotion recognition in borderline personality disorder: a facial electromyography study. Psychopathology. 2014;47(2):101-10. doi: 10.1159/000351122.

39. Limberg A, Barnow S, Freyberger HJ, Hamm AO. Emotional vulnerability in borderline personality disorder is cue specific and modulated by traumatization. Biol Psychiatry. 2011;69(6):574-82. doi: 10.1016/j.biopsych.2010.10.024.

40. Lobbestael J, Arntz A. Emotional hyperreactivity in response to childhood abuse by primary caregivers in patients with borderline personality disorder. J Behav Ther Exp Psychiatry. 2015;48:125-32. doi: 10.1016/j.jbtep.2015.03.005.
41. Karl A, Malta LS, Maercker A. Meta-analytic review of event-related potential studies in post-traumatic stress disorder. Biol Psychol. 2006;71(2):123-47. doi: 10.1016/j.biopsycho.2005.03.004.
42. Flasbeck V, Juckel G, Brüne M. Evidence for Altered Neural Processing in Patients With Borderline Personality Disorder A Review of Event-Related Potential Studies. Journal of Psychophysiology. 2020;35. doi: 10.1027/0269-8803/a000271.
43. Salas F, Nvo-Fernández M, Leiva-Bianchi M, Sáez DA, Páeza GS, García MV, et al. Components of event-related potentials and borderline personality disorder: a meta-analysis. Eur J Psychotraumatol. 2023;15(1):2297641. doi: 10.1080/20008066.2023.2297641.
44. Popkirov S, Flasbeck V, Schlegel U, Juckel G, Brüne M. Alexithymia in borderline personality disorder is not associated with deficits in automatic visual processing of negative emotional stimuli. Psychiatry Res. 2018;263:121-4. doi: 10.1016/j.psychres.2018.03.009.
45. Andermann M, Izurieta Hidalgo NA, Rupp A, Schmahl C, Herpertz SC, Bertsch K. Behavioral and neurophysiological correlates of emotional face processing in borderline personality disorder: are there differences between men and women? Eur Arch Psychiatry Clin Neurosci. 2022;272(8):1583-94. doi: 10.1007/s00406-022-01434-4.
46. Izurieta Hidalgo NA, Oelkers-Ax R, Nagy K, Mancke F, Bohus M, Herpertz SC, et al. Time course of facial emotion processing in women with borderline personality disorder: an ERP study. J Psychiatry Neurosci. 2016;41(1):16-26. doi: 10.1503/jpn.140215.

47. Schneider I, Bertsch K, Izurieta Hidalgo NA, Müller LE, Schmahl C, Herpertz SC. Remnants and changes in facial emotion processing in women with remitted borderline personality disorder: an EEG study. Eur Arch Psychiatry Clin Neurosci. 2018;268(4):429-39. doi: 10.1007/s00406-017-0841-7.
48. Saar-Ashkenazy R, Shalev H, Kanthak MK, Guez J, Friedman A, Cohen JE. Altered processing of visual emotional stimuli in posttraumatic stress disorder: an event-related potential study. Psychiatry Res. 2015;233(2):165-74. doi: 10.1016/j.pscychresns.2015.05.015.
49. Fitzgerald JM, MacNamara A, DiGangi JA, Kennedy AE, Rabinak CA, Patwell R, et al. An electrocortical investigation of voluntary emotion regulation in combat-related posttraumatic stress disorder. Psychiatry Res Neuroimaging. 2016;249:113-21. doi: 10.1016/j.pscychresns.2015.12.001.
50. Woodward SH, Shurick AA, Alvarez J, Kuo J, Nonyieva Y, Blechert J, et al. A psychophysiological investigation of emotion regulation in chronic severe posttraumatic stress disorder. Psychophysiology. 2015;52(5):667-78. doi: https://doi.org/10.1111/psyp.12392.
51. Sorgi-Wilson KM, McCloskey MS. Emotion regulation strategies among individuals with borderline personality disorder relative to other groups: A review. 2022;29(5):1655-78. doi: https://doi.org/10.1002/cpp.2738.
52. Christ NM, Elhai JD, Forbes CN, Gratz KL, Tull MT. A machine learning approach to modeling PTSD and difficulties in emotion regulation. Psychiatry Research. 2021;297:113712. doi: https://doi.org/10.1016/j.psychres.2021.113712.
53. Koenigsberg HW, Fan J, Ochsner KN, Liu X, Guise KG, Pizzarello S, et al. Neural correlates of the use of psychological distancing to regulate responses to negative social

cues: a study of patients with borderline personality disorder. Biol Psychiatry. 2009;66(9):854-63. doi: 10.1016/j.biopsych.2009.06.010.

54. Krause-Utz A, Walther J-C, Lis S, Schmahl C, Bohus M. Heart rate variability during a cognitive reappraisal task in female patients with borderline personality disorder: the role of comorbid posttraumatic stress disorder and dissociation. Psychological Medicine. 2019;49(11):1810-21. doi: 10.1017/S0033291718002489.
55. McLaughlin KA, Colich NL, Rodman AM, Weissman DG. Mechanisms linking childhood trauma exposure and psychopathology: a transdiagnostic model of risk and resilience. BMC Medicine. 2020;18(1):96. doi: 10.1186/s12916-020-01561-6.
56. Girme YU, Jones RE, Fleck C, Simpson JA, Overall NC. Infants' attachment insecurity predicts attachment-relevant emotion regulation strategies in adulthood. Emotion. 2021;21(2):260-72. doi: 10.1037/emo0000721.
57. Brumariu LE, Owen MT, Dyer N, Lyons-Ruth K. Developmental Pathways to BPD-Related Features in Adolescence: Infancy to Age 15. J Pers Disord. 2020;34(Suppl B):104-29. doi: 10.1521/pedi_2020_34_480.
58. Flechsenhar A, Seitz KI, Bertsch K, Herpertz SC. The association between psychopathology, childhood trauma, and emotion processing. Psychol Trauma. 2024;16(Suppl 1):S190-s203. doi: 10.1037/tra0001261.
59. Assed MM, Khafif TC, Belizario GO, Fatorelli R, Rocca CCdA, de Pádua Serafim A. Facial Emotion Recognition in Maltreated Children: A Systematic Review. J Child Fam Stud. 2020;29(5):1493-509. doi: 10.1007/s10826-019-01636-w.
60. Nemeroff CB. Neurobiological consequences of childhood trauma. J Clin Psychiatry. 2004;65 Suppl 1:18-28.

61. Cross D, Fani N, Powers A, Bradley B. Neurobiological Development in the Context of Childhood Trauma. Clin Psychol (New York). 2017;24(2):111-24. doi: 10.1111/cpsp.12198.
62. Flasbeck V, Björn E, and Brüne M. Childhood trauma affects processing of social interactions in borderline personality disorder: An event-related potential study investigating empathy for pain. The World Journal of Biological Psychiatry. 2019;20(4):278-88. doi: 10.1080/15622975.2017.1333147.
63. Wu J, Liu Y, Zhang L, Wang N, Kohn N, Duan H. Integrating the pattern of negative emotion processing and acute stress response with childhood stress among healthy young adults. Stress. 2023;26. doi: 10.1080/10253890.2023.2195503.
64. Clarke SD, Riser DK, Schmidt MS. Electrocortical Correlates of Emotion Processing and Resilience in Individuals with Adverse Childhood Experiences. J Child Adolesc Trauma. 2024;17(3):1-16. doi: 10.1007/s40653-024-00621-w.
65. Sill J, Popov T, Schauer M, Elbert T. Rapid brain responses to affective pictures indicate dimensions of trauma-related psychopathology in adolescents. Psychophysiology. 2020;57(1):e13353. doi: 10.1111/psyp.13353.
66. Pincham HL, Bryce D, Kokorikou D, Fonagy P, Fearon RMP. Psychosocial Intervention Is Associated with Altered Emotion Processing: An Event-Related Potential Study in At-Risk Adolescents. PLOS ONE. 2016;11(1):e0147357. doi: 10.1371/journal.pone.0147357.
67. Loranger AW, Janca A, Sartorius N. Assessment and diagnosis of personality disorders: The ICD-10 international personality disorder examination (IPDE). Cambridge University Press; 1997.
68. Carcone D, Tokarz V, Ruocco A. A Systematic Review on the Reliability and Validity of Semistructured Diagnostic Interviews for Borderline Personality Disorder. Canadian Psychology/Psychologie canadienne. 2015;56:208-26. doi: 10.1037/cap0000026.

69. Weathers FW, Bovin MJ, Lee DJ, Sloan DM, Schnurr PP, Kaloupek DG, et al. The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5): Development and initial psychometric evaluation in military veterans. Psychol Assess. 2018;30(3):383-95. doi: 10.1037/pas0000486.

70. Müller-Engelmann M, Schnyder U, Dittmann C, Priebe K, Bohus M, Thome J, et al. Psychometric Properties and Factor Structure of the German Version of the Clinician-Administered PTSD Scale for DSM-5. Assessment. 2020;27(6):1128-38. doi: 10.1177/1073191118774840.

71. First MB, Williams JBW, Karg RS, Spitzer RL. Structured Clinical Interview for DSM-5 Disorders, Clinician Version (SCID-5-CV). American Psychiatric Association. 2016.

72. Osório FL, Loureiro SR, Hallak JEC, Machado-de-Sousa JP, Ushirohira JM, Baes CVW, et al. Clinical validity and intrarater and test–retest reliability of the Structured Clinical Interview for DSM-5 – Clinician Version (SCID-5-CV). Psychiatry and Clinical Neurosciences. 2019;73(12):754-60. doi: https://doi.org/10.1111/pcn.12931.

73. Bernstein DP, Stein JA, Newcomb MD, Walker E, Pogge D, Ahluvalia T, et al. Development and validation of a brief screening version of the Childhood Trauma Questionnaire. Child Abuse Negl. 2003;27(2):169-90. doi: 10.1016/s0145-2134(02)00541-0.

74. Wingenfeld K, Spitzer C, Mensebach C, Grabe HJ, Hill A, Gast U, et al. The German version of the Childhood Trauma Questionnaire (CTQ): preliminary psychometric properties. Psychotherapie, Psychosomatik, Medizinische Psychologie. 2010;60(11):442-50. doi: 10.1055/s-0030-1247564.

75. Spinhoven P, Penninx BW, Hickendorff M, van Hemert AM, Bernstein DP, Elzinga BM. Childhood Trauma Questionnaire: factor structure, measurement invariance, and validity across emotional disorders. Psychol Assess. 2014;26(3):717-29. doi: 10.1037/pas0000002.

76. Hallion LS, Steinman SA, Tolin DF, Diefenbach GJ. Psychometric Properties of the Difficulties in Emotion Regulation Scale (DERS) and Its Short Forms in Adults With Emotional Disorders. Frontiers in Psychology. 2018;Volume 9 - 2018. doi: 10.3389/fpsyg.2018.00539.
77. Ritschel L, Tone E, Schoemann A, Lim N. Psychometric Properties of the Difficulties in Emotion Regulation Scale Across Demographic Groups. Psychological assessment. 2015;27. doi: 10.1037/pas0000099.
78. Hautzinger M, Keller F, Kühner C. BDI-II. Beck-Depressions-Inventar. Revision. 2. Auflage. . Pearson Assessment. 2009.
79. Wang YP, Gorenstein C. Psychometric properties of the Beck Depression Inventory-II: a comprehensive review. Braz J Psychiatry. 2013;35(4):416-31. doi: 10.1590/1516-4446-2012-1048.
80. Kanske P, Heissler J, Schönfelder S, Bongers A, Wessa M. How to regulate emotion? Neural networks for reappraisal and distraction. Cereb Cortex. 2011;21(6):1379-88. doi: 10.1093/cercor/bhq216.
81. Moser JS, Hajcak G, Bukay E, Simons RF. Intentional modulation of emotional responding to unpleasant pictures: an ERP study. Psychophysiology. 2006;43(3):292-6. doi: 10.1111/j.1469-8986.2006.00402.x.
82. Bradley MM, Lang PJ. Measuring emotion: The self-assessment manikin and the semantic differential. Journal of Behavior Therapy and Experimental Psychiatry. 1994;25(1):49-59. doi: https://doi.org/10.1016/0005-7916(94)90063-9.
83. Lang PJ, Bradley MM, Cuthbert BN. International Affective Picture System (IAPS): Instruction manual and affective ratings. Gainesville: The Center for Research in Psychophysiology, University of Florida.: Technical report A-8.; 2008.

84. Maris E, Oostenveld R. Nonparametric statistical testing of EEG- and MEG-data. J Neurosci Methods. 2007;164(1):177-90. doi: 10.1016/j.jneumeth.2007.03.024.

85. Sackeim HA. The definition and meaning of treatment-resistant depression. J Clin Psychiatry. 2001;62 Suppl 16:10-7.

86. Lobo I, David IA, Figueira I, Campagnoli RR, Volchan E, Pereira MG, et al. Brain reactivity to unpleasant stimuli is associated with severity of posttraumatic stress symptoms. Biol Psychol. 2014;103:233-41. doi: 10.1016/j.biopsycho.2014.09.002.

87. Pfaltz MC, Schumacher S, Wilhelm FH, Dammann G, Seifritz E, Martin-Soelch C. Acoustic Emotional Processing in Patients With Borderline Personality Disorder: Hyper- or Hyporeactivity? J Pers Disord. 2015;29(6):809-27. doi: 10.1521/pedi_2015_29_176.

88. Herpertz SC, Kunert HJ, Schwenger UB, Sass H. Affective Responsiveness in Borderline Personality Disorder: A Psychophysiological Approach. The American journal of psychiatry. 1999;156(10):1550-6. doi: 10.1176/ajp.156.10.1550.

89. Zukerman G, Pinhas M, Icht M. Hypervigilance or shutdown? Electrophysiological processing of trauma-unrelated aversive stimuli after traumatic life events. Exp Brain Res. 2023;241(4):1185-97. doi: 10.1007/s00221-023-06578-w.

90. Adenauer H, Pinösch S, Catani C, Gola H, Keil J, Kissler J, et al. Early processing of threat cues in posttraumatic stress disorder-evidence for a cortical vigilance-avoidance reaction. Biol Psychiatry. 2010;68(5):451-8. doi: 10.1016/j.biopsych.2010.05.015.

91. Lanius RA, Vermetten E, Loewenstein RJ, Brand B, Schmahl C, Bremner JD, et al. Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. Am J Psychiatry. 2010;167(6):640-7. doi: 10.1176/appi.ajp.2009.09081168.

92. Links PS, Heslegrave R, van Reekum R. Impulsivity: core aspect of borderline personality disorder. J Pers Disord. 1999;13(1):1-9. doi: 10.1521/pedi.1999.13.1.1.

93. Sauer C, Sheppes G, Lackner HK, Arens EA, Tarrasch R, Barnow S. Emotion regulation choice in female patients with borderline personality disorder: Findings from self-reports and experimental measures. Psychiatry Res. 2016;242:375-84. doi: 10.1016/j.psychres.2016.04.113.

94. Aldao A, Nolen-Hoeksema S, Schweizer S. Emotion-regulation strategies across psychopathology: A meta-analytic review. Clin Psychol Rev. 2010;30(2):217-37. doi: 10.1016/j.cpr.2009.11.004.

95. Daros AR, Zakzanis KK, Ruocco AC. Facial emotion recognition in borderline personality disorder. Psychological Medicine. 2013;43(9):1953-63. doi: 10.1017/S0033291712002607.

96. Kerbage H, Purper-Ouakil D. How is trauma a transdiagnostic risk factor? A biopsychosocial model of risk and protective mechanisms following childhood trauma. European Psychiatry. 2024;67:S20 - S1.

97. Nolen-Hoeksema S. Emotion Regulation and Psychopathology: The Role of Gender. Annual Review of Clinical Psychology. 2012;8(Volume 8, 2012):161-87. doi: https://doi.org/10.1146/annurev-clinpsy-032511-143109.

98. Joormann J, Vanderlind WM. Emotion Regulation in Depression:The Role of Biased Cognition and Reduced Cognitive Control. Clinical Psychological Science. 2014;2(4):402-21. doi: 10.1177/2167702614536163.

99. Short NA, Boffa JW, Clancy K, Schmidt NB. Effects of emotion regulation strategy use in response to stressors on PTSD symptoms: An ecological momentary assessment study. Journal of Affective Disorders. 2018;230:77-83. doi: https://doi.org/10.1016/j.jad.2017.12.063.

100. Chaudhury SR, Galfalvy H, Biggs E, Choo T-H, Mann JJ, Stanley B. Affect in response to stressors and coping strategies: an ecological momentary assessment study of borderline

personality disorder. Borderline Personality Disorder and Emotion Dysregulation. 2017;4(1):8. doi: 10.1186/s40479-017-0059-3.

101. Hepp J, Schmitz SE, Urbild J, Zauner K, Niedtfeld I. Childhood maltreatment is associated with distrust and negatively biased emotion processing. Borderline Personality Disorder and Emotion Dysregulation. 2021;8(1):5. doi: 10.1186/s40479-020-00143-5.

Supplementary Material

International Affective Picture System (IAPS) codes of stimuli used in the emotion regulation experiment

Negative pictures

Low normative valence ratings (M=1.97; SD=1.30)

Codes: 2095, 2141, 2688, 2703, 2800, 2900, 3010, 3015, 3051, 3060, 3080, 3170, 3220, 3230, 3280, 3301, 3350, 3500, 6211, 6212, 6312, 6370, 6560, 6571, 6821, 6831, 6834, 6836, 6838, 9050, 9220, 9254, 9410, 9421, 9428, 9429, 9530, 9635.1, 9910

Neutral pictures

Moderate normative valence ratings (M=5.27; SD=1.40)

Codes: 2102, 2191, 2381, 2396, 2480, 2514, 2579, 2593, 2595, 2745.1, 2749, 2840, 2850, 5410, 5470, 7493, 7550, 7700

Positive Bilder

High normative valence ratings (M=7.71; SD=1.52)

Codes: 2030, 2058, 2071, 2208, 2209, 2216, 2310, 2340, 2352, 2360, 4623, 4626, 8120, 8350, 8420, 8490, 8496, 8540

Control analyses

Since 21.4% (n = 9) of the BPD patients also had a co-morbid diagnosis of PTSD, we wanted to check if potential confounding effects influenced the results and therefore repeated all main analyses after excluding these participants (N = 126).

Experimental measures of emotional reactivity and regulation

Emotional reactivity

Table S1 presents the results of the 3×3 rmANOVAs examining main and interaction effects of emotion (positive, neutral, negative) and group (HC, BPD, PTSD) on valence ratings and ERP amplitudes (P3 and LPP). The pattern of results mirrored the main analyses. For valence ratings, there were significant main effects of group and emotion (both p<.001), as well as a group × emotion interaction (p<.001): BPD and PTSD participants reported lower emotional valence after viewing positive and neutral images than HC participants, with no differences for negative images. ERP analyses revealed significant main effects of emotion for both P3 and LPP amplitudes (p<.001). P3 amplitudes were reduced in BPD and PTSD participants in response to neutral images, and in PTSD participants also to negative images, compared to HC.

Table S1 Statistics of the 3x3 rmANOVAs and post-hoc comparisons for valence ratings and P3 and LPP amplitude for the effect of emotion (positive, neutral, negative)

Dependent Variable	Effect	F(df)	p-value	η^2_p	Post-hoc comparisons (Bonferroni corrected)
Valence Ratings	Group	F(2, 122) = 14.48	**<.001**	.192	HC > BPD: d=0.55, p<.001; HC > PTSD: d=0.62, p<.001
	Emotion	F(2, 244) = 476.40	**<.001**	.796	Pos > Neu: d=0.81, Pos > Neg: d=3.17; Neu > Neg, d=2.36; all ps<.001
	Group × Emotion	F(4, 244) = 11.41	**<.001**	.158	Pos: BPD < HC: d=-1.35, p<.01; PTSD < HC: d=-1.36, p<.01;

					Neu: BPD < HC: *d*=-0.51, *p*<.05; PTSD < HC: *d*=-0.58, *p*<.05
P3 Amplitude	Group	*F*(2,123) = 1.85	.161	.029	
	Emotion	*F*(2, 246) = 34.74	**<.001**	.220	Pos > Neu, *d*= 0.56µV, *p*<.001 Neg> Neu, *d*= 0.59µV, *p*<.001
	Group × Emotion	*F*(4,246) = 4.99	**<.001**	.075	Neu: PTSD < HC: *d*=-0.96µV, *p*<.01; PTSD < BPD: *d*=-0.83µV, *p*<.01; Neg: PTSD < HC: *d*= -0.51µV, *p*<.01
LPP Amplitude	Group	*F*(2,123) = 1.60	.206	.025	
	Emotion	*F*(2, 246) = 36.62	**<.001**	.229	Pos > Neu, *d*=0.66µV, *p*<.001; Neg> Neu, *d*=0.78µV, *p*<.001
	Group × Emotion	*F*(4,246) = 1.80	.129	.028	

Note. Significant effects (*p*<.05) are reported in bold. *d*, mean difference

Instructed emotional regulation

Table S2 presents the results of the 2×3 rmANOVAs examining the effects of instructed emotion regulation (view vs. regulate) and group (HC, BPD, PTSD) on valence ratings and ERP amplitudes (P3 and LPP). As in the main analysis, significant main effects of regulation were found for valence ratings, P3, and LPP amplitudes (all *ps*<.001). No significant group effects or group × regulation interactions emerged, suggesting that BPD and PTSD patients did not differ from HC in the effect of instructed emotion regulation on valence ratings or ERPs (P3, LPP).

Table S2 Statistics of the 2x3 rmANOVAs and post-hoc comparisons for valence ratings and P3 and LPP amplitude for the instructed emotion regulation effect

Dependent Variable	Effect	F(df)	p-value	η^2 (partial)	Post-hoc comparisons (Bonferroni corrected)

Valence Ratings	Group	$F(2,122) = 0.54$	.586	.009	
	Regulation	$F(1,122) = 149.47$	**<.001**	.551	Reg > View, d=0.72, p<.001
	Group × Regulation	$F(2,122) = 2.55$	.083	.040	
P3 Amplitude	Group	$F(2,123) = 2.74$	.068	.043	
	Regulation	$F(1,123) = 24.30$	**<.001**	.165	Reg > View, d=0.38μV, p<.001
	Group × Regulation	$F(2,123) = 0.49$	.613	.008	
LPP Amplitude	Group	$F(2,123) = 0.31$	.733	.005	
	Regulation	$F(1,123) = 25.95$	**<.001**	.174	Reg > View, d=0.52, p<.001
	Group × Regulation	$F(2, 123) = 2.42$	.093	.038	

Note. Significant effects (p<.05) are reported in bold.

Self-reported measures of emotion regulation

Table S3 shows that, consistent with the main analysis, BPD and PTSD patients reported significantly greater emotion dysregulation than HC across all DERS subscales and the total score (all ps<.001). Additionally, BPD patients reported greater difficulties with impulse control (p=.001) compared to PTSD patients. Of note, the previously significant difference in total DERS scores between BPD and PTSD patients (p=.013) of the main analysis (see manuscript) became non-significant in the control analysis but approached significance (p=.053).

Table S3 Group comparisons of self-reported emotion dysregulation (DERS)

Characteristics *M (SD)*	**HC (*n* = 38)**	**BPD (*n* = 31)**	**PTSD (*n* = 45)**	**HC vs. BPD vs. PTSD**	**PTSD vs. BPD**
Difficulties in Emotion Regulation Scale, DERS					
Non-acceptance	10.84 (4.33)	21.61 (5.32)	19.36 (6.32)	***p*< .001**	*p*=.576
Emotional awareness	13.08 (3.09)	21.26 (3.98)	18.49 (5.67)	***p*<.001**	*p*=.076
Impulse control	8.11 (2.32)	20.13 (5.83)	13.71 (5.93)	***p*<.001**	***p*=.001**
Emotional clarity	8.26 (3.24)	17.58 (4.11)	14.51 (4.87)	***p*<.001**	*p*=.073
ER strategies	12.79 (5.27)	27.29 (7.24)	23.89 (6.78)	***p*<.001**	*p*=.426
Goal-directed behaviour	11.21 (4.05)	18.97 (4.32)	18.58 (5.08)	***p*<.001**	*p*=1.00
Total	64.29 (16.09)	126.84 (21.53)	108.53 (24.74)	***p*<.001**	*p*=.053

Note. Significant p-values are highlighted in bold. Bonferroni corrected.

Study III:

Fear of the Night: Links Between Emotion Dysregulation, Sleep Disturbances and Childhood Trauma Across Trauma-Related Disorders

Göhre, I., Back, S., Weiß, M., Ehring, T., Herpertz, S.C., Schmahl, C., Wolkenstein, L., Bertsch, K. (2025). Fear of the Night: Links Between Emotion Dysregulation, Sleep Disturbances and Childhood Trauma Across Trauma-Related Disorders.

Submitted for publication in the Journal of Aggression, Maltreatment & Trauma.

This chapter is a pre-copyedited version of a submitted article.

Fear of the Night: Links Between Emotion Dysregulation, Sleep Disturbances and Childhood Trauma Across Trauma-Related Disorders

Isabelle Göhre[1,2*], Sarah Back[1,2], Martin Weiß[3], Thomas Ehring[1,2], Sabine C. Herpertz[2,4,5], Christian Schmahl[2,5,6], Larissa Wolkenstein[1], Katja Bertsch[2,3]

[1] Department of Psychology, Ludwig-Maximilians-Universität München, München, Germany
[2] German Center for Mental Health (DZPG), München/Augsburg site
[3] Department of Psychology, Julius-Maximilians-Universität Würzburg, Germany
[4] Department of General Psychiatry, Center for Psychosocial Medicine, University of Heidelberg, Heidelberg, Germany
[5] German Center for Mental Health (DZPG), Mannheim/Heidelberg/Ulm site
[6] Department of Psychosomatic Medicine and Psychotherapy, Central Institute of Mental Health Mannheim, University of Heidelberg, Germany

* Corresponding Author: Isabelle Göhre, M.Sc., Dept of Psychology, LMU Munich, Germany, Leopoldstr. 13, 80802 Munich, Tel: 089-21805199, E-Mail: isabelle.goehre@psy.lmu.de

E-Mails address:
Isabelle Göhre, isabelle.goehre@psy.lmu.de
Sarah Back, Sarah.naomi.back@gmail.com
Martin Weiß, martin.weiss@uni-wuerzburg.de
Thomas Ehring, Thomas.Ehring@psy.lmu.de
Sabine C. Herpertz, sabine.herpertz@uni-heidelberg.de
Christian Schmahl, christian.schmahl@zi-mannheim.de
Larissa Wolkenstein, Larissa.Wolkenstein@psy.lmu.de
Katja Bertsch, katja.bertsch@uni-wuerzburg.de

Data availability statement: Data from Studies 1 and 3 cannot be shared due to restrictions on author permissions. The analysis code for these studies is available from the authors upon reasonable request. For Study 2, the data cannot be shared until all preregistered analyses have been completed.

Acknowledgments: This work was developed as part of the author's PhD dissertation at Ludwig-Maximilians-Universität München, Germany.

Funding statement: This work was funded by grants from the German Research Foundation (Deutsche Forschungsgemeinschaft, DFG), awarded to Katja Bertsch for her work on interoceptive processing in Borderline Personality Disorder (BE5292/ 3-2 and BE5292/5-1). I.G., S.B., T.E., S.C.H., C.S., and K.B. are supported by the German Center for Mental Health (DZPG) funded by the Federal Ministry of Education and Research (Bundesministerium für Bildung und Forschung [BMBF]) and the Ministry of Bavaria (01EE2303A).

Conflict of interest disclosure: We have no conflict of interest to disclose.

Ethical standards statement: This research was conducted in accordance with the ethical standards of the Ethics Committee of the Department of Psychology at LMU Munich and the Ethics Committees of the Medical Faculties of the University of Heidelberg, and adheres to the principles of the Declaration of Helsinki.

Patient consent statement: Informed consent was obtained from all participants included in the three studies.

Permission to reproduce material from other sources: Not applicable.

Clinical trial registration: Study 2 was pre-registered in the German Clinical Trials Register (ID: DRKS00019945, https://www.drks.de/search/de/trial/DRKS00019945/details). Study 3 was part of a larger project of the Clinical Research Unit KFO256 at Heidelberg University (https://gepris.dfg.de/gepris/projekt/190034061?language=en).

Abstract

Sleep disturbances are hallmark features of posttraumatic stress disorder (PTSD) and are associated with emotion dysregulation. This study examined links between emotion dysregulation and sleep in PTSD, while accounting for childhood trauma and hyperarousal and assessed whether these associations are PTSD-specific or extend to borderline personality disorder (BPD).

Three studies examined (i) whether emotion dysregulation is linked to sleep disturbances in PTSD beyond hyperarousal (Study 1), and (ii) whether this link is PTSD-specific (Studies 2, 3). Furthermore, (iii) the role of childhood trauma in sleep was investigated (Studies 1, 2, 3). Study 1 consisted of a naturalistic sample of *N*=128 PTSD-patients, while Studies 2 and 3 were case-control designs in individuals with PTSD (2: *n*=46, 3: *n*=23), BPD (2: *n*=42, 3: *n*=44), and healthy controls (HC, 2: *n*=48, 3: *n*=59).

Increased emotion dysregulation was linked to poorer sleep quality (β=.21, *p*=.018, R^2=.15) and higher fear of sleep (β=.35, *p*=.007, R^2=.33) beyond hyperarousal and childhood trauma (Study 1). Fear of sleep was highest in PTSD, while sleep disturbances were elevated in both PTSD and BPD versus HC (*ps*<.046, Studies 2,3). Emotion dysregulation predicted past-month and past-night sleep disturbances independently of diagnoses and childhood trauma (β=.25-.37; Studies 2,3). Correlations between childhood trauma and sleep vanished once emotion dysregulation was included.

Emotion dysregulation may significantly contribute to sleep disturbances in PTSD beyond hyperarousal. The link between childhood trauma and sleep disturbances may be mediated by emotion dysregulation. Sleep disturbances should be considered in standard diagnostic assessments and interventions of trauma-related disorders.

Keywords: sleep, emotion dysregulation, childhood trauma, PTSD, BPD

Introduction

Sleep disturbances are highly prevalent in posttraumatic stress disorder (PTSD). They manifest in insomnia (80–90%), nightmares (50–70%), altered objective sleep duration, efficiency and architecture, and reduced subjective sleep quality and nightmare distress (Harvey et al., 2003; Pigeon & Gallegos, 2015; Zhang et al., 2019). *Fear of sleep,* defined as the fear of being dangerously vulnerable during sleep and of recurrent nightmares and loss of control, is highly prevalent (Werner et al., 2020). Sleep disturbances are now considered hallmark features of PTSD due to their role in the development and maintenance and their links to poor outcomes such as lack of recovery and suicidality (Germain et al., 2017)

Hyperarousal models suggest that trauma triggers sustained physiological and psychological arousal, which interferes with sleep onset and continuity (Riemann et al., 2010; Semsar et al., 2021). However, hyperarousal does not fully account for nocturnal distress and is linked to difficulties modulating negative affect before sleep (Palagini et al., 2017; Werner et al., 2020). Therefore, a growing body of evidence highlights *emotion dysregulation*, defined as difficulties monitoring, evaluating, and modifying emotional states, as another potential mechanism in trauma-related sleep disturbances (Levin & Nielsen, 2009; Palmer & Alfano, 2017).

In healthy individuals, emotion dysregulation and sleep are bidirectionally linked across biopsychosocial domains (Palmer & Alfano, 2017). Daytime emotional dysregulation can spill into the evening, heightening pre-sleep hyperarousal, rumination and maladaptive behaviours (e.g., substance use) that impair sleep onset and maintenance (van Trigt et al., 2025; Werner et al., 2021). In turn, disrupted sleep can impair neural systems involved in affect regulation, reinforcing a vicious cycle (Goldstein & Walker, 2014). In trauma-exposed adults, global emotion dysregulation and maladaptive regulation strategies such as rumination (Wang

& Wang, 2025; Zhou et al., 2023) have been associated with poor *sleep quality* and *nightmares* (Fairholme et al., 2013).

However, neither sleep disturbances nor emotion dysregulation is unique to PTSD and is regarded as a transdiagnostic process (Fairholme et al., 2013; Harvey et al., 2011; van Trigt et al., 2025). For example, emotion dysregulation is the key feature of borderline personality disorder (BPD), a severe mental disorder characterized by instability in affect, self-image, and relationships (van Trigt et al., 2025). High prevalences of insomnia and nightmares have been found in BPD, and reduced *sleep quality* and *nightmares* have been associated with emotional sensitivity and dysregulation (Grove et al., 2016; Selby, 2013; Winsper et al., 2017). However, it remains unclear whether the association between emotion dysregulation and sleep disturbances is specific to PTSD, as direct comparisons with BPD are lacking.

Childhood trauma, including neglect and abuse before age 18, is a shared antecedent of PTSD and BPD and has been discussed as a transdiagnostic risk factor for both emotion dysregulation (McLaughlin et al., 2020) and sleep disturbances (Semsar et al., 2021). Longitudinal studies suggest that childhood trauma can exert life-long effects on neural systems involved in emotion regulation and sleep-wake regulation (Karatzoglou et al., 2024). However, it remains unclear whether childhood trauma is directly related to sleep-related problems in PTSD and BPD when emotion dysregulation and diagnostic status are controlled (Fairholme et al., 2013; Germain et al., 2017; Palmer & Alfano, 2017).

The present project aimed to address some of these open questions in three studies. In Study 1, we used a naturalistic outpatient PTSD sample to test whether emotion dysregulation is associated with past-month sleep disturbances (sleep quality, nightmare distress, fear of sleep) beyond hyperarousal. We additionally investigated the link between sleep disturbances and childhood trauma while controlling for emotion dysregulation. To test if the regulation-sleep link is specific to PTSD, we compared patients with PTSD to those with BPD and healthy

controls (HC) in Study 2. BPD was chosen as a clinical control group given the central role of emotion dysregulation and high childhood trauma prevalence. Again, associations between sleep disturbances and emotion dysregulation were explored and compared across groups. In Study 3, we aimed to replicate the findings of Study 2 on a different timescale by assessing sleep disturbances over the past night to capture both broad, trait-level patterns of habitual sleep (past-month) and state-level, short-term fluctuations (past-night).

Across studies, we hypothesized that emotion dysregulation is related to sleep disturbances in PTSD beyond hyperarousal, childhood trauma (Study 1) and diagnostic group (Study 2,3). We expected more sleep disturbances in PTSD and BPD compared to HC, reflected in lower sleep quality, higher nightmare distress, and fear of sleep (Study 2). Based on diagnostic criteria, we expected more pronounced alterations in individuals with PTSD than BPD. Given limited prior work, no a priori hypotheses could be formulated concerning differences in the strength of the regulation-sleep link between diagnoses. Furthermore, we expected childhood trauma to be associated with sleep disturbances when controlling for emotion dysregulation in PTSD, BPD and HC (Study 1,2,3).

Study 1

Study 1 investigated whether emotion dysregulation explains variance in sleep disturbances in PTSD beyond hyperarousal and whether associations between childhood trauma and sleep persist after accounting for emotion dysregulation. We analysed self-reported past-month sleep quality, nightmare distress and fear of sleep in treatment-seeking patients with PTSD.

Method

Participants

The final sample consisted of N=128 adult patients with current PTSD (65.6% women, M_{age}=34.77, SD=13.48). Participants were recruited from a specialized outpatient treatment

unit providing trauma-focused psychological treatment. Ethical approval for the study was obtained from a local institutional ethics committee. Patients presenting with acute suicidality or psychotic symptoms were not eligible for treatment. Comorbid disorders included affective disorders *(n*=51, 45.5%), anxiety disorders (*n*=8, 7.1%), eating disorders (*n*=4, 3.6%), and BPD (*n*=2, 1.7%). Power analysis indicated that N≥75 was required for an expected effect size of r=0.40 with 95% power and α=.05 (e.g., r=.38 in Zhou et al., 2023).

Procedure

As part of the standard diagnostic assessment, all patients took part in diagnostic interviews, including the Structured Clinical Interview for DSM-IV (SCID-CV, First et al., 2016) as well as the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5, Weathers et al., 2018) and filled in a questionnaire battery before the start of treatment.

Measures

Sleep disturbances.

(1) Sleep Quality. The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) was used to assess overall sleep quality during the past month. The 19 items use a four-point scale ranging from 0 (‘*not during the past month’*) to 3 (‘*three or more times a week’*). A general index is computed (range: 0-21), with higher scores indicating poorer sleep quality. An index score above 5 indicates a “*bad sleep quality*”. The PSQI has demonstrated good reliability and validity (Buysse et al., 1989) and acceptable internal consistency (Cronbach's alpha α=.76) in the present sample.

(2) Nightmare Distress. The German Version of the Nightmare Distress Questionnaire (NDQ) was used to examine various forms of distress induced by both posttraumatic and idiopathic nightmares (Böckermann et al., 2014). The NDQ includes 13 items scored from 1 (*'not at all'*) to 5 (*'often'*) with a higher total score (range: 13–65) indicating increased emotional distress caused by nightmares. The questionnaire has demonstrated satisfactory internal

consistency (Böckermann et al., 2014) and excellent reliability in the present sample (Cronbach's α=.88).

(3) Fear of Sleep. The Fear of Sleep Inventory – Short Form (FOSI-SF; Drexl et al., 2019) was used to measure fear of sleep. The FOSI-SF is a 13-item self-report instrument rated on a 5-point Likert scale ranging from 0 (*'not at all'*) to 4 (*'every night'*). The scale comprises two subcomponents: fear of loss of control and fear of darkness, with total scores ranging from 0-52 with higher scores reflecting greater fear of sleep. The German version (Drexl et al., 2019) has demonstrated excellent internal consistency in the general population (α=.87) and the current sample (Cronbach's α=0.95).

Emotion dysregulation. The Difficulties in Emotion Regulation Scale (DERS), developed by Gratz and Roemer (2004), was included to assess emotion dysregulation. Participants rated 36 items on a Likert-scale from 1="*almost never*" to 5="*almost always*", yielding a total score spanning from 36-180 with higher scores indicating greater severity of emotion dysregulation. The DERS has shown robust psychometric properties (Gratz & Roemer, 2004) and demonstrated good internal consistency in the current sample (Cronbach's α=.95).

Hyperarousal. The German version of the PTSD Checklist for DSM-5 (PCL-5; Weathers et al., 2013) was used as a self-report measure to estimate the severity of twenty PTSD symptoms within the past month. The PTSD symptom severity is rated on a Likert scale from 0="*not at all*" to 4="*extremely*" with higher values signifying more severe symptomatology. To assess hyperarousal severity, a total score was computed for items 15-19, excluding item 20 to avoid confounding with sleep measures. The PCL-5 is a valid, reliable and widely used measure with an excellent Cronbach alpha reliability coefficient of α=.95 in PTSD patients (Weathers et al., 2013). In the present sample, the hyperarousal cluster showed a Cronbach's alpha of α=.71.

Childhood Trauma. The Childhood Trauma Questionnaire – Short-Form (CTQ-SF) (Bernstein et al., 2003) is a 28-item retrospective self-report measure used to assess childhood trauma

before age 18 across five domains: emotional, sexual and physical abuse, and emotional and physical neglect. Items are rated on a five-point Likert scale (1 = "*never true*" to 5 = "*very often true*"), yielding a total score ranging from 28 to 140, where higher scores indicate greater trauma. The CTQ-SF has demonstrated solid psychometric properties (Bernstein et al., 2003) and high internal consistency in the current sample (Cronbach's α=.94).

Data Analysis

All analyses were conducted using R Studio (R version 4.5.1). We first ran Pearson correlations to investigate associations between sleep disturbances, emotion dysregulation, hyperarousal and childhood trauma. Second, we used multiple regression models to analyze whether emotion dysregulation explained variance in sleep disturbances while adjusting for hyperarousal. Finally, we looked at the relationship between childhood trauma and sleep disturbances when entering emotion regulation into the regression model.

Results

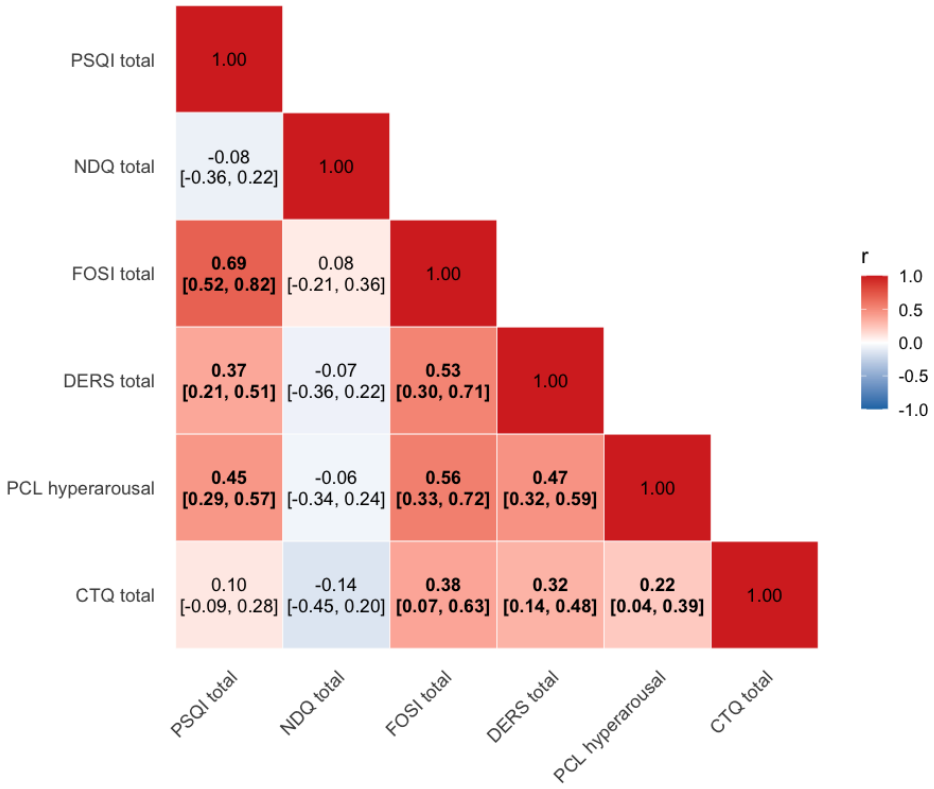
Participants showed high levels of sleep disturbance and emotion dysregulation (PSQI: M=10.02, SD=4.20; NDQ: M=40.11, SD=11.85; FOSI: M=12.26, SD=13.64; DERS: M=99.55, SD=28.88; PCL-5 hyperarousal: M=9.61, SD=4.32; CTQ: M=49.35, SD=20.69). Emotion dysregulation was significantly related to reduced sleep quality (r=.37, p<.001) and higher fear of sleep (r=.53, *p*<.001), but not to nightmare distress (r=-.07, *p*=.630). The same pattern emerged for hyperarousal (see Figure 1 for all correlations). As displayed in Table 1, emotion dysregulation remained a significant predictor of sleep quality (β=.21, *p*=.018) and fear of sleep (β=.35, *p*=.007) after adjusting for hyperarousal. Emotion dysregulation explained 15.4% of variance in sleep quality and 33.0% of variance in fear of sleep.

Finally, the significant association between childhood trauma and fear of sleep (r=.38, β=.38, *p*=.017) became insignificant (β=.17, *p*=.304) when emotion dysregulation was entered

into a regression model. Childhood trauma showed no significant associations with sleep quality (r=.10, *p*=.312) or nightmare distress (r=-.14, *p*=.412).

Figure 1

Correlations between Sleep, Emotion Dysregulation, Hyperarousal and Childhood Trauma



Note. Pairwise correlations. Bold values mark significant correlations, square brackets the 95% CI; PSQI: *n*=128, NDQ: *n*=35, FOSI: *n*=38.

Table 1

Multiple Regression Models Predicting Past-Month Sleep from Emotion Dysregulation and Hyperarousal in PTSD Patients

Model	*B*	*SE*	β [95% CI]	*t*	*p*	*F (df)*	*Adj. R^2*
Outcome: PSQI[a]						19.02 (2,125)	.22
DERS	0.03	0.01	.21 [.04, .39]	2.39	.018		
PCL hyp.	0.34	0.09	.35 [.17, .52]	3.92	<.001		
Outcome: NDQ[b]						0.13 (2,43)	-.04
DERS	-0.02	0.07	-.06 [-.41, .29]	-0.35	.730		
PCL hyp.	-0.09	0.47	-.03 [-.38, .31]	-0.19	.854		
Outcome: FOSI[c]						16.15 (2,47)	.38
DERS	0.17	0.06	.35 [.10, .62]	2.79	.007		
PCL hyp.	1.29	0.41	.40 [.15, .67]	3.13	.003		

Note. *B*=Unstandardized coefficient, *SE*=standard error, β=standardized coefficient, *t*=t-value, *p*=p-value, F(df_1, df_2) = overall model test, Adj. R^2=adjusted proportion of variance explained, PCL hyp=PCL hyperarousal subscale. [a] *n*=128, [b] *n*=46, [c] *n*=50.

Discussion

As hypothesized, lower sleep quality and higher fear of sleep were associated with emotion dysregulation even after controlling for hyperarousal in treatment-seeking PTSD patients. This confirms emotion dysregulation as a key factor in trauma-related sleep disturbances, complementing the well-established hyperarousal pathway (Levin & Nielsen, 2009; Palmer & Alfano, 2017; Riemann et al., 2010). Regression models further suggest that dysregulation may mediate links between childhood trauma and disturbed sleep as a transdiagnostic risk factor. In contrast to prior studies, neither emotion dysregulation nor hyperarousal was associated with nightmare distress. This null finding is difficult to interpret, given the lack of a healthy control group in the naturalistic design. It remains unclear whether these patterns are specific to PTSD or generalize to other trauma-exposed groups.

Study 2

Building on Study 1, we examined the specificity of sleep disturbances in a research sample of women with PTSD relative to women with BPD and HC in Study 2. We also investigated whether emotion dysregulation is linked to sleep disturbances independent of diagnosis and childhood trauma and whether these associations differ by group.

Method

Participants

Following the exclusion of 44 individuals with missing items or questionnaires, data from *n*=46 women with PTSD (M_{age}=27.5; *SD*=7.94), *n*=42 women with BPD (M_{age}=26.8; SD=7.65), and *n*=48 women without any psychiatric diagnosis (M_{age}=26.7, *SD*=4.46) were included in the current analysis. The study was part of a larger research project that was preregistered in a clinical trials registry. The data do not overlap with those used in Studies 1 or 3. Interview and self-report data were collected during an on-site visit prior to participation in laboratory experiments. Participants were recruited from the general population through advertisements and from in- and outpatient treatment centres, day clinics, and trauma support organisations. All participants received monetary compensation for their participation. Ethical approval was granted by the local Ethics Committee, and the study was conducted in accordance with the Declaration of Helsinki.

General exclusion criteria were: age<18 or >50 years, BMI>17 or <30, chronic medical illnesses or cardiovascular abnormalities, substance abuse/ dependence in the past 6 months, lifetime diagnosis of bipolar disorder or schizophrenia. Patients with PTSD had to have a DSM-5 diagnosis of PTSD, but were not allowed to fulfil more than three diagnostic criteria of BPD. Conversely, BPD patients had to fulfil a DSM-5 diagnosis of BPD (≥5 diagnostic criteria) but could not meet criteria for PTSD. The healthy individuals were not allowed to have any current or past mental disorders or current medication (except for contraceptives). The most prevalent

comorbid diagnoses were: affective disorders (PTSD: *n*=25, 54.3%; BPD: *n*=18, 42.9%), anxiety disorders (PTSD: *n*=7, 15.2%, BPD: *n*=7, 16.7%), eating disorders (PTSD: *n*=7, 15.2%, *n*=7, 16.7%), attention deficit hyperactivity disorder (PTSD: *n*=3, 6.5%; BPD: *n*=6, 14.3%), and obsessive compulsive disorder (PTSD: *n*=3, 6.5%; BPD: *n*=1, 2.4%). A power analysis indicated that ≥102 participants were required for 95% power to detect an expected effect of r=.40 (f^2=.19) at α=.05.

Procedure

After screening and informed consent, participants completed questionnaires and diagnostic interviews including SCID-CV, CAPS-5 (Weathers et al., 2018) and the BPD section of the International Personality Disorder Examination (IPDE, Loranger et al., 1997). Trained assessors performed all interviews.

Measures

The same questionnaires as in Study 1 assessed sleep quality (PSQI, Cronbach's α=.79), nightmare distress (NDQ, α=.93), fear of sleep (FoSI, α=.92), emotion dysregulation (DERS, α=.97), and childhood trauma (CTQ-SF, α=.95).

Data Analysis

Group differences were tested using Kruskal-Wallis rank sum tests with Bonferroni correction due to not-normally distributed data (Shapiro–Wilk test: p<.05). Pearson correlations examined associations between sleep disturbances and emotion dysregulation in the total sample. Multiple regression analyses tested the association between sleep disturbances and emotion dysregulation as well as childhood trauma while entering group as variable. Group × DERS interaction terms were evaluated with Type-III sums-of-squares tests.

Results

Descriptive statistics and results of the Kruskal–Wallis tests with Bonferroni-adjusted post hoc comparisons are summarized in Table 2. The PTSD and the BPD group reported

significantly lower sleep quality, higher nightmare distress, and higher fear of sleep compared to healthy controls (PSQI: H(2)=64.17, ε^2=.47; NDQ: H(2)=44.66, ε^2=.41; FOSI: H(2)=67.28, ε^2=.49, all *ps*<.001). Fear of sleep was higher in patients with PTSD than in those with BPD (*z*=-2.89, *p*=.012, *r*=-.25). Both patient groups also reported significantly higher levels of emotion dysregulation (H(2)=79.5, *p*<.001, ε^2=.58) and childhood trauma (H(2)=75.90, *p*<.001, ε^2=.56) compared to healthy controls. Emotion dysregulation was significantly higher in BPD than in PTSD (*z*=2.58, *p*=.029, *r*=.22). In the total sample, poor sleep quality, higher nightmare distress, and higher fear of sleep were significantly associated with emotion dysregulation and childhood trauma (all r≥.45, all *ps*<.001, see Figure 2).

Table 2

Group Comparisons of Past-Month Sleep, Emotion Dysregulation and Childhood Trauma

	HC (n = 46)		PTSD (n = 48)		BPD (n = 42)		HC vs. PTSD	HC vs. BPD	BPD vs. PTSD
	M	*SD*	*M*	*SD*	*M*	*SD*	*p*	*p*	*p*
Sleep variables									
PSQI[a]	3.87	1.72	9.60	3.59	7.71	2.81	<.001	<.001	.137
NDQ[b]	22.61	5.99	38.92	10.13	37.81	9.43	<.001	<.001	1.00
FOSI[c]	1.09	2.79	14.33	9.26	9.29	11.39	<.001	<.001	.012
DERS[a]	64.98	16.97	107.85	24.22	125.93	21.64	<.001	<.001	.029
CTQ[a]	31.26	9.26	65.58	16.40	55.90	15.91	<.001	<.001	.104

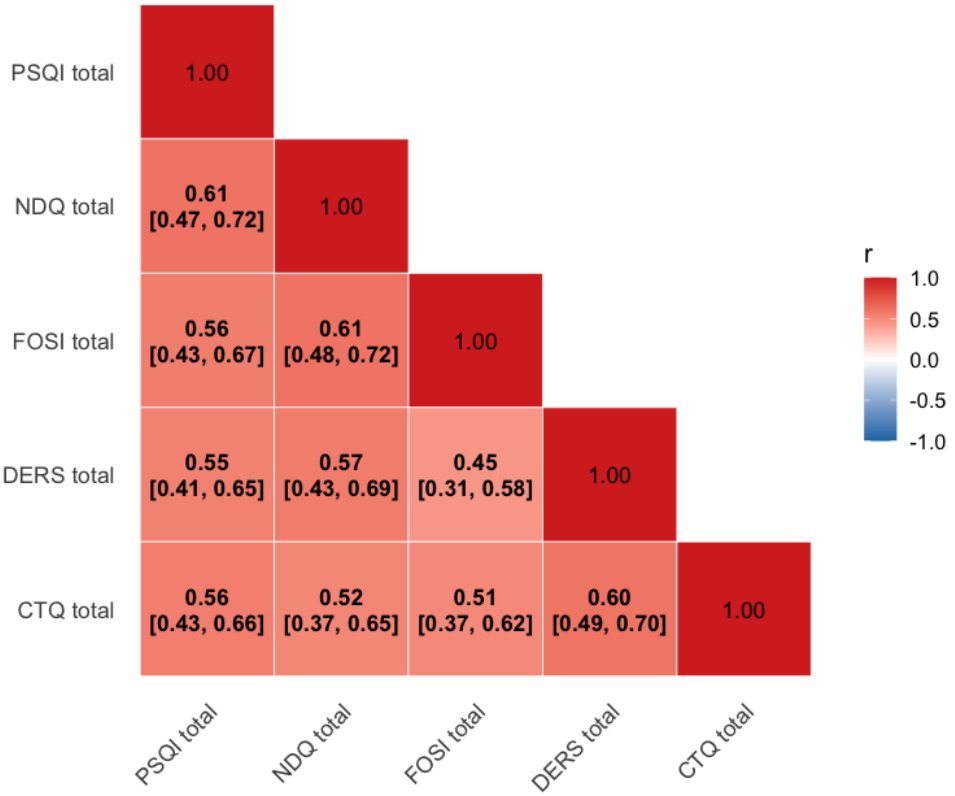
Note. PSQI, Pittsburgh Sleep Quality Inventory; FOSI, Fear of Sleep Inventory; NDQ, Nightmare Distress Questionnaire; DERS, Difficulties in Emotion Regulation Scale; CTQ, Childhood Trauma Questionnaire. Bonferroni correction was applied.

[a] *n*=136, [b] *n*=107, [c] *n*=135.

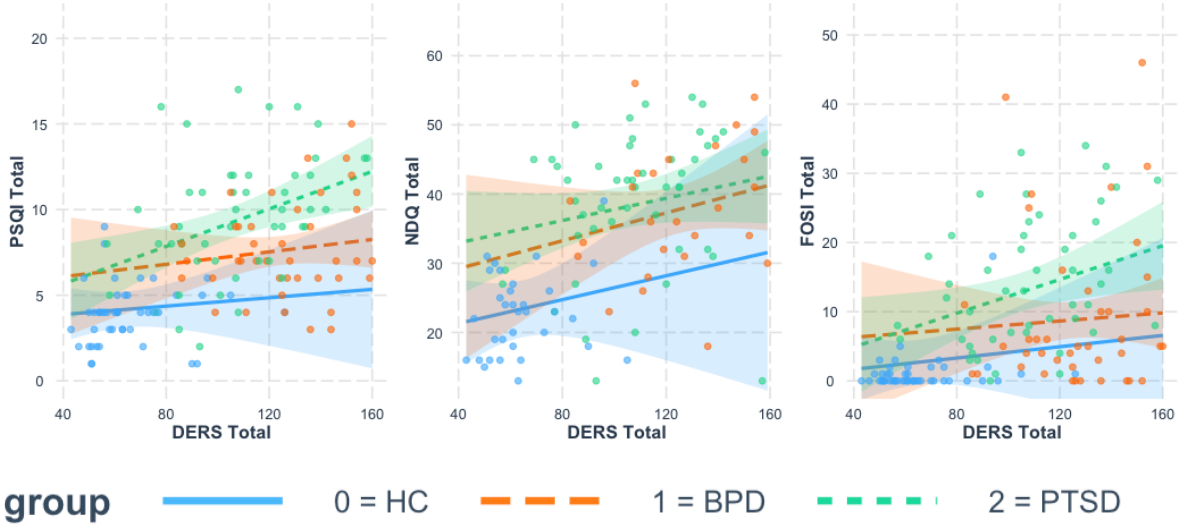
Figure 2

Associations Between Past-Month Sleep, Emotion Dysregulation and Childhood Trauma

A.



B.



Note. A. Pairwise correlations between sleep variables, DERS and CTQ; PSQI: *n*=136, NDQ: *n*=107, FOSI: *n*=135. B. Interaction plot of the relationship between sleep variables and emotion dysregulation by group (HC, BPD, PTSD). Shaded areas represent 95% CI.

As displayed in Table 3, results of multiple regression analyses revealed that emotion dysregulation remained significantly associated with sleep quality (β=.30, p=.005), nightmare distress (β=.24, p=.042), and fear of sleep (β=.25, p=.037) when controlling for diagnostic group and childhood trauma as additional predictors. Emotion dysregulation uniquely explained 5.8% of the variance in PSQI scores, 4.0% in nightmare distress, and 3.3% in fear of sleep. While PTSD consistently predicted poorer sleep relative to HC, individuals with BPD differed from HC only in nightmare distress (β=.34, p=.008). No association between childhood trauma and any sleep disturbances were found (all ps>.123) when group and emotion dysregulation were entered into the model. For all three types of sleep disturbances, the interaction between group and emotion dysregulation was not significant, indicating that the association between emotion dysregulation and sleep disturbances did not differ across the three groups (all ps>.218).

Table 3

Multiple Regression Models Predicting Past-Month Sleep from Emotion Dysregulation, Diagnostic Group, and Childhood Trauma

Model	*B*	*SE*	β [95% CI]	*t*	*p*	*F (df)*	*Adj. R^2*
Outcome: PSQI[a]						29.77 (4,131)	.46
DERS total	0.03	0.01	.30 [.09, .50]	2.85	.005		
HC vs. PTSD	3.71	0.86	1.00 [.54, 1.46]	4.30	< .001		
HC vs. BPD	1.38	0.92	.37 [-.12, .86]	1.50	.135		
CTQ total	0.02	0.02	.10 [-.09, .29]	0.99	.323		
Outcome: NDQ[b]						20.62 (4,102)	.43
DERS total	0.09	0.04	.25 [.01, .49]	2.06	.042		
HC vs. PTSD	11.22	3.10	.98 [.44, 1.51]	3.63	< .001		
HC vs. BPD	9.11	3.38	.79 [.21, 1.38]	2.70	.008		
CTQ total	0.04	0.06	.07 [-.16, .29]	0.59	.559		

Outcome: FOSI[c]						17.59 (4,130)	.33
DERS total	0.08	0.04	.25 [.01, .48]	2.10	.037		
HC vs. PTSD	7.16	2.66	.71 [.19, 1.23]	2.69	.008		
HC vs. BPD	1.43	2.87	.14 [-.42, .70]	0.50	.619		
CTQ total	0.08	0.05	.17 [-.05, .38]	1.55	.123		

Note. *B*=Unstandardized coefficient, *SE*=standard error, *β*=standardized coefficient, *t*=t-value, *p*=p-value, F(df_1, df_2) = overall model test, Adj. R^2=adjusted proportion of variance explained, Bold values represent significant predictors.

[a] *n*=136, [b] *n*=107, [c] *n*=135.

Discussion

Study 2 replicated and extended Study 1 using a case-control design. Consistent with our transdiagnostic hypothesis, PTSD and BPD groups reported higher sleep disturbances, including poorer sleep quality, higher nightmare distress and fear of sleep, than HC. In line with diagnostic profiles, fear of sleep was more pronounced in PTSD, while emotion dysregulation was higher in BPD. After entering emotion dysregulation and childhood trauma in regression models, differences between BPD and HC in sleep quality and fear of sleep were no longer significant. Childhood trauma was not associated with sleep disturbances beyond emotion dysregulation.

In line with our hypotheses, emotion dysregulation showed a significant and similarly strong association with sleep disturbances across groups, remaining significant after controlling for childhood trauma and group, indicating a transdiagnostic link. Unlike Study 1, significant correlations with nightmare distress emerged, possibly due to greater sample heterogeneity, increasing the power to detect an association. Taken together, Studies 1 and 2 established a consistent link between emotion dysregulation and past-month sleep disturbances in PTSD, BPD, and HC. However, it remains unclear whether these findings are affected by night-to-night variability of sleep.

Study 3

Study 3 tested whether the findings of Study 2 extend to state-level, short-term sleep fluctuations and to test diagnostic group differences (PTSD, BPD, HC). We compared groups on global sleep quality, restorative value of sleep, evening psychological balance, pre-sleep exhaustion and psychosomatic symptoms in the past night and examined associations between sleep disturbances, emotion dysregulation and childhood trauma.

Method

Participants

In total, n=23 patients with PTSD (M_{age}=33.84, SD=10.97), n=44 patients with BPD (M_{age}=27.43, SD=7.50), and n=59 healthy participants (M_{age}=29.59, SD=7.72) were included in the current study. The data were part of a larger project. Participants were recruited via municipal registration offices, advertisements, and clinical referrals (in- and outpatients). General exclusion criteria comprised: neurological disorders or severe medical illness, substance abuse in the past two months or dependence in the past 12 months, use of psychotropic medication in the past two weeks, diagnosis of schizophrenia, schizoaffective or bipolar disorder. Healthy participants were not allowed to have a history of psychiatric diagnoses or psychological/psychiatric treatment. Individuals with PTSD were not allowed to have a current or lifetime diagnosis of BPD. Participants with BPD had to meet at least five DSM-IV criteria for BPD, but were excluded if they currently fulfilled diagnostic criteria for PTSD. The study received approval from a local medical ethics committee, in accordance with the principles of the Declaration of Helsinki.

Participants were mostly women (HC: 67.8%, PTSD: 100%, BPD: 59.1%) and were between 18 and 54 years old. The most prevalent co-morbid diagnoses were: affective disorders (PTSD: n=5, 21.7%; BPD: n=26, 59.1%), anxiety disorders (PTSD: n=8, 34.8%; BPD: n=10,

22.7%), eating disorders (PTSD: *n*=4, 17.4%; BPD: *n*=10, 22.7%), somatoform disorders and hypochondria (PTSD: *n*=2, 8.7%; BPD: *n*=4, 9.1%) and OCD (PTSD: *n*=0; BPD: *n*=1, 2.3%).

Procedure

After in- and exclusion criteria had been checked in a telephone screening and informed consent had been signed, participants filled in a questionnaire battery and took part at a SCID-I interview for DSM-IV and the International Personality Disorder Examination (IPDE, Loranger et al., 1997) for the assessment of BPD. Participants completed a questionnaire battery and structured diagnostic interviews conducted by trained assessors. They were subsequently enrolled in additional experimental studies as part of a broader research consortium. Participation was financially compensated.

Measures

The same questionnaires were used as in Studies 1 and 2 to assess emotion dysregulation (DERS) and childhood trauma (CTQ-SF). However, since we were interested in sleep disturbances the previous night, the German Sleep Questionnaire ("Schlaffragebogen"-A, SF-A/R; Görtelmeyer, 2011) was used, which was completed in the morning by all participants. The SF-A is a 25-item inventory with five subscales: sleep quality (SQ), restorative value of sleep (GES), psychological balance/mood before sleep (PSYA), psychological exhaustion before sleep (PSYE), and psychosomatic symptoms at sleep onset (PSS). Items are scored on a Likert scale from 1-5, with lower scores reflecting more sleep disturbances, except for the variables "psychological exhaustion" and "psychosomatic symptoms", which are scored in reverse, i.e., higher scores reflecting more disturbances.

Data Analysis

To replicate the analyses of Study 2, we applied the same statistical procedures (Kruskal–Wallis rank-sum tests, Pearson correlations, multiple regression analyses) and power analysis. The authors do not have permission to share the data.

Results

Patients with PTSD and with BPD reported higher sleep disturbances in the past night compared to healthy controls (*ps*<.046, see Table 4): Sleep quality (SF-SQ: H(2)=48.07, *p*<.001, ε^2=.40) and restorative value of sleep (SF-GES: H(2)=45.68, *p*<.001, ε^2=.38) were lower, pre-sleep psychological balance (SF-PSYA: H(2)=66.57, *p*<.001, ε^2=.56) poorer, psychological exhaustion before sleep (SF-PSYE: H(2)=17.08, *p*<.001, ε^2=.13) and psychosomatic symptoms at sleep onset (SF-PSS: H(2)=25.96, *p*<.001, ε^2=.19) higher in both patient groups. No significant differences were found between the two patient groups in any of the past night's sleep disturbances (*ps*≥.162). Consistent with these patterns, both patient groups scored substantially higher than HC on emotion dysregulation (DERS: H(2)=85.15, *p*<.001, ε^2=.68) and childhood trauma (CTQ: H(2)=52.96, *p*<.001, ε^2=.41), whereas individuals with PTSD and BPD did not differ on either the DERS or CTQ scores (*ps*≥.346).

Table 4

Group Comparisons of Past-Night Sleep, Emotion Dysregulation and Childhood Trauma

	HC (n = 59)		PTSD (n = 23)		BPD (n = 44)		HC vs. PTSD	HC vs. BPD	BPD vs. PTSD
	M	*SD*	*M*	*SD*	*M*	*SD*	*p*	*p*	*p*
SF sleep variables									
SF-SQ	3.78	0.59	2.40	0.89	2.60	0.93	<.001	<.001	1.00
SF-GES	3.69	0.69	2.47	0.85	2.58	0.77	<.001	<.001	1.00
SF-PSYA	3.88	0.61	2.23	0.87	2.35	0.86	<.001	<.001	1.00
SF-PSYE	2.80	0.66	3.67	0.86	3.32	0.88	<.001	.046	.162
SF-PSS	1.18	0.32	1.76	0.65	1.52	0.48	<.001	<.001	.603
DERS	63.75	14.08	109.78	19.23	113.80	18.20	<.001	<.001	1.00
CTQ	36.05	13.36	74.04	28.09	58.20	19.79	<.001	<.001	.346

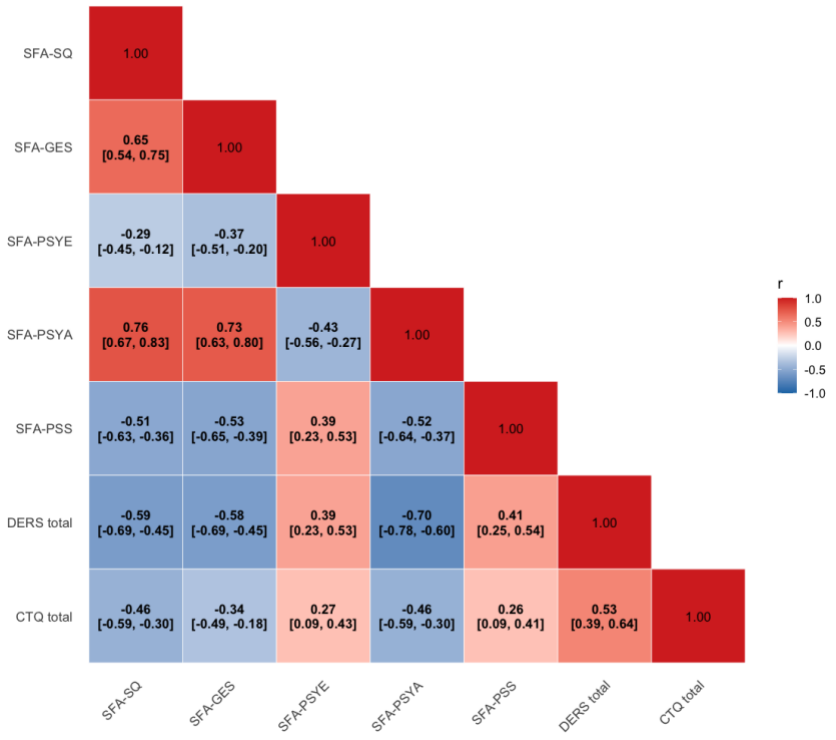
Note. SF-SQ, sleep quality; SF-GES, restorative value of sleep; PSYA, psychological balance before sleep; PSYE, psychological exhaustion before sleep; SF-PSS, psychosomatic symptoms at sleep onset. Bonferroni correction for multiple comparisons was used.

Emotion dysregulation was significantly associated with all types of past night's sleep disturbances. In the total sample, correlations were largest for the link to reduced psychological balance before sleep (r=-.70, *p*<.001), sleep quality (r=-.59, *p*<.001) and restorative value of sleep (r=-.58, *p*<.001). As depicted in Figure 3, all correlations among the sleep-related variables, emotion dysregulation, and childhood trauma were significant, ranging from weak to strong in magnitude (r≥.26, *ps*<.004).

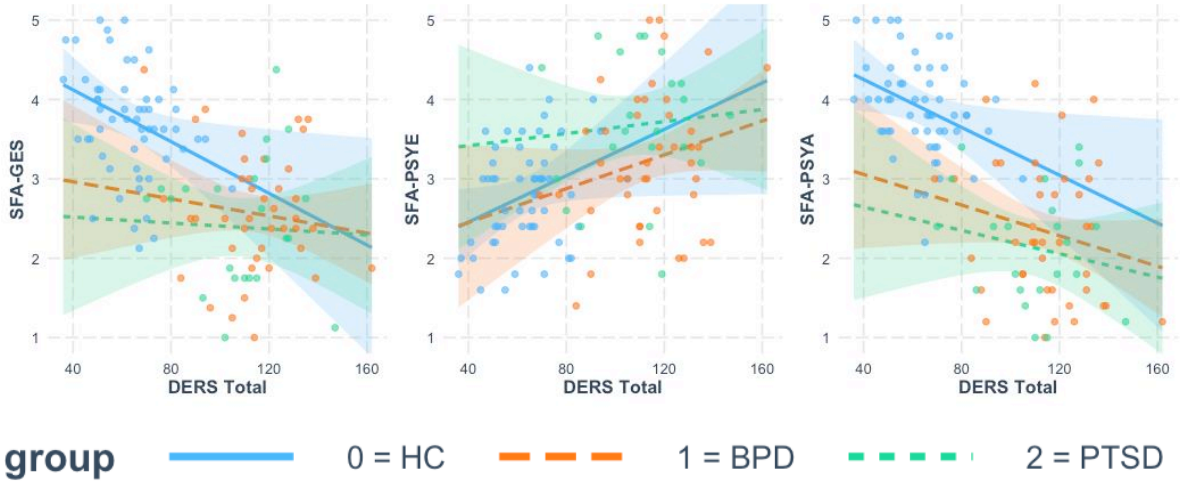
Figure 3

Associations Between Past-Night Sleep, DERS and CTQ

A.



B.



Note. A. Correlations between Sleep Variables, DERS and CTQ. Pairwise N varied from 118 to 126; B. Interaction Plot of the Relationship between Sleep Variables and Emotion Dysregulation by Group (HC, PTSD, BPD). Shaded areas represent 95% CI. SFA GES, restorative value of sleep; PSYA, psychological balance before sleep; PSYE, psychological exhaustion before sleep.

As shown in Table 5 and Figure 3, the multiple regression models revealed that higher emotion dysregulation was associated with a poorer restorative value of sleep (SFA-GES: β=.26, *p*=.048), a lower pre-sleep psychological balance (SFA-PSYA: β=–.30, *p*=.007), and a higher exhaustion before sleep (SFA-PSYE: β=.37, *p*=.016). Relative to HC, both patient groups reported significantly more disturbances in a lower restorative value and lower pre-sleep psychological balance as well as a lower global sleep quality (SFA-SQ) compared to healthy controls. Furthermore, the effect of childhood trauma was not significant for any type of sleep disturbance (all *ps*>.313). Since the interaction of group by emotion dysregulation did not reach statistical significance for any of the types of sleep disturbances, this suggests that the strength of the association between emotion dysregulation and past night's sleep disturbances did not differ between the three groups (all *ps*>.152).

Table 5

Multiple Regression Models Predicting Past-Night Sleep from Emotion Dysregulation, Diagnostic Group, and Childhood Trauma

Model	*B*	*SE*	β [95% CI]	*t*	*p*	*F(df)*	*Adj* R^2
Outcome: SFA-SQ[a], sleep quality						20.4 (4,113)	.40
DERS total	-0.01	0.00	-.21 [-.47, .04]	-1.68	.095		
HC vs. PTSD	-0.88	0.30	-.88 [-1.49, -.28]	-2.90	.005		
HC vs. BPD	-0.72	0.27	-.73 [-1.27, -.18]	-2.65	.009		
CTQ total	-0.00	0.00	-.10 [-.28, .09]	-1.01	.313		
Outcome: SFA-GES[b], restorative value of sleep						18.97 (4,114)	.38
DERS total	-0.01	0.00	-.26 [-.51, -.00]	-1.99	.048		
HC vs. PTSD	-0.97	0.29	-1.03 [-1.65, -0.42]	-3.34	.001		
HC vs. BPD	-0.76	0.26	-0.81 [-1.36, -0.25]	-2.90	.005		
CTQ total	0.00	0.00	.08 [-.11, .27]	0.82	.415		
Outcome: SFA-PSYA[b], pre-sleep psychological balance						39.28 (4, 114)	.56
DERS total	-0.01	0.00	-.30 [-.51, -.08]	-2.76	.007		
HC vs. PTSD	-1.26	0.28	-1.17 [-1.68, -0.65]	-4.51	<.001		
HC vs. BPD	-1.00	0.25	-0.93 [-1.39, -0.47]	-3.99	<.001		
CTQ total	0.00	0.00	.03 [-.13,.19]	0.34	.734		
Outcome: SFA-PSYE[c], pre-sleep psych. exhaustion						7.02 (4,115)	.17
DERS total	0.01	0.00	.37 [.07, .66]	2.46	.016		
HC vs. PTSD	0.42	0.30	.50 [-0.21, 1.21]	1.40	.164		
HC vs. BPD	-0.08	0.27	-.09 [-0.72, 0.54]	-0.29	.775		
CTQ total	-0.00	0.00	-.02 [-.24, .20]	-0.19	.850		
SFA-PSS[d], pre-sleep psychosomatic symptoms						8.69 (4,121)	.20
DERS total	0.00	0.00	.22 [-.07, .50]	1.50	.137		
HC vs. PTSD	0.46	0.17	.92 [.24, 1.59]	2.69	.008		
HC vs. BPD	0.19	0.15	.37 [-.24, .98]	1.21	.228		
CTQ total	-0.00	0.00	-.06 [-.27, .14]	-0.59	.556		

Note. *B*=Unstandardized coefficient, *SE*=standard error, *β*=standardized coefficient; *t*=t-value; *p*=p-value; $F(df_1, df_2)$ = overall model test; Adj. R^2=adjusted proportion of variance explained; bold values represent significant predictors.

[a] *n*=118, [b] *n*=119, [c] *n*=120, [d] *n*=126.

Discussion

Using past-night sleep indices, Study 3 replicated the findings of Studies 1 and 2. Patients with PTSD and BPD reported more sleep disturbances than HC, including lower sleep quality and restorative value, reduced psychological balance and greater exhaustion and psychosomatic symptoms. Unlike Study 2, we found no disorder-specific alterations, suggesting similar levels of acute sleep-related disturbances. Consistent with Study 2, emotion dysregulation was significantly associated with all past-night sleep variables, particularly affect-related components such as restorative value, psychological balance, and pre-sleep exhaustion. These associations did not differ between groups, again supporting a transdiagnostic link. Childhood trauma correlated with sleep disturbances, but lost significance after controlling for emotion dysregulation and group.

General Discussion

This project aimed to investigate the association between emotion dysregulation and sleep disturbances in PTSD, considering childhood trauma and hyperarousal, and to explore whether these relationships are disorder-specific or transdiagnostic. To enhance the generalizability of results, data from three studies were analysed, one naturalistic PTSD sample and two case–control studies of patients with PTSD and BPD and HC. Across studies, individuals with PTSD and BPD showed marked past-month and past-night sleep disturbances, with PTSD additionally characterised by elevated fear of sleep (Study 1,2,3). Emotion dysregulation was consistently related to sleep disturbances beyond hyperarousal in PTSD (Study 1) and beyond childhood trauma and diagnostic group (Studies 2,3). These findings highlight a link between emotion dysregulation and sleep disturbances in PTSD, BPD and HC.

Our findings support extending hyperarousal-centric models of trauma-related sleep problems to include emotion dysregulation as a key mechanism (Fairholme et al., 2013; Levin & Nielsen, 2009; Palmer & Alfano, 2017). This aligns with growing evidence linking emotion

dysregulation and sleep in trauma-related disorders, where direct comparisons between disorders were missing (Grove et al., 2016; Selby, 2013; Short et al., 2017; van Trigt et al., 2025). The regulation-sleep interaction may serve as a fundamental transdiagnostic mechanism across the spectrum of stress and trauma-related disorders, supporting dimensional models of psychopathology (Fairholme et al., 2013; Goldstein & Walker, 2014; Harvey et al., 2011; Palmer & Alfano, 2017).

Notably, emotion dysregulation was most strongly associated with the pre-sleep window, reflected in fear of sleep, lower psychological balance, and greater exhaustion at bedtime. Daytime dysregulation may spill into the evening, driving sleep-disruptive behaviours (e.g., self-harm) and pre-sleep worry or rumination (Palmer & Alfano, 2017; van Trigt et al., 2025). These states may intensify hyperarousal, disrupt sleep onset and continuity and increase nightmare risk (Werner et al., 2021). In turn, disrupted sleep may sustain negative affect, threat sensitivity and impair next-day regulation, reinforcing a vicious cycle (Goldstein & Walker, 2014). The durability of this loop is underscored in our replication across both past-month and past-night assessments (Studies 2,3).

In Studies 2 and 3, in line with our hypotheses, individuals with PTSD and BPD reported markedly worse sleep than HCs at both time scales, which is consistent with prior evidence (Harvey et al., 2003; Werner et al., 2020; Winsper et al., 2017). Yet, disorder-specific patterns also emerged, especially after controlling for emotion dysregulation, as PTSD patients continued to show elevated fear of sleep compared to BPD and HC. Fear of sleep may arise from PTSD-specific mechanisms such as dysfunctional trauma appraisals that heighten perceived threat and vulnerability during sleep. The perceived need to remain hypervigilant and the expectation of nightmares may foster safety behaviours (e.g. sleep avoidance) that worsen sleep (Werner et al., 2021). Fear of sleep has been linked to PTSD severity and

hyperarousal, which is less prominent in BPD (Reffi et al., 2023). Thus, affective evaluations of sleep may be more pronounced in PTSD and call for specific treatment and further research.

In line with previous knowledge and our hypotheses, childhood trauma was positively correlated with sleep disturbances and emotion dysregulation (Karatzoglou et al., 2024; McLaughlin et al., 2020), but its effect on sleep diminished when emotion dysregulation was included, suggesting a potential mediating role. Childhood trauma may operate as a distal risk factor through lasting effects on neurobiological emotion regulation circuits, thereby amplifying vulnerabilities to sleep disturbances (Karatzoglou et al., 2024). However, the cross-sectional design and the lack of prior research limit causal inferences. Longitudinal studies are needed to clarify this interplay. Future studies could explore whether manipulating pre-sleep emotion regulation reduces daytime dysregulation and improves sleep.

Clinical Implications

Our findings suggest that, in PTSD, combining hyperarousal-reducing interventions (e.g., relaxation, prazosin) with emotion-regulation strategies (e.g., mindfulness) during the day and at bedtime may lower fear of sleep and produce broader sleep improvements. In both PTSD and BPD, clinicians should systematically monitor and target sleep and emotional dysregulation. Evidence-based options include combining Cognitive Behavioral Therapy for Insomnia (CBT-I) to regulate pre-sleep affect, Imagery Rescripting & Reprocessing Therapy (IRRT) for nightmares, and sleep-hygiene modules of Dialectical Behavior Therapy (DBT) (Pigeon & Gallegos, 2015; van Trigt et al., 2025). Such integrated interventions may help reduce both disorder-specific symptoms (e.g., fear of sleep in PTSD) and transdiagnostic burdens (general sleep impairment and dysregulation).

Limitations and Future Directions

To the best of our knowledge, this research is the first to compare PTSD and BPD patients across multiple sleep disturbances and time frames and to examine their

transdiagnostic links with emotion dysregulation in naturalistic and transdiagnostic samples. However, several limitations should be acknowledged.

Firstly, all measures relied on self-report, which can diverge from objective sleep assessments and experimental measures of emotion regulation (Slightam et al., 2018). Future studies should adopt multimethod approaches, combining subjective, objective (e.g., polysomnography), and ecological momentary assessment (EMA) data (Short et al., 2017). Secondly, most of the patients were women, medicated, undergoing therapy and had comorbid diagnoses such as depression. As these factors can influence sleep and emotion dysregulation, they may have confounded results (Ehring et al., 2008). Future studies should control for them while examining disorder-specific contributors such as threat sensitivity in PTSD (van Trigt et al., 2025). Lastly, reliance on total scores may have obscured domain-specific associations. Future research should examine whether specific sleep disturbances (e.g., efficiency) relate differentially to facets of dysregulation (e.g., impulse control) or trauma types (e.g., sexual abuse). Although we focused mainly on dysregulation's influence on sleep, its bidirectionality is well established (Palmer & Alfano, 2017). Longitudinal and experimental designs are needed to examine bidirectional pathways and clarify causal mechanisms.

Conclusion

This research offers compelling evidence that emotion dysregulation is linked to sleep disturbances beyond hyperarousal and childhood trauma in PTSD. This link generalized to individuals with BPD and HC and did not show disorder-specific differences in strength. Both clinical groups showed pronounced sleep disturbances, with PTSD additionally marked by elevated fear of sleep. Childhood trauma correlated with sleep, but did not exert a direct effect once emotion dysregulation and group were considered. These findings emphasize the need for routine sleep assessments in trauma-focused care and support, integrating transdiagnostic emotion regulation modules with targeted sleep-focused interventions in PTSD and BPD.

Future longitudinal, multimethod designs, including EMA and objective sleep measures, are needed to elucidate causal dynamics linking sleep, dysregulation and early trauma.

Acknowledgments: This work was developed as part of the author's PhD dissertation at Ludwig-Maximilians-University Munich, Germany.

Declaration of Interest: We have no conflict of interest to disclose.

References

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T.,…Zule, W. (2003). Development and validation of a brief screening version of the childhood trauma questionnaire. *Child Abuse & Neglect*, *27*(2), 169-190. https://doi.org/10.1016/s0145-2134(02)00541-0

Böckermann, M., Gieselmann, A., & Pietrowsky, R. (2014). What does nightmare distress mean? Factorial structure and psychometric properties of the Nightmare Distress Questionnaire (NDQ). *Dreaming*, *24*(4), 279-289. https://doi.org/10.1037/a0037749

Buysse, D. J., Reynolds 3rd, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*, *28*(2), 193-213. https://doi.org/10.1016/0165-1781(89)90047-4

Drexl, K., Kunze, A. E., & Werner, G. G. (2019). The German version of the Fear of Sleep Inventory - Short Form: A psychometric study. *European Journal of Trauma & Dissociation*, *3*(4), 221-228. https://doi.org/10.1016/j.ejtd.2019.05.004

Ehring, T., Fischer, S., Schnülle, J., Bösterling, A., & Tuschen-Caffier, B. (2008). Characteristics of emotion regulation in recovered depressed versus never depressed individuals. *Personality and Individual Differences*, *44*(7), 1574-1584. https://doi.org//10.1016/j.paid.2008.01.013

Fairholme, C. P., Nosen, E. L., Nillni, Y. I., Schumacher, J. A., Tull, M. T., & Coffey, S. F. (2013). Sleep disturbance and emotion dysregulation as transdiagnostic processes in a comorbid sample. *Behaviour Research and Therapy*, *51*(9), 540-546. https://doi.org/10.1016/j.brat.2013.05.014

First, M. B., Williams, J. B. W., Karg, R. S., & Spitzer, R. L. (2016). Structured Clinical Interview for DSM-5 Disorders, Clinician Version (SCID-5-CV). *American Psychiatric Association*.

Germain, A., McKeon, A. B., & Campbell, R. L. (2017). Sleep in PTSD: Conceptual model and novel directions in brain-based research and interventions. *Curr Opin Psychol*, *14*, 84-89. https://doi.org/10.1016/j.copsyc.2016.12.004

Goldstein, A. N., & Walker, M. P. (2014). The role of sleep in emotional brain function. *Annual Review of Clinical Psychology*, *10*, 679-708. https://doi.org/10.1146/annurev-clinpsy-032813-153716

Görtelmeyer, R. (2011). SF-A/R und SF-B/R: Schlaffragebogen A und B - Revidierte Fassung. . *Hogrefe, Göttingen*.

Gratz, K. L., & Roemer, L. (2004). Multidimensional Assessment of Emotion Regulation and Dysregulation: Development, Factor Structure, and Initial Validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment*, *26*(1), 41-54. https://doi.org/10.1023/B:JOBA.0000007455.08539.94

Grove, J. L., Smith, T. W., Crowell, S. E., & Ellis, J. H. (2016). Preliminary Evidence for Emotion Dysregulation as a Mechanism Underlying Poor Sleep Quality in Borderline Personality Disorder. *Journal of Personality Disorders*, *31*(4), 542-552. https://doi.org/10.1521/pedi_2016_30_261

Harvey, A. G., Jones, C., & Schmidt, D. A. (2003). Sleep and posttraumatic stress disorder: a review. *Clinical Psychology Review*, *23*(3), 377-407. https://doi.org/10.1016/s0272-7358(03)00032-1

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. *Clin Psychol Rev*, *31*(2), 225-235. https://doi.org/10.1016/j.cpr.2010.04.003

Karatzoglou, V., Carollo, A., Karagiannopoulou, E., Esposito, G., Séaghdha, X. T. Ó., & Dimitriou, D. (2024). A scientometric review of the association between childhood

trauma and sleep. *Acta Psychologica*, *250*, 104488. https://doi.org/10.1016/j.actpsy.2024.104488

Levin, R., & Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation: A review and new neurocognitive model of dreaming. *Current Directions in Psychological Science*, *18*(2), 84-88. https://doi.org/10.1111/j.1467-8721.2009.01614.x

Loranger, A. W., Janca, A., & Sartorius, N. (1997). *Assessment and diagnosis of personality disorders: The ICD-10 international personality disorder examination (IPDE)*. Cambridge University Press.

McLaughlin, K. A., Colich, N. L., Rodman, A. M., & Weissman, D. G. (2020). Mechanisms linking childhood trauma exposure and psychopathology: A transdiagnostic model of risk and resilience. *BMC Medicine*, *18*(1), 96. https://doi.org/10.1186/s12916-020-01561-6

Palagini, L., Moretto, U., Dell'Osso, L., & Carney, C. (2017). Sleep-related cognitive processes, arousal, and emotion dysregulation in insomnia disorder: the role of insomnia-specific rumination. *Sleep Medicine*, *30*, 97-104. https://doi.org/10.1016/j.sleep.2016.11.004

Palmer, C. A., & Alfano, C. A. (2017). Sleep and emotion regulation: An organizing, integrative review. *Sleep Med Rev*, *31*, 6-16. https://doi.org/10.1016/j.smrv.2015.12.006

Pigeon, W. R., & Gallegos, A. M. (2015). Posttraumatic Stress Disorder and Sleep. *Sleep Medicine Clinics*, *10*(1), 41-48. https://doi.org/10.1016/j.jsmc.2014.11.010

Reffi, A. N., Kalmbach, D. A., Cheng, P., Tappenden, P., Valentine, J., Drake, C. L.,…Lilly, M. M. (2023). Fear of sleep in first responders: associations with trauma types, psychopathology, and sleep disturbances. *SLEEP Advances*, *4*(1), zpad053. https://doi.org/10.1093/sleepadvances/zpad053

Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its

evidence. *Sleep Medicine Reviews*, *14*(1), 19-31. https://doi.org/10.1016/j.smrv.2009.04.002

Selby, E. A. (2013). Chronic sleep disturbances and borderline personality disorder symptoms. *Journal of Consulting and Clinical Psychology*, *81*(5), 941-947. https://doi.org/10.1037/a0033201

Semsar, N., Mousavi, Z., Tran, M.-L. M., & Kuhlman, K. R. (2021). Dysregulated arousal as a pathway linking childhood neglect and clinical sleep disturbances in adulthood. *Child Abuse & Neglect*, *122*, 105306. https://doi.org/10.1016/j.chiabu.2021.105306

Short, N. A., Allan, N. P., & Schmidt, N. B. (2017). Sleep disturbance as a predictor of affective functioning and symptom severity among individuals with PTSD: An ecological momentary assessment study. *Behaviour Research and Therapy*, *97*, 146-153. https://doi.org/10.1016/j.brat.2017.07.014

Slightam, C., Petrowski, K., Jamison, A. L., Keller, M., Bertram, F., Kim, S., & Roth, W. T. (2018). Assessing sleep quality using self-report and actigraphy in PTSD. *Journal of Sleep Research*, *27*(3), e12632-e12632. https://doi.org/10.1111/jsr.12632

van Trigt, S., van der Zweerde, T., van Someren, E. J. W., van Straten, A., & van Marle, H. J. F. (2025). A theoretical perspective on the role of sleep in borderline personality disorder: From causative factor to treatment target. *Sleep Medicine Reviews*, *81*, 102089. https://doi.org/10.1016/j.smrv.2025.102089

Wang, W., & Wang, X. (2025). Sleep disturbance among Chinese survivors of childhood sexual abuse: associations with perceived discrimination and rumination [Original Research]. *Frontiers in Psychiatry*, *Volume 16 - 2025*. https://doi.org/10.3389/fpsyt.2025.1431839

Weathers, F. W., Bovin, M. J., Lee, D. J., Sloan, D. M., Schnurr, P. P., Kaloupek, D. G.,…Marx, B. P. (2018). The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5):

Development and initial psychometric evaluation in military veterans. *Psychol Assess*, *30*(3), 383-395. https://doi.org/10.1037/pas0000486

Weathers, F. W., Litz, B. T., Keane, T. M., Palmieri, P. A., Marx, B. P., & Schnurr, P. P. (2013). The PTSD Checklist for DSM-5 (PCL-5). *National Center for PTSD*.

Werner, G. G., Danböck, S. K., Metodiev, S., & Kunze, A. E. (2020). Pre-Sleep Arousal and Fear of Sleep in Trauma-Related Sleep Disturbances: A Cluster-Analytic Approach. *Clinical Psychology in Europe*, *2*(2), 1-20. https://doi.org/10.32872/cpe.v2i2.2699

Werner, G. G., Riemann, D., & Ehring, T. (2021). Fear of sleep and trauma-induced insomnia: A review and conceptual model. *Sleep Medicine Reviews*, *55*, 101383. https://doi.org/10.1016/j.smrv.2020.101383

Winsper, C., Tang, N. K., Marwaha, S., Lereya, S. T., Gibbs, M., Thompson, A., & Singh, S. P. (2017). The sleep phenotype of Borderline Personality Disorder: A systematic review and meta-analysis. *Neurosci Biobehav Rev*, *73*, 48-67. https://doi.org/10.1016/j.neubiorev.2016.12.008

Zhang, Y., Ren, R., Sanford, L. D., Yang, L., Zhou, J., Zhang, J.,…Tang, X. (2019). Sleep in posttraumatic stress disorder: A systematic review and meta-analysis of polysomnographic findings. *Sleep Medicine Reviews*, *48*, 101210. https://doi.org/10.1016/j.smrv.2019.08.004

Zhou, A., McDaniel, M., Hong, X., Mattin, M., Wang, X., & Shih, C. H. (2023). Emotion dysregulation mediates the association between acute sleep disturbance and later posttraumatic stress symptoms in trauma exposed adults. *Eur J Psychotraumatol*, *14*(2), 2202056. https://doi.org/10.1080/20008066.2023.2202056

3 General Discussion

The development of psychopathology is thought to be influenced by complex biopsychosocial processes that cut across diagnostic categories. As a result, classification systems are increasingly adopting dimensional, transdiagnostic frameworks based on shared risk factors and underlying mechanisms of disorders (Dalgleish et al., 2020; Harvey et al., 2004). Traumatic childhood experiences (TCE) might operate as a *distal risk factor* for mental disorders such as PTSD and BPD which share a high TCE prevalence. TCE might contribute to both shared symptoms and disorder-specific patterns of PTSD and BPD by influencing more *proximal* mechanisms (Nolen-Hoeksema & Watkins, 2011). Therefore, exploring the role of TCE in mental disorders, such as PTSD and BPD, as well as possible biopsychological mechanisms is urgently needed in order to improve prevention and intervention across diagnostic boundaries. In this thesis, the link between TCE and three possible mechanisms, namely, *social functioning*, *emotion processing*, and *sleep*, were investigated in individuals with PTSD, BPD as well as healthy controls in three complementary studies.

Study 1 was a systematic literature review exploring the transdiagnostic relationship between TCE and *social functioning*, including the mechanisms underlying this link, in adults with and without psychopathology. Study 2 investigated the association between TCE and *emotional processing* (reactivity and regulation) in individuals with PTSD, BPD and healthy controls. Study 3 examined the link between TCE and *sleep disturbances* (sleep quality, fear of sleep, nightmares) in individuals with PTSD, BPD and healthy controls. The role of emotion dysregulation within the association between TCE and sleep was further investigated.

In the following sections, the findings of the three studies will be summarised in light of the overarching aim of this thesis and systematically integrated into previous research. Moreover, the strengths and limitations of this thesis, as well as future directions and clinical implications, will be discussed.

3.1 Summary of Findings

Study 1 revealed that TCE were associated with various social impairments in adults, in line with previous systematic reviews (Carr et al., 2020; Haslam & Taylor, 2022). Across seven domains, the most robust links emerged with intimate partner violence (IPV), aggression, early maladaptive schemas, attachment insecurity, and reduced social connectedness. Evidence for domains such as sexuality, intimate partner relationships, and social-cognitive processes was comparatively inconsistent which highlights the need for further research. The strength of associations varied by TCE type, as physical abuse was strongly correlated with aggression, emotional abuse with attachment insecurity and early maladaptive schemas, and sexual abuse with sexual health outcomes. The most prominent mechanisms linking TCE to social dysfunction were psychopathology, insecure attachment/ maladaptive schemas and *emotion regulation*. Specifically, broad difficulties in emotion recognition, integration, and regulation, heightened fear of emotions, and maladaptive strategies (e.g., anger rumination, alcohol use) were associated with reduced social functioning (e.g. lower availability of social support, increased aggression, and revictimization due to intimate partner violence). Consistent with models highlighting a coupling between social and emotional processing following TCE, these patterns motivated the need for direct testing of emotion processing as a further mechanism in Study 2 (Gross & John, 2003; Kim & Cicchetti, 2010; McNeil & Rehman, 2024).

Study 2 demonstrated that TCE were correlated with an emotional hypo-reactivity to positive and neutral stimuli, consistent with non-clinical research (Clarke et al., 2024; Sill et al., 2020; Wu et al., 2023). Crucially, this association disappeared once group differences (PTSD, BPD, HC) and medication use were controlled for. This suggests that TCE may primarily increase the risk for mental health disorders like PTSD and BPD, with more immediate factors explaining the observed hypo-reactivity. Another important finding of Study 2 was that the correlation between TCE and self-reported *emotion dysregulation* remained

significant even after controlling for group and medication use. Importantly, TCE did not correlate with neurophysiological variables of emotion processing or instructed emotion regulation (i.e., cognitive reappraisal) in the laboratory. In this sense, TCE appear to confer a more widespread, trait-level vulnerability for global dysregulation that manifests in self-reports but might not strongly shape immediate, state-level reactivity to experimental cues or the success of an instructed strategy (Daros et al., 2013; Flechsenhar et al., 2024; Messman-Moore & Bhuptani, 2017). The results uncovered both shared and disorder-specific patterns as women with PTSD displayed heightened emotional hypo-reactivity, while women with BPD showed elevated dysregulation and impulsivity. Models positing bidirectional links between emotion processing and sleep disturbances following trauma, motivated the examination of sleep as an additional mechanism in Study 3.

Study 3 established that TCE were significantly associated with past-month and past-night sleep disturbances, including poor sleep quality, nightmare distress, fear of sleep, reduced restorative sleep and pre-sleep psychological balance and increased exhaustion, and psychosomatic symptoms at bedtime. This association was consistent across three complementary studies: a naturalistic sample of treatment-seeking PTSD patients and two case-control samples involving individuals with PTSD, BPD, and healthy controls. This finding is in line with previous longitudinal studies stressing the long-term effects of TCE on adult sleep disturbances (Chapman et al., 2013; Chapman et al., 2011; Karatzoglou et al., 2024; Lind et al., 2016; Sullivan et al., 2019). However, the association between TCE and sleep became insignificant once emotion dysregulation was taken into account, implying a potential indirect pathway. This finding might indicate that TCE act as a distal risk factor for sleep disturbances through more proximal pathways of emotion dysregulation. Emotion dysregulation was consistently linked to poorer past-month sleep quality, (nightmare distress), fear of sleep and past-night disturbances above and beyond hyperarousal, group and TCE. Individuals with

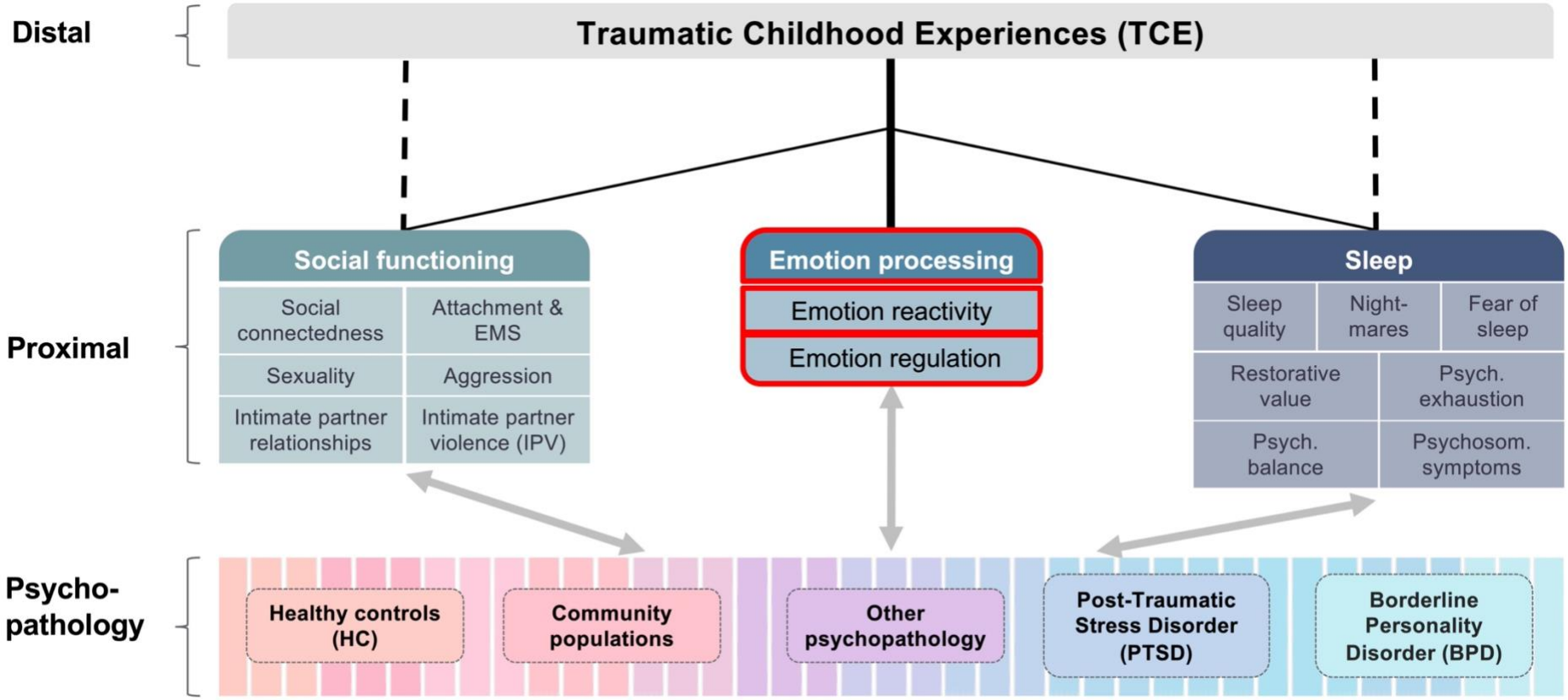
PTSD and BPD showed considerable overlap in sleep disturbances. However, fear of sleep remained markedly elevated in PTSD, while emotion dysregulation largely accounted for sleep issues in BPD.

3.2 Synthesis of Findings

Across this thesis, TCE emerged as a broad vulnerability factor, associated with disturbances in social functioning (Study 1), emotional processing (Study 2), and sleep (Study 3). Across all three studies, *emotion dysregulation* consistently emerged as a central mechanism linking TCE to impaired social functioning and sleep disturbances. It might serve as a transdiagnostic vulnerability and a key factor explaining overlapping symptoms across disorders. Therefore, this discussion will put a specific focus on emotion dysregulation as a mechanism. Figure 2 illustrates an integrative model, synthesising the key findings of this thesis as well as pathways that require more research. While the individual publications of the three studies discussed the results within their specific research areas, the following section will synthesize findings across studies and outline directions for future research.

Figure 2

Integrative Transdiagnostic Framework of Key Findings and Pathways for Future Research



Note. Dashed arrows indicate a possible indirect effect of TCE through emotion processing. Grey arrows represent a possible bidirectional interaction between mechanisms and psychopathology that requires further research.

3.2.1 The Role of Psychopathology

As indicated by Figure 2, all three studies raised the question of what role psychopathology plays in the complex interplay between TCE and mechanisms. In Study 1, psychopathology (e.g. PTSD and BPD features) was a mediator of the link between TCE and social functioning. In Studies 2 and 3, the associations between TCE and emotional hypo-reactivity, as well as between TCE and sleep disturbances, were substantially reduced once the presence of psychopathology (PTSD vs. HC and BPD vs. HC) was accounted for. This raises the question whether alterations in social functioning, emotion processing, and sleep after TCE (a) precede the onset of psychopathology, (b) emerge as a consequence of psychopathology or (c) become part of the disorder (e.g. nightmares in PTSD).

Longitudinal research suggests that all three mechanisms predict the onset and severity of trauma-related psychopathology following TCE (Gehrman et al., 2013; Liu et al., 2023; McLaughlin et al., 2020; Ozer et al., 2003; Pfaltz et al., 2022; Spoormaker & Montgomery, 2008; Weissman et al., 2019). Moreover, these features often persist and impact functional impairment after remission from trauma-related disorders, indicating their independent roles as lasting and self-sustaining processes (Boyd et al., 2020; Hjelseng et al., 2022; Soloff, 2019; Zayfert & DeViva, 2004). However, trauma-related psychopathology and mechanisms might also form vicious cycles after TCE through their complex bidirectional relationships (Liu et al., 2022; Lord et al., 2020). For example, in PTSD, trauma-related avoidance of emotions and people may lead to social estrangement and withdrawal, which diminishes social support and exacerbates PTSD symptoms (Calhoun et al., 2022; Johansen et al., 2022). Moreover, hyperarousal and emotion dysregulation may contribute to insomnia, nightmares, and fear of sleep, which can impair next-day regulation capacities and amplify re-experiencing (Werner et al., 2021). In BPD, heightened affective instability and reactivity (e.g. anger outbursts) might amplify volatile relationship dynamics (e.g. idealisation/ devaluation, fear of abandonment)

and recurrent conflict (Schulze et al., 2022). Moreover, the maladaptive regulation (e.g. self-harm, rumination) of inter- and intrapersonal distress might undermine sleep, which can increase next-day reactivity (van Trigt et al., 2025).

The scarcity of longitudinal research limits the ability to determine causal directionality. Similarly, the lack of case-control studies impedes understanding of whether the links between TCE and mechanisms vary between adults with and without psychopathology. This highlights the need for further research using longitudinal and real-life assessments to understand the complex temporal interplay among TCE, mechanisms and psychopathology (Glaser et al., 2006; Hein & Monk, 2017; McLaughlin et al., 2015). Moreover, as mental health processes unfold along a continuous spectrum and cannot be categorized into "healthy or pathological", both dimensional and categorical perspectives are needed, as discussed in the next section.

3.2.2 Dimensional versus Categorical Perspectives

This thesis aimed to develop an integrative account of categorical and dimensional perspectives to inform comprehensive models of trauma-related psychopathology (Dalgleish et al., 2020; Hofmann & Hayes, 2019; Nolen-Hoeksema & Watkins, 2011). The thesis explored (1) TCE as a *distal risk factor* that cuts across diagnostic boundaries and (2) *proximal mechanisms* that may explain overlapping and distinct illness trajectories.

From a dimensional perspective, TCE were linked with mechanisms across diverse populations such as community, forensic, and psychiatric (e.g., depression, schizophrenia, etc.) (Study 1) as well as individuals with PTSD, BPD and healthy controls (Studies 2 and 3). Moreover, individuals with PTSD and BPD showed overlapping alterations on all three mechanisms after TCE. In *Study 1*, both PTSD and BPD features mediated the association between TCE and social dysfunction, particularly violence perpetration (Liu et al., 2021; Liu

et al., 2012). Moreover, in *Study 2*, individuals with PTSD and BPD shared a hypo-reactivity to neutral stimuli, self-reported emotion dysregulation and an effective implementation of an emotion regulation strategy (i.e., cognitive reappraisal). Lastly, in *Study 3*, individuals with PTSD and BPD overlapped on heightened sleep disturbances on all sleep variables in comparison to healthy controls. Moreover, the strength of the link between emotion regulation and sleep did not differ across PTSD, BPD, and HC. Overall, these results align with the emerging shift to conceptualize psychopathology as a dimensional continuum rather than as a set of discrete disorders (Dalgleish et al., 2020; McLaughlin et al., 2020).

From a categorical perspective, distinct patterns appeared in PTSD and BPD, which indicate that shared TCE-related vulnerabilities can crystallize into disorder-specific profiles (Nolen-Hoeksema & Watkins, 2011). Table 2 provides an integrative summary of overlapping and disorder-specific patterns found across the three domains. In the following, these disorder-specific findings are integrated into established theoretical models of PTSD and BPD.

Table 2

Disorder-Specific and Overlapping Alterations in TCE, Social Functioning, Emotion Processing and Sleep Disturbances in PTSD and BPD

	PTSD-specific	Overlap	BPD-specific
Traumatic childhood experiences (TCE)	• Physical abuse (PTSD > BPD)	• Overall TCE severity • Emotional and sexual abuse • Emotional and physical neglect	
Social functioning		• Physical violence, aggression & hostility • Intrafamilial physical violence perpetration	
Emotion processing	• Decreased reactivity (P3 amplitudes) to neutral pictures (PTSD < BPD) and negative pictures (PTSD < HC)	• Deficits in self-reported emotional acceptance, awareness, clarity, ER strategies, goal-directed behaviour • Decreased valence ratings to positive and neutral pictures • Decreased P3 amplitudes to neutral pictures (PTSD < BPD < HC) • No deficits in instructed cognitive reappraisal (valence ratings and ERP amplitudes)	• Global emotion dysregulation (BPD > PTSD) • Impulse control difficulties (BPD > PTSD)
Sleep	• Increased Fear of sleep (PTSD > BPD)	• Decreased sleep quality • Decreased restorative value of sleep • Decreased psychological balance before sleep • Increased nightmare distress • Increased psychological exhaustion before sleep • Increased psychosomatic symptoms at sleep onset	• Disturbances (quality, fear of sleep) largely explained by emotion dysregulation

Note. Overlap denotes findings where PTSD and BPD both differed from healthy controls (HC) but not from one another.

The Cognitive model of PTSD: Disorder-Specific Alterations Following TCE

Ehlers and Clark (2000) cognitive model of PTSD proposes that PTSD arises when individuals process trauma in a way that creates a sense of ongoing threat. This sense of threat is maintained by (a) dysfunctional appraisals of TCE and its consequences (e.g., "*I am permanently damaged*") and (b) disturbances in autobiographical memory. Moreover, (c) the use of maladaptive coping strategies such as avoidance, rumination or safety-seeking behaviors prevents the disconfirmation of negative beliefs.

In line with component (a), Study 1 identified early maladaptive schemas as key mediators between TCE and violence/aggression. These findings indicate that trauma-related interpretations and internalized narratives can profoundly shape social functioning (Aafjes-van Doorn et al., 2020) (Lobbestael & McNally, 2016). Self- and other-directed negative beliefs (e.g. "*I am unable to feel close to anyone*", "*no one can be trusted*") may foster perceptions of others as untrustworthy, rejecting, or hostile, and social contexts as unsafe, prompting withdrawal or hypervigilance (Fertuck et al., 2013; Hébert et al., 2021; Martinez et al., 2025). Consistent with this, Study 1 linked TCE to heightened hostility and aggression in PTSD, with PTSD symptoms mediating associations between TCE and intimate partner violence, reduced relationship satisfaction and parental competence, and elevated child abuse potential.

In Study 2, individuals with PTSD exhibited greater emotional hypo-reactivity (P3 amplitude), especially to trauma-unrelated neutral and (negative) stimuli, than individuals with BPD and HC. This emotional numbing may reflect the avoidance processes outlined in component (c). It has been proposed that in individuals with TCE, the perception of threat might be heightened, while the processing of stimuli that are unrelated to the trauma is suppressed as a protective mechanism (Adenauer et al., 2010; Hein & Monk, 2017; McLaughlin et al., 2015; Zukerman et al., 2023).

Lastly, individuals with PTSD expressed a higher fear of sleep than individuals with BPD in Study 3. Fear of sleep is thought to develop due to (a) negative beliefs about the loss of safety and control during sleep (e.g., "*It is dangerous to fall asleep*") (Werner et al., 2021). Consequently, sleep can become associated with feelings of vulnerability and with trauma reminders (nightmares) after TCE. This might be especially relevant for survivors of childhood sexual abuse, where assaults often occurred at bedtime (Steine et al., 2019). The consequential fear of sleep can lead to (c) maladaptive regulation strategies such as avoidance of sleep, rumination on threat and safety behaviours (e.g., sleeping with lights on) that further disturb sleep (Palmer & Alfano, 2017; van Trigt et al., 2025; Wang & Wang, 2025). A factor that was not investigated in this thesis but that requires more attention is the impact of (b) disturbances in autobiographical memory on sleep disturbances and nightmares which might be more specific to PTSD (Pace-Schott et al., 2015).

The Biosocial Model of BPD: Disorder-Specific Alterations Following TCE

According to the *biosocial model* of Linehan (1993) and its extensions (Crowell et al., 2009), BPD arises from the interaction between genetic vulnerabilities (e.g., emotion sensitivity and impulsivity) and environmental factors such as emotional invalidation. In such environments, a child's emotional expressions are dismissed, punished, or ignored, preventing the acquisition of effective regulation strategies. Early attachment disruptions due to TCE, in which a child alternates between seeking closeness (approach) and withdrawing for self-protection (avoidance) can further produce disorganized attachment patterns (Holmes, 2004). Over time, the interplay of these factors fosters enduring difficulties in *emotion regulation* and *interpersonal functioning*, which constitute the core features of BPD (Carpenter & Trull, 2013).

In line with the biosocial model, Study 1 identified insecure attachment as a key mediator between TCE and social dysfunction, particularly in relationship quality and

satisfaction. Insecure attachment manifests as anxious attachment, characterised by a fear of abandonment and excessive efforts to maintain closeness, or avoidant attachment, marked by emotional distancing and discomfort with intimacy (Bowlby, 1982). Consistent with this, individuals with BPD showed higher levels of insecure attachment and greater relationship difficulties, including lower satisfaction compared to healthy controls. Moreover, in individuals with BPD, TCE were associated with heightened rejection sensitivity and loneliness, as well as reduced perspective-taking and empathic concern, reflecting the interpersonal consequences of TCE proposed by Linehan's model.

Extending these patterns, Study 2 found that individuals with BPD exhibited greater overall emotion dysregulation and more pronounced impulsivity in response to negative emotions compared to individuals with PTSD. This is consistent with the model's assumption of heightened emotional sensitivity coupled with limited regulatory capacities (Linehan, 1993). Similarly, Study 3 demonstrated that disturbances in sleep quality and fear of sleep among individuals with BPD were largely accounted for by emotion dysregulation. This again stresses emotion dysregulation at the core of the disorder and indicates disorder-specific elevations on top of the TCE-related vulnerability shared with PTSD (Links et al., 1999).

Taken together, the findings for PTSD and BPD indicate both shared vulnerabilities after TCE and disorder-specific alterations. PTSD and BPD might diverge in their predominant etiological mechanisms after TCE, with PTSD being dominated by cognitive trauma appraisals and avoidant coping mechanisms and BPD by the dominance of affective and interpersonal mechanisms.

3.2.3 The Role of Complex PTSD and Personality Functioning

Complex Post-Traumatic Stress Disorder (cPTSD)

The diagnostic construct of complex post-traumatic stress disorder (cPTSD) might provide a useful framework for understanding these shared and distinct pathways following TCE identified in this thesis. Introduced in the ICD-11, cPTSD is thought to arise predominantly from prolonged or repeated interpersonal traumas such as emotional, physical or sexual childhood abuse (World Health Organization, 2019). Moreover, the diagnostic criteria of cPTSD emphasize that the consequences of TCE extend beyond the core PTSD symptoms to chronic "disturbances in self-organization (DSO)", namely: (1) difficulties in affect regulation, (2) negative self-concept and (3) interpersonal impairment. This conceptualization closely mirrors the findings of this thesis that TCE are linked to emotion dysregulation and social dysfunction. Therefore, the new diagnosis cPTSD might provide a conceptual bridge for exploring underlying mechanisms after TCE.

As outlined in the previous section, next to strong overlaps between PTSD and BPD (dimensional perspective), distinct profiles emerged consistent with existing disorder-specific models of Ehlers and Clark (2000) and Linehan (1993) (categorical perspective). In PTSD, emotional numbing, avoidance (e.g. hypo-reactivity) and threat-related appraisals (e.g. fear of sleep) might have played a larger role, while in BPD, attachment-related distress and higher emotion dysregulation (e.g. impulsivity) appeared as more central processes. cPTSD might integrate elements of both profiles and may capture the enduring disruptions in self-organization observed in individuals exposed to severe, prolonged TCE.

Empirical findings support this position. Research indicates that more chronic, repeated interpersonal trauma such as childhood abuse is more predictive of cPTSD and BPD, while single-event trauma (e.g. accident) is more associated with PTSD (Cloitre et al., 2013; Cloitre et al., 2018; Hyland et al., 2019). PTSD and cPTSD can be differentiated most sharply by

sexual trauma histories based on conditional prevalences (PTSD: 20.0% kidnapping; cPTSD: 7.4% childhood sexual abuse), with childhood sexual abuse also reported more frequently in cPTSD than in BPD (Cloitre et al., 2014; Maercker et al., 2018). This pattern aligns with the findings of this thesis that more severe TCE is linked to more severe disturbances in social functioning and emotion regulation. Moreover, latent class and network analyses have identified four separable trauma-related profiles, suggesting partial but not complete overlap: (a) low endorsement of all symptoms; (b) PTSD class with low levels of cPTSD and BPD symptoms; (c) cPTSD class marked by high PTSD symptoms and low BPD features; and (d) BPD class characterized primarily by BPD symptoms (Cloitre et al., 2013; Cloitre et al., 2014; Frost et al., 2020; Knefel et al., 2016).

Within these profiles, phenomenological differences appear in how common symptoms manifest when comparing cPTSD, BPD and PTSD (Cloitre et al., 2014; Frost et al., 2020). For example, individuals with BPD show more (1) emotional hyperreactivity, impulsiveness, self-harming and suicidal behaviour, angry outbursts that spiral into loss of control, (2) a fluctuating and unstable sense of self and (3) frantic efforts to avoid abandonment and unstable and intense relationships (Cloitre et al., 2014; Hyland et al., 2019; Powers et al., 2022). In comparison, individuals with cPTSD presents a more (1) persistent pattern of emotional numbing, dissociative symptoms and avoidance of internal and external trauma reminders (2) a persistent negative self-concept (feelings of shame, guilt or self-loathing) and (3) social withdrawal, driven by challenges in maintaining relationships, feelings of detachment or estrangement (Bohus & Vonderlin, 2024; Cloitre et al., 2014; Hyland et al., 2019; McBride et al., 2025; McCann et al., 2023; Rüsch et al., 2007; Rüsch et al., 2011). Lastly, PTSD might be most strongly linked with classic PTSD symptoms such as re-experiencing, avoidance, alterations in cognitions and mood and hyperarousal , including sleep disturbances and nightmares (Frost et al., 2020; Hyland et al., 2019; Powers et al., 2022; Redican et al., 2021).

These differences parallel the pattern observed in this thesis, as individuals with BPD patients reported stronger emotional dysregulation while individuals with PTSD showed stronger avoidance and numbing (Cloitre et al., 2014; Hyland et al., 2019; Powers et al., 2022). Therefore, PTSD and BPD may vary on a spectrum from hyper- to hypo-reactivity to emotions, as well as from under- to over-regulation of emotions as described in cPTSD (Ford & Courtois, 2021; Río-Casanova et al., 2016). This aligns with neurobiological studies identifying two PTSD profiles after severe trauma: a reexperiencing/hyperarousal subtype with under-regulation (e.g., anger, self-destruction; reduced PFC inhibition of limbic regions) and a dissociative subtype with over-regulation (e.g., avoidance, numbing; heightened PFC inhibition) (Lanius et al., 2010; Litz et al., 2002; Steuwe et al., 2012). Moreover, the strongest bridge between BPD and cPTSD is thought to be through overlap on affective dysregulation, namely alternations between emotional hyperreactivity and numbness (Frost et al., 2020; Owczarek et al., 2023; Powers et al., 2022).

Taken together, these findings emphasize that cPTSD represents a diagnostic refinement that describes the enduring effects of chronic trauma, such as TCE, on interpersonal and emotional dysregulation. However, even though the diagnosis of cPTSD is already used in clinical practice, research on a disorder-specific, theory-driven model that can be directly translated into interventions is still lacking. Along with the findings of this thesis, this highlights the need to investigate trauma-related psychopathology through a hybrid approach, combining categorical distinctions (to delineate disorder-specific features) with dimensional mechanisms (to identify shared processes such as emotion dysregulation and social dysfunction). Future research should therefore directly compare PTSD, BPD, and cPTSD on the effects of different trauma types on various proximal mechanisms using longitudinal and multimodal designs. Alterations of these mechanisms should be compared across multiple trauma-related conditions with and without comorbidity. This dual approach can help to

identify disorder-specific features for targeted interventions and shared mechanisms after TCE, suited for transdiagnostic treatments (Hofmann & Hayes, 2019).

Levels of Personality Functioning

Based on this thesis, social functioning and particularly emotion dysregulation may be a central transdiagnostic factor linking TCE to various other mechanisms (e.g., sleep). Moreover, it might explain overlapping and disorder-specific alterations after TCE found across PTSD, BPD and cPTSD. However, another mechanism also merits closer attention. TCE may also shape more fundamental aspects of *personality functioning* that integrate emotional and interpersonal domains (Back et al., 2021; Back et al., 2020; Lock et al., 2025). In this context, levels of personality functioning, as defined in the Alternative Model for Personality Disorders (AMPD), may offer an additional dimensional framework for understanding the link between TCE and trauma-related psychopathology.

The AMPD was introduced in Section III of the DSM-5 and conceptually adopted in the ICD-11 and defines personality pathology using a hybrid model of dimensional and categorical assessments (American Psychiatric Association, 2013). Personality functioning is rated along dimensional levels of impairments in self-functioning (i.e., identity, self-direction) and interpersonal functioning (i.e., empathy, intimacy) as well as categorical pathological traits (e.g., antagonism) (Bender et al., 2011). These AMPD dimensions inherently overlap with the DSO criteria of complex PTSD and the mechanisms examined in this thesis: interpersonal impairments align with social dysfunction, while self-functioning deficits connect to emotion dysregulation and alterations in self-concept.

Given this inherent overlap, personality functioning may represent a higher-order integrative mechanism through which TCE exert long-term effects on trauma-related psychopathology. Personality functioning has been shown to mediate the link between TCE and transdiagnostic psychopathology, such as depression and anxiety (d'Huart et al., 2022;

Freier et al., 2022; Kerber et al., 2023; Krakau et al., 2021). Moreover, a study by Kampling et al. (2022) indicated that personality functioning may serve as a key factor in the development of PTSD and especially cPTSD symptoms, in adults exposed to TCE. Future research could therefore examine whether impairments in personality functioning constitute a core vulnerability factor, bridging dimensional mechanisms and categorical diagnoses in trauma-related disorders.

3.3 General Strengths of this Thesis

A major strength of this thesis is the integration of categorical and dimensional perspectives, which addresses a critical gap in trauma research. Whereas most studies focus on isolated mechanisms within single diagnostic groups, this work examines the interplay of three mechanisms within a unified framework of trauma-related psychopathology. The inclusion of a wide range of populations, such as community, forensic, and patient samples (Study 1), case–control groups (Studies 2 and 3), and naturalistic, treatment-seeking patients (Study 3), enhances the ecological validity and generalizability of findings. A further strength is the direct comparison of PTSD and BPD with healthy controls, which remains rare despite their significant overlap. By examining TCE-related mechanisms across clinical and non-clinical populations, the thesis contributes to a more integrative understanding of how early adversity shapes shared and disorder-specific pathways to psychopathology. Moreover, to add significant theoretical value, this thesis emphasizes understudied mechanisms such as sleep disturbances and moves beyond dominant explanatory frameworks (e.g., hyperarousal model) to explore their link to emotion dysregulation.

Methodologically, the thesis is further strengthened by its multimethod approach, integrating evidence from a systematic review, experimental task-based research, and clinical patient data. Moreover, combining multiple levels of analysis, such as self-report, behavioural,

and neurophysiological (Study 2), is particularly important due to the multidimensional biopsychosocial impact of TCE. Additionally, this thesis employed extensive and standardised diagnostic assessments through structured clinical interviews (e.g., SCID, CAPS, IPDE) and solely validated psychometric measures in all three studies. To ensure conceptual clarity and comparability, the same definition of TCE was followed in each of the three studies based on the Childhood Trauma Questionnaire (CTQ) (Bernstein et al., 2003) and the same validated measure of emotion dysregulation (DERS) was used in Studies 2 and 3. Taken together, the thesis advances the field by integrating transdiagnostic, multimethod, and clinically grounded approaches.

3.4 General Limitations and Implications for Future Studies

Despite these strengths, the current findings should be evaluated while acknowledging limitations that should be addressed in future research. This thesis mainly explored associations between TCE and mechanisms in individuals with PTSD and BPD, and not further disorders that are also linked to TCE. In this regard, a key limitation of this thesis is that PTSD was not further distinguished from cPTSD, despite its particular relevance to TCE, social functioning, and emotion dysregulation described above. In the review, not a single study differentiated between PTSD and cPTSD or drew comparisons to BPD. Moreover, in Studies 2 and 3, we relied on DSM-5 definitions of PTSD, although severe childhood trauma was prevalent, and some individuals may have met ICD-11 criteria for cPTSD. Collapsing such cases into a single PTSD category likely increased heterogeneity and may have obscured cPTSD-specific patterns. Future research should therefore measure disturbances in self-organisation directly, to clarify whether the strongest effects concentrate on cPTSD and investigate the impact of the comorbidity of disorders.

Next to cPTSD, other disorders with a strong association to TCE and the three mechanisms are affective (depression, bipolar), anxiety and psychotic disorders (Hailes et al., 2019; Hjelseng et al., 2022; Li et al., 2020). In these populations, meta-analyses found a link between TCE and reduced social functioning, interpersonal relations and social motivation and a higher threat perception with more aggressive behaviour, with emotion processing serving as mediator (Fares-Otero, Alameda, et al., 2023; Fares-Otero, De Prisco, et al., 2023). Moreover, difficulties in emotion regulation mediate the link between TCE (particularly emotional abuse) and depression, anxiety, and psychosis (Feiler et al., 2023; Li et al., 2020). Similarly, TCE-related sleep disturbances have been found in schizophrenia and bipolar disorder (Laskemoen et al., 2021) and may partly mediate the association between TCE and symptoms of depression and anxiety (Javakhishvili & Widom, 2021). Future research should systematically compare the link between TCE and mechanisms across further diagnostic boundaries and dimensional constructs of psychopathology (e.g., personality functioning). This could advance transdiagnostic frameworks that conceptualise psychopathology along a continuum of shared risk factors and mechanisms.

Another key methodological limitation is our primary reliance on cross-sectional data with retrospective self-report measures of TCE, social impairment, emotion dysregulation and sleep disturbances. A meta-analysis by Baldwin et al. (2019) indicated that prospective and retrospective assessments of TCE showed poor agreement and captured distinct groups of individuals. Moreover, interpersonal impairment, emotion dysregulation and sleep disturbances have shown discrepancies when measured with subjective (i.e., self-report) versus more objective (e.g., experimental) or informant-rated (e.g., clinician) measures, which was also found in study 2 (Arditte Hall et al., 2023; Krause-Utz et al., 2019; Sauer et al., 2016; Woodward et al., 2015). Consequently, future research should address these methodological constraints by combining prospective longitudinal designs with multi-method assessments of

mechanisms in transdiagnostic samples. Integrating prospective documentations of TCE (e.g., child protection or medical records) with retrospective self-reports could help disentangle distinct risk pathways to later psychopathology (Baldwin et al., 2019). Longitudinal cohort studies are needed in which children with TCE are systematically followed into adolescence and adulthood to capture the causal and dynamic temporal interplays between TCE, mechanisms and psychopathology (McLaughlin et al., 2020). Moreover, assessing mechanisms through a combination of self-report, experimental paradigms, clinician ratings, and ecological momentary assessment (e.g., actigraphy or physiological markers of arousal and regulation) would provide a more nuanced and ecologically valid picture. Since this thesis primarily involved young, female participants (Studies 2 and 3), future research should also include more generalizable samples, as younger age and female gender are consistently linked to more severe rates of PTSD, cPTSD, BPD, and various psychopathologies (Hyland et al., 2019).

Additionally, the operationalisation of TCE itself presents further limitations. This thesis adhered to the definition by Bernstein et al. (2003) which does not include other forms of childhood adversity such as witnessing violence within the family, bullying, war exposure, dating violence in adolescence or prenatal substance use. Additionally, traumatic experiences after the age of 18 (e.g., sexual violence, accidents, etc.) were not controlled for, which could have influenced the symptomology and mechanisms. Moreover, this thesis only differentially examined TCE types in relation to subdomains of social functioning in Study 1. In studies 2 and 3, total CTQ scores were used to assess associations with global emotion dysregulation and sleep disturbances, possibly masking differential effects of specific trauma types (Cheng & Langevin, 2023; Hatkevich et al., 2021).

Trauma subtypes that seemed to be particularly overlooked in previous research, according to Study 1, were emotional abuse and neglect. Emotional maltreatment is often overlooked in clinical and research contexts due to its invisibility, definitional ambiguity and

lack of clear diagnostic criteria (i.e., trauma criterion A of PTSD) compared to physical/sexual abuse (d'Huart et al., 2022; Xiao et al., 2023). However, it is associated with a wide spectrum of internalising and externalising psychopathology and plays a critical role in etiological models and treatment approaches of BPD, PTSD/ cPTSD and depression, among others (Li et al., 2020; Linehan, 1993; Lortye et al., 2024). Emotional maltreatment has shown a strong impact on key transdiagnostic mechanisms like attachment insecurity, maladaptive schemas, emotion processing and sleep disturbances (Burns et al., 2010; Harb et al., 2025; Huang et al., 2025; Rosenstein et al., 2018).

To address these limitations, future research should adopt more nuanced, data-driven methods. Examples include CTQ-based clusters (Goerigk et al., 2023) or network analyses based on longitudinal data (Huang et al., 2025). These approaches explore links between varied patterns of childhood trauma exposures and transdiagnostic symptom dimensions. Cluster analyses group individuals by *natural co-occurrence patterns* of trauma types (e.g., emotional neglect and sexual abuse), rather than on arbitrary or predefined categories. This captures the heterogeneity typically seen in real-world trauma histories. Moreover, these clusters may also offer clinical value as they could guide personalised interventions for the consequences of TCE (Goerigk et al., 2024). Network models conceptualize psychopathology as interconnected symptom systems, instead of diagnostic categories (Epskamp et al., 2018). This may help to reveal how specific trauma patterns activate particular symptom clusters across diagnostic boundaries (Ciringione et al., 2025; Huang et al., 2025). Moreover, temporal and directional links among symptoms and mechanisms can be explored (e.g., whether emotion dysregulation precedes sleep disturbances or vice versa) (Liu et al., 2024). Within these models, factors like the onset (developmental timing), number and severity of TCE (e.g., chronic, multi-subtype), as well as adult trauma re-exposure should also be considered as they might differentially

impact the development of psychopathology and mechanisms (Ehring & Quack, 2010; Russotti et al., 2021; Warmingham et al., 2023).

3.5 Clinical Applications

To establish robust clinical implications, the mechanisms investigated in this thesis require replication and longitudinal evaluation to clarify the complex temporal interactions with TCE and psychopathology. Nevertheless, this thesis offers preliminary insights for further development of clinical assessments and transdiagnostic treatment approaches, particularly for PTSD and BPD.

Based on the findings of this thesis, it might be important to integrate mechanism-focused assessments of emotion dysregulation, social functioning, and sleep along with standard diagnostics in routine clinical care. These could be supplemented by ecological momentary assessments (EMA) via mobile health apps to track day-to-day coupling between mechanisms (e.g., daytime affect and nighttime sleep) on biopsychosocial levels (e.g. REM sleep, heart-rate variability; Hensler et al., 2021). Building on this, EMA have the potential to generate personalised symptom networks to visualise the dynamic interactions among daily triggers, trauma symptoms, and coping mechanisms, providing temporal priorities for intervention and treatment evaluation (Epskamp et al., 2018).

Findings from study 2 indicate that individuals with PTSD and BPD can successfully implement adaptive emotion regulation strategies under instruction. Mobile health apps like "*PTSD Coach*" for PTSD and "*Priovi*" for BPD can track distress due to daily triggers and deliver real-time interventions of adaptive regulation (e.g., relaxation exercises, cognitive restructuring) (Assmann et al., 2025; Bröcker et al., 2023; Hensler et al., 2021). Moreover, results of Study 3 suggested that interventions targeting pre-bedtime hyperarousal and maladaptive regulation might be able to reduce sleep disturbances. Consequently, by stabilising

emotion regulation early, patients can mitigate downstream interpersonal problems and sleep disturbances (van Trigt et al., 2025).

Furthermore, this thesis underscores emotion dysregulation as a pivotal and transdiagnostic mechanism influencing social functioning and sleep in individuals with TCE. While research indicates that PTSD, cPTSD, and BPD are distinct disorders with limited symptom network overlap, emotion dysregulation consistently emerges as a bridging mechanism (Cloitre et al., 2014; Knefel et al., 2016; Owczarek et al., 2023). Therefore, multiple interventions have been developed to target emotion dysregulation in TCE-related PTSD, cPTSD, and BPD to disrupt symptom interplay across these disorders. Next to disorder-specific treatments (e.g. DBT), these can be subdivided into *stage-based interventions* (e.g., DBT-PTSD) in which BPD treatment and PTSD treatment are combined or sequentially delivered and *transdiagnostic interventions* (e.g. BPD Compass), which are purely based on dimensional models of psychopathology (Sauer-Zavala et al., 2017; Zeifman et al., 2021).

DBT-PTSD, a stage-based treatment for cPTSD with or without comorbid BPD following childhood trauma, integrates DBT skills with exposure-based techniques to regulate trauma-associated primary (e.g., fear, disgust) and secondary emotions (e.g., guilt, shame). Randomised controlled trials (RCTs) demonstrate large, sustained effect sizes compared to Cognitive Processing Therapy and treatment-as-usual, improving cPTSD symptoms, emotion regulation, and interpersonal functioning (Bohus et al., 2013; Bohus et al., 2020; Bohus & Vonderlin, 2024; Vonderlin et al., 2024). Similarly, the skills training in affect and interpersonal regulation (STAIR) was designed for PTSD after childhood abuse (Cloitre et al., 2002). It combines skills training in emotion recognition, regulation and distress tolerance with exposure therapy, while interpersonal sessions address maladaptive schemas, assertive communication, and social dynamics (Cloitre et al., 2010). RCTs confirm STAIR's superiority

over control groups in enhancing PTSD symptoms, emotion regulation, and interpersonal functioning (Lorbeer et al., 2023).

Alternatively, *transdiagnostic treatment approaches* have been developed that also target the mechanisms found in this thesis, including Mentalization-Based Therapy (MBT; Bateman & Fonagy, 2016), schema therapy (Young et al., 2003) and DBT Compass. Trauma-Focused MBT (MBT-TF) targets emotion dysregulation by emphasising embodied mentalizing (i.e., linking bodily sensations to emotional states), which is vital for trauma survivors with cPTSD, PTSD, and BPD prone to dissociation and avoidance (Bateman et al., 2024; Smits et al., 2024). Schema therapy addresses emotion dysregulation by helping patients identify, understand, and modify maladaptive schemas, as deeply rooted patterns stemming from TCE, that underlie chronic patterns of emotional distress and reactivity. Additionally, BPD Compass, designed for comorbid PTSD-BPD, combines cognitive, behavioural, and mindfulness techniques to address negative affectivity, disinhibition, and antagonism

Based on the results of this thesis, both disorder-specific (e.g., impulsivity) and transdiagnostic alterations (e.g., emotion regulation) have been found following TCE, specifically in PTSD and BPD. Consequently, the decision between disorder-specific, stage-based or transdiagnostic interventions should be based on the individual phenomenology of the patient. For example, when affect lability, self-harm risk, and interpersonal instability are pronounced, stage-based protocols (e.g., DBT-PTSD; STAIR) might provide a more tolerable route into trauma processing. When problems span PTSD and BPD features, transdiagnostic frameworks (e.g., MBT, Schema therapy, BPD Compass) might target the shared and interrelated mechanisms.

3.6 **Conclusion**

This thesis elucidates the transdiagnostic link between TCE as a distal risk factor and social functioning, emotion processing, and sleep as proximal mechanisms in trauma-related

psychopathology. The findings of three studies extend current knowledge on how TCE shape overlapping and disorder-specific pathways, specifically in PTSD and BPD. The systematic review of Study 1 established TCE as a transdiagnostic risk factor for adult social impairment, mediated by psychopathology, emotion dysregulation, and attachment insecurity/ maladaptive schemas across diverse populations. The multimethod experimental findings of study 2 revealed that TCE was associated with self-reported emotion dysregulation across PTSD, BPD, and healthy controls, while laboratory-assessed emotional reactivity was tied more closely with current psychopathology. Lastly, the clinical and case–control investigations of study 3 indicated that TCE might exert an indirect effect on sleep disturbances through emotion dysregulation. Across this thesis, emotion dysregulation emerged as the most consistent pathway linking TCE to social and sleep disturbances. Together, these three processes appear to account for overlapping vulnerabilities across trauma-related disorders and disorder-specific features (e.g. fear of sleep in PTSD; impulsivity in BPD). These findings support dimensional frameworks of psychopathology while also supporting the identification of disorder-specific clinical profiles after TCE. Limited by the cross-sectional design and focus on two disorders, future research should employ longitudinal, ecologically valid studies in transdiagnostic samples to further explore features of complex PTSD and personality functioning. This thesis underscores the importance of mechanism-focused assessments for individuals with TCE in clinical practice, and it advocates targeting emotion dysregulation with transdiagnostic treatment approaches while also using disorder-specific interventions.

4 Deutsche Zusammenfassung

Traumatisierende Kindheitserfahrungen und Psychopathologie

Die Rolle von sozialer Funktionsfähigkeit, emotionaler Verarbeitung und Schlaf bei der Post-Traumatischen Belastungsstörung und Borderline-Persönlichkeitsstörung

Etablierte kategoriale Diagnosesysteme ermöglichen eine klare Abgrenzung psychischer Störungen, berücksichtigen jedoch nicht die hohe Komorbidität, Heterogenität und übergreifenden Entstehungsmechanismen psychischer Erkrankungen (Dalgleish et al., 2020). Forschungsergebnisse deuten darauf hin, dass psychische Prozesse nicht strikt durch willkürliche Grenzen zwischen „gesund“ und „pathologisch“ getrennt sind, sondern sich eher entlang eines kontinuierlichen Spektrums entfalten könnten.(Haslam et al., 2012; Haslam et al., 2020). In den letzten Jahren ist es daher zu einem Paradigmenwechsel hin zu dimensionalen und transdiagnostischen Ansätzen gekommen, die psychische Störungen anhand gemeinsamer Risikofaktoren und Mechanismen verstehen (Harvey et al., 2004).

Traumatische Kindheitserfahrungen gelten als zentraler transdiagnostischer Risikofaktor für die Entwicklung zahlreicher psychischer Erkrankungen (Jaffee, 2017; Lewis et al., 2021; Walsh et al., 2017). Sie werden definiert als emotionaler, physischer und sexueller Missbrauch sowie als emotionale und physische Vernachlässigung (Bernstein et al., 2003). Traumatische Kindheitserfahrungen können als *distaler Risikofaktor* verstanden werden, der *proximale biopsychosoziale Mechanismen* beeinflusst. Diese Mechanismen könnten gemeinsame Symptome und Komorbiditäten zwischen Störungen (*Multifinalität*) sowie die Entwicklung unterschiedlicher Störungsbilder (*divergente Entwicklungsverläufe*) erklären (Nolen-Hoeksema & Watkins, 2011). Traumatische Kindheitserfahrungen sind ein besonders bedeutsamer ätiologischer Risikofaktor für das Auftreten, den Schweregrad sowie die Komorbidität traumaassozierter Störungen wie der posttraumatischen Belastungsstörung (PTBS) und der Borderline-Persönlichkeitsstörung (BPS) (Hailes et al., 2019; Jowett et al., 2020; Krause-Utz, 2021).

Die PTBS ist eine psychische Störung, die als Folge der Konfrontation mit einem traumatischen Ereignis von extremer Belastung oder Lebensbedrohung auftreten kann (American Psychiatric Association, 2013). Die PTBS ist gekennzeichnet durch Intrusionen (z.

B. Albträume, Flashbacks), Vermeidung traumaassoziierter Reize, negative Veränderungen von Kognition und Affekt sowie Veränderungen der Reaktivität (z. B. Schlafstörungen, Hypervigilanz) (Bisson, 2007). Die Borderline-Persönlichkeitsstörung (BPS) zählt zu den schwerwiegenden psychischen Störungen und beginnt häufig in der frühen Adoleszenz (Bradley et al., 2007). Kennzeichen der BPS sind tiefgreifende Instabilität in zwischenmenschlichen Beziehungen, im Selbstbild, im Affekt sowie in der Impulsivität (American Psychiatric Association, 2013). Ein zentrales Merkmal der Störung ist eine ausgeprägte emotionale Dysregulation, die sich in erhöhter emotionaler Sensitivität, instabilen negativen Affekten sowie Defiziten adaptiver Emotionsregulationsstrategien äußert (Carpenter & Trull, 2013).

Die PTBS und BPS weisen eine hohe Prävalenz früher Traumatisierungen auf und zeigen starke Symptomüberlappungen (z. B. Veränderungen der Emotionsregulation und interpersoneller Beziehungen) sowie störungsspezifische Muster (z. B. Schlafstörungen, Impulsivität). Bisher mangelt es jedoch an umfassender Forschung, die die Auswirkungen traumatischer Kindheitserfahrungen auf gemeinsame biopsychosoziale Mechanismen der PTBS und BPS untersucht. Dieses Wissen wäre jedoch bedeutsam für die Weiterentwicklung von Präventions- und Interventionsansätzen, die gezielt übergreifende Beeinträchtigungen nach traumatischen Kindheitserfahrungen adressieren. Vor diesem Hintergrund untersuchte die vorliegende Dissertation Zusammenhänge zwischen traumatischen Kindheitserfahrungen und drei proximalen Mechanismen bei Personen mit PTBS und BPS: *sozialer Funktionsfähigkeit, emotionaler Verarbeitung und Schlaf.*

In Studie 1 wurden in einer systematischen Übersichtsarbeit die Zusammenhänge zwischen verschiedenen Arten traumatischer Kindheitserfahrungen und Beeinträchtigungen der sozialen Funktionsfähigkeit im Erwachsenenalter untersucht. Traumatische Kindheitserfahrungen waren mit verschiedenen Domänen der sozialen Funktionsfähigkeit

sowohl in klinischen als auch in nicht-klinischen Populationen assoziiert. Die stärkste Evidenz zeigte sich für Zusammenhänge mit Gewalt in Paarbeziehungen, Aggression, reduzierte soziale Verbundenheit, unsichere Bindung und frühe maladaptive Schemata, wohingegen die Evidenz für Verbindungen mit Sexualität, Paarbeziehungen und sozial-kognitiven Prozessen Inkonsistenzen aufwies. Psychopathologie (z.B. PTBS- und BPS-Symptome), unsichere Bindung, frühe maladaptive Schemata sowie Emotionsdysregulation fungierten als zentrale vermittelnde Mechanismen zwischen traumatischen Kindheitserfahrungen und sozialer Dysfunktion. Diese Ergebnisse lassen vermuten, dass traumatische Kindheitserfahrungen mit einer langfristigen Beeinträchtigung der sozialen Funktionsfähigkeit in Zusammenhang stehen könnten, was auf einen potenziellen transdiagnostischen Mechanismus hinweist.

In Studie 2 wurde mithilfe eines multimethodischen experimentellen Designs der Zusammenhang zwischen traumatischen Kindheitserfahrungen und emotionaler Reaktivität sowie der emotionalen Regulation untersucht. Neben Selbstberichten wurden Verhaltensdaten und neurophysiologische Parameter (ereigniskorrelierte Potenziale, EKP) bei Frauen mit PTBS und BPS sowie bei gesunden Kontrollprobanden erfasst. Traumatische Kindheitserfahrungen waren mit einer reduzierten emotionalen Reaktivität auf positive (r = -.35, p < .001) und neutrale Reize (r = -.22, p = .011) assoziiert, was auf eine emotionale Abstumpfung hindeuten könnte (Clarke et al., 2024; Sill et al., 2020; Wu et al., 2023). Nach Kontrolle der Gruppenzugehörigkeit und der Medikation erwies sich dieser Effekt jedoch als insignifikant. Darüber hinaus zeigten traumatische Kindheitserfahrungen keine Zusammenhänge mit neurophysiologischen Korrelaten der Emotionsverarbeitung (EKPs) oder mit den Effekten instruierter Emotionsregulation (z. B. kognitiver Neubewertung). Der Zusammenhang zwischen traumatischen Kindheitserfahrungen und selbstberichteter Emotionsdysregulation (β = .24, p = .004) blieb jedoch nach Berücksichtigung des Einflusses der Gruppe und der Medikation robust. Diese Ergebnisse deuten darauf hin, dass traumatische

Kindheitserfahrungen möglicherweise eine Vulnerabilität für emotionale Dysregulation begünstigen, die sich in beiden Störungsbildern zeigte, jedoch keinen starken Einfluss auf die situationsabhängige Reaktivität und Regulation haben (Daros et al., 2013). Zugleich zeigten sich störungsspezifische Muster. Patientinnen mit PTBS wiesen bei neutralen Reizen eine stärkere emotionale Hyporeaktivität auf, während Patientinnen mit BPS eine ausgeprägtere globale Dysregulation und Impulsivität berichteten.

Studie 3 widmete sich dem Zusammenhang zwischen traumatischen Kindheitserfahrungen und Schlafstörungen des letzten Monats und der letzten Nacht anhand dreier komplementärer Stichproben. Die Analysen basierten auf Queerschnittdaten aus Selbstberichten von behandlungssuchenden Patientinnen mit PTBS sowie aus Fall-Kontroll-Gruppen mit PTBS und BPS sowie gesunden Kontrollprobanden. Traumatische Kindheitserfahrungen waren signifikant mit verschiedenen Formen von Schlafstörungen korreliert (r = .26 bis .56, p < .05): niedrigere Schlafqualität, (stärkere Belastung durch Albträume), größere Angst vor dem Schlaf, verringerte Erholung, psychische Ausgeglichenheit und verstärkte Erschöpfung am Abend und psychosomatische Symptome in der Schlafphase. Wurde jedoch Emotionsdysregulation als Kovariate berücksichtigt, verlor der direkte Zusammenhang seine Signifikanz, was auf eine indirekte Wirkung über emotionale Dysregulation hindeutet. Emotionale Dysregulation war mit einer verringerten Schlafqualität, Albträumen, Angst vor dem Schlafen sowie Schlafstörungen in der letzten Nacht verbunden, unabhängig von Symptomen der Hypervigilanz, Gruppenzugehörigkeit und traumatischen Kindheitserfahrungen (β = .21 bis .37; p < .046). Personen mit PTBS und BPS zeigten ähnliche Schlafbeeinträchtigungen, wobei Angst vor dem Schlaf bei PTBS-Patientinnen stärker ausgeprägt war, und Schlafstörungen bei BPS-Patientinnen überwiegend durch ausgeprägtere Emotionsdysregulation erklärt wurden.

In der integrativen Betrachtung der drei Studien zeigt sich, dass traumatische Kindheitserfahrungen eine breite, transdiagnostische Vulnerabilität erzeugen, die sich in Störungen der sozialen Funktionsfähigkeit, der Emotionsverarbeitung und des Schlafs niederschlägt. Emotionsdysregulation erwies sich dabei als zentraler Mechanismus, der traumatische Kindheitserfahrungen mit sozialer Dysfunktion und Schlafstörungen verbindet. Gleichzeitig ließen sich übergreifende Vulnerabilitäten sowie störungsspezifische Muster identifizieren (z. B. Angst vor Schlaf bei PTBS, Impulsivität bei BPS), die im Einklang mit den theoretischen Modellen von Ehlers, Clark & KollegInnen (2000) für die PTBS und Linehan & KollegInnen (1993) für die BPS stehen. Bei der PTBS könnten kognitive Bewertungen des Traumas und vermeidende Bewältigungsmechanismen eine größere Rolle spielen, während bei der BPS affektive und interpersonelle Mechanismen stärker beteiligt sein könnten.

Die Befunde unterstützen somit einen hybriden dimensional–kategorialen Ansatz, der sowohl gemeinsame Mechanismen als auch störungsspezifische Ausprägungen nach traumatischen Kindheitserfahrungen abbildet. Besonders die anhaltenden *Störungen der Selbstorganisation* (DSO; Affektregulation, Selbstkonzept, Beziehungsfähigkeit), die in der *komplexen PTBS* (kPTBS) auftreten, bieten hierfür ein nützliches Bezugsmodell (Ford & Courtois, 2021; Río-Casanova et al., 2016). Zudem wird Personlichkeitspathologie im „Alternative Model for Personality Disorder“ (AMPD) dimensional entlang des *Funktionsniveaus der Persönlichkeit* definiert (Bender et al., 2011). Erste Forschungsergebnisse weisen darauf hin, dass Beeinträchtigungen der Selbst- und der zwischenmenschlichen Funktionen einen übergeordneten Mechanismus abbilden, über den traumatische Kindheitserfahrungen transdiagnostische Vulnerabilität vermitteln könnten (d’Huart et al., 2022; Freier et al., 2022; Kampling et al., 2022; Kerber et al., 2023; Krakau et al., 2021).

Zu den Stärken dieser Dissertation zählen die Kombination aus mehreren methodischen Ansätzen (systematischem Review, experimenteller Laborstudie, klinischer Stichproben) und Beobachtungsebenen (Selbstbericht, Verhaltensdaten, neurophysiologische Parameter) sowie der Einsatz standardisierter diagnostischer Interviews und ausschließlich validierter psychometrischer Messinstrumente. Zudem wurden kategoriale und dimensionale Perspektiven integriert und Zusammenhänge über mehrere Populationen hinweg untersucht (u.a. klinische und Nicht-Klinische Gruppen, PTBS, BPS, etc.).

Methodische Einschränkungen ergeben sich aus dem querschnittlichen Design, der retrospektiven Erfassung traumatischer Kindheitserfahrungen mittels Selbstberichts und der Fokussierung auf die PTBS und BPS als zwei modellhafte Störungsbilder. Dadurch sind kausale und transdiagnostische Zusammenhänge nur eingeschränkt abbildbar. Weitere Forschung ist daher erforderlich, um die Befunde zu replizieren und die komplexen zeitlichen Wechselwirkungen zwischen traumatischen Kindheitserfahrungen, Mechanismen und Psychopathologie zu klären. Die Zusammenhänge sollten systematisch in weiteren Störungsbildern (z. B. Depressionen) sowie entlang dimensionaler Konstrukte (z. B. Störungen der Selbstorganisation, Funktionsniveau der Persönlichkeit) untersucht werden, um störungsübergreifende und spezifische Mechanismen klarer zu differenzieren. Zukünftige Studien sollten längsschnittliche, transdiagnostische und multimodale Designs verwenden und datengetriebene Ansätze (z. B. Cluster- oder Netzwerkmodelle) einsetzen.

Die Ergebnisse dieser Dissertation könnten erste Hinweise auf potenzielle klinische Implikationen liefern. Auf ihrer Grundlage erscheint es bedeutsam, Mechanismen der emotionalen Regulation, sozialen Funktionsfähigkeit und des Schlafs in Patientinnen mit traumatischen Kindheitserfahrungen zu erfassen. Diese könnten durch „Ecological Momentary Assessments“ (EMA) mithilfe mobiler Apps ergänzt werden, um die alltäglichen Wechselwirkungen zwischen Mechanismen (z. B. Affekt und Schlaf) auf biopsychosozialer

Ebene zu erfassen (Hensler et al., 2021). Eine frühzeitige Stabilisierung der Emotionsregulation könnte dazu beitragen, interpersonelle und schlafbezogene Probleme zu reduzieren. Je nach individueller Symptomkonstellation können transdiagnostische Behandlungsansätze oder störungsspezifische Interventionen eingesetzt werden, um sowohl gemeinsame Mechanismen als auch Störungsausprägungen zu adressieren (Bohus & Vonderlin, 2024; Cloitre et al., 2002).

Zusammenfassend legen die Ergebnisse dieser Dissertation nahe, dass traumatische Kindheitserfahrungen mit Beeinträchtigungen der sozialen Funktionsfähigkeit, der emotionalen Verarbeitung und des Schlafs in Zusammenhang stehen könnten. Emotionsdysregulation erwies sich dabei als zentrale transdiagnostische Schnittstelle, die übergreifende wie auch spezifische Verläufe nach traumatischen Kindheitserfahrungen erklären könnte. Damit könnte die Dissertation einen Beitrag zu einem integrativen, mechanismenbasierten Verständnis traumaassoziierter Psychopathologie leisten.

References

Aafjes-van Doorn, K., Kamsteeg, C., & Silberschatz, G. (2020). Cognitive mediators of the relationship between adverse childhood experiences and adult psychopathology: A systematic review. *Dev Psychopathol*, *32*(3), 1017-1029. https://doi.org/10.1017/s0954579419001317

Adenauer, H., Pinösch, S., Catani, C., Gola, H., Keil, J., Kissler, J., & Neuner, F. (2010). Early processing of threat cues in posttraumatic stress disorder-evidence for a cortical vigilance-avoidance reaction. *Biol Psychiatry*, *68*(5), 451-458. https://doi.org/10.1016/j.biopsych.2010.05.015

Agorastos, A., & Olff, M. (2020). Traumatic stress and the circadian system: neurobiology, timing and treatment of posttraumatic chronodisruption. *Eur J Psychotraumatol*, *11*(1), 1833644. https://doi.org/10.1080/20008198.2020.1833644

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clin Psychol Rev*, *30*(2), 217-237. https://doi.org/10.1016/j.cpr.2009.11.004

Alisic, E., Zalta, A. K., van Wesel, F., Larsen, S. E., Hafstad, G. S., Hassanpour, K., & Smid, G. E. (2014). Rates of post-traumatic stress disorder in trauma-exposed children and adolescents: meta-analysis. *British Journal of Psychiatry*, *204*(5), 335-340. https://doi.org/10.1192/bjp.bp.113.131227

Alonso, J., Angermeyer, M. C., Bernert, S., Bruffaerts, R., Brugha, T. S., Bryson, H.,…Vollebergh, W. A. M. (2004). Prevalence of mental disorders in Europe: results from the European Study of the Epidemiology of Mental Disorders (ESEMeD) project. *Acta Psychiatrica Scandinavica*, *109*(s420), 21-27. https://doi.org/10.1111/j.1600-0047.2004.00327.x

Amad, A., Radua, J., Vaiva, G., Williams, S. C. R., & Fovet, T. (2019). Similarities between borderline personality disorder and post traumatic stress disorder: Evidence from resting-state meta-analysis [Review]. *Neuroscience and biobehavioral reviews*, *105*, 52-59. https://doi.org/10.1016/j.neubiorev.2019.07.018

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. American Psychiatric Publishing. https://doi.org/10.1176/appi.books.9780890425596

Andrews, S., & Hanna, P. (2020). Investigating the psychological mechanisms underlying the relationship between nightmares, suicide and self-harm. *Sleep Medicine Reviews*, *54*, 101352. https://doi.org/10.1016/j.smrv.2020.101352

APA. (2025). APA Clinical Practice Guideline for the Treatment of Posttraumatic Stress Disorder (PTSD) in Adults. (https://www.apa.org/about/policy/guideline-ptsd-in-adults.pdf)

Arditte Hall, K. A., B., W. K., G., G. M., & and Galovski, T. E. (2023). Exploring Predictors of Sleep State Misperception in Women with Posttraumatic Stress Disorder. *Behavioral Sleep Medicine*, *21*(1), 22-32. https://doi.org/10.1080/15402002.2021.2024193

Arntz, A., Weertman, A., & Salet, S. (2011). Interpretation bias in Cluster-C and borderline personality disorders. *Behav Res Ther*, *49*(8), 472-481. https://doi.org/10.1016/j.brat.2011.05.002

Assed, M. M., Khafif, T. C., Belizario, G. O., Fatorelli, R., Rocca, C. C. d. A., & de Pádua Serafim, A. (2020). Facial Emotion Recognition in Maltreated Children: A Systematic Review. *Journal of Child and Family Studies*, *29*(5), 1493-1509. https://doi.org/10.1007/s10826-019-01636-w

Assmann, N., Jacob, G., Schaich, A., Berger, T., Zindler, T., Betz, L.,…Klein, J. P. (2025). A digital therapeutic for people with borderline personality disorder in Germany

(EPADIP-BPD): a pragmatic, assessor-blind, parallel-group, randomised controlled trial. *The Lancet Psychiatry*, *12*(5), 366-376. https://doi.org/10.1016/S2215-0366(25)00063-X

Back, S. N., Flechsenhar, A., Bertsch, K., & Zettl, M. (2021). Childhood Traumatic Experiences and Dimensional Models of Personality Disorder in DSM-5 and ICD-11: Opportunities and Challenges. *Current Psychiatry Reports*, *23*(9), 60. https://doi.org/10.1007/s11920-021-01265-5

Back, S. N., Zettl, M., Bertsch, K., & Taubner, S. (2020). Persönlichkeitsfunktionsniveau, maladaptive Traits und Kindheitstraumata. *Psychotherapeut*, *65*(5), 374-382. https://doi.org/10.1007/s00278-020-00445-7

Bader, K., Schäfer, V., Schenkel, M., Nissen, L., & Schwander, J. (2007). Adverse childhood experiences associated with sleep in primary insomnia. *Journal of Sleep Research*, *16*(3), 285-296. https://doi.org/10.1111/j.1365-2869.2007.00608.x

Baldwin, J. R., Reuben, A., Newbury, J. B., & Danese, A. (2019). Agreement Between Prospective and Retrospective Measures of Childhood Maltreatment: A Systematic Review and Meta-analysis. *JAMA Psychiatry*, *76*(6), 584-593. https://doi.org/10.1001/jamapsychiatry.2019.0097

Bateman, A., & Fonagy, P. (2016). *Mentalization-based treatment for personality disorders: A practical guide*. Oxford University Press. https://doi.org/10.1093/med:psych/9780199680375.001.0001

Bateman, A., Rüfenacht, E., Perroud, N., Debbané, M., Nolte, T., Shaverin, L., & Fonagy, P. (2024). Childhood maltreatment, dissociation and borderline personality disorder: Preliminary data on the mediational role of mentalizing in complex post-traumatic stress disorder. *Psychol Psychother*, *97 Suppl 1*, 58-74. https://doi.org/10.1111/papt.12514

Ben Simon, E., Vallat, R., Barnes, C. M., & Walker, M. P. (2020). Sleep Loss and the Socio-Emotional Brain. *Trends in Cognitive Sciences*, *24*(6), 435-450. https://doi.org/10.1016/j.tics.2020.02.003

Bender, D. S., Morey, L. C., & Skodol, A. E. (2011). Toward a Model for Assessing Level of Personality Functioning in DSM–5, Part I: A Review of Theory and Methods. *Journal of Personality Assessment*, *93*(4), 332-346. https://doi.org/10.1080/00223891.2011.583808

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T.,…Zule, W. (2003). Development and validation of a brief screening version of the childhood trauma questionnaire. *Child Abuse & Neglect*, *27*(2), 169-190. https://doi.org/10.1016/s0145-2134(02)00541-0

Bertsch, K., Hillmann, K., & Herpertz, Sabine C. (2018). Behavioral and Neurobiological Correlates of Disturbed Emotion Processing in Borderline Personality Disorder. *Psychopathology*, *51*(2), 76-82. https://doi.org/10.1159/000487363

Bérubé, A., Turgeon, J., Blais, C., & Fiset, D. (2023). Emotion Recognition in Adults With a History of Childhood Maltreatment: A Systematic Review. *Trauma, Violence, & Abuse*, *24*(1), 278-294. https://doi.org/10.1177/15248380211029403

Bisson, J. I. (2007). Post-traumatic stress disorder. *Occupational Medicine*, *334*(7597), 789-793. https://doi.org/10.1093/occmed/kqm069

Bohus, M., Dyer, A. S., Priebe, K., Krüger, A., Kleindienst, N., Schmahl, C.,…Steil, R. (2013). Dialectical behaviour therapy for post-traumatic stress disorder after childhood sexual abuse in patients with and without borderline personality disorder: a randomised controlled trial. *Psychother Psychosom*, *82*(4), 221-233. https://doi.org/10.1159/000348451

Bohus, M., Kleindienst, N., Hahn, C., Müller-Engelmann, M., Ludäscher, P., Steil, R.,…Priebe, K. (2020). Dialectical Behavior Therapy for Posttraumatic Stress Disorder (DBT-PTSD) Compared With Cognitive Processing Therapy (CPT) in Complex Presentations of PTSD in Women Survivors of Childhood Abuse: A Randomized Clinical Trial. *JAMA Psychiatry*, *77*(12), 1235-1245. https://doi.org/10.1001/jamapsychiatry.2020.2148

Bohus, M., & Vonderlin, R. (2024). Dialektisch behaviorale Therapie für komplexe posttraumatische Belastungsstörung (DBT-PTBS): ein evidenzbasiertes störungsspezfisches Behandlungsprogramm. *Der Nervenarzt*, *95*(7), 630-638. https://doi.org/10.1007/s00115-024-01680-y

Bortolla, R., Cavicchioli, M., Fossati, A., & Maffei, C. (2020). Emotional Reactivity in Borderline Personality Disorder: Theoretical Considerations Based on a Meta-Analytic Review of Laboratory Studies. *J Pers Disord*, *34*(1), 64-87. https://doi.org/10.1521/pedi_2018_32_382

Bowlby, J. (1982). Attachment and loss: Retrospect and prospect. *American Journal of Orthopsychiatry*, *52*(4), 664-678. https://doi.org/10.1111/j.1939-0025.1982.tb01456.x

Boyd, J. E., O'Connor, C., Protopopescu, A., Jetly, R., Lanius, R. A., & McKinnon, M. C. (2020). The contributions of emotion regulation difficulties and dissociative symptoms to functional impairment among civilian inpatients with posttraumatic stress symptoms. *Psychol Trauma*, *12*(7), 739-749. https://doi.org/10.1037/tra0000576

Bradley, R., Conklin, C. Z., & Westen, D. (2007). Borderline Personality Disorder. In W. O'Donohue, K. Fowler, S. Lilienfeld, & R. Bradley (Eds.), *Personality disorders: Toward the DSM-V.* (pp. 167–201). SAGE Publications, Inc. https://doi.org/10.4135/9781483328980.n7

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder. *Psychol Rev*, *103*(4), 670-686. https://doi.org/10.1037/0033-295x.103.4.670

Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clin Psychol Rev*, *23*(3), 339-376. https://doi.org/10.1016/s0272-7358(03)00033-3

Briggs-Gowan, M. J., Grasso, D., Bar-Haim, Y., Voss, J., McCarthy, K. J., Pine, D. S., & Wakschlag, L. S. (2016). Attention bias in the developmental unfolding of post-traumatic stress symptoms in young children at risk. *Journal of Child Psychology and Psychiatry*, *57*(9), 1083-1091. https://doi.org/10.1111/jcpp.12577

Bröcker, E., Suliman, S., Olff, M., & Seedat, S. (2023). Feasibility, acceptability, and effectiveness of web-based and mobile PTSD Coach: a systematic review and meta-analysis. *European Journal of Psychotraumatology*, *14*(2), 2209469. https://doi.org/10.1080/20008066.2023.2209469

Brumariu, L. E., Owen, M. T., Dyer, N., & Lyons-Ruth, K. (2020). Developmental Pathways to BPD-Related Features in Adolescence: Infancy to Age 15. *J Pers Disord*, *34*(Suppl B), 104-129. https://doi.org/10.1521/pedi_2020_34_480

Bryant, R. A., Galatzer-Levy, I., & Hadzi-Pavlovic, D. (2023). The Heterogeneity of Posttraumatic Stress Disorder in DSM-5. *JAMA Psychiatry*, *80*(2), 189-191. https://doi.org/10.1001/jamapsychiatry.2022.4092

Burns, E. E., Jackson, J. L., & Harding, H. G. (2010). Child Maltreatment, Emotion Regulation, and Posttraumatic Stress: The Impact of Emotional Abuse. *Journal of Aggression, Maltreatment & Trauma*, *19*(8), 801-819. https://doi.org/10.1080/10926771.2010.522947

Calhoun, C. D., Stone, K. J., Cobb, A. R., Patterson, M. W., Danielson, C. K., & Bendezú, J. J. (2022). The Role of Social Support in Coping with Psychological Trauma: An

Integrated Biopsychosocial Model for Posttraumatic Stress Recovery. *Psychiatr Q*, *93*(4), 949-970. https://doi.org/10.1007/s11126-022-10003-w

Carlson, E. M., Cox, D. W., Kealy, D., Chapman, A. L., & Ogrodniczuk, J. S. (2020). Social role dysfunction and coping in borderline personality disorder. *Personality and Mental Health*, *14*(2), 227-239. https://doi.org/10.1002/pmh.1476

Carpenter, R. W., & Trull, T. J. (2013). Components of emotion dysregulation in borderline personality disorder: a review. *Curr Psychiatry Rep*, *15*(1), 335. https://doi.org/10.1007/s11920-012-0335-2

Carr, A., Duff, H., & Craddock, F. (2020). A systematic review of reviews of the outcome of noninstitutional child maltreatment. *Trauma Violence Abuse*, *21*(4), 828-843. https://doi.org/10.1177/1524838018801334

Cattane, N., Rossi, R., Lanfredi, M., & Cattaneo, A. (2017). Borderline personality disorder and childhood trauma: exploring the affected biological systems and mechanisms. *BMC Psychiatry*, *17*(1), 221. https://doi.org/10.1186/s12888-017-1383-2

Chapman, D. P., Liu, Y., Presley-Cantrell, L. R., Edwards, V. J., Wheaton, A. G., Perry, G. S., & Croft, J. B. (2013). Adverse childhood experiences and frequent insufficient sleep in 5 U.S. States, 2009: a retrospective cohort study. *BMC Public Health*, *13*, 3. https://doi.org/10.1186/1471-2458-13-3

Chapman, D. P., Wheaton, A. G., Anda, R. F., Croft, J. B., Edwards, V. J., Liu, Y.,…Perry, G. S. (2011). Adverse childhood experiences and sleep disturbances in adults. *Sleep Medicine*, *12*(8), 773-779. https://doi.org/10.1016/j.sleep.2011.03.013

Cheng, P., & Langevin, R. (2023). Unpacking the effects of child maltreatment subtypes on emotional competence in emerging adults. *Psychol Trauma*, *15*(Suppl 1), S102-s111. https://doi.org/10.1037/tra0001322

Christ, N. M., Elhai, J. D., Forbes, C. N., Gratz, K. L., & Tull, M. T. (2021). A machine learning approach to modeling PTSD and difficulties in emotion regulation. *Psychiatry Research*, *297*, 113712. https://doi.org/10.1016/j.psychres.2021.113712

Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*(4), 597-600. https://doi.org/10.1017/S0954579400007318

Ciringione, L., Perinelli, E., Mancini, F., & Prunetti, E. (2025). Beyond the Scars: An Analysis of Adverse Childhood Experiences and the Interconnections Between Emotion Dysregulation, Dissociation, and Trauma in Patients with Borderline Personality Disorder. *Brain Sci*, *15*(8). https://doi.org/10.3390/brainsci15080889

Clarke, S. D., Riser, D. K., & Schmidt, M. S. (2024). Electrocortical Correlates of Emotion Processing and Resilience in Individuals with Adverse Childhood Experiences. *Journal of Child & Adolescent Trauma*, *17*(3), 1-16. https://doi.org/10.1007/s40653-024-00621-w

Cloitre, M. (2020). ICD-11 complex post-traumatic stress disorder: simplifying diagnosis in trauma populations. *The British Journal of Psychiatry*, *216*(3), 129-131. https://doi.org/10.1192/bjp.2020.43

Cloitre, M., Garvert, D. W., Brewin, C. R., Bryant, R. A., & Maercker, A. (2013). Evidence for proposed ICD-11 PTSD and complex PTSD: a latent profile analysis. *European Journal of Psychotraumatology*, *4*(1), 20706. https://doi.org/10.3402/ejpt.v4i0.20706

Cloitre, M., Garvert, D. W., Weiss, B., Carlson, E. B., & Bryant, R. A. (2014). Distinguishing PTSD, Complex PTSD, and Borderline Personality Disorder: A latent class analysis. *Eur J Psychotraumatol*, *5*. https://doi.org/10.3402/ejpt.v5.25097

Cloitre, M., Koenen, K. C., Cohen, L. R., & Han, H. (2002). Skills training in affective and interpersonal regulation followed by exposure: a phase-based treatment for PTSD

related to childhood abuse. *J Consult Clin Psychol*, *70*(5), 1067-1074. https://doi.org/10.1037//0022-006x.70.5.1067

Cloitre, M., Shevlin, M., Brewin, C. R., Bisson, J. I., Roberts, N. P., Maercker, A.,…Hyland, P. (2018). The International Trauma Questionnaire: development of a self-report measure of ICD-11 PTSD and complex PTSD. *Acta Psychiatrica Scandinavica*, *138*(6), 536-546. https://doi.org/10.1111/acps.12956

Cloitre, M., Stovall-McClough, K. C., Nooner, K., Zorbas, P., Cherry, S., Jackson, C. L.,…Petkova, E. (2010). Treatment for PTSD related to childhood abuse: a randomized controlled trial. *Am J Psychiatry*, *167*(8), 915-924. https://doi.org/10.1176/appi.ajp.2010.09081247

Cludius, B., Mennin, D., & Ehring, T. (2020). Emotion regulation as a transdiagnostic process. *Emotion*, *20*(1), 37-42. https://doi.org/10.1037/emo0000646

Couette, M., Mouchabac, S., Bourla, A., Nuss, P., & Ferreri, F. (2020). Social cognition in post-traumatic stress disorder: A systematic review. *British Journal of Clinical Psychology*, *59*(2), 117-138. https://doi.org/10.1111/bjc.12238

Cross, D., Fani, N., Powers, A., & Bradley, B. (2017). Neurobiological Development in the Context of Childhood Trauma. *Clin Psychol (New York)*, *24*(2), 111-124. https://doi.org/10.1111/cpsp.12198

Crow, T. M., Levy, K. N., Bradley, B., Fani, N., & Powers, A. (2021). The roles of attachment and emotion dysregulation in the association between childhood maltreatment and PTSD in an inner-city sample. *Child Abuse & Neglect*, *118*, 105139. https://doi.org/10.1016/j.chiabu.2021.105139

Crowell, S., Beauchaine, T., & Linehan, M. (2009). A Biosocial Developmental Model of Borderline Personality: Elaborating and Extending Linehan's Theory. *Psychological Bulletin*, *135*, 495-510. https://doi.org/10.1037/a0015616

Cyr, C., Euser, E. M., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2010). Attachment security and disorganization in maltreating and high-risk families: A series of meta-analyses. *Development and Psychopathology*, *22*(1), 87-108. https://doi.org/10.1017/S0954579409990289

d'Huart, D., Hutsebaut, J., Seker, S., Schmid, M., Schmeck, K., Bürgin, D., & Boonmann, C. (2022). Personality functioning and the pathogenic effect of childhood maltreatment in a high-risk sample. *Child and Adolescent Psychiatry and Mental Health*, *16*(1), 95. https://doi.org/10.1186/s13034-022-00527-1

da Silva, H. C., Vilete, L., Coutinho, E. S. F., Luz, M. P., Mendlowicz, M., Portela, C. M.,…Berger, W. (2024). The role of childhood cumulative trauma in the risk of lifetime PTSD: An epidemiological study. *Psychiatry Research*, *336*, 115887. https://doi.org/10.1016/j.psychres.2024.115887

Dalgleish, T., Black, M., Johnston, D., & Bevan, A. (2020). Transdiagnostic approaches to mental health problems: Current status and future directions. 179-195. https://doi.org/10.1037/ccp0000482

Daros, A. R., Guevara, M. A., Uliaszek, A. A., McMain, S. F., & Ruocco, A. C. (2018). Cognitive Emotion Regulation Strategies in Borderline Personality Disorder: Diagnostic Comparisons and Associations with Potentially Harmful Behaviors. *Psychopathology*, *51*(2), 83-95. https://doi.org/10.1159/000487008

Daros, A. R., & Williams, G. E. (2019). A Meta-analysis and Systematic Review of Emotion-Regulation Strategies in Borderline Personality Disorder. *Harv Rev Psychiatry*, *27*(4), 217-232. https://doi.org/10.1097/hrp.0000000000000212

Daros, A. R., Zakzanis, K. K., & Ruocco, A. C. (2013). Facial emotion recognition in borderline personality disorder. *Psychological Medicine*, *43*(9), 1953-1963. https://doi.org/10.1017/S0033291712002607

DGPPN. (2022). *S3-Leitlinie Borderline-Persönlichkeitsstörung* (Vol. AWMF-Register Nr. 038-015). AWMF.

Dixon-Gordon, K. L., Gratz, K. L., & Tull, M. T. (2013). Multimodal assessment of emotional reactivity in borderline personality pathology: the moderating role of posttraumatic stress disorder symptoms. *Compr Psychiatry*, *54*(6), 639-648. https://doi.org/10.1016/j.comppsych.2012.12.019

Dolan, M., Slavish, D. C., Weiss, N. H., & Contractor, A. A. (2023). The Role of Emotion Dysregulation in the Relationship Between Sleep Disturbances and PTSD Symptom Severity. *J Nerv Ment Dis*, *211*(3), 203-215. https://doi.org/10.1097/nmd.0000000000001631

Drews, E., Fertuck, E. A., Koenig, J., Kaess, M., & Arntz, A. (2019). Hypothalamic-pituitary-adrenal axis functioning in borderline personality disorder: A meta-analysis. *Neuroscience & Biobehavioral Reviews*, *96*, 316-334. https://doi.org/10.1016/j.neubiorev.2018.11.008

Dvir, Y., Ford, J. D., Hill, M., & Frazier, J. A. (2014). Childhood maltreatment, emotional dysregulation, and psychiatric comorbidities. *Harv Rev Psychiatry*, *22*(3), 149-161. https://doi.org/10.1097/hrp.0000000000000014

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behav Res Ther*, *38*(4), 319-345. https://doi.org/10.1016/s0005-7967(99)00123-0

Ehring, T., & Quack, D. (2010). Emotion Regulation Difficulties in Trauma Survivors: The Role of Trauma Type and PTSD Symptom Severity. *Behavior Therapy*, *41*(4), 587-598. https://doi.org/10.1016/j.beth.2010.04.004

Epskamp, S., van Borkulo, C. D., van der Veen, D. C., Servaas, M. N., Isvoranu, A.-M., Riese, H., & Cramer, A. O. J. (2018). Personalized Network Modeling in Psychopathology:

The Importance of Contemporaneous and Temporal Connections. *Clinical Psychological Science*, *6*(3), 416-427. https://doi.org/10.1177/2167702617744325

Fairholme, C. P., Nosen, E. L., Nillni, Y. I., Schumacher, J. A., Tull, M. T., & Coffey, S. F. (2013). Sleep disturbance and emotion dysregulation as transdiagnostic processes in a comorbid sample. *Behaviour Research and Therapy*, *51*(9), 540-546. https://doi.org/10.1016/j.brat.2013.05.014

Fares-Otero, N. E., Alameda, L., Pfaltz, M. C., Martinez-Aran, A., Schäfer, I., & Vieta, E. (2023). Examining associations, moderators and mediators between childhood maltreatment, social functioning, and social cognition in psychotic disorders: A systematic review and meta-analysis. *Psychological Medicine*, *53*(13), 5909-5932. https://doi.org/10.1017/S0033291723001678

Fares-Otero, N. E., De Prisco, M., Oliva, V., Radua, J., Halligan, S. L., Vieta, E., & Martínez-Arán, A. (2023). Association between childhood maltreatment and social functioning in individuals with affective disorders: A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, *148*(2), 142-164. https://doi.org/10.1111/acps.13557

Feiler, T., Vanacore, S., & Dolbier, C. (2023). Relationships Among Adverse and Benevolent Childhood Experiences, Emotion Dysregulation, and Psychopathology Symptoms. *Adversity and Resilience Science*, *4*(3), 273-289. https://doi.org/10.1007/s42844-023-00094-0

Fertuck, E. A., Grinband, J., & Stanley, B. (2013). Facial trust appraisal negatively biased in borderline personality disorder. *Psychiatry Res*, *207*(3), 195-202. https://doi.org/10.1016/j.psychres.2013.01.004

Flechsenhar, A., Seitz, K. I., Bertsch, K., & Herpertz, S. C. (2024). The association between psychopathology, childhood trauma, and emotion processing. *Psychol Trauma*, *16*(Suppl 1), S190-s203. https://doi.org/10.1037/tra0001261

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy*, *20*(2), 155-176. https://doi.org/10.1016/S0005-7894(89)80067-X

Fonagy, P., & Bateman, A. (2008). The Development of Borderline Personality Disorder—A Mentalizing Model. *Journal of Personality Disorders*, *22*(1), 4-21. https://doi.org/10.1521/pedi.2008.22.1.4

Ford, J. D., & Courtois, C. A. (2021). Complex PTSD and borderline personality disorder. *Borderline Personality Disorder and Emotion Dysregulation*, *8*(1), 16. https://doi.org/10.1186/s40479-021-00155-9

Freier, A., Kruse, J., Schmalbach, B., Zara, S., Werner, S., Brähler, E.,…Kampling, H. (2022). The mediation effect of personality functioning between different types of child maltreatment and the development of depression/anxiety symptoms – A German representative study. *Journal of Affective Disorders*, *299*, 408-415. https://doi.org/10.1016/j.jad.2021.12.020

Frías, Á., & Palma, C. (2014). Comorbidity between Post-Traumatic Stress Disorder and Borderline Personality Disorder: A Review. *Psychopathology*, *48*(1), 1-10. https://doi.org/10.1159/000363145

Frost, R., Murphy, J., Hyland, P., Shevlin, M., Ben-Ezra, M., Hansen, M.,…McDonagh, T. (2020). Revealing what is distinct by recognising what is common: distinguishing between complex PTSD and Borderline Personality Disorder symptoms using bifactor modelling. *Eur J Psychotraumatol*, *11*(1), 1836864. https://doi.org/10.1080/20008198.2020.1836864

Gehrman, P., Seelig, A. D., Jacobson, I. G., Boyko, E. J., Hooper, T. I., Gackstetter, G. D.,…Smith, T. C. (2013). Predeployment Sleep Duration and Insomnia Symptoms as

Risk Factors for New-Onset Mental Health Disorders Following Military Deployment. *Sleep*, *36*(7), 1009-1018. https://doi.org/10.5665/sleep.2798

Gieselmann, A., Ait Aoudia, M., Carr, M., Germain, A., Gorzka, R., Holzinger, B.,…Pietrowsky, R. (2019). Aetiology and treatment of nightmare disorder: State of the art and future perspectives. *Journal of Sleep Research*, *28*(4), e12820-e12820. https://doi.org/10.1111/jsr.12820

Glaser, J. P., van Os, J., Portegijs, P. J., & Myin-Germeys, I. (2006). Childhood trauma and emotional reactivity to daily life stress in adult frequent attenders of general practitioners. *J Psychosom Res*, *61*(2), 229-236. https://doi.org/10.1016/j.jpsychores.2006.04.014

Goerigk, S., Elsaesser, M., Reinhard, M. A., Kriston, L., Härter, M., Hautzinger, M.,…Padberg, F. (2024). Childhood Trauma Questionnaire-based child maltreatment profiles to predict efficacy of the Cognitive Behavioral Analysis System of Psychotherapy versus non-specific psychotherapy in adults with early-onset chronic depression: cluster analysis of data from a randomised controlled trial. *Lancet Psychiatry*, *11*(9), 709-719. https://doi.org/10.1016/s2215-0366(24)00209-8

Goerigk, S., Reinhard, M. A., Barton, B. B., Burkhardt, G., Ehring, T., Bertsch, K.,…Padberg, F. (2023). Transdiagnostic analysis of adverse childhood experiences and their association with psychopathology-A TRANS-D conform study. *Psychiatry Research*, *329*, 115545. https://doi.org/10.1016/j.psychres.2023.115545

Goldstein, A. N., & Walker, M. P. (2014). The role of sleep in emotional brain function. *Annual Review of Clinical Psychology*, *10*, 679-708. https://doi.org/10.1146/annurev-clinpsy-032813-153716

Gordon, A. M., & Chen, S. (2014). The role of sleep in interpersonal conflict: Do sleepless nights mean worse fights? *Social Psychological and Personality Science*, *5*(2), 168-175. https://doi.org/10.1177/1948550613488952

Gratz, K. L., Dixon-Gordon, K. L., Breetz, A., & Tull, M. (2013). A Laboratory-Based Examination of Responses to Social Rejection in Borderline Personality Disorder: The Mediating Role of Emotion Dysregulation. *Journal of Personality Disorders*, *27*(2), 157-171. https://doi.org/10.1521/pedi.2013.27.2.157

Gratz, K. L., & Roemer, L. (2004). Multidimensional Assessment of Emotion Regulation and Dysregulation: Development, Factor Structure, and Initial Validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment*, *26*(1), 41-54. https://doi.org/10.1023/B:JOBA.0000007455.08539.94

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J Pers Soc Psychol*, *74*(1), 224-237. https://doi.org/10.1037//0022-3514.74.1.224

Gross, J. J. (1998b). The emerging field of emotion regulation: An integrative review. *Review of General Psychology*, *2*(3), 271-299. https://doi.org/10.1037/1089-2680.2.3.271

Gross, J. J. (2015). Emotion regulation: Current status and future prospects. *Psychological Inquiry*, *26*(1), 1-26. https://doi.org/10.1080/1047840X.2014.940781

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *J Pers Soc Psychol*, *85*(2), 348-362. https://doi.org/10.1037/0022-3514.85.2.348

Grove, J. L., Smith, T. W., Crowell, S. E., & Ellis, J. H. (2016). Preliminary Evidence for Emotion Dysregulation as a Mechanism Underlying Poor Sleep Quality in Borderline Personality Disorder. *Journal of Personality Disorders*, *31*(4), 542-552. https://doi.org/10.1521/pedi_2016_30_261

Guay, S., Billette, V., & Marchand, A. (2006). Exploring the links between posttraumatic stress disorder and social support: Processes and potential research avenues. *Journal of Traumatic Stress*, *19*(3), 327-338. https://doi.org/10.1002/jts.20124

Gunderson, J. G., Herpertz, S. C., Skodol, A. E., Torgersen, S., & Zanarini, M. C. (2018). Borderline personality disorder. *Nat Rev Dis Primers*, *4*, 18029. https://doi.org/10.1038/nrdp.2018.29

Gupta, M. A., & Sheridan, A. D. (2018). Fear of Sleep May Be a Core Symptom of Sympathetic Activation and the Drive for Vigilance in Posttraumatic Stress Disorder. *J Clin Sleep Med*, *14*(12), 2093. https://doi.org/10.5664/jcsm.7550

Hafizi, S. (2013). Sleep and borderline personality disorder: a review. *Asian J Psychiatr*, *6*(6), 452-459. https://doi.org/10.1016/j.ajp.2013.06.016

Hailes, H. P., Yu, R., Danese, A., & Fazel, S. (2019). Long-term outcomes of childhood sexual abuse: an umbrella review. *Lancet Psychiatry*, *6*(10), 830-839. https://doi.org/10.1016/s2215-0366(19)30286-x

Harb, F., González-Van Wart, A., Brzezinski, J., deRoon-Cassini, T. A., & Larson, C. L. (2025). Subtypes of childhood maltreatment and posttraumatic stress disorder symptoms in an adult trauma sample: The mechanistic role of sleep. *Psychol Trauma*, *17*(2), 353-362. https://doi.org/10.1037/tra0001716

Harned, M. S., Korslund, K. E., Foa, E. B., & Linehan, M. M. (2012). Treating PTSD in suicidal and self-injuring women with borderline personality disorder: development and preliminary evaluation of a Dialectical Behavior Therapy Prolonged Exposure Protocol. *Behav Res Ther*, *50*(6), 381-386. https://doi.org/10.1016/j.brat.2012.02.011

Harty, L., D., F. R., Aaron, T., Jeffrey, S., & and Tangney, J. P. (2010). Are inmates' subjective sleep problems associated with borderline personality, psychopathy, and antisocial personality independent of depression and substance dependence? *The Journal of*

Forensic Psychiatry & Psychology, *21*(1), 23-38. https://doi.org/10.1080/14789940903194095

Harvey, A., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive Behavioural Processes across Psychological Disorders: A transdiagnostic approach to research and treatment*. Oxford University Press. https://doi.org/10.1093/med:psych/9780198528883.001.0001

Harvey, A. G., Jones, C., & Schmidt, D. A. (2003). Sleep and posttraumatic stress disorder: a review. *Clinical Psychology Review*, *23*(3), 377-407. https://doi.org/10.1016/s0272-7358(03)00032-1

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. *Clin Psychol Rev*, *31*(2), 225-235. https://doi.org/10.1016/j.cpr.2010.04.003

Haslam, N., Holland, E., & Kuppens, P. (2012). Categories versus dimensions in personality and psychopathology: a quantitative review of taxometric research. *Psychol Med*, *42*(5), 903-920. https://doi.org/10.1017/s0033291711001966

Haslam, N., McGrath, M. J., Viechtbauer, W., & Kuppens, P. (2020). Dimensions over categories: a meta-analysis of taxometric research. *Psychological Medicine*, *50*(9), 1418-1432. https://doi.org/10.1017/S003329172000183X

Haslam, Z., & Taylor, E. P. (2022). The relationship between child neglect and adolescent interpersonal functioning: A systematic review. *Child Abuse & Neglect*, *125*, 105510. https://doi.org/10.1016/j.chiabu.2022.105510

Hatkevich, C., Sumlin, E., & Sharp, C. (2021). Examining Associations Between Child Abuse and Neglect Experiences With Emotion Regulation Difficulties Indicative of Adolescent Suicidal Ideation Risk [Original Research]. *Frontiers in Psychiatry*, *Volume 12 - 2021*. https://doi.org/10.3389/fpsyt.2021.630697

Hébert, M., Tremblay-Perreault, A., & Myre, G. (2021). The Interplay of Depression and Hostile Attributions in the Link Between PTSD Symptoms and Peer Victimization in Child Victims of Sexual Abuse. *Child Psychiatry & Human Development*, *52*(2), 291-300. https://doi.org/10.1007/s10578-020-01011-7

Heffernan, K., & Cloitre, M. (2000). A comparison of posttraumatic stress disorder with and without borderline personality disorder among women with a history of childhood sexual abuse: etiological and clinical characteristics. *J Nerv Ment Dis*, *188*(9), 589-595. https://doi.org/10.1097/00005053-200009000-00005

Hein, T. C., & Monk, C. S. (2017). Research Review: Neural response to threat in children, adolescents, and adults after child maltreatment - a quantitative meta-analysis. *J Child Psychol Psychiatry*, *58*(3), 222-230. https://doi.org/10.1111/jcpp.12651

Heleniak, C., Jenness, J. L., Vander Stoep, A., McCauley, E., & McLaughlin, K. A. (2016). Childhood Maltreatment Exposure and Disruptions in Emotion Regulation: A Transdiagnostic Pathway to Adolescent Internalizing and Externalizing Psychopathology. *Cognitive Therapy and Research*, *40*(3), 394-415. https://doi.org/10.1007/s10608-015-9735-z

Hensler, I., Sveen, J., Cernvall, M., & Arnberg, F. K. (2021). Ecological momentary assessment of self-rated health, daily strategies and self-management app use among trauma-exposed adults. *European Journal of Psychotraumatology*, *12*(1), 1920204. https://doi.org/10.1080/20008198.2021.1920204

Herringa, R. J. (2017). Trauma, PTSD, and the Developing Brain. *Current Psychiatry Reports*, *19*(10), 69. https://doi.org/10.1007/s11920-017-0825-3

Hjelseng, I. V., Vaskinn, A., Ueland, T., Lunding, S. H., Reponen, E. J., Steen, N. E.,…Aas, M. (2022). Childhood trauma is associated with poorer social functioning in severe

mental disorders both during an active illness phase and in remission. *Schizophrenia Research*, *243*, 241-246. https://doi.org/10.1016/j.schres.2020.03.015

Hofmann, S. G., & Hayes, S. C. (2019). The Future of Intervention Science: Process-Based Therapy. *Clinical Psychological Science*, *7*(1), 37-50. https://doi.org/10.1177/2167702618772296

Holmes, J. (2004). Disorganized attachment and Borderline Personality Disorder: A clinical perspective. *ATTACHMENT & HUMAN DEVELOPMENT*, *6*(2), 181-190. https://doi.org/10.1080/14616730410001688202

Hood, C. O., Southward, M. W., Badour, C. L., & Sauer-Zavala, S. (2024). BPD Compass: Using a dimensional model of psychopathology to treat co-occurring borderline personality disorder and posttraumatic stress symptoms. *Journal of Traumatic Stress*, *37*(3), 422-432. https://doi.org/10.1002/jts.23024

Huang, Y. H., Liu, C., Zhang, J. B., Li, S. B., Wang, L. L., Hu, H. X.,…Chan, R. C. K. (2025). A Transdiagnostic Network Analysis of Childhood Trauma and Psychopathology. *Schizophr Bull*, *51*(3), 792-803. https://doi.org/10.1093/schbul/sbae137

Huang, Yu L., Fonagy, P., Feigenbaum, J., Montague, P. R., Nolte, T., & and London Personality and Mood Disorder Research Consortium. (2020). Multidirectional Pathways between Attachment, Mentalizing, and Posttraumatic Stress Symptomatology in the Context of Childhood Trauma. *Psychopathology*, *53*(1), 48-58. https://doi.org/10.1159/000506406

Hyland, P., Karatzias, T., Shevlin, M., & Cloitre, M. (2019). Examining the Discriminant Validity of Complex Posttraumatic Stress Disorder and Borderline Personality Disorder Symptoms: Results From a United Kingdom Population Sample. *J Trauma Stress*, *32*(6), 855-863. https://doi.org/10.1002/jts.22444

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K.,…Wang, P. (2010). Research Domain Criteria (RDoC): Toward a New Classification Framework for Research on Mental Disorders. *American Journal of Psychiatry*, *167*(7), 748-751. https://doi.org/10.1176/appi.ajp.2010.09091379

Jaffee, S. R. (2017). Child Maltreatment and Risk for Psychopathology in Childhood and Adulthood. *Annual Review of Clinical Psychology*, *13*(Volume 13, 2017), 525-551. https://doi.org/10.1146/annurev-clinpsy-032816-045005

Javakhishvili, M., & Widom, C. S. (2021). Childhood maltreatment, sleep disturbances, and anxiety and depression: A prospective longitudinal investigation. *Journal of Applied Developmental Psychology*, *77*. https://doi.org/10.1016/j.appdev.2021.101351

Javanbakht, A., Liberzon, I., Amirsadri, A., Gjini, K., & Boutros, N. N. (2011). Event-related potential studies of post-traumatic stress disorder: a critical review and synthesis. *Biol Mood Anxiety Disord*, *1*(1), 5. https://doi.org/10.1186/2045-5380-1-5

Jenkins, C. A., Thompson, K. N., Nicholas, C. L., Hartmann, J. A., & Chanen, A. M. (2022). Potential mechanisms underlying sleep disturbance in young people with borderline personality disorder features: an exploratory study. *Borderline Personality Disorder and Emotion Dysregulation*, *9*(1), 10. https://doi.org/10.1186/s40479-022-00180-2

Johansen, V. A., Milde, A. M., Nilsen, R. M., Breivik, K., Nordanger, D. Ø., Stormark, K. M., & Weisæth, L. (2022). The Relationship Between Perceived Social Support and PTSD Symptoms After Exposure to Physical Assault: An 8 Years Longitudinal Study. *Journal of Interpersonal Violence*, *37*(9-10), NP7679-NP7706. https://doi.org/10.1177/0886260520970314

Johnson-Motoyama, M., & Davis, S. (2020). Child Maltreatment Prevention. *Social Work*. https://doi.org/10.1093/obo/9780195389678-0282

Jowett, S., Karatzias, T., & Albert, I. (2020). Multiple and interpersonal trauma are risk factors for both post-traumatic stress disorder and borderline personality disorder: A systematic review on the traumatic backgrounds and clinical characteristics of comorbid post-traumatic stress disorder/borderline personality disorder groups versus single-disorder groups. *Psychol Psychother*, *93*(3), 621-638. https://doi.org/10.1111/papt.12248

Kajeepeta, S., Gelaye, B., Jackson, C. L., & Williams, M. A. (2015). Adverse childhood experiences are associated with adult sleep disorders: a systematic review. *Sleep Med*, *16*(3), 320-330. https://doi.org/10.1016/j.sleep.2014.12.013

Kampling, H., Kruse, J., Lampe, A., Nolte, T., Hettich, N., Brähler, E.,…Riedl, D. (2022). Epistemic trust and personality functioning mediate the association between adverse childhood experiences and posttraumatic stress disorder and complex posttraumatic stress disorder in adulthood. *Front Psychiatry*, *13*, 919191. https://doi.org/10.3389/fpsyt.2022.919191

Karatzoglou, V., Carollo, A., Karagiannopoulou, E., Esposito, G., Séaghdha, X. T. Ó., & Dimitriou, D. (2024). A scientometric review of the association between childhood trauma and sleep. *Acta Psychologica*, *250*, 104488. https://doi.org/10.1016/j.actpsy.2024.104488

Keepers, G. A., Fochtmann, L. J., Anzia, J. M., Benjamin, S., Lyness, J. M., Mojtabai, R.,…Medicus, J. (2024). The American Psychiatric Association Practice Guideline for the Treatment of Patients With Borderline Personality Disorder. *Am J Psychiatry*, *181*(11), 1024-1028. https://doi.org/10.1176/appi.ajp.24181010

Kerber, A., Gewehr, E., Zimmermann, J., Sachser, C., J, M. F., Knaevelsrud, C., & Spitzer, C. (2023). Adverse childhood experiences and personality functioning interact substantially in predicting depression, anxiety, and somatization. *Personal Ment Health*, *17*(3), 246-258. https://doi.org/10.1002/pmh.1578

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, *62*(6), 593-602. https://doi.org/10.1001/archpsyc.62.6.593

Kim, J., & Cicchetti, D. (2010). Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology. *Journal of Child Psychology and Psychiatry*, *51*(6), 706-716. https://doi.org/10.1111/j.1469-7610.2009.02202.x

Klinger-König, J., Erhardt, A., Streit, F., Völker, M. P., Schulze, M. B., Keil, T.,…Grabe, H. J. (2024). Childhood Trauma and Somatic and Mental Illness in Adulthood. *Dtsch Arztebl Int*, *121*(1), 1-8. https://doi.org/10.3238/arztebl.m2023.0225

Knefel, M., Tran, U. S., & Lueger-Schuster, B. (2016). The association of posttraumatic stress disorder, complex posttraumatic stress disorder, and borderline personality disorder from a network analytical perspective. *J Anxiety Disord*, *43*, 70-78. https://doi.org/10.1016/j.janxdis.2016.09.002

Kockler, T. D., Tschacher, W., Santangelo, P. S., Limberger, M. F., & Ebner-Priemer, U. W. (2017). Specificity of emotion sequences in borderline personality disorder compared to posttraumatic stress disorder, bulimia nervosa, and healthy controls: an e-diary study. *Borderline Personal Disord Emot Dysregul*, *4*, 26. https://doi.org/10.1186/s40479-017-0077-1

Koenigsberg, H. W., Fan, J., Ochsner, K. N., Liu, X., Guise, K. G., Pizzarello, S.,…Siever, L. J. (2009). Neural correlates of the use of psychological distancing to regulate responses to negative social cues: a study of patients with borderline personality disorder. *Biol Psychiatry*, *66*(9), 854-863. https://doi.org/10.1016/j.biopsych.2009.06.010

Kolb, B., & Gibb, R. (2014). Searching for the principles of brain plasticity and behavior. *Cortex*, *58*, 251-260. https://doi.org/10.1016/j.cortex.2013.11.012

Kotov, R., Krueger, R. F., & Watson, D. (2018). A paradigm shift in psychiatric classification: the hierarchical taxonomy of psychopathology (HiTOP). *World Psychiatry*, *17*(1), 24-25. https://doi.org/10.1002/wps.20478

Krakau, L., Tibubos, A. N., Beutel, M. E., Ehrenthal, J. C., Gieler, U., & Brähler, E. (2021). Personality functioning as a mediator of adult mental health following child maltreatment. *Journal of Affective Disorders*, *291*, 126-134. https://doi.org/10.1016/j.jad.2021.05.006

Krause-Utz, A. (2021). Stimulating research on childhood adversities, borderline personality disorder, and complex post-traumatic stress disorder. *Borderline Personality Disorder and Emotion Dysregulation*, *8*(1), 11. https://doi.org/10.1186/s40479-021-00152-y

Krause-Utz, A., Walther, J.-C., Lis, S., Schmahl, C., & Bohus, M. (2019). Heart rate variability during a cognitive reappraisal task in female patients with borderline personality disorder: the role of comorbid posttraumatic stress disorder and dissociation. *Psychological Medicine*, *49*(11), 1810-1821. https://doi.org/10.1017/S0033291718002489

Kühner, C., de Waal, M., Steenkamp, L., van Zwol, I., Goudriaan, A., & Thomaes, K. (2025). The impact of types of childhood maltreatment on the severity of PTSD and comorbid personality disorder symptoms. *European Journal of Psychotraumatology*, *16*(1), 2491172. https://doi.org/10.1080/20008066.2025.2491172

Kuo, J. R., Khoury, J. E., Metcalfe, R., Fitzpatrick, S., & Goodwill, A. (2015). An examination of the relationship between childhood emotional abuse and borderline personality disorder features: The role of difficulties with emotion regulation. *Child Abuse & Neglect*, *39*, 147-155. https://doi.org/10.1016/j.chiabu.2014.08.008

Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion modulation in PTSD: Clinical and neurobiological

evidence for a dissociative subtype. *Am J Psychiatry*, *167*(6), 640-647. https://doi.org/10.1176/appi.ajp.2009.09081168

Laskemoen, J. F., Aas, M., Vaskinn, A., Berg, A. O., Lunding, S. H., Barrett, E. A.,…Simonsen, C. (2021). Sleep disturbance mediates the link between childhood trauma and clinical outcome in severe mental disorders. *Psychol Med*, *51*(14), 2337-2346. https://doi.org/10.1017/s0033291720000914

Lazarus, S. A., Cheavens, J. S., Festa, F., & Zachary Rosenthal, M. (2014). Interpersonal functioning in borderline personality disorder: A systematic review of behavioral and laboratory-based assessments. *Clinical Psychology Review*, *34*(3), 193-205. https://doi.org/10.1016/j.cpr.2014.01.007

Lee, D. J., Witte, T. K., Weathers, F. W., & Davis, M. T. (2015). Emotion Regulation Strategy Use and Posttraumatic Stress Disorder: Associations Between Multiple Strategies and Specific Symptom Clusters. *Journal of Psychopathology and Behavioral Assessment*, *37*(3), 533-544. https://doi.org/10.1007/s10862-014-9477-3

Leskin, G. A., Woodward, S. H., Young, H. E., & Sheikh, J. I. (2002). Effects of comorbid diagnoses on sleep disturbance in PTSD. *Journal of Psychiatric Research*. https://doi.org/10.1016/S0022-3956(02)00025-0

Levin, R., & Nielsen, T. (2009). Nightmares, bad dreams, and emotion dysregulation: A review and new neurocognitive model of dreaming. *Current Directions in Psychological Science*, *18*(2), 84-88. https://doi.org/10.1111/j.1467-8721.2009.01614.x

Lewis, S. J., Koenen, K. C., Ambler, A., Arseneault, L., Caspi, A., Fisher, H. L.,…Danese, A. (2021). Unravelling the contribution of complex trauma to psychopathology and cognitive deficits: a cohort study. *The British Journal of Psychiatry*, *219*(2), 448-455. https://doi.org/10.1192/bjp.2021.57

Li, E. T., Luyten, P., & Midgley, N. (2020). Psychological Mediators of the Association Between Childhood Emotional Abuse and Depression: A Systematic Review [Systematic Review]. *Frontiers in Psychiatry*, *Volume 11 - 2020*. https://doi.org/10.3389/fpsyt.2020.559213

Limberg, A., Barnow, S., Freyberger, H. J., & Hamm, A. O. (2011). Emotional vulnerability in borderline personality disorder is cue specific and modulated by traumatization. *Biol Psychiatry*, *69*(6), 574-582. https://doi.org/10.1016/j.biopsych.2010.10.024

Lind, M. J., Aggen, S. H., Kendler, K. S., York, T. P., & Amstadter, A. B. (2016). An epidemiologic study of childhood sexual abuse and adult sleep disturbances. *Psychol Trauma*, *8*(2), 198-205. https://doi.org/10.1037/tra0000080

Linehan, M. (2014). *DBT? Skills training manual*. Guilford Publications.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. Guilford Press.

Links, P. S., Heslegrave, R., & van Reekum, R. (1999). Impulsivity: core aspect of borderline personality disorder. *J Pers Disord*, *13*(1), 1-9. https://doi.org/10.1521/pedi.1999.13.1.1

Litz, B. T., Litz, B. T., & Gray, M. J. (2002). Emotional Numbing in Posttraumatic Stress Disorder: Current and Future Research Directions. *Australian & New Zealand Journal of Psychiatry*, *36*(2), 198-204. https://doi.org/10.1046/j.1440-1614.2002.01002.x

Liu, A., Liu, M., Ren, Y., Zhang, L. M., & Peng, Y. (2024). Dynamic networks of complex posttraumatic stress disorder and depression among college students with childhood trauma: insights from cross-sectional and cross-lagged panel network analysis. *Eur J Psychotraumatol*, *15*(1), 2391656. https://doi.org/10.1080/20008066.2024.2391656

Liu, J., Teh, W. L., Tan, R. H. S., Tan, Y. B., Tang, C., Chandwani, N., & Subramaniam, M. (2023). Sleep disturbance as transdiagnostic mediator between adverse childhood

experiences and psychopathology in children and adolescents: A structural equation modeling meta-analysis. *JCPP Adv*, *3*(3), e12156. https://doi.org/10.1002/jcv2.12156

Liu, N., Cao, Y., Qiao, H., Ma, H., Li, J., Luo, X.,…Zhang, N. (2021). Traumatic experiences and PTSD symptoms in the Chinese male intrafamilial physical violence perpetrators: A comparative and structural equation modeling study. *Journal of Interpersonal Violence*, *36*(5-6), 2841-2861. https://doi.org/10.1177/0886260518764103

Liu, N., Zhang, Y. L., Brady, H. J., Cao, Y. P., & He, Y. (2012). Relation Between Childhood Maltreatment and Severe Intrafamilial Male-Perpetrated Physical Violence in Chinese Community: The Mediating Role of Borderline and Antisocial Personality Disorder Features. *Aggressive Behavior*, *38*(1), 64-76. https://doi.org/10.1002/ab.20417

Liu, S. Y., Li, J., Leon, L. F., Schwarzer, R., & Cone, J. E. (2022). The Bidirectional Relationship between Posttraumatic Stress Symptoms and Social Support in a 9/11-Exposed Cohort: A Longitudinal Cross-Lagged Analysis. *Int J Environ Res Public Health*, *19*(5). https://doi.org/10.3390/ijerph19052604

Lobbestael, J., & Arntz, A. (2015). Emotional hyperreactivity in response to childhood abuse by primary caregivers in patients with borderline personality disorder. *J Behav Ther Exp Psychiatry*, *48*, 125-132. https://doi.org/10.1016/j.jbtep.2015.03.005

Lobbestael, J., & McNally, R. J. (2016). An Empirical Test of Rejection- and Anger-Related Interpretation Bias in Borderline Personality Disorder. *J Pers Disord*, *30*(3), 307-319. https://doi.org/10.1521/pedi_2015_29_194

Lock, J., Yang, M., & Tyrer, P. (2025). Establishing the relationship between history of childhood trauma and personality disorder using the ICD-11 classification system. *Personality and Mental Health*, *19*(1), e1648. https://doi.org/10.1002/pmh.1648

Lorbeer, N., Knaevelsrud, C., & Niemeyer, H. (2023). STAIR and STAIR/NT as a Treatment for Posttraumatic Stress: A Systematic Review. *Verhaltenstherapie*, *33*(2-3), 53-63. https://doi.org/10.1159/000526592

Lord, K. A., Suvak, M. K., Holmes, S., Shields, N., Lane, J. E. M., Sijercic, I.,…Monson, C. M. (2020). Bidirectional Relationships Between Posttraumatic Stress Disorder and Social Functioning During Cognitive Processing Therapy. *Behav Ther*, *51*(3), 447-460. https://doi.org/10.1016/j.beth.2019.08.002

Lortye, S. A., Will, J. P., Rameckers, S. A., Marquenie, L. A., Goudriaan, A. E., Arntz, A., & de Waal, M. M. (2024). Types of childhood maltreatment as predictors of posttraumatic stress disorder severity and complex posttraumatic stress disorder in patients with substance use disorders. *Eur J Psychotraumatol*, *15*(1), 2367179. https://doi.org/10.1080/20008066.2024.2367179

Machlin, L., Sheridan, M. A., Lurie, L. A., Kasparek, S. W., Kim, S. G., Peverill, M.,…McLaughlin, K. A. (2024). Alterations in fear learning as a mechanism linking childhood exposure to violence with PTSD symptoms: a longitudinal study. *Psychological Medicine*, *54*(12), 3389-3397. https://doi.org/10.1017/S0033291724001569

Maercker, A., Hecker, T., Augsburger, M., & Kliem, S. (2018). ICD-11 Prevalence Rates of Posttraumatic Stress Disorder and Complex Posttraumatic Stress Disorder in a German Nationwide Sample. *J Nerv Ment Dis*, *206*(4), 270-276. https://doi.org/10.1097/nmd.0000000000000790

Marissen, M. A., Meuleman, L., & Franken, I. H. (2010). Altered emotional information processing in borderline personality disorder: an electrophysiological study. *Psychiatry Res*, *181*(3), 226-232. https://doi.org/10.1016/j.pscychresns.2009.10.006

Martín-Blanco, A., Ferrer, M., Soler, J., Arranz, M. J., Vega, D., Calvo, N.,…Pascual, J. C. (2016). The role of hypothalamus-pituitary-adrenal genes and childhood trauma in borderline personality disorder. *Eur Arch Psychiatry Clin Neurosci*, *266*(4), 307-316. https://doi.org/10.1007/s00406-015-0612-2

Martinez, A. P., Milne, E., Rowse, G., & Bentall, R. P. (2025). Manipulating self and other schemas to explore psychological processes associated with paranoid beliefs: an online experimental study [Original Research]. *Frontiers in Psychology*, *Volume 15 - 2024*. https://doi.org/10.3389/fpsyg.2024.1474562

Martínez-Priego, C., García-Noblejas, B. P., & Roca, P. (2024). Strategies and goals in Emotion Regulation models: a systematic review [Systematic Review]. *Frontiers in Psychology*, *Volume 15 - 2024*. https://doi.org/10.3389/fpsyg.2024.1425465

Marusak, H. A., Martin, K. R., Etkin, A., & Thomason, M. E. (2015). Childhood trauma exposure disrupts the automatic regulation of emotional processing. *Neuropsychopharmacology*, *40*(5), 1250-1258. https://doi.org/10.1038/npp.2014.311

Masten, C. L., Guyer, A. E., Hodgdon, H. B., McClure, E. B., Charney, D. S., Ernst, M.,…Monk, C. S. (2008). Recognition of facial emotions among maltreated children with high rates of post-traumatic stress disorder. *Child Abuse & Neglect*, *32*(1), 139-153. https://doi.org/10.1016/j.chiabu.2007.09.006

Mauss, I. B., Troy, A. S., & LeBourgeois, M. K. (2013). Poorer sleep quality is associated with lower emotion-regulation ability in a laboratory paradigm. *Cogn Emot*, *27*(3), 567-576. https://doi.org/10.1080/02699931.2012.727783

McBride, S., Goulden, N., Barnicot, K., Corrigan, K., Shen, S., Guillemard, S.,…Crawford, M. J. (2025). Mental Health and Personality Functioning of People With Probable Personality Disorder Who Have Coexisting Complex Post Traumatic Stress Disorder. *Personal Ment Health*, *19*(1), e70010. https://doi.org/10.1002/pmh.70010

McCann, J. P., Tipsword, J. M., Brake, C. A., & Badour, C. L. (2023). Trauma-Related Shame and Guilt as Prospective Predictors of Daily Mental Contamination and PTSD Symptoms in Survivors of Sexual Trauma. *Journal of Interpersonal Violence*, *38*(19-20), 11117-11137. https://doi.org/10.1177/08862605231179721

McCrory, E., De Brito, S. A., & Viding, E. (2011). The impact of childhood maltreatment: a review of neurobiological and genetic factors. *Front Psychiatry*, *2*, 48. https://doi.org/10.3389/fpsyt.2011.00048

McLaughlin, K. A., Colich, N. L., Rodman, A. M., & Weissman, D. G. (2020). Mechanisms linking childhood trauma exposure and psychopathology: A transdiagnostic model of risk and resilience. *BMC Medicine*, *18*(1), 96. https://doi.org/10.1186/s12916-020-01561-6

McLaughlin, K. A., Koenen, K. C., Bromet, E. J., Karam, E. G., Liu, H., Petukhova, M.,…Kessler, R. C. (2017). Childhood adversities and post-traumatic stress disorder: evidence for stress sensitisation in the World Mental Health Surveys. *Br J Psychiatry*, *211*(5), 280-288. https://doi.org/10.1192/bjp.bp.116.197640

McLaughlin, K. A., & Lambert, H. K. (2017). Child trauma exposure and psychopathology: mechanisms of risk and resilience. *Current Opinion in Psychology*, *14*, 29-34. https://doi.org/10.1016/j.copsyc.2016.10.004

McLaughlin, K. A., Peverill, M., Gold, A. L., Alves, S., & Sheridan, M. A. (2015). Child Maltreatment and Neural Systems Underlying Emotion Regulation. *J Am Acad Child Adolesc Psychiatry*, *54*(9), 753-762. https://doi.org/10.1016/j.jaac.2015.06.010

McNeil, J., & Rehman, U. S. (2024). The Role of Emotional Processes in Explaining the Link Between Severity of Childhood Maltreatment and Relationship Difficulties. *Journal of Interpersonal Violence*, *39*(9-10), 2165-2188. https://doi.org/10.1177/08862605231216695

McRae, K. (2013). Emotion Regulation Frequency and Success: Separating Constructs from Methods and Time Scale. *Social and Personality Psychology Compass*, *7*(5), 289-302. https://doi.org/10.1111/spc3.12027

Messman, B. A., Fentem, A., Compton, S. E., Griffith, E. L., Blumenthal, H., Contractor, A. A., & Slavish, D. C. (2023). The role of affect in associations between sleep disturbances and posttraumatic stress disorder symptoms: A systematic review. *Sleep Medicine*, *110*, 287-296. https://doi.org/10.1016/j.sleep.2023.08.025

Messman-Moore, T. L., & Bhuptani, P. H. (2017). A review of the long-term impact of child maltreatment on posttraumatic stress disorder and its comorbidities: An emotion dysregulation perspective. *Clinical Psychology: Science and Practice*, *24*(2), 154-169. https://doi.org/10.1111/cpsp.12193

Milojevich, H. M., Machlin, L., & Sheridan, M. A. (2020). Early adversity and children's emotion regulation: Differential roles of parent emotion regulation and adversity exposure. *Development and Psychopathology*, *32*(5), 1788-1798. https://doi.org/10.1017/S0954579420001273

Miu, A. C., Szentágotai-Tătar, A., Balázsi, R., Nechita, D., Bunea, I., & Pollak, S. D. (2022). Emotion regulation as mediator between childhood adversity and psychopathology: A meta-analysis. *Clinical Psychology Review*, *93*, 102141. https://doi.org/10.1016/j.cpr.2022.102141

Morales-Muñoz, I., Durdurak, B. B., Bilgin, A., Marwaha, S., & Winsper, C. (2021). Understanding the Relationship Between Sleep Problems in Early Childhood and Borderline Personality Disorder: A Narrative Review. *Nat Sci Sleep*, *13*, 2175-2202. https://doi.org/10.2147/nss.S311672

Nemeroff, C. B. (2004). Neurobiological consequences of childhood trauma. *J Clin Psychiatry*, *65 Suppl 1*, 18-28.

Nielsen, T., & Levin, R. (2007). Nightmares: A new neurocognitive model. *Sleep Medicine Reviews*, *11*(4), 295-310. https://doi.org/10.1016/j.smrv.2007.03.004

Nietlisbach, G., Maercker, A., Rösler, W., & Haker, H. (2010). Are Empathic Abilities Impaired in Posttraumatic Stress Disorder? *Psychological Reports*, *106*(3), 832-844. https://doi.org/10.2466/pr0.106.3.832-844

NIH. (2005). National Institutes of Health State of the Science Conference statement on Manifestations and Management of Chronic Insomnia in Adults, June 13-15, 2005. *Sleep*, *28*(9), 1049-1057. https://doi.org/10.1093/sleep/28.9.1049

Nolen-Hoeksema, S., & Watkins, E. R. (2011). A Heuristic for Developing Transdiagnostic Models of Psychopathology: Explaining Multifinality and Divergent Trajectories. *Perspect Psychol Sci*, *6*(6), 589-609. https://doi.org/10.1177/1745691611419672

O'Brien, H., Kalokerinos, E. K., Felmingham, K., Lau, W., & O'Donnell, M. (2023). Emotion regulation strategy use in PTSD: A daily life study. *Journal of Affective Disorders*, *338*, 365-372. https://doi.org/10.1016/j.jad.2023.06.023

Owczarek, M., Karatzias, T., McElroy, E., Hyland, P., Cloitre, M., Kratzer, L.,…Shevlin, M. (2023). Borderline Personality Disorder (BPD) and Complex Posttraumatic Stress Disorder (CPTSD): A Network Analysis in a Highly Traumatized Clinical Sample. *J Pers Disord*, *37*(1), 112-129. https://doi.org/10.1521/pedi.2023.37.1.112

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychol Bull*, *129*(1), 52-73. https://doi.org/10.1037/0033-2909.129.1.52

Pace-Schott, E. F., Germain, A., & Milad, M. R. (2015). Sleep and REM sleep disturbance in the pathophysiology of PTSD: The role of extinction memory. *Biology of Mood and Anxiety Disorders*, *5*, 3-3. https://doi.org/10.1186/s13587-015-0018-9

Pagura, J., Stein, M. B., Bolton, J. M., Cox, B. J., Grant, B., & Sareen, J. (2010). Comorbidity of borderline personality disorder and posttraumatic stress disorder in the U.S. population. *J Psychiatr Res*, *44*(16), 1190-1198. https://doi.org/10.1016/j.jpsychires.2010.04.016

Palagini, L., Moretto, U., Dell'Osso, L., & Carney, C. (2017). Sleep-related cognitive processes, arousal, and emotion dysregulation in insomnia disorder: the role of insomnia-specific rumination. *Sleep Medicine*, *30*, 97-104. https://doi.org/10.1016/j.sleep.2016.11.004

Palmer, C. A., & Alfano, C. A. (2017). Sleep and emotion regulation: An organizing, integrative review. *Sleep Med Rev*, *31*, 6-16. https://doi.org/10.1016/j.smrv.2015.12.006

Peterson, C., Florence, C., & Klevens, J. (2018). The economic burden of child maltreatment in the United States, 2015. *Child Abuse & Neglect*, *86*, 178-183. https://doi.org/10.1016/j.chiabu.2018.09.018

Pfaltz, M. C., Halligan, S. L., Haim-Nachum, S., Sopp, M. R., Åhs, F., Bachem, R.,…Seedat, S. (2022). Social functioning in individuals affected by childhood maltreatment: Establishing a research agenda to inform interventions. *Psychotherapy and Psychosomatics*, *91*(4), 238-251. https://doi.org/10.1159/000523667

Popkirov, S., Flasbeck, V., Schlegel, U., Juckel, G., & Brüne, M. (2018). Alexithymia in borderline personality disorder is not associated with deficits in automatic visual processing of negative emotional stimuli. *Psychiatry Res*, *263*, 121-124. https://doi.org/10.1016/j.psychres.2018.03.009

Porter, C., Palmier-Claus, J., Branitsky, A., Mansell, W., Warwick, H., & Varese, F. (2020). Childhood adversity and borderline personality disorder: a meta-analysis. *Acta Psychiatrica Scandinavica*, *141*(1), 6-20. https://doi.org/10.1111/acps.13118

Powers, A., Petri, J. M., Sleep, C., Mekawi, Y., Lathan, E. C., Shebuski, K.,…Fani, N. (2022). Distinguishing PTSD, complex PTSD, and borderline personality disorder using exploratory structural equation modeling in a trauma-exposed urban sample. *J Anxiety Disord*, *88*, 102558. https://doi.org/10.1016/j.janxdis.2022.102558

Redican, E., Nolan, E., Hyland, P., Cloitre, M., McBride, O., Karatzias, T.,…Shevlin, M. (2021). A systematic literature review of factor analytic and mixture models of ICD-11 PTSD and CPTSD using the International Trauma Questionnaire. *Journal of Anxiety Disorders*, *79*, 102381. https://doi.org/10.1016/j.janxdis.2021.102381

Riemann, D., Spiegelhalder, K., Feige, B., Voderholzer, U., Berger, M., Perlis, M., & Nissen, C. (2010). The hyperarousal model of insomnia: A review of the concept and its evidence. *Sleep Medicine Reviews*, *14*(1), 19-31. https://doi.org/10.1016/j.smrv.2009.04.002

Río-Casanova, L. d., González, A., Páramo, M., Dijke, A. V., & Brenlla, J. (2016). Emotion regulation strategies in trauma-related disorders: pathways linking neurobiology and clinical manifestations. *Reviews in the Neurosciences*, *27*(4), 385-395. https://doi.org/doi:10.1515/revneuro-2015-0045

Rosenstein, L. K., Ellison, W. D., Walsh, E., Chelminski, I., Dalrymple, K., & Zimmerman, M. (2018). The role of emotion regulation difficulties in the connection between childhood emotional abuse and borderline personality features. *Personal Disord*, *9*(6), 590-594. https://doi.org/10.1037/per0000294

Rüsch, N., Corrigan, P. W., Bohus, M., Kühler, T., Jacob, G. A., & Lieb, K. (2007). The impact of posttraumatic stress disorder on dysfunctional implicit and explicit emotions among women with borderline personality disorder. *J Nerv Ment Dis*, *195*(6), 537-539. https://doi.org/10.1097/NMD.0b013e318064e7fc

Rüsch, N., Schulz, D., Valerius, G., Steil, R., Bohus, M., & Schmahl, C. (2011). Disgust and implicit self-concept in women with borderline personality disorder and posttraumatic stress disorder. *Eur Arch Psychiatry Clin Neurosci*, *261*(5), 369-376. https://doi.org/10.1007/s00406-010-0174-2

Russotti, J., Warmingham, J. M., Duprey, E. B., Handley, E. D., Manly, J. T., Rogosch, F. A., & Cicchetti, D. (2021). Child maltreatment and the development of psychopathology: The role of developmental timing and chronicity. *Child Abuse & Neglect*, *120*, 105215. https://doi.org/10.1016/j.chiabu.2021.105215

Sauer, C., Sheppes, G., Lackner, H. K., Arens, E. A., Tarrasch, R., & Barnow, S. (2016). Emotion regulation choice in female patients with borderline personality disorder: Findings from self-reports and experimental measures. *Psychiatry Res*, *242*, 375-384. https://doi.org/10.1016/j.psychres.2016.04.113

Sauer-Zavala, S., Gutner, C. A., Farchione, T. J., Boettcher, H. T., Bullis, J. R., & Barlow, D. H. (2017). Current Definitions of "Transdiagnostic" in Treatment Development: A Search for Consensus. *Behavior Therapy*, *48*(1), 128-138. https://doi.org/10.1016/j.beth.2016.09.004

Schäfer, I., Gast, U., Hofmann, A., Knaevelsrud, C., Lampe, A., Liebermann, P.,…Wöller, W. (2019). *S3-Leitlinie Posttraumatische Belastungsstörung*. https://doi.org/10.1007/978-3-662-59783-5

Schäfer, V., & Bader, K. (2013). Relationship Between Early-life Stress Load and Sleep in Psychiatric Outpatients: A Sleep Diary and Actigraphy Study. *Stress and Health*, *29*(3), 177-189. https://doi.org/10.1002/smi.2438

Scheiderer, E. M., Wood, P. K., & Trull, T. J. (2015). The comorbidity of borderline personality disorder and posttraumatic stress disorder: revisiting the prevalence and associations in

a general population sample. *Borderline Personality Disorder and Emotion Dysregulation*, *2*(1), 11. https://doi.org/10.1186/s40479-015-0032-y

Schredl, M., Paul, F., Reinhard, I., Ebner-Priemer, U. W., Schmahl, C., & Bohus, M. (2012). Sleep and dreaming in patients with borderline personality disorder: a polysomnographic study. *Psychiatry Res*, *200*(2-3), 430-436. https://doi.org/10.1016/j.psychres.2012.04.036

Schulze, A., Cloos, L., Zdravkovic, M., Lis, S., & Krause-Utz, A. (2022). On the interplay of borderline personality features, childhood trauma severity, attachment types, and social support. *Borderline Personal Disord Emot Dysregul*, *9*(1), 35. https://doi.org/10.1186/s40479-022-00206-9

Schulze, L., Schulze, A., Renneberg, B., Schmahl, C., & Niedtfeld, I. (2019). Neural Correlates of Affective Disturbances: A Comparative Meta-analysis of Negative Affect Processing in Borderline Personality Disorder, Major Depressive Disorder, and Posttraumatic Stress Disorder. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *4*(3), 220-232. https://doi.org/10.1016/j.bpsc.2018.11.004

Schumacher, S., Niemeyer, H., Engel, S., Cwik, J. C., Laufer, S., Klusmann, H., & Knaevelsrud, C. (2019). HPA axis regulation in posttraumatic stress disorder: A meta-analysis focusing on potential moderators. *Neuroscience & Biobehavioral Reviews*, *100*, 35-57. https://doi.org/10.1016/j.neubiorev.2019.02.005

Scoglio, A. A. J., Reilly, E. D., Girouard, C., Quigley, K. S., Carnes, S., & Kelly, M. M. (2022). Social Functioning in Individuals With Post-Traumatic Stress Disorder: A Systematic Review. *Trauma, Violence, & Abuse*, *23*(2), 356-371. https://doi.org/10.1177/1524838020946800

Selby, E., Ribeiro, J., & Jr, T. (2013). What Dreams May Come: Emotional Cascades and Nightmares in Borderline Personality Disorder. *Dreaming*, *23*, 126. https://doi.org/10.1037/a0032208

Selby, E. A. (2013). Chronic sleep disturbances and borderline personality disorder symptoms. *Journal of Consulting and Clinical Psychology*, *81*(5), 941-947. https://doi.org/10.1037/a0033201

Selby, E. A., Anestis, M. D., Bender, T. W., & Joiner, T. E., Jr. (2009). An exploration of the emotional cascade model in borderline personality disorder. *J Abnorm Psychol*, *118*(2), 375-387. https://doi.org/10.1037/a0015711

Seligowski, A. V., Lee, D. J., Bardeen, J. R., & Orcutt, H. K. (2015). Emotion regulation and posttraumatic stress symptoms: a meta-analysis. *Cogn Behav Ther*, *44*(2), 87-102. https://doi.org/10.1080/16506073.2014.980753

Semiz, U. B., Basoglu, C., Ebrinc, S., & Cetin, M. (2008). Nightmare disorder, dream anxiety, and subjective sleep quality in patients with borderline personality disorder. *Psychiatry Clin Neurosci*, *62*(1), 48-55. https://doi.org/10.1111/j.1440-1819.2007.01789.x

Semsar, N., Mousavi, Z., Tran, M.-L. M., & Kuhlman, K. R. (2021). Dysregulated arousal as a pathway linking childhood neglect and clinical sleep disturbances in adulthood. *Child Abuse & Neglect*, *122*, 105306. https://doi.org/10.1016/j.chiabu.2021.105306

Shahab, M. K., de Ridder, J. A., Spinhoven, P., Penninx, B. W. J. H., Mook-Kanamori, D. O., & Elzinga, B. M. (2021). A tangled start: The link between childhood maltreatment, psychopathology, and relationships in adulthood. *Child Abuse & Neglect*, *121*, 105228. https://doi.org/10.1016/j.chiabu.2021.105228

Short, N. A., Allan, N. P., Stentz, L., Portero, A. K., & Schmidt, N. B. (2018). Predictors of insomnia symptoms and nightmares among individuals with post-traumatic stress

disorder: an ecological momentary assessment study. *Journal of Sleep Research*, *27*(1), 64-72. https://doi.org/10.1111/jsr.12589

Sill, J., Popov, T., Schauer, M., & Elbert, T. (2020). Rapid brain responses to affective pictures indicate dimensions of trauma-related psychopathology in adolescents. *Psychophysiology*, *57*(1), e13353. https://doi.org/10.1111/psyp.13353

Sinha, S. S. (2016). Trauma-induced insomnia: A novel model for trauma and sleep research. *Sleep Medicine Reviews*, *25*, 74-83. https://doi.org/10.1016/j.smrv.2015.01.008

Smits, M. L., de Vos, J., Rüfenacht, E., Nijssens, L., Shaverin, L., Nolte, T.,…Bateman, A. (2024). Breaking the cycle with trauma-focused mentalization-based treatment: theory and practice of a trauma-focused group intervention. *Front Psychol*, *15*, 1426092. https://doi.org/10.3389/fpsyg.2024.1426092

Socci, V., Festucci, F., Barlattani, T., Salfi, F., D'Aurizio, G., Rossi, R.,…Tempesta, D. (2024). Sleep quality and emotional reactivity in patients with borderline personality disorder [Brief Research Report]. *Frontiers in Sleep*, *Volume 3 - 2024*. https://doi.org/10.3389/frsle.2024.1394979

Soloff, P. H. (2019). Bridging the Gap Between Remission and Recovery in BPD: Qualitative Versus Quantitative Perspectives. *Journal of Personality Disorders*, *35*(1), 21-40. https://doi.org/10.1521/pedi_2019_33_419

Sorgi-Wilson, K. M., & McCloskey, M. S. (2022). Emotion regulation strategies among individuals with borderline personality disorder relative to other groups: A review. *29*(5), 1655-1678. https://doi.org/10.1002/cpp.2738

Spilsbury, J. C. (2009). Sleep as a Mediator in the Pathway From Violence-Induced Traumatic Stress to Poorer Health and Functioning: A Review of the Literature and Proposed Conceptual Model. *Behavioral Sleep Medicine*, *7*(4), 223-244. https://doi.org/10.1080/15402000903190207

Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: Secondary symptom or core feature? *Sleep Medicine Reviews*, *12*(3), 169-184. https://doi.org/10.1016/j.smrv.2007.08.008

Steine, I. M., Skogen, J. C., Krystal, J. H., Winje, D., Milde, A. M., Grønli, J.,…Pallesen, S. (2019). Insomnia symptom trajectories among adult survivors of childhood sexual abuse: A longitudinal study. *Child Abuse & Neglect*, *93*, 263-276. https://doi.org/10.1016/j.chiabu.2019.05.009

Steuwe, C., Lanius, R. A., & Frewen, P. A. (2012). Evidence for a dissociative subtype of PTSD by latent profile and confirmatory factor analyses in a civilian sample. *Depress Anxiety*, *29*(8), 689-700. https://doi.org/10.1002/da.21944

Sullivan, K., Rochani, H., Huang, L.-T., Donley, D. K., & Zhang, J. (2019). Adverse childhood experiences affect sleep duration for up to 50 years later. *Sleep*, *42*(7). https://doi.org/10.1093/sleep/zsz087

Temes, C. M., Magni, L. R., Aguirre, B. A., Goodman, M., Ridolfi, M. E., & Zanarini, M. C. (2020). Parameters of reported childhood sexual abuse and assault in adolescents and adults with borderline personality disorder. *Personality and Mental Health*, *14*(3), 254-262. https://doi.org/10.1002/pmh.1475

Thompson, R. A. (1991). Emotional regulation and emotional development. *Educational Psychology Review*, *3*(4), 269-307. https://doi.org/10.1007/BF01319934

Tull, M. T., Vidaña, A. G., & Betts, J. E. (2020). Chapter 10 - Emotion regulation difficulties in PTSD. In M. T. Tull & N. A. Kimbrel (Eds.), *Emotion in Posttraumatic Stress Disorder* (pp. 295-310). Academic Press. https://doi.org/10.1016/B978-0-12-816022-0.00010-7

van Trigt, S., van der Zweerde, T., van Someren, E. J. W., van Straten, A., & van Marle, H. J. F. (2025). A theoretical perspective on the role of sleep in borderline personality

disorder: From causative factor to treatment target. *Sleep Medicine Reviews*, *81*, 102089. https://doi.org/10.1016/j.smrv.2025.102089

Vanek, J., Prasko, J., Ociskova, M., Hodny, F., Holubova, M., Minarikova, K.,…Nesnidal, V. (2021). Insomnia in Patients with Borderline Personality Disorder. *Nat Sci Sleep*, *13*, 239-250. https://doi.org/10.2147/nss.S295030

Vonderlin, R., Priebe, K., Müller-Engelmann, M., Fydrich, T., Steil, R., Resick, P. A.,…Bohus, M. (2024). Long-term effects of dialectical behaviour therapy for posttraumatic stress disorder and cognitive processing therapy 9 months after treatment termination. *European Journal of Psychotraumatology*, *15*(1), 2393061. https://doi.org/10.1080/20008066.2024.2393061

Walker, H. E., & Wamser-Nanney, R. (2023). Revictimization Risk Factors Following Childhood Maltreatment: A Literature Review. *Trauma, Violence, & Abuse*, *24*(4), 2319-2332. https://doi.org/10.1177/15248380221093692

Walsh, K., McLaughlin, K. A., Hamilton, A., & Keyes, K. M. (2017). Trauma exposure, incident psychiatric disorders, and disorder transitions in a longitudinal population representative sample. *J Psychiatr Res*, *92*, 212-218. https://doi.org/10.1016/j.jpsychires.2017.05.001

Wang, Q., Shelton, R. C., & Dwivedi, Y. (2018). Interaction between early-life stress and FKBP5 gene variants in major depressive disorder and post-traumatic stress disorder: A systematic review and meta-analysis. *J Affect Disord*, *225*, 422-428. https://doi.org/10.1016/j.jad.2017.08.066

Wang, W., & Wang, X. (2025). Sleep disturbance among Chinese survivors of childhood sexual abuse: associations with perceived discrimination and rumination [Original Research]. *Frontiers in Psychiatry*, *Volume 16 - 2025*. https://doi.org/10.3389/fpsyt.2025.1431839

Wang, Y., Chung, M. C., Wang, N., Yu, X., & Kenardy, J. (2021). Social support and posttraumatic stress disorder: A meta-analysis of longitudinal studies. *Clin Psychol Rev*, *85*, 101998. https://doi.org/10.1016/j.cpr.2021.101998

Warmingham, J. M., Duprey, E. B., Handley, E. D., Rogosch, F. A., & Cicchetti, D. (2023). Patterns of childhood maltreatment predict emotion processing and regulation in emerging adulthood. *Development and Psychopathology*, *35*(2), 766-781. https://doi.org/10.1017/S0954579422000025

Weissman, D. G., Bitran, D., Miller, A. B., Schaefer, J. D., Sheridan, M. A., & McLaughlin, K. A. (2019). Difficulties with emotion regulation as a transdiagnostic mechanism linking child maltreatment with the emergence of psychopathology. *Dev Psychopathol*, *31*(3), 899-915. https://doi.org/10.1017/s0954579419000348

Weissman, M. M. (1975). The assessment of social adjustment: A review of techniques. *Archives of General Psychiatry*, *32*(3), 357-365. https://doi.org/10.1001/archpsyc.1975.01760210091006

Weltz, S. M., Armeli, S., Ford, J. D., & Tennen, H. (2016). A daily process examination of the relationship between childhood trauma and stress-reactivity. *Child Abuse & Neglect*, *60*, 1-9. https://doi.org/10.1016/j.chiabu.2016.08.005

Werner, G. G., Danböck, S. K., Metodiev, S., & Kunze, A. E. (2020). Pre-Sleep Arousal and Fear of Sleep in Trauma-Related Sleep Disturbances: A Cluster-Analytic Approach. *Clinical Psychology in Europe*, *2*(2), 1-20. https://doi.org/10.32872/cpe.v2i2.2699

Werner, G. G., Riemann, D., & Ehring, T. (2021). Fear of sleep and trauma-induced insomnia: A review and conceptual model. *Sleep Medicine Reviews*, *55*, 101383. https://doi.org/10.1016/j.smrv.2020.101383

Widom, C. S. (1999). Posttraumatic stress disorder in abused and neglected children grown up. *Am J Psychiatry*, *156*(8), 1223-1229. https://doi.org/10.1176/ajp.156.8.1223

Winsper, C., Tang, N. K., Marwaha, S., Lereya, S. T., Gibbs, M., Thompson, A., & Singh, S. P. (2017). The sleep phenotype of Borderline Personality Disorder: A systematic review and meta-analysis. *Neurosci Biobehav Rev*, *73*, 48-67. https://doi.org/10.1016/j.neubiorev.2016.12.008

Witt, A., Brown, R. C., Plener, P. L., Brähler, E., & Fegert, J. M. (2017). Child maltreatment in Germany: prevalence rates in the general population. *Child Adolesc Psychiatry Ment Health*, *11*, 47. https://doi.org/10.1186/s13034-017-0185-0

Wolf, R., & Herringa, R. (2016). Prefrontal–Amygdala Dysregulation to Threat in Pediatric Posttraumatic Stress Disorder. *Neuropsychopharmacology*, *41*, 822-831. https://doi.org/10.1038/npp.2015.209

Wood, A., Brooks, R., & Beynon-Pindar, C. (2015). The experience of sleep for women with borderline personality disorder: An occupational perspective. *British Journal of Occupational Therapy*, *78*(12), 750-756. https://doi.org/10.1177/0308022615587864

Woodward, S. H., Shurick, A. A., Alvarez, J., Kuo, J., Nonyieva, Y., Blechert, J.,…Gross, J. J. (2015). A psychophysiological investigation of emotion regulation in chronic severe posttraumatic stress disorder. *Psychophysiology*, *52*(5), 667-678. https://doi.org/10.1111/psyp.12392

World Health Organization. (2019). International statistical classification of diseases and related health problems (11th ed.). https://icd.who.int/

Wu, J., Liu, Y., Zhang, L., Wang, N., Kohn, N., & Duan, H. (2023). Integrating the pattern of negative emotion processing and acute stress response with childhood stress among healthy young adults. *Stress*, *26*. https://doi.org/10.1080/10253890.2023.2195503

Xiao, Z., Murat Baldwin, M., Wong, S. C., Obsuth, I., Meinck, F., & Murray, A. L. (2023). The Impact of Childhood Psychological Maltreatment on Mental Health Outcomes in

Adulthood: A Systematic Review and Meta-Analysis. *Trauma, Violence, & Abuse*, *24*(5), 3049-3064. https://doi.org/10.1177/15248380221122816

Young, J. E., Klosko, J. S., & Weishaar, M. E. (2003). *Schema therapy: A practitioner's guide*. Guilford Press.

Zayfert, C., & DeViva, J. C. (2004). Residual insomnia following cognitive behavioral therapy for PTSD. *Journal of Traumatic Stress*, *17*(1), 69-73. https://doi.org/10.1023/B:JOTS.0000014679.31799.e7

Zeifman, R. J., Landy, M. S. H., Liebman, R. E., Fitzpatrick, S., & Monson, C. M. (2021). Optimizing treatment for comorbid borderline personality disorder and posttraumatic stress disorder: A systematic review of psychotherapeutic approaches and treatment efficacy. *Clinical Psychology Review*, *86*, 102030. https://doi.org/10.1016/j.cpr.2021.102030

Zhang, Y., Ren, R., Sanford, L. D., Yang, L., Zhou, J., Zhang, J.,…Tang, X. (2019). Sleep in posttraumatic stress disorder: A systematic review and meta-analysis of polysomnographic findings. *Sleep Medicine Reviews*, *48*, 101210. https://doi.org/10.1016/j.smrv.2019.08.004

Zhu, W., Chen, Y., & Xia, L.-X. (2020). Childhood maltreatment and aggression: The mediating roles of hostile attribution bias and anger rumination. *Personality and Individual Differences*, *162*, 110007. https://doi.org/10.1016/j.paid.2020.110007

Zukerman, G., Pinhas, M., & Icht, M. (2023). Hypervigilance or shutdown? Electrophysiological processing of trauma-unrelated aversive stimuli after traumatic life events. *Exp Brain Res*, *241*(4), 1185-1197. https://doi.org/10.1007/s00221-023-06578-w