

The Nature and Mechanisms of Psychotic Experiences in Borderline Personality Disorder



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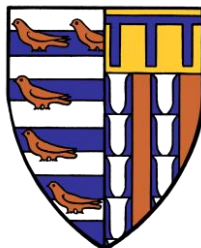
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Declaration

I hereby declare that this thesis is the result of my own work and includes nothing which is the outcome of work done in collaboration except as declared in the Acknowledgements and specified in the text. I further state that this dissertation has not been submitted in whole or in part for consideration for any such degree, diploma, or other qualification at the University of Cambridge or any other university or similar institution. It does not exceed the prescribed word limit for the relevant Degree Committee.

Charlotte Peitzmeier

8 November, 2023

Abstract

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Borderline personality disorder (BPD) is a diagnostic label ascribed to a markedly heterogenous psychiatric phenotype, with symptoms ranging from intense emotions to disturbed relatedness, from impulsivity and self-harm to chronic feelings of guilt, shame, and emptiness. While often regarded as an affective disorder, psychotic symptoms such as hallucinations and delusional convictions are also frequently experienced by people with BPD but are habitually dismissed in both research and clinical practice. Their aetiology and the mechanisms underlying them remain poorly understood, largely due to historical and prevailing stigma towards the disorder. By building on recurring findings of psychotic symptom correlates, and by drawing on causal and cognitive models of psychosis in other mental health conditions it is possible to explore potential risk factors and create a preliminary aetiological framework to better inform and direct future research.

This dissertation aims to expand the currently sparse literature on psychotic experiences in BPD, by providing a detailed assessment of the prevalence (Chapter 4), type, and range of hallucinations and delusions (Chapter 5), as well as investigating key factors hypothesised to influence their occurrence or severity (Chapter 6 and 7) with two empirical studies – an online experiment recruiting BPD participants identified by self-report, and a laboratory study of people with a confirmed clinical diagnosis. Both hallucinatory and delusional experiences were significantly more common in people with BPD, presenting on a broad spectrum of frequency and severity – ranging from non-pathological to comparable with psychotic disorders. Dissociation was the strongest predictor for psychotic symptom prevalence and severity for both, perceptual and delusional experiences. Several other factors (including trauma and adversity, disorder severity, psychiatric comorbidities, and

common mental distress) were found to generally correlate with (but have limited predictive value for) psychotic experiences. Additionally, current stress levels appeared to influence dissociative and psychotic state symptoms. Results did not support a causal role of aberrant prediction error signalling (as postulated by predictive processing theories) for psychosis proneness in people with borderline personality disorder.

In summary, this thesis furthers our understanding of the prevalence and nature of psychotic symptoms in BPD and provides novel insights into the possible cognitive and aetiological mechanisms underlying these experiences.

*"Wenn Du vor mir stehst und mich ansiehst,
was weißt Du von den Schmerzen, die in mir sind
und was weiß ich von den Deinen.
Und wenn ich mich vor Dir niederwerfen würde und weinen,
und erzählen,
was wüsstest Du von mir mehr als von der Hölle,
wenn Dir jemand erzählt, sie ist heiß und fürchterlich.
Schon darum sollten wir Menschen voreinander
so ehrfürchtig,
so nachdenklich,
so liebend stehn,
wie vor dem Eingang zur Hölle."*

Franz Kafka, Brief an Oscar Pollak, 8 November 1903

*"When you stand before me and look at me,
what do you know of the pains inside me
and what do I know of yours.
And if I were to throw myself at your feet and cry,
and tell,
what more would you know of me than of hell,
having been told that it is scorching and frightful.
For that alone, us humans should stand before one another
as reverently,
as reflective,
as loving,
as at the gates of hell."*

Franz Kafka, Letter to Oscar Pollak, 8 November 1903

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Nomenclature

Acronyms/Abbreviations

ADD/ADHD	Attention Deficit (Hyperactivity) Disorder
ANCOVA	Analysis of Covariance
ANOVA	Analysis of Variance
AQ-10	Autism Quotient
ASC	Autism Spectrum Condition
ASSIST	Alcohol, Substance and Smoking Involvement Test
AVH	Auditory-Verbal Hallucinations
BAVQ-R	Beliefs About Voices Questionnaire (Revised)
BDI	The Beck Depression Inventory
BIC	Bayesian Information Criterion
BPD	Borderline Personality Disorder
BSL	The Borderline Symptom List (Short-Form)
CADSS	Clinician-Administered Dissociative States Scale
CAPE	The Community Assessment of Psychic Experiences
CAPS	Cardiff Anomalous Perception Scale
CBQp	Cognitive Biases Questionnaire for Psychosis
CBT	Cognitive Behavioural Therapy
CFI	Comparative Fit Index
CI	Confidence Interval
CFA	Confirmatory Factor Analysis
CMD	Common Mental Distress
cPTSD	complex Post Traumatic Stress Disorder

CTQ	Childhood Trauma Questionnaire
CTQ-SA	Sexual Abuse
CTQ-PA	Physical Abuse
CTQ-EA	Emotional Abuse
CTQ-EN	Emotional Neglect
CTQ-PN	Physical Neglect
DEMO	The Dissociative Experiences Measure, Oxford
DES-II	Dissociative Experiences Scale
DIB-R	Diagnostic Interview for Borderlines (Revised)
DSM	Diagnostic and Statistical Manual of Mental Disorders
ED	Eating Disorder
EUPD	Emotionally Unstable Personality Disorder
FRS/SFR	First-Rank Symptoms
GAD	Generalised Anxiety Disorder Questionnaire
GMV	Grey Matter Volume
HPA	Hypothalamic-Pituitary-Adrenal Axis
ICD	International Classification for Diseases
IQR	Interquartile Range
LSC-R	Life Stressor Checklist – Revised
LV	Latent Variable
MINI-Plus	Mini-International Neuropsychiatric Interview (Plus)
ML	Maximum Likelihood (estimator)
MSI-BPD	McLean Screening Instrument for BPD
NHS	National Health Service
OCD	Obsessive Compulsive Disorder
PANSS	Positive and Negative Syndrome Scale
PCA	Principal Component Analysis
PCL	PTSD checklist for DSM-5
PD	Personality Disorder

PDCS	Personality Disorder Community Service
PDI	Peter's Delusion Inventory
PE	Psychotic Experiences
PPI	Patient and Public Involvement
PSE	Present State Examination
PSI	The Psychotomimetic States Inventory
PSP	Personal and Social Performance Scale
PSQ	Perceived Stress Questionnaire
PsychREC	University of Cambridge Psychology Ethics Committee
PSYRATS-H	Psychotic Symptom Rating Scales (Hallucinations)
PTPD	Post Traumatic Personality Disorder/Disorganisation
PTSD	Post Traumatic Stress Disorder
QIDS-SR	Quick Inventory of Depressive Symptomology
QPE	Questionnaire for Psychotic Experiences
RMSEA	Root Square Error of Approximation
RT	Reaction Time
RSS	Residual Sum of Squares
SAM	Self-Assessment Manikin
SCID	Structured Clinical Interview for DSM
SCL-90	Symptom Checklist-90
SEM	Structural Equation Model
SFR/ FRS	Schneider's First Rank (Symptoms)
SSI	Schizotypal Symptom Inventory
SRMR	Standardized Root Mean Squared Residual
SSRIs	Selective Serotonin Reuptake Inhibitors
STAI	The State-Trait Anxiety Inventory
TSST	Trier Social Stress Test
UHR	Ultra-High Risk
WHO	World Health Organization

Chapter 1 The art of hugging a porcupine – an introduction

“Die Kunst, ein Stachelschwein zu umarmen“¹

This poignant title of a novel by author and BPD expert-by-experience Laura Adrian vividly describes both subjective experience of and sentiment towards a condition that is notoriously difficult to explain to (or be understood by) those not afflicted:

1.1 What is borderline personality disorder?

At its core, borderline personality disorder (BPD) may perhaps best be described as a disorder of relatedness – to society, to other people, and (possibly most prominently) to oneself. Sufferers consistently disclose histories of trauma and abuse, often complex and ongoing. Over time, these have shaped the way they perceive and react to the world – emotionally and cognitively. The symptoms of such a fundamental disturbance are manifold and range from dissociation and chronic feelings of emptiness to intense emotions and affective instability, from social withdrawal and self-harm to impulsive behaviour and outward hostility. In clinical practice, this translates to a vast variety of presentations with supposedly over 250 different combinations of criteria yielding the same diagnosis (Biskin and Paris, 2012).

Ironically, the casual understanding of the condition is far more basic and steeped in prejudice. If asked to describe the typical (generally assumed to be female) patient, people

¹ “The art of hugging a porcupine” (Adrian, 2016)

across the societal spectrum from lay persons, over academics, to medical professionals will paint a badly tainted picture of emotional outbursts, manipulative behaviour, and violence. When talking to experts-by-experience for any amount of time, evidence of this stigma and its impact is practically inescapable. They share anecdotes of how, as recent as 2016, the Canadian government enquired about their personality disorder (PD) diagnosis as part of its electronic travel authorization application process, reserving the right to declare people with BPD inadmissible to the country due to a ‘danger to public safety’ under section 38 (medical inadmissibility) of the immigration act². Private psychiatrists are said to limit the amount of people with BPD they accept (Kreisman and Straus, 1989), and many health practitioners will avoid (Sulzer et al., 2016) or even refuse (McGrath and Dowling, 2012) patients with the diagnosis, who are commonly depreciated as “difficult”, “attention-seeking” (Sulzer, 2015), and inherently treatment resistant. Fortuitously, a growing body of research has recently begun to consider this prevailing prejudice against BPD in societal, structural, and clinical settings, much of which has been succinctly summarised by Klein et al. (2022).

Despite a wealth of theories on what might be considered the central, driving feature of the disorder, it remains relatively elusive. Early attempts to differentiate borderline pathology from other psychiatric diagnoses found that interpersonal functioning, affect modulation, and impulsivity most reliably identified borderline patients (Sheehy et al., 1980; Clarkin et al., 1983). The latter two were subsequently adopted as the pathology’s major phenotypes, while interpersonal difficulties were considered to be primarily (mal)adaptive behaviours (Siever and Davis, 1991). This understanding began to shift when Gunderson (2007) proposed a reconceptualization of the BPD paradigm which integrated *disturbed relatedness* as a third, fundamental attribute of the disorder, based on observations from clinical practice and developments in cognitive behavioural theories and treatment approaches (Gunderson, 2007). However, whether disturbed relatedness constitutes the

² The question’s phrasing now reads “*untreated mental health condition with psychosis*” (Government of Canada, 2017; earlier versions publicly not available). The overall construct of medical inadmissibility has received severe backlash due to its discrimination of people with a wide range of disabilities (Wilton et al., 2017), and is set to receive a series of updates.

primary source of all borderline symptomology or whether it is also simply the symptom of a more profound, underlying pathology remains unclear.

While some (Trull et al., 2011) argue that there is no single primary “borderline” identity and that all its pathology should be considered solely along spectra of genotypic and phenotypic presentations, an intriguing (unifying) hypothesis stems from Meares et al. (2011), who (using factor analysis) suggested that at the heart of the disorder lies a deeply fragmented sense of self – a “painful incoherence” reflected by chronic emptiness, fear of abandonment, and intense emotional pain. Within this framework, all other significant borderline features (including high-cyclothymic temperament, recurrent self-injurious behaviour, and reduced self-control) would merely be externalised by-products of the tremendously distressing and inescapable inner turmoil.

1.1.1 Diagnostic conceptualisations

Strictly speaking, the term “borderline personality disorder” is an American construct, first introduced into the Diagnostic and Statistical Manual of Mental Disorders (DSM) in 1980, and in the United Kingdom – which uses the International Classification for Diseases (ICD), currently still applied in its tenth edition (World Health Organization, 2016) – the label does not exist per se. However, the ICD-10’s (phenotypical) equivalent “emotionally unstable personality disorder” (EUPD) uses diagnostic criteria that closely resemble those of BPD, and the two are routinely used interchangeably by patients, clinicians, and researchers alike.

In its most recent (fifth) rendition, the DSM classifies personality disorders as persistent and inflexible patterns of traits and behaviours that cause significant impairment and are not better explained by another condition and/or substance use (American Psychiatric Association, 2013). There are currently ten specific PDs, grouped into three clusters based on their symptomatic similarities: odd and eccentric disorders (A), dramatic or emotional disorders (B), and anxious or fearful disorders (C). The manual additionally includes three further diagnostic labels for pathological patterns that do not match any category but are nevertheless characteristic of a personality disorder.

BPD is considered part of cluster B, and is typically thought to be characterised by three major traits (Sanislow et al., 2000; Sanislow et al., 2002):

1. Emotional dysregulation (emotional instability, raised affect intensity, reduced affect control, and chronic feelings of emptiness)
2. Impulsivity (including recklessness and self-damaging behaviour)
3. Disturbed Relatedness (identity disturbance, hypermentalization, dichotomous thinking, intense or unstable interpersonal relationships, and fear of abandonment)

The ICD-10 construct EUPD further distinguishes between two subtypes: the impulsive type, marked predominantly by reduced impulse control and emotional instability, and the borderline type, marked additionally by disturbances in the relationship with self and others, chronic feelings of emptiness, and self-harming behaviour.

However, with the publishing of the ICD's most recent (eleventh) revision (World Health Organization, 2019; published in 2022) the official model of personality disorders underwent a radical change. Replacing the categorical classification, personality disturbance will henceforth be evaluated on a continuum – from mild to severe. Subsequently, the diagnosing clinician can specify up to five disorder “traits” to qualify the severity with relevant psychopathological aspects. An additional descriptor (“*personality difficulty*”) was also introduced as a non-disease factor (a variable which is not clinically relevant but may nevertheless affect health status), thereby explicitly expanding the scope of experiences recognised under the PD umbrella. After much (and despite ongoing) debate (Tyrer et al., 2019), the 11th revision preserved a borderline pattern qualifier, based on symptom profile of previous ICD-10 and DSM-5 diagnostic criteria. Officially published in January 2022, these changes are not expected to become fully implemented in England before April 2026 (NHS Digital, 2023) and most people currently living with a diagnosis of BPD/EUPD will have been assessed using DSM-5 or ICD-10 criteria.

1.1.2 Morbidity and comorbidity

Psychiatric comorbidities in patients with a primary diagnosis of borderline personality disorder are extremely common and well documented (Hudziak et al., 1997; Zanarini et al., 1998; Zimmerman and Mattia, 1999; Becker et al., 2000; Pagura et al., 2010; Loas et al., 2013). In fact, the pathology is associated with an increased risk for almost every other common mental disorder – including anxiety, depression, post-traumatic stress disorder (PTSD), substance-use disorder, and other PDs – to such an extent that people with BPD and *no* psychiatric comorbidities may be considered the exception. Moreover, borderline symptomology is associated with a high lifetime risk of suicide with up to ten percent of sufferers ending their own life (Skodol et al., 2002; Paris, 2019), often after long courses of unsuccessful treatment (Paris, 2003). Men appear to be especially at risk, likely due a decreased likelihood of seeking treatment and increased rates of non- or misdiagnosis (McGirr et al., 2007).

In addition to severe impairment in mental and emotional functioning, BPD is frequently accompanied by substantial physical health problems (El-Gabalawy et al., 2010; Dokucu and Cloninger, 2019), with an on average twenty years shorter life-expectancy (Castle, 2019). A connection between bodily and mental health has long been established in other psychiatric conditions, including depression (Jaremka et al., 2013) and PTSD (Ryder et al., 2018). Now, a growing body of research suggests a similar but unique association between BPD and several physical health conditions, including cardiovascular disease, arthritis, gastro-intestinal problems, and a range of chronic pain conditions (Grant et al., 2008; Frankenburg and Zanarini, 2006). Preliminary evidence has also linked physical comorbidities – especially chronic pain – with poorer outcomes and lower remission rates for both BPD and the chronic physical illness (Frankenburg and Zanarini, 2006; El-Gabalawy et al., 2010).

There are likely several factors mediating the complex relationship between physical and psychological health in BPD, and emotional dysregulation has been suggested as one possible connection (Gratz et al., 2017) since heightened emotional reaction could be associated with a stronger physiological response to stressors. Neuroimmunology paradims

similarly postulate that chronically elevated levels of inflammation (a physiological correlate of stress and depression) can increase the risk for infection (Cohen et al., 1991), cardio-vascular disease (Schulz et al., 2000; Joynt et al., 2003), and further metabolic and neurodegenerative disorders (Hansson, 2005; Hotamisligil, 2006; Whisman, 2010; Pan et al., 2012). Empirically, affect instability (and other aspects of borderline pathology) appear to be impacted by a disturbance in interoception (the ability to recognise and describe changes of one's internal bodily states), which suggests that physiological underpinnings may, in turn, also influence psychological pathology (Löffler et al., 2018). The hypothesised link between interoception and emotion dysregulation is equally supported by recent findings that people with BPD showed reduced cortical representation of cardiac signs (Schmitz et al., 2020; Flasbeck et al., 2020), which implies a deficit in the processing of internal bodily signals. However, a previous study comparing people with borderline PD to a control sample and to people with depression found the BPD group to have overall greater interoceptive (heart beat perception) abilities (Mussgay et al., 1999), and others yet did not observe any significant differences in objective measures of interoceptive awareness between individuals with BPD and healthy controls (Hart et al., 2013). Consequently, the role of interoception in emotion regulation (and borderline pathology) remains poorly understood.

1.1.3 Problems, nothing but problems - Conceptual and consequential

Despite the recent revisions to the diagnostic criteria for (borderline) personality disorder significant issues remain with the BPD construct, both conceptual and consequential. The vast diversity of borderline pathology is often insufficiently considered in diagnostic and treatment approaches and the resulting high rate of misdiagnosis and inappropriate care carries detrimental consequences for those affected (Porr, 2017). This becomes remarkably apparent when considering psychotic experiences in BPD.

Persistent auditory hallucinations in BPD can be phenomenologically indistinguishable from those in schizophrenia (Slotema et al., 2012; Tschoeke et al., 2014) and may result in a primary diagnosis of psychotic disorder. Indeed, some studies suggest that BPD patients experiencing hallucinations or delusions are wrongfully treated for schizophrenia

(Beatson, 2019; Beatson et al., 2019), and a growing body of research considers the substantial rate of misdiagnosis with bipolar disorder³ blamed on the affective similarities and cycling nature of both pathologies (Ruggero et al., 2010; Paris and Black 2015).

Unfortunately, one of BPD's fundamental complexities stems from the lack of scientific, societal, and clinical consensus surrounding its causes, mechanisms, and even its basic construct and symptom structure: Is it an affective disorder, defined by emotional vulnerability which is driving all other symptoms as frantic attempts to avoid negative affect? The ICD-10's diagnostic label of emotionally unstable personality disorder certainly endorsed this classification for almost thirty years and many service users with a primary diagnosis of BPD report that researchers and health professionals alike lay the strongest (and at times the only) emphasis on their emotional and impulsive symptoms, dismissing other experiences (such as physiological comorbidities or perceptual disturbances) as exaggerations – an overstated by-product of their affective pathology.

Or is it perhaps a grossly misunderstood form of complex PTSD, and affective symptoms and extreme behaviours are coping- and defence mechanisms acquired in the face of adversity? This model is increasingly supported by a large body of research that overwhelmingly connects BPD with histories of childhood abuse and neglect (Zanarini et al., 2006), which appear to be more prevalent than in any other personality disorder (Battle et al., 2004; Yen et al., 2002) and seem to correlate to borderline symptom severity (Martín-Blanco et al., 2014; Gunderson et al., 2006). Consequently, many of those diagnosed chose to reframe their condition in the context of previous adverse experiences as a form of complex trauma (cPTSD), and the label “*personality disorder*” has received considerable backlash due to its insinuation that the fault lies with the affected. To tackle the significant overlap between BPD, PTSD, and cPTSD, Giourou et al. (2018) proposed the concept of a comorbidity cluster that reflects the continuum of adversity and its common clinical features. Similar concepts, including a post-traumatic personality disorder (PTPD), have previously been proposed as possible reformulations of the overlap (Classen et al., 2006).

³ Similarly, many people may be wrongfully diagnosed with BPD when in fact bipolar disorder would be a more suitable diagnosis.

A mere change in name, however, is unlikely to erase decades of ingrained prejudice and may risk simply replacing the label on the proverbial box into which patients are placed.

Whether the ICD-11's new dimensional model can resolve some of these persisting conceptional issues and pave the way for more accurate diagnosis and better prognosis (and perchance less stigma) remains to be seen. In a 2019 commentary, Jay Watts described the potential pitfalls of the new classification under which more people than ever could be described as having a personality disorder or disturbance (Watts, 2019). Admittedly, several studies have highlighted the relevance of sub-clinical personality difficulties for service-use and social functioning (Yang et al., 2010; Sanatinia et al., 2016), as well as in detecting adolescent personality pathology (Ramleth et al., 2017). However, it also risks exposing increased numbers of people to the PD label and its associated prejudice and discrimination. Moreover, the sudden drastic overhaul and wider definition of the concept threatens to further diminish the societal acceptance of an already questioned diagnosis and to divert resources away from those on the more severe spectrum of the symptomology. While the dimensional approach to personality pathology is undeniably the superior theoretical framework (compared to a categorical one) we can but wait and hope that applicability and implications for clinical practice follow suit.

1.2 Crossing the line – Psychotic experiences in BPD

“I describe my life as living somewhere 'just outside of reality' and sometimes I feel like I can see the 'layers' of reality; I've seen flies that are 'faded' as if they are obscured by layers of thick plastic that aren't there.

All these experiences are exacerbated by me spending time alone observing myself and my feelings; good for my mood, really bad for all this. It feels like the more socially disconnected I am from the world I am part of, the more I can connect with this other world instead.”

1.2.1 An origin story

The relationship between borderline personality and psychotic experiences is complicated, complex, and was written into the very foundation of their theoretical constructs. In its earliest applications, *psychosis* was suggested as a synonym for *psychopathy* (then broadly used to describe personality disorders), due to the drastic changes in personality it appeared to incite (Feuchtersleben, 1845). Development of the concept that personality dysfunction might constitute its own separate pathology was later (largely) driven by psychiatrist Kurt Schneider, who in his clinical practice observed what he termed “*abnormal personalities*” (Schneider, 1950)⁴. Notably, Schneider made a clear distinction between pathological personalities (that caused suffering to the individual and others around them) and unusual; but non-pathological personalities, such as eccentric creatives or exceedingly intelligent people. He proposed a classification system that differentiated between ten distinct types of personality pathology. While most of them have long been superseded by other (arguably more accurate) definitions, his “*emotionally unstable personality*” would later

⁴ Schneider reportedly first proposed and used the term in 1923, but the theory was most effectively summarised and evaluated in his 1950 publication *Die psychopathischen Persönlichkeiten*.

evolve into (and become a frequent descriptor of) what is now categorised as borderline or EUPD.⁵

Similarly, on the historical flipside of the borderline-psychosis coin, the etymological and conceptual aetiology of BPD further strengthens the initial and ongoing link between the two psychopathologies. Predating its clinical applications in the 1980s, the first official use of the descriptor “*borderline*” occurred in 1938. By then, the term psychosis had been widely established and in combination with neurosis was used alongside neurosis to describe the full range of virtually all mental disorders. Diagnostic differentiation between the two categories was strict and could be broadly summarised as follows: psychoses were illnesses with incomprehensible symptoms but an underlying somatic process, whereas neuroses were a psychological phenomenon, the perversion of a healthy psyche by environmental or biographical factors (Jaspers, 1913). In other words, psychoses were considered a disease of the brain, and neuroses a disease of the mind.

American psychiatrist and psychoanalyst Adolf Stern however observed that a group of patients did not appear to fit into either category. Their histories of trauma and general affective instability would have called for a neurotic classification, however drastic changes in behaviour affecting every aspect of their personality, as well as frequently reported “*psychic*” symptoms such as hallucinations and delusions would have suggested a psychotic diagnosis. The term “*borderline*” was thus coined to describe people falling in-between the prevailing diagnostic categories (Stern, 1938), and psychotic experiences were considered a core feature of its symptomology. Yet, for a long time the borderline construct remained controversial (see Gunderson, 2009 for a comprehensive ontogeny of the diagnosis).

⁵ Aside from his significant influence on our current understanding of personality pathology, right down to the DSM-5 and ICD-10 definitions and categories of PDs, Schneider also played a crucial role in establishing differential diagnostic procedures for psychotic disorders. Emphasising the importance of form over content, he formulated a series of “*first-rank symptoms*”, positive symptoms of psychosis (primarily including auditory hallucinations, unusual thought experiences, and strongly held delusions), long believed to be uniquely characteristic of schizophrenia (Schneider, 1959). Their specificity has now largely been disproven and they are no longer used diagnostically but Schneiderian first-rank symptoms (SFR/FRS) retain an important place in descriptive psychopathology and clinical practice.

Inevitably, further psychopathologies emerged that did not fit within the dichotomous mental health framework and by the time BPD came into general clinical use (with the publication of the DSM-III in 1980) the classification of mental health disorders had incorporated (most of) the diagnostic categories known today. Psychotic experiences became a symptom of Major Axis I disorders while BPD joined the cluster B of personality disorders (on Axis II, alongside “*mental retardation*”)⁶. For years any association between them was in part forgotten, and in part purposefully ignored, and the ICD-10 (diagnostic tool for EUPD in the UK since 1994) does not mention psychotic experiences as an aspect of borderline pathology⁷ leaving an incomplete clinical picture, an obsolete name, and many questions.

1.2.2 Psychotic or psychosis-like

With the dawn of the twenty-first century the theoretical conceptualisation of psychosis began to shift from a categorial all-or-nothing state towards a more dimensional understanding, with pathological symptoms reflecting the extreme end of an expansive spectrum. Explorations into whether, how, and why hallucinations and delusions (attenuated or otherwise) permeate ultra-high risk (UHR) and non-clinical populations subsequently exploded (Johns and Van Os, 2001; Van Os, 2003; Cougnard et al., 2007; Nuevo et al., 2012; Laurens et al., 2012; Linscott and Van Os, 2013; to name but a few). The continuum model of psychosis has now been widely established – albeit not withstanding considerable debate (David, 2010; Lawrie et al., 2010). Justifiable concerns over the idea of a single continuum have questioned the predominantly quantitative, questionnaire-based approach to assessing PEs in the general population, which tend to disregard experience quality, frequency, and associated distress (Sommer, 2010). Daalman et al. (2011) reported markedly little phenomenological overlap between the hallucinations of clinical and non-clinical voice hearers (with predominantly negative content in patients, but predominantly positive content in the general population). Consequently, a conceptual

⁶ The DSM-5 (published in 2013) finally abolished the multi-axial system and classified personality disorders alongside other mental health diagnoses. Similarly, “*mental retardation*” received a long-overdue reconceptualization as “intellectual disabilities (intellectual developmental disorders)”.

⁷ The DSM similarly only considers “transient, stress-related paranoid ideation or severe dissociation”.

compromise might be an extended psychosis phenotype (Howes and Kapur, 2009; Kaymaz and Van Os, 2010), representing risk factors rather than attenuated pathologies, with potentially differing neurobiological mechanisms underlying clinical and non-clinical experiences (Garrison et al., 2019).

Along with the burgeoning hypothesis of a psychosis spectrum, psychotic experiences in BPD also received renewed attention. Clinicians started publishing accounts of their patients' symptoms (Yee et al., 2005; Adams and Sanders, 2011) and qualitative assessments began to compare such experiences to the symptoms of schizophrenia (Kingdon et al., 2010; Slotema et al., 2012; Tschoeke et al., 2014; Cavelti et al., 2019; see Merrett et al., 2016 for a comprehensive review) and (to a lesser extent) bipolar disorder (Benvenuti et al., 2005; Henriques-Calado et al., 2023). Further studies set out to investigate the efficacy of antipsychotic treatment for people with BPD (see Slotema et al., 2018 for a systematic review), while others researched the impact of borderline personality traits on psychotic symptom development in populations with a clinically high risk for psychosis (Thompson et al., 2012).

However, despite this sudden increase in interest there remains a persisting clinical and academic stance towards the validity and nature of these symptoms, unparalleled in psychiatric research. The final decades of the twentieth century commonly discredited patients' experiences altogether, reporting them as factitious (Pope, 1983; Jonas and Pope, 1984; Links et al., 1989) or voluntary (Pope et al., 1985), likely influenced by the prevailing notion that people with BPD are prone to lying. Later views credited them with truth but applied labels such as "*pseudo-*" or "*quasi-psychotic*" (as used by Zanarini et al., 1990; Oliva et al., 2014; Zanarini et al., 2013; similarly, Adam and Sanders, 2011 reported frequent use of these descriptors in patient case notes), thereby inadvertently downplaying the severity of such symptoms and disregarding people's (often substantial) distress. An ostensibly shorter (when compared to schizophrenia) duration, and an increased insight into the reality of the experiences are often cited as reasons for this etymological distinction. In its most recent development, this conceptual differentiation is frequently made between *psychotic* and *psychosis-like* experiences (e.g., Schroeder et al., 2018). While this is arguably less demeaning than previous iterations of the term, it nevertheless

begs the question of appropriateness – and of relevance. A review of the concept of “pseudo-hallucinations” (Van Der Zwaard and Polak, 2001) highlights its low construct-validity and lack of clinical value. In a theoretical context, the distinction makes equally little sense: Any experience falling along a continuum of psychosis should, by definition, be considered “*psychotic*”⁸. This inanity becomes particularly apparent when compared to any other form of psychopathology: the brevity of a panic attack would not make it anxiety-like, just as an insight into the reality of a depressive episode does not cause it to be depression-like. Finally, in practice, the separation is unnecessary, inconsistent, equivocal, and impeding the comparability and reproducibility of research findings due to the subjective and arbitrary thresholds for what is considered a “true” psychotic symptom.

1.2.3 An incomplete picture, and many questions

A long series of periodically recurring reviews and commentaries (so many in fact that they appear to almost outnumber the limited research they are based on, and with an embarrassing number of articles predominantly citing previous reviews)⁹ have attempted to paint a coherent picture of the prevalence and predictors of psychotic experiences in BPD but have so far established only the outlines.

Prevalence estimates fluctuate between extremes (Table 1.1), with studies reporting “true” psychotic symptoms in none (Zanarini et al., 1990; Nishizono-Maher et al., 1993) to around half of their participants (Chopra and Beatson, 1988; Oliva et al., 2014). Subjectively placed thresholds between *psychotic* and *psychosis-like* further complicate interpretation of results, driving prevalence estimates up to 80-100% when including purportedly “pseudo-psychotic” symptoms (Chopra and Beatson, 1988; Oliva et al., 2014; Pearse et al., 2014).

⁸ Even in light of the ongoing debate surrounding the psychosis spectrum, this remains true for psychotic experiences in BPD which are consistently shown to have significant phenomenological overlap with those observed in schizophrenia (e.g., Kingdon et al., 2010; Slotema et al., 2012), thereby meeting both the quantitative and qualitative requirements of a psychosis continuum.

⁹ Barnow et al., 2010 ; Schroeder et al., 2013; Gras et al., 2014; Zonnenberg et al., 2016; Schultz and Hong, 2017; Frías, 2018 ; Beatson, 2019; D’Agostino et al., 2019; Cavelti et al., 2021 ; Minarikova et al, 2022

Differences in methodology (including psychometric measures, sample population, and sample size), as well as a lack of consensus on how to report symptoms or symptom categories (ranging from modality-specific assessments to broadly and vaguely defined groupings like “perceptual symptoms”), make direct comparisons between studies virtually impossible and prevent the formulation and generalisability of an overall prevalence estimate.

Auditory-verbal hallucinations (AVH) constitute the most extensively researched type of symptom with somewhat more consistent frequencies of around 30 (Yee et al., 2005; Slotema et al., 2017) to 50% (Kingdon et al., 2010; Pearse et al., 2014), depending on sample size and participant population. These experiences have been predominantly assessed in comparison to positive symptoms in patients with schizophrenia and appear remarkably similar with regard to (predominantly abusive) content, conviction, distress, and perceived location of the voice (Kingdon et al., 2010; Slotema et al., 2012; Hepworth et al., 2013; Tschoeke et al., 2014). Notable differences between the disorders include a greater emotional resistance to the experience in people with BPD (Hepworth et al., 2013) and an (on average) earlier age of onset (Tschoeke et al., 2014; Slotema et al., 2012). Although, considering the persisting discrepancies between studies in this field of research, it is unclear whether these findings are generalisable, or sample specific.

Other types of perceptual experiences have occasionally also been reported (typically alongside AVH) but have received much less attention (see Table 1.1). Hallucinations observed in other modalities include visual (11-32%), tactile (14-18%), and olfactory (10-28%) experiences, with very limited exploration of their phenomenology. It would be interesting to consider to what extent these might reflect sensory elements of post-traumatic phenomena in a group of people with high rates of (childhood) adversity.

Research on delusional symptoms remains almost entirely absent with only a handful of studies including their specific assessment, and an estimated prevalence of 20-30% (Kingdon et al., 2010; Oliva et al., 2014; Pearse et al., 2014, see Table 1.1). Again, a delusional versus quasi-delusional divide may be hampering accurate ascertainment of their true frequency.

Explorations into the exact causes or consequences of these symptoms are equally limited. Niemantsverdriet et al. (2017) found a significant association between the presence of hallucinations and the number of psychiatric comorbidities (with an apparent link to PTSD). A particularly intriguing correlation between hallucination severity and loneliness was reported by Slotema et al. (2019), and the directionality of this relationship certainly bears further exploration. Impact assessments report an increased risk for self-harm (Cavelti et al., 2019), more incidences of suicide plans and attempts (Kelleher et al., 2017; Slotema et al., 2017), and increased risk of hospitalisation and readmission (Furnes et al., 2021) in people with BPD who experience psychotic symptoms compared to those who do not.

Table 1.1

Prevalence of psychotic symptoms in previous studies (non-exhaustive). Types of experience are listed verbatim, as reported by the authors. Prevalence estimates are shown as percentages (either reported or calculated from the observed case numbers).

For full titles of the symptom measures see Nomenclature.

Study	Sample	n	Measure (s)	Experience	Prevalence
<i>Adams and Sanders, 2011</i>	service users	7	SCID	Any disturbed belief	100%
				Unusual experiences	100%
<i>Chopra and Beatson, 1988</i>	inpatients	13	DIB psychosis	Any psychotic experience	84.6 - 100%
				Brief paranoid experiences	76.9%
				Brief depressive experiences	76.9%
				True hallucinations or delusions	53.8%
<i>George and Soloff, 1986</i>	in- and outpatients	48	SSI	Perceptual symptoms	67%
				Cognitive symptoms	92%
<i>Kelleher et al., 2017</i>	community	7403	PSQ	Hallucinations	13.7%
<i>Kingdon et al., 2010</i>	in- and outpatients	33	BAVQ-R	Auditory hallucinations	49.5%
			PSYRATS-H	Paranoid delusions	29.7%
<i>Miller et al., 1993</i>	inpatient records	92	DSM-III Case notes	Any auditory or visual hallucination	27%
<i>Niemantsverdriet et al., 2017</i>	outpatients	323	PSYRATS-H	Any psychotic symptom	43%
			PANSS	Auditory	27 %
				Auditory verbal	21%
				Visual	11%
				Gustatory	8%
				Olfactory	17%
				Tactile	15%

<i>Nishizono-Maher et al., 1993</i>	outpatients	15	DIB	True psychotic symptoms	0%
				Brief depressed experience	13.3%
				Brief paranoid experience	13.3%
				Transient psychosis	26.7%
<i>Oliva et al., 2014</i>	outpatients	28	DIB-R PSP	Any psychotic symptom	85.7%
				True psychotic thought	46.4%
				Quasi-psychotic thought	82.1%
				Non-delusional paranoia	100%
<i>Pearse et al., 2014</i>	outpatients	30	PSE	Any psychotic symptom	60-80%
				Auditory hallucinations	50%
				Visual hallucinations	30%
				Delusions	20%
				Tactile hallucinations	14%
				Olfactory hallucinations	10%
				Ideas of Reference	27%
				Sensory distortion	12%
<i>Schroeder et al., 2018</i>	outpatients	95	SCID-I (German)	Perceptual alterations	36%
				Delusions	21%
<i>Slotema et al., 2017</i>	outpatients	89	PSYRATS MINI Plus	Auditory verbal hallucinations	30%
<i>Slotema et al., 2019</i>	outpatients	60	QPE	Any hallucinations	62%
				Auditory	40%
				Visual	32%
				Tactile	18%
				Olfactory	28%
<i>Yee et al., 2005</i>	BPD research & treatment programme	171	SCL-90 SCID-D	Auditory hallucinations/voices	29.2%
<i>Zanarini et al., 1990</i>	N/A	50	DIB-R	Non-psychotic disturbed thought	100%
				Quasi-psychotic thought	40%
				True psychotic thought past 2 years	0%
				True psychotic thought lifetime	14%
<i>Zanarini et al., 2013**</i>	inpatients	290	DIB-R SCID cognition	Any odd thinking	85.5% (37.2%)
				Any quasi-psychotic symptom	56.6% (7.4%)
				Quasi-delusions	48.6% (6.1%)
				Quasi-hallucinations	41% (3.5%)
				Unusual perceptions	76.2% (26.4%)
				Non-delusional paranoia	86.6% (43.3%)
				Ideas of reference	61.7% (28.6%)
				Paranoid ideation	51.7% (17.8%)
				Any true psychotic symptom	3.8% (1.7%)
				True delusions	3.5% (0.9%)
				True hallucinations	2.1% (1.7%)

** Zanarini et al. (2013) conducted a longitudinal study over 16 years with follow-up occurring every four years. The percentages presented are from the first and last (presented in brackets) data collection, respectively.

1.3 Aims of this dissertation

Considering the stark dissensus on something as conceptually basic as symptom prevalence it comes as no surprise that evaluations of possible causes of psychotic experiences in BPD are few and far between, and research into their neurological mechanisms still entirely absent. Current hypotheses on their aetiology are predominantly informed by research findings in other psychiatric populations and necessitate verification in the context of borderline pathology. Thus, the aim of this dissertation is to disentangle some of the uncertainties surrounding the nature and range of psychotic experiences in BPD and to assess their potential risk factors and mechanisms. Previously observed associations between psychosis and other aspects of psychopathology will be reassessed, and experimental paradigms (established and novel) investigating the cognitive underpinnings of (abnormal) perception will be implemented in people with BPD. Symptom prevalence and severity will be measured with validated psychometric instruments (with independent assessments of hallucinations and delusions), to fill some of the current gaps in the understanding of delusional experiences in particular. Of course, such scoping explorations cannot provide a definitive answer to the many questions still clouding our understanding of psychotic symptoms in BPD. Rather, they are intended to propose a viable aetiological model of these experiences that may lay the foundation for more targeted future research.

Chapter 2 summarises some of the current (and most relevant) theories on the development of psychotic symptoms, which are largely drawn from models proposed for and tested in other psychiatric conditions but may nevertheless be applicable in the context of BPD. Chapter 3 presents the research rationale and methodology for the (online) Screening survey, Study 1 (international online sample), and Study 2 (clinical in-person sample), including an overview of the questionnaire and task measures used, study procedures, and population demographics. In Chapter 4, the prevalence and severity of psychotic experiences is examined in people with and without BPD using the Screening data, and potential differences between people with BPD and high versus low psychotic symptom scores are investigated. Chapter 5 describes the range, frequency, and severity of hallucinations and delusions in people with BPD (and non-psychiatric controls), comparing

an online, self-report population (Study 1) to a clinical sample (Study 2). Chapter 6 evaluates possible risk factors and cognitive mechanisms, assessing correlations and determining the most reliable predictors for psychotic experiences (again, drawing comparisons between the two BPD populations and their respective control groups). The chapter additionally explores an aetiological structural equation model, defining latent variables to investigate the relationships between key predictors and their direct and indirect influence on symptom prevalence. Finally, in Chapter 7, the impact of stress on psychotic and dissociative experiences is investigated using a stress induction paradigm to compare people with BPD to control participants with regards to stress reactivity and changes in current state symptoms.

Chapter 2 On causes and cognition - a theoretical framework

“When I am extremely low or suicidal, I have visual hallucinations. I also experience paraesthesia when acutely distressed. The auditory hallucinations involve one voice, usually telling me to hang myself and asking how long it would take to ligature myself from every-day, inconspicuous points, like the back of a chair.”

Broadly hypothesised to arise from the catch-all combination of genetic predisposition and environmental influences, no definite causes of psychotic experiences in BPD have been identified. Equally, ascertaining their underlying mechanisms (be these neurobiological, or psychological in nature) has proved challenging. Considering the numerous models of psychotic experiences in schizophrenia, it is surprising how few comparable efforts have been made for personality disorders. Nevertheless, it is possible to extract several recurring factors that could account for the occurrence of hallucinations and delusions in people with BPD. These are not necessarily mutually exclusive and may instead reflect different causal and mechanistic levels, rather than conflicting theories:

2.1 Common mental distress and the p-factor

Conceptually, severe mental disorders are categorised as distinct entities with (at times somewhat arbitrary) diagnostic thresholds. Yet, in research and clinical practice they appear to follow a correlational structure and many patients present with significant lifetime comorbidities (McGrath et al., 2020), leading to calls for a more transdiagnostic approach to mental health problems (Dalglish et al., 2020). To explain the phenomenon,

Lahey et al. (2012) proposed the concept of a p-factor – a single common factor underlying psychopathology. In addition to more explicit variables that influence predisposition to specific disorders, the p-factor summarises a person's susceptibility to any and all types of mental distress (Caspi et al., 2014), which allows for a more spectrum-like picture of psychopathology and can account for meaningful symptom variation below and above diagnostic thresholds. Similar general factors have long been proposed in personality (and especially intelligence) research (Spearman, 1904), and Sharp et al. (2015) argued in favour of an underlying latent dimension to account for the correlational nature of PD traits – a model which is set to be actualised in diagnostic practice with the impending implementation of the ICD-11. Congruently, individuals with high p-factor scores have been suggested to exhibit marked difficulties in self-control when confronted with others, the environment, and themselves (Caspi et al., 2014) – a feature commonly associated with personality disorders. While some of the model's potential pitfalls have been discussed elsewhere (van Bork et al., 2017), a single latent variable to explain the vast correlational range of psychopathological observations certainly seems intriguing.

Another key construct within this dimensional framework is common mental distress (CMD), broadly depression and anxiety, as the directionality of increased psychopathological susceptibility is typically assumed to move from common symptoms (i.e., depression and anxiety) to more rare experiences (e.g., hallucinations and delusions) (Polek et al., 2020). Epidemiological studies have shown a consistent correlation between mental distress and the presence of psychotic experiences, with such symptoms appearing at the extreme end of the CMD continuum (Wigman et al., 2012; Kelleher et al., 2015; Perez et al., 2018; Heinze et al., 2018; Bhavsar et al., 2019). In the context of BPD, the high prevalence of ongoing, severe CMD (depression: Southwick et al., 1995; Levy et al., 2007; anxiety: Gratz et al., 2008, Howard et al., 2021; both: Comtois et al., 1999) could therefore increase psychosis proneness. Additionally (or alternatively), given the radical affect instability associated with the disorder and the time spent in intensely emotional states, it may be that individuals develop transient psychotic symptoms at the extreme ends of their mood spectrum.

2.2 (Childhood) trauma and dissociation

The impact of childhood trauma on brain development is well documented (Tarullo and Gunnar, 2006; Teicher et al., 2016) and various structural and functional alterations have been suggested to drive dissociation, emotion dysregulation, and impaired functioning in later life (Dunn et al., 2017; Cross et al., 2017).

Hallucinations and delusions have consistently been demonstrated to correlate with childhood abuse, in psychotic disorders as well as the general population (Ross and Joshi, 1992; Read et al., 2003; Daalman et al., 2012; Gaudiano and Zimmerman, 2009). Similarly, in BPD, childhood adversity appears to be the most frequently observed (and promising) predictor of psychotic experiences (Ross and Joshi, 1992; Tschoeke et al., 2014; Beatson et al., 2019), with patients reporting hallucinatory content specifically associated with past abuse (Yee et al., 2005; Adams and Sanders, 2011).

Considering the widely established (and possibly aetiological) relationship between different types of childhood trauma and borderline pathology (Zanarini et al., 1997; Johnson et al., 1999; Hengartner et al., 2013; Schulze et al., 2022) it may be that early-life adversity incites neurological changes that do not only increase the risk for developing BPD but further drive psychosis proneness in (some) people with this disorder.

Multiple MRI studies suggest that individuals with borderline PD show hyper-responsivity of the (left) amygdala (Herpertz et al., 2001; Donegan et al., 2003, Schulze et al., 2016) and posterior cingulate cortex (Schulze et al., 2016), as well as decreased responsiveness of the bilateral dorsolateral prefrontal cortex (compared with healthy controls), when processing negative emotional stimuli. These findings would suggest a greater (possibly affect-related) reactivity to negative stimuli, with reduced frontal cortical inhibition or moderation. Curiously, stimulation of the amygdala has previously been shown to evoke hallucinatory experiences in people with temporal lobe epilepsy (Gloor et al., 1982).

Conversely, a 2013 meta-analysis looking at contrasts of negative versus neutral emotion conditions in people with BPD (versus control participants) noted greater activation in the insula and posterior cingulate cortex, but overall reduced activation in the (right) amygdala

and frontal regions (Ruocco et al., 2013). This is further in line with reports of bilateral grey matter volume (GMV) reductions in both the hippocampus and amygdala (Nunes et al., 2009, Ruocco et al., 2012) of people with BPD. In turn, these findings seem to emulate some of the neuroanatomical changes observed in psychotic disorder, such as the amygdala volume reductions (repeatedly) associated with schizophrenia (Okada et al., 2016; Van Erp et al., 2016; Barth et al., 2021), as well as the significantly smaller hippocampal volumes of people with psychosis as identified by a meta-analysis in over 18,000 subjects (Hajima et al., 2013). However, in the context of borderline pathology results of hippocampal volume reductions have been inconsistent, and in some studies the amygdala volume of individuals with BPD (and comorbid depression) even appears to be *increased* (Zetsche et al., 2006). Notably, several studies (Brambilla et al., 2004; New et al., 2007; Schmahl et al., 2009; Sala et al., 2011) only found significantly reduced amygdala and/or hippocampal volumes in BPD patients with a history of childhood abuse. This of course would support the theory that such differences might indeed be neurobiological consequences of early-life adversity and may be differentially applicable to those who experienced certain types (or a certain severity) of trauma, which could further explain why psychotic experiences are reported by many, but not all people with BPD.¹⁰

Finally, the relationship between trauma and psychosis may not be direct but mediated or modulated by the presence and severity of dissociative experiences. The link between childhood adversity and the development of dissociative symptoms, both during and following the traumatic event, is well researched and overwhelmingly supported by two recent meta-analyses (Rafiq et al., 2018; Vonderlin et al., 2018). Moreover, a growing body of research is exploring the connection between dissociation and positive psychotic symptoms in patients with psychotic disorder (Perona-Garcelán et al., 2010; Černis et al., 2022), and supporting its role as a mediator for hallucination proneness (Longden et al., 2012; Perona-Garcelán et al., 2012; Varese et al., 2012).

¹⁰ To date, there have been no MRI studies comparing people with BPD with and without psychosis. In future, such studies might perhaps shed a light on associations between neuroanatomy and symptom prevalence and would enable better comparison with known neurobiological differences in schizophrenia or bipolar disorder.

In clinical populations, dissociative experiences are observed trans-diagnostically and can occur in the context of practically any major mental health disorder including depression (Parlar et al., 2017), bipolar disorder (Tuineag et al., 2020), and schizophrenia (Holowka et al., 2003). They are also typically (albeit not exclusively) associated with previous trauma. Phenomenologically, there is considerable overlap between dissociative experiences, identity disturbance, and temporal lobe abnormalities (Schenk and Bear, 1981; Devinsky et al., 1989; Kuyk et al., 1999; Blanke et al., 2005; Hara et al., 2015). Equally, several studies have associated borderline PD with elevated limbic irritability, a key measure of altered temporal lobe function (Harris et al., 2002; Schiavone et al., 2018), which may provide a plausible neurobiological bridge from trauma, over structural change, to dissociative (and potentially psychotic) symptoms. Besides its evident prominence in dissociative disorders, dissociation is frequently considered a core symptom of PTSD and BPD (Lyssenko et al., 2018) – despite its role in the latter being subject to ongoing debate. For instance, a 2017 meta-analytic review argues that dissociative symptoms should not be considered central to BPD symptomology and instead exists on a severity continuum (within the disorder) comparable to that observed in other psychiatric populations (Scalabrini et al., 2017). Furthermore, the aetiology of dissociative experiences appears to be less clear cut in the context of BPD where emotional vulnerability, affect instability, and stress, may be important additional risk factors for dissociation (Krause-Utz, 2022; Al-Shamali et al., 2022).

2.3 Cognitive biases and predictive processing

In 1991, Westen described aspects of how people with BPD relate to others and the world as what he termed “borderline attributional style” – the tendency to ascribed egocentric motivations to other’s thoughts and actions. This observation subsequently led him to formulate the much-quoted hypothesis that in BPD “the self is viewed as the causal centre of the social universe” (Westen, 1991). And while his theory originated from a psychoanalytic approach of the disorder, its central idea is still reflected within the framework of BPD associated cognitive biases.

Distinct from cognitive impairment (i.e., a deficit), a cognitive bias is a marked alteration in cognitive functioning, or more precisely, a distortion in the interpretation and integration of perceptual stimuli. Cognitive biases influence a person's subjective reality, causing it to diverge (at times severely) from the objective input which in turn may shape their behaviour. While many cognitive biases are prevalent throughout the population and form an intrinsic part of human psychology¹¹, they can also morph into rigid and pathological thinking patterns that drive psychiatric symptomology and can offer an insight into the cognitive underpinnings of mental health disorders (Millan et al., 2012; see Aue and Okon-Singer, 2020 for a comprehensive overview of cognitive biases and their implications for health and psychiatric pathologies).

In line with Westen's theory, cognitive biases in BPD are typically assessed in an interpersonal or social context. However, they may also factor into the development of psychotic experiences (Garety and Freeman, 1999; Bell et al., 2006; Freeman, 2007). Very specific types of cognitive bias are frequently observed in patients with schizophrenia, such as the tendency to reach conclusions quickly, confidently, and based on little evidence ("jumping-to-conclusions"), or the inability to adopt a critical stance towards an original guess despite evidence to the contrary ("overconfidence in errors") (Moritz et al., 2005; Van Dael et al., 2006; Menon et al., 2008). Interestingly, many of these (or comparable) thought patterns are also associated with borderline pathology (Ruocco, 2005; LeGris and Van Reekum, 2006; Mak and Lam, 2013). A process called "hypermentalization" (the attribution of exaggerated, complex, and typically malicious mental states to other people) is especially prevalent among people with BPD (Sharp et al., 2011; 2013). In perpetuity, this could perhaps lead to an inferential cycle of fear and suspicion centring around the (negative) thoughts and intentions of others, which (in extreme cases) might ultimately result in paranoid ideations of delusional intensity.

¹¹ Examples of wide-spread, non-pathological cognitive biases include *confirmation bias* (the tendency to overemphasise information that reaffirms an initial assumption and discredit information that challenges it), *priming bias* (the tendency to assign the greatest weight to an initial perception), and *congruence bias* (the tendency to test hypotheses directly, rather than testing alternative hypotheses), all of which of course are fallacies commonly affecting the quality of scientific research.

During a small-scale 2011 study assessing psychosis-related cognitive biases, participants with BPD scored high on the Cognitive Biases Questionnaire for Psychosis (CBQp) and were markedly more likely to relate external events to themselves (Moritz et al., 2011), a tendency which correlated with borderline symptom severity. CBQp scores have equally been found to correlate with symptoms of hallucinations and delusions (Peters et al., 2014). While this suggests an (at least partial) overlap between the cognitive profile of BPD and psychotic disorders such as schizophrenia it should be mentioned that cognitive biases in the latter are typically assessed with tasks, and questionnaire results may not be entirely transferable. In fact, when evaluating the reliability and validity of their instrument, the authors of the CBQp (Peters et al., 2014) remarked that none of the five biases measured related to existing experimental tasks and might possibly capture a separate cognitive construct. Nevertheless, marked alterations in cognitive functioning could still play a role in the aetiology of psychotic experiences in BPD: Rigid tendencies, such as to jumping to conclusions or overconfidence in errors, may simply be features of an overreliance on established beliefs over new evidence. This of course would tie in seamlessly with the increasingly auspicious theories of predictive processing¹² and its implications for (aberrant) perception.

The predictive processing framework is built on the understanding that our knowledge and (sensory) experience of the world is influenced by both prior knowledge and incoming information. It postulates that rather than simply receiving input passively, our brain actively provides (and updates) guesses about the input's potential causes and consequences. The result is a subjective reality, shaped by a balance between prior expectations (based on the current understanding of the world), and sensory signals that either confirm or reject these.

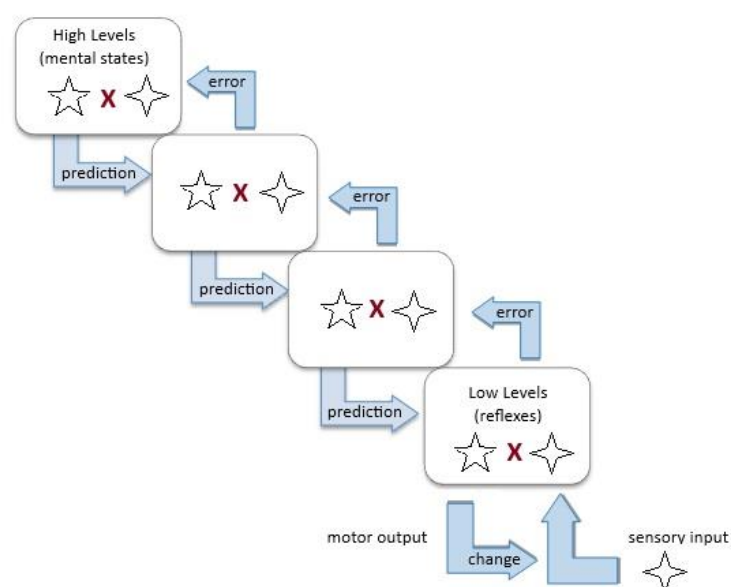
¹² So many theories in fact, that what may at first glance appear as one unified predictive processing framework is perhaps better understood as a series of closely related but distinct hypotheses, all of which (of course) have their own name. These include “predictive processing” (Clark, 2016), “predictive coding” (Friston and Kiebel, 2009), “the predictive mind” (Hohwy, 2013), and the “Bayesian brain” (Knill and Pouget, 2004). For the purpose of this dissertation the above terms (and further variations thereof) will be used more or less synonymously to refer to an overarching concept of hierarchical prediction error signalling.

On this basis, some of the most drastically computational proponents of predictive processing theories have argued that all that is encoded neuronally are predictions, and prediction errors (mismatches between expectation and input), which are passed up and down a cortical hierarchy of Bayesian inference in a sole effort to resolve and avoid disparities between expected and actual signals (Friston, 2005). Sensory input at low levels generates prediction errors which are forwarded upwards, while simultaneously, high levels pass down their predictions (Figure 2.1) until the error can be resolved. To decrease the risk of further mismatches the brain can either update its expectations in line with the new sensory evidence or try to change the sensory input through action.

Of course, such a theory wields the power to explain at least some of the cognitive processes underlying psychotic symptoms: If our perception and knowledge of the world is based on inference, then misperceptions and incorrect beliefs (i.e., hallucinations and delusions) could perhaps be caused by incorrect inference (Fletcher and Frith, 2009).

Figure 2.1

Illustration of the hypothesised Bayesian hierarchy of prediction error signalling.



Disturbed prediction error signalling (possibly due to dopaminergic dysregulation) could result in excessively strong bottom-up signalling and challenge existing predictions that are unable to account for this (Corlett et al., 2007; Corlett et al., 2016). Moreover, this unreliable and amplified perceptual “noise” may result in a maladaptation of the learning rate (as observed in people with first episode psychosis, see Suetani et al., 2022; Knolle et al., 2023) and could explain patterns of initial overfitting (e.g., quickly reaching a conclusion) and subsequent downregulation (e.g., inability to adopt a critical stance towards that conclusion) of the learning response (Griffin and Fletcher, 2017).

As such, a pronounced and persistent alteration in the balance between expectations about signals and the signals themselves may ultimately lead to aberrant salience (the incorrect assignment of importance to stimuli that would normally be considered neutral or irrelevant) which in turn would drive the development of profoundly altered models of the world – a distorted version of reality that manifests psychiatrically as positive psychotic symptoms.

The predictive processing theory of psychosis has been experimentally evidenced by a series of studies which found that individuals at high risk for psychosis (and already experiencing attenuated hallucinations) show a greater reliance on prior beliefs (Teufel et al., 2015) when interpreting ambiguous stimuli. It further aligns with findings of basic perceptual (visual) disambiguation deficits in people with psychosis (proneness), which may drive the necessity for an increased reliance on prior expectations to make sense of incoming information (Aleman et al., 2003; Leitman et al., 2010).

In absence of any tangible theories about the cognitive underpinnings of psychotic experiences in BPD, the predictive processing model of psychosis therefore offers a plausible (and experimentally viable) framework for assessing and understanding these symptoms. Perhaps people with BPD share a similar mechanistic perturbation of their Bayesian hierarchy, where unreliable bottom-up signalling causes an overreliance on prior information to form coherent mental states. Alternatively (or additionally), their expectations (and by extension predictions) may be significantly influenced by past trauma. This would profoundly affect inference accuracy and distort incoming information

in accordance with a threatening and unstable view of the world, thus driving the development of hallucinations and delusions.

2.4 Emotional vulnerability and borderline personality traits

It has been suggested that even premorbid personality disturbance could already increase a person's vulnerability to psychotic experiences or have a pathoplastic¹³ effect (Cuesta et al., 2002), and there is preliminary evidence that links psychotic experiences to BPD: borderline traits are prevalent among groups at high risk for psychosis (Sevilla-Llewellyn-Jones et al., 2018)¹⁴, and borderline symptom severity has been associated with greater likelihood of developing psychotic symptoms (Baryshnikov et al., 2018). Similarly, high levels of affect intensity correlate with an increase in almost all other aspects of BPD (Yen et al., 2002) which, by extension, might also include psychotic experiences. Individuals with the disorder typically experience negative feelings very strongly and are highly sensitive to social threat, especially to cues that signal rejection. Fears of abandonment can be intense and can give rise to unfounded beliefs that may almost be described as delusional, due to their baseless but unshakable conviction, often held in face of evidence to the contrary. Since threat evaluation is greatly influenced by affect, such non-bizarre (but severely distressing) convictions could possibly facilitate the transition to clinically relevant psychotic symptoms during heightened emotional states.

Furthermore, emotional vulnerability could also play a role in the pathological significance of auditory-verbal hallucinations in BPD. Non-pathological voice hearing is increasingly recognised as a benevolent form of perceptual abnormality (Iudici et al., 2019; Toh et al., 2022). Rising numbers of those experiencing the phenomenon embrace it as a form of neurodiversity and organisations like the Hearing Voices Network campaign to increase

¹³ The pathoplastic model describes personality as a major influence on psychopathology. It postulates that rather than simply pre-disposing a person to a mental illness, personality affects the way they interact with their environment, thereby shaping how the condition manifests (Clark et al., 1994; and Clark, 2005). Contrary to the vulnerability model, pathoplasticity considers both the positive and negative effects individual personality traits can have on the development of a disorder.

¹⁴ However, the predictive value for transition to a clinically relevant psychotic disorder is low.

visibility and normalise the experience (Styron et al., 2017). As a prominent example, Dr Eleanor Longden – psychologist and psychiatric survivor – eminently describes how the benign voices she started to hear as a young adult turned threatening and destructive only after she confided in friends and clinicians who unanimously declared her ill. In numerous interviews and a poignant TED-talk, she chronicles her journey from healthy voice hearer to branded schizophrenic (and back), emphasising the impact her emotional life had on the hallucinations. Indeed, a significant difference between healthy and clinical voice hearers appears to be the appraisal and emotional valence of the experience (Johns et al., 2014; Tsang et al., 2021; Buck et al., 2022). As such, the emotional vulnerability characteristic of BPD could perhaps make those affected by auditory hallucinations much more susceptible to distressing and clinically significant experiences.

2.5 The role of stress

Finally, the connection between psychotic experiences and stress (especially social stressors) is frequently noted in case studies (Yee et al., 2005; Adams and Sanders, 2011; Schultz and Hong, 2017) and reviews habitually list it among the causal or influencing factors (compare for example Schroeder et al., 2013; D’Agostino et al., 2019; Minarikova et al., 2022), yet strikingly few studies have researched this. By means of experience sampling, Glaser et al., 2010 investigated psychotic reactivity to everyday life stressors and reported higher reactivity to both major interpersonal and minor environmental stressors in people with BPD, compared to other personality disorders, healthy controls, and (interestingly) people with psychotic disorders. Another study (Niemantsverdriet et al., 2017) reported a positive correlation between psychotic experiences and the number of adult life stressors. These were measured with the Life Stressor Checklist (LSC-R), which includes a range of potentially impactful experiences such as natural disasters, financial troubles, and assault. Confoundingly, results were reported as “number of current life stressors”, even though the LSC-R measures any event that occurred after the age of eighteen on a dichotomous scale and is in many ways is better categorised as a measure of adult-life trauma than of stress.

In possibly related findings, studies have noted significant increases in state dissociation (Graumann et al., 2023) and heightened negative emotions (Deckers et al., 2015) in response to the Trier Social Stress Test (TSST) (in people with BPD). Considering the repeatedly observed relationship between dissociative and psychotic experiences these results may point towards a mediating role of dissociation, not just for overall psychosis proneness but perhaps also in the transition to acutely psychotic states under stress.

The influence of stress and stress responses on psychosis has similarly been of interest in the context of prodromal ultra-high risk (UHR) states and first episode psychosis, with a dysregulation of the hypothalamic-pituitary-adrenal axis (HPA) (and by extension, cortisol levels) as the hypothesised link between past stressful experiences and psychotic reactivity (Cullen et al., 2022; Vaessen et al., 2023; see Chaumette et al., 2016 for a comprehensive meta-analysis of earlier publications).

Unfortunately, a fundamental difficulty with assessing the relationship between stress, BPD, and psychosis arises from the fact that “*everyone knows what stress is, but nobody really knows about stress*” – as Hans Selye (originator of the term in its clinical sense) appropriately summarises the dilemma (Selye, 1973). An inherently subjective experience, stress can be emotional, psychological, physiological, or any combination of the above. Continuous low-level (or post-traumatic) distress and acutely stressful situations may have very different effects on BPD symptomology and (by extension) psychotic experiences. Numerous studies have established its implication in the dopaminergic system (Pani et al., 2000; Baik, 2020), as well as in the aetiology of depression where chronic stress is hypothesised to blunt reward sensitivity and increase anhedonia (Hammen, 2005; Ironside et al., 2018). Dopamine dysregulation in the ventral tegmental region has similarly emerged as a robust finding in studies on psychosis in schizophrenia (Howes et al., 2007) and it may be possible that intense stress produces transient dopamine dysregulation thereby acutely facilitating the development of (clinically relevant) psychotic experiences, however this remains speculative. Stress alone is of course unlikely to account for the full range of psychotic symptoms observed in people with BPD. However, in light of previous findings it may nevertheless be an important variable that could ultimately drive episodes of hallucinations and delusions in those already predisposed by other risk factors.

Chapter 3 Forth into the unknown - research rationale and methodology

“[These experiences] used to be full of light and wonder when I was a child, but as an adult, between the light and wonder was a lot of darkness and demons. The demons used to say, "Come with us to the kingdom of death and we will show you the Treasures of Darkness". I was very intrigued, and I went with them almost all the way to death 20 times but in the end, I decided this was illness rather than good spirituality, so I decided to stop following them.”

3.1 Introduction

Bearing in mind the sparsity of empirical studies, their inconsistencies, and their tainting by historic and ongoing stigma, an original assessment of the prevalence and characteristics of psychotic experiences in people with BPD was plainly necessary. With a multitude of variables hypothesised to influence the occurrence, intensity, or frequency of these symptoms, but no reliable aetiological or mechanistic model, the objective was furthermore to explore the relationship between trauma, psychopathology, and cognitive function, and to determine the most suitable subset of predictors for psychotic experiences in BPD.

Two studies were conducted (comparing people with BPD to volunteers with no known history of major psychiatric disorder), to address some of the cardinal topics that are still contested or unresolved:

1. The prevalence of psychotic experiences in people with BPD.
2. The range of psychotic symptoms experienced by people with BPD, including their severity and symptom type (hallucinations and delusions).

3. The relationship between borderline psychopathology, psychotic symptoms, and possible risk factors for psychosis: trauma, dissociation, common mental distress, the emotional valence of ambiguous sensory experiences, and changes in predictive processing.
4. The impact of stress on psychotic experiences in BPD.

Both studies shared a significant methodological overlap, and each included a variety of questionnaire and task measures pertaining to different aspects of the above areas of interest. As such, data was analysed (and will be presented) according to the topic it addresses, rather than by study. The following seeks to first provide an overview of the instruments used to assess psychopathology and cognitive mechanisms and then describe the experimental procedures of both studies, including the participant demographics of their respective populations. Further details of analytical methods will be given prior to description of the respective results, in each chapter.

3.2 Questionnaire measures

For a complete list of questionnaires (including full name, a brief description of their respective scoring systems, and reference) see Table 3.1.

3.2.1 Screening questionnaires

Preceding each study, volunteers interested in participating were asked to complete a brief online screening survey, which served as an assessment of inclusion and exclusion criteria (Appendix A.1). Participants were asked to confirm whether they had been diagnosed with BPD. They then completed a demographic assessment, and a series of questions about their psychiatric comorbidities. The CAPE-15 was included as a measure of psychotic experiences. Screening for Study 1 (an online study) furthermore included the MSI-BPD¹⁵

¹⁵ For the purpose of this study, MSI-BPD item 2 “*Have you deliberately hurt yourself physically (e.g. punched yourself, cut yourself, burned yourself)? How about made a suicide attempt?*” was separated into two questions. Endorsement of either item (as well as of both items) resulted in a score of one towards the overall total.

as a brief confirmatory assessment of their self-reported diagnosis. This was omitted in the screening survey for Study 2 (an in-person study), since eligible participants were asked to bring confirmation of their diagnosis to the testing session. Screening questionnaires took approximately five to ten minutes to complete.

3.2.2 Study questionnaires

A general demographic assessment of the study populations was included in both studies. Psychotic experiences were measured using the Cardiff Anomalous Perception Scale (CAPS) and Peters Delusion Inventory (PDI). General psychopathology was assessed with the following instruments:

- **Study 1:** QIDS (depression), GAD-7 (anxiety), DES-II (dissociation), CTQ (childhood trauma), PCL-5 (post-traumatic stress), ASSIST (risk for psychoactive substance use)
- **Study 2:** BDI (depression), STAI (anxiety), DEMO (dissociation), CTQ (childhood trauma), PCL-5 (post-traumatic stress), LSC-R (exposure to major life-stressors), BSL-23 (borderline symptom severity)

Study 2 additionally incorporated current state symptom measures of dissociation (CADSS) and psychotic experiences (PSI).

In addition to the validated measures of psychopathology listed above, both studies also involved custom questionnaires and rating scales:

1. A brief psychotic experiences follow-up questionnaire presented to participants who endorsed one or more symptoms on the CAPS and PDI (both studies), asking about perceptual symptom duration, delusional belief frequency, and any known influencing or triggering factors (Appendix A.2).
2. A ‘Stress-Check’ (Likert scale of 1-5), asking participants to rate their current feelings of stress (“*How stressed do you feel right now?*”), alertness (“*How alert do you feel right now?*”), and perceived level of control (“*How in-control do you feel right now?*”) (Study 2).

Table 3.1

Overview of questionnaire measures.

Instrument	Type of assessment	Authors
<i>Borderline symptom measures</i>		
McLean screening instrument for BPD (MSI-BPD)*	10-item self-report scale based on the DSM-IV criteria for BPD, with a dichotomous scale (yes/no) and total score as sum of items endorsed. A clinical cut-off of 7 indicates possible BPD	Zanarini et al., 2003
The Borderline Symptom List Short-Form (BSL-23)	23-item self-report scale to assess BPD symptom severity, with a 5-point (0-4) Likert scale. The average score (sum of item scores divided by 23) is calculated. Higher scores indicate more severe borderline symptom traits	Bohus et al., 2009
<i>Psychotic experiences measures</i>		
Community Assessment of Psychic Experiences (CAPE 15)*	15-item self-report scale to assess frequency and severity of psychotic experiences in the past three months, with two 5-point (0-4) Likert scales (assessing frequency and distress), and three subscales (persecutory ideation, perceptual abnormalities, and bizarre experiences). Scores are summed per scale and divided by the total number of items.	Capra et al., 2013
Cardiff Anomalous Perception Scale (CAPS)	32-item self-report scale assessing perceptual anomalies in the past three months, with a dichotomous (yes/no) primary dimension, followed by three 5-point (0-4) Likert scales (distress, intrusiveness, frequency), across nine symptom categories. Scores are obtained for the Yes/No total, and for each subscale.	Bell et al., 2006
Peter's Delusion Index (PDI)	21-item self-report scale of delusional ideation in the past three months, with a dichotomous (yes/no) primary dimension, followed by three 5-point (0-4) Likert scales (distress, conviction, preoccupation), measuring a single dimension. Scores are obtained for the Yes/No total, and for each subscale.	Peters et al., 2004
The Psychotomimetic States Inventory (PSI)	48-item self-report scale of current state psychotic-type experiences (developed in the context of cannabis and ketamine use), using a 4-point (0-3) Likert scale, with 6 subscales (delusions, perception, cognition, anhedonia, mania, paranoia). An overall total is obtained by summing all item scores.	Mason et al., 2008
<i>Dissociation measures</i>		
Dissociative Experiences Scale (DES-II)	28-item self-report scale on dissociative experiences, using a 10-step percentage (0-100) scale. Item scores are totalled and divided by 28 to calculate average.	Carlson & Putnam, 1993
The Dissociative Experiences Measure, Oxford (DEMO)	30-item self-report assessment of dissociative experiences in the past two weeks, with a 5-point (1-5) Likert scale. Item scores are summed to create an overall total.	Černis et al., 2018
Clinician-Administered Dissociative States Scale (CADSS)	A clinician administered 28-item interview to assess current dissociative states, rated on a 5-point (0-4) Likert scale. Items 1-23 are subjectively answered by the participant, items 24-28 are objectively observed by the clinician. All scores are summed for an overall total.	Bremner et al., 1998

<i>Common mental distress measures</i>		
Quick Inventory of Depressive Symptomology (QIDS-SR)	16-item self-report scale on depressive symptom severity, with a 4-point (0-3) Likert scale and nine item categories. The highest scores of each category are summed to create an overall total. Higher scores reflect depression severity.	Rush et al., 2003
The Beck Depression Inventory (BDI-II)	21-item self-report scale on depressive symptom severity, with a 4-point (0-3) Likert scale. Scores are summed to give an overall estimate of depression severity.	Beck, 1996
Generalised Anxiety Disorder Questionnaire (GAD-7)	7-item self-report anxiety screen for the four most common anxiety disorders found in primary care (panic, generalised, social, post-traumatic), with a 4-point (0-3) Likert scale. Scores are summed to give an overall estimate of anxiety severity.	Roy-Byrne et al., 2009
The State-Trait Anxiety Inventory (STAI) – Form Y	40-item self-report scale, including 20 items for assessing trait anxiety and 20 for state anxiety, with a 4-point (1-4) Likert scale. Items are summed for state (1-20) and trait (21-40) symptoms to give two total scores.	Spielberger, 1983
<i>Trauma and PTSD measures</i>		
Childhood Trauma Questionnaire (CTQ)	A 28-item, retrospective self-report tool measuring exposure to childhood adversity, with five subscales: physical abuse, sexual abuse, emotional abuse, physical neglect, emotional neglect. Items are rated on a 5-point (1-5) Likert scale and scores are summed for each subscale, and an overall total.	Bernstein et al., 1994
Life Stressor Checklist – Revised (LSC-R)	30-item self-report measure, assessing exposure to traumatic or stressful life events, with a dichotomous (yes/no) primary dimension and follow-up 5-point (1-5) intensity scales measuring subjective, ongoing impact for each item endorsed.	Wolfe et al., 1997
PTSD checklist for DSM-5 (PCL5)	20-item self-report measure of current (past month) PTSD symptoms, corresponding to DSM-5 diagnostic criteria, with a 5-point (0-4) Likert scale. Item scores are summed to create an overall total. A score of 30 or more is used as clinical cut-off.	Blevins et al., 2015
<i>Additional measures and custom questionnaires</i>		
Alcohol, Substance and Smoking Involvement Test (ASSIST)	Self-report screening measure to detect the risk for psychoactive substance. Answers are associated with individual scores, which are totalled by substance. Higher scores indicate higher risk for abuse of that substance and fall within three categories (lower risk, moderate risk, high risk).	WHO ASSIST working group, 2002
CAPS and PDI follow-up questionnaire	Brief follow-up survey to the CAPS and PDI, asking about the duration of perceptual experiences, the frequency of recurrence of delusional thoughts, and any known triggers of these symptoms.	Custom
Stress-check	5-point (1-5) Likert scale assessing current levels of stress (“How stressed do you feel right now?”), alertness (“How alert do you feel right now?”), and feelings of being in control (“How in-control do you feel right now?”)	Custom

3.3 Task measures

In addition to these questionnaire measures, participants of both studies were asked to complete a series of computerised cognitive tasks. The following briefly describes their experimental paradigms and procedures.

3.3.1 The two-tone image task

Two-tone images are high-contrast, black-and-white pictures. They are created through digitally binarization of photographs depicting people or animals, by intensifying brightness thresholds until the original scene is virtually unrecognisable without prior knowledge (“priors”) of the image content. Viewing the original photograph enables subsequent disambiguation of the two-tone image and even a single, brief exposure to the template can lead to a long-term advantage in recognising the underlying picture (Ludmer et al., 2011). This makes two-tone images an ideal instrument for inducing contextual visual priors to assess top-down influences on the processing of ambiguous sensory stimuli (thereby examining the balance between prior information and incoming sensory evidence) and they have now been widely used in this context (Dolan et al., 1997; Teufel et al., 2018).



Figure 3.1

Example of a two-tone image (top) and its coloured template (bottom).

Considering the possible causal role of aberrant prediction in the development of psychotic symptoms (Murray et al., 2008; Fletcher and Frith, 2009), multiple studies have begun to explore two-tone disambiguation capacities in populations with schizotypal traits (Batty et al., 2014) and schizophrenia (Rivolta et al., 2014; Teufel et al., 2015). While inconsistent, findings largely indicate a correlation between psychotic experiences and *decreased*

baseline performance (lower rates of recognising image content without context) (Batty et al., 2014; Rivolta et al., 2014), but an *increased* benefit from prior information (a more pronounced difference in recognition improvement after seeing the template pictures) (Teufel et al., 2015). These observations generally support the theory that cognitive deficits could lead to an imbalance between bottom-up and top-down information processing, causing an overreliance on prior information and thereby driving the development of hallucinations and delusions in schizophrenia (Aleman et al., 2003; Leitman et al., 2010).

The two-tone task employed in the context of this dissertation was developed (and has previously been applied in predictive processing studies) by Davies et al. (2018)¹⁶. It was adapted for online use by Lucie Daniel-Watanabe¹⁷ and was conducted via the experiment platform Gorilla. The task is divided into two-tone blocks (during which participants see the black-and-white images) and template blocks (that show the grayscale image templates). Two-tone blocks are paired as pre-template and post-template trials, which show the same stimuli before and after original image exposure (Figure 3.2). Seeing the template offers participants new prior information with which to make sense of the ambiguous black-and-white patterns and is expected to improve their overall performance. The degree of improvement (i.e. the difference between correctly identified images after versus before template exposure) provides a measure of the relative balance between prior information and sensory information in the context of perceptual decision making (Teufel et al., 2015; Davies et al 2018).

Task procedure

Participants were instructed that they would be presented with black-and-white images, and that these images contain figures (people or animals). They had three seconds to judge (yes/no) whether a red target dot in the picture is on the figure or on the background, by clicking the corresponding button with the cursor. The location of the dot was equidistant from the image centre for both the figure and the background condition. The experiment

¹⁶ A full breakdown of task development and stimuli can be accessed in the original study's supplementary materials, available online (PMCID: PMC6192467)

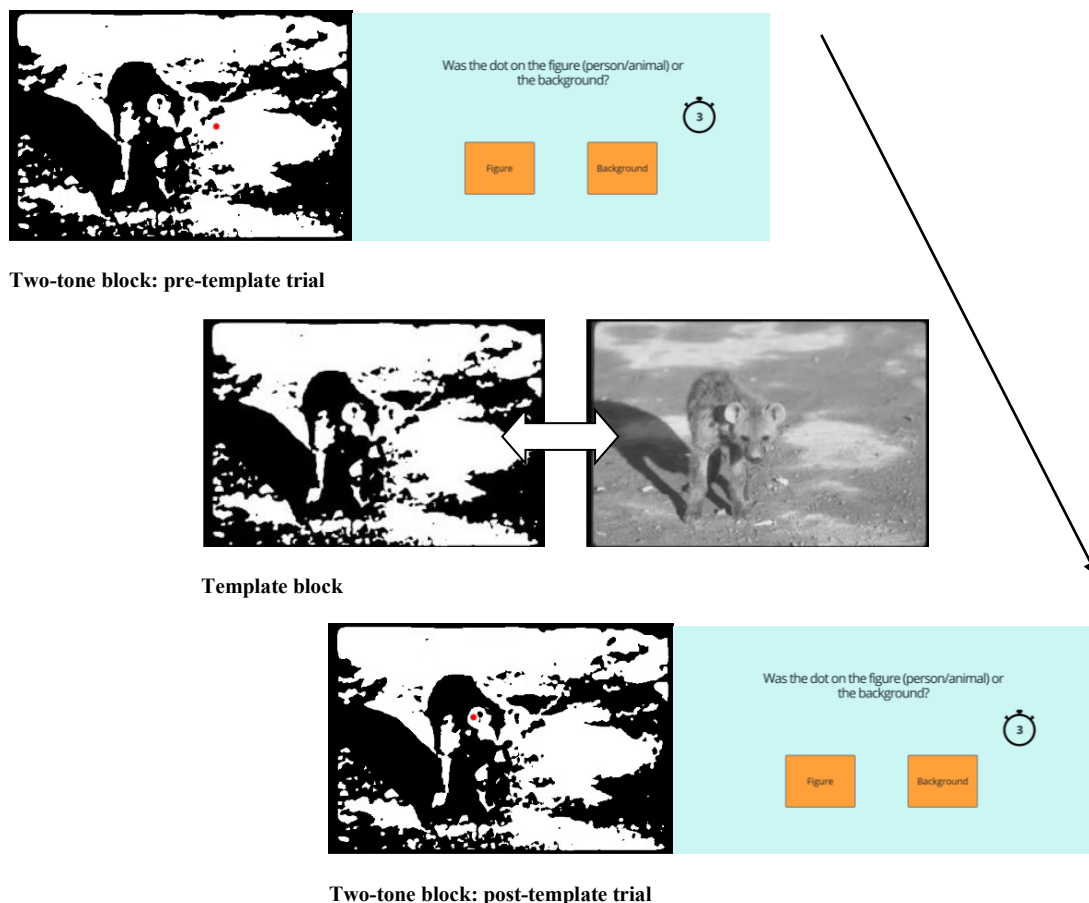
¹⁷ Health Neuroscience Lab, Department of Psychiatry, University of Cambridge

consisted of eight blocks, with three different images per block. Each image was presented four times, twice with the dot on the figure (once on a black patch, once on a white patch), and twice with the dot on the background (also once on a black patch, once on a white patch), totalling twelve stimuli per block.

Stimuli were presented for 1.5 seconds, with the red dot appearing after 200 milliseconds. After responding to all twelve stimuli, participants were asked to carefully observe as the two-tone images cross-faded with their grayscale templates. Each fade lasted for a total of thirteen seconds and morphed twice from two-tone to template. Subsequently, the twelve black-and-white stimuli were presented a second time (in randomised order), again asking participants to judge the location of the red dot. Following each block participants received the message that they were about to progress to the next set of trials.

Figure 3.2

Illustration of the two-tone image task procedure.



Prior to task begin, all participants completed a practice round with two example images. At the end of the experiment, participants were asked if they had faced any problems loading the images (none/some/many), and to indicate whether they had previously performed this task as part of another study, since prior exposure to the image templates may confound the results.

3.3.2 The phantom words task

Phantom word illusions were discovered by psychologist and speech and music researcher Diana Deutsch, who first reported the intriguing sound phenomenon on her 2003 CD *“Phantom Words and Other Curiosities”*¹⁸. They are created by vocoding a two-syllable word and looping it on a mono track. The same sound-loop is then copied onto a second track, but slightly offset in time, so that the first syllable of the first word is played simultaneously with the second syllable of the second word and vice versa. When presented via stereo loudspeaker, the words merge, creating an ambiguous and somewhat eerie sound effect that initially appears meaningless but soon morphs into recognisable words and phrases. As a strange consequence of the illusion, people rarely recognise the true underlying signal but instead hear new words, which (as Deutsch notes) tend to be greatly influenced by their personal experience, expectations, and current state of mind: *“I can guess who is likely to be on a diet, as they report words like ‘I’m hungry’, ‘diet coke’, or ‘feel fat’. And [...] if I play these sounds close to exam time, students hear phrases like ‘I’m tired’, ‘no brain’, or ‘no time’.”* (Deutsch, 2019).

Contextualised as a cognitive task, this phenomenon could perhaps provide a measure of a person’s tendency to experience meaningful speech in structured noise, and to ascribe emotional weight to the experience. Since the emotional valence of psychotic experiences (especially hearing voices) has been postulated as a possible driving factor for clinically significant auditory hallucinations in BPD, phantom word illusions provide fruitful ground

¹⁸ Examples of Diana Deutsch’s original phantom word illusions can be found on her website under www.deutsch.ucsd.edu.

for assessing the affective processing of ambiguous auditory stimuli and the novel phantom words task was developed accordingly.

Development of the task

Loosely modelled on visual emotion recognition tasks that capture sensitivity to ambiguous facial expressions and potential bias to perceive them positively or negatively, the aim in developing the phantom words task was to determine whether similar biases may influence the perception (and meaning) of abstruse sounds. In line with Deutsch's observations of predominantly egocentric and current-state driven interpretations of the words, and Westen's hypothesis on the borderline attributional style (Westen, 1991), one would expect people with BPD to exhibit an increased tendency to ascribe negative meaning to these illusions. With this in mind, the objective of the phantom words task was to assess the following:

1. Whether people with BPD are more likely to ascribe negative meaning to ambiguous auditory stimuli than control participants and,
2. If yes, whether this tendency is strong enough to supersede correct identification of a true signal (i.e., hearing a negative word even when the underlying stimulus is positive or neutral).

The following briefly describes the task's development. Supplementary materials can be found in Appendix B.

Emotional valence survey

Using the *Affective Norms for English Words* (Bradley and Lang, 1999), fifty-nine two-syllable word-pairs (107 words) that share a sound basis (i.e., rhyme) were identified. These words were then re-assessed for their emotional valence in a brief survey, in which participants were asked to rate how the words make them feel on a nine-point sliding scale, ranging from very unhappy (1) to very happy (9). The rating scale was adapted from the Self-Assessment Manikin (SAM) affective valence scale employed by Bradley and Lang in 1999. The aim of the pilot was to uncover any significant changes in rating between the original development sample and a contemporary one. In total, 162 participants completed

the questionnaire on the experiment platform Qualtrics (for demographics, see Appendix B.1).

Words with an average rating above six were categorised as *positive*, words with an average rating below four as *negative*, and those between four and six as *neutral* (for examples, see Appendix B.2). A standard deviation cut-off of 2.0 was used to determine consensus and words with an SD above this threshold were excluded from further task development (for examples, see Appendix B.3). The remaining words were matched phonetically to create *positive/negative*, as well as *positive/neutral*, and *negative/neutral* sound-pairs. Overall, this resulted in forty-six word-pairs for which sound files were created in the style of Diana Deutsch's phantom word illusions. Twenty-two audio clips had a *true signal*, meaning that one word in each pair was used to create the sound file (e.g., the phantom word for the only/lonely pair contained the true signal "only"), while the other twenty-four had a *non-signal*. Here, the shared phonetic basis of a pair was used to create the sound, however neither word was fully represented in the audio (e.g., the phantom word of the furious/curious pair used the ambiguous signal "urious"). The minimum difference between average ratings of non-signal word pairs was set at 2.5 to ensure a sufficient gap in emotional valence. Non-signals were intended to measure an emotional bias towards either positive or negative interpretation of ambiguous auditory stimuli, whereas true signals were meant to assess whether such a bias (if it did exist) could persist in the face of an objectively correct alternative option.

Phantom words pilots 1 and 2

Thirty-one volunteers took part in a preliminary pilot of the task on the experiment platform Gorilla. Participants were asked to listen to the audio of each of the forty-six phantom word pairs and were asked (forced choice) which sound they heard. They were also given a third ("Neither") option to indicate that they could not identify either word. The average reaction time was 4009ms for selecting a word option and 6985ms for selecting *Neither*. Pairs for which more than 90% of participants could not identify a stimulus, as well as non-signal pairs for which more than 90% of participants perceived the same word, were excluded from the task. This preserved thirty-four word-pairs for which both words were either

identified at equal rates (inclusion criterion for non-signal pairs), or predominantly correctly identified (inclusion criterion for true-signal pairs).

These remaining phantom words were tested during in a second pilot, which abolished the *Neither* response option so that participants had to decide between the two words presented. A further twenty volunteers participated in the testing. The average reaction time was 4002ms. Previous inclusion and exclusion criteria for word selection were retained. Additionally, pairs were excluded if their average reaction time was >7000ms, using the “*Neither*” reaction time from the first pilot as cut-off. This ultimately resulted in a selection of twenty word-pairs (eleven non-signals, nine true signals) which were subsequently used in the final version of the phantom words task (Figure 3.3, Appendix B.4).

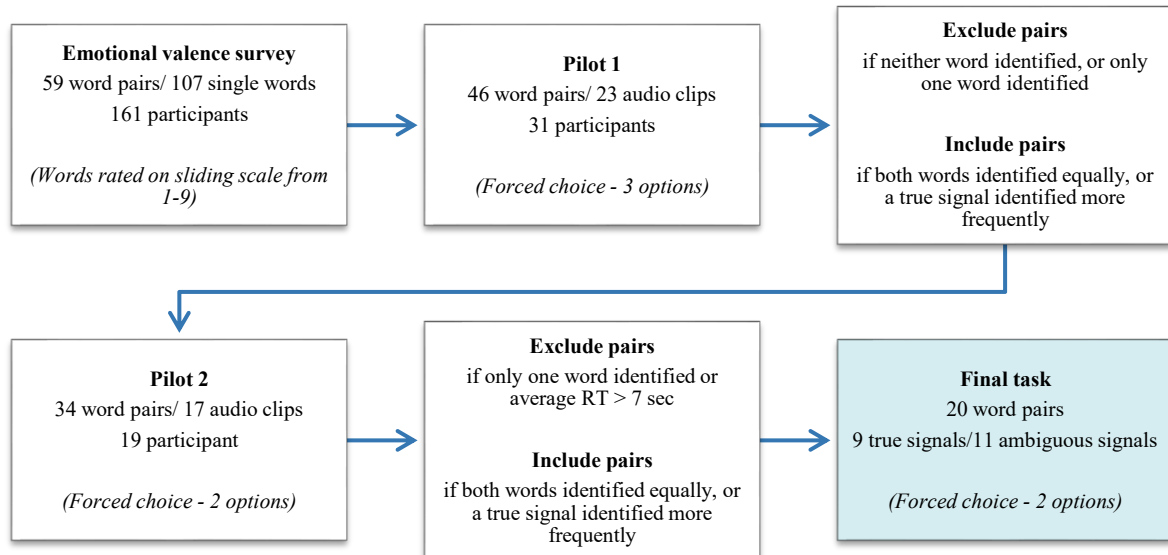


Figure 3.3

Illustration of the procedure of developing the phantom words task.

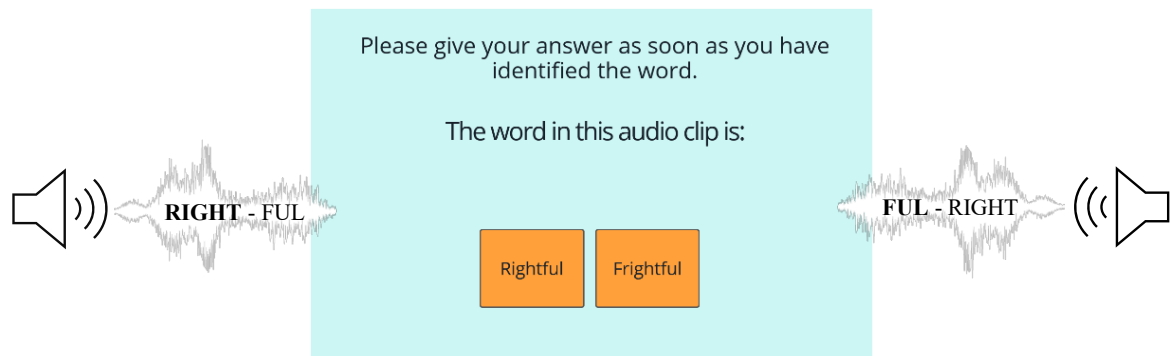


Figure 3.4

Illustration of the phantom words task experimental procedure.

Task procedure

Participants were instructed that they would be presented with a series of audio clips, and that they would be given a choice of two words and asked to identify which of them is said in the sound clip. They were told that both their accuracy and their speed would be recorded and were asked to make their decision as quickly as possible. They were also advised to not use headphones, and to ensure that their speakers were on and at a comfortable volume. Participants then completed a brief practice round with five examples of phantom words, which mirrored the structure of the task. The experiment consisted of twenty trials (nine true-signal, eleven non-signal), presented consecutively and in randomised order. Answer-button selection triggered progression, with a fixation cross (displayed for 250ms) between trials. Sound files started playing automatically after 100ms, for a maximum of thirty seconds, following which the trial was recorded as *timeout* and the participant was moved onto the next phantom word.

3.3.3 The stress induction task

Owing to the considerable theoretical interest in (but scarce empirical evidence for) the influence of stress on psychotic experiences in BPD, a stress induction procedure was central to the second study's experimental paradigm. Such experiments have long played a crucial role in evaluating and disentangling the nuances of the human stress response,

and a suitable range of physiological and psychological stressors of varying intensities (and varying ethical considerations) are habitually used in imaging studies (Dedovic et al., 2005; Quaedflieg et al., 2013), physiological studies (Nater et al., 2006; Fleischhauer et al., 2023), and assessments of their effect on psychopathology (Petrowski et al., 2010; Monteleone et al., 2012). In the context of BPD, acute stress inductions have predominantly been performed to investigate changes in social cognition and emotion processing (Deckers et al., 2015; Graumann et al., 2021).

One of the best validated and most reliable inductions of moderate psychological stress in laboratory settings is the Trier Social Stress Test (TSST, Kirschbaum et al., 1993), which involves a combination of stress anticipation and stress exposure through public speaking and mental arithmetic. While undeniably effective in raising both subjective and objective (cortisol and heart rate) measures of stress, the TSST is highly time and resource consuming and often not feasible within the ethical, financial, and practical constraints of a study. As such, many researchers (Dedovic et al., 2005) resort to modified versions, which typically retain the mental arithmetic component of the test while omitting the more complex social speaking part. The task that was presented to participants in Study 2 also followed this approach. It was adapted from Westwater et al. (2021), preserving the original stimuli but reprogramming their aesthetic and structural presentation¹⁹. The task was coded in Gorilla.

Task procedure

Participants were informed that they would be tested on their overall cognitive abilities, which would require them to solve mathematical problems. They were instructed to answer as quickly and accurately as possible, as both measures would establish their overall score. Participants were further told that their performance would be judged against an expected, standardised average obtained during piloting and that this was meant to prevent them from selecting answers randomly and would give the researchers higher confidence in their results. They were cautioned that if they were to fall more than 10% below this average, their data could not be used, and they might forfeit payment.

¹⁹ In its original iteration the stress induction involved mild electric shocks (in addition to the mental arithmetic), as well as a control task. Both were omitted in the current study.

Participants then complete a practice round of twenty-five equations, including ten easy questions, nine moderately difficult questions, and six difficult questions. Each equation had three answer options, one of which was correct. The button order was randomised between trials. Equations and answer options were displayed after 100ms, for a maximum of fifteen seconds. A pulsing countdown was displayed at the bottom of the screen for the five final seconds, after which participants received the warning “*Too slow!*” (displayed for 1000ms) before advancing to the next question. Response selection triggered progression, with a fixation cross (250ms) between trials. Positive feedback (a small green checkmark) was given for correct responses, and negative feedback (a small red cross) for incorrect responses. Both feedback symbols were displayed in the same location (centre of screen), at equal size and duration (500ms). At the end of the trial block participants were informed that they had now completed the practice round and were presented with their overall total, as well as a fictitious group score. This group score was personalised, and was calculated by summing only the scores of the *easy* and *moderate* questions (participants’ own scores additionally included their performance on hard questions), typically causing it to appear slightly higher. Participants were also reminded to make sure to score within 10% of the group average during the following test round.

The test round retained the overall task structure of the practice, however, the maximum time allowed per equation was adjusted based on participants’ individual performance and assigned as follows:

- Practice score <5: *Very Easy*, 15 sec, (no change)
- Practice score <10: *Easy*, 12.5 sec (-2.5)
- Practice score <15: *Low difficulty*, 10 sec (-5)
- Practice score <20: *High difficulty*, 7.5 sec (-7.5)
- Practice score <25: *Hard*, 5 sec (-10)
- Practice score =25: *Extreme*, 3 sec (-12)

The experiment consisted of two blocks with twenty-five trials per block. In between blocks, participants were presented with their current score and correspondingly calculated

(factitious) group average. At the end of the task, participants were shown their overall total score, but not the group average.

The stress induction was used in conjunction with a brief subjective assessment, asking participants to rate (1-5) their current level of stress immediately prior to and following the task.

3.4 Design and experiment procedure – Study 1

3.4.1 Study design and recruitment

Study 1 was initially conceived as an in-person assessment, aiming to recruit BPD participants from the NHS Cambridgeshire and Peterborough Personality Disorder Community Service (PDCS), however the COVID-19 pandemic put drastic limitations on the study's design and target population. It was subsequently adapted into an online experiment during which BPD and control participants completed a series questionnaires and cognitive tasks on established digital research platforms. Study protocols were approved by the University of Cambridge Psychology Ethics Committee (PsychREC), and all participants gave digital informed consent.

Participants between the ages of 18-65 were recruited internationally, through advertisements distributed via email to BPD support groups and experts-by-experience mailing lists, social media, mental health charity websites, existing departmental databases, and word of mouth. Individuals interested in participating were asked to complete a screening questionnaire on the survey platform Qualtrics to determine eligibility.

BPD participants were included if they self-reported a medical diagnosis of borderline personality disorder/emotionally unstable personality disorder and met the clinical cut-off on the McLean screening instrument for BPD.

Control participants were included if they self-reported no history of significant psychiatric illness requiring treatment. Volunteers from both groups were excluded if they reported the presence of a neurological disorder affecting brain functioning, or a history of serious

head injury. BPD participants were also excluded if they reported a current clinical diagnosis of psychotic disorder, including schizophrenia, schizoaffective disorder, or bipolar disorder.

3.4.2 Materials and study procedure

During the screening, participants were presented with a general demographic assessment, as well as the CAPE-15. People with a self-reported diagnosis of BPD also completed the MSI-BPD, as well as questions about their current and previous treatment and psychiatric comorbidities. The complete screening questionnaire can be found in Appendix A.1.1.

Following the screening, participants were sent a link and a unique participant ID to log on to the experiment platform Gorilla. For more detailed information on the questionnaires and tasks involved in this study please refer to Chapter 3.2 (questionnaire measures) and Chapter 3.3 (task measures).

Briefly, psychotic experiences were assessed using the CAPS and PDI, including a custom questionnaire about additional aspects of these symptoms. Psychological measures were collected using the QIDS for depressive symptoms, the GAD-7 for anxiety, and the DES-II for dissociation. Experiences of childhood adversity and current post-traumatic stress were measured with the CTQ and PCL-5 respectively. Further instruments included the AQ-10 (as an assessment of autism spectrum traits), as well as the ASSIST (to control for psychoactive substance use).

In addition to the questionnaire measures, participants were asked to complete a series of tasks that concern interpreting sensory information (visual and auditory). These included the two-tone image task, which assesses the balance between prior information and sensory evidence thereby specifically investigating the predictive processing model of psychosis in BPD, and the phantom words task, a novel task that aims to identify a tendency to attribute emotional valence to ambiguous stimuli. The study session lasted on average between sixty and ninety minutes. For a copy of the experiment tree including the order of tasks and questionnaires see Appendix C.1.

3.4.3 Participants

Screening population

The screening questionnaire for Study 1 was completed by 211 volunteers. Main exclusion criteria were programmed directly into Qualtrics, however after manual verification of eligibility a further twenty-three participants were excluded from analysis: Four BPD-group participants who did not meet the MSI-BPD clinical cut-off, seventeen BPD-group participants who reported a comorbid diagnosis of bipolar disorder and/or schizophrenia, and two controls with a history of Psychotic Disorder Not Otherwise Specified. Thirty-six BPD participants reported taking antidepressants at the time of data collection, fourteen people were receiving antipsychotic tablets, and eleven sedatives. Seven participants had been prescribed mood stabilisers, and five ADHD medication. None of the control group volunteers reported psychoactive substance treatment. As it is unclear whether or how antipsychotic medication might affect some of the neuro-cognitive mechanisms under investigation, participants currently undergoing antipsychotic treatment were rejected from the main study. However, their data was included in the analysis of the Screening survey with the aim to assess PE prevalence in a representative sample of individuals with BPD and psychosis.

Table 3.2

Population demographics and comorbidities of the participant population for the Qualtrics (screening) survey of Study 1. ED=Eating Disorders, OCD=Obsessive-compulsive disorder, PTSD=post-traumatic stress disorder, ASC=Autism spectrum condition, f=female, m=male, d=diverse (other gender identity). Diagnostic status (self-report) of autism was only assessed the second study and is therefore denoted as NA.

	Age mean (sd)	Age range	Gender (f:m:d)	Anxiety (%)	Depression (%)	ED (%)	OCD (%)	PTSD (%)	ASC (%)
BPD (N=94)	27.87 (9.16)	18-64	67:20:7	32 (34.0)	36 (56.3)	13 (13.8)	6 (6.4)	13 (13.8)	NA
Control (N=94)	27.26 (5.89)	18-58	65:27:2	15 (16.0)	16 (17.0)	6 (6.3)	NA	NA	NA

Study population

Of the 211 volunteers who completed the screening, 174 met all inclusion criteria and were invited to participate in the main study on the experiment platform Gorilla. In addition to the twenty-three participants excluded for reasons described above, a further fourteen people from the BPD group were prevented from taking part due to current use of antipsychotic medication. Thirty-three people (19.5%) withdrew pre-study, with a further sixteen (9.2%) terminating their testing session before its conclusion. Due to the delicate nature of ensuring continuing consent in an anonymous, digital environment, non-completion was interpreted as wishing to withdraw and partially complete datasets were discarded. In total, 124 participants completed the study, including fifty-four people with BPD and seventy controls.

Groups were well matched for age and gender. Twenty of the BPD participants stated that they were currently taking antidepressants, nine people were treated with mood stabilisers, and four took sedatives. None of the control group participants reported medicinal treatment. Participants demographics and current diagnoses of psychiatric comorbidities are summarised in Table 3.3. Additionally, four BPD participants reported a current diagnosis of *Any Other Mental Health Condition(s)*, including ADHD ($n=3$), cPTSD ($n=1$), and dissociative disorder ($n=1$). One control participant also disclosed an ADHD diagnosis.

The substance involvement screening test (ASSIST) revealed higher rates of alcohol ($t_{89}=2.65$, $p<.01$), tobacco ($t_{70}=4.24$, $p<.001$), and cannabis ($t_{91}=2.37$, $p=.01$) use in the BPD group, however average scores remained in the low-risk range, indicating occasional use. All other substances (including hallucinogens, cocaine, sedatives, amphetamines, and opioids) had a median score of zero in both groups, and no repeated outliers (i.e., no participant fell outside the median for all substances).

Both groups were screened for outliers using interquartile range (IQR) and z-score methods, as well as Grubbs' test. Two controls were identified as scoring consistently outside the expected range on each of the main psychopathology measures (CAPS, PDI, DES-II, QIDS, CTQ, and PCL-5) and their data was excluded from further analysis.

Table 3.3

Population demographics and comorbidities of the participant population for the experiment session of Study 1. ED=Eating Disorders, OCD=Obsessive-compulsive disorder, PTSD=post-traumatic stress disorder, ASC=Autism spectrum condition, f=female, m=male, d=diverse (other gender identity). Assessment of autism spectrum conditions was only implemented during the second study and is therefore denoted as NA.

	Age mean (<i>sd</i>)	Age range	Gender (f:m:d)	Anxiety (%)	Depression (%)	ED (%)	OCD (%)	PTSD (%)	ASC (%)
BPD (<i>N</i> =54)	27.91 (9.10)	18-64	36:15:3	16 (30%)	21 (39%)	7 (13%)	5 (9%)	6 (11%)	NA
Control (<i>N</i> =68)	26.13 (5.10)	18-56	48:19:1	2 (3%)	2 (3%)	0 (0%)	NA	NA	NA

3.5 Design and experiment procedure – Study 2

3.5.1 Study design and recruitment

Stress is often cited as a precipitant and potential trigger of psychotic experiences in BPD, with much anecdotal evidence from case studies and patient testimony. However, to date only few studies have investigated the relationship.

The purpose of the second study was therefore to further the understanding of whether and how the burden of lifetime stressors and acutely stressful situations might influence susceptibility to psychotic symptoms in people with BPD, under consideration of a possible link to dissociative states. Specifically, the aim was to answer the following questions:

1. Does the number of life stressors experienced and/or their ongoing subjective impact predict an increased risk for psychotic experiences (i.e., can the findings of Niemantsverdriet et al., 2017 be replicated?)
2. Is the severity of dissociative and psychotic state symptoms influenced by an acutely (psychosocially) stressful experience and, if so, are these symptoms correlated?
3. Are the neurocognitive mechanisms hypothesised to underlie (some) psychotic experiences (specifically, predictive processing mechanisms) altered by stress?

Moreover, the second study offered the opportunity to further explore and confirm the findings of Study 1 in a population with a confirmed clinical diagnosis of BPD.

In addition to the psychopathological questionnaires and two-tone image task previously utilised in the online study, Study 2 also assessed participants' exposure to significant life-stressors, as well as their cognitive and emotional responses to an acute social stressor – with a particular focus on psychotic reactivity and dissociative traits. Study protocols were approved by PsychREC, and all participants gave written informed consent. Data was collected during an in-person testing session at the Department of Psychiatry, University of Cambridge.

Participants between the ages of 18-65 were recruited from the Cambridgeshire and Peterborough area, with advertisements distributed via email, social media, printed ads, and word of mouth. Individuals interested in taking part were asked to complete a screening questionnaire on Qualtrics, to determine eligibility. BPD participants were included if they had a current medical diagnosis of borderline personality disorder/emotionally unstable personality disorder and were asked to bring confirmation of their diagnosis to the study session. Control participants were matched with the BPD group for age and gender and were included if they reported no history of significant psychiatric illness requiring treatment (not including subclinical depression or anxiety). Volunteers from both groups were excluded if they reported a neurological disorder affecting brain functioning, or a history of serious head injury. BPD participants were also excluded if they reported a current clinical diagnosis of psychotic disorder, including schizophrenia, schizoaffective disorder, or bipolar disorder, or if they were currently treated with antipsychotic medication.

3.5.2 Materials and study procedure

Before being invited to the testing sessions, participants were asked to complete a short online screening to determine their eligibility.

The screening questionnaire can be found in Appendix A.1.2 and included the following:

- Demographics (age, gender, residence, diagnosis status)
- Psychotic symptom screen (CAPE-15)
- Details of current care and treatment (medications and/or psychological care)
- Exclusion criteria

People who were eligible to participate were then contacted by email to schedule a study session at the Department of Psychiatry, University of Cambridge.

At the start of the study, participants were presented with a series of ‘pre stress’ measures. These included assessments for current anxiety (STAI), dissociative (CADSS) and psychosis-like experiences (PSI), as well as the two-tone image task and an emotion processing task. Next, participants completed a series of questionnaires (for more information on the questionnaires and tasks involved please refer to Chapter 3.2 and 3.3). Psychotic experiences were assessed using the CAPS and PDI, including custom additions on the subjective experience of these symptoms. Additional psychological measures were collected using the BDI for depressive symptoms, the DEMO for dissociation, and the BSL-23 for BPD symptom severity. Experiences of childhood adversity, exposure to major life-stressors, and current post-traumatic stress were measured with the CTQ, LSC-R, and PCL-5 respectively.

Subsequently, participants took part in a computerised, acute stress induction paradigm involving mental arithmetic. Adapted from Westwater et al. (2021) this paradigm is a modification of the Montreal Imaging Stress Task (Dedovic et al., 2005), which in turn was based on the Trier Social Stress Test (Kirschbaum et al., 1993). Participants were instructed that the next task would be a cognitive task that will involve solving mathematical problems under time pressure and that they will be judged against a group average (for more details on the stress induction paradigm, see Chapter 3.3.3). Subjective stress levels were assessed immediately prior to and following the induction, as well as at four other points throughout the study (see Appendix C.2 for information on the order in which questionnaires and task measures were presented).

In the final part of the study, participants were again presented with the ‘pre stress’ measures (questionnaires and tasks) to assess any changes in their levels of anxiety, dissociation, or psychotic experiences, as well as perceptual (predictive) processing capacity.

3.5.3 Participants

Screening population

Screening for the second study was completed by forty-four volunteers with BPD and ninety-two control participants. All inclusion criteria were programmed directly into Qualtrics, and manual verification of eligibility returned no further exclusions. Participant demographics and comorbidities are summarised in Table 3.4. Additionally, three people reported a current diagnosis of ADHD (BPD=2, control=1).

Table 3.4

Population demographics and comorbidities of the participant population from the Qualtrics (screening) survey of Study 2. ED=Eating Disorders, OCD=Obsessive-compulsive disorder, PTSD=post-traumatic stress disorder, ASC=Autism spectrum condition, f=female, m=male, d=diverse (other gender identity).

	Age mean (<i>sd</i>)	Age range	Gender (f:m:d)	Anxiety (%)	Depression (%)	ED (%)	OCD (%)	PTSD (%)	ASC (%)
BPD (<i>N</i> =44)	31.18 (10.97)	18-54	34:3:7	24 (54.5)	23 (52.3)	13 (29.5)	6 (13.6)	6 (13.6)	4 (9%)
Control (<i>N</i> =92)	32.24 (12.60)	18-65	71:18:3	14 (14.9)	16 (17.0)	3 (3.3)	NA	NA	0 (0)

Study population

Following screening, all forty-four BPD volunteers and thirty-nine controls (matched to the BPD group for age and gender) were invited to the in-person testing session at the Department of Psychiatry, University of Cambridge. Fifty people, including twenty

individuals with a clinical diagnosis of BPD, took part in the study. Participant demographics and psychiatric comorbidities are summarised in Table 3.5. At the time of testing, thirteen BPD participants were taking antidepressants, two were receiving benzodiazepines, four mood stabilisers, and one person was treated with ADHD medication. Three control group volunteers were also taking antidepressant medication. Three of the participants with BPD reported infrequent cannabis use (around once a month), and one person reported weekly consumption. The data of one control group participant was removed from analysis after being identified as an outlier on all the main psychopathology measures (CAPS, PDI, MSI-BPD, DEMO, BDI, CTQ, PCL, and LSC-R), using IQR and z-score methods, and Grubb's test.

Table 3.5

Population demographics and comorbidities of the participant population for the experiment session of Study 2. ED=Eating Disorders, OCD=Obsessive-compulsive disorder, PTSD=post-traumatic stress disorder, ASC=Autism spectrum condition, f=female, m=male, d=diverse (other gender identity).

	Age mean (<i>sd</i>)	Age range	Gender (f:m:d)	Anxiety (%)	Depression (%)	ED (%)	OCD (%)	PTSD (%)	ASC (%)
BPD (<i>N</i> =20)	30.2 (10.12)	18-54	18:1:1	11 (55%)	11 (55%)	6 (30%)	4 (20%)	3 (15%)	4 (20%)
Control (<i>N</i> =29)	33.3 (12.23)	21-61	27:2:1	2 (6%)	3 (10%)	1 (3%)	NA	NA	0 (0%)

3.6 The grand scheme of things

To address the principal questions and aims of this dissertation, data from the screening questionnaires was combined to estimate the overall prevalence of psychotic experiences in the wider (BPD) population, and to compare people with low and high psychotic experience scores with regard to the types of symptoms they endorsed, their comorbidities, and reports of self-harm and suicide attempts (Chapter 4). The range of psychotic

experiences (including perceptual abnormalities as well as delusional beliefs, their severity and frequency) were assessed using the PE symptom measures (CAPS and PDI) of both studies, comparing people with BPD to controls, as well as participants of Study 1 (an online self-report sample) to participants of Study 2 (an in-person sample with a verified clinical diagnosis), thereby endeavouring an instant replication of the research findings (Chapter 5). The relationship between borderline psychopathology, psychotic symptoms, and known risk factors for psychosis was also assessed within and between participant populations, using the questionnaire and task data from both studies (Chapter 6). Finally, the remaining data obtained during Study 2 (specifically, the repeat-measure tasks and questionnaires that assessed pre versus post stress symptomology) was analysed to investigate the impact of stress on psychotic experiences in BPD (Chapter 7). Figure 3.5 offers an illustration of how the different study populations inform the ensuing chapters.

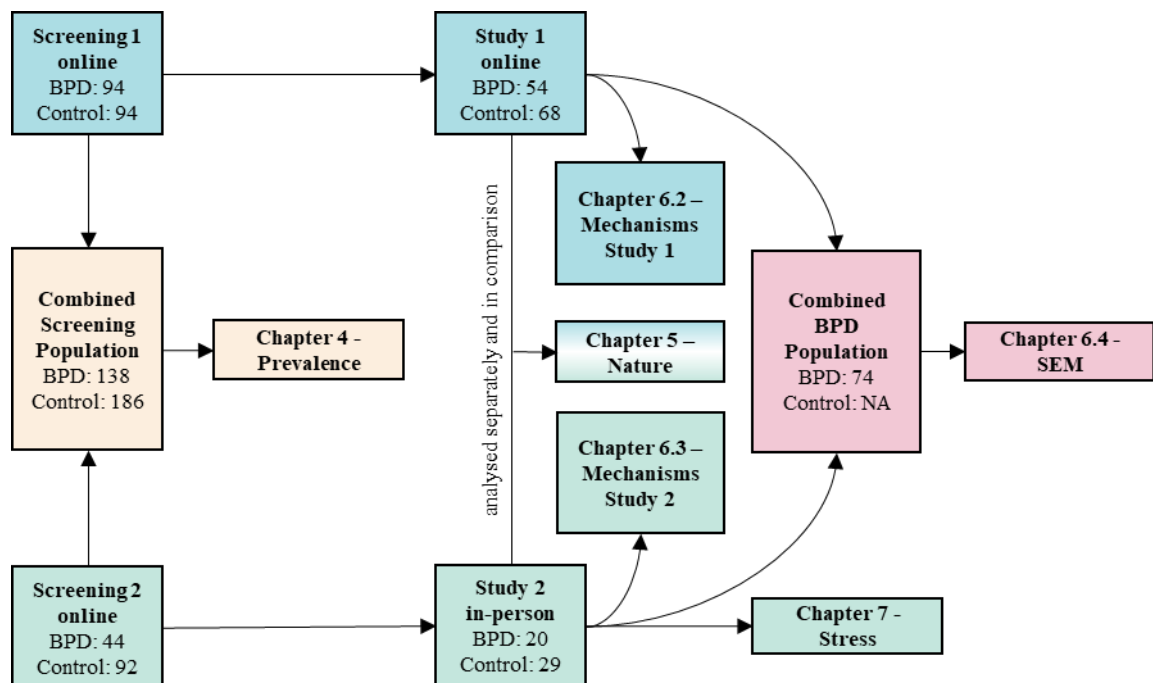


Figure 3.5

Illustration of the different study populations (separate and in combination) and their corresponding chapters.

Chapter 4 (Don't) agree to disagree – prevalence of psychotic experiences

“I struggle to cope with these symptoms and experience and am afraid to talk about them with other people, which often makes them worse. I get frustrated if people tell me it's not true when I know it is.”

4.1 Introduction

A fundamental challenge of determining replicable PE prevalence estimates lies in the selection of a validated, reliable, and adequately suited instrument. With no measures specifically designed to capture psychotic symptoms in the context of BPD, previous studies have employed a wide range of questionnaires, clinical interviews, and at times patient medical records (e.g., Miller et al., 1993; Adam and Sanders, 2011). While structured clinical interviews (which are frequently used to assess psychotic symptoms in BPD) can paint a more in-depth and qualitative picture of the experience, they are limited by their partial subjectivity, and their samples are often small and not generalisable. The (revised) diagnostic interview for borderlines (DIB-R; Zanarini et al., 1989) is a particularly biased assessment, as the assumption that psychosis in BPD is inherently different and less “true” than in other disorders is built into its very structure. The DIB’s cognition section (which is used to measure perceptual symptoms and unusual beliefs) explicitly uses the terminology “quasipsychotic” (summary statement 8) and “nondelusional” (summary statement 7) to evaluate experiences. Moreover, four items concerning psychotic symptoms associated with schizophrenia and/or bipolar disorder (including mania, widespread/persistent hallucinations, and nihilistic, grandiose, or bizarre delusions) were specifically excluded during the instrument’s revision in 1989 thereby further restricting the types of experiences considered relevant in the context of BPD. Nevertheless, the DIB has repeatedly been used to examine PEs in BPD (Zanarini et al.,

1990; Nishizono-Maher et al., 1993; Zanarini et al., 2013; Oliva et al., 2014), which simply serves to further highlight its potential issues, since the reported symptoms and prevalence rates differ wildly between these studies.

By contrast, (self-report) questionnaire measures are not as qualitatively detailed and often cover a broader range of experiences. However, they provide a quantifiable measure of both prevalence and symptom severity in a way that may be more readily reproducible and comparable across diagnostic thresholds. When exploring hallucinations and delusions in BPD, community assessments of psychotic ideation are of particular interest. These questionnaires generally evaluate symptom frequency and severity along a continuum: from unusual but relatively common to severely distressing and potentially pathological. This structure lends itself well to assessing the full range of psychotic experiences in people with BPD, as well as comparing it to the prevalence of sub-clinical symptoms expected to be found in the general population.

4.1.1 Assessing prevalence and severity

The Community Assessment of Psychic Experiences (CAPE-P15) was originally designed to measure the range and severity of psychotic experiences in the general population, with the aim of assessing sub-threshold symptoms in adolescents as predictors for adult-life psychiatric disorders (Capra et al., 2017). In its original version the CAPE screened for lifetime prevalence, whereas the “current” version asks participant specifically about the past three months, thereby providing a more recent measure of psychotic symptoms. Each item is assessed for its frequency and associated distress, and experiences are grouped into three subscales (persecutory ideation, bizarre experiences, and perceptual abnormalities). While the CAPE was primarily conceived as a severity measure, Bukenaitė et al. (2017) explored its potential as a screening tool, to identify people at ultra-high risk for psychosis (UHR) in clinical settings. They postulated a mean-weighted frequency score cut-off of 1.47²⁰ (77% sensitivity, 58% specificity), based on a sample of 165 participants between

²⁰ Using a mean-weighted score that divides the total (0-45) by the number of items (21).

the ages of thirteen and eighteen (Bukenaitė et al., 2017).²¹ This threshold has now been widely used for studies on psychotic experiences in the general population (Núñez et al., 2021; Rozental et al., 2022), in people with common mental disorders (Knight et al., 2020b); and in people in UHR states (Pena-Garijo et al., 2023). Therefore, applying it to a BPD population may provide a reliable estimate of the prevalence of clinically significant psychotic experiences in people with this diagnosis.

Overall, these features make the CAPE-15 an ideal measure for exploring psychotic symptoms in BPD, as it allows comprehensive assessment of the multiple dimensions that are of interest for this thesis:

1. Are psychotic experiences more frequent in BPD than in the general population?
2. Are psychotic experiences in BPD more clinically relevant than in the general population?
3. Are psychotic experiences perceived as more distressing to people with BPD than to the general population?
4. Do these observations differ depending on the type of psychotic symptom?

Furthermore, the clinical cut-off additionally offered the chance to explore differences between BPD participants above and below the significance threshold. Since the presence of psychotic symptoms has been linked to an increase in self-harm (Cavelti et al., 2019), a higher risk of suicide (Kelleher et al., 2017; Slotema et al., 2017), and greater borderline symptom severity (Baryshnikov et al., 2018), the following questions were also explored:

1. Do people above the clinical threshold have higher BPD symptom scores?
2. Are people above the clinical threshold more likely to report incidences of self-harm and/or suicide attempts?
3. Do the types of psychotic symptoms experienced differ between people above and below the clinical threshold?

²¹ The same research group later reassessed the cut-off in an adult population of 1131 individuals with common mental distress (mean age = 39.7 years), and subsequently suggested a revised the score of 1.3 (Knight et al, 2020a) to capture more people with potentially clinical symptoms. However, they maintained that this should not replace the original cut-off but simply provide an additional, more sensitive assessment.

4.1.2 Data analysis

To address the above questions, this chapter draws on the Screening data. See Chapter 3.2.1 for details on the questionnaires, inclusion and exclusion criteria, and participant demographics.

Since both screening questionnaires employed the same questions (including the CAPE-15 as a measure of psychotic experiences), assigned group (BPD versus Control) identity based on self-report, and were collected via the same research platform (Qualtrics), their data were combined for the prevalence analysis. The screening step did not collect any identifiable information and it is unknown whether the BPD or control populations overlapped. However, Study 2 was advertised locally in the Cambridgeshire area whereas Study 1 comprised a much larger sample that was recruited online and internationally, and an automated scan for duplicate IP addresses did not suggest substantial overlap between the samples.

Potential differences in CAPE-15 scores between BPD participants and controls were assessed with independent two-sample t-tests. Where equal variance could not be assumed, Welch's test was employed. The BPD sample was subsequently split into two groups (high-PE and low-PE) using the CAPE's clinical cut-off of 1.47 (Bukenaitė et al., 2017). BPD subgroups were compared on CAPE-15 subscales and symptoms, on their relative (MSI) BPD score, comorbidities, and lifetime prevalence of self-harm and suicide attempts. MSI-BPD items 2.1 and 2.2²² were used as a measure of incidences of self-harm and attempted suicide (respectively) and while such a binary measure is of course unable to consider event frequency or severity, it may still offer a useful comparison of the overall (group-level) prevalence or self-harming and suicidal behaviour. Differences in the number of symptoms and symptom-type prevalence were analysed using Pearson's Chi-squared tests with Yates' continuity correction. *P* values of all serial comparisons were adjusted with the Bonferroni correction. Since Study 2 involved a visual confirmation of participants' diagnosis, as well as a BPD symptom severity measure (BSL), the MSI-BPD

²² As previously explained in Chapter 4 the MSI-BPD item 2 was split into two separate questions to obtain a more detailed assessment.

was not included in Screening 2. Therefore, the results pertaining to this questionnaire (Figure 4.4) were obtained from Screening 1 only ($N=188$, BPD=94, Control=94). Distribution of the data was assessed visually (histograms and Q-Q plots) and using Kolmogorov-Smirnov normality tests with Lilliefors correction. Non-normally distributed datasets are reported with the Shapiro-Wilk statistic. Unless otherwise stated, approximately normal distribution of the relevant measures was observed. All analyses and graphs were performed in R.

4.2 Results

A total data sample of 138 BPD and 186 control participants was analysed, including ninety-four people with BPD and ninety-four controls from the screening of Study 1 (Table 3.2), as well as forty-four people with BPD and ninety-two controls from the screening of Study 2 (Table 3.4).

4.2.1 Main analysis: Psychotic experiences in BPD & control participants

Mean CAPE-15 scores differed significantly (at a level of $p<.001$, using Bonferroni correction) between BPD participants and controls on frequency total, distress total, and across all three subscales (Table 4.1).

97.8% of the BPD population (135/138) endorsed one or more CAPE symptoms, compared to 55.9% (104/186) of controls. BPD participants on average endorsed more items (Figure 4.1) and experienced these more frequently (based on the CAPE frequency score, Figure 4.2, left). Additionally, the BPD group perceived the symptoms as considerably more distressing, a difference that remained significant even when controlling for the number of items endorsed (Figure 4.2, right). When considering the mean-weighted frequency threshold (>1.47) as an indication for clinically relevant psychotic experiences (Bukenaite et al., 2017), ninety-one BPD participants (65.9%) scored above this cut-off, indicating that around two-thirds of the BPD sample experience psychotic symptoms that are persistent, distressing, and comparable in severity to clinical populations with psychosis. Twenty-one of the controls (12.5%) also crossed the significance threshold.

Table 4.1

CAPE symptom scores for BPD and control participants for the three CAPE subscales, as well as the frequency and distress (total and weighted mean) of each endorsed item. The *Frequency total* and the *Distress total* consider the cumulative score of all symptoms experienced, whereas *Frequency mean*, and *Distress mean* denote the number-of-items-weighted mean.

	Number of items	Persecutory ideation	Bizarre experiences	Perceptual abnormalities	Frequency total	Frequency mean	Distress total	Distress mean
BPD (n=138) mean(sd)	7.60 (4.08)	2.10 (0.66)	1.74 (0.62)	1.50 (0.70)	12.17 (8.37)	1.79 (0.61)	13.16 (8.87)	1.64 (0.60)
Control (n=186) mean(sd)	2.12 (3.07)	1.29 (0.43)	1.14 (0.32)	1.08 (0.26)	2.69 (4.70)	0.74 (0.71)	2.13 (4.82)	0.40 (0.56)
Significance *Welch's	t ₂₄₄ =13.2, p<.001*	t ₃₂₂ =13.3, p<.001	t ₃₂₂ =11.3, p<.001	t ₁₆₆ =6.8, p<.001*	t ₂₀₀ =12.0, p<.001*	t ₂₀₀ =12.0, p<.001*	t ₁₉₇ =13.2, p<.001*	t ₃₂₂ =19.1, p<.001*

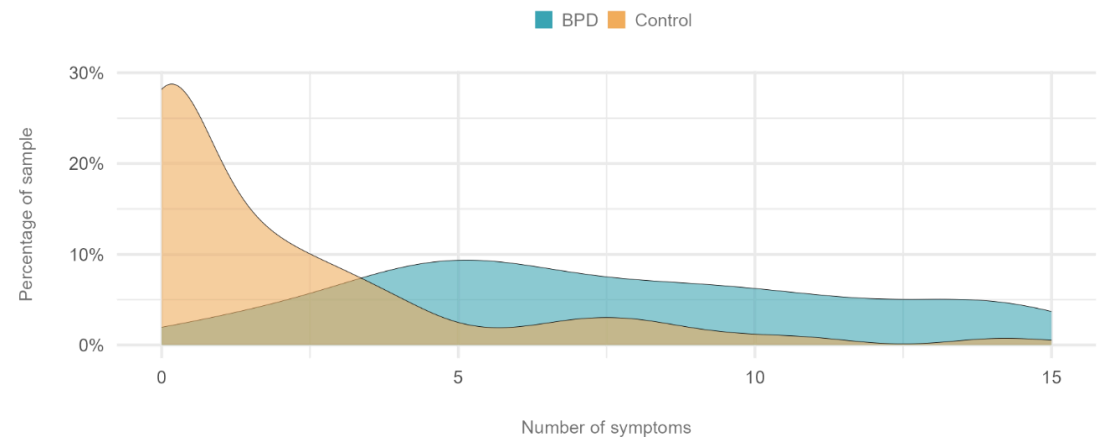


Figure 4.1

Number of psychotic experiences endorsed by BPD and control group participants. BPD: mean 7.60 (SD 4.08); control: mean 2.12 (SD 3.07), Welch's $t_{244}=13.25$, $p<.001$. For all further descriptive comparisons see Table 4.1 (above).

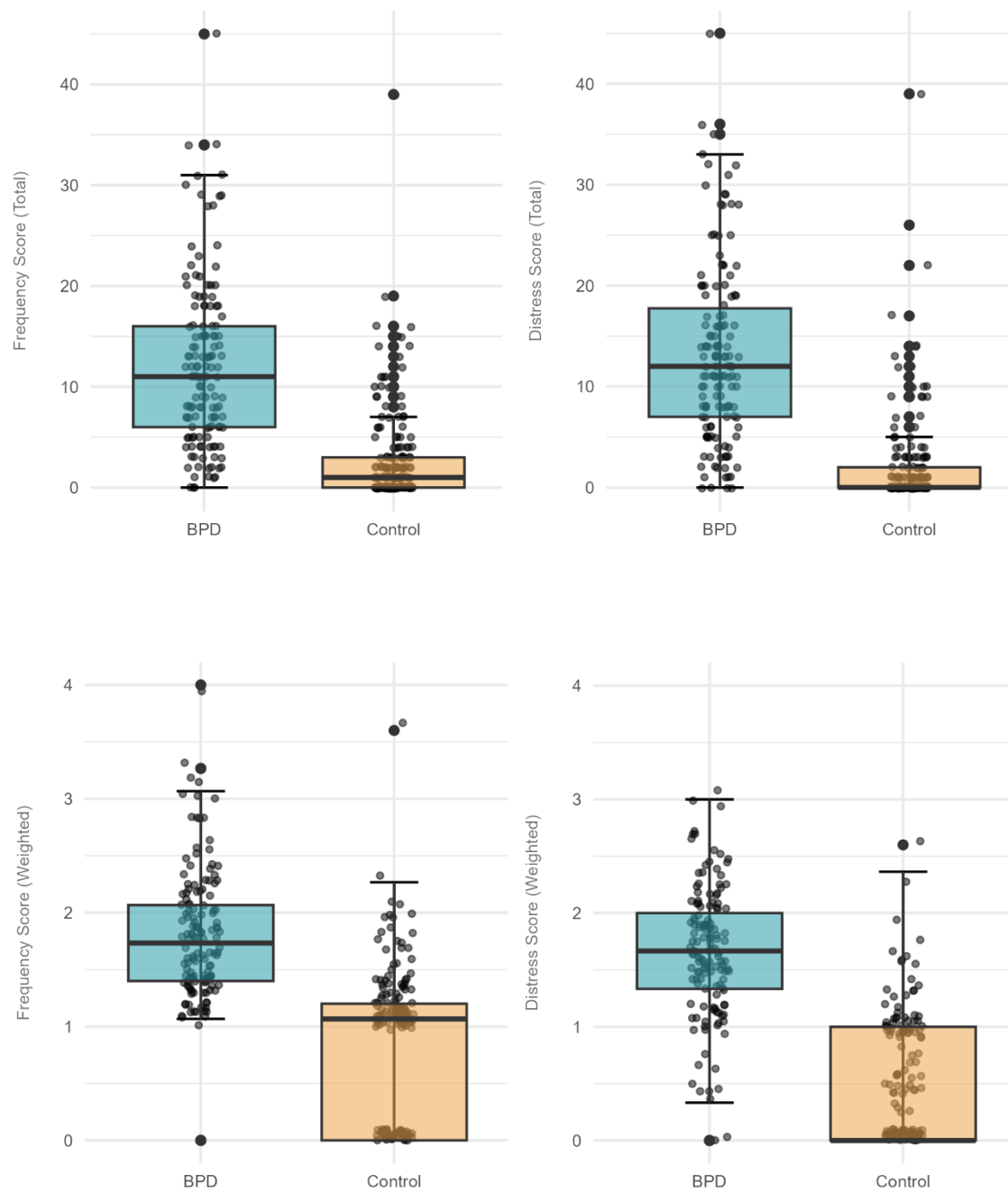


Figure 4.2

CAPE frequency total (top left) and item-weighted mean (bottom left), as well as distress score (top right), and item-weighted distress mean (bottom right) for BPD ($n=138$) and control group ($n=186$) participants. For descriptive statistics see Table 4.1)

4.2.2 Exploratory analysis: Symptoms above versus below the clinical threshold

Differences in symptom type and associated distress

On average, people with BPD above the threshold endorsed more items than those below (with a median of nine and four, respectively, Welch's $t_{136}=15.63, p<.001$), which suggests that in addition to symptom frequency, the range and number of symptoms experienced may contribute to clinical significance of psychosis in BPD (Figure 4.3). Group differences were observed for each of the three subscales (at $p<.001$), with the largest difference on the bizarre experiences score (Welsh's $t_{113}=13.00, p<.001$). All items were endorsed at significantly higher frequency by the high-PE group than by the low-PE group (at $p<.001$, Pearson's Chi-squared tests with Yates' continuity correction and Bonferroni adjustment for multiple comparisons), and the high-PE group reported more severe mean-weighted distress ($t_{136}=3.63, p<.001$).

For eleven out of the fifteen items, symptom prevalence (i.e., percentage of people endorsing that symptom) in the low-PE BPD group matched that of the control group, with only items one²³ ($\chi^2_1=13.5, p=.004$), two²⁴ ($\chi^2_1=17.5, p<.001$), three²⁵ ($\chi^2_1=17.2, p<.001$), and six²⁶ ($\chi^2_1=9.2, p=.036$) significantly more frequently reported by participants with BPD above *and* below the threshold (Figure 4.3). The first three of these symptoms load onto a common factor of "*paranoid ideation*" (Nunez et al., 2015). They are also the items most frequently endorsed by the high-PE group and may point towards a latent dimension of paranoia and suspiciousness that underlies (psychotic) experiences in BPD across its full spectrum of severity.

²³ Item 1: "...have you felt as if people seem to drop hints about you or say things with a double meaning?"

²⁴ Item 2: "...have you felt as if some people are not what they seem to be?"

²⁵ Item 3: "...have you felt that you are being persecuted in anyway?"

²⁶ Item 6: "...have you felt as if electrical devices such as computers can influence the way you think?"

Table 4.2

Questions and subscales of the current CAPE-P15

CAPE Item	Subscales
1. In the past 3 months, have you felt as if people seem to drop hints about you or say things with a double meaning? 2. In the past 3 months, have you felt as if some people are not what they seem to be? 3. In the past 3 months, have you felt that you are being persecuted in anyway? 4. In the past 3 months, have you felt as if there is a conspiracy against you? 5. In the past 3 months, have you felt that people look at you oddly because of your appearance?	<i>Persecutory Ideation</i>
6. In the past 3 months, have you felt as if electrical devices such as computers can influence the way you think? 7. In the past 3 months, have you felt as if the thoughts in your head are being taken away from you? 8. In the past 3 months, have you felt as if the thoughts in your head are not your own? 9. In the past 3 months, have you ever been so vivid that you were worried other people would hear them? 10. In the past 3 months, have you heard your thoughts being echoed back at you? 11. In the past 3 months, have you felt as if you are under the control of some force or power other than yourself? 12. In the past 3 months, have you felt as if a double has taken the place of a family member, friend or acquaintance?	<i>Bizarre Experiences</i>
13. In the past 3 months, have you heard voices when you are alone? 14. In the past 3 months, have you heard voiced talking to each other when you are alone? 15. In the past 3 months, have you seen objects, people or animals that other people can't see?	<i>Perceptual Abnormalities</i>

Differences in psychopathology, self-harm, and suicide attempts

On average, participants in the high-PE group had a greater MSI-BPD score ($t_{75}=2.31$ $p=.024$; Figure 4.4, left), although groups did not differ on any of the ten individual items. Similarly, for BPD participants of Study 2 (who completed the BSL, $n=20$) borderline symptom scores correlated significantly with the CAPE distress total ($r=0.614$, $p=.004$). More severe BSL symptomology seemed to also be associated with a higher CAPE frequency score, though this was non-significant ($r=0.419$, $p=.066$). There was no relationship between BSL, and the number of CAPE items endorsed ($r=0.349$, $p=.131$). In contrast to previous findings that have linked psychotic experiences in BPD to an increased risk for self-harm and suicide (Kelleher et al., 2017; Slotema et al., 2017; Cavelti et al.,

2019), there was no difference in average endorsement of the MSI items relating to self-harm²⁷ ($\chi^2_1=0.08$, $p=.779$) and suicide attempts²⁸ ($\chi^2_1<0.00$, $p=1$) (Figure 4.4, right). However, it must be noted that the McLean Screening Instrument only offers a yes/no answer option to assess any or all previous incidences and does not assess their frequency or severity.

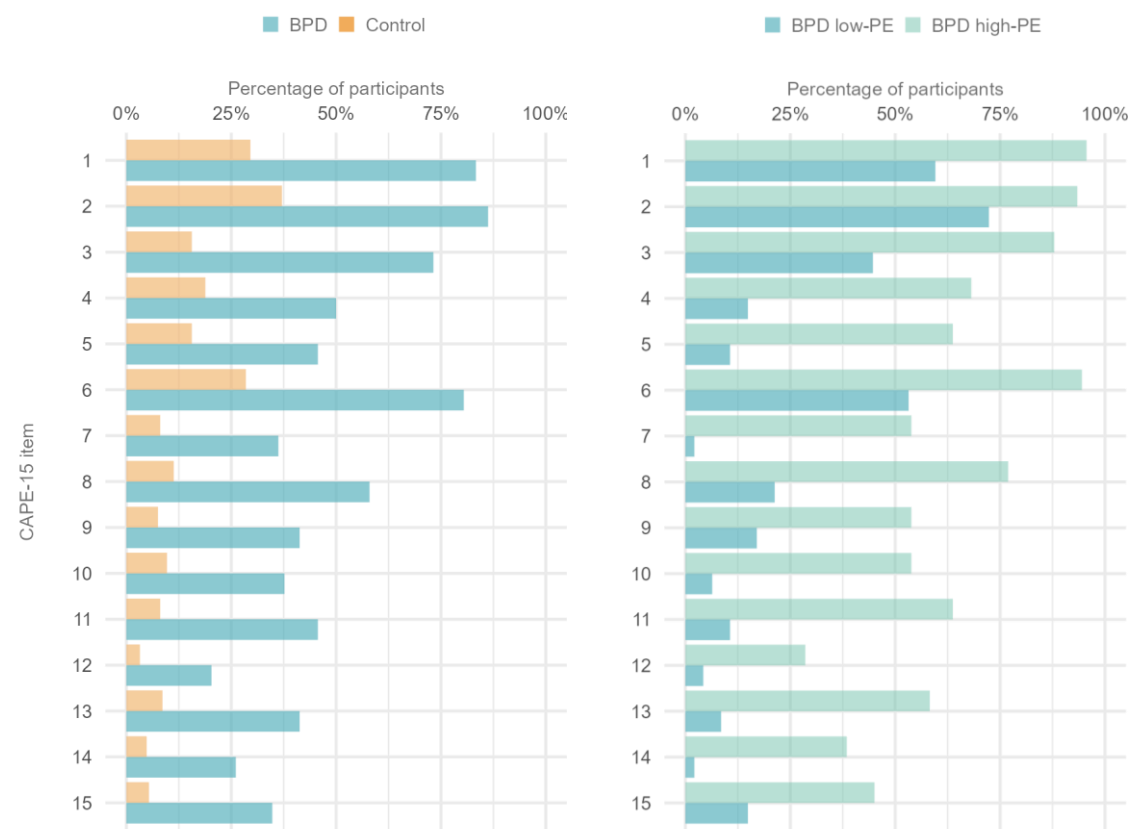


Figure 4.3

Proportion of sample endorsing individual CAPE items for the BPD group versus control participants (left), as well as for people with BPD and low versus high psychotic-experience scores (right). Control group: $n=186$, overall BPD group: $n=138$, BPD low-PE: $n=47$, BPD high-PE: $n=91$.

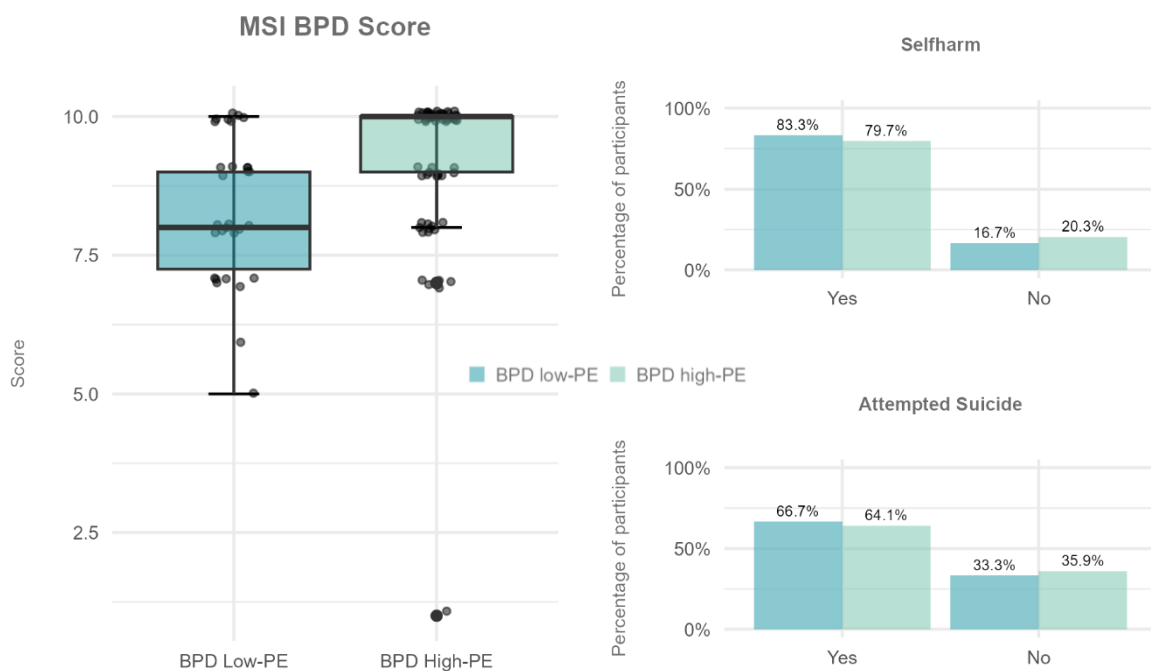
²⁷ “Have you deliberately hurt yourself physically (e.g. punched yourself, cut yourself, burned yourself)?”

²⁸ “Have you ever made a suicide attempt?”

Table 4.3

Number and type of comorbidities for BPD participants above (BPD high-PE) and below (BPD low-PE) the CAPE clinical significance cut-off (1.47). Any=at least one psychiatric comorbidity, 2+= two or more psychiatric comorbidities, ED=Eating Disorders, OCD=Obsessive-compulsive disorder, PTSD=post-traumatic stress disorder

	Any	2+	Anxiety	Depression	ED	OCD	PTSD
BPD low-PE (n=47)	44 (93.6%)	16 (34.0%)	12 (25.5%)	15 (31.9%)	7 (14.9%)	3 (6.4%)	7 (14.9%)
BPD high-PE (n=91)	88 (96.7%)	43 (47.3%)	44 (48.4%)	40 (44.0%)	19 (20.9%)	9 (9.9%)	22 (24.2%)
Significance							

**Figure 4.4**

Differences in BPD score (left), self-reported rates of self-harm (right, top), and suicide attempts (right, bottom) between people with BPD and high (n=42) versus low (n=52) psychotic experience scores

Over 90% of participants in both groups were currently diagnosed with a psychiatric comorbidity. People above the threshold appeared more likely to suffer from two or more additional disorders, and while the difference was non-significant at group level ($\chi^2_1=1.70$, $p=.191$), there was a weak correlation between number of reported diagnoses and CAPE (mean-weighted) symptom score ($r=0.205$; $p<.047$). The likelihood of being diagnosed with a particular comorbidity did not vary between groups for the disorders assessed (Table 4.2).

4.3 Discussion

The principal aim of collecting and examining the Screening data was to provide a quantifiable and reproducible estimate of the overall prevalence and clinical significance of psychotic experiences in BPD. It was the first assessment of these symptoms using the CAPE-15.

The data from Screening 1 and Screening 2 were combined into a total participant population of 324 people (138 BPD, 186 Control). This was possible for multiple reasons: First, both surveys employed the same questions (including using the CAPE-15 as psychotic symptom screen) and collected responses online via the experiment platform Qualtrics, effectively offering participants the same experimental procedure. Second, all study advertisements were disseminated in the wider community and both screening populations were assigned to the BPD versus control group identity based on self-report. As such, the screening data may be more accurately understood as reflecting one overall convenience sample (rather than two distinctly separate cohorts) and were thus merged. One important caveat concerning this approach is of course the MSI which was only presented to (BPD) participants of the first screening survey. Unfortunately, this precluded complete assessment of borderline symptom severity across the full sample, and (while unlikely) it is unknown whether the populations of Screening 1 and 2 may have differed on this measure.

Compared to the control group, BPD participants endorsed significantly more (and more severe) psychotic experiences, with close to two-thirds (65.9%) of the sample scoring

similar to patients with psychotic disorder. BPD participants with high versus low psychotic experience scores differed across the amount and types of symptoms reported, as well as regarding their associated distress. Higher CAPE scores additionally correlated with more severe borderline pathology, and a greater number of currently diagnosed comorbidities.

BPD and control participants differed significantly across all levels of measurement, including the sum of symptoms, the frequency of the experiences, and their associated distress. Notably, the difference in distress remained significant even after controlling for the number of items endorsed, indicating that both the quantity of symptoms and the (negative) quality of the experiences may contribute to their overall impact. This is in line with findings of marked differences in the qualitative appraisal of hallucinations in clinical versus non-clinical populations, the latter of which typically report benign, non-disturbing content (Sommer et al., 2010).

Increased symptom prevalence and severity in BPD participants was observed for all three subscales (i.e., “types” of psychotic experiences), including persecutory ideation, perceptual abnormalities, and (most significantly differing from the control group) bizarre experiences.

Across both the BPD and the control group, items one²⁹, two³⁰, and six³¹ were the most frequently endorsed symptoms. This reflects findings by Núñez et al. (2021) who reported the greatest answer variability for the first two symptoms (with most participants choosing never for all other questions) in a community sample of adolescents. Item six is categorised as a “*bizarre experience*”, aiming to assess the extent participants believe that they are under the control of electrical devices. However, since the question’s conception in 2002 (as part of the original CAPE-40 questionnaire) much has changed in how technology dictates and influences our daily lives. What was intended as a psychopathological measure of the phantastic paranoid delusions one would associate with schizophrenia may instead

²⁹ Item 1: “...have you felt as if people seem to drop hints about you or say things with a double meaning?”

³⁰ Item 2: “...have you felt as if some people are not what they seem to be?”

³¹ Item 6: “...have you felt as if electrical devices such as computers can influence the way you think?”

now be interpreted within a cultural framework of social media, data-mining, and subtle commercial coercion. As such, endorsement could in fact reflect a more sceptical and distrustful stance towards information technology, rather than a highly specific bizarre delusion. This in turn would also explain why item six was the third most frequently selected symptom in the control group.

The CAPE-15 has previously been suggested (Bukenaitė et al., 2017) and applied as screening tool for detecting people at ultra-high risk for psychosis in the general population (Núñez et al., 2021), and in people with common mental disorders (Knight et al., 2020b), with a postulated cut-off of 1.47. Two recent studies applying the measure to different clinical samples individuals with common mental disorder consistently found the ratio of participants above the threshold to be around 20-30% in patients across health services in three different counties in the East of England, including 26.5% in an overall sample of 1131 people with CMD (Knight et al., 2020b), as well as 22.5% (133/590) in CPFT patients (the Cambridgeshire and Peterborough foundation trust operating in the local region from which the second study's participant population was also recruited) (Knight et al., 2020c). By comparison, in the current study 65.9% (91/138) of BPD participants scored above this threshold (as well as 12.5% (21/168) of the control group), which further underlines the increased pathological impact and clinical relevance of PEs in people with BPD compared to not only the general population but also to other mental health disorders.

Interestingly, for the majority (11 out of 15) of experiences, the percentage of BPD participants in low-PE group endorsing those items did not significantly differ from the control group. While this needs to be considered with the important caveat that group affiliation (low versus high-PE) was determined by the CAPE clinical cut-off and thereby naturally biased towards more extreme pathologies, the threshold score is calculated by averaging frequency ratings and is not influenced by the number of items or their associated distress (i.e., it is possible for participants to endorse all fifteen symptoms but still score below 1.47). As such, the overlap between BPD low-PE and control participants (and lack thereof between the low- and high-PE groups) may certainly point toward possible (phenomenological, aetiological, mechanistic) differences in how and why different people with BPD experience psychotic symptoms.

Congruently, people with BPD who scored above the CAPE threshold of 1.47 experienced a greater number of symptoms more frequently and found them more distressing. This raises interesting questions about the exact nature of the psychosis continuum in BPD, as it suggests that experiences below the threshold are neither “the same symptoms but less distressing”, nor “equally distressing, just fewer symptoms”. Rather they appear to present a dimension that is both quantitatively and qualitatively less severe and may be more similar to the experience of psychotic symptoms in the general population – whereas the experience of people above the threshold may be more comparable to psychotic disorders. Consequently, it would be beneficial to assess high-PE and low-PE BPD populations on some of the factors previously found to distinguish clinical from non-clinical psychotic experiences, including content and emotional appraisal, age of onset (Sommer et al., 2010), and possibly neurological differences (Garrison et al., 2019).

Assessment of further differences between participants with BPD above and below the cut-off revealed a link between psychotic symptom severity and MSI BPD scores. This essentially replicates findings by Baryshnikov et al. (2018) who reported moderate correlation between the frequency of positive symptoms from the CAPE-42³², and MSI total. While these results suggest a relationship between psychotic experiences and BPD symptom severity, it should be noted the McLean Screening Instrument is a brief, ten-item measure, which provides only a limited depth of assessment. Further investigations using more detailed measures of borderline pathology must be applied to ascertain the nature and directionality of this correlation. The increased rates of self-harm and suicide attempts in people with BPD and high psychotic symptom scores reported by others (Cavelti et al., 2019; Kelleher et al., 2017; Slotema et al., 2017) were not observed, however due to the broad, dichotomous nature of the assessment in the current study (i.e., two simple yes/no questions) it would be foolish to suggest that the present observations contradict previous findings. There was, however, a significant (albeit weak) correlation between psychotic experiences and the number of psychiatric comorbidities (as also found by

³² The *Community Assessment of Psychic Experiences-42* (CAPE-42) is a 42-item self-report questionnaire that measures the lifetime prevalence of psychotic symptoms (both positive and negative) and forms the basis from which the (current) CAPE-15 (measuring only positive psychotic experiences within the past three months) was developed (Stefanis et al., 2002).

Niemantsverdriet et al., 2017), with notably higher rates of depression and anxiety, but no statistically relevant differences in the prevalence of any particular disorder. Future experiments may choose to further investigate this relationship and determine if it is the number, type, or severity of psychiatric comorbidities that is most strongly associated with the co-occurrence of psychotic experiences in BPD.

Overall, these findings broadly point towards a possible relationship between the severity of psychiatric morbidity and psychosis proneness, as high (clinically relevant) CAPE scores appear to delineate the more extreme end of borderline pathology (as reflected by greater MSI scores), as well as higher rates of comorbid mental health disorders.

This could sustain the conceptualisation of psychotic experiences in BPD within a p-factor framework, as well as the hypothesis that such symptoms may develop at the extreme ends of CMD and might therefore be more common in people with more severe borderline pathology (who might spend more time in intense emotional states) and those with higher rates of depression and anxiety.

Nevertheless, it is interesting to note that higher CAPE scores were characterised not just by increased symptom frequency and distress, but also by a broader range of (more) unusual experiences. This would equally support the theory that among people with a BPD diagnosis there are inherent (aetiological or mechanistic) differences in the risk for and struggle with psychotic symptoms, with some individuals' predisposition and response to the experience similar to that of non-clinical populations, while others are affected at a level of personal and clinical significance akin to psychotic disorder. Biographical factors such as trauma and post-traumatic stress, as well as genetic or cognitive vulnerabilities could be further spurring such divergence.

Chapter 5 The Psychic Neuroses – exploring symptom range

“Sometimes I feel the presence of pure evil watching me, usually from a window or a doorway (a 'gateway' or 'entrance' I suppose?), and most commonly when I'm in bed with my back to the gateway. It fills me with fear and dread, and I cannot move. It usually happens during the night in the dark [...]. Often around 3-4am, too, which I always found rather funny/ironic as 3am is known as the devil's hour.”

5.1 Introduction

A chapter ostensibly claiming to assess the range of psychotic experiences (in BPD) begs a frank disclaimer: it is virtually impossible to assess the actual range of psychotic experiences (in any context). What is now liberally defined as hallucinations and delusions began in 1841 as the shorthand for “*psychic neurosis*”, at a time when neurosis broadly referred to any disorder of the nervous system (Bürky, 2008). Over the course of the following century the term morphed into a much more explicit definition and for a while was used synonymously with schizophrenia, before again devolving into the extensive, abstract concept it is today. Rather than a single condition, psychosis describes a syndromal construct that refers to a state of mind (transient or permanent) during which thought, and perception are harshly distorted, thus diverting from consensus reality. The experience of such a state is of course highly personal, and so are the multitudinous symptom presentations that reflect it. Hallucinations and delusions, the positive symptoms of

psychosis, are often merely the tip of a colossal iceberg of thought and speech disorder, difficulties with motor-control, and negative affect. Few studies purporting to investigate “psychotic experiences” in BPD have looked beyond positive symptoms and this dissertation is no exception. Nonetheless, by investigating and understanding the perceptual and cognitive aspects of psychosis in BPD it may be possible to gauge whether future explorations of further psychotic symptom dimensions are advisable.

5.1.1 Assessing perceptual abnormalities and delusional beliefs

While the CAPE is well-suited to estimate the overall prevalence and severity of psychotic symptoms in people with BPD, exploring their nature and risk factors necessitates a more thorough evaluation. Experiences of delusional beliefs may differ from those of perceptual abnormalities (subjectively and mechanistically), and both require individual assessment. Given the high variance of reported symptoms within the BPD group, dimensional measures are preferable over strictly clinical instruments, especially since the latter have typically been designed for use psychotic (in)patient populations and are often inclined towards highly specific types of symptoms.

The Peters Delusions Inventory (PDI; Peters et al., 2004) measures delusional ideation, with items derived from a long-established clinical measure (the Present State Examination; Wing et al., 1975) but adjusted for use in the general population. Like the CAPE, it assesses symptom presence and frequency, as well as associated distress, preoccupation with-, and conviction of the beliefs. Modelled on this same structure, Bell et al. (2006) later devised the Cardiff Anomalous Perceptions Scale (CAPS), a (fairly analogous) measure of perceptual symptoms, which includes items pertaining to nine different experience categories. In combination, both instruments permit assessment and comparison of BPD and control participants across a wide range of perceptual and delusional symptoms.

Unlike the CAPS, which was designed as a multifactorial construct using principal component analysis, the PDI was created under the assumption of a single dimension of delusional ideation (Peters et al., 1999). Subsequent attempts to identify multi-factor

solutions (Jones and Fernyhough, 2007; Fonseca-Pedrero et al., 2012) have repeatedly fallen short and the unidimensional structure was later reassessed and reindorsed by Wang et al. (2017). With this in mind, comparing different types of delusional thinking between participant populations proves somewhat challenging. However, of the eleven components originally proposed by the authors, seven were identified as having clearly recognisable, distinct themes³³, which could allow for reliable and replicable comparison of participants across these belief categories.

Of course, (even in combination) these two instruments are unlikely to capture the full range of psychotic symptoms experienced by people with BPD. However, they can provide a solid conceptual foundation by assessing different experience categories and may serve as an estimate for the prevalence and severity of specific types of hallucinatory and delusional experiences, thereby facilitating subsequent investigation of the risk factors and mechanisms associated with these symptoms.

5.1.2 BPD populations and replicability

Considering the immense inconsistencies in the reported prevalence rates (see Chapter 1.2) of psychotic experiences in BPD populations (including between studies employing the same PE measure), it is only prudent to consider sample selection as a potential confounding factor: While some researchers worked with inpatient populations (Zanarini et al., 2013), others assessed outpatients (Pearse et al., 2014; Slotema et al., 2017), and others yet investigated borderline symptom traits in the general population (Kelleher et al., 2017). It is therefore possible that unknown variables such as BPD severity, the number of comorbidities, or simply diagnostic differences between countries have a latent effect on the presentation of psychotic symptoms and are hindering replicability. Coincidentally (or more accurately due to the COVID-19 restrictions on in-person research), participants for the first study were recruited online and internationally, whilst Study 2 was conducted

³³ Persecution (items 4 and 5), suspiciousness (items 1 and 3), paranoid ideation (items 13 and 15), religiosity (items 8 and 11), grandiosity (items 6 and 7), paranormal beliefs (items 9 and 12), and thought disturbances (items 18 and 20).

locally in the Cambridgeshire area. Additionally, BPD participants of the latter were asked to provide an official confirmation of their diagnosis whereas online participants were assigned to the BPD group based on self-identification (in conjunction with the MSI-BPD cut-off). These circumstances provide the interesting opportunity to investigate to what extent prevalence and severity estimates may be affected by sample selection, by comparing a verified clinical sample, to a self-report populational group.

Accordingly, this chapter aims to answer the following questions:

1. Are there marked differences in the prevalence or severity between perceptual and delusional experiences?
2. What are the prevalence rates of different symptom clusters and are there certain experience categories that are especially (un)common in people with BPD?
3. Do symptom prevalence and severity differ between a local, clinical BPD population, and a more international BPD population identified by self-report?

5.2 Analysis

This chapter presents analyses of the questionnaire data concerning the experience of psychotic symptoms, specifically perceptual abnormalities as measured by the CAPS and delusional beliefs as measured by the PDI, including brief supplementary surveys on symptom duration and triggering factors.

Unlike their respective screening surveys (the data of which was combined due to significant methodological and sampling overlap), the main experiment sessions of Study 1 and Study 2 differed apropos procedure (online versus in-person), methodology (partially diverging instruments) and most importantly, study population (self-report/self-identified versus verified diagnosis). As such, results from Study 1 and Study 2 were analysed independently and in comparison. In addition to their psychotic symptom measures, both experimental procedures included a series of further questionnaires and cognitive tasks. See Chapter 3.2.2 for more details on the CAPS and the PDI, and Chapter 3.5 (Study 1)

and 3.6 (Study 2) for individual study procedures. For participant demographics see Chapter 3.4.3 (Study 1) and 3.5.3 (Study 2).

For both CAPS and PDI, four separate scores were calculated: A total score (calculated by summing the number of symptoms experienced) as well as three subscales which measure distress, intrusiveness, and frequency (for the CAPS), and distress, preoccupation, and conviction (for the PDI). Since these additional scores are obtained by considering non-endorsed items to have a value of zero, additional mean-weighted scores were calculated by dividing participants' subscale total by the number of symptoms endorsed, to assess distress levels independent of symptom count.

CAPS and PDI scores were compared between BPD participants and controls, as well as between the self-report (Study 1) and the clinical (Study 2) BPD population. Significance was assessed with independent two-sample t-tests (Welch's *t* in case of unequal variances). Differences in the prevalence of specific types of PE were explored using Pearson's Chi-squared tests with Yates' continuity correction. For the CAPS, assessments were made for the nine symptom categories suggested by the original authors (Bell et al., 2006). Additionally, four further symptom groups³⁴ were defined, since these were of interest for judging prevalence against previous findings, independent of instrument. For the PDI, participants were compared on seven of its eleven components, which were identified by the authors as dependably distinct factors (Peters et al., 2004).

P values of all serial comparisons were adjusted with Bonferroni correction. Distribution of the data was assessed visually (histograms and Q-Q plots) and using Kolmogorov-Smirnov normality tests with Lilliefors correction. Non-normally distributed datasets are reported with the Shapiro-Wilk statistic. Unless otherwise stated, approximately normal distribution of the relevant measures was observed. All analyses and graphs were done in R.

³⁴ Visual distortion, visual hallucinations, auditory distortion, auditory non-verbal hallucinations

5.3 Results

5.3.1 Main analysis: Frequency and severity of perceptions and beliefs

Results from Study 1 – Self-report BPD sample

BPD participants scored significantly higher than controls (at a significance level of $p < .001$, with Bonferroni correction) across all four dimensions of both CAPS, and PDI (Figure 5.1, bottom). Descriptive statistics are summarised in Table 5.1. The distribution of CAPS total scores for the control group was markedly left-skewed (Figure 5.1, top), which is in line with previous assessments of non-clinical samples (Bell et al., 2006) and has been postulated as a true reflection of how anomalous perceptual experiences occur within the general population (Johns and Van Os, 2001). By contrast, BPD group totals were distributed along the full range of CAPS scores, which mirrors the distributional pattern typically observed in inpatients with psychosis (Bell et al., 2006; Bell et al., 2011).

Subscale differences between groups remained significant at $p < .001$ for the mean-weighted scores, which again indicates that not just the increased number of psychotic symptoms, but also their subjective experience (including higher frequency, stronger intensity, and greater distress) may contribute to the prominence of these symptoms in people with BPD.

While the PDI total is intended to capture the full range of delusional ideation (and not as a screening tool), a clinical significance threshold of eight has previously been proposed (Preti et al., 2007). Using this cut-off, 12% of the control sample (8/68) and 59% of the BPD sample (32/54) in Study 1 were experiencing symptoms of potentially pathological significance. In the second study, 55% of BPD participants (11/20) scored above the threshold, compared to 3% (1/29) of controls.

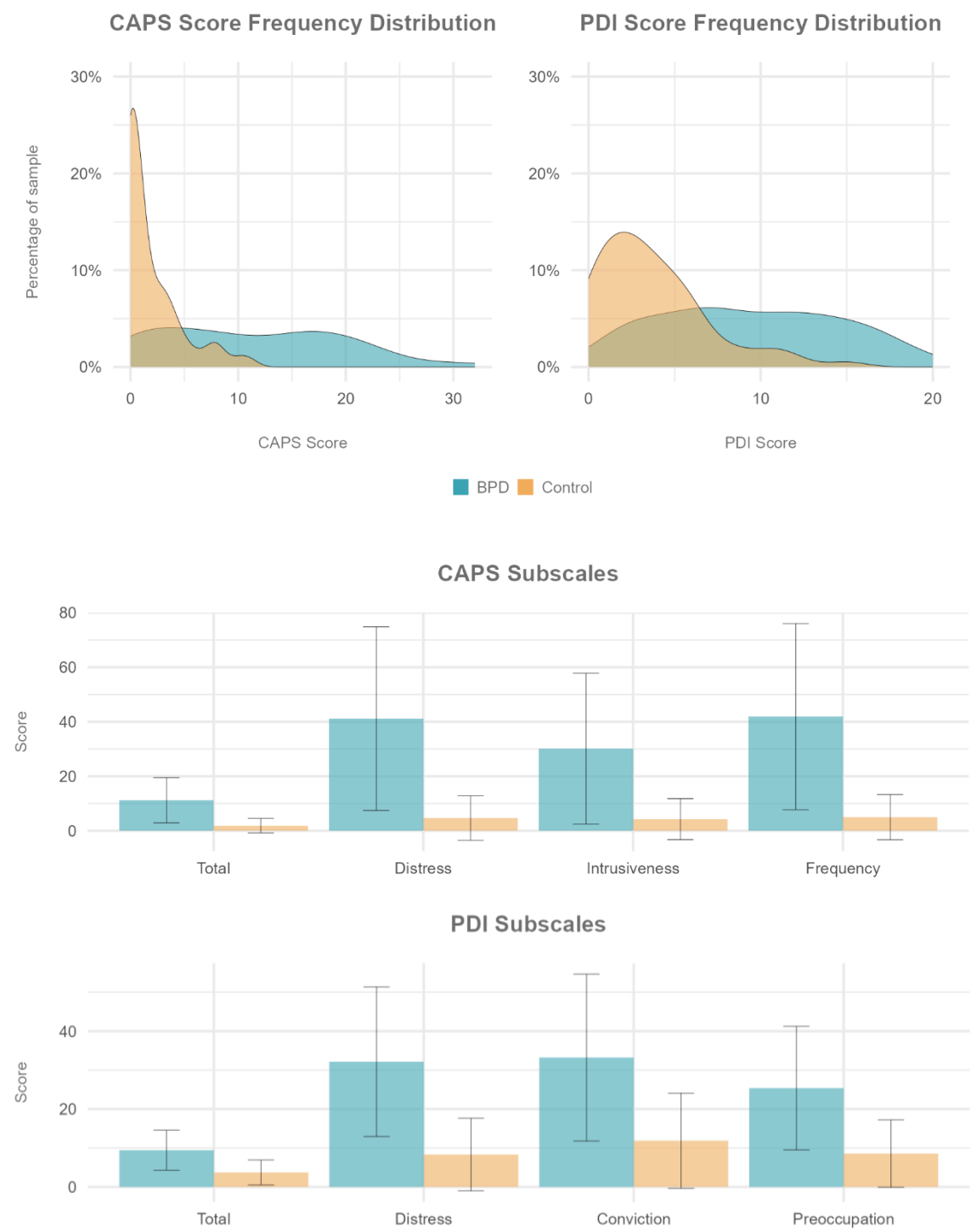


Figure 5.1

Distributions of CAPS (perceptual, top left) and PDI (delusional, top right) scores, as well as bar plots of the means and standard deviations of the total and subscale scores of CAPS (top) and PDI (bottom) for BPD and control participants of Study 1. For descriptive statistics see Table 5.1.

Table 5.1

Descriptive statistics of the CAPS and PDI total and subscale scores for Study 1 and Study 2. BPD participants scored significantly higher than the controls on each of the measures, across both studies. There were no significant differences between BPD populations.

CAPS						PDI			
Study 1		Total	Distress	Intrusiveness	Frequency	Total	Distress	Conviction	Preoccupation
BPD (<i>n</i> =54)	mean(<i>sd</i>)	11.2(8.3)	41.1(33.7)	30.1(27.7)	41.9(34.2)	9.4(5.1)	32.2(19.2)	33.2(21.4)	25.4(15.9)
Control (<i>n</i> =68)	mean(<i>sd</i>)	1.8(2.7)	4.7(8.2)	4.2(7.5)	5.0(8.3)	3.7(3.2)	8.3(9.3)	11.9(12.2)	8.6(8.7)
	Welch's	t ₆₂ =7.94, p<.001	t ₅₃ =8.70, p<.001*	t ₁₂₀ =6.43, p<.001*	t ₁₂₀ =8.45, p<.001*	t ₈₅ =7.13, p<.001*	t ₇₃ =8.37, p<.001*	t ₈₀ =6.53, p<.001*	t ₁₁₄ =10.54, p<.001*
Study 2									
BPD (<i>n</i> =20)	mean(<i>sd</i>)	13.7(8.5)	42.7(31.4)	35.0(22.6)	46.9(34.6)	8.5(5.1)	28.2(18.1)	27.9(17.8)	26.9(17.8)
Control (<i>n</i> =29)	mean(<i>sd</i>)	1.1(1.5)	2(2.8)	1.7(2.6)	2.4(4)	1.8(2.4)	3.6(5.3)	5.5(7.7)	4.1(6.4)
	Welch's	t ₁₉ =6.60, p<.001	t ₁₉ =5.77, p<.001*	t ₁₉ =6.55, p<.001*	t ₁₉ =5.72, p<.001*	t ₂₄ =5.56, p<.001*	t ₂₁ =5.91, p<.001*	t ₂₄ =5.29, p<.001*	t ₂₂ =5.50, p<.001*

Comparison with Study 2 – Clinical sample

All differences between BPD participants and controls observed in Study 1 (including item totals, subscales, and weighted scores) were replicated in the clinical sample, at a significance level of $p<.001$ for both the CAPS, and the PDI (see Table 5.1 for descriptive statistics of Study 2).

While the BPD population of Study 2 scored marginally higher on all of the CAPS scales, this was non-significant and the two groups did not differ regarding the number of symptoms experienced ($t_{72}=1.16$, $p=.249$), nor their frequency ($t_{72}=0.55$, $p=.582$), associated distress ($t_{72}=0.18$, $p=.860$), or intrusiveness ($t_{72}=0.70$, $p=.487$). The PDI also showed no differences between participants with BPD of Study 1 and Study 2, neither for the total score ($t_{72}=0.69$, $p<.491$), nor any of the three subscales ($t_{72}=0.80$, $p<.428$, Welch's $t_{41}=1.09$, $p<.282$, and Welch's $t_{31}=0.34$, $p<.738$ for distress, conviction, and preoccupation respectively) (Figure 5.2).



Figure 5.2

Top: Distributions of CAPS (top) and PDI (bottom) scores for BPD participants of Study 1 ($n=54$) and Study 2 ($n=20$). Bottom: Violin plots of the CAPS (left) and PDI (right) total and subscales. The stark overlap of scores' means and range, as well as the nearly identical distribution between the international self-report (Study 1) and the local clinical sample (Study 2) suggest (at least partial) generalisability and replicability across different BPD populations.

Curiously, control participants in Study 1 and Study 2 differed on the PDI (Welch's $t_{70}=3.33, p<.4.28$). This could (in part) be explained by the (lack of) social desirability bias (Paulhus, 1984), such that people feel more comfortable to disclose unusual beliefs from the anonymity and safety of their own home than in the presence of an experimenter. On the other hand, there is a distinct possibility that the (much larger and less controllable) online sample included a higher number of people with undisclosed or unrecognised underlying psychopathology. Assumption of the latter is especially reasonable when considering that rates of clinically significant delusional ideation in the general population are estimated to be around 1-5% (compare Freeman, 2006; Freeman, 2007; Linscott and Van Os, 2013), which is much more in line with the findings of Study 2. CAPS scores of the control group in Study 1 were also elevated, though the difference was not significant at a 95% CI.

5.3.2 Further analysis: Differences in prevalence of symptom type

Perceptual experiences (CAPS items)

In both studies, symptom prevalence differed significantly between the BPD group and their respective controls, for each of the nine CAPS experience clusters (at $p<.001$, using Bonferroni correction for multiple comparison, Appendix D.1) (Table 5.2). Changes in the level of sensory intensity (e.g., “*Do you ever notice that sounds are much louder than they normally would be?*”) and temporal lobe associated perceptual abnormalities (e.g., “*Do you ever sense the presence of another being, despite being unable to see any evidence?*”) were the most frequently reported experiences, followed by sensory flooding (e.g., “*Do you ever find that sensations happen all at once and flood you with information?*”) and sensory distortion (e.g., “*Do you ever find the appearance of things or people seems to change in a puzzling way, e.g. distorted shapes or sizes or colour?*”).

Overall, 72% of BPD participants in Study 1 and 80% of participants in Study 2 endorsed one or more of Schneider's first-rank symptoms. Historically, FRS were conceptualised as a symptom marker for schizophrenia and were frequently used to distinguish the disorder from other psychiatric conditions (Kendler and Mishara, 2019), with an apparent

specificity of 81.4% according to a 2015 review (Soares-Weiser et al., 2015). However, since FRS are frequently observed in non-schizophrenic psychosis (Peralta and Cuesta, 1999; Gonzales-Pinto et al., 2003), their diagnostic validity has repeatedly been called into question (Peralta and Cuesta, 2023) and they are typically no longer applied in an official, clinical context – albeit calls for their reinstatement (Heinz et al., 2016; Moscarelli et al., 2020). Even so, the concept still finds its way into non-diagnostic clinical (and research) contexts as a common indicator for what “counts” as a real psychotic symptom.

Table 5.2

Prevalences of perceptual experiences in the BPD and control groups of Study 1 and Study 2, for the nine CAPS symptom categories (a), other relevant symptom groups (as often reported in the literature) (b), and Schneider’s first rank symptoms (FRS) of psychosis (c). BPD samples did not differ between online- and clinical population on any of the symptom categories or items. All differences between BPD and control groups significant at $p < .001$, with Bonferroni correction for multiple comparison (Appendix D.1).

		relevant items	Study 1		Study 2	
			BPD ($n=54$)	Control ($n=70$)	BPD 2 ($n=20$)	Control ($n=59$)
a)	Having a Nonshared Sensory Experience	13, 29, 31, 32	30 (56%)	2 (3%)	13 (65%)	1 (3%)
	Unusual or Distorted Sensory Experience	5, 16, 25, 26, 30	36 (67%)	12 (18%)	12 (60%)	3 (10%)
	Sensory Experience from Unexplained Source	4, 6, 8, 12, 14, 28	35 (65%)	13 (19%)	15 (75%)	3 (10%)
	Distortion of Form (Own Body and World)	9, 10, 19, 22	35 (65%)	11 (16%)	12 (60%)	2 (7%)
	Changes in Levels of Sensory Intensity	1, 18, 20, 21, 23	45 (83%)	23 (34%)	18 (90%)	6 (21%)
	Sensory Flooding	15, 17	37 (69%)	6 (9%)	17 (85%)	4 (14%)
	Thought Echo and Hearing Thoughts	3, 7	24 (44%)	5 (7%)	13 (65%)	1 (3%)
	Temporal Lobe Abnormalities	2, 10, 24, 27	38 (70%)	16 (24%)	18 (90%)	4 (14%)
	Auditory Verbal Hallucinations	11, 13, 28	21 (39%)	5 (7%)	11 (55%)	1 (3%)
b)	Visual distortion	4, 26, 19	32 (59%)	6 (9%)	13 (65%)	0 (0%)
	Visual Hallucination	31	19 (35%)	2 (3%)	10 (50%)	0 (0%)
	Auditory distortion	16	16 (30%)	2 (3%)	5 (25%)	0 (0%)
	Auditory non-verbal hallucination	6, 32	30 (56%)	5 (7%)	9 (45%)	2 (7%)
c)	FRS	3, 5, 7, 11	39 (72%)	9 (13%)	16 (80%)	5 (17%)

In both BPD groups, visual distortion was significantly more widespread than auditory distortion ($\chi^2_1=8.44$, $p=.003$), while visual and auditory hallucinations appeared to be equally common. Similarly, there was no difference in the rates of auditory verbal, and auditory non-verbal hallucinations ($\chi^2_1=2.38$, $p=.123$). BPD samples did not differ between online- and clinical population on any of the symptom categories (Appendix D.1.3).

Delusional experiences (PDI items)

In contrast to the CAPS (where BPD participants and controls differed across the full range of anomalous experiences) the differences observed on the PDI were highly symptom dependent. In Study 1, BPD participants scored significantly higher than controls on thought disturbances, as well as persecution, suspiciousness, and paranoid ideation (at $p<.001$, using Bonferroni correction for multiple comparisons). The latter three have been suggested to converge on a central theme of paranoia (Peters et al., 2004) and thus mirror the findings of the Screening analysis (Chapter 4.2.1). There was no difference between groups for the grandiosity ($\chi^2_1=1.29$, $p=1$), religiosity ($\chi^2_1=0.65$, $p=1$), and paranormal beliefs ($\chi^2_1=5.57$, $p=.127$) components. Findings were replicated in Study 2 (Appendix D.2), with exception of the persecution factor, although this appears to be due to a change in control group, rather than BPD group endorsement. The BPD populations again did not differ significantly on any of the reported measures (Appendix D.3).

Table 5.3

Prevalences of delusional beliefs in the BPD and control groups of Study 1 and Study 2, for seven of the PDI symptom-type components. BPD samples did not differ between online- and clinical population on any of the symptom categories or items.

*Difference between BPD and respective control group significant at $p<.05$ (see Appendix D.2)

	Study 1		Study 2	
	BPD ($n=54$)	Control ($n=68$)	BPD ($n=20$)	Control ($n=29$)
*persecution (items 4, 5)	31 (57%)	8 (12%)	12 (60%)	10 (34%)
*suspiciousness (items 1,3)	48 (89%)	40 (60%)	17 (85%)	8 (28%)
*paranoid ideation (items 13, 15)	41 (76%)	24 (35%)	16 (80%)	8 (28%)
religiosity (items 8, 11)	18 (33%)	17 (25%)	3 (15%)	3 (10%)
grandiosity (items 6, 7)	35 (65%)	36 (53%)	11 (55%)	8 (28%)
paranormal beliefs (items 9, 12)	29 (54%)	21 (31%)	9 (45%)	5 (17%)
*thought disturbances (items 18,	35 (65%)	5 (7%)	10 (50%)	3 (10%)

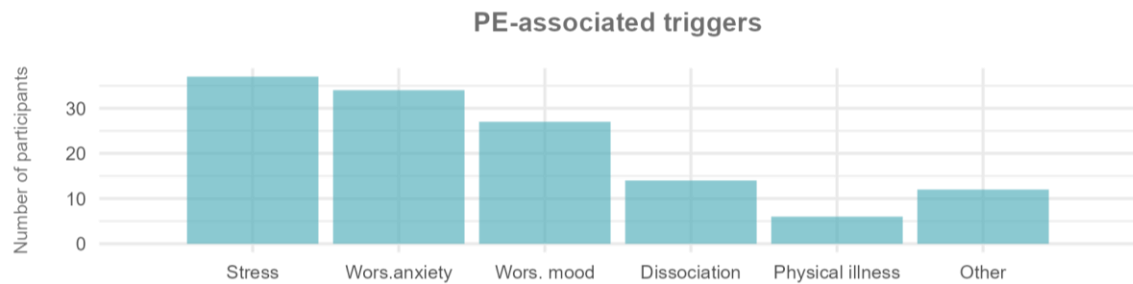


Figure 5.3

Self-reported triggers for psychotic experiences endorsed by BPD participants ($N=74$) on the CAPS and PDI follow-up questionnaire. Participants had the option to endorse multiple triggers. ‘Other’ triggers included poor sleep and sleep deprivation ($n=4$), social isolation ($n=5$), and an association between psychotic symptoms and menstrual cycle ($n=7$).

5.3.3 Exploratory analysis: Symptom frequency, duration, and triggers

While CAPS and PDI can provide an estimate of the prevalence, frequency, and severity of psychotic experiences, they do not consider symptom duration or permanence. Due to a lack of validated psychopathology measures assessing these dimensions, BPD participants who endorsed one or more items on the above questionnaires were presented with a brief survey on the general length and longevity of their experiences. It was furthermore of interest whether such symptoms persisted with relative stability, or whether they might be affected by external or internal events such as stress, which is often reported as a potential trigger but has rarely been assessed in research settings. Considering their substantial overlap on both psychotic symptom measures (i.e., CAPS and PDI), the BPD groups of Study 1 and Study 2 were combined into one sample population ($N=74$) and are reported as such.

61% (45/74) of participants were aware of one or more triggers for their experiences and an additional 34% (25/74) reported factors that they thought might be influencing the symptoms but were unsure. Only 5% (4/74) reported that they were unaware of any specific causes. Of the potential triggers assessed, stress was most frequently endorsed (50%, 37/74), followed by worsening anxiety (45%, 33/74), and mood deterioration (35%, 26/74) (Figure 5.3). Twelve people reported additional ‘Other’ influences, including poor sleep

and sleep deprivation ($n=4$), social isolation ($n=5$), and an association between their symptoms and their menstrual cycle ($n=7$). 35% of participants (26/74) experienced their delusional symptoms daily, with a decreasing number of people describing them as weekly (30%, 22/74), monthly (15%, 11/74), occasional (11%, 8/74), or rare and circumstantial (9%, 7/74) (Figure 5.4, top). CAPS symptom duration followed a less predictable pattern, with most people's perceptual experiences lasting a few minutes (30%, 22/74), followed by day-long episodes (22%, 16/74). Fourteen people (19%) reported continuous perceptual symptoms, and only a few participants described them as lasting hours (11%, 8/74), up to a week (7%, 5/74), or up to a month (7%, 5/74) (Figure 5.4, bottom). Since participants were encouraged to answer with regard to the perceptual experience they perceived as the most the distressing and impactful, these observations could suggest that PE duration could perhaps vary between different types of symptoms.

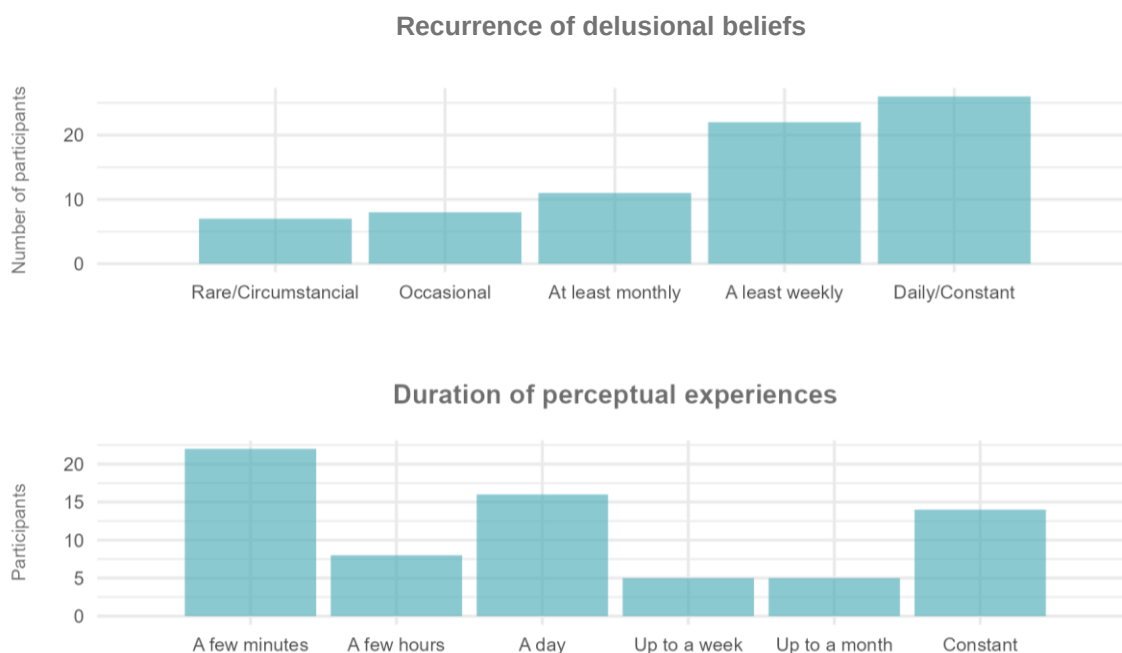


Figure 5.4

Self-reported frequency of unusual beliefs and convictions (top) and perceptual symptoms (bottom) endorsed by BPD participants ($N=74$) on the CAPS and PDI follow-up questionnaire. Participants were encouraged to answer regarding the CAPS and PDI item(s) they perceived as the most distressing and/or impactful.

5.4 Discussion

Compared to the control group, BPD participants reported a significantly higher number of psychotic experiences which occurred more frequently, were perceived as more intrusive, and associated with greater levels of distress (even after correcting for the number of symptoms endorsed). For hallucinatory experiences, the distribution of symptoms in the BPD group was remarkably similar to the pattern typically observed in inpatients with psychosis (Bell et al., 2006; Bell et al., 2011), whereas scores in the control group reflected the expected distribution of a non-clinical sample (Bell et al., 2006; Johns and Van Os, 2001). When asked about the average duration of their perceptual experiences, answers diverged, with 35% (26/74) of participants reporting predominantly minute-long symptoms, 27% (16/74) describing day-long experiences, and 19% (14/74) saying their perceptual symptoms are continuous. Since participants were encouraged to answer with regard to whichever perceptual symptom(s) they found the most distressing, this suggests that different types of hallucinatory experiences may be associated with different durations (e.g., AVH may be more likely to be continuous, whereas visual distortions could be more transient). Since participants were not asked to identify to which specific symptom(s) their answers pertain, it was unfortunately not possible to assess such link between symptom type and duration in the current study. In future, probing individual experiences for their onset, duration, and phenomenology, may provide crucial information about differences in aetiology or underlying mechanisms, and help eradicate the misconception that most psychotic experiences in BPD are short-lived.

Since previous research has largely focused on hallucinatory experiences, the present assessment with the PDI provided one of the first dedicated and detailed (quantitative) measures of delusional experiences in a BPD population. The distributions of PDI scores were in line with the original findings from Peters et al. (2004) for both, the control group, and the BPD sample, which followed the pattern of people with clinical delusions. Remarkably, BPD participants of both Studies scored higher than in-patients with psychosis on the PDI total (mean 8.5, SD 5.1 versus 6.7, SD 4.4) and all three subscales (Peters et al., 2004 in-patients: distress 15.5, SD (14.1); preoccupation 15.4, SD 14.1;

conviction 20.4, SD 16; see Table 5.1 for study results). This is vastly contrary to the assumption that psychotic symptoms in borderline are predominantly perceptual, and that prevalence of delusional ideation is low or pathologically insignificant. In a follow-up questionnaire that enquired about how frequently these beliefs recur, 65% of participants (48/74) described them as constant, daily, or at least weekly, further underlining the potentially substantial impact they can have on day-to-day life. Similarly, between 55-59% of the BPD sample scored above the proposed cut-off (Preti et al., 2007), indicating that more than half of the participants experienced unusual and disturbing thoughts of clinical significance. By comparison, previous findings have described the prevalence of regular delusional ideations in the general population as 10–15%, with 1-3% presenting with a severity comparable to clinical cases (Freeman, 2006; Freeman, 2007). McGrath et al. (2015) equally estimated the lifetime prevalence of delusional experiences to be around 1.3%. Overall, these results expose considerable shortfalls in the theoretical and experimental approaches to psychosis in BPD and emphasise the need for more thorough considerations of the delusional and thought-disturbance aspects of these experiences.

Bearing in mind the immense discrepancies regarding the prevalence of psychotic experiences, this study further attempted an instant replication of findings across two different BPD populations: a larger, international, self-report sample, and a smaller, local, clinical group. Outcomes of the comparison were striking with almost complete overlap for the number of experiences endorsed, as well as their impact (i.e., distress, invasiveness, and regularity). In light of these findings, it appears baffling how little phenomenological overlap there is between previous assessments (compare Table 1.1 in Chapter 1.2), and the evident troubles with replicability may perhaps be less influenced by sample type, size, or selection, and more due to the erratic use of a large variety of PE measures – an issue which can easily be remedied in future research by ascertaining the most appropriate instrument(s) and applying them consistently. Since measures designed for use in the general population (such as the CAPE, CAPS, and PDI) may not be able to fully capture the (clearly prevalent) clinically relevant dimensions of psychotic experiences in BPD (and measures designed for use in people with psychotic disorder are often biased towards very specific symptom presentations), it may be worth considering the development of an

instrument specifically designed to capture and reflect the nature and range of these symptoms in the context of borderline pathology.

Across both studies, participants with BPD were significantly more likely to endorse delusional ideations of persecution, thought disturbances, paranoia, and suspiciousness. The final two categories were especially prevalent (76-80% and 85-89% of the sample respectively) and are very much in line with the borderline associated emotional states of constant agitation, caution, and mistrust. By contrast, BPD and control group participants did not differ on the symptom categories religiosity, grandiosity, and paranormal beliefs. Notably, the symptom dimension “*religiosity*” was infrequently and inconsistently endorsed across all four participant groups and begs the question of a perhaps necessary update. Already accounting for the highest proportion of variance during the questionnaire’s validation in 2004, societal and personal stances towards religion have changed markedly over the past twenty years. The specifically Christianity-associated phrasing of the items may not feel relatable to people of other faiths, not to mention the increasing number of atheist or agnostic participants likely to be partaking in the experiment. Conversely, a number of participants who did identify with the Christian belief system approached the experimenters during and after the study to voice their concerns and dissatisfaction about seeing their faith labelled as delusional. Reminiscent of the “electrical devices” dilemma that befell interpretation of the CAPE results in the previous study, in this case a phrasing that during a time of more widespread Christian belief would have made sense to assess potentially grandiose levels of faith, now only serves to alienate both those who do and those who do not ascribe to religion.

Rates of auditory non-verbal hallucinations (45-56%) in the BPD sample were comparable to previous findings by Kingdon et al., (2010), Pearse et al. (2014) (both 50%), and Slotema et al. (2019; 40%). By contrast, the prevalence of auditory verbal hallucinations was higher than observed in previous studies, with 39% of BPD participants in Study 1, and 55% of those in Study 2 endorsing one or more types of AVH. By comparison, Niemantsverdriet et al. (2017) reported that 21% of their sample was hearing voices, Slotema et al. (2017) 30%, and Yee et al. (2005) 29%. It is possible that the decreased sample size of Study 2 (20 participants, in contrast to 323, 89, and 171 in the three studies above, respectively)

had an impact on the estimate. However, it would also be interesting to know whether the definition of this symptom category might have differed between studies. For instance, the CAPS summarises three items (11, 13, 28) as verbal, one of which (11, persistent commentary) would be considered a first-rank symptom while the other two pertain to hearing multiple voices converse with each other (28), and simply “hearing a voice when alone” (13). As such, some questionnaire measures or structured clinical interviews may be stricter in their definition of AVH, driving prevalence estimates down.

Two perceptual experience categories that are rarely considered but were overwhelming endorsed by people with BPD are “*changes in levels of sensory intensity*” (83-90%) and “*temporal lobe abnormalities*” (70-90%). The former points towards a dysregulation in sensory gating – the process of filtering incoming sensory stimuli and removing redundancies to prevent an information overload. Deficits in (especially auditory) gating have repeatedly been associated with both post-traumatic stress (Neylan et al., 1999; Ghisolfi et al., 2004; Meteran et al., 2019), and schizophrenia (Thoma et al., 2003; Hamilton et al., 2018). If people with BPD may be affected by a similar perturbation of their sensory filter, this would not only provide a mechanistic link between the psychotic experiences of both disorders but could also offer a plausible explanatory pathway from trauma to psychotic experiences, via gating dysregulation. Therefore, future investigations of this process (or the neural pathways it involves) may shed further light on the cognitive underpinnings of psychotic symptoms in BPD.

An alternative (or perhaps complimentary) mechanistic process may be evidenced by another perceptual symptom category overwhelmingly endorsed by participants with BPD: *Temporal lobe abnormalities* were so named by the creators of the CAPS due to their similarities with the visual and auditory phenomena observed in the context of temporal lobe epilepsy (Bell et al., 2006). Their high prevalence in both BPD populations could point towards (some) shared neurological mechanisms between psychotic experiences in epilepsy and those in borderline. Similar comparisons have previously been drawn by Harris et al. (2002), but no further investigations of their phenomenological or neurological overlap have been attempted since. Overall, while there are a series of undeniably more apparent aetiological and mechanistic models that perhaps require foremost attention, this

observation should be considered in future ventures into the neurological basis of psychotic experiences in people with BPD.

Finally, the exploratory assessment of self-identified “triggers” for people’s psychotic experiences yielded preliminary evidence in support of the often-claimed link between PE and stress in BPD (Yee et al., 2005; Adams and Sanders, 2011; Schroeder et al., 2013; Schultz and Hong, 2017; D’Agostino et al., 2019; Minarikova et al., 2022): Half of the participants (37/74) reported that their experiences can be brought on or exacerbated by stress. Negative affect (including worsening mood and worsening anxiety) was equally cited as a major factor, which further supports the hypothesis that common mental distress and heightened emotional states may play a role in the aetiology of these symptoms.

Chapter 6 What lies beneath – assessing risk factors and mechanisms

“Most experiences happen in rooms where I felt unsafe or where I experienced trauma. I have seen things like snakes and spiders that weren't there, and they have made me doubt reality. In these places I sometimes forget where I am and briefly see my childhood bedroom, even though I know it has been changed multiple times since I have left. I hear my parents calling my name from outside, even though I know they're nowhere near. These tend to happen more when I am stressed.”

6.1 Introduction

One might argue that, under the right circumstances, anyone could (and would) develop psychotic symptoms, and the list of ways in which the mind can be coerced into creating new realities is as long as it is varied. They range from dementia (Cerejeira et al., 2012) and brain injury (McAllister, 1998) to poisoning (Ekino et al., 2007) and infection (Kulaga and Miller, 2021), from extreme physiological states, such as starvation (Mittal and Khan, 2010) and sleep deprivation (Waters et al., 2018) to psychological states such as trauma (Gibson et al., 2016) and bereavement (Castelnovo et al., 2015) – needless to mention many psychoactive drugs, both recreational and prescriptive. Ascertaining which and how many of these (or other) factors might underlie the development of psychotic symptoms in BPD is a vital step toward understanding the complexities of borderline symptomology and its implications for treatment, care, and quality of life.

Predictive processing – a cognitive framework

Predictive processing models of psychosis broadly conceive its core features – delusions and hallucinations – as a consequence of altered balance between prior expectations about incoming sensory signals and the signals themselves (Fletcher and Frith, 2009). By framing perception as a Bayesian process of prediction error minimisation and probabilistic inferences, this approach offers a plausible explanation for the positive symptoms of psychosis: If our subjective experience of the world is merely the brain’s “best guess” about the causes of signals, then erroneous guesses might lead to aberrant salience (the incorrect assignment of importance to stimuli that would normally be considered neutral or irrelevant), driving the development of a distorted version of reality that may manifest psychiatically as hallucinations and delusions. On a neurochemical level, the implications of aberrant salience are evidenced by the robust body of literature linking dopaminergic (dys)function to psychotic symptoms, including imaging studies of the dopamine system in patients with schizophrenia (Zakzanis and Hansen, 1998; Kegeles et al., 2010), and the palpable efficacy of dopamine D2 receptor blockers as antipsychotic treatments (Kapur, 2003; Seeman, 2010). Unlike other cognitive models (which tend to rely on separate explanatory mechanisms for hallucinations versus delusions), the predictive processing theory thereby grants a unified understanding of both, perceptual symptoms and unusual beliefs, as disruptions of a shared core mechanism, while also providing a link to one of the earliest and most widely established pharmacological theories of psychosis.

In the context of borderline PD, the predictive processing model offers a mechanistic framework for assessing underlying cognitive differences that may increase the risk of psychotic experiences. Altered prior expectations (for example, due to past trauma) could profoundly bias how sensory signals are processed, distorting incoming information in accordance with a threatening and unstable view of the world and thereby leading to the development of psychotic experiences. Experimentally, this would be evidenced by people with BPD showing greater reliance on top-down influences from prior knowledge (over bottom-up influences from sensory information), compared to healthy controls – a shift that has previously been observed in people with early psychosis, and psychosis prone individuals (Teufel et al., 2015). Furthermore, the greater reliance on prior information

would be more pronounced in individuals with BPD and high psychotic (especially perceptual) symptom scores. It would also be expected that the degree of this shift correlates with the severity of BPD symptomatology, as well as with adversity and trauma.

PTSD and dissociation – a trauma-response framework

The link between trauma and psychotic experiences has long been described by survivors of trauma, who relate both the emergence, and the content of their symptoms to past (or ongoing) adversity. This includes qualitative descriptions and statements made by participants of the current studies (*“Most experiences happened in rooms where I felt unsafe or where I experienced trauma.”*, *“Triggers influence the experience: usually something like shouting, aggressive behaviour, or loud noises - I believe due to past trauma”*³⁵), as well as from previous accounts (*“I think that’s why I hear voices, it’s because I was sexually abused [...] and I can see how these different mental health experiences are really clearly linked to what happened to me, through like the content of my voices.”*, Campodonico et al., 2022; *“One service user heard their abuser saying: ‘you’re fat, stupid and no one will believe you’. Another experienced images and voices relating to the loss of their children. [...] One participant heard a crying baby which she related to a termination.”*, Adam and Sanders, 2011). The connection has also repeatedly been reported across quantitative studies in individuals with psychotic disorder (Daalman et al., 2012; see Bailey et al., 2018 for a meta-analysis), major depression (Gaudiano and Zimmerman, 2009), as well as in the general population (Read et al., 2003; Daalman et al., 2012; Van Den Berg et al., 2023). Whether this relationship is causal or modulating, direct or mediated, and how it applies in the context of BPD remains unclear. Moreover, it is uncertain whether (and to what extent), the number of adverse events, the type of trauma, or the age at which it was experienced factor into this relationship. Adam and Sanders’ (2011) observations suggest a particular connection between hallucinations and childhood sexual abuse. Others similarly found a direct (as well as an indirect) effect of childhood abuse on PEs, possibly mediated by adulthood adverse life events (Bhavsar et al., 2019). It may be that early-life adversity leads to fundamental neurodevelopmental

³⁵ Comments from the CAPS follow-up questionnaire, submitted by two BPD participants of Study 2..

changes which subsequently increase the risk for psychotic symptoms. Alternatively, PEs could be more closely linked to the ongoing psychological impact of trauma (e.g., in form of PTSD), and both hypotheses are subject to continued investigation (Read et al., 2014; Stanton et al., 2020; Barnes et al., 2023; Allardyce et al., 2023; see Inyang et al., 2022 for a recent systematic review on the subject).

The search for potential mediators has uncovered similarly conflicting findings. While some studies reported a potentially mediating role of dissociative experiences for childhood trauma and hallucinations (Varese et al., 2012) but not delusions (Perona-Garcelán et al., 2012; Muenzenmaier et al., 2015), others contrarily found an indirect effect of dissociation on the relationship with delusions, but not hallucinations (Sun et al., 2018), and one research group observed no significantly mediating effect of either dissociation or any other hypothesised (affective) predictors at the point of transition from UHR to psychosis (Thompson et al., 2016). Despite these gaps in our understanding of the exact nature of the relationship, it is plainly apparent that trauma and dissociation (independently or in interaction) have an influence on whether and how psychotic symptoms manifest. Therefore, considering the high prevalence of both factors in people with BPD, it is exceedingly plausible that they may also affect hallucinatory and delusional experiences in this population.

Common mental distress and symptom severity – a p-factor framework

Along with the burgeoning theory of a psychosis spectrum came an increase in explorations of the nature and prevalence of PEs in community samples. Research into population level risk factors followed suit and common mental distress (severity) soon emerged as a (relatively) consistent predictor for psychotic experiences in non-clinical populations (Perez et al., 2018), non-psychotic clinical populations (Wigman et al., 2012; Bhavsar et al., 2019), and people in UHR states (Heinze et al., 2018). Of course, the directionality of this relationship is highly contentious and it is much too early to confirm whether high levels of depression and anxiety truly increase the risk for psychotic symptoms – or whether such experiences are simply so distressing that they raise the levels of depression and anxiety. However, these findings do point towards a covert dimension of underlying

psychiatric vulnerability, in which mental distress begets further distress, conceptualised by Lahey et al. (2012) as the “*p-factor*”. Originally devised to explain the significant overlap (and frequent comorbidities) between internalising and externalising disorders, the paradigm was subsequently extended to also include psychotic symptoms (Caspi et al., 2014). Remarkably, of all assessed psychiatric diagnoses, BPD consistently shows one of the highest loadings onto this latent factor of overall psychopathology (Caspi et al., 2014; Ringwald et al., 2021; Southward et al., 2023), suggesting that vulnerability to psychiatric symptoms (including psychosis) may be markedly higher than in other conditions. Empirically, this is evidenced by the high rates of comorbidities in patients with BPD. In this context, the association between psychotic experiences and number of psychiatric diagnoses (Niemantsverdriet et al., 2017) – which was (in part) replicated by the current study (Chapter 4.2.2.) – is equally noteworthy.

Besides, the high prevalence of (severe) common mental distress may have a more direct influence on psychotic experiences. While psychotic disorders such as schizophrenia are typically associated with negative symptoms (including anhedonia, apathy, and emotional blunting), affective psychosis has long been recognised as a theoretical and clinical construct (most commonly in the context of bipolar disorder) in which heightened affect appears to accompany the development of hallucinations and delusions. Given that people with BPD spend considerable amounts of time in intense emotional states, that are often indistinguishable from bipolar disorder (so much so that the similarities in phenomenology bear the risk of misdiagnosis (Ruggero et al., 2010), psychotic experiences in borderline may be driven by similar (affective) mechanisms.

So many theories, so little time

Patently, the above does not reflect the full scale of neuroanatomical, neurological, cognitive, genetic, psychological, and pharmacological (not to mention spiritual) models of psychosis. Nor does it come close to relaying all the risk factors thought to be involved in its aetiology, in BPD and beyond. The intention is rather, to collate and probe some of the most common, current, and promising paradigms to narrow down the scope of theories and carve out a plausible foundation of the mechanisms of psychotic experiences in BPD

on which to build future research. With this in mind, the current chapter will focus on three main, conceptual targets:

1. The relationship between (childhood) trauma, post-traumatic stress, dissociation, and psychotic experiences.
2. The relationship between cognitive (predictive processing) mechanisms and psychotic experiences.
3. The relationship between BPD severity, common mental distress, and psychotic experiences.

As in the previous chapter, investigations into the causes and mechanisms of psychotic experiences draws on the data from both Study 1, and Study 2. Notably, some measures were applied in both experiments (CAPS, PDI, CTQ, PCL, and the two-tone image task), while others were swapped for a different instrument assessing the same pathology (depressive symptoms, anxiety symptoms, dissociative symptoms). Aside from establishing symptom prevalence and correlations, both studies gathered measures of the variables hypothesised to influence PE risk or severity. They were then used to inform a best-subset regression model and determine which factors might best explain (and most reliably predict) psychotic symptoms in BPD.

While any similarities and differences between study populations will be reported, the primary aim of the analysis was not to compare the psychopathology of different BPD samples, but rather to determine whether consistent and reproducible predictors for psychotic experiences can be ascertained. As such, results from Study 1 and Study 2 will be dissected independently, and comparisons will be reserved for the ensuing discussion.

6.1.1 Analysis

Analyses in this chapter were performed on the following data sets:

- *Psychopathology measures Study 1*: QIDS (depression), GAD-7 (anxiety), DES-II (dissociation), CTQ (childhood trauma), PCL (post-traumatic stress)

- *Psychopathology measures Study 2*: BDI (depression), STAI (anxiety), DEMO (dissociation), CTQ (childhood trauma), PCL (post-traumatic stress), LSC-R (life-time stressors)
- *Cognitive task measures Study 1*: the phantom words task, the two-tone image task
- *Cognitive task measure Study 2*: the phantom words task (pre stress condition)

For more information on the task and questionnaire measures see Chapter 3.2 (questionnaires) and Chapter 3.3 (tasks). Study procedures can be found in Chapter 3.4 (Study 1) and 3.5 (Study 2). Participant demographics are summarised in Table 3.3 (Study 1) and 3.5 (Study 2). Distribution of the data was assessed visually (histograms and Q-Q plots) and using Kolmogorov-Smirnov normality tests with Lilliefors correction. Non-normally distributed datasets are reported with the Shapiro-Wilk statistic. Unless otherwise stated, approximately normal distribution of the relevant measures was observed. All analyses and graphs were done in R.

Analysis of questionnaire measures

In Study 1, total symptom scores were obtained for the QIDS (depression), GAD-7 (anxiety), DES-II (dissociation), and PCL-5 (post-traumatic stress). For the CTQ, subscale scores for emotional abuse (EA), emotional neglect (EN), physical abuse (PA), physical neglect (PN), and sexual abuse (SA) were calculated in addition to the overall total.

In Study 2, total symptom scores were obtained for the BDI (depression), STAI (anxiety), and DEMO (dissociation). PCL-5 and CTQ were scored as in Study 1. For the LSC-R (life-stressor checklist), two scores were obtained: An item score, totalling the number of stressful events experiences (Scoring Option 1), and a subjective score that additionally considers the ongoing impact of these events on participants' everyday life (Scoring Option 2). Group differences for individual LSC-R items were assessed visually, and only obviously diverging item counts were selected for significance testing.

All scores were analysed as independent two-sample t-tests with diagnostic status as grouping variable. Welch's t-tests were employed in cases of unequal variance and are denoted as such. Furthermore, CTQ and PCL averages were compared between the BPD

samples of Study 1 and Study 2. *P* values of all serial comparisons were adjusted with Bonferroni correction.

Associations between symptom scores and psychotic experiences (as measured by CAPS and PDI yes/no scores) were analysed using Pearson's product-moment correlation. For the CTQ, correlations were assessed using the total score and the SA subscale. Correlations between psychotic symptoms and LSC-R were calculated using both scoring options.

Analysis of task measures

For the phantom words task (in the true-signal condition), *hits* were defined as correct identifications of the word underlying the sound illusion, *misses* were incorrect responses, *incorrect positives* were trials on which participants failed to identify a negative word and chose the positive option instead, and *incorrect negatives* were trials where a negative word was falsely selected instead of the positive option. In the non-signal condition (neither word option fully represented the underlying sound signal), negative word selections were categorised as *negative bias* and positive word selections as *positive bias*. True-signal *hits*, and non-signal *bias* were analysed as two independent t-tests with diagnostic status as grouping variable. The rates of *incorrect positives* and *incorrect negatives* were equally assessed with independent t-tests, both within and between groups.

For the two-tone image task, accurate identifications of the location of the red dot were defined as *hits* if the dot was on the figure, and as *correct rejections* when the dot was on the background. Trials in which participants falsely reported the dot to be on the figure were classed as *false alarms* and those in which they falsely reported the dot to be on the background when it was, in fact, on the figure were described as *misses*. Visual discriminability (d') and response bias towards reporting more dots on the figure than on the background (β) were assessed at baseline and after template exposure. Baseline performance, and differences in d' and β before and after template exposure were analysed as independent samples t-tests, comparing BPD participants to the control group. The R package [psycho] was employed to create d' and β estimates.

Best subsets selection

Best subsets regression is a model selection approach that facilitates identification of a set of explanatory variables that most effectively predict the response variable. Unlike stepwise selection (which only compares a limited number of models), best subsets regression evaluates each combination of explanatory variables and returns a selection of n models (where n is the sum of predictors incorporated), proposing the best subset for each factor level (i.e., the best 1-variable model, the best 2-variable model, etc).

To ascertain the most reliable predictors for psychotic symptoms in each study population, four best subsets selections (two per data set) were performed, with perceptual experiences (CAPS total) and delusional thoughts (PDI total) as the respective response variables. Since linear regression models assume an association between explanatory and response variables, only outcome measures that were found to significantly correlate with CAPS and PDI (including full-sample correlations) were included as possible predictors. Best subsets were computed using the `regsubsets` function from the R package `[leaps]`.

For increased statistical power, regressions were performed using the full participant populations (BPD and control group), with diagnostic status as grouping variable in Study 1. In Study 2 this was replaced by the Borderline Symptoms List (BSL) since a (dimensional) measure of BPD pathology was better suited to regression analysis. Similarly, the LSC-R was substituted for the PCL as a measure of post-traumatic stress, as it assesses both the number of traumatic events and their (accumulative) current impact and was more strongly correlated with psychotic experience scores.

The resulting subsets were compared on three indices to determine the model with the best overall fit. The following metrics were used:

1. Adjusted R^2 (proportion of variance in the response variable explained by the predictors, adjusted for the number of predictors; a higher R^2 indicates better fit)
2. Mallows' C_p (estimate of the sum squared prediction error; a lower C_p indicates better fit)

3. The Bayesian information criterion (BIC; based on maximum likelihood, with penalties for the number of predictor variables; a lower BIC indicates better fit)

Non-consensus between the selection metrics (as is routinely the case in best subsets selections) was resolved by using k -fold cross-validation ($k=5$)³⁶ to compute each model's prediction error, which was consulted as an additional goodness-of-fit indicator. Cross validations were performed using the `train` function of the R package [`caret`].

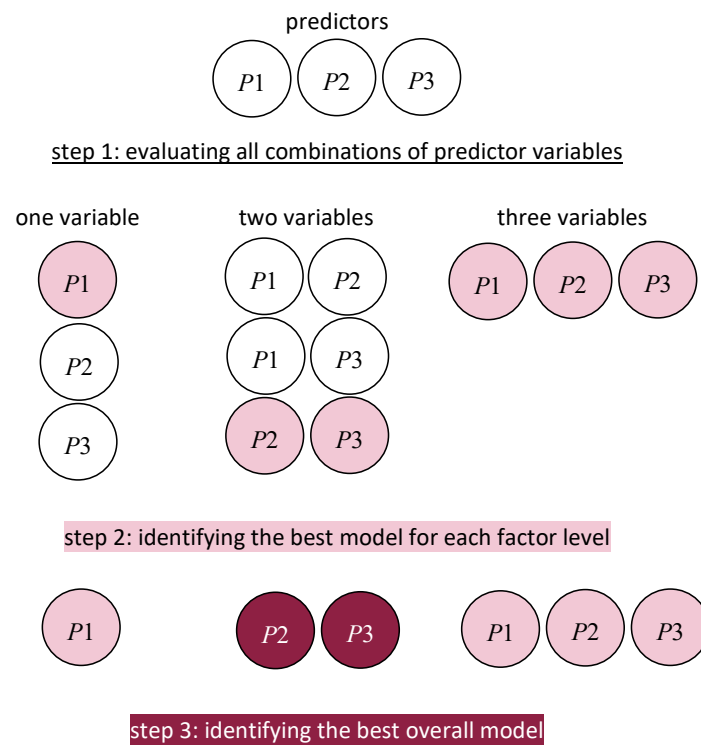


Figure 6.1

Illustration of the best-subset regression computational procedure (top to bottom).

³⁶ Cross validation techniques can be used to create a new data set, and then apply the model to calculate its prediction error. The k -fold cross-validation achieves this by dividing the original data into k -subsets, each of which subsequently serves as a “test” data set that is cross-validated with the remaining $k-1$ “training” data sets. The mean error of all comparisons is subsequently returned as the model’s overall prediction error. Compared to other model selection criteria (including adjusted R^2 , BIC, and Marlow’s C_p) which are computed using the training data, cross validation is less prone to overfitting. K-fold techniques are typically computed with $k=5$ (Arlot and Celisse, 2010).

Following subset selection, the chosen model was tested for overall suitability, significance, and variance inflation factor. Model residuals were assessed visually for normality (histograms and Q-Q plots), linearity (residuals plot), and homoscedasticity (scale-location plot). Suspected heteroscedasticity was assessed using Breusch-Pagan test. If significant, weighted least squares regression was performed and the model was reassessed. All reported statistics and graphs are of the final regression.

Structural equation modelling

Structural equation models (SEM) can be used to quantify the interactions and causal relationships between latent variables (LVs), by combining confirmatory factor analysis (CFA) and linear path models. This makes SEMs uniquely suitable for evaluating links between unobserved psychopathological constructs (e.g. “*trauma*” and “*common mental distress*”) and test their direct, indirect, and accumulative effects on a (latent) response variable (e.g. “*psychosis*”). Structural equation modelling involves two steps:

1. Defining and validating a measurement model. This is typically accomplished by CFA during which latent constructs are specified using observed variables (“factor loadings”), covariances are analysed (and removed, if necessary), and fit indices are consulted to ensure that the hypothesised factors are reliable enough to be included in the structural model.
2. Applying the hypothesised structure to the CFA model (i.e. fitting the SEM), including all relevant paths and any additional observed variables not already included as factor loadings. The structural model is then evaluated for overall fit (using the same metrics as CFA), and its structural coefficients can be interpreted.

While an SEM can almost always be improved a posteriori, good practice typically demands little to no post-hoc modification to the originally postulated structure, unless severe misfit of the model necessitates re-specification. For a comprehensive overview of SEM theory and applications see “*The principles and practice of structural equation modelling*” (Kline, 2016).

To guarantee sufficient statistical power, minimal bias, and meaningful solutions, SEMs typically require large sample sizes – with no consensus on what size exactly can be considered large enough. Absolute estimates have repeatedly been set at 300 (Comrey and Lee, 1992; Tabachnick and Fidell, 2019), however requirements appear to vary drastically (between 30-460 cases according to Wolf et al., 2013) and are likely to be influenced by patterns of association between parameters and observations. Common rules of thumb include a minimum of 10:1 (Schreiber et al., 2006) or 20:1 (Kline, 2016) participants per estimated (latent) variable.

Despite the limitations of a (comparatively) small sample, the significant overlap in psychiatric symptomology between the two studies' BPD groups, their (largely) shared methodologies, as well as the consistent and transferable findings of the best subset (and subsequent multiple-linear) regression models, afforded the intriguing opportunity to combine both populations and explore the relationships between the various psychopathological risk factors for PEs in BPD within a structural equation framework.

Accordingly, data sets of the BPD groups from Study 1 and Study 2 were aggregated ($N=74$) and the following measures were extracted as indicator variables:

- *Psychotic experiences*: CAPS and PDI total (yes/no) scores
- *Trauma and post-traumatic stress*: PCL and CTQ
- *Common mental distress*: QIDS and GAD (Study 1), BDI and STAI (Study 2)
- *Dissociation*: DES (Study1), and DEMO (Study 2)

Measures that had not been repeated (or substituted) across both studies were excluded from analysis (LSRC and BSL, specifically). An initial principal component analysis (PCA) was performed for each set of indicators (e.g., PCL and CTQ for the LV *Trauma*), with the aim of identifying potentially significant factor loadings, however no reasonable number of components with a sufficient cumulative proportion of the total variance could be ascertained for any of the latent variables. Instead, total scores were scaled using z-score normalisation, which served to standardise the features of those psychopathology measures that differed between studies (DEMO/DES, BDI/QIDS, and GAD/STAI), as well as to

rescale the ranges of all predictors, thus ensuring approximately proportional contribution to the model.

Convergent validity of the latent constructs was assessed with CFA using Maximum Likelihood (ML) estimation. The following metric indices were consulted to ascertain adequate model fit:

1. χ^2_M – Absolute fit index, measuring the specified test model against a theoretical baseline model. If the chi-square value is $>.05$ we fail to reject the null hypothesis that the models are equal, therefore, non-significance indicates adequate fit of the test model. The χ^2 test is highly sensitive to non-normality, covariance of observations, standardisation of the sample covariance matrix, and very small (<75) or large (>400) sample sizes, making it the least reliable estimator.
2. Root Mean Square Error of Approximation (RMSEA) - Absolute fit index, measuring the average difference between predicted and actual values of the model. The RMSEA is the most commonly reported metric for CFA and SEM, with values of <0.05 indicating good, and values of <0.01 excellent fit, while values up to <0.08 are still considered acceptable (MacCallum et al., 1996). The measure is sensitive to low degrees of freedom and is positively biased (more likely to be too high). Typically, a 90% confidence interval is computed to assess precision of the RMSEA estimate.
3. Standardised Root Mean Square Residual (SRMR) - Absolute fit index, measuring the standardised difference between observed and predicted correlation. A value of $<.08$ suggests adequate fit (Hu and Bentler, 1999) with zero indicating perfect fit. SRMR uses no penalty for model complexity and is also prone to positive bias, which may increase with smaller sample size.
4. Comparative Fit Index (CFI) – Incremental fit index, measuring non-centrality dependent on the correlations between variables. Values close to 1 indicate good model fit.

Following assessment of the measurement model, its structural components were estimated within an SEM framework. In addition to the latent constructs, the observed variable

Dissociation (standardised scores of the Study 1 DES and Study 2 DEMO) was included as mediator. The structure was specified as follows:

CMD	=~	Anxiety + Depression	=~ defines latent variables by their indicators
Trauma	=~	CTQ + PCL	
Psychosis	=~	CAPS + PDI	
CMD	~	Trauma	~ describes a regression path
Dissociation	~~	CMD	
Psychosis	~	CMD	~~ describes a correlation
Psychosis	~	a*Trauma	
Dissociation	~	b*Trauma	
Psychosis	~	c*Dissociation	
indirect effect	=	b*c	
total effect	=	c+ (b*c)	

All variables followed a multivariate normal distribution (assessed visually and using Mardia's test for multivariate skew and kurtosis) and there was no missing data. Model parameters were expressed in `lavaan` and computed using the `sem` command, with ML estimation. Model visualisations were created with `semtools`. All analyses and graphs were done in R.

6.2 Results of Study 1

6.2.1 Analysis of questionnaire and task measures

Common Mental Distress (QIDS and GAD-7)

As expected, participants with BPD reported higher rates of common mental distress, and groups differed on the QIDS ($t_{94}=9.21, p < .001$) and the GAD ($t_{90}=6.02, p < .001$). In the BPD group, severity of depression and anxiety symptoms correlated moderately with both the CAPS (QIDS $r=0.323, p=.017$, and GAD $r=0.372, p=.006$), and the PDI ($r=0.310, p=.023$, and $r=0.360, p=.007$ for QIDS and GAD respectively). Similar correlations were observed in the control group (QIDS $r=0.409, p<.001$, and GAD $r=0.329, p=.006$, as well as QIDS $r=0.315, p=.009$, and GAD $r=0.273, p=.024$, for correlations with CAPS and PDI respectively).

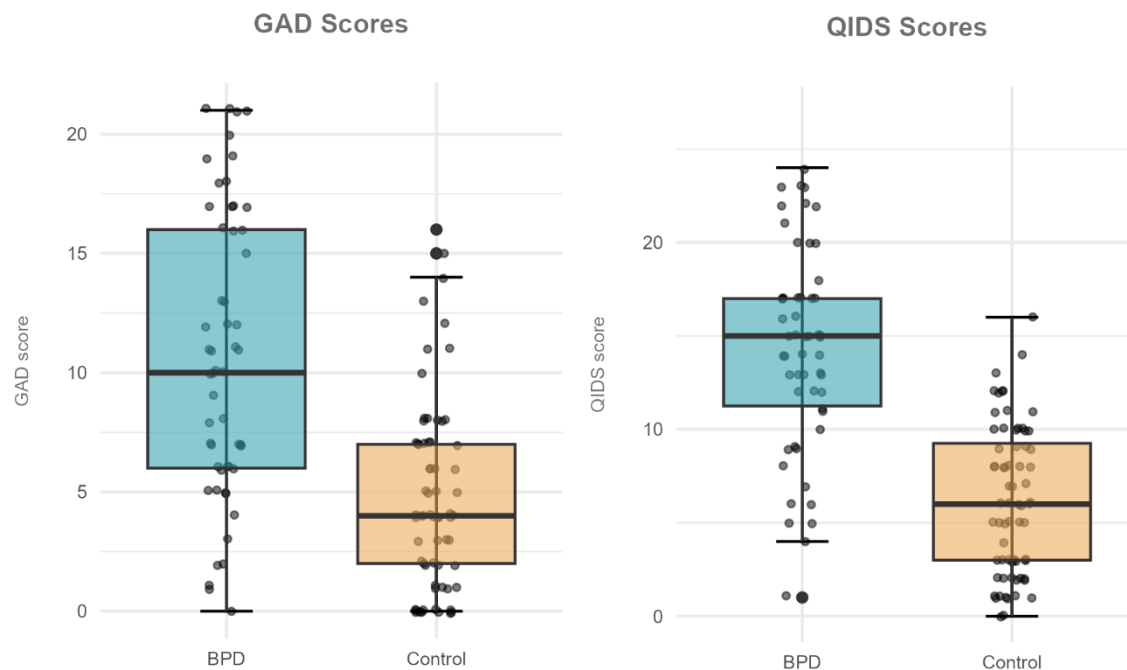


Figure 6.2

Box plots depicting mean and interquartile range of the GAD (anxiety; left) and QIDS (depression; right) scores of Study 1.

Differences were significant for both QIDS ($t_{94}=9.21, p < .001$) and GAD ($t_{90}=6.02, p < .001$).

Childhood trauma and post-traumatic stress (CTQ and PCL-5)

On the CTQ, BPD participants reported significantly greater childhood adversity ($t_{93}=7.53, p < .001$), and had higher incidence rates than controls across all five experience categories³⁷ (at $p < .001$) (Figure 6.3). The most frequently endorsed types of trauma were emotional abuse and emotional neglect, while physical or sexual abuse appeared proportionately less common (Table 6.3, in comparison with Study 2).

In the BPD group, the CTQ total ($r=0.375, p < .005$) and the SA subscale ($r=0.311, p < .022$) were moderately correlated with CAPS scores. This is in line with previous findings linking hallucinations to incidences of trauma and especially to childhood sexual abuse (Varese et al., 2012). In the control group no association between CTQ and perceptual

³⁷ CTQ subscales: Physical Abuse (PA), Physical Neglect (PN), Emotional Abuse (EA), Emotional Neglect (EN), Sexual Abuse (SA)

abnormalities could be determined, although the large percentage of participants scoring (near) zero may have precluded an accurate measure of dispersion in this sample. There was no discernible relationship between CTQ and PDI scores (at a 95% CI) in either population which suggests that in the aetiology of psychotic experiences in BPD, childhood trauma may play a different role for perceptual symptoms, than for delusional ideation.

The groups also differed on the PCL-5 ($t_{103}=9.83$, $p < .001$) with BPD participants endorsing significantly more symptoms associated with current post-traumatic distress. A previously recommended clinical cut-off (≥ 31 , Bovin et al., 2016) suggested that 77.8% of BPD participants (42/54) could meet diagnostic criteria for comorbid PTSD. 17.6% of controls (12/68) also scored above the threshold. However, it should be noted that this cut-off was determined in a veteran population and may have limited generalisability, especially when assessing people with psychiatric disorders. PCL scores were moderately correlated with CAPS ($r=0.371$, $p=.006$) and PDI ($r=0.444$, $p<.001$) in both populations³⁸.

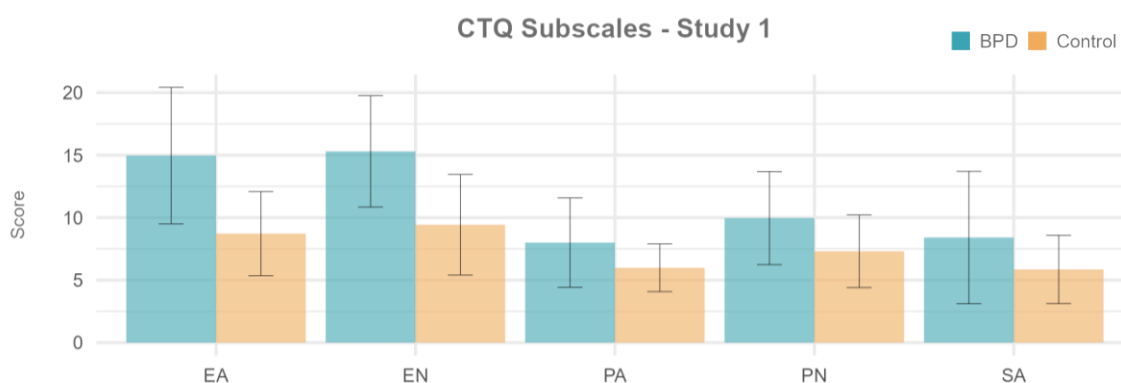


Figure 6.3

Bar plots with error bars for CTQ subscale scores of Study 1. EA=emotional abuse, EN=emotional neglect, PA=physical abuse, PN=physical neglect, SA=sexual abuse. BPD and control participants differed significantly across all five categories (EA: $t_{84}=7.37$, $p < .001$; EN: $t_{108}=7.54$, $p < .001$; PN: $t_{98}=4.30$, $p < .001$; PA: $t_{77}=3.74$, $p < .001$; SA: $t_{75}=3.22$, $p = .002$).

³⁸ BPD group coefficients shown in brackets. Control group correlations: $r=0.479$, $p<.001$, and $r=0.412$, $p<.001$ for CAPS and PDI, respectively.

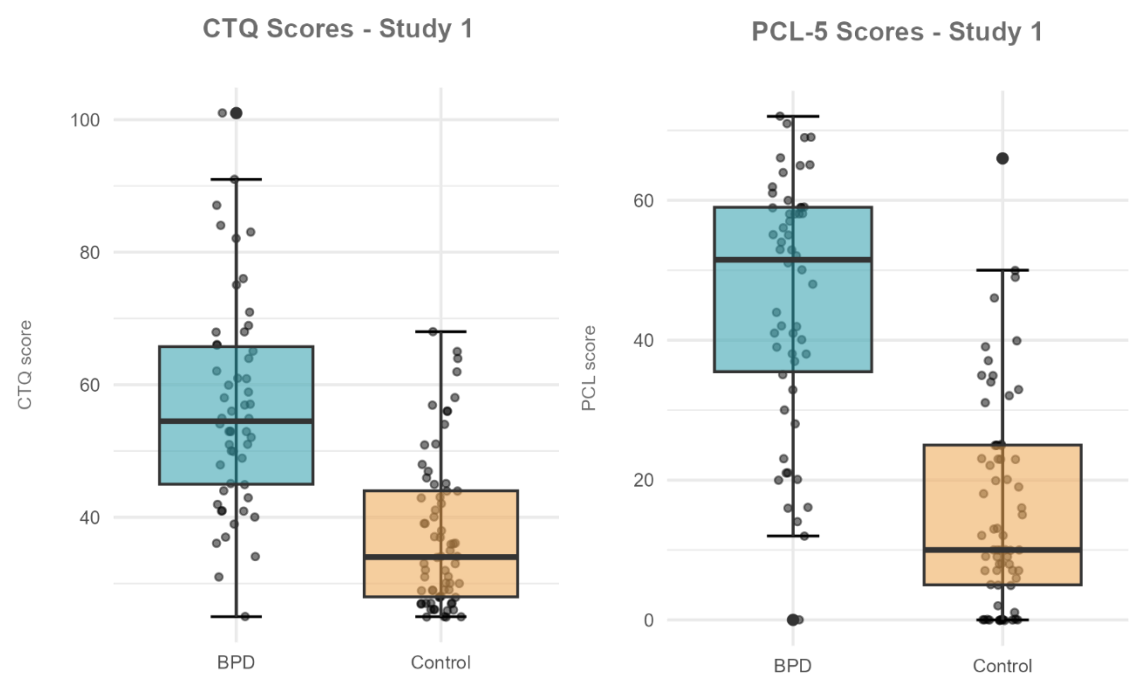


Figure 6.4

Box plots depicting mean and interquartile range of CTQ (childhood adversity, left) ($t_{93}=7.53, p < .001$) and PCL (post-traumatic stress, right) ($t_{103}=9.83, p < .001$) scores of Study 1.

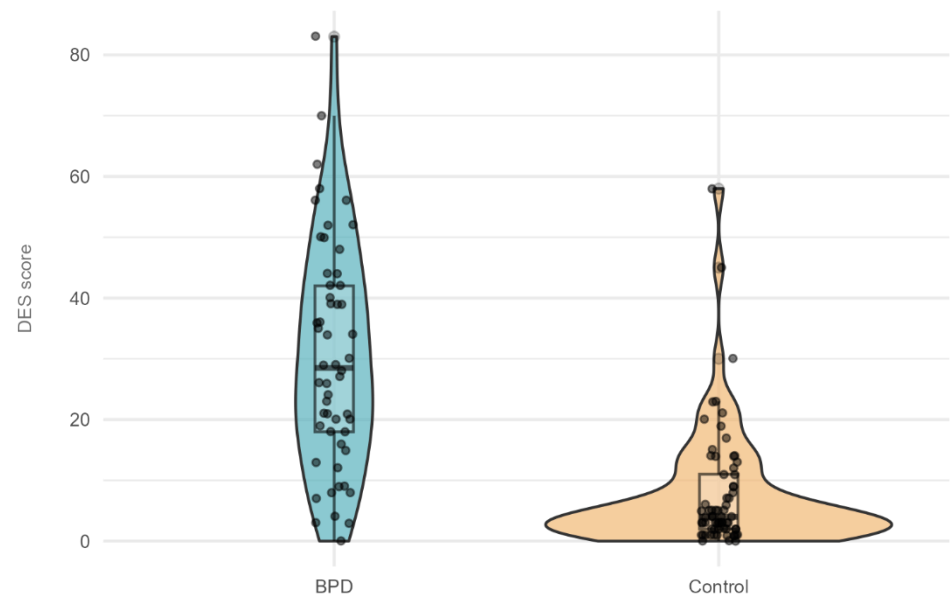


Figure 6.5

Violin plots for the DES (dissociation) scores of BPD and control participants of Study 1 ($t_{78}=8.06, p < .001$).

Dissociation (DES)

The strongest relationship between psychotic experiences and other aspects of psychopathology was observed for dissociative symptoms. DES scores were positively correlated with both CAPS ($r=0.733$, $p<.001$) and PDI ($r=0.761$, $p<.001$), and moderate positive relationships were retained at $p<.001$ in both groups (Figure 6.7 – CAPS, and Figure 6.8 – PDI).

Symptom frequency differed significantly between BPD participants and controls ($t_{78}=8.06$, $p<.001$) and the BPD group presented with a median DES score of 28.5 (mean 30.5, SD 18.5), comparatively higher than earlier findings (e.g., Coons et al., 1989³⁹; and Carlson, 1994⁴⁰; summarised in Table 1 of Carlson and Putnam, 1993). A total score of thirty has previously been proposed as a clinical cut-off for identifying people with dissociative disorder, or disorders with a considerable dissociative component (Carlson et al., 1993). In the present study, nearly half of the BPD participants (46.3%, 25/54) scored above this threshold, with an additional 24.1% (13/54) scoring between twenty and thirty, and 29.6% (16/54) scoring below twenty. By contrast, 91.2% of the control group (62/68) scored below twenty, 4.4% (3/68) between twenty and thirty, and 4.4% (3/68) above the clinical cut-off. However, since the DES measures both pathological and non-pathological forms of dissociation, the authors advise that high scores do not necessarily reflect severe psychopathology and may necessitate different interpretation in clinical versus non-clinical populations (Carlson and Putnam, 1993).

Ambiguous visual stimuli and the benefit of prior knowledge (two-tone image task)

The data from the two-tone image task was screened for technical errors, using the question about loading difficulties as indicator (none/some/many) for potentially biased results. Four of the BPD participants and seven controls reported “some” issues, however excluding their data did not affect the results and they were retained in the analysis. As expected, both groups showed improved perceptual discriminability (d') after exposure to the image templates ($t_{67}=5.71$, $p<.001$ and $t_{82}=-6.41$, $p<.001$ for BPD and control group,

³⁹ $n=13$, median 18.2

⁴⁰ $n=19$, median 20.1

respectively, Figure 6.6). Neither sample displayed a significant shift in response bias (β) towards reporting more dots on the figure ($t_{180}=0.22$, $p=.83$). The difference between before and after template exposure did not differ between BPD and control participants, neither for the d' ($t_{92}=-0.27$, $p=.78$), nor the β ($t_{92}=1.02$, $p=.31$). Previous findings have related two-tone image task performance (and by extension the ability to effectively integrate prior information to disambiguate low-information sensory stimuli) to an increased risk for psychosis (Teufel et al., 2015). These correlations could not be replicated in the current study, with no discernible association between CAPS and d' difference ($r=0.072$, $p=.49$). Equally, PDI scores did also not correlate with the rate of disambiguation improvement ($r=-0.091$, $p=.38$). Furthermore, the reduced baseline performance previously observed in people high schizotypal traits (Batty et al., 2014) and experiences of psychosis (Rivolta et al., 2014) did not apply to the BPD sample, with no differences in image disambiguation ability during pre-template trials, between groups ($t_{92}=0.12$, $p=.91$).

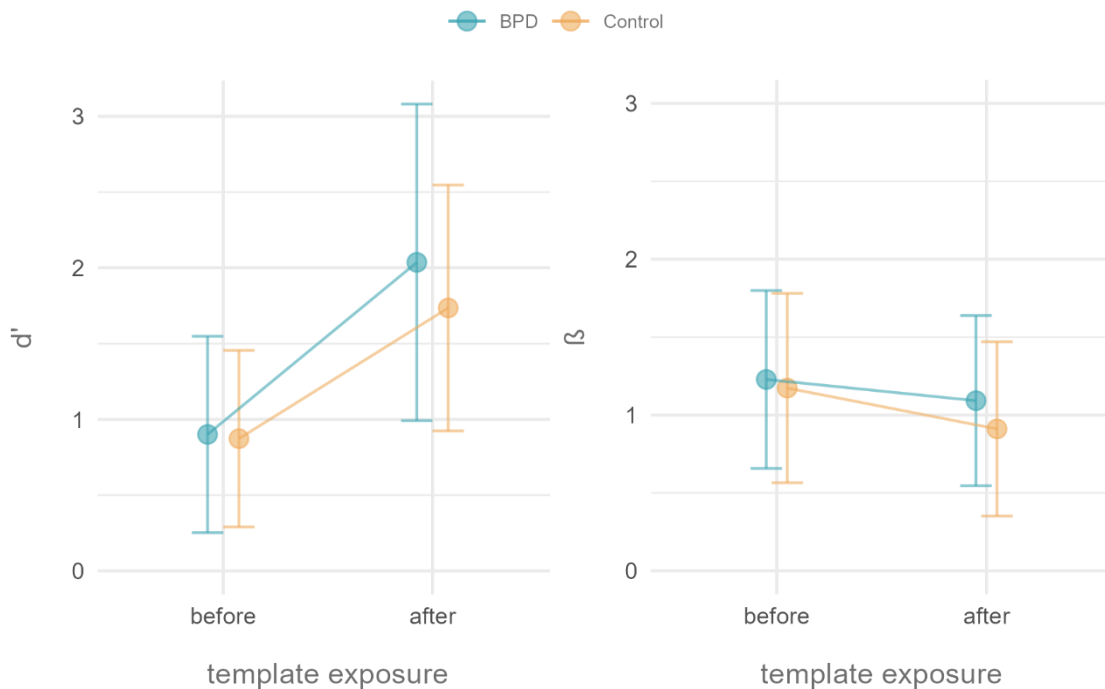
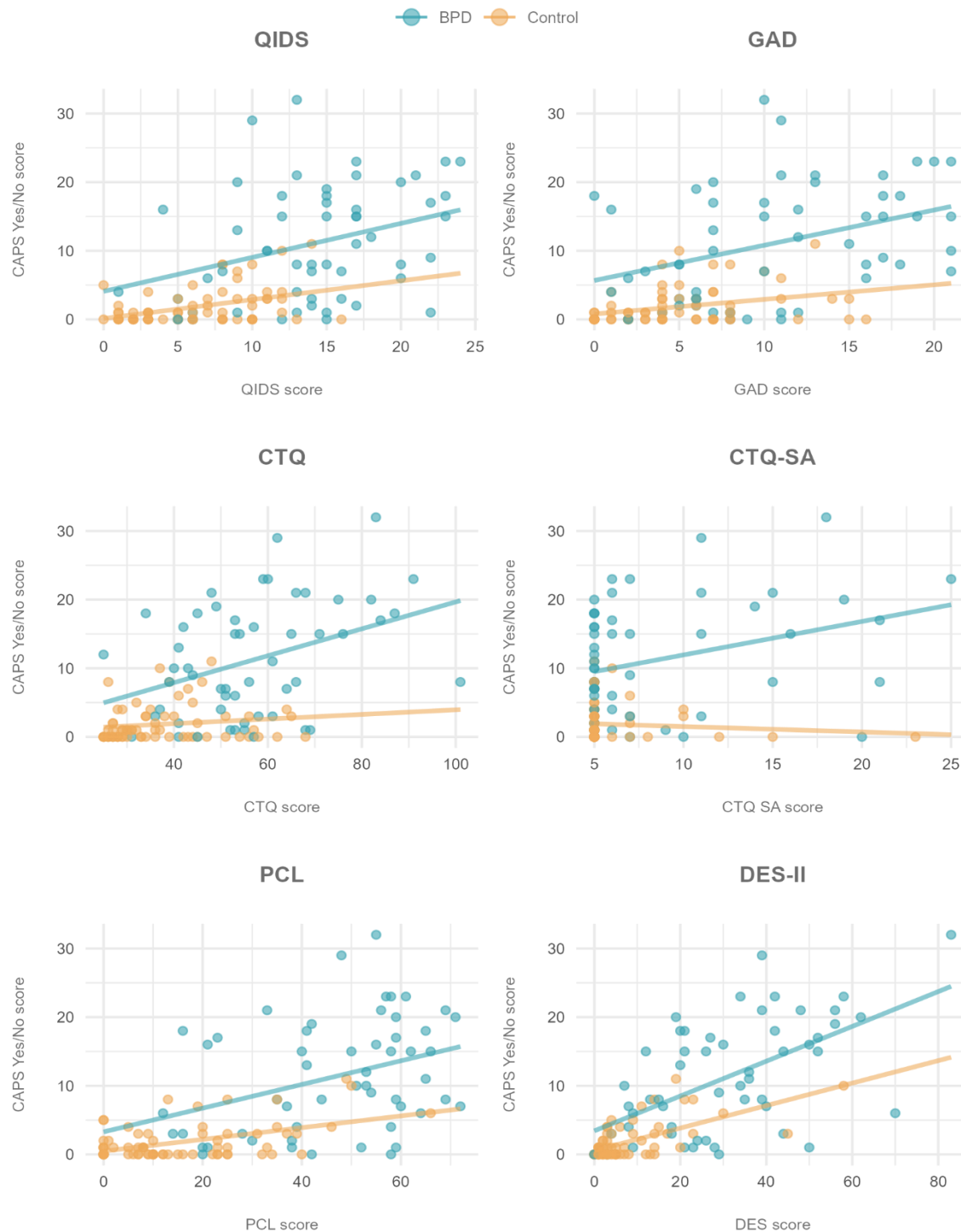
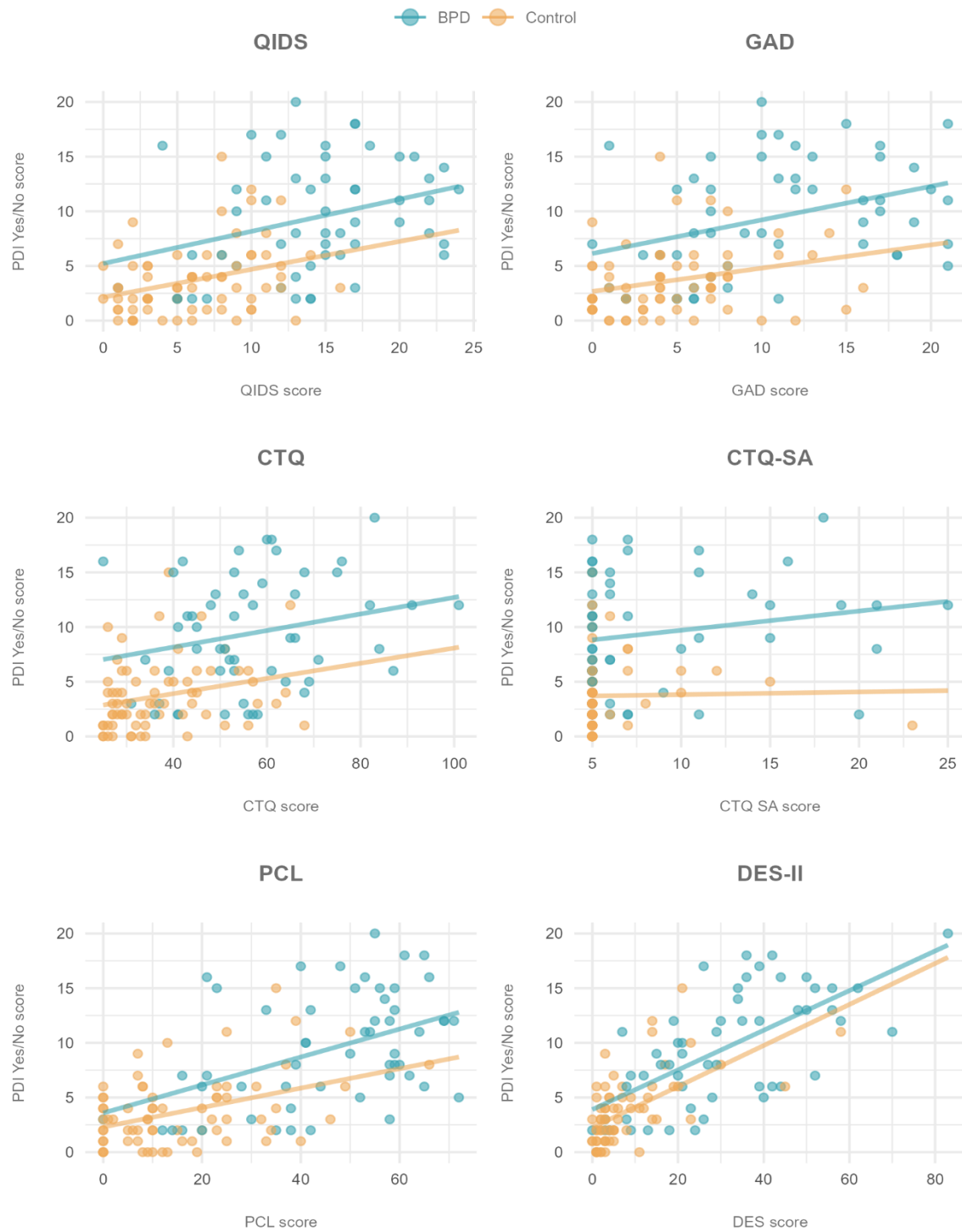


Figure 6.6

Perceptual discriminability (d' , left) of the two-tone images, and response bias (β) before and after template exposure for BPD and control participants of Study 1. To avoid overplotting, graphs are presented offset (jittered). There was no difference between groups.

**Figure 6.7**

Correlations between CAPS (yes/no) scores and other psychopathology measures. QIDS=depression (BPD $r=0.323$, $p=.017$, Control $r=0.409$, $p<.001$), GAD=anxiety (BPD $r=0.372$, $p=.006$, Control $r=0.329$, $p=.006$), CTQ=childhood adversity (BPD $r=0.375$, $p<.005$), CTQ-SA=childhood sexual assault (BPD $r=0.311$, $p<.022$), PCL=post-traumatic stress (BPD $r=0.371$, $p=.006$, Control $r=0.479$, $p<.001$), DES-II=dissociation (BPD $r=0.733$, $p<.001$, Control $r=0.629$, $p<.001$)

**Figure 6.8**

Correlations between PDI (yes/no) scores and other psychopathology measures. QIDS=depression (BPD $r=0.310$, $p=.023$, Control $r=0.315$, $p=.009$), GAD=anxiety (BPD $r=0.360$, $p=.007$, Control $r=0.329$, $p=.006$), CTQ=childhood adversity (BPD $r=0.234$, $p=.088$, Control $r=0.247$, $p=.062$), CTQ-SA=childhood sexual assault (BPD $r=0.181$, $p=.189$, Control $r=0.021$, $p=.867$), PCL=post-traumatic stress (BPD $r=0.444$, $p<.001$, Control $r=0.412$, $p<.001$), DES-II=dissociation (BPD $r=0.655$, $p<.001$, Control $r=0.596$, $p<.001$)

Ambiguous auditory stimuli and their emotional valence (phantom words task)

Reaction times to positive and negative stimuli did not differ between ($t_{98}=0.77$, $p=.44$), nor within groups ($t_{103}=-0.31$, $p=.76$ and $t_{128}=-0.22$, $p=.83$, for BPD and control group respectively).

In the non-signal condition (neither answer option corresponded to the true auditory signal) BPD participants showed a marked bias towards selecting the word with negative emotional valence ($t_{106}=-6.24$, $p<.001$) while control participants chose positive and negative words at equal rates ($t_{134}=0.48$, $p=.6$). This is consistent with the results of the phantom words pilot, which was conducted in a general population cohort (see Chapter 3.3.2). A Chi-square test of independence indicated that the proportions of positive and negative words also differed significantly between the BPD group and controls ($\chi^2_1=15.2$, $p<.001$) (Figure 6.9).

For word pairs containing a true signal, BPD participants were less proficient at identifying the correct stimuli ($t_{120}=-3.05$, $p=.003$) and more likely to instead endorse an ‘*incorrect negative*’⁴¹. The shift was significant when compared to the rate of incorrect negatives in control participants ($t_{120}=4.09$, $p<.001$), as well as to the rate of ‘*incorrect positives*’⁴² in the BPD group ($t_{106}=3.51$, $p<.001$). The control group exhibited no tendency towards either incorrect positives or incorrect negatives on their misses ($t_{134}=-1.24$, $p=.22$), which again mirrors pilot findings.

Though significant, this negativity bias appeared to be unrelated to both perceptual ($r=0.115$, $p=.41$) and delusional ($r=0.127$, $p=.36$) psychotic experiences (correlations in the control group were also non-significant). Instead, it may reflect a broader dimension of impairments in social cognition and emotion processing, frequently observed in people with BPD (Domes et al., 2008) and typically assessed in the context of visual perception (specifically facial recognition) – with shared mechanisms potentially underlying a similar deficit in the context of ambiguous auditory (verbal) information.

⁴¹ Selecting the negative word when the true signal is a positive word.

⁴² Selecting the positive word when the true signal is a negative word.

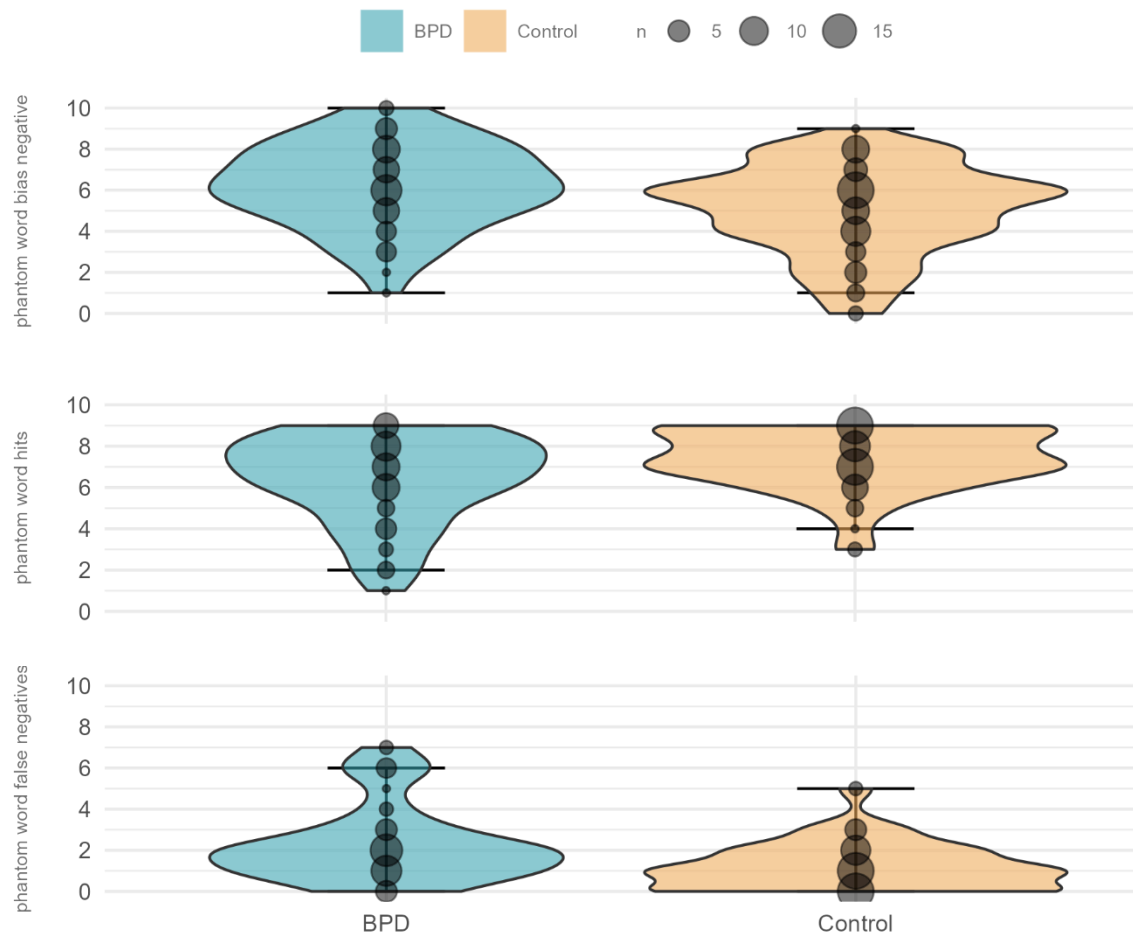


Figure 6.9

Violin plots of the number of phantom word *negative bias* selections (top) ($\chi^2_1=15.2, p<.001$), phantom word *hits* (centre) ($t_{120}=-3.05, p=.003$), and phantom word *incorrect negatives* (bottom) ($t_{120}=4.09, p<.001$) for BPD and control participants.

6.2.2 Further analysis: Predictors of psychotic experiences (best subset)

Based on the results from the correlation analyses, psychological outcome measures that were significantly associated with psychotic experiences were incorporated into a best subset regression, to identify which (and how many) variables most effectively predict delusional experiences. Models for perceptual experiences (CAPS) and delusional

thoughts (PDI) were assessed separately, since hallucinations and delusions may have different contributing factors (considering for instance the conflicting findings on the role of dissociation: Perona-Garcelán et al., 2012 and Muenzenmaier et al., 2015 versus Sun et al., 2018 and Thompson et al., 2016). As BPD participants (on average) scored significantly higher than controls across all psychopathology measures, diagnostic status was additionally added as a grouping variable.

Best predictors for delusional thoughts

Based on the PDI correlates, subset selection for delusional experiences was computed as follows: $PDI \sim DES + GAD + QIDS + CTQ + PCL + \text{diagnostic status}$. The maximum number of variables was specified as six (all), and none were forced in or out. The computation output rendered the best set of predictors for each model size (1-6, summarised in Table 6.1 and Figure 6.10). Models were compared on three metrics of accuracy (adjusted R^2 , C_p , and BIC), all of which endorsed a two-factor model (Model 2) incorporating dissociative experiences (DES) and post-traumatic stress (PCL).

Table 6.1

Best subsets of predictors for PDI (yes/no) scores in Study 1. Adj R^2 = adjusted R-squared, BIC=Bayesian information criterion, DS=diagnostic status, DES=dissociation, GAD=anxiety, QIDS=depression, CTQ=childhood adversity, PCL=post-traumatic stress.
*=best model based on the respective selection criterion, highlighted model=best subset overall. Xs denote which predictors are part of each model.

M	Adj R^2	Mallow's C_p	BIC	DS	DES	GAD	QIDS	CTQ	PCL
1	0.5762415	8.951826	-96.15259		X				
2	0.6030123*	1.940584*	-100.33103*		X				X
3	0.6021590	3.200886	-96.29458	X	X				X
4	0.6013672	4.438916	-92.28631		X	X	X		X
5	0.6027980	5.029368	-88.96817	X	X	X	X		X
6	0.5994464	7.000000	-84.19530	X	X	X	X	X	X

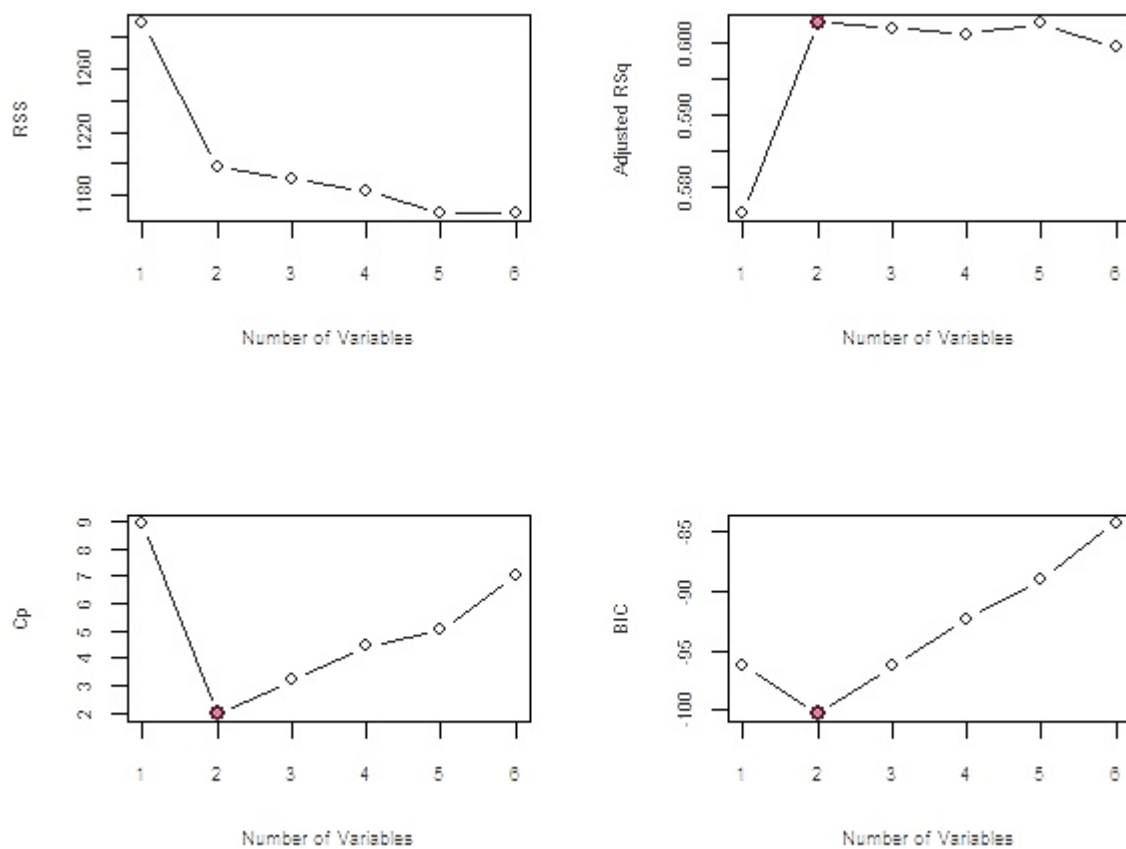


Figure 6.10

Comparison of the best subset(s) of predictor variables for the PDI (Study 1) according to different selection criteria. Starting top-left, clockwise: Residual sum of squares of the regression models (RSS); Adjusted R^2 , Mallows' C_p , Bayesian Information Criterion (BIC). X-axes denote the number of variables (i.e., the models 1-6). Best model per metric highlighted in red.

Model 2 was subsequently tested using multiple linear regression. Visual assessment indicated a linear pattern (Figure 6.12, centre left) and an approximately normal distribution of the residuals (Figure 6.12, top and centre right). A scatterplot of residual leverage did not reveal any influential data points (Figure 6.12, bottom right), as well as adequate homoscedasticity (Figure 6.12, bottom left) which was confirmed by a Breusch Pagan test ($BP=5.02_{(2)}, p=.081$). Overall, the findings suggested that a linear model is likely appropriate to explain the relationship between dissociation, post-traumatic stress, and

delusions. The regression was statistically significant ($R^2 = 0.610$, $F_{(2,119)}=92.9$, $p<.001$), with both dissociative (DES-II score, $\beta=0.166$, $p<.001$), and post-traumatic symptoms (PCL-5 score, $\beta=0.054$, $p=.003$) predicting delusional experiences (PDI total). While the predictor variables were moderately correlated ($r=0.675$, $p<.001$), the variance inflation factor (VIF 1.84) was within an acceptable range (<5).

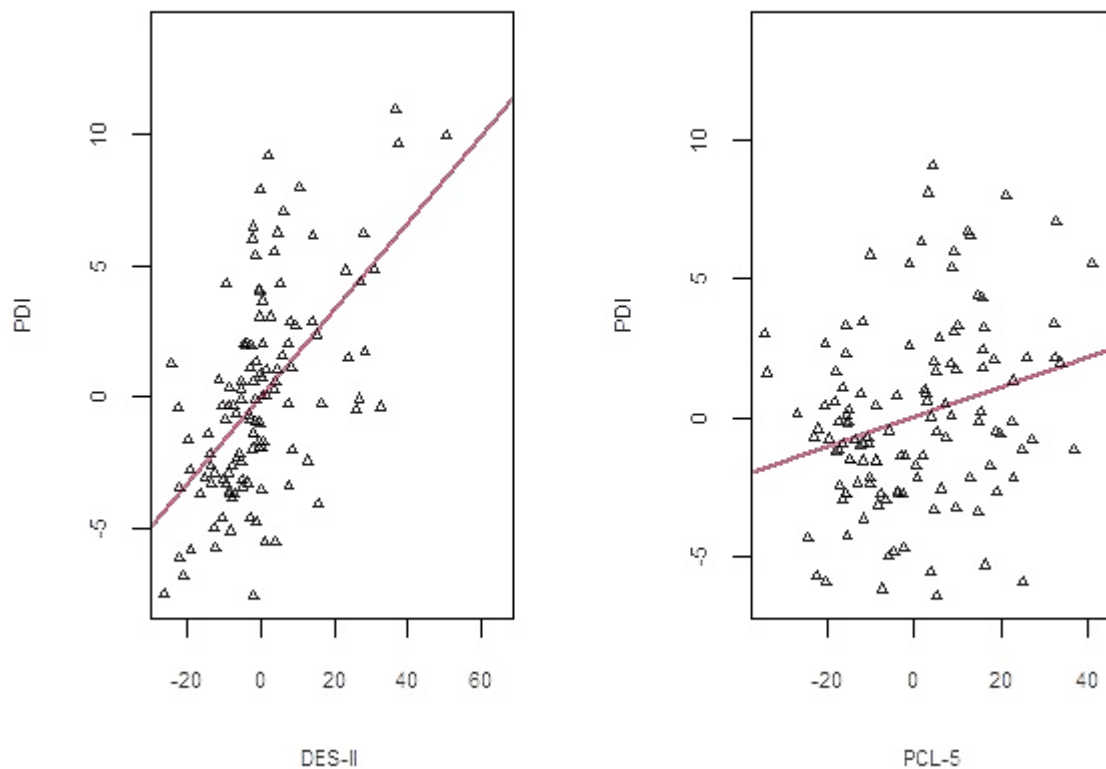


Figure 6.11

Added variable (partial regression) plots of Model 2 for the PDI in Study 1. Left: partial regression of the DES (dissociation), controlled for PCL-5 (post-traumatic stress) scores. Right: partial regression of the PCL-5, controlled for DES scores.

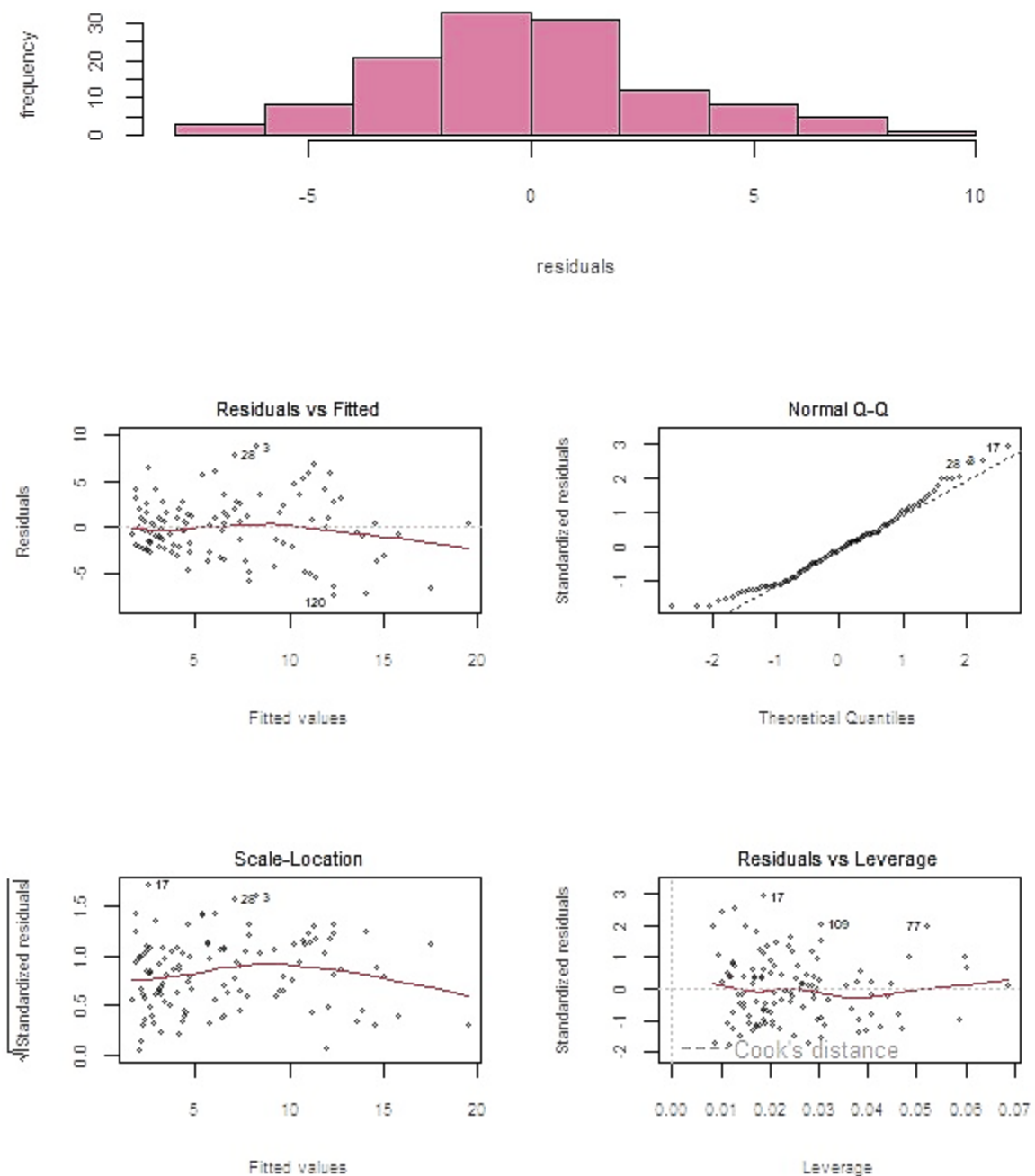


Figure 6.12

Diagnostic statistics for the best-subset selected model (Model 2) for the PDI in Study 1. Top: histogram of the model's residuals, assessing normal distribution; Centre left: residuals vs. fitted plot, assessing (non-)linear patterns; Centre right: Q-Q plot, assessing normal distribution of the model's residuals; Bottom left: scale-location plot, assessing homoscedasticity; Bottom right: Leverage plot, assessing influential datapoints (using Cook's distance).

Table 6.2

Best subsets of predictors for CAPS (yes/no) scores in Study 1. Adj R^2 = adjusted R-squared, BIC=Bayesian information criterion, DS=diagnostic status, DES=dissociation, GAD=anxiety, QIDS=depression, CTQ=childhood adversity, PCL=post-traumatic stress. *=best model based on the respective selection criterion, dark highlighted model=selected subset, light highlighted model=alternative subset (also fitted), Xs denote which predictors are part of each model.

M	Adj R^2	Mallow's Cp	BIC	cv error	D.S.	DES	GAD	QIDS	CTQ	PCL
1	0.5328104	19.240158	-84.24887	5.050070		X				
2	0.5771176	7.189403	-92.62199*	4.822338*	X	X				
3	0.5898233	4.484041	-92.56923	4.903548	X	X	X			
4	0.5936523	4.383256*	-89.94774	4.947427	X	X	X		X	
5	0.5945104*	5.144852	-86.44884	4.972067	X	X	X	X	X	
6	0.5914989	7.000000	-81.79839	5.013146	X	X	X	X	X	X

Best predictors for perceptual experiences

Since each of the psychometric variables (DES, GAD, QIDS, CTQ, and PCL) had also shown significant correlations with the CAPS symptom score, best subset selection for perceptual experiences was computed matching the previous parameters: six predictors, no variables forced in or out, and diagnostic status added as covariate. The fitted model was computed as follows: CAPS ~ DES + GAD + QIDS + CTQ + PCL + diagnostic status. All resulting models are summarised in Table 6.2 and Figure 6.13. Comparison of the three main accuracy metrics did not offer an obvious best candidate (adjusted R^2 endorsed the 5-factor model, Marlow's Cp the 4-factor one, and BIC lead to a two-factor solution) and k-fold cross-validation was consulted to identify the model that minimises prediction error. Two models were subsequently fitted for direct comparison: Model 2 which was identified as the best model by two metrics (BIC and cross-validation error), and Model 4 which had been consistently identified as one of the top three models across all metric evaluations (Table 6.2).

The four-factor model was statistically significant ($r^2=0.561$, $F_{(4,117)}=37.44$, $p<.001$), with DES ($\beta=0.19$, $p<.001$) and diagnostic status ($\beta=-2.98$, $p=.015$), but neither GAD ($\beta=0.06$,

$p=.292$), nor CTQ ($\beta=0.01$, $p=.573$) significantly influencing CAPS symptom scores. The latter were subsequently dropped from the regression analysis, leaving Model 2 (with DES and diagnostic status as predictors) as the most prospective subset. This model was tested independently to evaluate its explanatory and predictive capacities. Visual assessment indicated an approximately linear pattern and no significantly influential data points; however, residuals were non-normally distributed, and heteroscedasticity was present in the data set (Breusch-Pagan statistic: $BP=33.97$, $p<.001$). Accordingly, a weighted least squares regression was performed which adjusted all parameters to fit assumptions (Figure 6.15).

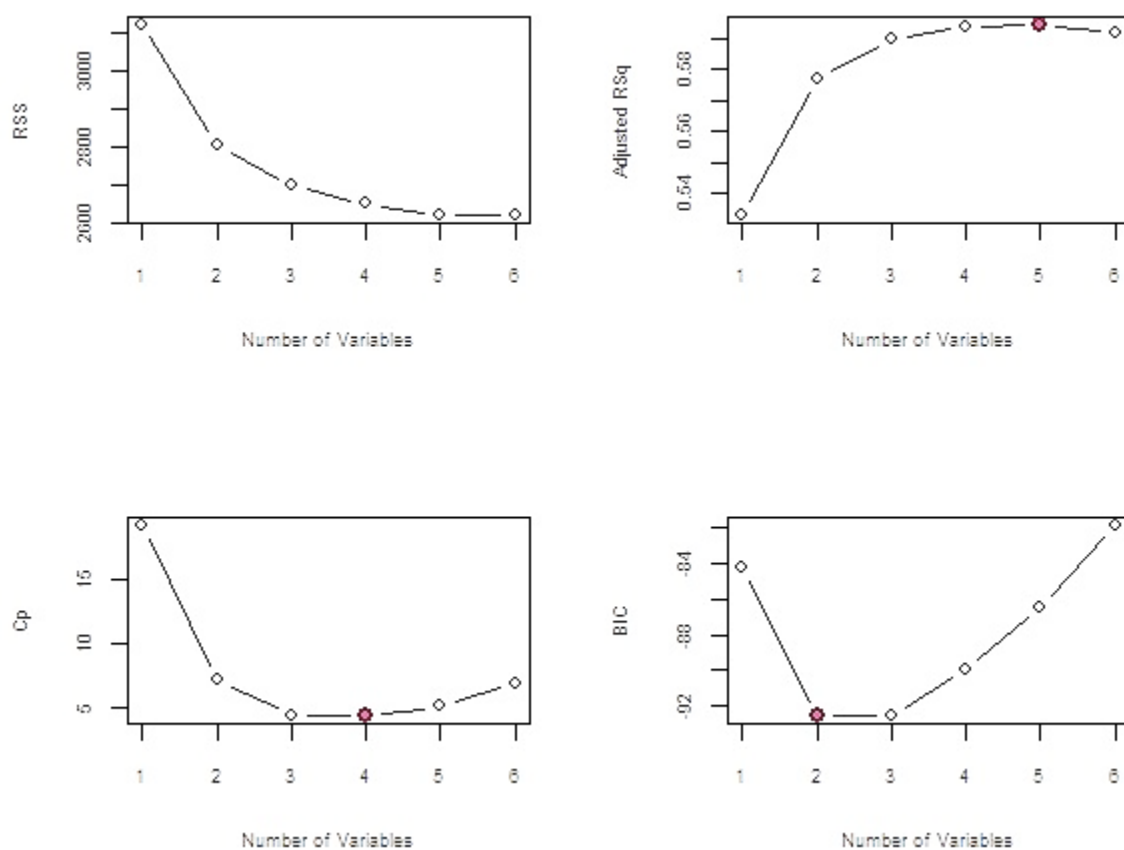


Figure 6.13

Comparison of the best subset(s) of predictor variables for the CAPS (Study 1) according to different selection criteria. Starting top-left, clockwise: Residual sum of squares of the regression models (RSS); Adjusted R^2 , Mallows' C_p , Bayesian Information Criterion (BIC). X-axes denote the number of variables (i.e., the models 1-6). Best model per metric highlighted in red.

The final model was statistically significant ($r^2=0.558$, $F_{(2,119)}=75.23$, $p<.001$), with perceptual experiences influenced by dissociative symptoms independent of diagnostic status ($\beta=0.244$, $p<.001$), as well as by diagnostic status unrelated to dissociative symptoms ($\beta=-3.241$, $p<.001$). There was no interaction between the predictors (Figure 6.14, right). While dissociative experience scores were moderately associated ($r=0.675$, $p<.001$) with diagnostic status, the model's variance inflation factor (VIF 1.61) was within the acceptable range (<5).

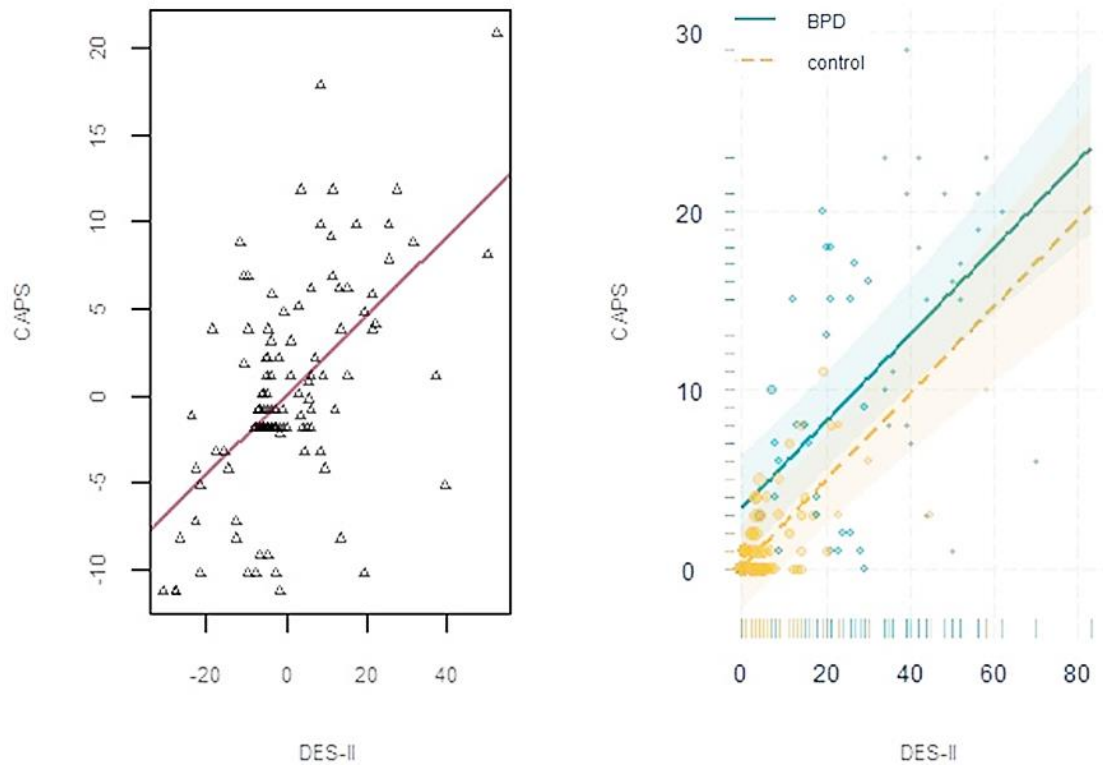


Figure 6.14

Added variable (partial regression) and interaction plots of Model 2 for the CAPS in Study 1. Left: partial regression of the DES (dissociation), controlled for diagnostic status. Right: interaction plot of diagnostic status and dissociative experiences as individual and combined predictors of CAPS scores.

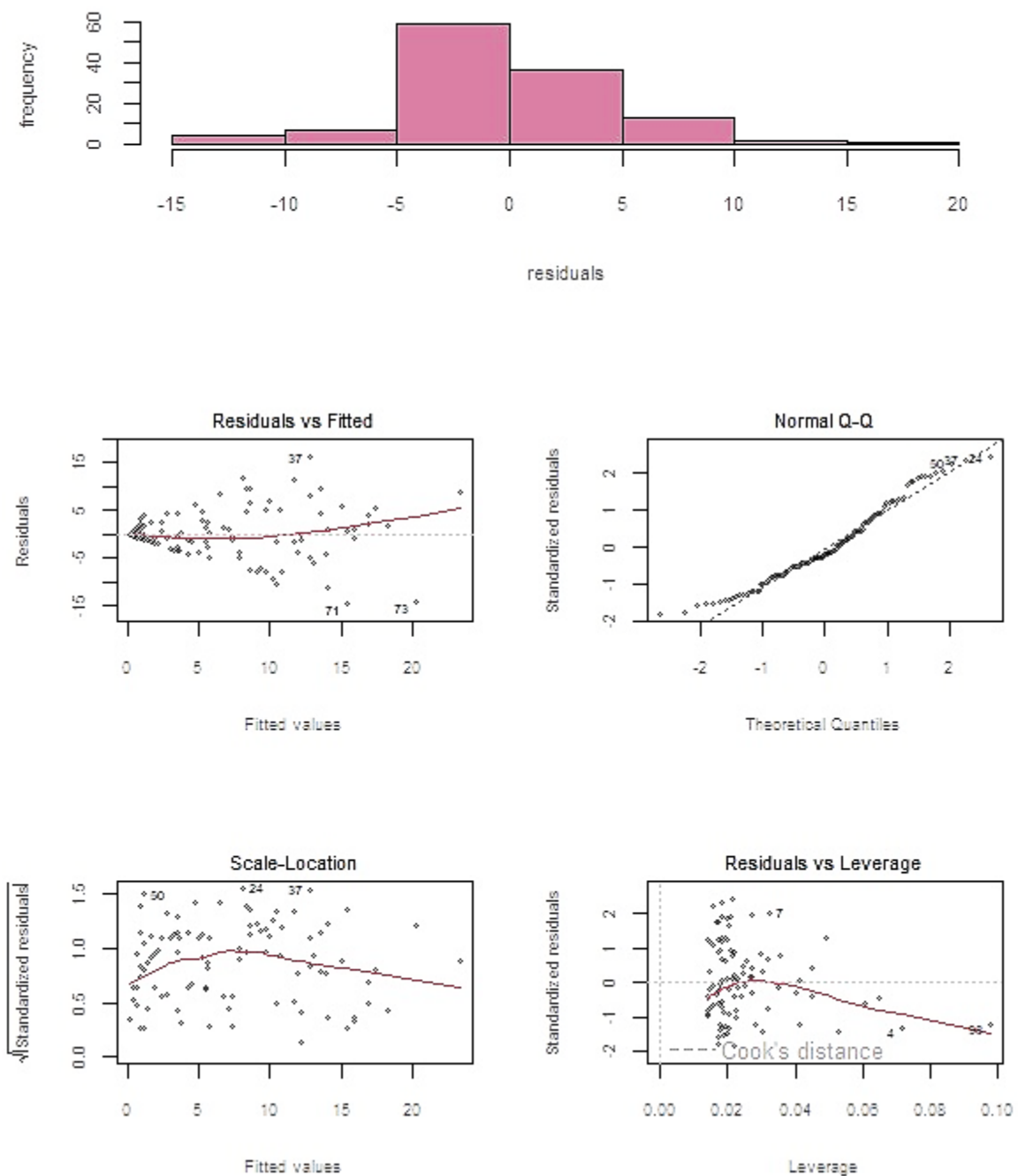


Figure 6.15

Diagnostic statistics for the best-subset selected model (Model 2) for the CAPS in Study 1. Top: histogram of the model's residuals, assessing normal distribution; Centre left: residuals vs. fitted plot, assessing (non-)linear patterns; Centre right: Q-Q plot, assessing normal distribution of the model's residuals; Bottom left: scale-location plot, assessing homoscedasticity; Bottom right: Leverage plot, assessing influential datapoints (using Cook's distance).

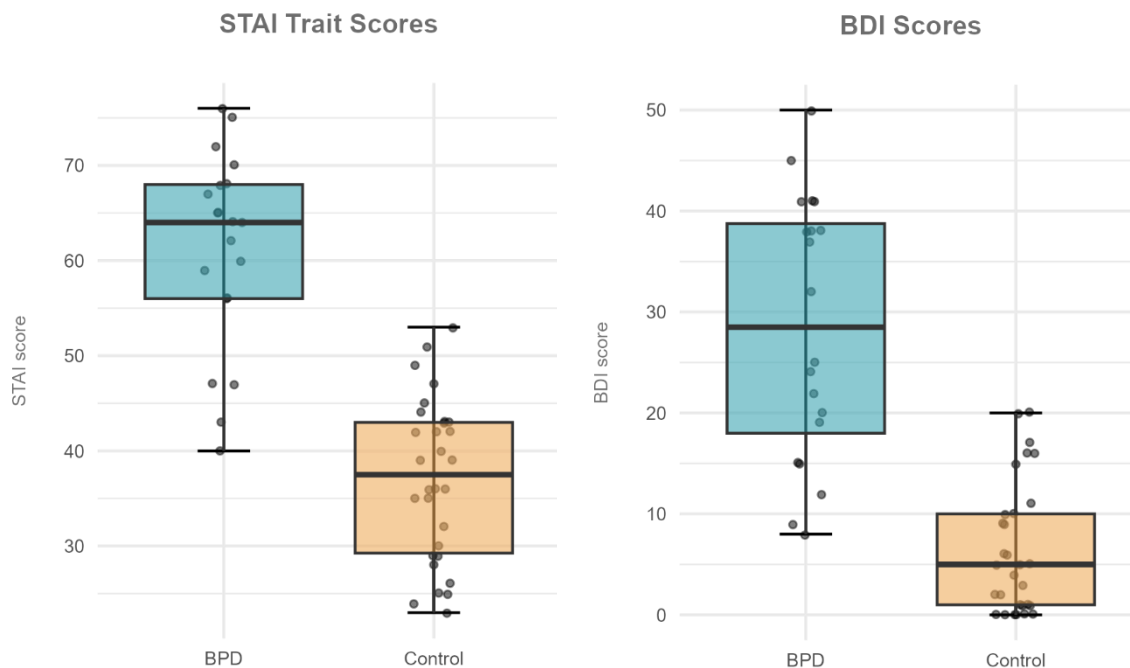


Figure 6.16

Box plots depicting median and interquartile range of STAI (anxiety, trait score; left) ($t_{36}=8.64$, $p < .001$) and BDI (depression; right) ($t_{26}=6.99$, $p < .001$), for Study 2.

6.3 Results of Study 2

6.3.1 Analysis of questionnaire and task measures

Common mental distress (BDI and STAI) and borderline symptom severity (BSL)

In line with the findings of Study 1, BPD and control participants differed significantly on both BDI ($t_{26}=6.99$, $p < .001$) and STAI ($t_{36}=8.64$, $p < .001$). However, their correlations with CAPS and PDI were comparatively weaker (than those between PE scores and QIDS and GAD-7 in Study 1). An association between depression and hallucinations ($r=0.646$, $p < .001$), and delusions ($r=0.606$, $p < .001$), was observed for the full participant population – but not within groups (see Figure 6.23 and Figure 6.24 for within-group correlations). Similarly, anxiety scores were also correlated with PEs in the overall sample only ($r=0.669$, $p < .001$ and $r=0.670$, $p < .001$ for CAPS and PDI, respectively). While unexpected, this is most likely due to the significantly smaller sample size of Study 2.

Unsurprisingly, the BPD group presented with significantly higher borderline symptom scores ($t_{47}=8.37, p<.001$). While the BSL did not correlate with either CAPS ($r=0.066, p=.78$) or PDI ($r=0.101, p=.67$) symptom prevalence, higher BPD symptom scores were associated with more distress for perceptual experiences ($r=0.562, p=0.010$), but not delusional beliefs ($r=0.362, p=.12$). Correlations in the control sample were non-significant.

Childhood trauma and post-traumatic stress

BPD participants scored higher than controls on the CTQ total (Welch's $t_{33}=3.36, p=.002$), as well as the EN (Welch's $t_{43}=3.41, p=.007$) and EA (Welch's $t_{42}=4.03, p=.001$) subscales. Conversely, no significant differences were observed for the PN (Welch's $t_{27}=2.61, p=.07$), PA (Welch's $t_{34}=1.01, p=.32$), and SA (Welch's $t_{31}=0.99, p=.33$) scores. Unlike in Study 1 (and mirroring CMD findings), no correlation between CTQ and CAPS scores could be ascertained in the BPD group, neither for the total score ($r=0.260, p=.27$), nor the SA subscale ($r=0.162, p=.27$). This may be partially due to the overall lower prevalence of childhood adversity among participants of Study 2. There were also no correlations between CTQ and PDI at 95% CI (see Figure 6.24 for correlation coefficients).

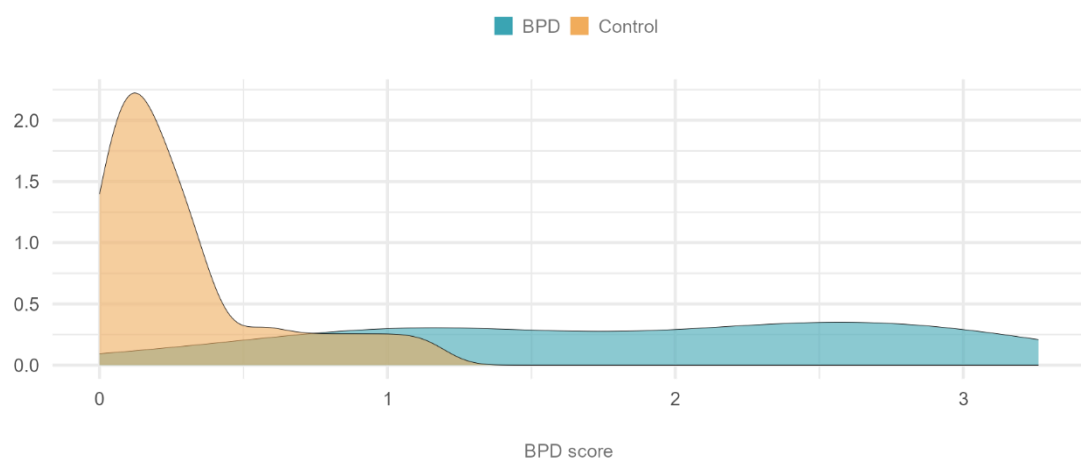


Figure 6.17

Distribution of BSL (BPD pathology) scores for BPD and control participants ($t_{47}=8.37, p<.001$).

The BPD group again scored higher on the PCL-5 ($t_{103}=9.83, p < .001$), with 65% (13/20) of participants above the threshold for clinically significant post-traumatic symptoms – compared to 10% of controls (3/29). As with the CTQ, the correlational findings of Study 1 could not be replicated for neither the CAPS nor the PDI, in either group (CAPS: Figure 6.23; and PDI: Figure 6.24). Direct comparison of the CTQ and PCL scores between the BPD populations of Study 1 and Study 2 revealed remarkably similar rates of childhood adverse experiences and post-traumatic stress (Table 6.3), although groups differed on the physical neglect subscale⁴³ ($t_{58}=6.75, p < .001$). This may perhaps be influenced by the wider range of socioeconomic backgrounds represented in an international, online sample, compared to a Cambridgeshire population.

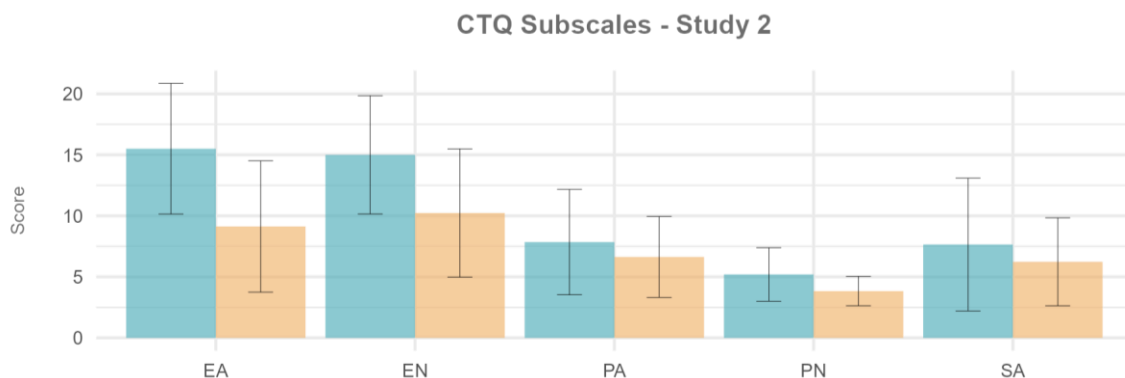


Figure 6.18

Bar plots with error bars for CTQ subscale scores of Study 2. EA=emotional abuse, EN=emotional neglect, PA=physical abuse, PN=physical neglect, SA=sexual abuse. BPD and control participants differed significantly on the EA ($t_{42}=4.03, p=.001$) and EN ($t_{43}=3.41, p=.007$), but not on the PA ($t_{34}=1.01, p=.32$), PN ($t_{27}=2.61, p=.07$), or the SA ($r=0.162, p=.27$) subscales.

⁴³ Physical neglect subscale: Item 1 (“I didn’t have enough to eat”), item 2 (“I knew that there was someone to take care of me and protect me”), item 4 (“My parents were too drunk or high to take care of the family”), item 6 (“I had to wear dirty clothes”), item 26 (“There was someone to take me to the doctor if I needed it”)

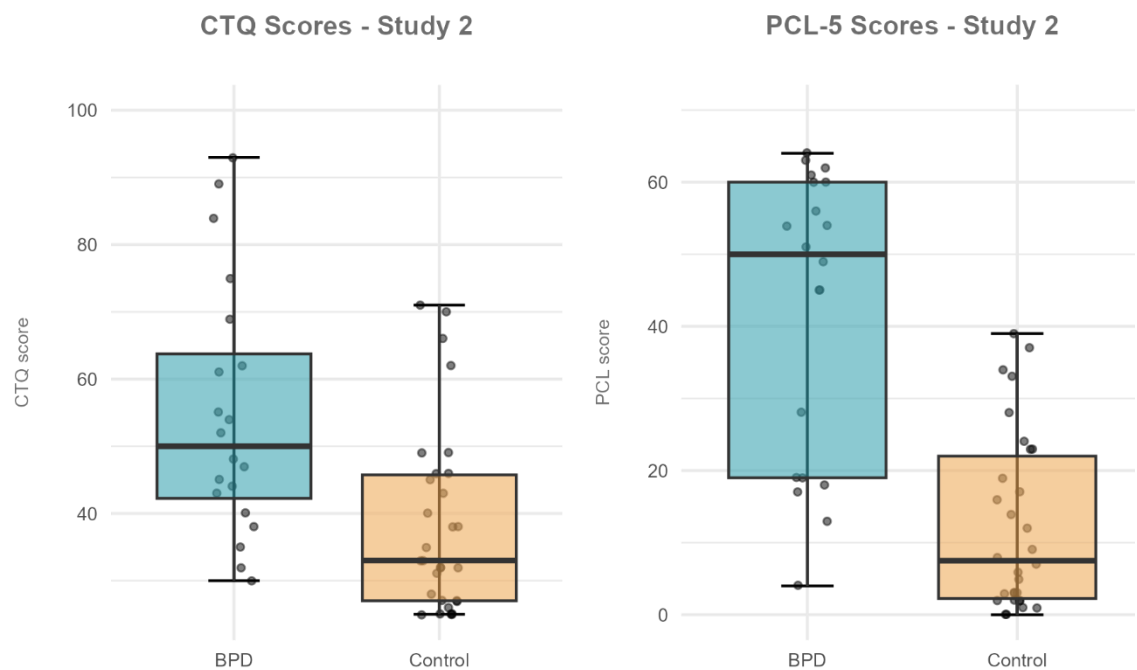


Figure 6.19

Box plots depicting median and interquartile range of CTQ (childhood adversity; left) ($t_{33}=3.36, p=.002$) and PCL (post-traumatic stress; right) ($t_{103}=9.83, p<.001$), for Study 2.

Table 6.3

Comparison of CTQ (childhood adversity) and PCL (post-traumatic stress) descriptive statistics between the BPD groups of Study 1 ($n=54$) and Study 2 ($n=20$). EA=emotional abuse, EN=emotional neglect, PA=physical abuse, PN=physical neglect, SA=sexual abuse. Scores did not differ significantly between groups, with exception of the physical neglect subscale ($t_{58}=6.75, p<.001$).

	CTQ Total	PN	PA	EN	EA	SA	PCL
Study 1 mean(sd)	56.6(15.9)	10.0(3.7)	8.0(3.6)	15.3(4.5)	15.0(5.5)	8.4(5.3)	45.7(17.8)
Study 2 mean(sd)	54.8(18.7)	5.2(2.2)	7.9(4.3)	15.0(4.8)	15.5(5.4)	7.6(5.4)	42.1(20.1)

Life-time stressors and adversity

In addition to presenting with a higher prevalence of childhood trauma, BPD participants were also more likely to have lived through a greater number of potentially stressful or traumatic events in their (adult) lifetime, with a median of eight reported incidences in the BPD, and four in the control group ($t_{47}=4.02, p <.001$). Furthermore, BPD participants reported being more strongly affected by these experiences in their day-to-day lives, over the past year ($t_{29}=5.38, p <.001$).

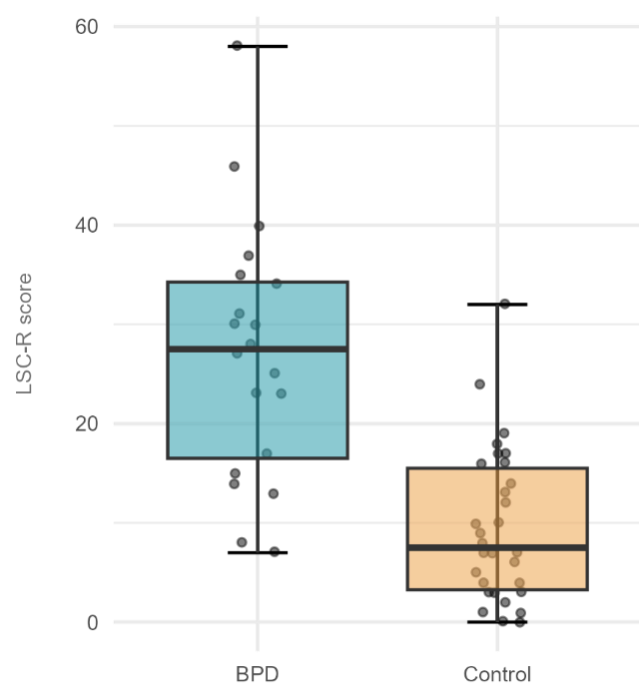
Events for which the rate of endorsement differed between groups are summarised in Table 6.4. The most poignant contrast between groups was observed for item eleven (*“Have you ever been emotionally abused or neglected (for example, being frequently shamed, embarrassed, ignored, or repeatedly told that you were “no good”)?”*) which was reported by 95% of the BPD group (19/20). Curiously, one of thirty stressors (item 18, non-sudden death of a relation) was endorsed more frequently by the control group. While unexpected, this may be due to several factors: Firstly, the control group’s slightly higher age average (for participant demographics see Table 3.5) increases the likelihood of participants having lost elderly relatives. Secondly, the item’s phrasing (*“Has someone close to you died (do NOT include those who died suddenly or unexpectedly)?”*) lacks clear definition and could encompass anyone, from immediate family to neighbours and colleagues. Owing to the tumultuous and unstable interpersonal relationships associated with BPD, control participants may be considerably less isolated and more likely to have a wider social circle in which such non-sudden deaths could occur.

Despite the small sample size, the number of LSC-R items endorsed (Scoring Option 1) correlated positively with both, the CAPS ($r=0.592, p=.006$) and the PDI ($r=0.533, p=.016$) in the BPD group – but only with the PDI in the control group ($r=-0.090, p=.643$ and $r=0.433, p=.019$ for CAPS and PDI, respectively). Similarly, subjective impact scores (Option 2) were also associated with both, perceptual ($r=0.474, p=.035$) and delusional experiences ($r=0.043, p=.037$) in BPD participants, but again only with PDI scores in controls ($r=0.043, p=.824$ and $r=0.527, p=.003$ for CAPS and PDI, respectively).

Table 6.4 (left) and Figure 6.20 (right)

Table (left): LSC-R items for which the frequency of endorsement differed between BPD and control participants (assessed visually), including number of endorsements and percentage per group. For each question, participants had the option to abstain (“prefer not to answer”). Some items additionally offered the option “not applicable” (e.g., questions on parenthood or pregnancy). Both types of answer were recorded as NA. **Figure (right):** LSC-R subjective scores (Scoring Option 2, number of items x ongoing distress associated with the event), for BPD and control participants ($t_{29}=5.38, p < .001$).

Item	Yes		NA	
	BPD	Control	BPD	Control
7	8 (40%)	5 (17%)	-	-
9	7 (35%)	1 (3%)	-	-
11	19 (95%)	10 (35%)	-	1 (3.4%)
18	7 (35%)	20 (69%)	2 (10%)	-
23	8 (40%)	5 (17%)	-	-
24	10 (50%)	7 (24%)	-	1 (3%)
27	6 (30%)	-	-	-
29*	9 (45%)	-	-	1 (3%)



Item 7: Did your parents ever separate or divorce while you were living with them?

Item 9: Have you ever had serious money problems (for example, not enough money for food or place to live)?

Item 11: Have you ever been emotionally abused or neglected (for example, being frequently shamed, embarrassed, ignored, or repeatedly told that you were “no good”)?

Item 18: Has someone close to you died (do NOT include those who died suddenly or unexpectedly)?

Item 23: After age 16, were you ever abused or physically attacked (not sexually) by someone you knew (for example, a parent, boyfriend, or husband hit, slapped, choked, burned, or beat you up)?

Item 24: Have you ever been bothered or harassed by sexual remarks, jokes, or demands for sexual favours by someone at work or school (for example, a coworker, a boss, a customer, another student, a teacher)?

Item 27: Before age 16, did you ever have sex (oral, anal, genital) when you didn’t want to because someone forced you in some way or threatened to hurt you if you didn’t?

Item 29: Are there any events we did not include that you would like to mention? Answers to this item overwhelmingly included peer abuse (“bullying”, $n=7$). One person reported being rejected from their family for being homosexual, one person reported attempted trafficking, and one person reported a forced marriage.



Figure 6.21

Top: Correlations between the LSC-R quantitative (Option 1, top) and qualitative (Option 2, centre) scores and PDI (delusions, right) and CAPS (hallucinations, left) scores. Bottom: Distribution of the number of life-stressors experienced for the BPD and control groups ($t_{47}=4.02$, $p < .001$).

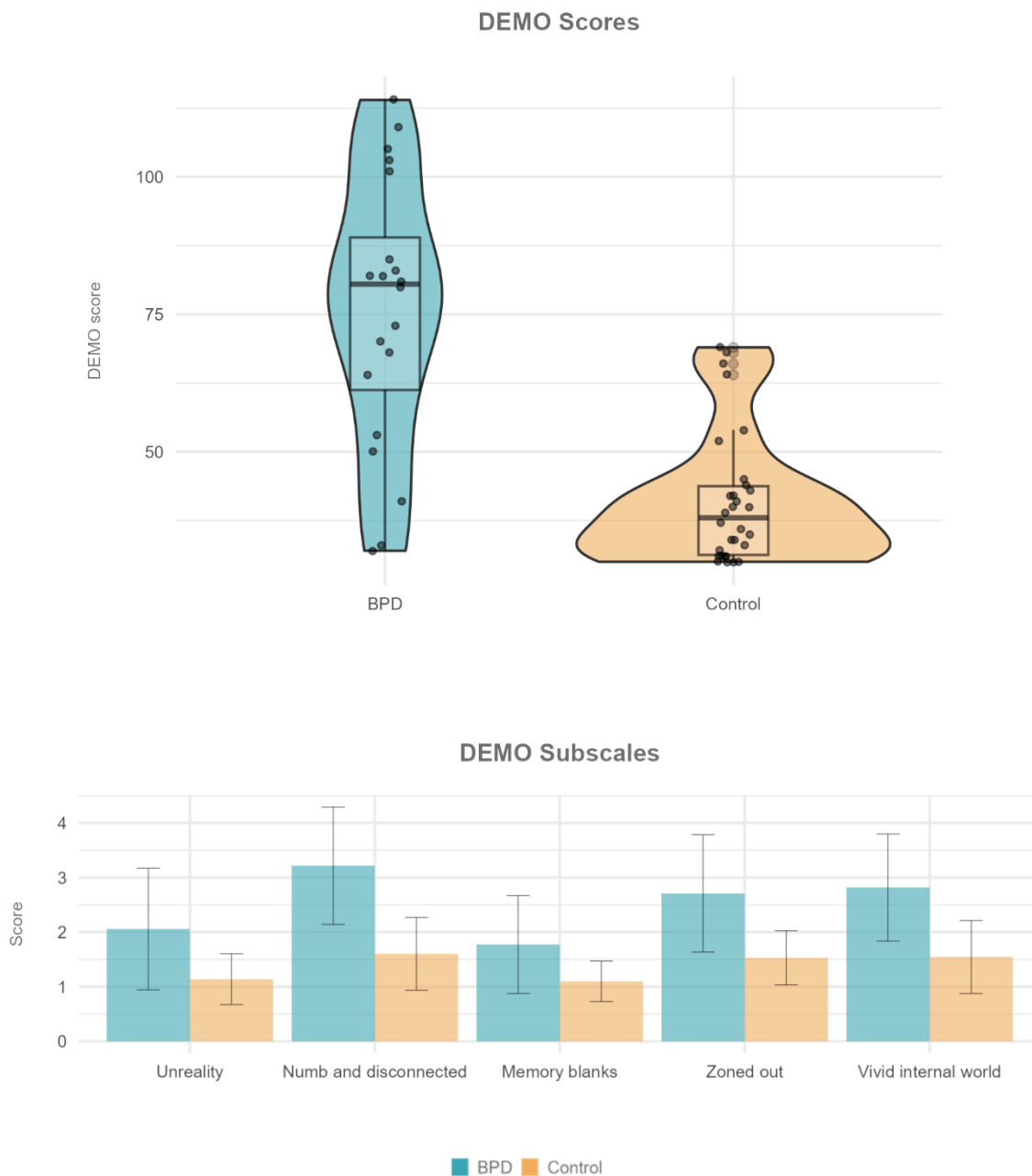
Dissociation (DEMO)

BPD participants scored significantly higher than controls on the DEMO total ($t_{25}=5.73$, $p<.001$) and all five subscales (at $p<.01$ with Bonferroni correction, see Table 6.5), with the most pronounced differences observed for the *Numb and Disconnected*, *Zoned Out*, and *Vivid Internal World* experience categories. Due to the relative novelty of the measure (Černis et al., 2018), a clinical cut-off has not yet been suggested and few comparable findings have been reported. Strachan et al. (2023) described an average score of 62.75 (SD 22.13) in a non-clinical undergraduate population ($n=528$) with 65.7% prevalence of previous trauma. In this context, results from the present study are consistent with what might reasonably be expected: Comparatively higher levels of dissociation in BPD participants, who represent a more clinical sample (mean 75.5, SD 24.5), and comparatively lower levels of dissociation in control participants, who had a lower prevalence of lifetime trauma and mental distress (mean 41.5, SD 12.1). Dissociative experiences were correlated with psychotic symptoms for both BPD ($r=0.472$, $p=.036$ and $r=0.513$, $p=.021$ for CAPS and PDI, respectively) and control group ($r=0.518$, $p=.002$ and $r=0.675$, $p<.001$ for CAPS and PDI, respectively).

Table 6.5

Descriptive statistics of the DEMO total and weighted subscale scores for BPD and control participants.

	Total	Unreality	Numb and Disconnected	Memory Blanks	Zoned out	Vivid Internal World
BPD ($n=20$)	75.4	2.06	3.22	1.78	2.71	2.82
mean(sd)	(24.5)	(1.11)	(1.07)	(0.90)	(1.07)	(0.98)
Control ($n=29$)	41.5	1.14	1.60	1.10	1.53	1.55
mean(sd)	(12.1)	(0.47)	(0.67)	(0.37)	(0.50)	(0.67)
*Welch's	$t_{25}=5.73$, $p<.001^*$	$t_{24}=3.50$, $p=.009^*$	$t_{29}=5.97$, $p<.001^*$	$t_{24}=3.20$, $p=.02^*$	$t_{25}=4.60$, $p<.001^*$	$t_{47}=5.41$, $p<.001$

**Figure 6.22**

Top: Violin plots, depicting distribution, median, and interquartile range of DEMO scores for BPD and control participants.

Bottom: Bar plots showing the mean and standard deviation of the five DEMO subscales for BPD and control participants. Groups differed significantly across all five categories. For descriptive statistics, see Table 6.5.

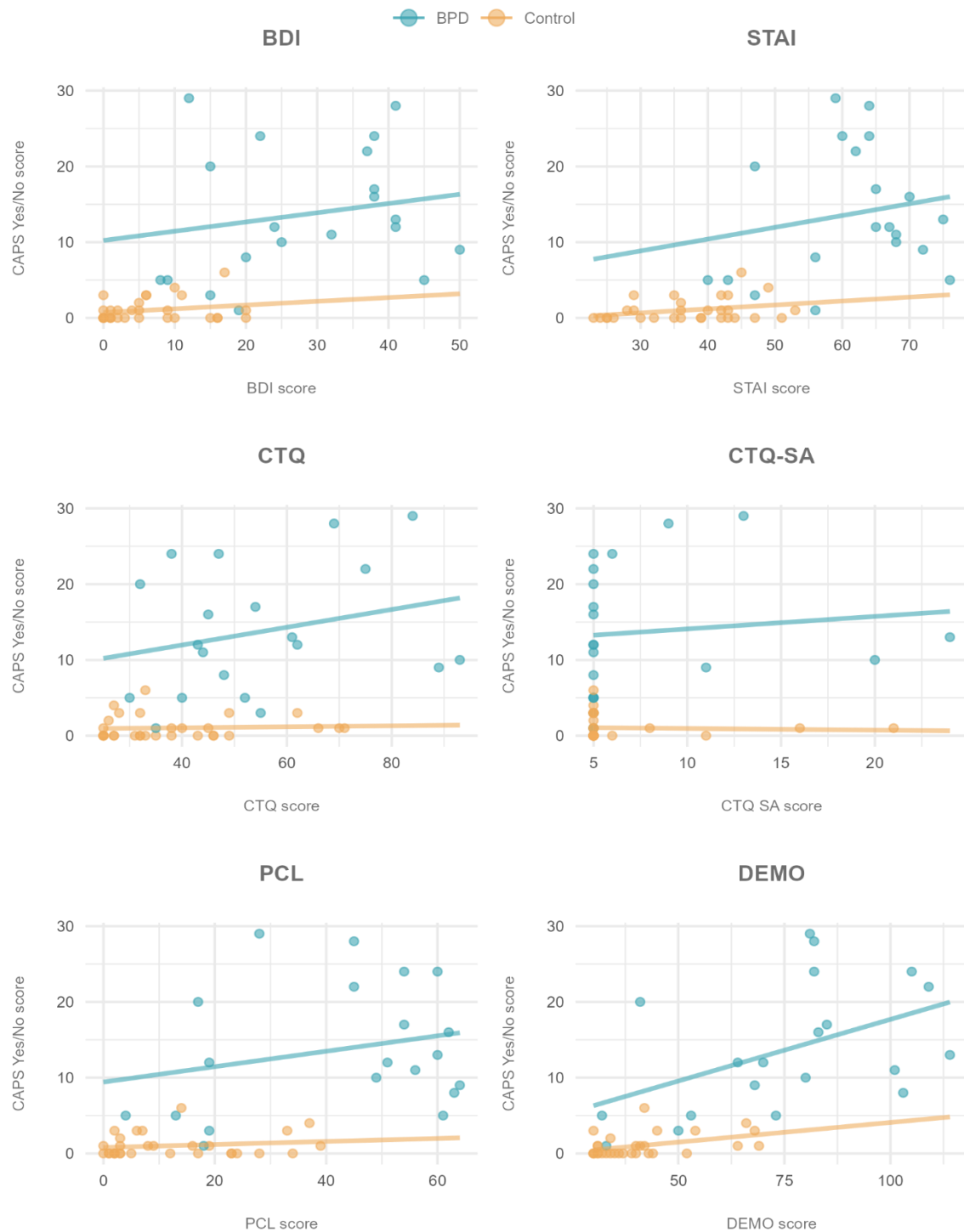


Figure 6.23

Correlations between CAPS (yes/no) scores and other psychopathology measures in Study 2. BDI=depression (BPD $r=0.188$, $p=.428$, Control $r=0.227$, $p=.236$), STAI=anxiety (trait score) (BPD $r=0.190$, $p=.423$, Control $r=0.298$, $p=.116$), CTQ=childhood adversity (BPD $r=0.260$, $p=.270$), CTQ-SA=childhood sexual assault (BPD $r=0.162$, $p=.271$), PCL=post-traumatic stress (BPD $r=0.242$, $p=.304$, Control $r=0.144$, $p=.458$), DEMO=dissociation (BPD $r=0.472$, $p=.036$, Control $r=0.518$, $p=.002$).

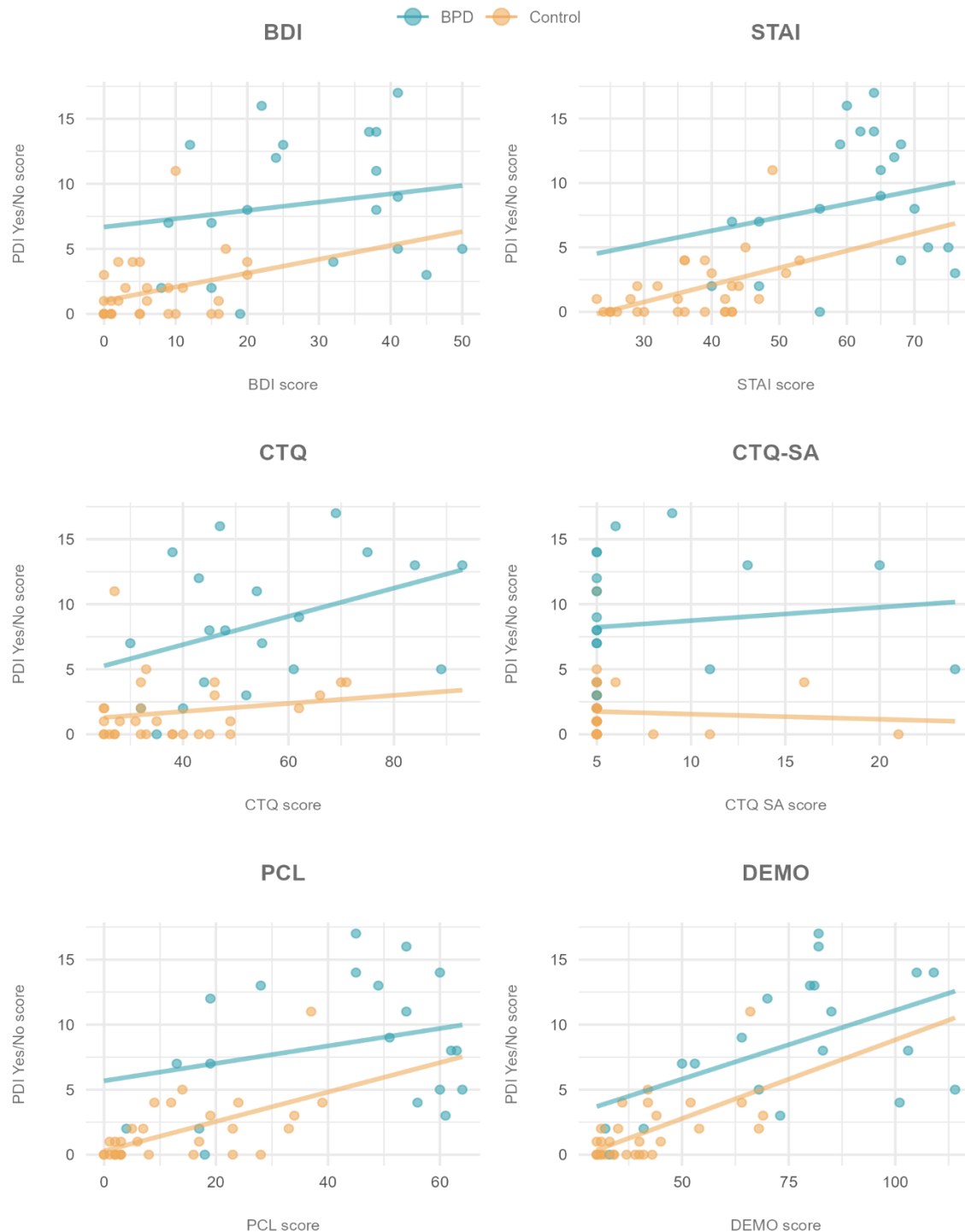


Figure 6.24

Correlations between PDI (yes/no) scores and other psychopathology measures in Study 2. BDI=depression (BPD $r=0.165, p=.488$, Control $r=0.311, p=.101$), STAI=anxiety (trait score) (BPD $r=0.212, p=.369$, Control $r=0.486, p=.124$), CTQ=childhood adversity (BPD $r=0.403, p=.079$, Control $r=0.193, p=.317$), CTQ-SA=childhood sexual assault (BPD $r=0.110, p=.644$), PCL=post-traumatic stress (BPD $r=0.267, p=.254$, Control $r=0.58, p=.021$), DEMO=dissociation (BPD $r=0.513, p=.021$, Control $r=0.675, p<.001$).

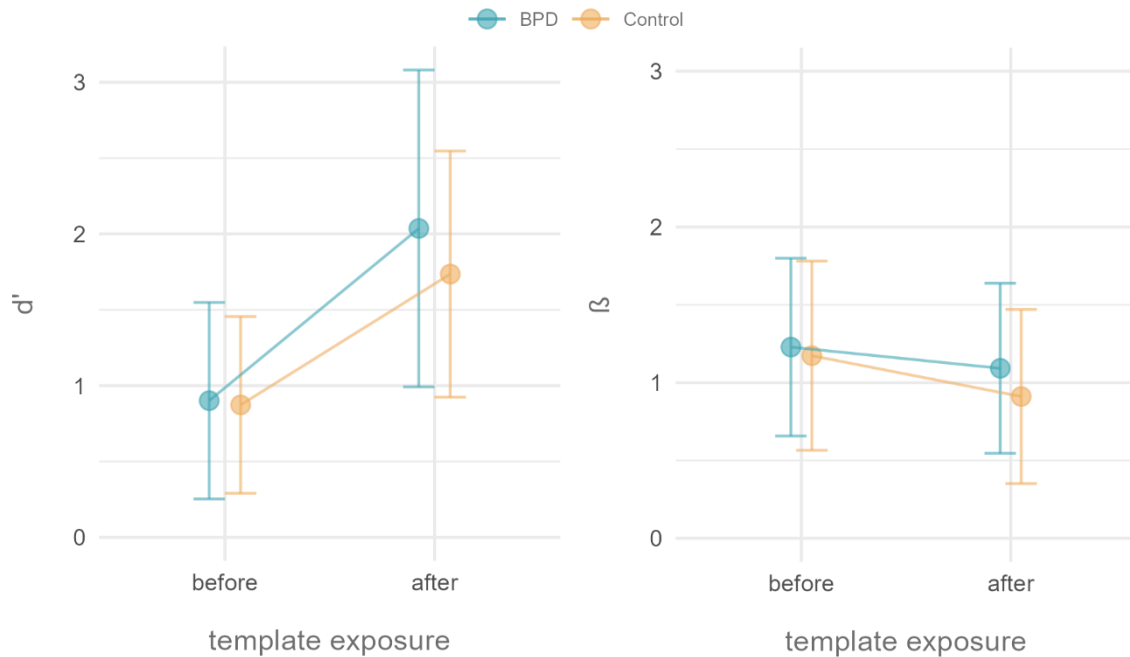


Figure 6.25

Perceptual discriminability (d' , left) of the two-tone images, and response bias (β) before and after template exposure for BPD and control participants of Study 2 (at first assessment, before stress induction). To avoid overplotting, graphs are presented offset (jittered). There was no significant difference between the groups.

Ambiguous visual stimuli and benefit of prior knowledge (the two-tone image task)

One BPD group participant indicated previous exposure to the two-tone image task in a different study, however removing their data did not affect results and they were included in the analysis. Both groups showed improved performance after exposure to the template images ($t_{38}=-4.13$, $p<.001$ and $t_{56}=-4.51$, $p<.001$ for BPD and control group respectively). The rate of change between before and after template exposure did not differ between groups for both d' ($t_{47}=1.21$, $p=.23$) and β ($t_{47}=0.61$, $p=.55$), and there was no discernible shift in response bias (β) towards reporting more dots on the figure for neither BPD ($t_{38}=0.77$, $p=.44$), nor control participants ($t_{56}=1.75$, $p=.09$).

Analogous to study one (and in contrast to previous findings that correlated task performance with schizotypy; e.g., Teufel et al., 2015) no correlation between CAPS and d' difference ($r=0.101$, $p=.48$), or PDI and d' difference ($r=-0.036$, $p=.81$) could be

demonstrated. Equally, baseline performance did not correlate with psychotic experience scores (compare Batty et al., 2014; Rivolta et al., 2014), and there was no difference in the ratio of correct hits prior to template exposure, between groups ($t_{49}=0.43$, $p=.68$)

6.3.2 Further analysis: Predictors of psychotic experiences (best subset)

Best predictors for delusional thoughts

Mirroring the analysis of Study 1 a best subset regression was performed, using psychopathology measures that correlated with the PDI total. To retain an equal number of variables across both studies while also allowing for potentially more reliable (i.e., more strongly correlated) predictors, the LSC-R (Score Option 2) was substituted for the PCL (both measures of post-traumatic distress). Similarly, diagnostic status was replaced by the BSL as covariate, since a dimensional measure of borderline pathology is better suited for regression analysis than a binary, categorical variable.

The maximum number of predictors was specified as six (all), no variables were forced in or out, and the computation output again rendered the best set of predictors for each model size (1-6, summarised in Table 6.6 and Figure 6.26). In line with the findings of Study 1, all three metrics of accuracy (adjusted R^2 , Cp, and BIC) indicated that a two-factor model (Model 2) incorporating dissociative experiences (DEMO) and post-traumatic stress (LSC-R) would offer the best fit. The model's scale-location plot indicated heteroscedasticity (Figure 6.27, bottom left; Breusch-Pagan statistic: $BP=14.32$, $p<.001$), and as such, a weighted least squares regression was performed. Visual assessment of the model's residuals showed approximately normal distribution of (Figure 6.27, top and centre right) and a linear pattern (Figure 6.27, centre left), without influential data points (Figure 6.27, bottom right). The overall regression was statistically significant ($R^2=0.636$, $F_{(2,47)}=41.0$, $p<.001$), with both dissociation (DEMO score, $\beta=0.119$, $p<.001$), as well as life-time trauma and associated distress (LSC-R score, $\beta=0.100$, $p=.029$) significantly predicting delusional experiences (PDI total). While the variables again were correlated ($r=0.698$, $p<.001$), their variance inflation factor (VIF 2.05) was well within the acceptable range (<5).

Table 6.6

Best subsets of predictors for PDI (yes/no) scores in Study 2. Adj R^2 = adjusted R-squared, BIC=Bayesian information criterion, BSL=borderline pathology, DEMO=dissociation, STAI=anxiety, BDI=depression, CTQ=childhood adversity, LSC-R=life-time stressors (Option 2). *=best model based on the respective selection criterion, highlighted model=best subset overall. Xs denote which predictors are part of each model.

M	Adj R^2	Mallow's C_p	BIC	BSL	DEMO	STAI	BDI	CTQ	LSC-R
1	0.5610277	7.0863707	-34.37287		X				
2	0.6218811*	0.774512*	-38.97490*		X				X
3	0.6200818	2.0303991	-35.90082	X	X				X
4	0.6190043	3.1953720	-32.94614	X	X	X			X
5	0.6120298	5.0086408	-29.25073	X	X	X		X	X
6	0.6030870	7.0000000	-25.34876	X	X	X	X	X	X

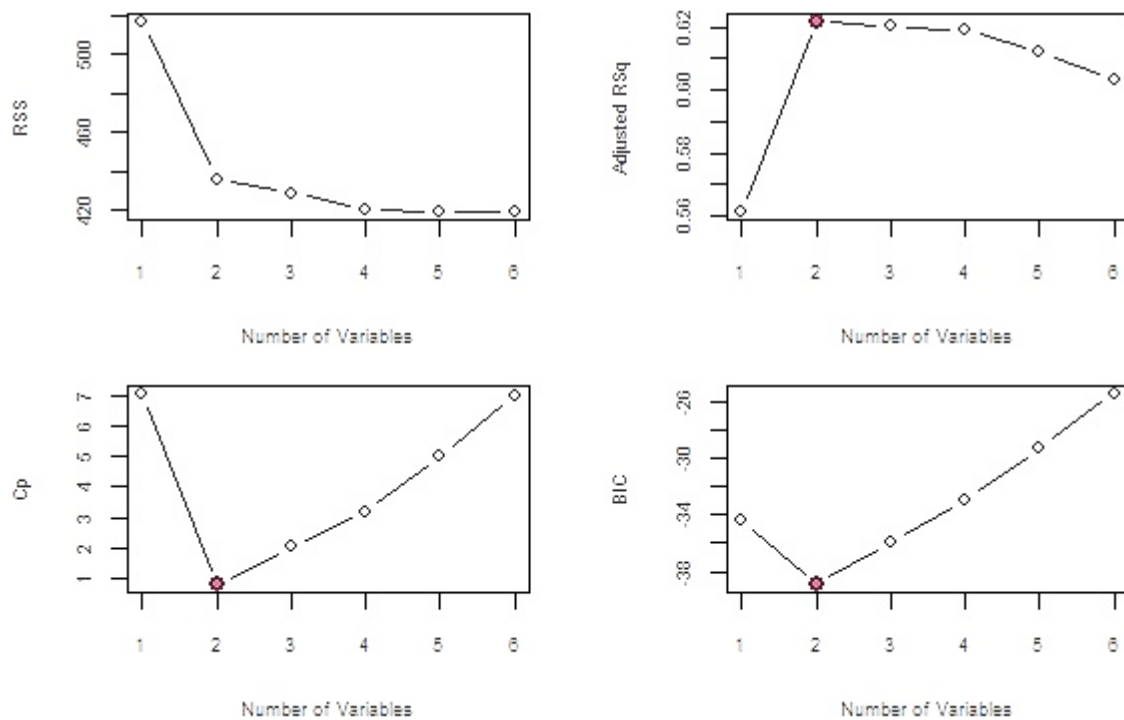


Figure 6.26

Comparison of the best subset(s) of predictor variables for the PDI (Study 2), according to different selection criteria. Starting top-left, clockwise: Residual sum of squares of the regression models (RSS); Adjusted R^2 , Mallow's C_p , Bayesian Information Criterion (BIC). The X-axes denote the number of variables (i.e., the models 1-6). Best model per metric highlighted in red.

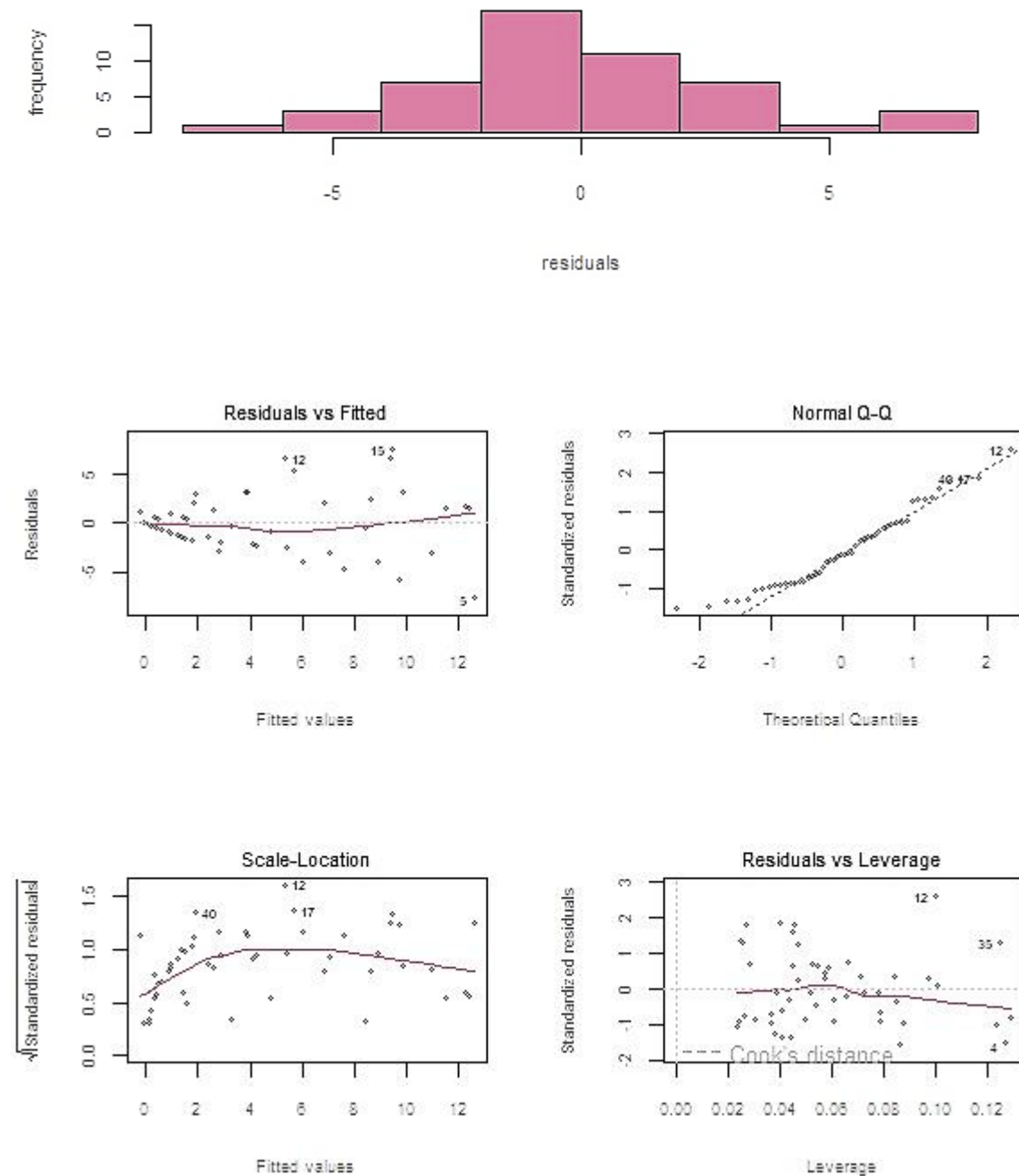


Figure 6.27

Diagnostic statistics for the best-subset selected model (Model 2) for the PDI in Study 2. Top: histogram of the model's residuals, assessing normal distribution; Centre left: residuals vs. fitted plot, assessing (non-)linear patterns; Centre right: Q-Q plot, assessing normal distribution of the model's residuals; Bottom left: scale-location plot, assessing homoscedasticity; Bottom right: Leverage plot, assessing influential datapoints (using Cook's distance).

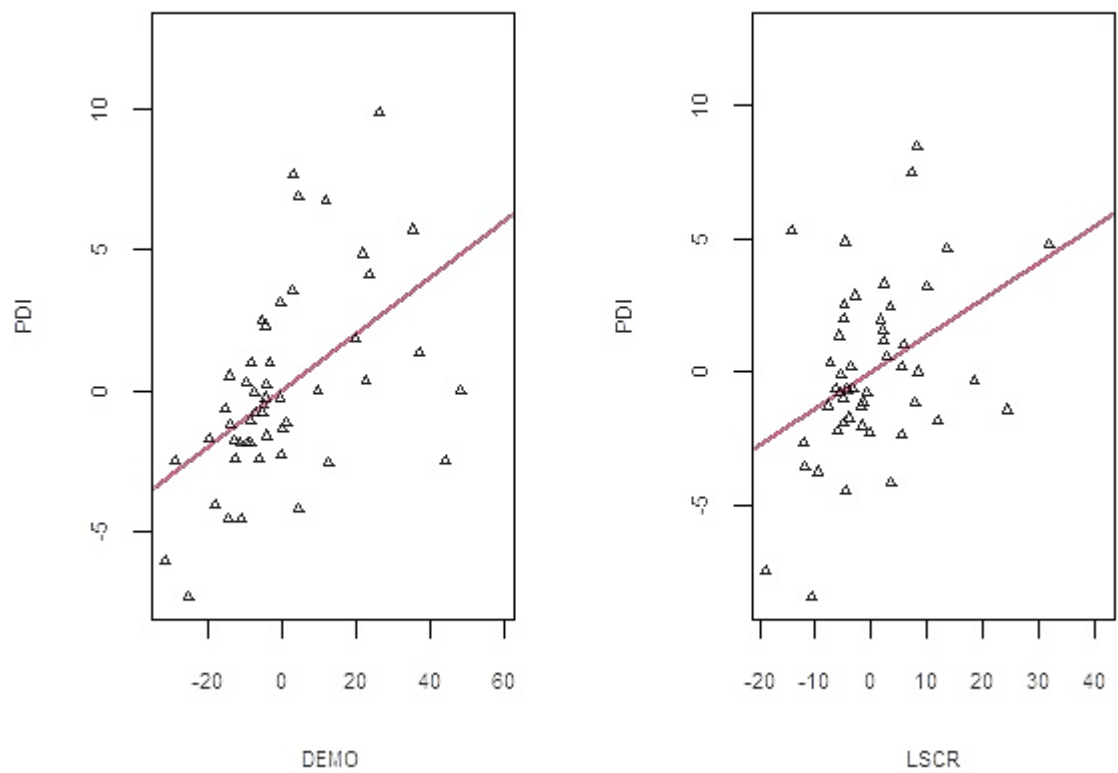


Figure 6.28

Added variable (partial regression) plots of Model 2 for the PDI in Study 2. Left: partial regression of the DEMO (dissociation), controlled for LSC-R (Option 2, number of life-time stressors and current impact) scores. Right: partial regression of the LSC-R, controlled for DEMO scores.

Best predictors for perceptual experiences

Like the best subset selection for perceptual experiences in Study 1, comparison of the three main accuracy metrics again did not offer an obvious candidate model for the CAPS (adjusted R^2 endorsed the four-factor-, Marlow's C_p and BIC the two-factor model). Confoundingly, k-fold cross-validation additionally suggested a one-factor (dissociation only) solution. Model 2 was ultimately selected for two reasons:

1. It was consistently identified as one of the most suitable subsets with a notably better C_p and BIC (compared to the second-best models on these statistics) and an only marginally weaker R^2 and cross-validation error (compared to the front-runners on these statistics).
2. While the one-factor model had the lowest prediction error, it also explained the least amount of variance ($R^2=52.36$), giving it limited explanatory value. Additionally, its C_p was above the threshold of fit (number of variables negative one), meaning that on this metric it is not just less suitable, but fully unsuitable.

Visual assessment of the residuals of Model 2 indicated severe non-normality and heteroscedasticity (Breusch-Pagan statistic: $BP=20.61$, $p<.001$), therefore, a weighted least squares regression was performed.

Table 6.7

Best subsets of predictors for CAPS (yes/no) scores in Study 2. Adj R^2 = adjusted R-squared, BIC=Bayesian information criterion, BSL=borderline pathology, DEMO=dissociation, STAI=anxiety, BDI=depression, CTQ=childhood adversity, LSC-R=life-time stressors (Option 2). *=best model based on the respective selection criterion, highlighted model=best subset overall. Xs denote which predictors are part of each model.

M	Adj R^2	Mallow's C_p	BIC	cv error	BSL	DEMO	STAI	BDI	CTQ	LSC-R
1	0.5235922	6.537704	-30.28098	5.410850*		X				
2	0.5739104	2.009742*	-33.00285*	5.930012		X				X
3	0.5730824	3.118322	-30.06906	5.912231		X			X	X
4	0.5747689*	3.963126	-27.45390	6.348615	X	X		X		X
5	0.5744743	5.015948	-24.63089	6.312723	X	X		X	X	X
6	0.5647398	7.000000	-20.73741	6.394685	X	X	X	X	X	X

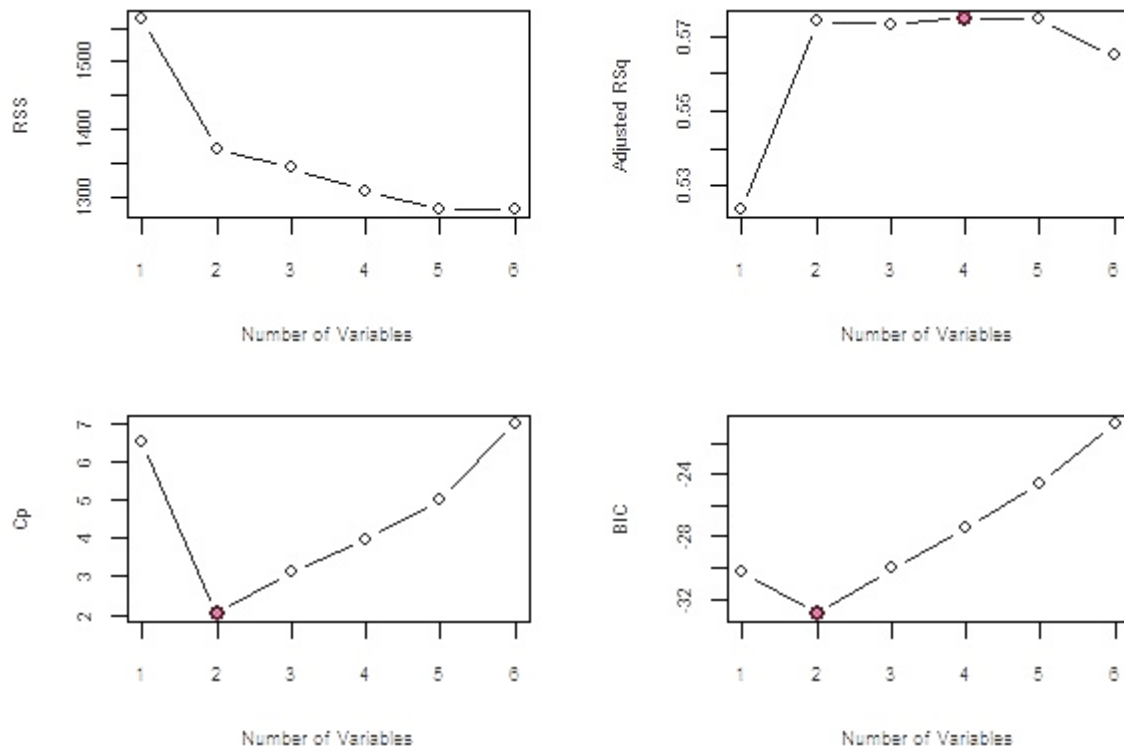


Figure 6.29

Comparison of the best subset(s) of predictor variables for the CAPS (Study 2) according to different selection criteria. Starting top-left, clockwise: Residual sum of squares of the regression models (RSS); Adjusted R^2 , Mallows' Cp, Bayesian Information Criterion (BIC). The X-axes denote the number of variables (i.e., the models 1-6). Best model per metric highlighted in red.

The overall model fit was significant ($R^2=0.466$, $F_{(2,47)}=20.48$, $p<.001$), with the DEMO ($\beta=0.158$, $p=.001$), but not LSC-R ($\beta=0.109$, $p=.154$), significantly predicting CAPS symptom scores. Visual assessment indicated an approximately linear pattern (Figure 6.30, centre left), and adequate homoscedasticity (Figure 6.30, bottom left), however residual distribution was still non-normal (Figure 6.30, top and centre right) and two data points were approaching Cook's distance of potential leverage (Figure 6.30, bottom right), though excluding them did not substantially alter the results. While the predictor variables were correlated ($r=0.698$, $p<.001$), their variance inflation factor (VIF 2.00) was again of no concern.

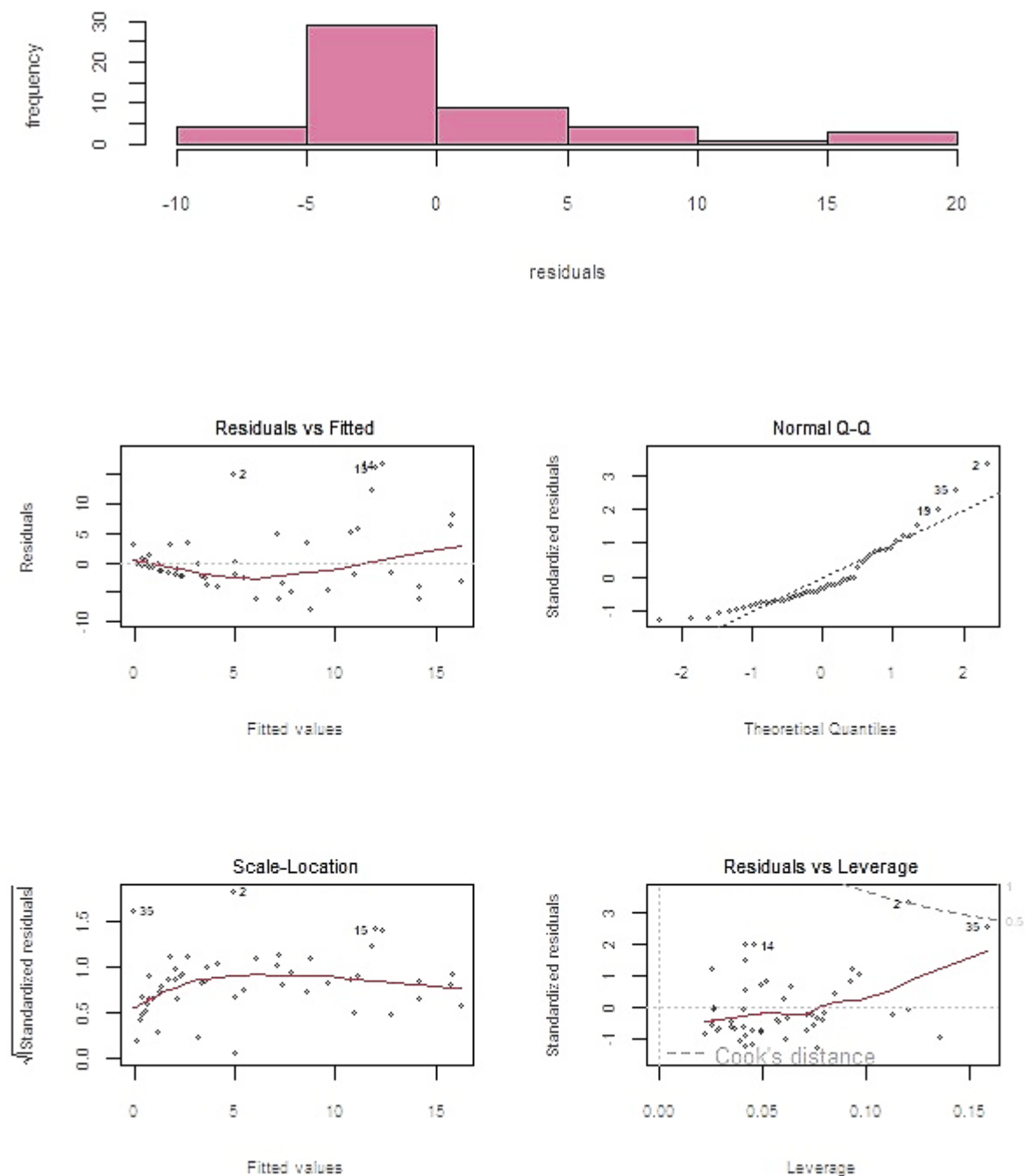


Figure 6.30

Diagnostic statistics for the best-subset selected model (Model 2) for the CAPS in Study 2. Top: histogram of the model's residuals, assessing normal distribution; Centre left: residuals vs. fitted plot, assessing (non-)linear patterns; Centre right: Q-Q plot, assessing normal distribution of the model's residuals; Bottom left: scale-location plot, assessing homoscedasticity; Bottom right: Leverage plot, assessing influential datapoints (using Cook's distance).

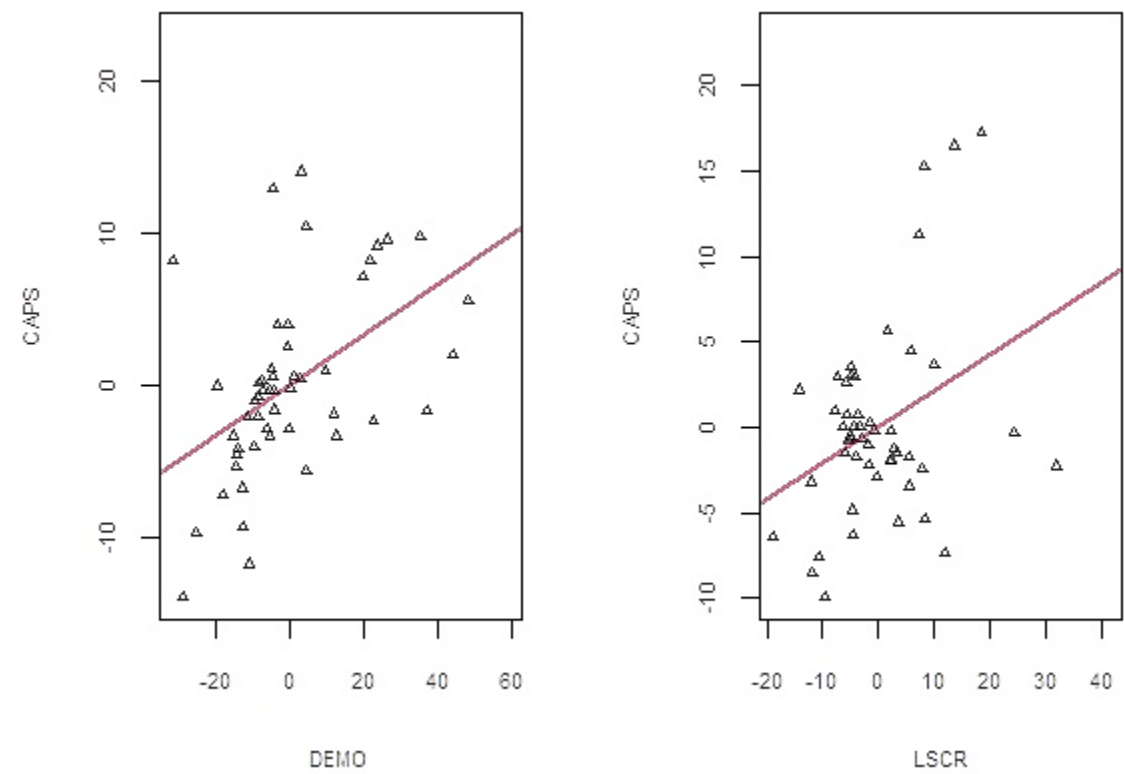


Figure 6.31

Added variable (partial regression) plots of Model 2 for the CAPS in Study 2. Left: partial regression of the DEMO (dissociation), controlled for LSC-R (Option 2, number of life-time stressors and current impact) scores. Right: partial regression of the LSC-R, controlled for DEMO scores.

6.4 Exploratory analysis: Structural equation modelling

Recalling the theoretical framework of psychosis in BPD (constructed in light of the literature as well as the current findings) it appears that several of the hypothesised factors may indeed be contributing (independently or in combination) to the development of these experiences: Trauma and post-traumatic stress are likely to form a central aspect of the paradigm, evidenced not only by a substantial body of research generally linking incidences of childhood trauma and lifetime adversity (Daalman et al., 2012; Niemantsverdriet et al., 2017) to the prevalence and severity of psychotic symptoms, but further demonstrated by results of regression analyses performed in the populations at hand. The latter additionally point towards the unequivocal significance of dissociative experiences as a possible mediator of the relationship between trauma and PEs (or as an modulating factor, independent of trauma), a hypothesis equally supported by the literature (Varese et al., 2012; Perona-Garcelán et al., 2012; Sun et al., 2018). Finally, frequently reported correlations of hallucinations and delusions with depression and anxiety (including those observed in Study 1) suggest a link (be that causal or correlational) between common mental distress and psychotic symptoms (Perez et al., 2018).

Arising from the above considerations, the following aetiological model with three latent variables (LVs) may be considered:

Could *Psychotic Experiences* (LV 1) in BPD be explained as an indirect consequence of *Trauma* (LV 2), mediated by *Dissociation* (observed variable), and perhaps further by *CMD* (LV 3) (Figure 6.32)?

6.4.1 Latent variable specifications and measurement model

Since the aim was to examine the relationship between different variables hypothesised to influence the risk for psychotic experiences in people with borderline personality disorder, only data from the BPD groups was included in the analysis. Convergent validity of the latent constructs was assessed with confirmatory factor analysis (CFA), using Maximum Likelihood (ML) estimation.

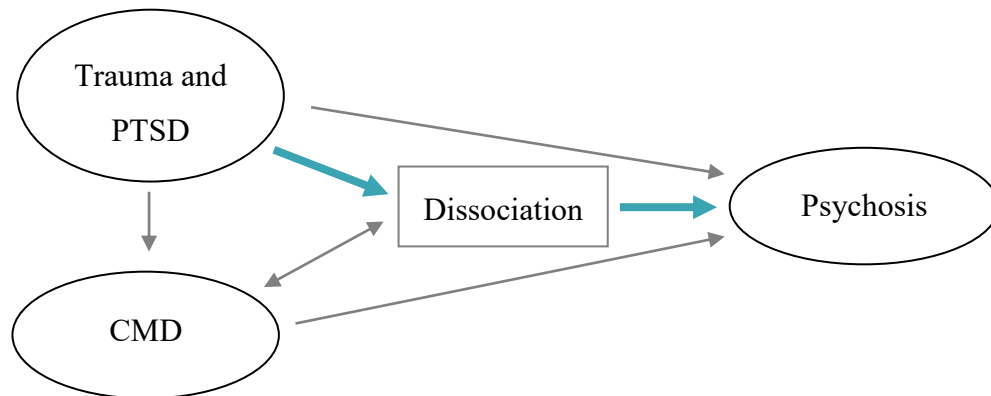


Figure 6.32

Illustration of the hypothesised interactions and primary aetiological pathway of key risk factors for psychotic experiences in BPD.

The proposed model offered an adequate fit for the data ($\chi^2_M=5.746$, $p=.452$ on 6 degrees of freedom), with all standardised factor loadings ≥ 0.5 (Appendix E.2). It should be mentioned that the chi-square statistic is only reliable when using a non-standardised covariance matrix, however alternative fit indexes also indicated good fit, including the RMSEA (0.000, CI 0.00-0.15), CFI (1.000), and SRMR (0.037). Examination of the residuals (Appendix E.2) equally confirmed the measurement model to be an acceptable approximation but did indicate a markedly high difference in covariances for the relationship between CAPS and CTQ. Notably, the CTQ was the indicator with the lowest factor loading (0.496) and (by extension) the highest unique variance. While this was likely due to the retrospective nature of the measure, removing the CTQ did not improve overall fit and it was included in the model. Similarly (and as expected), the latent variables *Trauma* and *CMD* were significantly correlated (0.827, $p=.002$) and a combination of both constructs was explored. However, this would ultimately have prevented any meaningful assessment of the independent or interactive effects of trauma and mental distress on psychotic experiences, and both LVs (and their indicators) were maintained as defined a priori. Univariate score tests were computed to identify potential sources of misfit, however no further modification indices were returned. A Lagrange Multiplier tests did not reveal any equality constraints in the model. Overall, the CFA indicated that all latent variables were supported by the data and suitable for inclusion in the structural model.

6.4.2 Structural equation model

Following assessment of the measurement model, its structural components (as illustrated in Figure 6.32) were estimated within an SEM framework. In addition to the previously defined latent constructs, the observed variable *Dissociation* (standardised scores of the Study 1 DES and Study 2 DEMO) was included as a possible mediator.

The chi-square test was non-significant ($\chi^2_M=10.212$, $p=.334$ on 9 degrees of freedom), indicating adequate goodness of fit, which was further supported by the CFI (0.994), RMSEA (0.043, CI 0.00-0.14), and SRMR (0.035) equally suggested adequate model fit. Assessment of residuals reflected the findings from the measurement model, with all (standardised) residuals at <0.05 apart from a notable (0.121) divergence between the sample and model-implied covariances of CTQ and CAPS (see Appendix E.1 for SEM parameters).

Univariate score tests identified modification indices that proposed a potential covariance of the PCL and Anxiety residuals, which was in line with the previously observed covariance of the *Trauma* and *CMD* latent constructs (as well as with a phenomenological theoretical overlap between symptoms of post-traumatic stress and generalised anxiety). Consequently, one alternative model was fitted to include the additional path. This marginally improved RMSEA (0.000, 90% CI 0.00-0.11), CFI (1.00), and SRMR (0.034). However, the model offered an improper solution to the defined parameters (specifically, a standardised coefficient of >1 for the estimated total effect of trauma and dissociation) and was therefore abandoned (see Appendix E.2 for details on the alternative model's fit).

Significant relationships (regressions) were detected between *CMD* and *Trauma* (0.84, $p<.001$), *Trauma* and *Dissociation* (0.61, $p=.001$), and *Dissociation* and *Psychotic Experiences* (0.59, $p<.001$). Furthermore, there was an indirect effect of *Trauma* on *Psychotic Experiences* (0.36, $p=.006$) (via *Dissociation*), as well as a significant accumulative effect of both *Trauma* and *Dissociation*, which appeared to explain most of the observed variance (0.95, $p=.001$). By contrast, neither *Trauma*, nor *CMD* showed a direct influence on *Psychotic Experiences*, and the residual variance of *CMD* and

Dissociation did not correlate (Figure 6.33, see Appendix E.2 for a full account of all SEM parameters).

R^2 values for the endogenous variables *CMD* (0.71), *Dissociation* (0.37), and *Psychosis* (0.50) indicated moderate determination (Chin, 1998; Hair et al., 2011).

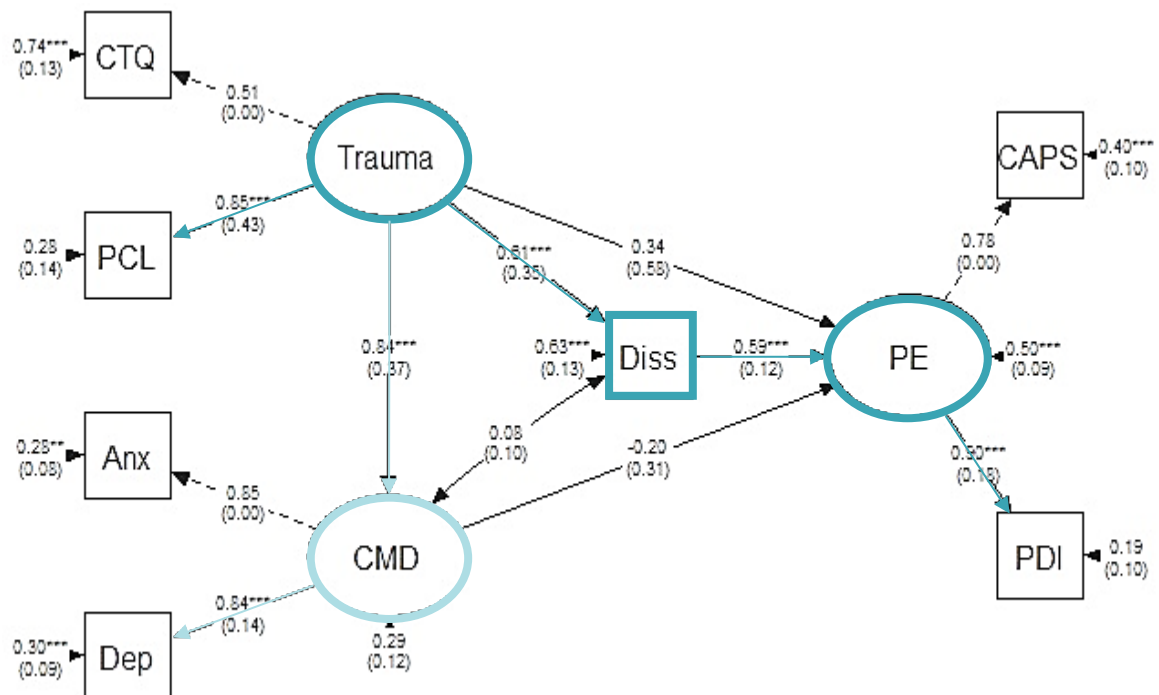


Figure 6.33

Exploratory structural equation model of key risk factors for psychotic experiences in BPD. Significant pathways highlighted in blue. Significance levels: *=.05, **=.01, ***=.001. CTQ=childhood adversity, PCL=post-traumatic stress, Anx=anxiety (GAD+STAI trait score), Dep=depression (QIDS+BDI), CMD=common mental distress, Diss=dissociation, PE=psychotic experiences, CAPS=perceptual experiences, PDI=unusual beliefs and delusions. Residual variance and standard error (in brackets) included for manifest and endogenous latent variables. Parameter estimates denoted with standard error (in brackets).

6.5 Discussion

Psychotic experiences, common mental distress, and borderline symptom severity

Symptoms of common mental distress (depression and anxiety) were moderately associated with psychotic experiences in both groups in Study 1 (but not Study 2), however, the correlations were neither strong nor consistent enough to support a causal link. Rather, they could be the result of shared aetiological underpinnings (such as trauma) that independently raises the risk for both forms of psychopathology. This is further supported by the results of the (best subset) regression modelling which seem to render the relationship insignificant once controlled for other (better) predictors.

While the results did not show a relationship between BSL score and CAPS, nor PDI symptom prevalence (within the BPD group), borderline symptom severity did appear to influence the level of distress derived from perceptual experiences, though not from beliefs. These findings are interesting in multiple regards: First, they support the hypothesis that more severe borderline pathology may not necessarily increase the number of psychotic (hallucinatory) experiences, but rather affect their qualitative appraisal – which is typically postulated as a hallmark of clinical relevance. Secondly, they do not support the hypothesis of an intrinsic link between BPD severity and overall psychosis proneness, as has previously been observed by others (Baryshnikov et al., 2018) as well as in the context of the prevalence study discussed in Chapter 4. While the smaller sample size of the BPD participant group ($n=20$, compared to $n=54$ for the previous study, and $n=282$ for Baryshnikov et al., 2018) may certainly have biased observation, it stands to mention that both other studies employed the MSI (a ten-item, yes/no screening questionnaire) whereas the current assessment used the BSL, an arguably more detailed measure of the BPD pathology spectrum. Furthermore, Baryshnikov et al. (2018) assessed a wider clinical population of patients with mood disorders, rather than a BPD specific sample. As such, their findings may more accurately indicate that an *overall* increase in borderline pathology is linked to a higher risk for psychosis – rather than symptom severity within the diagnosis. This is similarly evidenced by the current results which show an overwhelmingly significant association ($r=.624, p<.001$) when considering the full participant sample (i.e.,

BPD and control group). We must therefore consider the concrete possibility that psychotic experiences are associated with BPD symptomology per se (rather than with symptom severity), and may occur at any level of disorder presentation. This in turn, would have significant implications for diagnostic and treatment approaches.

Psychotic experiences and cognitive processes

In light of previous theoretical and experimental work that postulated abnormalities in prediction error signalling as a powerful cognitive framework for the development of hallucinations and delusions (e.g., Fletcher and Frith, 2009), one might have expected to find similarly aberrant processes in people with BPD – especially in association with psychotic experiences. By contrast, the results suggest that the Bayesian prediction apparatus is unaffected in borderline pathology: During the two-tone image task, both BPD and control participants equally benefited from the perceptual advantage granted by template exposure, and neither group showed a change in response bias (the bias towards reporting more dots on the figures than on the background). Additionally, there was no correlation between the rate of disambiguation improvement (before versus after seeing the templates) and psychotic symptoms scores (neither CAPS nor PDI).

Of course (as with any experimental paradigm) the translation of an abstract construct such as “prior knowledge” into an empirical measure such as the two-tone image task may not be complete, and improved performance could instead be invoked by something as mundane as basic priming effects. However, findings from imaging studies (Van Loon et al., 2016; González-García et al., 2018; Flounders et al., 2019) suggest that the cortical representation of two-tone images seen *post-template* more closely resembles the neural response patterns for the greyscale pictures than those for unidentified black-and-white stimuli which indicates that such tasks do measure at least some form of Bayesian perceptual reorganisation.

Instead, it appears that (some) of the cognitive mechanism underlying psychotic experiences in BPD may be fundamentally different from those associated with schizophrenia, with symptoms predominantly driven by biographical and psychopathological factors, rather than by an innate imbalance in perceptual processing.

Unfortunately, cognitive appraisals of (aberrant) prediction error signalling in people with bipolar disorder have not yet been published. It would be beneficial to understand whether their reliance on prior information resembles that of people with schizophrenia, or whether (as in BPD) the balance between priors and sensory information is largely intact. The latter could suggest a closer mechanistic relationship between the hallucinations and delusions in BPD and affective psychosis (cognitively distinct from schizophreniform disorders) and would merit further investigations of their mechanistic and phenomenological overlap.

Similarly intriguing findings were brought forth by the novel phantom words task that assessed the interpretation and emotional valence of distorted auditory verbal cues. Participants with BPD were significantly less likely to correctly identify the underlying word, and significantly more likely to endorse the negatively connotated answer option instead. Similarly, when presented with truly ambiguous stimuli (i.e., sounds that contained the shared phonetic root of a word pair but did not fully reflect either answer option), participants with BPD chose negative words more frequently than their positive counterparts, while the control group chose positive and negative options at an equal rate. These results are especially noteworthy considering the limited number of stimuli (ten per condition), that nevertheless yielded significant results.

Although striking, this tendency to ascribe negative meaning to ambiguous speech, did not correlate with psychotic experiences (neither with CAPS, nor PDI symptom scores). Instead, it may reflect a broader dimension of impairments in social cognition and emotion processing, frequently observed in people with BPD. Domes et al. (2008) investigated recognition of facial affect in people with BPD and identified a marked bias towards attributing anger to ambiguous expressions. Daros et al. (2013) further reported misattributions of emotions to faces depicting neutral expressions. While the above studies suggest that cognitive biases may be a fundamental aspect of (BPD associated) hyperemotionality, others observed such deficit in emotion recognition only under time-pressure (Dyck et al., 2009). Similarly, 2015 study investigating the influence of stress on social cognition in patients with BPD (Deckers et al., 2015) found an increase in negative evaluations of ambiguous facial emotions after stress but observed no differences between BPD and controls participants. Despite their inconsistencies, these findings broadly point

towards (borderline related) irregularities in the processing of ambiguous stimuli with a tendency to ascribe negative affect to neutral emotions, and while this has solely been assessed in the context of visual perception (and specifically facial recognition) comparable mechanisms could be at work when interpreting ambiguous auditory information. The phantom words task proves a potentially useful measure for investigating such bias, and further validation of the instrument in clinical and non-clinical populations may cement it as a valuable experimental tool.

Psychotic experiences, (childhood) trauma and dissociation

Of the three factors (common mental distress, predictive processing, and trauma) hypothesised to play a role in the aetiology of psychotic experiences in BPD, trauma and post-traumatic stress provided the strongest link to both hallucinations, and delusions. Incidences of childhood adversity (especially emotional abuse and emotional neglect) were significantly more prevalent in the BPD population than in the control group, with striking similarities across both BPD samples (which only differed on the physical neglect subscale). CAPS (but not PDI) scores were positively correlated with the CTQ total which further adds to a wide body of literature linking childhood trauma to hallucination proneness in BPD, and beyond (Ross and Joshi, 1992; Ross et al., 1994; Read et al., 2003; Daalman et al., 2012; Gaudiano and Zimmerman, 2009). Previous claims of a particularly strong association between psychotic symptoms and childhood sexual abuse (Varese et al., 2012) were (tentatively) supported by the first, but not by the second study. Of course, results of the latter may have been biased by a comparatively small sample size and a low incidence rate of SA. However, in Study 1, each of the five other CTQ subscales also correlated with psychotic experiences scores – which somewhat undermines the assumed explicit importance of sexual trauma.

PCL scores (measuring current symptoms of PTSD) were significantly elevated in the BPD groups, and correlated with both the CAPS, and (especially) the PDI. This raises the interesting (and often posed) question whether in the aetiology of psychosis the prevalence and number of adverse experiences alone (e.g., through neurodevelopmental changes), or rather their persisting effects (i.e., post-traumatic stress and its associated symptoms) is a

better predictor of the relationship between trauma and PEs. Equally, wide ranges of studies have shown both direct and mediated influences of past traumatic events.

In this context, the results of the LSC-R are particularly intriguing: Firstly, number and severity of life-time stressful events were more strongly correlated with PEs than the CTQ, or any of its subscales. Considering that the LSC-R is predominantly a measure of adult-life stressors (and not just events that occurred during childhood), it raises the question if perhaps unwarranted emphasis is being placed on early-life events when consideration of overall trauma may be more accurate. Secondly, psychotic experience prevalence (for both CAPS and PDI) was also more strongly correlated with the *number* of previously experienced life-stressors, than with their subjective, ongoing (past-year) effect. These findings indicate a relationship between traumatic or stressful events and psychosis proneness irrespective of their perceived psychological impact. Overall, the results replicated previous findings (Niemantverdriet et al., 2017) of a greater number of life stressors as correlate for more (severe) psychotic experiences.

Notably, Kelleher et al. (2013) reported that cessation of trauma predicts cessation of psychotic experiences, which may further underline the impact of adult life (and especially ongoing) adversity. The same study also observed a dose-response relationship between peer abuse (“bullying”) and risk for psychosis. Similar findings have been reported in a community sample of adolescents (Strauss et al., 2018), a nonclinical sample of young adults (Cristóbal-Narváez, 2016), as well as by a longitudinal, population-based cohort study repeatedly assessing young people from the ages of eight to eighteen years (Wolke et al., 2014). A 2012 systematic review and meta-analysis (Van Dam et al., 2012) determined that non-clinical studies provided (predominantly) consistent evidence supporting the correlation between (school) peer abuse and the development of (clinically not significant) psychotic experiences – an association which strengthened with increased frequency, severity, and duration of the victimisation. By contrast, results of the studies in clinical populations were found to be inconsistent and they advised that the literature (at the time) was too sparse to draw definite conclusions. Interestingly, while not included as part of the original twenty-eight LSC-R life-stressors, 35% of the BPD sample reported bullying under item twenty-nine (“*any other events*”), a number likely to be under-

representative of the true incidence rate considering responses on this factor were not prompted. Overall, this highlights the historic and continued failure to consider peer abuse as a potentially devastating influence on psychological development as well as a possible factor in the aetiology of BPD, and (potentially) of psychotic experiences.

If the influence of trauma on psychosis is indeed (or in part) indirect, then dissociation is a promising candidate for mediating the relationship. It appears increasingly associated with psychotic symptoms across clinical and non-clinical populations, and several studies have found partial (or even full) moderation of the correlation between trauma and hallucinations (Varese et al., 2012; Muenzenmaier et al., 2015) – as well as delusions (Sun et al., 2018) – by dissociative experiences. Correspondingly, in the context of this dissertation, the strongest association between psychotic experiences and psychopathology were observed for the DES (Study 1) and DEMO (Study 2), respectively. This relationship was especially strong for delusional experiences, and (unlike other all other measures) permeated the control groups, with remarkably similar coefficients but a significantly lower baseline. This suggests that psychotic experiences in people with BPD may (to a substantial extent) be driven by general factors that increase the risk for psychosis in anyone but are especially prevalent in the context of this disorder, rather than by distinct, borderline-specific mechanisms.

Best (subset) predictors for psychotic experiences

Considering the abundance of hypothesised risk factors – but lack of aetiological or mechanistic models for how exactly they may influence psychotic experiences in BPD – the aim of the best subset regression was to ascertain which (and how many) of the correlates most comprehensively explain symptom variance and can most reliably predict the prevalence of hallucinations and delusions.

Results from the PDI analysis were especially noteworthy, with dissociative experiences and trauma as the unswervingly most significant predictors across all metrics, different instruments, and both study populations. Moreover, the final regressions of Study 1 and Study 2 explained a remarkably similar amount of PDI variance (61% and 63.3%, respectively), further supporting the reliability of the model. Interestingly, in both cases

the second-best solution was a three-factor model additionally considering BPD pathology (diagnostic status in Study 1 and BSL in Study 2). This suggests that, while a substantial part of the risk for psychosis can be explained by transdiagnostic factors, there may still be an additional variable innately associated with borderline psychopathology. The sizable proportion of unexplained variance also points in this direction.

By contrast, ascertaining the best subset of predictors for perceptual experiences proved much less straightforward since different metrics yielded conflicting solutions. Furthermore (and unlike the PDI), CAPS findings appeared to diverge between studies, with results from Study 1 proposing a two-factor solution of dissociation and diagnostic status (with post-traumatic symptoms not considered until the 5-factor model), while Study 2 suggested a two-factor model of dissociation and trauma (with BPD pathology not considered until the 4-factor solution). However, in both cases correcting the second variable for the influence of dissociation negated its statistical significance, so that dissociative experiences equally emerged as the most reliable predictor for hallucinations.

One possible explanation for the difference (between PDI and CAPS) is the impact of specific types of trauma on specific types of psychotic experiences. A 2019 study on the relationship between the nature of the traumatic event and the psychotic symptom displayed reported that childhood neglect was first and foremost associated with paranoia, suspiciousness, and persecutory ideas (Ered and Ellman, 2019). Sexual abuse has been associated with perceptual abnormalities (Thompson et al., 2010; Velthorst et al., 2013), and physical abuse with perceptual abnormalities and grandiose belief (Velthorst et al., 2013). Considering the results of both current studies in which the principal types of abuse reported by BPD participants were emotional abuse and emotional neglect – and the principal types of psychotic beliefs paranoia and suspicion – it is perhaps not surprising that the aetiological model for the PDI (via trauma and dissociation) is highly consistent, and the model for the CAPS perhaps simply underpowered. Yet, it also begs the question if a linear dose-response relationship is strictly appropriate for estimating the link between dissociation (and trauma) and perceptual experiences. Instead, the association might be better understood as exponential – a stark surge in risk driven by a few significant (perhaps

early-life) adverse events followed by a levelling with no additional increase in psychosis proneness, despite further traumatic experiences. In the current literature, there is little evidence to support or refute this hypothesis. Frequent claims of a dose-response relationship (e.g., as purported in a recent review by Giannopoulou et al., 2023) are upon closer inspection more accurately described as a dose-threshold relationship. Several studies noted an association between cumulative trauma exposure and (life-time) presence of psychotic symptoms (yes/no) (Whitfield et al., 2005; Anda et al., 2006; Spauwen et al., 2006; Shevlin et al., 2011), while others assessed the influence of trauma frequency or severity on the presence psychotic symptoms (Janssen et al., 2004; Lataster et al., 2006; Schreier et al., 2009). One research group (in a somewhat different approach) assessed the relationship between psychotic experience severity and presence of previous trauma-exposure (yes/no) (Heins et al., 2011). Most of these studies were conducted in community samples in which trauma may not be as prevalent as in a BPD sample and the trauma-psychosis relationship therefore more direct. Two experiments that did assess cumulative effects of multiple versus single trauma-exposure in a group of people with psychotic disorder (Fisher et al., 2009; Fisher et al., 2010) found no evidence for a dose-response effect, whereas Loewy et al. (2019) reported a direct (albeit weak, $r=.29$, $p=.002$) correlation between number of traumatic events and severity of perceptual abnormalities. Given the inconclusive literature and the present findings a non-linear relationship between trauma and hallucinations remains a distinct possibility and should be given further consideration.

Structural equation modelling of psychosis risk factors

To delve deeper into the hypothesised interactions between different risk factors, and to ascertain their direct and indirect effects on psychotic experiences, an explorative analysis using structural equation modelling was conducted on a combined BPD sample, composed of the participant populations from both studies. The measurement model was statistically significant and indicated good fit of the three latent variables (trauma, CMD, and psychotic experiences) and their respective loading factors. Subsequently, a structural model was defined to test the principal hypothesis resulting from the preceding correlation and regression analyses; specifically, that psychotic experiences in BPD could be the result of

trauma and post-traumatic stress, mediated by dissociation. The severity of common mental distress (in the form of depression and anxiety) may further exacerbate symptoms, or perhaps contribute to dissociative experience prevalence. The overall model showed adequate fit on all principal metrics. The primary aetiological pathway (trauma - dissociation - psychosis) was statistically significant with a direct effect of dissociation, as well as an indirect (but no direct) effect of trauma on psychotic experiences. Trauma additionally had a significant influence on common mental distress, however CMD was not associated with either dissociation or psychotic experiences (neither directly, nor indirectly). This further supports the earlier conclusion that the high levels of depression and anxiety in people with BPD who experience psychotic symptoms are most likely the artefact of a shared causal mechanism, rather than predictors of a causal relationship.

In line with outcomes from the best subset regression, the PDI (but not the CAPS) loaded significantly on the psychosis latent construct. The observed variables CTQ and (more surprisingly) Anxiety did also not achieve significance level. This was likely an issue of computing power (typically SEMs are more effective for sample sizes of three-hundred or more observations), which was anticipated and acquiesced in light of the explorative nature of the analysis. However, it does denote that the model is perhaps more accurately described as the relationship between post-traumatic stress, dissociation, depression, and delusions. Interestingly, residuals revealed the (by far) largest divergence between the data-observed and the model-implied covariance between the CTQ and CAPS, which further affirms the earlier proposition that the relationship between childhood trauma and hallucination (severity) may be more complex than its (non-) association with delusions.

Of course, this particular structural model is evidently explorative in nature and any inference should be made with due diligence. Nevertheless, its statistical significance in spite of the obvious methodological limitations, and the fact that the proposed relationships reflect not only the cumulative results of the present studies but the broader understanding of how and why psychotic experiences may arise from previous trauma, speak volumes to the model's potential explanatory value. As such, it would be of great interest to further explore (and possibly extend) the postulated structure with a sample and sampling method more adequately suited to SEM analysis.

Chapter 7 Under pressure? The role of stress

“Answers [of not endorsing CAPE-15 symptoms] are true for last few months but it might be worth mentioning that I have experienced severe psychosis in the past. Stress/major life events/overwhelm etc seem to trigger the sudden onset of symptoms. I don’t know if that’s relevant.”

7.1 Introduction

The relationship between stress, and the physiological and psychological changes it incites is notoriously complex. A myriad of different physical, psychological, and emotional events can trigger a stress response and in turn, one stressful event can spur a cascade of physiological, psychological, and emotional reactions that differ drastically between individuals and situations.

In the context of psychotic experiences in BPD, the mention of stress as a potential causal or triggering factor can often be accompanied by a nagging feeling of devaluation: if these symptoms are (predominantly) present in times of stress they must be less real, less relevant, and therefore less worthy of clinical or theoretical attention. A subtle insinuation of it being “just another borderline overreaction” (and that people might be able to control their symptoms, if they would only control their stress levels) is still reminiscent of the ostensibly “voluntary” psychotic experiences purported forty years ago (Pope et al., 1985). This risks invalidating the often considerable distress accompanying such symptoms, and may further alienate patients who might not agree with the framing of their suffering as merely transient, stress-related peculiarities. This dilemma is especially unfortunate considering the increasing evidence for the devastating and long-lasting effects that stress

(past, acute, and chronic) can have on physical health, cognition, emotion regulation, and psychopathology – including psychotic symptom.

7.1.1 Stress and psychosis

Most current theories on the contribution of stress to psychotic symptom development are rooted in the traumagenic neurodevelopmental model of schizophrenia (Read et al., 2001), also described as the stress-vulnerability model. Born out of the observed similarities between effects of early-life traumatic events on the developing brain and some of the chemical, cognitive, and structural changes found in people with schizophrenia, the model offers a pathway from genetic vulnerability, over trauma and adversity, to psychosis – via dysregulation of the body's stress response. One of the neurobiological pathways central to this paradigm is the hypothalamic-pituitary-adrenal axis (HPA), a complex neuroendocrine system of directional influences and negative feedback loops between the paraventricular nucleus of the hypothalamus, the anterior lobe of the pituitary gland, and the adrenal cortex, that regulates reactions to stress (alongside a myriad of further bodily functions including emotional reactivity, immune responses, mood, and digestion). In humans, its principal “output” is the glucocorticoid cortisol, a steroid hormone often measured as a physiological indicator for stress (Dziurkowska and Wesolowski, 2021).

In one of the earliest assessments of neuroendocrine functioning in people with schizophrenia, Sachar (1975) observed that prior to psychotic relapse, cortisol levels were extensively elevated (by up to 250%), and that this was frequently immediately preceded by stressful events. Similarly, a reduction in cortisol levels has been linked to an improvement of (negative) psychotic symptoms (Zhang et al., 2005). Longitudinal studies additionally reported that higher levels of cortisol expression in at-risk adolescents can predict later conversion to psychosis (Walker et al., 2010), and are associated with psychotic symptom severity (Walker et al., 2013). Moreover, basal cortisol levels appear to be increased in UHR populations, compared to controls (Chaumette et al., 2016). Despite inconsistencies in results on whether (Fries et al., 2005), and when (e.g., diurnally: Braehler et al., 2005; in the morning: Girshkin et al., 2014; throughout the day: Mondelli et al., 2010; in the afternoon: Christie et al., 1986; or during the night: Jiang and Wang, 1998) cortisol

expression is elevated in populations with psychosis, these (and further) findings generally point towards an overactivity of the HPA axis as an important factor in the pathway from stress to psychosis.

This is further supported by the fact that raised glucocorticoid levels (either as a consequence of acutely stressful events or due to such an underlying dysregulation of the HPA system) are associated with an increase in dopamine levels (De Weerth et al., 2003; possibly to facilitate cue-reward learning: Lopez and Flagel, 2021), which have long been linked to the prevalence and severity of hallucinations and delusions. The levels of other neurochemical factors (glutamate, and serotonin) thought to be implicated in the pathogenesis of schizophrenia are similarly known to correlate with stress and endocrine functioning (glutamate: Moghaddam, 1993; Lowy et al., 1993; serotonin: Meijer and De Kloet, 1998; Chaouloff et al., 1999). Dopamine agonists in turn, can further elevate HPA activity (Piacentini et al., 2004; Madden et al., 2005) thereby possibly creating a feedback loop of chronic stress and aberrant salience that increases the risk for (or transition to) psychotic psychopathology. Interestingly, correlations between the increase of striatal dopamine and cortisol levels in response to psychosocial stress have also been observed in people with a history of interpersonal trauma (Pruessner et al., 2004), offering a possible neuroendocrine pathway from trauma to psychosis.

7.1.2 Stress in BPD

As previously lamented, few experimental paradigms have explored the impact of stress on psychotic experiences in BPD. However, parallels may be drawn from a series of investigations into other aspects of the relationship between stress reactivity and (borderline) psychopathology. A 2013 study on emotional reactivity and emotion regulation strategies in response to a fear stressor found that people with high borderline personality features exhibited greater levels of fear, distress, and hostility following the stress induction (compared to people with low BPD traits), as well as greater use of emotion suppression and lower emotional acceptance (Chapman et al., 2013). While the authors measured dissociative symptoms before and after the event (as covariate), assessments of changes in state dissociation were unfortunately not reported and it is unclear whether these

symptoms may have increased in response to the fear stressor. Nevertheless, several other studies have examined the impact of psychosocial stress on dissociative experiences with findings largely indicating an association between dissociation (frequency and severity) and the duration and intensity of the tension (Stiglmayer et al., 2001), as well as incidences of every-day stressors (Stiglmayer et al., 2008). Graumann et al. (2023) examined the association between baseline dissociation levels and stress-induced dissociative symptoms across different diagnostic categories (people with BPD/PTSD or major depressive disorder, compared to non-clinical controls), and reported significant increases in somatoform dissociation and derealisation in response to the Trier Social Stress Test (observed in both clinical groups), as well as an association between the level of baseline dissociation and the rate of symptom increase. The sole known study on stress-related psychotic reactivity in people with BPD (as well as participants with cluster C personality disorder and participants with psychotic disorder) reported a significant increase in psychotic experiences in relation to daily life stress in all three patient samples (but not in the control group) with the BPD group showing a markedly stronger increase (comparable to the psychotic disorder group) in hallucinatory experiences than people with cluster C PD (Glaser et al., 2010).

Overall, these findings suggest that a dysregulated HPA system, including a heightened stress response may be yet another link in the long chain from trauma, to dissociation, to psychotic experiences in people with BPD. Evidence from the literature is further corroborated by the analysis presented in Chapter 5.3.3, in which participants overwhelmingly endorsed stress as a potential trigger for their experiences. As such, it must be determined whether perhaps a chronically elevated stress baseline, or rather an enhanced stress-sensitivity and reactivity (or both) might increase the risk for psychotic experiences. Equally, the mediating role of dissociation (not just as a general predictor for psychosis proneness, but as a potential real-time modulator of changes in state symptoms) must be investigated. Furthermore, given the widely recognised impact that stress has on cognitive functioning (Kirschbaum et al., 1996; Lupien and Lepage, 2001; Li et al., 2008; Sandi, 2013) it is of value to explore whether cognitive (predictive processing) mechanisms

thought to be implemented in the pathogenesis of psychosis (but previously found to be unaffected in people with BPD), may be “activated” during acute stress.

Accordingly, the aim of the final analysis was to ascertain differences in baseline stress, stress-reactivity, and changes in psychotic and dissociative symptom states (between participants with BPD and non-clinical controls), by addressing the following questions:

1. Do participants with BPD report higher baseline stress-levels than control group participants and/or exhibit a more pronounced increase in (subjective) stress in response to a psychosocial stressor?
2. Does stress exposure affect the severity of dissociative and/or psychotic state symptoms, either independently or in correlation?
3. Are predictive processing mechanisms affected by stress exposure in such a way that a previous balance of prior versus sensory information could become aberrant?

7.2 Analysis

This chapter focusses on the state symptom measures and tasks that formed part of Study 2 assess potential changes in symptomology and cognition following an acutely stressful event. Specifically, it evaluates differences in psychological symptom measures before versus after stress induction (within and between groups), using data from the six self-report stress surveys (“stress-checks”), as well as the PSI (as a measure of psychotic experiences), the two-tone image task⁴⁴ (perceptual processing), and the CADSS interview (dissociative states). For more information about the task and questionnaire measures, see Chapter 3.2 (questionnaires) and Chapter 3.3 (tasks). The study procedures can be found in Chapter 3.5.2 (or abridged as study-flow in Appendix C.2) Participant demographics are summarised in Chapter 3.5.3, Table 3.5.

⁴⁴ Two-tone image results from the pre stress condition were previously discussed as part of the assessment of risk factors and mechanisms in Chapter 6.3.1. This chapter will report on the post stress findings and assess any changes in task performance following the stress induction.

To explore potential differences in stress levels, a repeat-measure mixed model analysis of variance (ANOVA) was conducted with diagnostic status as the between-subject and time (number of stress-checkpoint) as the within-subject variable. Post-hoc comparisons of the relevant data points (beginning of study, as well as immediately pre and post induction) were performed using paired t-tests (within subjects) and independent two-sample t-tests (between subjects). Changes in dissociative and psychotic state symptoms were also assessed with paired and independent t-tests. Between-group findings were followed up by a one-way analysis of covariance (ANCOVA) to ascertain if the observed differences remained significant when controlling for participants' dissociative and psychotic trait symptoms (using DEMO scores as covariate for the CADSS, as well as PDI and CAPS scores as covariates for the PSI).

Assumptions of ANOVA and ANCOVA models were verified as follows: The relevant measures were screened for outliers, using the interquartile range and Grubb's test. Normality was assessed visually (histograms and Q-Q plots), and using Kolmogorov-Smirnov normality tests with Lilliefors correction. Homogeneity of variances was ascertained using Levene's test ($p > .05$), and homogeneity of covariances was evaluated with Box's M test ($p > .001$). The R package [rstatix] was used to test for sphericity (Mauchly's test, with automatic application of Greenhouse-Geisser correction to factors violating the assumption), and to compute the models.

Unless otherwise stated, approximately normal distribution of the relevant measures was observed. All analyses and graphs were done in R.

7.3 Results

7.3.1 Baseline stress and response to stress induction

Both groups showed a rise in stress ratings following the stress induction event, between checkpoints four and five (Figure 7.1, top). A repeat-measures mixed ANOVA (checkpoints 1-6, between and within groups) indicated significant differences in stress levels between groups ($F_{(1,48)}=9.09$, $p=.004$) and between checkpoints ($F_{(4,202)}=18.02$,

$p < .001$), but no interaction between diagnostic status and changes in stress ratings ($F_{(4,202)} = 0.45$, $p = .078$). Post-hoc pairwise comparison revealed significant group differences in baseline stress ($F_{(1,48)} = 5.42$, $p = .024$), which remained significant (at $p < .05$, see Appendix F.1) throughout the study with the exception of checkpoint 5, immediately after stress induction ($F_{(1,48)} = 3.38$, $p = .072$).

Paired t-tests assessing the direct increase in stress following the induction event were significant in the control group ($t_{29} = 4.56$, $p = .001$), but not in the BPD group ($t_{19} = 2.18$, $p = .631$), which may have been due to their premature slight increase in stress at the previous checkpoint. Notably, immediately prior to checkpoint four participants were asked to complete the CTQ, LSC-R, and PCL, measures of trauma and associated symptoms that involve recalling potentially distressing memories and may have contributed to an earlier rise in stress. Since control participants reported significantly fewer adverse experiences, BPD participants would have been disproportionately affected by this effect. Nevertheless, BPD group stress levels after the induction event differed significantly from the baseline recorded at the beginning of the study ($t_{38} = 3.21$, $p = .005$). Since all tasks and questionnaires were first performed between checkpoints one and two, and then repeated after checkpoint five, the state manipulation retains validity. As suggested by the ANOVA, there was no difference in the rate of increase pre to post manipulation between BPD and control participants ($t_{47} = 0.80$, $p = .43$).

On average, BPD participants felt slightly less alert throughout the study (Figure 7.1, bottom). A repeat-measures mixed ANOVA (checkpoints 1-6, between and within groups) indicated significant differences between checkpoints for the control group ($F_{(3,97)} = 2.86$, $p = .035$), however this did not remain significant during post-hoc comparisons with pairwise t-test (Appendix F.2). There were no differences between groups ($F_{(1,48)} = 1.43$, $p = .238$) and no interaction between diagnostic status and changes in the level of alertness ($F_{(1,198)} = 0.775$, $p = .546$). Subjective feelings of being ‘in-control’ were significantly affected by both group ($F_{(1,48)} = 18.35$, $p < .001$) and number of checkpoint ($F_{(1,240)} = 9.22$, $p < .001$) and the mixed ANOVA additionally indicated an interaction between diagnostic status and changes in ratings ($F_{(1,240)} = 4.23$, $p < .001$). Post-hoc comparison revealed that, while there was no difference between groups at the beginning of the experiment session

($F_{(1,48)}=2.23, p<.142$), BPD participants experienced an unexpected and significant (paired t-test $t_{47}=-4.34, p<.001$) drop in their sense of control between checkpoints one and two⁴⁵ (Figure 7.1, centre). The resulting difference between the groups remained significant at $p<.01$ throughout the remainder of the study session (see Appendix F.3). Ratings were not affected by the stress induction for neither the BPD ($t_{19}=0.24, p=1$) nor the control group ($t_{29}=1.76, p=1$).

7.3.2 Main analysis: Impact of stress induction

Psychotic state symptoms (PSI)

Surprisingly, participants of both groups had an on average lower PSI total on their repeat measure, compared to their initial (paired $t_{19}=2.04, p=.056$ and $t_{28}=2.91, p=.005$ for BPD and control group respectively) (Figure 7.2, left).

Compared to controls, BPD participants reported significantly more symptoms on both assessments ($t_{25}=5.01, p<.001$ and $t_{30}=4.97, p<.001$ before and after stress, respectively). A one-way ANCOVA (controlling for participants' PDI and CAPS scores) was still significant for the first ($F_{(1,46)}=4.37, p=.042$) but not for the second PSI score ($F_{(1,46)}=2.66, p=.110$). Curiously however, the covariates were not significantly related to PSI outcome, neither before ($F_{(1,46)}=0.627, p=.432$ and $F_{(1,46)}=1.30, p=.261$ for CAPS and PDI, respectively) nor after stress ($F_{(1,46)}=1.05, p=.312$ and $F_{(1,46)}=1.67, p=.202$ for CAPS and PDI, respectively). Psychotic symptom trait scores also had no influence on the PSI pre versus post stress difference ($F_{(1,46)}=0.01, p=.915$ and $F_{(1,46)}=0.01, p=.929$ for CAPS and PDI, respectively).

⁴⁵ On average, this would have been within the first five to ten minutes of the study, around fifteen to twenty minutes after arriving at the test site. There were no differences in set-up or experimenter conduct that would provide an immediate plausible explanation for this observation. For more details on the experiment procedure and questionnaires that were presented to participants between the two checkpoints, see Chapter 3.5.2, or Appendix C.2.

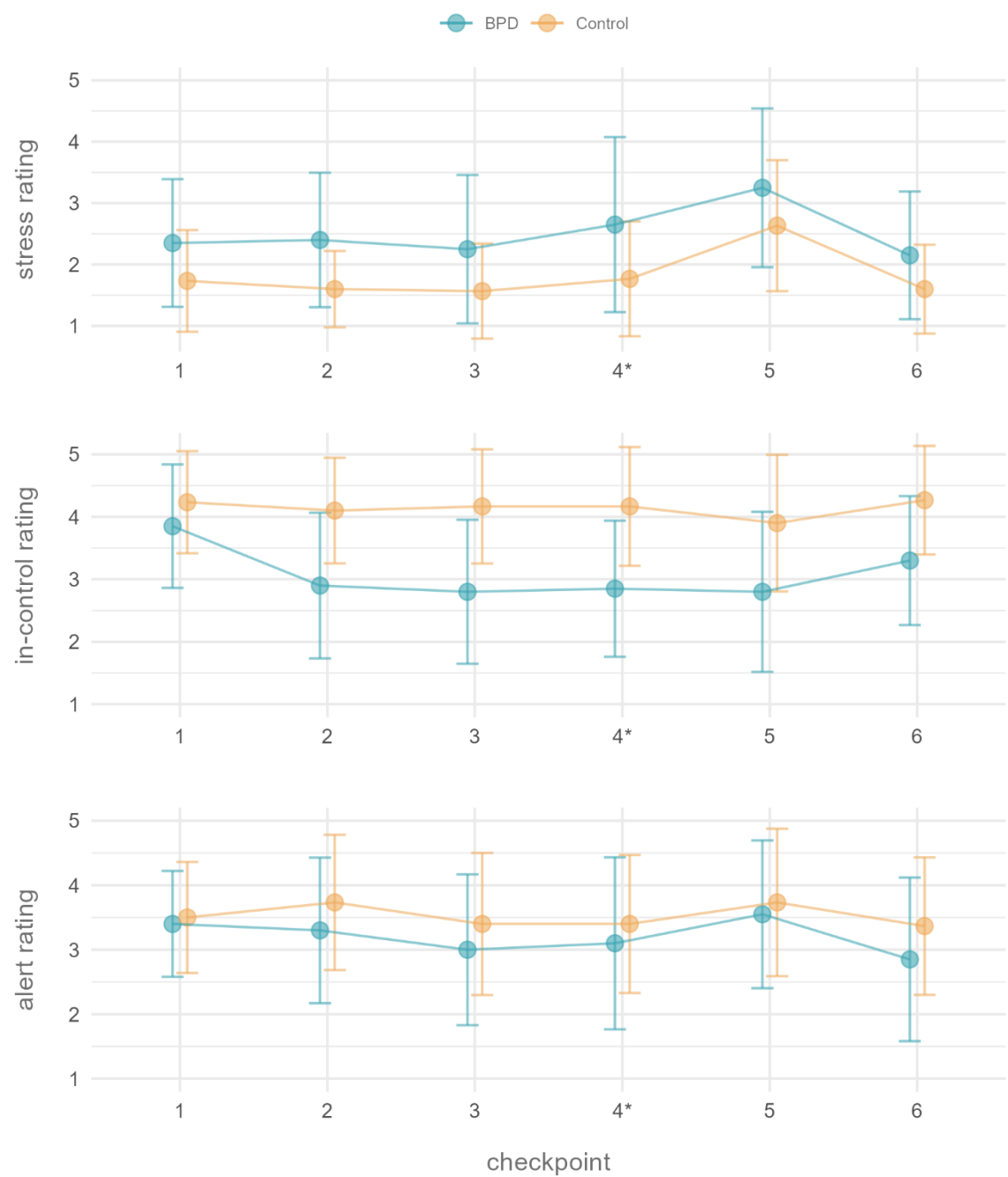


Figure 7.1

Mean and standard deviation for BPD and control participants at each of the six self-report checkpoints for stress (“How stressed do you feel right now?”, top), feeling in-control (“How in control do you feel right now?”, centre), and alertness (“How alert do you feel right now?”, bottom). *Stress induction event occurred between checkpoints four and five. For complete pairwise comparisons see Appendix F.

PSI scores were distributed normally for both groups (before as well as after the stress induction), but the assumption of homogeneity was not met (Levene $F_1=1.55$, $p=.220$ and Levene $F_1=0.09$, $p=.76$ for pre and post stress, respectively) and the data was transformed into weighted averages before fitting the ANCOVA model. Using the interquartile range, no outliers were identified.

There was no correlation between the difference in PSI scores and the rate of stress increase between checkpoint 1 and checkpoint 5 ($r=-0.08$, $p=.583$). Interestingly however, there was a negative correlation between the baseline PSI total and the pre versus post stress difference in the BPD group ($r=-0.624$, $p=.003$), though not in control participants ($r=-0.03$, $p=.867$) (Figure 7.4, left).

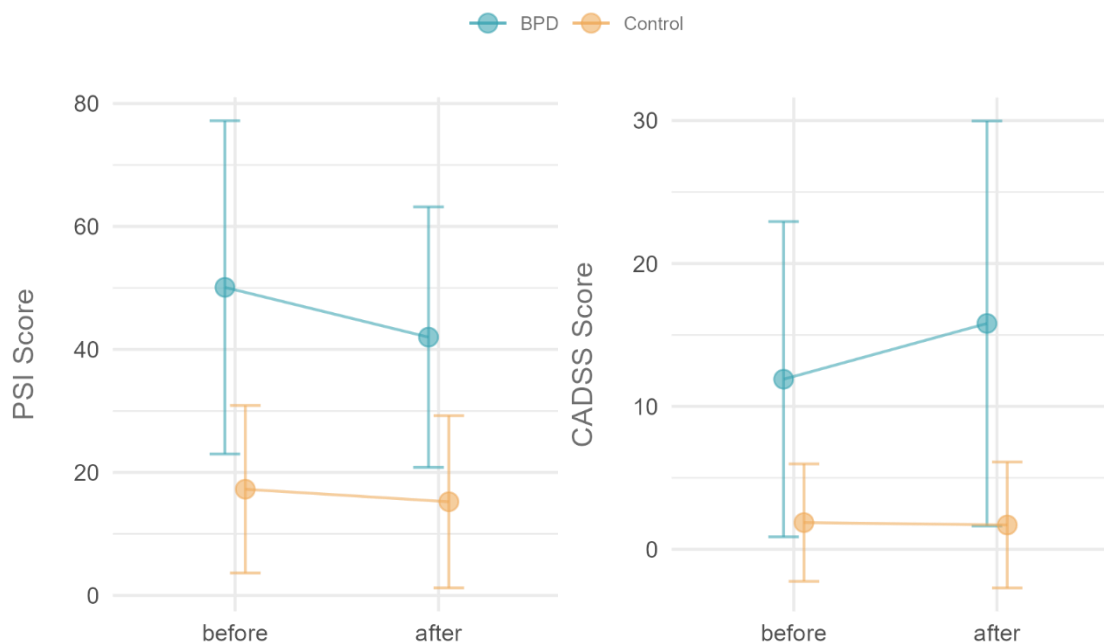


Figure 7.2

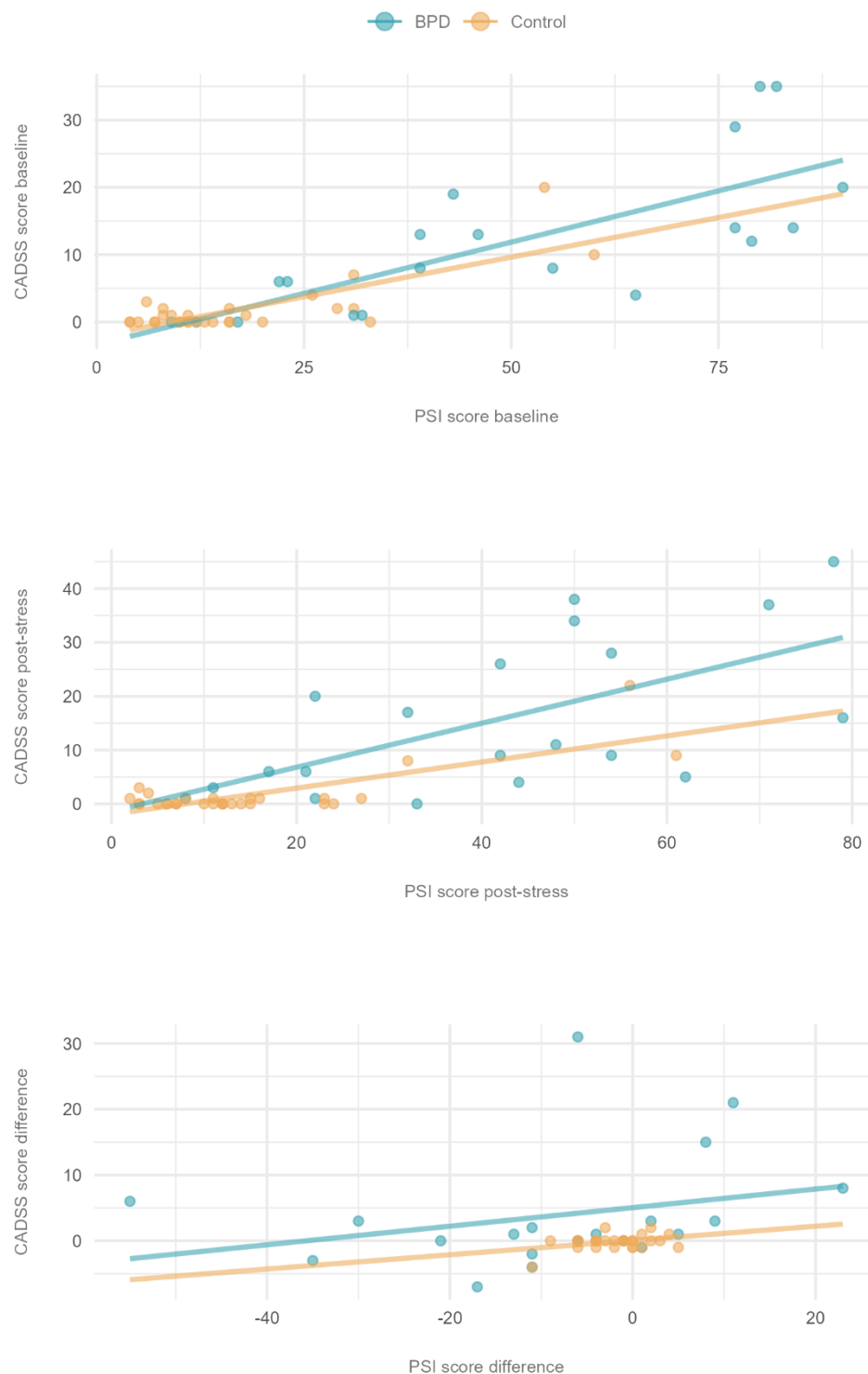
Changes in PSI (psychotic experiences, left) and CADSS (dissociative experiences, right) scores for BPD and control participants before (measured between stress checkpoint 1 and 2) and after (measured between stress checkpoint 5 and 6) stress induction. To avoid overplotting, graphs are presented offset (jittered). PSI pre stress: BPD (mean=50.1, SD=27.1), control (mean=17.24, SD=13.9); PSI post stress: BPD (mean=41, SD=21.2), control (mean=15.21, SD=14.25); CADSS pre stress: BPD (mean=11.9, SD=11.03), control (mean=1.93, SD=4.17); CADSS post stress: BPD (mean=15.8, SD=14.17), control (mean=1.76, SD=4.47).

Dissociative state symptoms (CADSS)

Compared to the control group, BPD participants reported significantly more current-state dissociative experiences pre stress (Welch's $t_{21}=3.89$, $p<.001$), as well as post stress (Welch's $t_{21}=4.31$, $p<.001$). While the control group data was non-normally distributed (Shapiro: $W=0.521$, $p<.001$), Mann-Whitney U tests with continuity correction were still significant at $p<.001$ ($U=494$ and $U=540$ for CADSS scores one and two, respectively). For control participants, differences in CADSS scores in response to the stress induction were also non-normally distributed (with a mean and median of zero) (Shapiro: $W=0.772$, $p<.001$), and a Mann-Whitney U test showed no difference before and after stress induction in this group ($U=47$, $p=.537$) (Figure 7.2).

Differences in the BPD group were approximately normally distributed, with a trend towards reporting more symptoms after stress – although a paired t-test did not indicate significance at a 95% CI (paired $t_{19}=1.94$, $p=.067$). Similarly, there was a tendency towards an overall greater increase of CADSS scores in response to the stress induction for the BPD group, compared to control participants (Welch's $t_{19}=2.02$, $p=.058$), which again was non-significant.

In contrast to the PSI, CADSS score differences between BPD and control group were no longer significant after correcting for the effect of 'trait' dissociation (DEMO) ($F_{(1,47)}=0.006$, $p=.938$ and $F_{(1,47)}=0.204$, $p=.654$ for CADSS scores one and two, respectively), and DEMO scores significantly predicted dissociative state symptoms both before ($F_{(1,47)}=45.76$, $p<.001$) and after ($F_{(1,47)}=47.56$, $p<.001$) stress. Four outliers (two in the BPD and two in the control group) were identified prior to fitting the ANCOVA model, however removing them did not affect results and their data was included in the analysis. An increase in CADSS score was significantly related to the increase in stress levels between checkpoint 1 and checkpoint 5 in the BPD ($r=0.476$, $p=.034$), but not in the control group ($r=-0.016$, $p=.931$). Higher DEMO scores did not predict a larger increase in CADSS score following stress induction in neither BPD ($r=0.314$, $p=.177$), nor control participants ($r=-0.27$, $p=.149$). There was also no correlation between baseline dissociative symptoms and the difference in CADSS scores before versus after stress ($r=-0.185$, $p=.198$) (Figure 7.4, right).

**Figure 7.3**

Correlations between PSI (psychotic) and CADSS (dissociative) symptom scores at baseline (top) (BPD: $r=0.748$, $p<.001$, Control: $r=0.783$, $p<.001$), after stress induction (centre) (BPD: $r=0.611$, $p=.025$, Control: $r=0.774$, $p<.001$), as well as between their respective pre versus post stress differences (bottom) (BPD $r=0.279$, $p=.233$, Control $r=0.380$, $p=.252$).

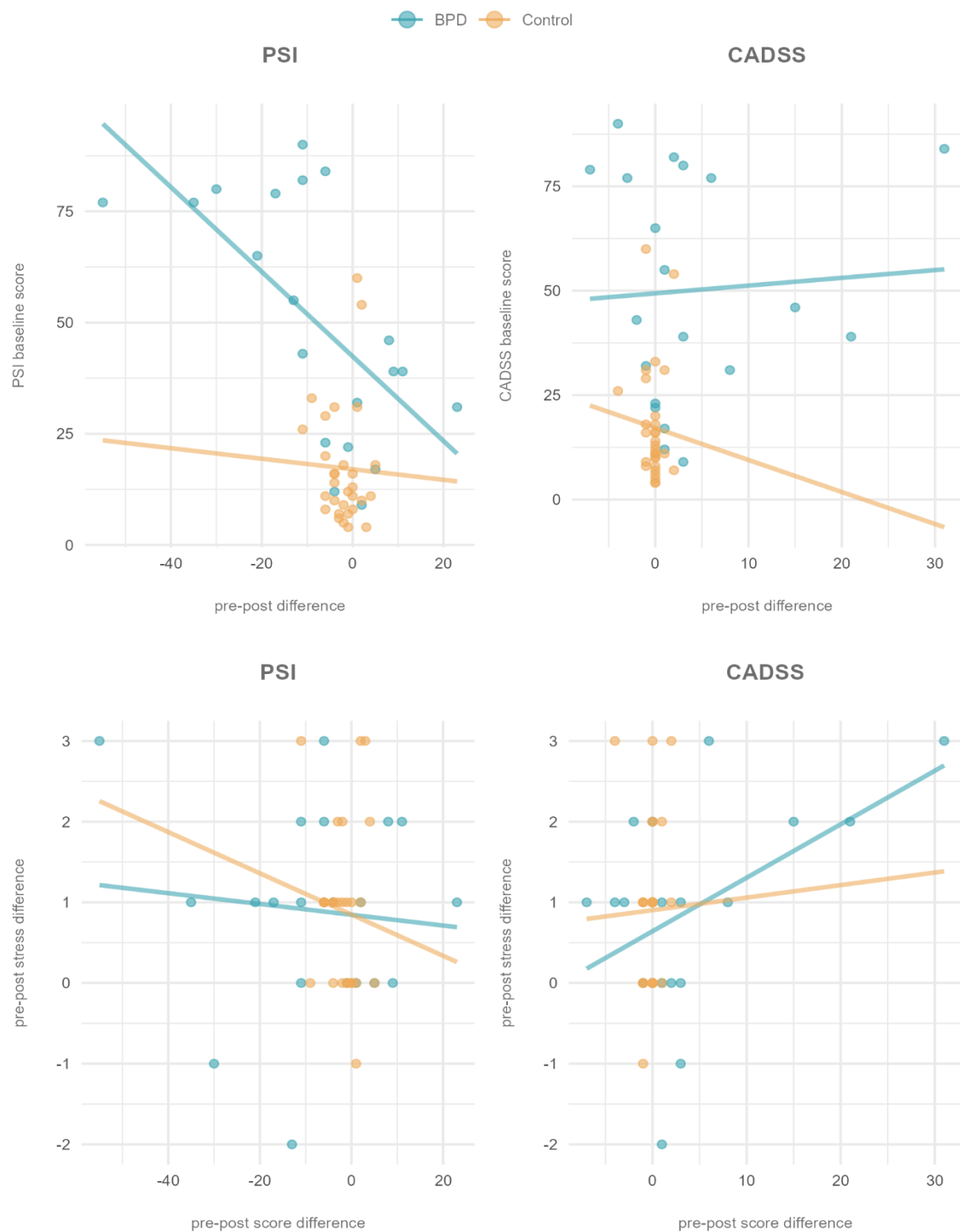


Figure 7.4

Correlations between the pre versus post stress induction difference of PSI (psychotic experiences, left) and CADSS (dissociation, right) scores, with their respective baselines (top), as well as with the pre versus post induction stress ratings (bottom). Top left: BPD $r = -0.624, p = .003$, Control $r = -0.03, p = .867$; Top right: Full group $r = -0.185, p = .198$; Bottom left: Full group $r = -0.08, p = .583$; Bottom right: BPD $r = 0.476, p = .034$, Control $r = 0.016, p = .931$.

Correlations between dissociative and psychotic state symptoms

Across both groups, PSI and CADSS scores showed a marked positive correlation at baseline (BPD: $r=0.748$, $p<.001$, Control: $r=0.783$, $p<.001$), as well as after the stress induction event (BPD: $r=0.611$, $p=.025$, Control: $r=0.774$, $p<.001$). There was no significant relationship between the pre versus post stress difference of psychotic and dissociative experiences for neither the BPD ($r=0.279$, $p=.233$), nor the control group ($r=0.380$, $p=.252$) (Figure 7.3).

Visual perception and prior knowledge

During the repeated measure of the two-tone image task, both groups gained the expected advantage (increase in d') from their exposure to the template images (paired $t_{19}=-5.84$, $p<.001$ and $t_{29}=-6.40$, $p<.001$ for BPD and control participants respectively). Equally, both groups again showed no difference in response bias (β , the bias towards reporting more dots on the figure) in response to template exposure (paired $t_{19}=-0.67$, $p=.510$ and $t_{29}=-0.89$, $p=.381$ for BPD and control participants, respectively) (Figure 7.5). There was no difference between groups for neither the increase in d' ($t_{48}=-0.41$, $p=.681$), nor the change in β ($t_{48}=-0.17$, $p=.868$) after seeing the templates.

Paired t-tests comparing task performance before and after stress induction showed no significant changes in d' difference, neither within the BPD group ($t_{19}=-0.46$, $p=.650$), nor within the control group ($t_{29}=0.36$, $p=.719$), nor between the groups ($t_{48}=0.45$, $p=.653$). Equally, there was no difference (before versus after stress) in the increase in β (before versus after template exposure), neither between groups ($t_{48}=0.36$, $p=.717$), nor within the BPD group ($t_{19}=0.92$, $p=.367$), nor the control group ($t_{29}=1.66$, $p=.107$).

On the second (repeated) measure, the control group (but not the BPD participants) had a significantly improved baseline performance ($t_{19}=1.47$, $p=.16$ and $t_{29}=2.83$, $p=.008$ for BPD and control group, respectively), compared to before the stress induction. There was no difference in baseline performance between groups ($t_{48}=-0.49$, $p=.623$).

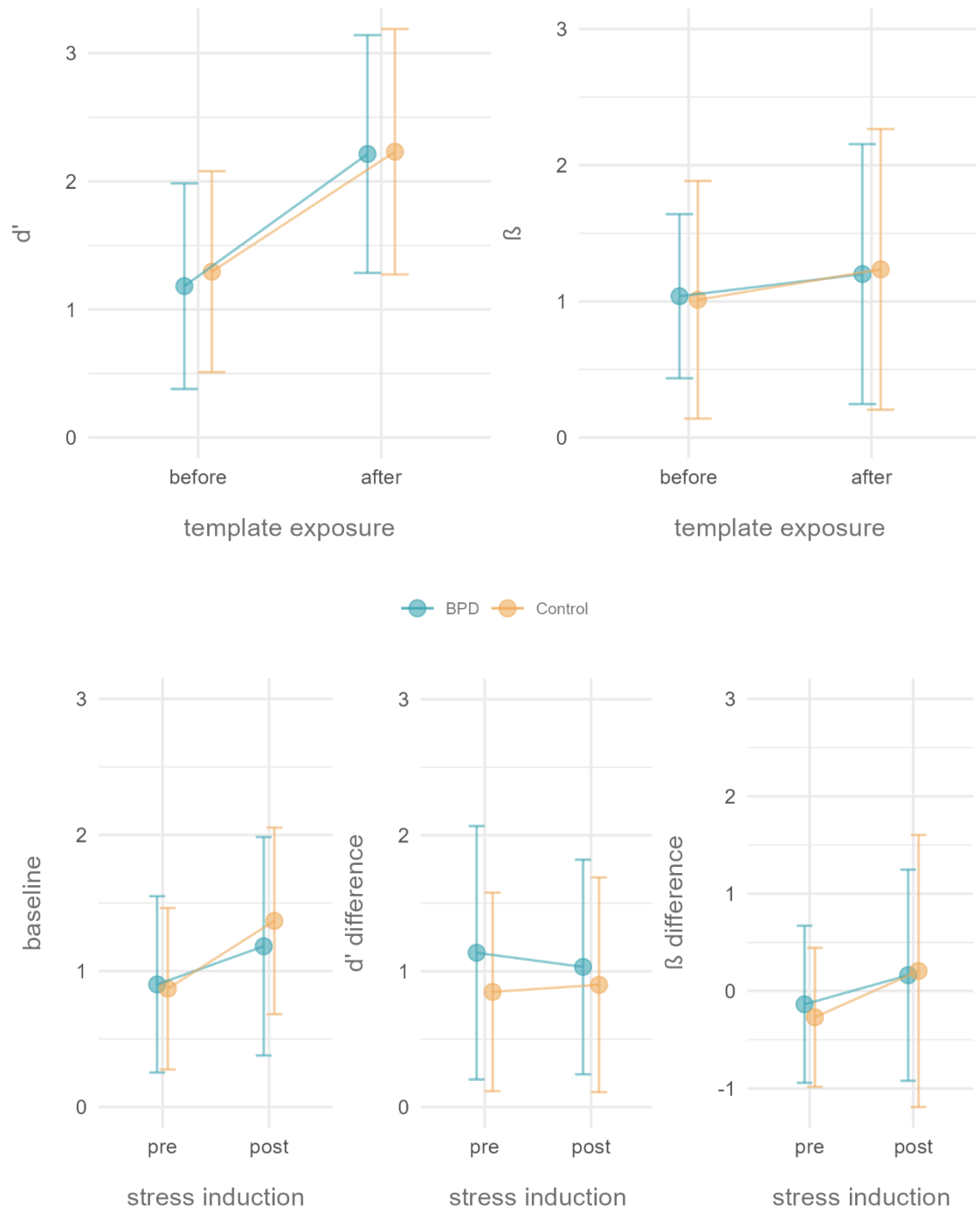


Figure 7.5

Top: Perceptual discriminability (d' , left) and response bias (β) before and after template exposure for BPD and control participants of Study 2 (at second assessment, after stress induction). Bottom: Changes in baseline performance (left), d' difference before versus after template exposure (“improvement”, centre), and β difference before versus after template exposure (“change in bias”, right) pre versus post stress induction event. To avoid overplotting, the graphs are presented offset (jittered).

Ability to utilise visual priors to discern ambiguous stimuli (as measured by the difference in d' before versus after template exposure) was not associated with the change in stress levels between checkpoint 1 and checkpoint 5, neither in the BPD ($r=0.339$, $p=.144$), nor the control group ($r=0.10$, $p=.584$). Corresponding to the two preceding analyses, no correlation between CAPS and d' difference ($r=-0.50$, $p=.729$) or PDI and d' difference ($r=-0.14$, $p=.344$) could be demonstrated. As before, one BPD group participant indicated previous exposure to the task in a different study, however removing their data did not affect results and they were included in the analysis.

7.3.3 Exploratory analysis: The role of greater overall stress

When the experiment was designed (as a before-versus-after stress comparison assessing stress reactivity and potential changes in state symptoms), the immediate (and persisting) significant difference in stress ratings between BPD and control participants had not been anticipated. The BPD group scored significantly higher (than controls) at every checkpoint leading up to the stress induction (at $p<.01$, Appendix F.1), which (considering the equally higher PSI and CADSS scores in this group) presented the intriguing opportunity to explore whether perhaps an *overall* stress level, was associated with an *overall* increased risk for these experiences.

Ratings of each of the six stress checks were aggregated to obtain a stress total (a score between 6-30), which (as expected) differed significantly between groups (Welch's $t_{30}=2.80$, $p=.009$). The pre and post stress CADSS and PSI scores were summed and divided by two, to obtain mean weighted "total" more reflective of the participants' symptom state over the course of the entire study.

For both groups, the stress total was positively correlated with psychotic experiences (mean PSI score; $r=0.634$, $p=.003$ and $r=0.422$, $p=.020$ for BPD and control group, respectively) (Figure 7.6), which in turn were associated with an increase in dissociative (state) symptoms (mean CADSS score; $r=0.716$, $p<.001$ and $r=0.782$, $p<.001$ for BPD and control group, respectively).

Following this observation (and in light of the potentially causal and/or mediating relationship between dissociation and psychosis explored in Chapter 6), it was of interest to determine whether *current* stress, and *current* dissociative states could perhaps predict *acute* psychotic experiences.

This premise was tested using multiple linear regression, controlling for participants' underlying psychosis proneness (CAPS and PDI), as well as their diagnostic status. The fitted model was:

$$\text{mean PSI score} = 4.36 + 1.36 * (\text{stress total}) + 1.19 * (\text{mean CADSS score}) + 0.25 * (\text{CAPS score}) + 0.16 * (\text{PDI score}) - 5.59 * (\text{diagnostic status=control})$$

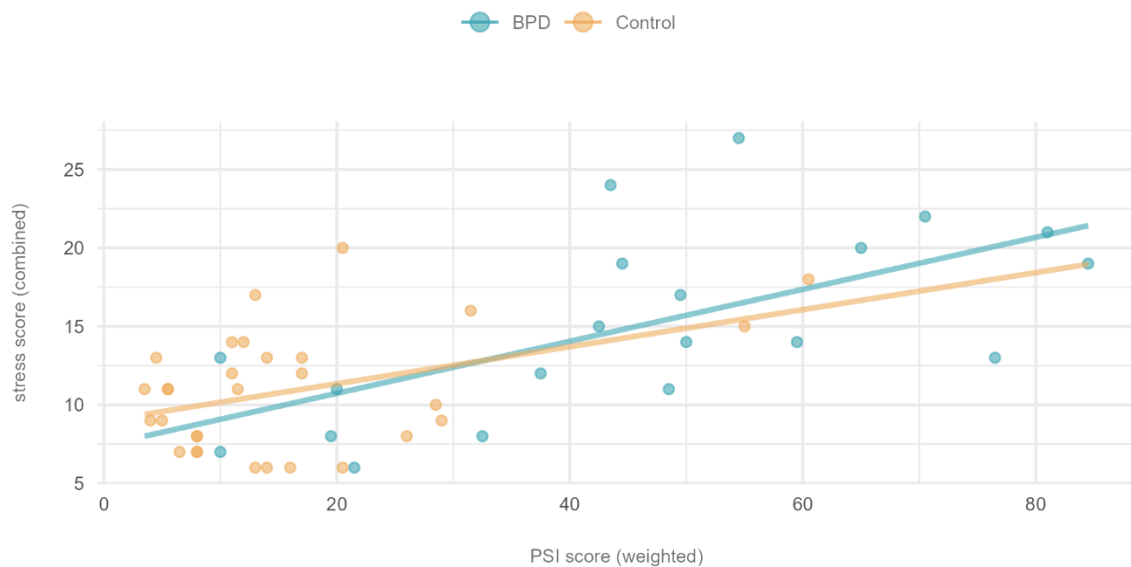


Figure 7.6

Exploratory correlation between the stress total over the course of the participation (sum of all six stress checkpoints), and the weighted overall PSI (psychotic experiences) score (sum of assessments one and two, divided by two), for BPD and control participants (BPD $r=0.634$, $p=.003$, Control $r=0.422$, $p=.020$).

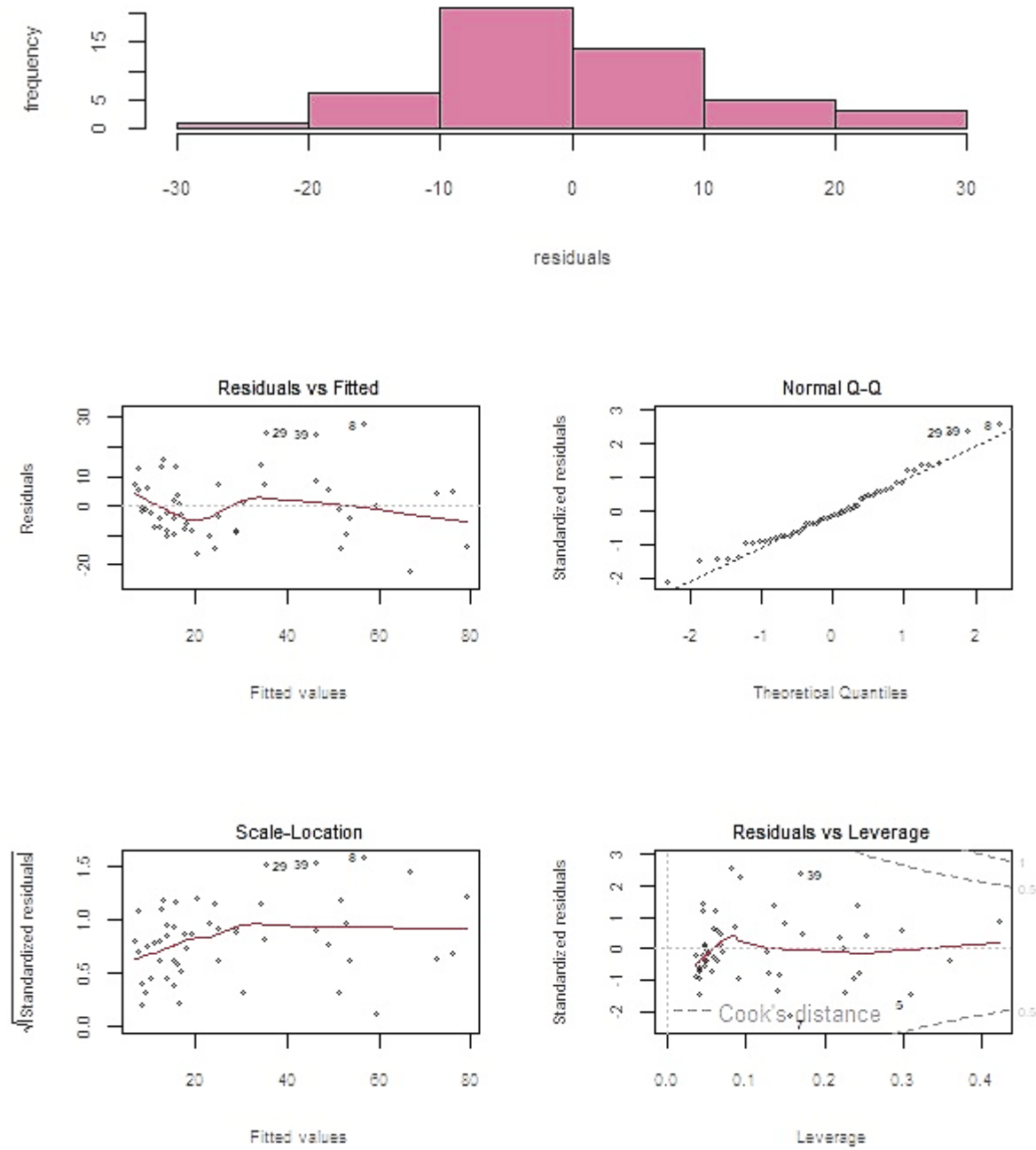


Figure 7.7

Diagnostic statistics for the linear regression model of stress total and weighted overall PSI score. Top: histogram of the model's residuals, assessing normal distribution; Centre left: residuals vs. fitted plot, assessing (non-)linear patterns; Centre right: Q-Q plot, assessing normal distribution of the model's residuals; Bottom left: scale-location plot, assessing homoscedasticity; Bottom right: Leverage plot, assessing influential datapoints (using Cook's distance)

Prior to fitting the model, mean weighted PSI scores were screened for outliers (using interquartile range method and Grubb's test), with none identified. All assumptions of regression models were met, including approximately normal distribution (Figure 7.7, top and centre right), an overall linear pattern (Figure 7.7, centre left), and reasonably consistent variance (Figure 7.7 bottom left) of the residuals. Cook's distance did not indicate any influential observations (Figure 7.7, bottom right).

The overall regression was statistically significant ($r^2=0.78$, $F_{(5,44)}=31.42$, $p<.001$), with both stress ($\beta=1.36$, $p<.001$), and current dissociative symptoms ($\beta=1.19$, $p<.001$) predicting PE scores. By contrast, neither CAPS ($\beta=0.25$, $p=.562$), nor PDI ($\beta=0.16$, $p=.798$), nor diagnostic status ($\beta=-5.59$, $p=.292$), were associated with the severity of acute psychotic states (PSI scores) experienced during the study session. While the coefficient of determination implied the model to be of adequate *explanatory* value (up to 78% of the variance in PSI scores could be explained by stress and dissociation), the prediction interval was relatively large ($S=11.33_{(44)}$), therefore offering only limited *predictive* value.

7.4 Discussion

With introduction of the ICD-11, transient, stress-related dissociative and psychotic experiences became part of the diagnostic criteria for determining personality disorder severity⁴⁶ – reiterated under the borderline pattern identifier as “transient dissociative symptoms or psychotic-like features (e.g., brief hallucinations, paranoia) in situations of high affective arousal” (First et al., 2015). Considering the central (and exclusive) role stress has thus been assigned in the aetiology of these experiences, one might expect this to be backed by scores of empirical research. Instead, the literature on the subject may at best be described as a work in progress. The aim of the current study was therefore to assess whether (and to what extent) acute stress may influence psychotic experiences, and how dissociative states might further affect the relationship.

⁴⁶ “Under stress, there are extreme distortions in the individual's situational and interpersonal appraisals [...], dissociative states or psychotic-like beliefs or perceptions (e.g., extreme paranoid reactions).”, criterium for moderate and severe personality disorder, ICD-11 (World Health Organization, 2019).

Following the stress induction, average stress ratings increased for both BPD and control participants. While the rate of increase (i.e., the “stress-reactivity”) did not differ between groups, BPD participants reported significantly higher baseline ratings which remained elevated throughout the course of the study. Similarly, baseline levels of psychotic (PSI) and dissociative (CADSS) state symptoms were also increased in BPD participants. While differences in PSI scores (pre stress) retained statistical significance after correcting for people’s ‘trait’ psychotic symptoms (i.e., CAPS and PDI scores), differences in CADSS scores were no longer relevant after correcting for the effect of participants’ ‘trait’ dissociation (DEMO scores). Both PSI and CADSS symptoms were unaffected by the stress induction in the control group, while BPD participants showed a trend towards a more pronounced (compared to controls, $p=.058$) increase in dissociation ($p=.067$), as well as a decrease in psychotic ($p=.056$) experiences. For the PSI, there was no correlation between stress-reactivity and changes in PE scores pre versus post stress. However, in the BPD group, baseline PSI total and pre versus post PSI difference were negatively correlated. By contrast, the difference in CADSS scores (pre versus post stress) correlated with the difference in stress ratings (for BPD but not control participants), but not with the severity of dissociative symptoms at baseline. Strong positive associations between PSI and CADSS scores (though not between their pre versus post stress differences) were observed in both groups. Furthermore, participants’ stress total (sum of all stress ratings) was associated with their overall (mean-weighted) PSI scores and an exploratory linear regression model suggested that both, people’s overall (subjective) level of stress, as well as their dissociative symptom severity significantly affected psychotic experience scores over the course of the current study.

Impact of stress on two-tone image task performance

Participants’ ability to utilise visual priors to discern ambiguous stimuli (two-tone image task) was unaffected by the stress induction and there were no differences between groups, neither for the rate of improvement post template exposure (d' difference), nor for the response bias (β). This corresponds to task performance before stress induction (Chapter 6.3.1), as well as to the two-tone task results of Study 1 (Chapter 6.2.1), and further supports the conclusion that there are no discernible differences in the balance between

prior knowledge and incoming sensory stimuli between people with and without BPD. Notably, both groups' baseline scores improved (non-significantly for BPD and significantly for control participants, with no statistical difference between the groups) on the second exposure to the task – despite being presented with a novel set of stimuli (i.e., none of the two-tone images from the pre stress task were presented post stress and vice versa). This raises the question whether (and to what extent) improved discriminability may be due not just to template exposure, but also perhaps (in part) to acutely sharpened attentional mechanisms, (prolonged) learning effects, or increased pattern awareness. While not necessarily upsetting within-study analysis and reliability of the data (at least in the current context, since both participant groups appear to have been equally affected), this may be an important consideration for future studies, especially if participants are (not) known to have completed the task before, or if recruiting from volunteer catchment areas in which the task has previously been presented. Additionally, considering the relatively short amount of time between the first and second two-tone task in this study, it may be of interest to investigate whether length of the test-retest interval might offset the observed effect.

Impact of stress on dissociative state symptoms

As expected, dissociative experience scores of BPD (but not of control) participants increased in response to the stress induction, and the rate of increase correlated (moderately) with the self-reported change in stress levels. On the verge (but falling short) of statistical significance ($p=.067$), these findings are in line with a series of previous studies (Stiglmayer et al., 2001, Stiglmayer et al., 2008, Graumann et al., 2023) that have found a link between stress and dissociation frequency (or severity) in people with borderline pathology. Current dissociative symptoms were substantially predicted by individuals' DEMO scores, and the differences in CADSS rating initially observed between BPD and control participants lost significance when correcting for this 'trait' dissociation. This (reassuringly) suggests that at any given time the risk for acute dissociative symptoms appears first and foremost driven by a general dissociation proneness. Interestingly, neither DEMO nor CADSS baseline scores were associated with the changes in dissociation in response to the stress induction – however, considering the

non-significance of this change and the comparatively small sample size it is doubtful whether any useful conclusions can be drawn from this observation.

Impact of stress on psychotic state symptoms

Unexpectedly, BPD participants' PSI scores decreased after the stress induction event, with the difference again marginally non-significant ($p=.056$). While this appears to contradict findings by Glaser et al. (2010), who reported an increase in psychotic experiences (especially paranoia and hallucinations) in relation to daily life stress, there are several notable differences concerning experiment design and methodology that render these opposing results not necessarily mutually exclusive. Firstly, Glaser et al employed an experience sampling method, prompting participants to fill in a questionnaire ten times a day for a period of six days and measured their response to naturally stressful events. Secondly, they assessed psychotic symptoms with a brief rating scale of eight items generally associated with psychotic experiences, while in the current study, participants completed a comprehensive, forty-eight item questionnaire specifically designed for testing experimentally manipulated mental states. Perhaps most importantly, Study 2 used a single, manufactured stress induction to compare immediate changes in symptoms before versus after the manipulation, which is arguably very different from real-life (psychologically and/or physiologically) stressors. By contrast, Glaser et al. (2010) investigated the relationship between the overall amount and severity of stressful events, and the total number of psychotic symptoms reported – not their immediate increase or decrease. This in turn is very much in line with the exploratory analysis of participants' stress-total and averaged PSI scores, which showed a significant positive correlation.

Consequently, the unforeseen decrease in psychotic state symptoms remains an intriguing curiosity. Of course, the limited sample size may have biased the results. Furthermore, the PSI was originally designed to assess experiences before, during, and after a laboratory-controlled, ketamine-induced psychotic state. As such, the phrasing of some of the questions may be more applicable in their intended context and might have been confusing – for example item 6 “*You feel close to people*” may not have seemed particularly relevant in the sole company of the researcher conducting the experiment. However, this limitation

should affect all participants equally, and would be applicable both before, and after stress. Notably, mean PSI scores (pre and post stress) of the control group were in line with findings from the instrument's original placebo group ($n=16$, mean 19.2, SD 10.7), as well as with the ketamine ($n=16$, mean 17.2, SD 10.3) and Cannabis ($n=138$ mean 17.0, SD 11.0) groups before drug exposure (Mason et al., 2008). Overall, this supports the assumption that the measure itself served its purpose as intended. Remarkably, BPD participants did not only score significantly higher than the control and placebo groups, but also than the ketamine ($n=16$, mean 24.0, SD 10.9) and cannabis (mean 33.3, SD 19.5) groups *after* infusion (Mason et al., 2008). This begs the question whether the association between stress and psychotic experiences is perhaps not entirely linear and instead plateaus or perhaps even subsides again (irrespective of sustained or further increasing stress levels), either as a temporally limited symptom or due to external factors. While of course entirely speculative, this hypothesis may receive preliminary support from the fact that the BPD group began the study at a stress level comparable to the control group's post-induction average, and that baseline psychotic experiences showed a moderate, negative correlation with the PSI score pre versus post stress difference (in the BPD group). In other words, the lower participants' original PSI score, the stronger the symptom increase in response to the stress induction. Additionally, dissociative experiences (which increased in the BPD group in response to the stress induction), though likely to be a significant risk factor in the aetiology of psychosis in BPD, could perhaps acutely confer a (partial) protective advantage against more severe psychotic symptoms by cognitively and emotionally removing the person from their current experience.

Neither CAPS nor PDI were significantly related to PSI scores (neither before, nor after stress, nor to the pre versus post stress difference). This means that unlike the dissociative state symptoms (which were largely predicted by participants underlying trait dissociation), the likelihood of displaying current psychotic experiences was not necessarily influenced by a person's general psychotic symptom frequency or severity. This may in part be because the CAPS and PDI each only measure a very specific subset of psychotic experiences (perceptual symptoms and delusional ideations, respectively), whereas the PSI aims to capture both these categories and additionally includes items

pertaining to some of the negative symptoms of psychosis⁴⁷. As such, their correlational and predictive value is inherently limited.

An explorative multiple linear regression model did equally not show an association between CAPS or PDI, and the psychotic experiences reported during the study. Curiously however, it did suggest that stress total and dissociative state symptoms more significantly predicted PSI scores than diagnostic status. In the overall aetiological and mechanistic puzzle of psychosis and borderline pathology, this would once again support the idea that in BPD such symptoms could be on the extreme end of a spectrum that shares one or more of its causalities with similar experiences in the general population.

Thoughts on group differences at baseline and unexpected mental states

Starting with checkpoint one, BPD and control group participants differed significantly on their subjective stress ratings from the very beginning of the study. While originally unexpected, in retrospect this observation may simply reflect the higher rates of anxiety (Zimmerman and Mattia, 1999), rumination and post-traumatic stress (Dell’Osso et al., 2019), pain (Sansone and Sansone, 2012; Johnson et al., 2020), and a myriad of other psychological and physiological difficulties associated with borderline pathology and accompanied by chronically heightened feelings of stress and agitation. In the context of the current study, the finding was inconsequential and even beneficial to the research question and its analysis. However, it does mean that there may be further pervasive but fundamental differences in the (current) mental and emotional state of participants, subtly influencing psychological research results. Another pertinent example is the astonishing and unexplained drop in the BPD group’s sense of “*feeling in-control*”, the detailed discussion of which would severely depart from the scope of this dissertation as it appeared wholly unrelated to psychosis in BPD – and psychosis in general. However, perhaps future studies in people with (borderline) personality disorder may wish to consider including similar, brief assessments to perchance replicate (or even explain) this curious finding.

⁴⁷ Despite this thesis being primarily concerned with positive psychotic experiences, PSI scores were calculated including the negative symptom categories (specifically, “anhedonia” and “cognitive disorganisation”) to enable comparison with previously published scores.

Chapter 8 Onward and upward - general discussion and future directions

“I still have voices and visions, but I am comfortable with them now. I have an interpretation of them which allows me to live at peace with them. They are mine, they are me, and they come to help me, not to do me harm. I consider it a privilege to be able to hear and see information directly from my subconscious and the collective unconscious, which is hidden from most people. It no longer distresses me. I think of it as a gift.”

When one of the experts-by-experience involved in the design and Patient and Public Involvement (PPI) review of this dissertation’s studies first came to the attention of the mental health care system during her teens, her symptoms included hearing voices, frequent visual distortions and hallucinations, and a series of firmly held delusional beliefs ranging from mundane to fantastical. Without being told an official diagnosis, she recalls undergoing several years of counselling, cognitive behavioural therapy (CBT), and (ineffective) anti-psychotic medication, with health care professionals ubiquitously focused on her perceptual symptoms and largely disregarded her deteriorating mood, anxiety, and interpersonal difficulties. She also recalls how, when almost a decade later she was diagnosed with BPD, the tables turned practically overnight. Her psychotic experiences were no longer deemed real or relevant, an “exaggeration”, and “call for attention”. Today, she rarely mentions them – not to her GP, in counselling sessions or assessments, during emergencies or mental health crisis calls, not even to family and friends – for fear of being either called a liar or referred for yet another psychotic disorder assessment. For quite some time, she was convinced that these experiences must be a sign of schizophrenia, because nothing else seemed to explain her persistent paranoia and

auditory-verbal hallucinations, until learning that psychotic symptoms can also affect (some) people with borderline personality disorder.

While of course solely anecdotal within the scope of this thesis and beyond, the above experience echoes countless other accounts of people with BPD and psychosis as well as reflecting a growing body of research that has begun to uncover our limited understanding of such symptoms (for a recent review see Minarikova et al., 2022), the persisting ambivalence and prejudice against those reporting them (Adam and Sanders, 2011), and the devastating personal and clinical implications that ensue (Kelleher et al., 2017; Slotema et al., 2017; Cavelti et al., 2019; Furnes et al., 2019).

In view of this gaping abyss in knowledge regarding the nature (and especially the mechanisms) of such experiences, the primary aim of this dissertation was to ascertain the range and types of psychotic symptoms experienced by people with BPD, and to investigate potential risk factors that may be implicated in their prevalence, by considering some of the current (common mental distress-, trauma and dissociation-, as well as predictive processing) models of psychosis.

Extending a recently expanding list of similar findings (Pearse et al., 2014; Niemantsverdriet et al., 2017; Slotema et al., 2019), the results provide compelling evidence that a wide range of psychotic experiences (including perceptual abnormalities and delusional thoughts) are common in people with BPD, and at a level of clinical significance comparable to psychotic disorders. They further challenge wide-spread terminologies such as “psychosis-like” which have previously been called into question by others (Gras et al., 2014; D’Agostino et al., 2019). Moreover, this thesis offers a comprehensive assessment of potential aetiological and cognitive risk factors for PE development, identifying dissociative experiences (and to a lesser, limited extent trauma) as the strongest predictors for psychosis proneness, while suggesting a correlational (rather than causal) relationship with common mental distress and borderline symptom severity. Stress was found to have a potentially triggering or modulating role; however, this analysis was chiefly explorative. Finally, this dissertation marks the first consideration of BPD (and psychosis) within a predictive processing framework. Results consistently indicated that

basic sensory prediction mechanisms appear intact in this population, which suggests that the conceptualisation of hallucinations and delusions as the result of aberrant prediction error signalling may not be applicable in the context of borderline pathology.

8.1 Psychosis in BPD - A sum(mary) of many things

In parallel with the planning, research, and writing of this dissertation, the diagnostic construct of (borderline) personality disorder underwent an unprecedented reconceptualization: From a set of related but dichotomous categories to a symptom severity spectrum of (up to five) disordered traits (World Health Organization, 2019). Such a fundamental and momentous change may risk casting a doubt on the long-term relevance and future applicability of a thesis primarily concerned with one specific symptom feature of a soon-to-be archaic (in the UK) medical concept.

However, despite this theoretical (and impeding practical) overhaul, the ghost of PD-past is unlikely to be vacating research and clinical settings just yet. The retention of a borderline pattern identifier – undeterred by little empirical evidence for a unified BPD syndrome, and despite causing an apparent disintegration of the entire ICD-11 trait domain structure (Tyrer et al., 2019) – bears testament to the concept’s continued influence. This choice was possibly guided by the American Psychiatric Association’s 2013 decision (unchanged in the recent 2022 revision) to uphold the categorical classification of PDs (including BPD) in the DSM-V (American Psychiatric Association, 2013), after essentially rejecting a (more ICD-11 comparable) continuum approach as insufficiently evidenced and too complex for clinical practice (Zachar et al., 2016). In this regard, the continued use of a borderline identifier enables a higher degree of ongoing congruity and transferability between the two most frequently utilised (psychiatric) diagnostic standards in the (western) academic world. More significantly however, the term BPD is associated with considerable intellectual, societal, and personal implications, not to mention over half a century’s worth of research and millions of people currently living and/or identifying with the label – all of which may not be as straight-forward to supplant. Therefore, whether welcoming or scrutinising the recent developments, one cannot deny the lasting impact that past and

present studies on borderline pathology are destined to have on our understanding of this set of psychosocial difficulties – no matter its name or classification.

8.1.1 Methodological considerations

The past decade has seen a fundamental shift in the way psychological research is conducted, moving away from pen-and-paper assessments towards increasingly digitalised forms of data collection as well as (more recently, and exacerbated by the circumstances surrounding COVID-19) studies performed entirely online (Gillan and Daw, 2016). Beyond basic online surveys, a rising number of fully tooled behavioural experiment builders (e.g. Gorilla, developed by Anwyl-Irvine et al., 2020 and used in the context of this dissertation) now offer the chance to design complex study procedures that can accommodate a vast variety of questionnaires and cognitive tasks, and permit precise programming of study-flows, timings, and even rest breaks. This essentially allows researchers to curate and control remote experiments which closely resemble the experience of an in-person study session, with the literature supporting the (online) replicability of both task and questionnaire data collected under laboratory conditions (Crump et al., 2013; Gillan et al., 2015). The replication of the first study's findings by the subsequent in-person experiment (Study 2) may perhaps be regarded as further evidence of the fundamental suitability of virtual assessments.

Even though the online study in this dissertation was primarily motivated (and necessitated) by pandemic-related research restrictions, there might in fact be concrete benefits to assessing participants virtually, from the comfort of their own home. First and foremost, it can help widen the logistical bottleneck of convenience sampling, which typically sees large groups of people prevented from taking part in laboratory-based studies due to full-time work, geographical distance, and lack of transportation, or sometimes simply because the time and energy required for travel, necessary social interaction, and experiment completion does not appear worth the effort (or amount of compensation). By contrast, (single session) online studies can be completed at participants' own discretion and do not involve any commitment or considerations beyond the actual experiment procedure, thus potentially engaging and giving access to a broader and more diverse

sample population. At the same time, this may of course restrict involvement of other minority demographics including those without access to (or fundamental knowledge of) technology.

In addition to such generalisable benefits and disadvantages of online versus in-person testing, it is important to recognise how this may be amplified (or differ) in clinical (and in this context, borderline PD) populations. The drop-out rate for the BPD group was considerably higher for the lab-based study, with 56% (24/44) cancelling or not attending their scheduled appointment, compared to just 33% (26/80) non-completion for the online study. By contrast, control group drop-out rates were stable across both studies with 24% (23/94) non-completion online and 23% (9/39) non-attendance in-person. Unpredictable, frequent changes in mood and emotional well-being, as well as physical comorbidities (Frankenburg and Zanarini, 2006; Dokucu and Cloninger, 2019) may be preventing people from attending sessions that they had to arranged weeks in advance, possibly during a time of better health. Feelings of guilt and shame, prevalently associated with borderline pathology (Buchman-Wildbaum et al., 2021; Spitzer et al., 2021) could further hinder rescheduling, as participants might seek to avoid confrontation with the research team. As such, the flexible, self-directed schedule of online assessments can be more accommodating to fluctuations in daily functioning, as well as circumventing the emotional burden of stressful and potentially uncomfortable social interactions. In this regard, the BPD group's high stress ratings in Study 2 are particularly intriguing: Do they simply capture the chronically enhanced levels of stress and anxiety one might expect from borderline pathology? Or do they perhaps reflect BPD participants' response to the unfamiliar testing environment? While the significant (unexpected and unprompted) decrease in their perceived sense of control shortly after study begin certainly points towards an unknown psychological or environmental variable, these ratings were unfortunately not collected during Study 1, making direct comparison (and by extension any inference on what role the testing environment may have played) impossible.

On the other hand, the tangible, physical reality of an in-person study session could provide a much more grounded experience, with the presence of the experimenter offering additional validation and reassurance. However, the researcher-researchee relationship (so

far predominantly evaluated in the context of qualitative studies (e.g., Råheim et al., 2016) is exceptionally complex and appears to mainly revolve around conscious and subconscious power-dynamics (Dennis, 2014), and trust (Guillemin et al., 2018). Considering the central role (impairment of) the latter plays in BPD pathology, further explorations of this relationship may be of interest to better inform future research approaches and study designs.

Another notable limitation of both studies stems from their length, complexity, and the (at times) substantial cognitive and emotional investment they required, all of which may have given rise to an increasing sense of cognitive fatigue that could have affected certain tests (or certain people) disproportionately. Environmentally, this effect would likely be exacerbated in an online study, during which screen tiredness and lack of sensory stimuli can further decrease concentration and attention. Moreover, the severity of such fatigue would be subject to differences in technological set-up (e.g., screen size, luminosity, and lighting; Lee et al., 2021), and while device type and operating system were recorded (and assessed) as potential confounding factors, it was of course impossible to evaluate individuals' hardware. Similarly, the order of tasks and questionnaires could not be counter-balanced between participants⁴⁸. As such, cognitive assessments towards the end of a study (e.g., the two-tone image task; see Appendix C for the order of tasks and questionnaires) would have been impacted more severely. Yet, the fact that the second assessment of the two-tone images yielded the same results as the pre stress measure (from the beginning of the experiment procedure), and that both of these also replicated the online study's findings suggests that any occurring cognitive fatigue did not significantly affect (two-tone image task) data obtained towards the end of the session.

Cognitive exhaustion might additionally be experienced more quickly or more severely by people with attention deficit disorder (ADD) or traits, which have long been associated with mental (and chronic) fatigue (Byun et al., 2013; Rogers et al., 2017; Yamamoto, 2022). While it is doubtful that comorbid ADD diagnoses affected results due to their low

⁴⁸ In Study 1, this was largely due to technical limitations of the research platform, whilst in Study 2, the stress induction paradigm necessitated a strict assessment order to ensure that each task or questionnaire was completed at the same "level" of stress across all participants.

prevalence⁴⁹, the significant phenotypic (Weiner et al., 2019) and genotypic (Distel et al., 2011) overlap between attention deficit traits and borderline pathology raises the question whether such concerns may be pertinent to the overall BPD population. Unfortunately, neither study included a dedicated assessment of ADD symptomology, which would have helped to determine whether and how the prevalence of such traits might have influenced the (later) cognitive tests. Future studies may wish to consider the potential impact of ADD traits when designing (online) experiment procedures for participants with borderline PD.

Limitations specific to Study 1

Evidently, any (online) study using primarily self-report assessments of psychiatric (and related) symptoms is affected by “noise”, ranging from differences in the interpretation of questions, over technical difficulties, to an overall decreased level of standardisation of (and control over) individuals’ current physiological or mental state, and study environment. Most crucially perhaps, this includes the (COVID-19 necessitated) assigning of participants to the BPD group based on their self-reported diagnostic status, which comprised people who reported having a current diagnosis of borderline PD – as well as people who reported not having an official diagnosis but identifying with the medical construct (in conjunction with the MSI cut-off).

Such use of self-reported (not to mention self-identified) diagnoses undoubtably raises questions about the suitability and reliability of the sample population, especially in a disorder as heterogeneous as BPD. Naturally, a quick online search and a handful of questionnaires cannot and should not replace years of clinical expertise and hours of diagnostic interviews. While valid, such concerns may fail to consider the lived reality of (and manifold reasons for) self-diagnosis, which often involves years of illness, questioning, and searching, rather than simply a brief excursion on the internet. Some participants may not be able to obtain a diagnosis for financial reasons, especially in countries where (mental) health care is privatised. Others may have (repeatedly) been advised that they might suffer from BPD by people with substantial (clinical) experience but without the administrative power to offer a medical diagnosis (e.g., youth councillors

⁴⁹ Study 1: 4/124, including three BPD participants and one control; Study 2: one BPD participant (1/49).

or mental health nurses) and may currently be awaiting official assessment, facing years-long waiting lists, and a daunting number of referrals. This appears to have been the case for at least one participant, who contacted the research team a year after completing Study 1 to say that she took part as “self-identified” but had since been given a clinical diagnosis of EUPD and wanted to relay this information in case it impacted the results. Considering the high rates of misdiagnosis afflicting borderline pathology (Ruggero et al., 2010; Porr, 2017; Beatson et al., 2019), as well as its clinical under-representation in non-female, non-white populations, studies involving self-reported diagnoses may perhaps even enable a more accurate picture of the full range of affected individuals by avoiding the narrow and structurally biased lens of Western psychiatry.

Whilst the benefits and limitations of the different diagnostic and assessment tools for BPD/EUPD have been reviewed elsewhere (Thornton et al., 2023), so far there have been no dedicated investigations of self-diagnosis (or its research implications) in the context of borderline pathology. Fortuitously, an increasing body of literature is inspecting the rising trend towards (online) self-diagnosing for other psychiatric disorders and beyond (Hynes, 2013; Farnood et al., 2020). Notably, a recent cross-sectional study on self-diagnosis in common mental health disorders (Rutter et al., 2023) found that people who identify with a mental health condition (specifically anxiety, depression, mania, and alcohol use disorder) experience a degree of psychopathology comparable to those with an official diagnosis. Although more common and largely symptom-based labels such as anxiety and depression are arguably easier to identify (and self-identify with) than an abstract construct such as “personality disorder”, the significant overlap between the results of Study 1 and Study 2 (which only included participants with a verified clinical diagnosis), permeating virtually all aspects of the populations’ psychopathology, suggests that self-report may indeed be a viable assessment option for BPD research (current and future).

Limitations specific to Study 2

In contrast to the larger, but less regulated online assessment of Study 1, the second study enjoyed a much more controlled and detailed assessment, but was restricted in (BPD) sample size, with a high drop-out rate between screening and study participation. This

could have possibly in been a lingering effect of the pandemic, which likely affected people with BPD (many of whom reported physical comorbidities and may be clinically vulnerable) disproportionately. While the decreased of statistical power undoubtedly affected the significance of some of the results, the tangible overlap between virtually all task and questionnaire scores in the BPD groups of Study 1 and Study 2 strongly suggests, that it did not markedly alter the overall findings. Nevertheless, a more detailed assessment of borderline pathology (unfortunately the BSL was not included in the online study) across both populations would have allowed for a better comparison of disorder severity and how it may have related to the different factors examined. Similarly, further research extending the sample size of Study 2 may help to reaffirm the significance of the current findings and increase their level of generalisability.

The substitution of several of Study 1's instruments during the design of Study 2 was predominantly motivated by the desire for more detailed (STAI instead of GAD, BDI instead of the QIDS) and more up to date (DEMO instead of DES) measures. This exchange was possible since the primary aim was to assess the severity of the latent constructs of *Depression*, *Anxiety*, and *Dissociation* between BPD and control participants, rather than specifically comparing them between studies. Still, such direct contrast could have been a worthwhile continuation of the investigation of parallels between the international online BPD sample, and the Cambridgeshire-drawn clinical population, and it would have been interesting to ascertain if their near complete score overlap (observed across CTQ⁵⁰, PCL, CAPS, and PDI) might have extended to these measures. It is further possible, that the change in instruments had an unknown effect on the best subset selection, although considering that the resulting regression models (of the predictors for CAPS and PDI) were again virtually identical between studies this is unlikely.

Owing to these methodological differences (as well as to the studies' considerable structural and populational deviations) the data from Study 1 and Study 2 were analysed separately and in comparison. This additionally permitted an assessment of result replicability across multiple BPD samples, which was especially appropriate considering

⁵⁰ With exception of the physical neglect subscale.

the prevailing dissensus and lack of replicability among studies on psychosis in BPD. Since no substantial differences between the cohorts could be ascertained (neither for the psychotic experience measures, nor for any other aspect of psychopathology) the two datasets were subsequently merged to enable an explorative structural equation model of the hypothesised risk factors.

Combining participant populations has long been a viable option for increasing sample size and (by extension) statistical power (see Alhudiri et al., 2023 for a recent example using longitudinal COVID data) and is typically acceptable if the cohorts share the same population characteristics but can be assumed to be independent. Interestingly, in a comparison of data-merging and meta-analysis as alternative approaches for integrative investigation (in the context of gene expression), Taminiau et al. (2014) discovered that the combined dataset identified significantly more correct target genes (while also recognising all targets of the meta-analysis), suggesting that it may be possible to draw more generalisable and reliable conclusions with this method. While it is unknown whether or to what extent participant populations may have overlapped, automated IP-address comparison during the screening surveys, as well as participants' responses to the question *"Did you ever complete this task as part of a previous study?"* in the second study's two-tone image task both strongly suggested that the cohorts were independent⁵¹.

An explorative principal component analysis did not identify a suitable number of factor loadings; therefore, to mitigate the potential effects of differences in instruments and scoring ranges, CFA and SEM latent variables were defined using standardised z-scores of the relevant symptom measures. Measurement indices of the confirmatory factor analysis, and the structural model indicated adequate goodness of fit, suggesting that the hypothesised aetiological framework appears principally sound. Nevertheless, due to a (for latent factor models) comparatively small sample size, the SEM analysis and its results should be regarded as explorative, preliminary, and requiring confirmation from a study with more, and more adequately suited observations.

⁵¹ Participants that previously completed the online experiment would have been expected to reply "Yes", as the two-tone image task was part of both studies.

8.1.2 Prevalence and range

Across both studies and all three PE measures (CAPE, CAPS, and PDI) BPD participants scored consistently higher than controls regarding the number of symptoms experienced, the frequency at which they occur, and their associated distress. A previously suggested clinical cut-off for the CAPE (>1.47 , Bukenaite et al., 2017) indicated that 65.9% of BPD participants report a level of symptom severity comparable to in-patients with schizophrenia. These results provide further evidence that psychotic experiences in BPD are common, long-standing, and associated with significant distress, thereby extending a recently expanding list of studies with similar findings (Niemantsverdriet et al., 2017; Slotema et al., 2017; Slotema et al., 2019). They also strongly reject the assumption (and prejudice) that these symptoms are merely “psychosis-like”, an undefined, unnecessary, denigratory (and evidently unsuitable) label that is frequently employed by researchers and clinicians alike and will soon (with introduction of the ICD-11) become part of the official diagnostic criteria for the PD borderline trait identifier, thereby subtly but fundamentally shaping how such experiences should be regarded.

Strikingly, there were no significant differences for neither CAPS, nor PDI scores between the BPD participants of both studies (including total score, subscales, and prevalence rates of all symptom categories), however, previous findings were only partially replicated. Rates of auditory verbal hallucinations (39-55%) are notably above those reported by Niemantsverdriet et al. (2017) (21%), Slotema et al. (2017) (30%), and Yee et al. (2005) (29.2%). Auditory (non-verbal) hallucination prevalence (45-56%) on the other hand, was in line with findings by Kingdon et al. (2010) (49.5%), Pearse et al. (2014) (50%), and Slotema et al. (2019) (40%) and appears to be a good estimate of the true incidence-rate of these experiences. However, most of these studies simply report the category “auditory” without specifying whether this constitutes hearing voices, non-verbal auditory hallucinations, or both. The frequency of visual hallucinations was equally difficult to compare to previous reports, many of which describe “perceptual alterations” (which may also include distortions). However, current findings (35-50%) appear to approximately reflect results reported by Pearse et al. (2014) (30%), Slotema et al. (2019) (32%), and

Schroeder et al. (2018) (36%), while significantly above the estimates of Niemantsverdriet et al. (2017) (11%).

As one of the first dedicated and detailed examinations of delusional experiences in BPD, the observed prevalence rates – ranging from 45% (lowest symptom category rating, *paranormal beliefs*), to 89% (highest symptom category rating, *suspiciousness*) – strongly contradict the prevailing notion that delusional experiences are less common or less severe than perceptual symptoms. This was further emphasised by the PDI's clinical significance threshold (>8 , Preti et al., 2007), which indicated that 55-59% of BPD participants reached a PDI total typically expected to identify people with psychotic disorder. Surprisingly, BPD participants in both groups scored higher than the in-patients with delusion who took part in the instrument's validation study (Peters et al., 2004). However, it should be noted that this original sample was relatively small ($n=33$), included a range of diagnoses (schizophrenia, paranoid schizophrenia, bipolar disorder, schizoaffective disorder, and manic episode), and all participants were receiving antipsychotic treatment at the time of testing.

In the context of delusions, the items most frequently endorsed by people with BPD centred around a theme of paranoia (*persecution*, *paranoia*, and *suspiciousness*) which is in line with the expectations of (and diagnostic criteria for) borderline pathology. Curiously, when assessing differences in the endorsement frequency of CAPE items between people with BPD and high- versus low PE scores, symptoms of paranoid ideation were equally endorsed by both groups whereas all other psychotic experience categories were significantly more endorsed by the high-PE group – whilst the low-PE group did not differ from the control participants. This suggests a possible latent dimension of distrust and suspicion that underlies (psychotic) experiences in BPD across its full spectrum of severity. It also raises the question whether different symptoms may have different causal or mechanistic pathways, with paranoid experiences perhaps more closely linked to shared borderline features, emotionality, and common mental distress, while other types of unusual beliefs and perceptual symptoms could have additional cognitive or aetiological risk factors (possibly shared by people with a primary diagnosis of psychotic illness). Bentall et al. (2009) found an independent and combined effect of pessimistic thinking

style and impaired cognitive performance (especially the tendency to jump to conclusions) on paranoid delusions. A recent study on empathy, emotion recognition, and paranoia in the general population (Beals et al., 2022) reported an association between the PDI paranoia subscale and imaginative perspective taking (i.e., “fantasy-proneness”), but observed no link to empathy or emotion recognition, while Combs et al. (2006) found that increased levels of paranoia were linked to decreased emotion perception. Interestingly, a 2021 transdiagnostic study on the involvement of amygdala hyperconnectivity in paranoid states reported an increased functional connectivity between the right amygdala and the prefrontal cortex (Fan et al., 2021). Considering the inconsistent findings stemming from investigations of amygdala reactivity in BPD, including observed hyper- (Herpertz et al., 2001; Donegan et al., 2003, Schulze et al., 2016) as well as hypo-reactivity (see Ruocco et al., 2013 for a systematic review and meta-analysis), paranoid ideation may be one of the missing links to elucidating amygdala involvement, while also offering a possible aetiological link between (childhood) trauma, neurobiological changes, and delusional experiences.

First and foremost, the above explorations and comparisons emphasise the continued need for a more standardised approach to measuring and evaluating psychotic experiences in BPD. The current assessments were the first to use the CAPS and the PDI, both of which demonstrated remarkable reproducibility. Re-applying them in future (quantitative) studies would help to determine whether their (apparently replicable) prevalence findings remain robust. Nevertheless, the incredible variety of instruments that used to date make it challenging to tease apart the nuances between symptoms and symptom categories, population characteristics, as well as biased (and unbiased) interviewers drawing the arbitrary line between psychotic and non-psychotic. A careful and thorough meta-analysis may be a helpful step towards disentangling some of the apparent contradictions, not least by resolutely assessing study quality and weighing which findings should perhaps best be considered with caution. To enable a more accurate and generalisable examination of these experiences in the future, it may additionally be beneficial to develop a dedicated measure of PEs in the context of borderline pathology that specifically considers the broad spectrum of symptom type, range, and severity observed in and reported by people with BPD. Such

an endeavour must not simply draw on established psychometric measures (as is customary, see the development of the CAPS by Bell et al., 2006) but be led by experts-by-experience or else risk forfeiting its relevance, applicability, and ultimately worth.

8.1.3 Correlations, replications, and explorations

By and large, this thesis offers a replication and compilation of a series of correlational relationships with psychotic experiences previously observed or hypothesised by others, including associations of hallucinations (CAPS) and delusions (PDI) with depression and anxiety (Kelleher et al., 2015; Perez et al., 2018; Bhavsar et al., 2019), childhood trauma (Read et al., 2003; Daalman et al., 2012; Gaudiano and Zimmerman, 2009), and post-traumatic stress (Compean and Hamner, 2019). It also addresses the observed and hypothesised relationship between psychotic experiences and increased borderline pathology, as well as the potential importance of emotional valence in the context of PE severity.

Associations with high psychotic symptoms scores

On average, participants with BPD who scored above the CAPE clinical significance threshold of 1.47 (mean-weighted frequency) had significantly higher MSI (borderline pathology) scores than those below the threshold. This suggests a possible link between psychotic symptoms and disorder severity and resembles findings by Baryshnikov et al. (2018) who reported a correlation between MSI score and CAPE-42 (positive) symptom frequency. The result is particularly noteworthy, considering that the McLean Screening Instrument is a brief, ten-item measure, for which any given person with a diagnosis of borderline PD would be expected to obtain a score of seven or above (BPD group inclusion criterium in Study 1's). As such, a significantly higher average in the high-PE participant sample occurring within such a limited window is quite striking and certainly warrants further consideration, especially since direct comparison of item prevalence did not show any differences for specific symptom categories.

However, in Study 2, correlations between CAPE and BSL (assessing BPD symptoms on a 5-point Likert scale) were non-significant, and whether increased MSI scores may be

regarded as an accurate indication of disorder severity should stand to debate. Ten dichotomous questions, designed as a quick screening instrument, are unlikely to capture the full spectrum of a diagnosis (and experience) as multifaceted as BPD. Similarly, many of the symptoms assessed show phenotypical overlap with other psychiatric disorders (e.g., impulsivity, moodiness, unreality). As such, high MSI scores could perhaps be more accurately described as reflecting a broader range of (borderline and associated) psychopathology, possibly also indicating higher rates of common mental distress or post-traumatic symptoms.

This in turn would be in line with the link between CAPE score and number of (diagnosed) psychiatric comorbidities (also previously reported by Niemantsverdriet et al., 2017), as well as with the (moderate) correlations between psychotic experience scores (CAPS and PDI) and self-reported levels of depression and anxiety. Participants accounts of factors that trigger or influence their experiences equally suggest a relationship between psychotic symptoms and worsening mood (identified by 35%, 26/74), or worsening anxiety (identified by 45%, 33/74). Most prominently, 50% (37/74) of the combined BPD participant sample from studies one and two related their experiences (perceptual and delusional) to stress, thereby offering further (preliminary) evidence to support this often assumed but rarely assessed connection.

Emotional valence and the phantom words task

It has become increasingly evident that the emotional valence of a psychotic experience can have a significant impact on its severity and clinical significance, especially in the context of auditory hallucinations (Johns et al., 2014; Tsang et al., 2021; Buck et al., 2022). As such, the emotional vulnerability associated with BPD might make people with the disorder particularly susceptible to more distressing symptoms that require medical attention. Additionally, ambiguous or neutral sensory stimuli could perhaps be warped into meaningful (but ultimately false) perceptions and beliefs, if imbued with sufficient emotional valence. Consequently, the phantom words task – modelled on Diana Deutsch's phantom words speech illusions (Deutsch, 2019) – was developed specifically for this dissertation, to assess such potential tendencies. As theorised, people with BPD were

significantly more likely to ascribe negative meaning to ambiguous auditory stimuli, selecting words with negative associations at a higher rate than positively connotated and neutral words, as well as compared to control participants. Outstandingly, this tendency persisted even for stimuli that had an underlying “true” signal, which the BPD group was significantly less likely to correctly identify (if positive), with a higher rate of selecting the (incorrect) negative word option instead.

Although uncorrelated with psychotic symptoms, these findings are reminiscent of previous studies on emotion recognition in BPD, including by Daros et al. (2013), who reported misattributions of emotions to faces depicting neutral expressions. As possibly the first consideration of how such a bias might affect other modalities (e.g., interpretation speech and sound), the phantom words findings offer valuable insights into hitherto unassessed aspects of borderline pathology, and it would be interesting to investigate how this auditory bias might relate to other aspects of psychopathology such as trauma, or depression. The task itself also warrants further development, including adequate validation in clinical and non-clinical populations, additional stimuli, and (most crucially) an assessment and confirmation of what exactly it measures. Pending this, it may in future prove a useful tool for research into borderline personality disorder and beyond.

8.1.4 Risk factors, and mechanisms

This dissertation considered three theoretical frameworks that (separately and in combination) may offer possible aetiological and cognitive pathways towards psychotic symptom development in people with BPD:

1. The predictive processing model.

Conceiving the brain as a Bayesian prediction-error minimisation machine, the predictive processing model of psychosis considers its core symptoms (hallucinations and delusions) the result of aberrant salience between the expectations of incoming sensory signals and the signals themselves (Fletcher and Frith, 2009). Psychotic experiences in the context of borderline pathology may be linked to similar cognitive perturbations.

2. *The common-mental-distress/ p-factor model.*

Conceptualised as a latent dimension of overall psychiatric vulnerability, the *p*-factor model (Lahey et al., 2012) offers an explanation for the high rates of psychiatric comorbidities and transdiagnostic symptoms observed in clinical practice. BPD consistently has one of the highest *p*-factor loadings (Caspi et al., 2014; Ringwald et al., 2021; Southward et al., 2023), and people with this diagnosis may be vulnerable to more (or more severe) psychiatric symptoms, with a directionality from CMD to psychosis.

3. *The post-traumatic model.*

Finally, according to the post-traumatic model, psychosis could be the consequence of trauma-induced neurobiological (structural and functional) alterations (Teicher et al., 2016; Dunn et al., 2017), or a sign of ongoing post-traumatic stress. Dissociation, which is considered a symptom of both BPD (Krause-Utz et al., 2021) and PTSD (Lyssenko et al., 2018), and is equally prevalent in populations with psychosis (Perona-Garcelán et al., 2010; Varese et al., 2012; Černis et al., 2022), may be a key modulator of this relationship.

Predictive Processing

The current findings do not support the hypothesis of an alteration in the balance of expected and observed signals as an underlying mechanism of psychotic symptom development in people with BPD, as participants of both studies (including before, and after the stress induction in Study 2) did not differ from the control groups on any of the two-tone image task measures. Additionally, neither the rate of disambiguation improvement (d') – used as a proxy for the balance between prior information and incoming sensory signals – nor the shift in response bias (β) correlated with the psychotic symptom scores (CAPS and PDI), an association previously observed in people with first episode psychosis (Teufel et al., 2015).

Of course, the two-tone image task can only provide an intermediary approximation of (visual) sensory prediction, and other experimental paradigms (e.g., the force-matching task; Shergill et al., 2003) may uncover differences between people with and without BPD

(or with BPD but with and without psychotic symptoms) in future. Similarly, predictive processes are thought to occur along a cortical hierarchy, from elementary low-level priors to complex higher-order predictions involving for example the interpretation of interpersonal interactions and social clues. Since the current task (chosen specifically due to its established use in the context of psychotic illness) assesses just one (relatively low-order) level of this cortical hierarchy, subsequent endeavours might address possible impairments or alterations higher up the signalling cascade. Considering the borderline associated difficulties in social cognition (Fonagy and Bateman, 2008; Domes et al., 2009; Schulze et al., 2013) sensory prediction processes may perhaps be more affected at these levels – respective or irrespective of psychotic experiences. Nevertheless, in view of the current evidence it appears that the predictive processing apparatus remains intact in people with borderline pathology and is unlikely to play a part in the development of psychotic experiences in this disorder.

This in turn has significant implications for our aetiological and functional understanding of these symptoms: Despite their evident phenomenological overlap with experiences described by people with psychotic disorder, both qualitatively (Kingdon et al., 2010; Slotema et al., 2012; Hepworth et al., 2013; Tschoeke et al., 2014) and with regard to symptom frequency and severity (Pearse et al., 2014; Niemantsverdriet et al., 2017; as well as evidenced by the current findings), their mechanisms appear to diverge from the aberrant prediction paradigms increasingly evidenced in schizophrenia and first-episode psychosis (e.g., Teufel et al., 2015; Haarsma et al., 2020; Haarsma et al., 2021).

Common Mental Distress

As expected, and in line with previous findings (Levy et al., 2007; anxiety: Gratz et al., 2008, Howard et al., 2021), CMD scores were significantly higher among BPD than control participants, across both study populations. Depression (QIDS) and anxiety (GAD) scores showed (inconsistent) correlations with hallucinatory (CAPS) and delusional (PDI) experiences, however the association was not strong enough to explain the considerable variance in psychotic symptoms. Equally, the best-subset selection of variables most

reliably predicting psychotic experiences did not support a significant contribution of CMD to the development of neither perceptual symptoms, nor unusual beliefs, in either study.

Interestingly, the structural equation model of all associated (observed and latent) factors suggested significant directional pathways from *Trauma* (latent construct of CTQ and PCL) to *CMD* (latent construct of the anxiety and depression scores), as well as to *Psychotic Experiences* (latent construct of CAPS and PDI; via dissociation), but no direct link between *CMD* and *Psychotic Experiences*. This would imply that the most plausible explanation for the observed relationship may be a shared aetiological process originating in adverse experiences and post-traumatic stress, rather than a causal pathway. While any inferences based on the SEM are inherently limited by the explorative nature of the analysis and its (relatively) small sample size, these conclusions are principally in line with the remaining results and may be (cautiously) regarded as preliminary evidence against a primarily *p*-factor based model of psychosis in BPD.

However, these findings do not necessarily contradict a possible aetiological or mechanistic overlap with affective psychoses. Indeed, qualitative differences in emotional intensity may perhaps even account for some of the observed variance in the CMD-psychosis relationship: Self-report measures of psychopathology typically condense depression and anxiety severity into a summary score based on symptom number and frequency, and rarely consider the subjective reality of these emotional states. For example, between two people endorsing the BDI item “*I feel irritated all the time*” (Beck et al., 1961), the ferocity and intrusiveness of this irritability might differ tremendously. As such, psychotic experiences could be more specifically associated with depression and anxiety magnitude (possibly facilitating acute psychotic relapse during severely heightened emotional states) – which indubitably proves much harder to measure. The considerable number of participants identifying worsening mood and anxiety as triggers for their (most significant) CAPS and PDI symptoms also highlight this potentiality and prove, that further efforts to elucidate the interplay between depression, anxiety, and psychotic symptoms in borderline PD are needed. Perhaps experience sampling paradigms, asking people with BPD and psychotic experiences to rate emotion amplitude (rather than specific CMD

symptoms) and relating this to the waxing and waning of their hallucinations and delusions, could form a useful part of such future endeavours.

While research into the possibly pathological consequences of psychosis-associated distress is sparse, a 2017 study assessed the potentially post-traumatic effects of psychotic episodes and found that symptoms appeared to elicit increased emotions of anger, sadness, distrust, and helplessness (Lu et al., 2017). Notably, participants also reported traumatic aspects of their resulting (often coercive) treatment, including involuntary hospitalisations and forced medication. In light of the already substantial amount of discrimination, dismissal, and disbelief that people with BPD face in both societal and clinical settings (Markham, 2003) – and which appears to only be amplified by disclosing psychotic symptoms (Adams and Sanders, 2011) – it is possible that the higher rates of depression and anxiety are in fact a byproduct of the mental and managerial burden of psychotic experiences, rather than the other way around. This would also explain the relatively low strength of the correlations between PE and CMD observed in the current studies, due to individual differences in the levels of discrimination experienced versus support received. Consequently, one possible approach to investigating this relationship could be as simple as asking participants with BPD what attitudes towards their psychotic symptoms (by self and others) they have been met with, and assessing whether and how these might pertain to CMD severity.

In summary, broadly consistent with epidemiological studies (Wigman et al., 2012; Kelleher et al., 2015; Perez et al., 2018; Heinze et al., 2008; Bhavsar et al., 2019) that relate the presence of psychotic symptoms to mental distress severity, psychotic experiences (hallucinations and delusions) in participants with BPD were moderately correlated with depression and anxiety scores in Study 1 (but not in Study 2). While hitherto unassessed links between these symptoms (including the role of emotion intensity and the possible reverse-directionality of the relationship) warrant further consideration, current results suggest that previous trauma and post-traumatic stress may be involved in the causal pathways of both CMD and psychosis, thereby giving rise to their correlational association.

Trauma and dissociation

The association between psychotic symptoms in BPD and incidences of (childhood) trauma and adversity have previously been noted by both quantitative (Tschoeke et al., 2014; Bhavsar et al., 2019) and qualitative (Yee et al., 2005; Adams and Sanders, 2011; Campodonico et al., 2022) assessments. However, whether the *number* of adverse experiences, specific *types* of trauma (with childhood sexual abuse most frequently postulated), or perhaps their actively ongoing psychological repercussions (e.g., in the form of PTSD) are more significantly associated with psychosis proneness (in borderline PD) remains the subject of continued debate.

Life-time occurrence of PTSD in the general population ($n=5877$, age 15-54) has previously been estimated at around 7.8% (Kessler et al., 1995). By contrast, between 65% (13/20, Study 2) and 77.8% (42/54, Study 1) of BPD participants scored above the PCL threshold (>31) indicative of a possible post-traumatic stress disorder. Aside from its possible aetiological implications for psychotic symptom development, this striking prevalence of clinically significant, trauma-related symptoms reflects the widely established overlap between BPD and PTSD (Pagura et al., 2010; Dell’Osso et al., 2019; Johnson et al., 2020), and further emphasises the importance of considering experiences of adversity and post-traumatic stress when trying to understand and treat borderline pathology (Niemantsverdriet et al., 2017).

As expected, incidences of childhood trauma (CTQ score) were significantly more common among the BPD (compared to the control) groups of both studies, and were correlated with the CAPS, mirroring the numerous links between early-life adversity and hallucination proneness observed trans-diagnostically (Ross and Joshi, 1992; Ross et al., 1994; Read et al., 2003; Daalman et al., 2012; Gaudiano and Zimmerman, 2009). Previous qualitative (Adams and Sanders, 2011) and quantitative (Varese et al., 2012) studies proposing an especially strong association between perceptual experiences and childhood sexual abused were only partially replicated, with a weak correlation in the first- and no significant relationship in the second study and findings did not necessarily suggest a uniquely elevated importance of particular types of adverse experiences. However, the

sample size of Study 2 was small, and no qualitative assessment of the psychotic symptoms was made. Moreover, associations between CTQ, and perceptual and delusional experiences were assessed using the CAPS and PDI totals which cover a wide range of symptom categories and are based on the number of symptoms endorsed (yes/no) rather than their frequency or intensity. It is therefore possible that specific types of trauma do not necessarily influence overall psychosis proneness, but exert an effect on the risk for very specific psychotic experiences. For example, childhood sexual abuse has been predominantly reported in the context of auditory-verbal hallucinations (Read et al., 2003; Hammersley et al., 2003; Varese et al., 2012), physical abuse in the context of perceptual abnormalities and grandiose belief (Velthorst et al., 2013), and (emotional and/or physical) neglect has been observed to correlate with suspiciousness and persecutory ideation (Ered and Ellman, 2019).

Remarkably (considering the apparent emphasis on *early*-life adversity in both, BPD and psychosis literature), psychotic experiences correlated markedly more strongly with the LSC-R (a measure of life-time adversity) than with the CTQ, for both the quantitative (number of experiences) and the qualitative (ongoing impact of experiences) score. While possibly biased due to a limited sample size (the LSC-R was only applied in Study 2) and limited generalisability, this essentially replicates results by Niemantsverdriet et al. (2017), who equally observed a relationship between psychotic symptom severity and number of life stressors (LSC-R score), that was stronger than its association with childhood trauma (measured by the CTQ shortform questionnaire). These observations challenge the prevailing emphasis on an age-dependent effect of trauma on psychosis (proneness). They also have considerable implications for the aetiological and mechanistic conceptualisation of psychotic experiences in BPD: If ongoing adversity can predict psychotic experiences more significantly than childhood trauma, then perhaps trauma-induced, irreversible changes in (early) brain development may not be the key driving factor of these symptoms. This is further evidenced by a growing body of research linking cessation of ongoing adversity to cessation of psychotic experiences in adult- (Kelleher et al., 2013), young adult- (Cristóbal-Narváez, 2016), and adolescent populational samples (Wolke et al., 2014; Strauss et al., 2018).

Whether (and to what extent) structural or functional neurobiological changes might influence psychotic symptom development in BPD could perhaps be investigated with imaging (or related) studies that consider key regions thought to be involved in this relationship (e.g., the hippocampus, Haijma et al., 2013; or the amygdala, Okada et al., 2016; Van Erp et al., 2016; Barth et al., 2021). While previous attempts to assess these structures in people with BPD (Herpertz et al., 2001; Donegan et al., 2003, Nunes et al., 2009; Ruocco et al., 2012; Ruocco et al., 2013; Schulze et al., 2016) have heretofore produced conflicting results, potential differences between people with BPD with and without psychotic experiences have not yet been investigated. Consequently, an MRI study comparing people with BPD and high versus low psychotic symptoms scores (and healthy controls), relating potential structural abnormalities to participants' trauma history, may not only offer a better insight into the mechanisms of psychosis in this disorder but perhaps also disentangle the historically paradoxical excursions into borderline neurobiology.

While the association between (early-life) trauma and psychosis is often purported to be a dose-response relationship (including in reviews by Read et al., 2005 and more recently Giannopoulou et al., 2023), a dose-threshold conceptualisation may perhaps be more accurate since studies have predominantly assessed the link between cumulative trauma exposure and (life-time) presence of psychotic symptoms (yes/no) (Whitfield et al., 2005; Anda et al., 2006; Spauwen et al., 2006; Shevlin et al., 2011), or the influence of trauma frequency and severity on the presence psychotic symptoms (Janssen et al., 2004; Lataster et al., 2006; Schreier et al., 2009). Considering the limited strength (and predictive value) of the link between trauma and psychosis in the current findings, additional variables are likely to mediate or moderate the relationship – with dissociation as a tenable candidate.

Across both studies, dissociative experiences were the strongest and most significant correlate of psychotic symptoms (both hallucinations, and delusions). A best-subset selection approach to identifying the most reliable predictors for perceptual experiences and delusional beliefs, respectively, further established dissociation as a key variable across all suggested models. Curiously, in both studies, PDI scores were best estimated by a two-factor framework of dissociation (DES and DEMO for Study 1 and Study 2, respectively) and post-traumatic stress associated pathology (PCL and LSC-R-Option 2 for

Study 1 and Study 2, respectively), whereas CAPS scores were most accurately predicted by dissociative symptoms alone. Additionally, while the final regression models for delusional thoughts (which were practically identical between studies, despite considerable differences in BPD population and dissociative symptom measures) displayed good fit, models for perceptual experiences appeared less reliable – which raises the intriguing possibility of a non-linear relationship between trauma, dissociation, and hallucinatory symptoms.

Aetiologically, this association could perhaps be exponential with initial events of adversity drastically increasing psychosis proneness, while additional incidences of trauma (or trauma severity) only marginally influencing further symptoms. While supported by two previous studies that found no cumulative effects of multiple versus single trauma-exposure in a group of people with psychotic disorder (Fisher et al., 2009; Fisher et al., 2010), this hypothesis cannot explain the clear correlation between the number of adverse life events and PEs observed in this, and other studies (Niemantsverdriet et al., 2017; Loewy et al., 2019).

Instead, previously some of the mentioned theories postulating that different adverse events might induce different types of psychotic experiences, may offer a possible insight into this observation. Dissociation is an immensely multifaceted phenomenological construct, and its symptoms may vary widely between participants. Certain aspects of dissociation could be disparately related to specific perceptual symptoms, for example, depersonalisation to temporal lobe associated disturbances, absorption to visual or auditory hallucinations, and memory-related factors to thought echoes. Qualitative assessments relating the subjective reality of a person's dissociative experiences to specific aspects of their (perceptual) psychotic symptoms could help shine a light on this potential connection.

Stress

While explorative, results of the stress induction experiment also supported a complex, (possibly non-linear) link between dissociative states and the acute transition to psychotic experiences (under stress). CADSS and PSI symptom scores were significantly correlated before and after stress, in both participant groups. In the BPD sample, dissociative state

symptoms showed an *increase* (non-significant, $p=.067$) in response to the stress induction event, which was correlated with the change in (subjective) stress rating. PSI scores on the other hand appeared to *decrease* (non-significant, $p=.056$) after stress, and the rate of this change was significantly associated with participants' baseline PE scores, such that a higher original PSI score predicted a more pronounced symptom decrease and vice versa. Despite the study's methodological limitations (including a small sample size, an artificial stress induction paradigm, and the PSI's design as a measure of ketamine-induced psychotomimetic states), these findings may offer preliminary evidence of a direct, real-time interplay, between psychotic and dissociative states.

They also raise the thought-provoking possibility that dissociation may confer both vulnerability towards, and protection from psychotic experiences. Shared structural or functional pathways (possibly as a result of previous or ongoing adversity) may increase the overall risk for developing psychotic symptoms in the presence of (or facilitated by) severe dissociative states. Equally, highly reactive dissociative (defence) mechanisms could perhaps provide a transient advantage against descent into more severe psychotic pathologies, by offering emotional and cognitive detachment from the experience.

In light of the considerable sensory implications of dissociative (and psychotic) experiences, a potentially interesting pathway from dissociation to psychosis could involve models of sensory deprivation that have long been established (Bexton et al., 1954; Daniel et al., 2014; Daniel and Mason, 2015), but are rarely considered in clinical populations. Preliminary evidence (Demartini et al., 2021) suggests that dissociative experiences in people with psychiatric disorder may be triggered by (experimentally induced) conditions of deprivation. Similarly, the at times significant and persistent sensory loss accompanying (some) dissociative experiences (e.g., loss of pain and temperature sensation, diminished visual or auditory clarity, or even feeling entirely detached from one's own body) could perhaps trigger a sort of afferent overcompensation, giving rise to hallucinations and delusions to offset the lack of sensory input. An extreme case of this possible mechanism may be reflected in people with Charles Bonnet syndrome (Berrios and Brook, 1982), a curious condition in which people who lost their sight late in life begin to experience "compensational" hallucinations. Such theories of course circle back to predictive

processing frameworks (which offer a possible conceptualisation of this process as the overreliance on prior expectations balancing out the lack of sensory input), and others have previously noted the model's potential to explain sensory-deprivation related phenomena (Corlett et al., 2009).

In view of the implicit (as part of the upcoming ICD-11 symptom criteria) and explicit role stress is hypothesised to play in the aetiology of or transition into psychotic states in BPD (not least evidenced by 50% of participants endorsing it as a trigger of their experiences), an expansion of research into the relationship is direly needed. Future studies may consider utilising a more extensive stress induction paradigm (e.g., the full TSST) that explicitly focusses on the interaction between stress, (current) dissociative symptoms, and (current) psychotic experiences. The previously suggested development of a PE measure specifically designed for use in people with borderline PD may greatly assist in this endeavour. In addition to subjective stress ratings, physiological measures such as cortisol levels (previously found to be elevated in people with BPD: Rausch et al., 2015; and people at ultra-high risk for psychosis: Chaumette et al., 2016) may also provide a more comprehensive insight into the obscure and complex pathways linking stress, dissociation, and psychosis.

Building on the aetiological model presented in this dissertation, next steps towards understanding the role of stress within the causal and mechanistic framework of psychosis in BPD, might chose follow and dissect the following hypothesis: In people with BPD, trauma-induced, functional changes to the hypothalamic–pituitary–adrenal axis (Murphy et al., 2022), maintained and reinforced by ongoing adversity, could lead to a dysregulated stress response (Walter et al., 2008; Aleknaviciute et al., 2016; Bourvis et al., 2017) with potentially chronically heightened levels of stress (as suggested by current findings) and an ensuing blunting effect (Duesenberg et al., 2019). Stress-induced increases in dissociative experiences (Graumann et al., 2023; and as suggested by current findings) and their associated sensory loss, may subsequently drive the development of psychotic experiences to compensate for the perceived sensory deprivation (Corlett et al., 2009), thereby facilitating both underlying vulnerability and acute transition to psychotic symptom states.

8.2 The (certainly not) final word

Overall, the results of this dissertation strongly support the conceptualisation of psychotic experiences in BPD within a trauma-response framework, with childhood adversity and (more significantly) subsequently accruing stressful life events predicting psychosis proneness. Dissociative symptoms (both acute and general) appear to play a central role in mediating this relationship, as well as possibly exerting an additional effect, independent from trauma. Preliminary evidence similarly suggests that exposure to stress may exacerbate dissociative symptoms, possibly facilitating transition to acute psychotic states, and future studies should focus on clarifying its involvement. Common mental distress bears further consideration as a significant correlational (but likely non-causal) factor, though more detailed research is needed to determine its exact position, function, and consequence, within the overall structure of aetiological and convergent variables. Current results do not support the involvement of aberrant (low-level) sensory prediction in the aetiological or mechanistic underpinnings of psychotic experiences in BPD.

As apparent from the assorted suggestions for forthcoming journeys into the theoretical and phenomenological rabbit hole of psychosis in borderline pathology, the quest towards a comprehensive understanding of their nature and mechanisms has only just begun. A vital step towards ensuring its continued progress (and hopefully, eventual conclusion) requires the clinical and academic practice to relinquish their ineffectual, dubious, and devaluing conceptualisation as “psychosis-like”, a term that persists despite a growing list of evidence to the contrary (Adams and Sanders, 2011; Pearse et al., 2014; Niemantsverdriet et al., 2017; Slotema et al., 2017; Slotema et al., 2019) – now further expanded by the results of this dissertation. In light of the impending implementation of the ICD-11 (which will be introducing the label into UK clinical practice as an “official” descriptor of perceptual and delusional experiences in people with personality disorder), it remains to be seen whether the recently swelling calls for its abolition (Van der Zwaard et al., 2001; Gras et al., 2014; D’Agostino et al., 2019) will be able to effect the desired progress – or go unheeded. Most poignantly, the dilemma serves to highlight the historic and ongoing failure to place lived experience at the centre of research.

The majority of past (and current) ventures into the link between psychotic symptoms and borderline personality appear primarily motivated by the Big Conceptual Questions: *Are they real? How real are they?* And where exactly does their occurrence (or qualitative content) place BPD within a manufactured hierarchy of mental disturbance? Perhaps future endeavours may instead be driven by more primitive concerns, such as: How might psychotic experiences inform our overall understanding of borderline pathology and effective treatment?

Paramount to the still substantial gaps in knowledge surrounding psychosis in BPD is the prevailing notion that trying to understand the experiences of someone with the disorder (interpersonally, clinically, or phenomenologically) truly does resemble “*the art of hugging a porcupine*” (Adrian, 2016) – a presumed impossibility, largely blamed on the person(s) that need understanding. Fortunately, Laurie Isop author of the unrelated, but surprisingly befitting children’s book *How do you hug a porcupine?* (Isop, 2011) provides a detailed proposal of how to address the issue:

Firstly, any further purely theoretical explorations of psychosis in BPD uninformed by lived experience and unlikely to provide valuable contributions to an already biased debate should be suspended in favour of advancing our understanding of the aetiology and mechanisms of these symptoms and what might possibly help to prevent them or alleviate their impact. Secondly, patient and public involvement needs to become a core aspect of hypothesis formulation and experiment design, rather than a mere afterthought. Finally (and most crucially) however, researchers must be respectful of-, unbiased towards, and truly care about the lived reality of the experiences they study.

While undoubtedly intending to convey the same message, Isop managed to eloquently summarise the above into the simple,

succinct,

logical,

(and marginally more appropriate in a literary context):

“Carefully.”



Chapter 9 Bibliography

- Adams, B., & Sanders, T. (2011). Experiences of psychosis in borderline personality disorder: A qualitative analysis. *Journal of Mental Health (Abingdon, England)*, 20(4), 381–391. <https://doi.org/10.3109/09638237.2011.577846>
- Adrian, L. (2016). *Die Kunst, ein Stachelschwein zu umarmen* (Erstausgabe Taschenbuch). Masou-Verlag.
- Alekhin, A., & Isagulova, E. (2022). Abuse in childhood as a precondition for the development of borderline personality disorder in adolescence. *INTERACCION Y PERSPECTIVA*, 12(2), Article 2. <https://doi.org/10.5281/zenodo.7114636>
- Aleknaviciute, J., Tulen, J. H. M., Kamperman, A. M., De Rijke, Y. B., Kooiman, C. G., & Kushner, S. A. (2016). Borderline and cluster C personality disorders manifest distinct physiological responses to psychosocial stress. *Psychoneuroendocrinology*, 72, 131–138. <https://doi.org/10.1016/j.psyneuen.2016.06.010>
- Aleman, A., Böcker, K. B. E., Hijman, R., De Haan, E. H. F., & Kahn, R. S. (2003). Cognitive basis of hallucinations in schizophrenia: Role of top-down information processing. *Schizophrenia Research*, 64(2–3), 175–185. [https://doi.org/10.1016/S0920-9964\(03\)00060-4](https://doi.org/10.1016/S0920-9964(03)00060-4)
- Alhudiri, I., Abusrewil, Z., Dakhil, O., Zwaik, M. A., Awn, M. A., Jallul, M., Ahmed, A. I., Abugrara, R., & Elzagheid, A. (2023). Impact of vaccination and risk factors on COVID-19 mortality amid delta wave in Libya: A single center cohort study. *PLOS ONE*, 18(8), e0289490. <https://doi.org/10.1371/journal.pone.0289490>
- Allardyce, J., Hollander, A.-C., Rahman, S., Dalman, C., & Zammit, S. (2023). Association of trauma, post-traumatic stress disorder and non-affective psychosis across the life course: A nationwide prospective cohort study. *Psychological Medicine*, 53(4), 1620–1628. <https://doi.org/10.1017/S0033291721003287>

- Al-Shamali, H. F., Winkler, O., Talarico, F., Greenshaw, A. J., Forner, C., Zhang, Y., Vermetten, E., & Burbach, L. (2022). A systematic scoping review of dissociation in borderline personality disorder and implications for research and clinical practice: Exploring the fog. *Australian & New Zealand Journal of Psychiatry*, 56(10), 1252–1264. <https://doi.org/10.1177/00048674221077029>
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition). American Psychiatric Association. <https://doi.org/10.1176/appi.books.9780890425596>
- Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, Ch., Perry, B. D., Dube, Sh. R., & Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical Neuroscience*, 256(3), 174–186. <https://doi.org/10.1007/s00406-005-0624-4>
- Anwyl-Irvine, A. L., Massonnié, J., Flitton, A., Kirkham, N., & Evershed, J. K. (2020). Gorilla in our midst: An online behavioral experiment builder. *Behavior Research Methods*, 52(1), 388–407. <https://doi.org/10.3758/s13428-019-01237-x>
- Arlot, S., & Celisse, A. (2010). A survey of cross-validation procedures for model selection. *Statistics Surveys*, 4(none). <https://doi.org/10.1214/09-SS054>
- Arseneault, L., Cannon, M., Fisher, H. L., Polanczyk, G., Moffitt, T. E., & Caspi, A. (2011). Childhood Trauma and Children's Emerging Psychotic Symptoms: A Genetically Sensitive Longitudinal Cohort Study. *American Journal of Psychiatry*, 168(1), 65–72. <https://doi.org/10.1176/appi.ajp.2010.10040567>
- Aue, T., & Okon-Singer, H. (Eds.). (2020). *Cognitive biases in health and psychiatric disorders* (1st ed.). Academic press is an imprint of Elsevier.
- Baik, J.-H. (2020). Stress and the dopaminergic reward system. *Experimental & Molecular Medicine*, 52(12), 1879–1890. <https://doi.org/10.1038/s12276-020-00532-4>
- Bailey, T., Alvarez-Jimenez, M., Garcia-Sanchez, A. M., Hulbert, C., Barlow, E., & Bendall, S. (2018). Childhood Trauma Is Associated With Severity of Hallucinations and Delusions in Psychotic Disorders: A Systematic Review and Meta-Analysis. *Schizophrenia Bulletin*, 44(5), 1111–1122. <https://doi.org/10.1093/schbul/sbx161>

- Barnes, G. L., Emsley, R., Garety, P., & Hardy, A. (2023). Investigating Specific Associations Between Childhood Victimization Profiles and Positive Psychosis Symptoms: The Mediating Roles of Anxiety, Depression, and Schema. *Schizophrenia Bulletin Open*, 4(1), sgad017. <https://doi.org/10.1093/schizbullopen/sgad017>
- Barnow, S., Arens, E. A., Sieswerda, S., Dinu-Biringer, R., Spitzer, C., & Lang, S. (2010). Borderline Personality Disorder and Psychosis: A Review. *Current Psychiatry Reports*, 12(3), 186–195. <https://doi.org/10.1007/s11920-010-0107-9>
- Barth, C., Nerland, S., De Lange, A.-M. G., Wortinger, L. A., Hilland, E., Andreassen, O. A., Jørgensen, K. N., & Agartz, I. (2021). In Vivo Amygdala Nuclei Volumes in Schizophrenia and Bipolar Disorders. *Schizophrenia Bulletin*, 47(5), 1431–1441. <https://doi.org/10.1093/schbul/sbaa192>
- Baryshnikov, I., Aaltonen, K., Koivisto, M., Melartin, T., Näätänen, P., Suominen, K., Karpov, B., Heikkinen, M., Oksanen, J., Paunio, T., Joffe, G., & Isometsä, E. (2018). Self-reported psychosis-like experiences in patients with mood disorders. *European Psychiatry*, 51, 90–97. <https://doi.org/10.1016/j.eurpsy.2016.07.005>
- Battle, C. L., Shea, M. T., Johnson, D. M., Yen, S., Zlotnick, C., Zanarini, M. C., Sanislow, C. A., Skodol, A. E., Gunderson, J. G., Grilo, C. M., McGlashan, T. H., & Morey, L. C. (2004). Childhood Maltreatment Associated With Adult Personality Disorders: Findings From the Collaborative Longitudinal Personality Disorders Study. *Journal of Personality Disorders*, 18(2), 193–211. <https://doi.org/10.1521/pedi.18.2.193.32777>
- Batty, R. A., Francis, A. J. P., Innes-Brown, H., Joshua, N. R., & Rossell, S. L. (2014). Neurophysiological Correlates of Configural Face Processing in Schizotypy. *Frontiers in Psychiatry*, 5. <https://doi.org/10.3389/fpsy.2014.00101>
- Beals, K., Sperry, S. H., & Sheffield, J. M. (2022). Empathy, Emotion Recognition, and Paranoia in the General Population. *Frontiers in Psychology*, 13, 804178. <https://doi.org/10.3389/fpsyg.2022.804178>
- Beatson, J. (2019). Borderline personality disorder and auditory verbal hallucinations. *Australasian Psychiatry*, 27(6), 548–551. <https://doi.org/10.1177/1039856219859290>

- Beatson, J. A., Broadbear, J. H., Duncan, C., Bourton, D., & Rao, S. (2019). Avoiding Misdiagnosis When Auditory Verbal Hallucinations Are Present in Borderline Personality Disorder. *Journal of Nervous & Mental Disease*, 207(12), 1048–1055. <https://doi.org/10.1097/NMD.0000000000001073>
- Bebbington, P., & Freeman, D. (2017). Transdiagnostic Extension of Delusions: Schizophrenia and Beyond. *Schizophrenia Bulletin*, 43(2), 273–282. <https://doi.org/10.1093/schbul/sbw191>
- Beck, A. T., Steer, R. A., & Brown, G. (1996). *Beck Depression Inventory–II* [dataset]. <https://doi.org/10.1037/t00742-000>
- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561–571. <https://doi.org/10.1001/archpsyc.1961.01710120031004>
- Becker, D. (2000). When she was bad: Borderline personality disorder in a post-traumatic age. *American Journal of Orthopsychiatry*, 70(4), 422–432. Scopus. <https://doi.org/10.1037/h0087769>
- Bell, V., Halligan, P. W., & Ellis, H. D. (2006). The Cardiff Anomalous Perceptions Scale (CAPS): A New Validated Measure of Anomalous Perceptual Experience. *Schizophrenia Bulletin*, 32(2), 366–377. <https://doi.org/10.1093/schbul/sbj014>
- Bell, V., Halligan, P. W., Pugh, K., & Freeman, D. (2011). Correlates of perceptual distortions in clinical and non-clinical populations using the Cardiff Anomalous Perceptions Scale (CAPS): Associations with anxiety and depression and a re-validation using a representative population sample. *Psychiatry Research*, 189(3), 451–457. <https://doi.org/10.1016/j.psychres.2011.05.025>
- Bentall, R. P., Rowse, G., Shryane, N., Kinderman, P., Howard, R., Blackwood, N., Moore, R., & Corcoran, R. (2009). The Cognitive and Affective Structure of Paranoid Delusions: A Transdiagnostic Investigation of Patients With Schizophrenia Spectrum Disorders and Depression. *Archives of General Psychiatry*, 66(3), 236. <https://doi.org/10.1001/archgenpsychiatry.2009.1>
- Benvenuti, A., Rucci, P., Ravani, L., Gonnelli, C., Frank, Balestrieri, M., Sbrana, A., Dell’Osso, L., & Cassano, G. (2005). Psychotic features in borderline patients: Is

- there a connection to mood dysregulation? *Bipolar Disorders*, 7(4), 338–343. <https://doi.org/10.1111/j.1399-5618.2005.00217.x>
- Bernstein, D. P., Fink, L., Handelsman, L., Foote, J., Lovejoy, M., Wenzel, K., Sapareto, E., & Ruggiero, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *The American Journal of Psychiatry*, 151(8), 1132–1136. <https://doi.org/10.1176/ajp.151.8.1132>
- Berrios, G. E., & Brook, P. (1982). The Charles Bonnet syndrome and the problem of visual perceptual disorders in the elderly. *Age and Ageing*, 11(1), 17–23. <https://doi.org/10.1093/ageing/11.1.17>
- Bexton, W. H., Heron, W., & Scott, T. H. (1954). Effects of decreased variation in the sensory environment. *Canadian Journal of Psychology / Revue Canadienne de Psychologie*, 8(2), 70–76. <https://doi.org/10.1037/h0083596>
- Bhavsar, V., Boydell, J., McGuire, P., Harris, V., Hotopf, M., Hatch, S. L., MacCabe, J. H., & Morgan, C. (2019). Childhood abuse and psychotic experiences – evidence for mediation by adulthood adverse life events. *Epidemiology and Psychiatric Sciences*, 28(03), 300–309. <https://doi.org/10.1017/S2045796017000518>
- Bhavsar, V., Dorrington, S., Morgan, C., Hatch, S. L., McGuire, P., Fusar-Poli, P., Mills, J., MacCabe, J. H., & Hotopf, M. (2021). Psychotic experiences, psychiatric comorbidity and mental health need in the general population: A cross-sectional and cohort study in Southeast London. *Psychological Medicine*, 51(1), 147–157. <https://doi.org/10.1017/S0033291719003106>
- Biskin, R. S., & Paris, J. (2012). Diagnosing borderline personality disorder. *CMAJ*, 184(16), 1789–1794. Scopus. <https://doi.org/10.1503/cmaj.090618>
- Blanke, O., Mohr, C., Michel, C. M., Pascual-Leone, A., Brugger, P., Seeck, M., Landis, T., & Thut, G. (2005). Linking out-of-body experience and self processing to mental own-body imagery at the temporoparietal junction. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 25(3), 550–557. <https://doi.org/10.1523/JNEUROSCI.2612-04.2005>
- Blevins, C. A., Weathers, F. W., Davis, M. T., Witte, T. K., & Domino, J. L. (2015). The Posttraumatic Stress Disorder Checklist for *DSM-5* (PCL-5): Development and Initial

- Psychometric Evaluation: Posttraumatic Stress Disorder Checklist for *DSM-5*. *Journal of Traumatic Stress*, 28(6), 489–498. <https://doi.org/10.1002/jts.22059>
- Bohus, M., Kleindienst, N., Limberger, M. F., Stieglitz, R.-D., Domsalla, M., Chapman, A. L., Steil, R., Philipsen, A., & Wolf, M. (2009). The Short Version of the Borderline Symptom List (BSL-23): Development and Initial Data on Psychometric Properties. *Psychopathology*, 42(1), 32–39. <https://doi.org/10.1159/000173701>
- Bourvis, N., Aouidad, A., Cabelguen, C., Cohen, D., & Xavier, J. (2017). How Do Stress Exposure and Stress Regulation Relate to Borderline Personality Disorder? *Frontiers in Psychology*, 8, 2054. <https://doi.org/10.3389/fpsyg.2017.02054>
- Bovin, M. J., Marx, B. P., Weathers, F. W., Gallagher, M. W., Rodriguez, P., Schnurr, P. P., & Keane, T. M. (2016). Psychometric properties of the PTSD Checklist for Diagnostic and Statistical Manual of Mental Disorders–Fifth Edition (PCL-5) in veterans. *Psychological Assessment*, 28(11), 1379–1391. <https://doi.org/10.1037/pas0000254>
- Bradley, M. M., & Lang, P. J. (1999). *Affective Norms for English Words (ANEW): Instruction Manual and Affective Ratings*.
- Braehler, C., Holowka, D., Brunet, A., Beaulieu, S., Baptista, T., Debrulle, J., Walker, C., & King, S. (2005). Diurnal cortisol in schizophrenia patients with childhood trauma. *Schizophrenia Research*, 79(2–3), 353–354. <https://doi.org/10.1016/j.schres.2004.07.007>
- Brambilla, P., Soloff, P. H., Sala, M., Nicoletti, M. A., Keshavan, M. S., & Soares, J. C. (2004). Anatomical MRI study of borderline personality disorder patients. *Psychiatry Research: Neuroimaging*, 131(2), 125–133. <https://doi.org/10.1016/j.pscychresns.2004.04.003>
- Bremner, J. D., Krystal, J. H., Putnam, F. W., Southwick, S. M., Marmar, C., Charney, D. S., & Mazure, C. M. (1998). Measurement of dissociative states with the Clinician-Administered Dissociative States Scale (CADSS). *Journal of Traumatic Stress*, 11(1), 125–136. <https://doi.org/10.1023/A:1024465317902>
- Buchman-Wildbaum, T., Unoka, Z., Dudas, R., Vizin, G., Demetrovics, Z., & Richman, M. J. (2021). Shame in Borderline Personality Disorder: Meta-Analysis. *Journal of*

- Personality Disorders*, 35(Suppl A), 149–161.
https://doi.org/10.1521/pedi_2021_35_515
- Buck, B., Munson, J., Chander, A., Wang, W., Brenner, C. J., Campbell, A. T., & Ben-Zeev, D. (2022). The relationship between appraisals of auditory verbal hallucinations and real-time affect and social functioning. *Schizophrenia Research*, 250, 112–119. <https://doi.org/10.1016/j.schres.2022.10.015>
- Bukenaite, A., Stochl, J., Mossaheb, N., Schäfer, M. R., Klier, C. M., Becker, J., Schloegelhofer, M., Papageorgiou, K., Montejo, A. L., Russo, D. A., Jones, P. B., Perez, J., & Amminger, G. P. (2017). Usefulness of the CAPE-P15 for detecting people at ultra-high risk for psychosis: Psychometric properties and cut-off values. *Schizophrenia Research*, 189, 69–74. <https://doi.org/10.1016/j.schres.2017.02.017>
- Bürky, M. (2008). The Concept of Psychosis: Historical and Phenomenological Aspects. *Schizophrenia Bulletin*, 34(6), 1200–1210. <https://doi.org/10.1093/schbul/sbm136>
- Byun, Y.-H., Ha, M., Kwon, H.-J., Hong, Y.-C., Leem, J.-H., Sakong, J., Kim, S. Y., Lee, C. G., Kang, D., Choi, H.-D., & Kim, N. (2013). Mobile phone use, blood lead levels, and attention deficit hyperactivity symptoms in children: A longitudinal study. *PloS One*, 8(3), e59742. <https://doi.org/10.1371/journal.pone.0059742>
- Campodonico, C., Varese, F., & Berry, K. (2022). Trauma and psychosis: A qualitative study exploring the perspectives of people with psychosis on the influence of traumatic experiences on psychotic symptoms and quality of life. *BMC Psychiatry*, 22(1), 213. <https://doi.org/10.1186/s12888-022-03808-3>
- Capra, C., Kavanagh, D. J., Hides, L., & Scott, J. (2013). Brief screening for psychosis-like experiences. *Schizophrenia Research*, 149(1–3), 104–107. <https://doi.org/10.1016/j.schres.2013.05.020>
- Capra, C., Kavanagh, D. J., Hides, L., & Scott, J. G. (2017). Current CAPE-15: A measure of recent psychotic-like experiences and associated distress. *Early Intervention in Psychiatry*, 11(5), 411–417. <https://doi.org/10.1111/eip.12245>
- Carlson, E. B. (1994). Studying the interaction between physical and psychological states with the Dissociative Experiences Scale. In *Dissociation: Culture, mind, and body*. (pp. 41–58). American Psychiatric Association.

- Carlson, E. B., & Putnam, F. W. (1993). An update on the Dissociative Experiences Scale. *Dissociation: Progress in the Dissociative Disorders*, 6(1), 16–27.
- Carlson, E. B., Putnam, F. W., Ross, C. A., Torem, M., Coons, P., Dill, D. L., Loewenstein, R. J., & Braun, B. G. (1993). Validity of the Dissociative Experiences Scale in screening for multiple personality disorder: A multicenter study. *The American Journal of Psychiatry*, 150(7), 1030–1036. <https://doi.org/10.1176/ajp.150.7.1030>
- Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., Meier, M. H., Ramrakha, S., Shalev, I., Poulton, R., & Moffitt, T. E. (2014). The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders? *Clinical Psychological Science*, 2(2), 119–137. <https://doi.org/10.1177/2167702613497473>
- Castelnovo, A., Cavallotti, S., Gambini, O., & D’Agostino, A. (2015). Post-bereavement hallucinatory experiences: A critical overview of population and clinical studies. *Journal of Affective Disorders*, 186, 266–274. <https://doi.org/10.1016/j.jad.2015.07.032>
- Castle, D. J. (2019). The complexities of the borderline patient: How much more complex when considering physical health? *Australasian Psychiatry*, 27(6), 552–555. <https://doi.org/10.1177/1039856219848833>
- Cavelti, M., Thompson, K., Chanen, A. M., & Kaess, M. (2021). Psychotic symptoms in borderline personality disorder: Developmental aspects. *Current Opinion in Psychology*, 37, 26–31. <https://doi.org/10.1016/j.copsyc.2020.07.003>
- Cavelti, M., Thompson, K. N., Hulbert, C., Betts, J., Jackson, H., Francey, S., Homan, P., & Chanen, A. M. (2019). Exploratory comparison of auditory verbal hallucinations and other psychotic symptoms among youth with borderline personality disorder or schizophrenia spectrum disorder. *Early Intervention in Psychiatry*, 13(5), 1252–1262. <https://doi.org/10.1111/eip.12763>
- Cerejeira, J., Lagarto, L., & Mukaetova-Ladinska, E. B. (2012). Behavioral and Psychological Symptoms of Dementia. *Frontiers in Neurology*, 3. <https://doi.org/10.3389/fneur.2012.00073>

- Černis, E., Cooper, M., & Chan, C. (2018). Developing a new measure of dissociation: The Dissociative Experiences Measure, Oxford (DEMO). *Psychiatry Research*, 269, 229–236. <https://doi.org/10.1016/j.psychres.2018.08.060>
- Černis, E., Ehlers, A., & Freeman, D. (2022). Psychological mechanisms connected to dissociation: Generating hypotheses using network analyses. *Journal of Psychiatric Research*, 148, 165–173. <https://doi.org/10.1016/j.jpsychires.2022.01.049>
- Chaouloff, F. (1999). Serotonin and Stress. *Neuropsychopharmacology*, 21(2), 28S–32S. [https://doi.org/10.1016/S0893-133X\(99\)00008-1](https://doi.org/10.1016/S0893-133X(99)00008-1)
- Chapman, A. L., Dixon-Gordon, K. L., & Walters, K. N. (2013). Borderline Personality Features Moderate Emotion Reactivity and Emotion Regulation in Response to a Fear Stressor. *Journal of Experimental Psychopathology*, 4(5), 451–470. <https://doi.org/10.5127/jep.031612>
- Chaumette, B., Kebir, O., Mam-Lam-Fook, C., Morvan, Y., Bourgin, J., Godsil, B. P., Plaze, M., Gaillard, R., Jay, T. M., & Krebs, M.-O. (2016). Salivary cortisol in early psychosis: New findings and meta-analysis. *Psychoneuroendocrinology*, 63, 262–270. <https://doi.org/10.1016/j.psyneuen.2015.10.007>
- Chin, W. W. (1998). The partial least squares approach for structural equation modeling. In *Modern methods for business research*. (pp. 295–336). Lawrence Erlbaum Associates Publishers.
- Chopra, H. D., & Beatson, J. A. (1988). Some aspects of the phenomenology of borderline personality disorder. *Indian Journal of Psychiatry*, 30(3), 221–226.
- Christie, J. E., Whalley, L. J., Dick, H., Blackwood, D. M. R., Blackburn, I. M., & Fink, G. (1986). Raised Plasma Cortisol Concentrations a Feature of Drug-Free Psychotics and not Specific for Depression. *British Journal of Psychiatry*, 148(1), 58–65. <https://doi.org/10.1192/bjp.148.1.58>
- Clark, A. (2016). Attention alters predictive processing. *Behavioral and Brain Sciences*, 39, e234. <https://doi.org/10.1017/S0140525X15002472>
- Clark, L. A. (2005). Temperament as a Unifying Basis for Personality and Psychopathology. *Journal of Abnormal Psychology*, 114(4), 505–521. <https://doi.org/10.1037/0021-843X.114.4.505>

- Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology, 103*(1), 103–116. <https://doi.org/10.1037/0021-843X.103.1.103>
- Clarkin, J. F., Widiger, T. A., Frances, A., Hurt, S. W., & Gilmore, M. (1983). Prototypic typology and the Borderline Personality Disorder. *Journal of Abnormal Psychology, 92*(3), 263–275. <https://doi.org/10.1037/0021-843X.92.3.263>
- Classen, C. C., Pain, C., Field, N. P., & Woods, P. (2006). Posttraumatic Personality Disorder: A Reformulation of Complex Posttraumatic Stress Disorder and Borderline Personality Disorder. *Psychiatric Clinics of North America, 29*(1), 87–112. <https://doi.org/10.1016/j.psc.2005.11.001>
- Cohen, S., Tyrrell, D. A., & Smith, A. P. (1991). Psychological stress and susceptibility to the common cold. *The New England Journal of Medicine, 325*(9), 606–612. <https://doi.org/10.1056/NEJM199108293250903>
- Combs, D. R., Michael, C. O., & Penn, D. L. (2006). Paranoia and emotion perception across the continuum. *British Journal of Clinical Psychology, 45*(1), 19–31. <https://doi.org/10.1348/014466505X29099>
- Compean, E., & Hamner, M. (2019). Posttraumatic stress disorder with secondary psychotic features (PTSD-SP): Diagnostic and treatment challenges. *Progress in Neuro-Psychopharmacology & Biological Psychiatry, 88*, 265–275. <https://doi.org/10.1016/j.pnpbp.2018.08.001>
- Comrey, A. L., & Lee, H. B. (1992). *A First Course in Factor Analysis* (0 ed.). Psychology Press. <https://doi.org/10.4324/9781315827506>
- Comtois, K. A., Cowley, D. S., Dunner, D. L., & Roy-Byrne, P. P. (1999). Relationship Between Borderline Personality Disorder and Axis I Diagnosis in Severity of Depression and Anxiety. *The Journal of Clinical Psychiatry, 60*(11), 752–758. <https://doi.org/10.4088/JCP.v60n1106>
- Coons, P. M., Bowman, E. S., Pellow, T. A., & Schneider, P. (1989). Post-Traumatic Aspects of the Treatment of Victims of Sexual Abuse and Incest. *Psychiatric Clinics of North America, 12*(2), 325–335. [https://doi.org/10.1016/S0193-953X\(18\)30434-9](https://doi.org/10.1016/S0193-953X(18)30434-9)

- Corlett, P. R., Frith, C. D., & Fletcher, P. C. (2009). From drugs to deprivation: A Bayesian framework for understanding models of psychosis. *Psychopharmacology*, 206(4), 515–530. <https://doi.org/10.1007/s00213-009-1561-0>
- Corlett, P. R., Honey, G. D., & Fletcher, P. C. (2007). From prediction error to psychosis: Ketamine as a pharmacological model of delusions. *Journal of Psychopharmacology*, 21(3), 238–252. <https://doi.org/10.1177/0269881107077716>
- Corlett, P. R., Honey, G. D., & Fletcher, P. C. (2016). Prediction error, ketamine and psychosis: An updated model. *Journal of Psychopharmacology*, 30(11), 1145–1155. <https://doi.org/10.1177/0269881116650087>
- Cougnard, A., Marcelis, M., Myin-Germeys, I., De Graaf, R., Vollebergh, W., Krabbendam, L., Lieb, R., Wittchen, H.-U., Henquet, C., Spauwen, J., & Van Os, J. (2007). Does normal developmental expression of psychosis combine with environmental risk to cause persistence of psychosis? A psychosis proneness–persistence model. *Psychological Medicine*, 37(04), 513. <https://doi.org/10.1017/S0033291706009731>
- Cristóbal-Narváez, P., Sheinbaum, T., Ballespí, S., Mitjavila, M., Myin-Germeys, I., Kwapil, T. R., & Barrantes-Vidal, N. (2016). Impact of Adverse Childhood Experiences on Psychotic-Like Symptoms and Stress Reactivity in Daily Life in Nonclinical Young Adults. *PLOS ONE*, 11(4), e0153557. <https://doi.org/10.1371/journal.pone.0153557>
- Cross, D., Fani, N., Powers, A., & Bradley, B. (2017). Neurobiological development in the context of childhood trauma. *Clinical Psychology: Science and Practice*, 24(2), 111–124. <https://doi.org/10.1111/cpsp.12198>
- Crump, M. J. C., McDonnell, J. V., & Gureckis, T. M. (2013). Evaluating Amazon’s Mechanical Turk as a tool for experimental behavioral research. *PloS One*, 8(3), e57410. <https://doi.org/10.1371/journal.pone.0057410>
- Cuesta, M. J., Gil, P., Artamendi, M., Serrano, J. F., & Peralta, V. (2002). Premorbid personality and psychopathological dimensions in first-episode psychosis. *Schizophrenia Research*, 58(2–3), 273–280. [https://doi.org/10.1016/s0920-9964\(01\)00395-4](https://doi.org/10.1016/s0920-9964(01)00395-4)

-
- Cullen, A. E., Fisher, H. L., Gullet, N., Fraser, E. R., Roberts, R. E., Zahid, U., To, M., Yap, N. H., Zunszain, P. A., Pariante, C. M., Wood, S. J., McGuire, P., Murray, R. M., Mondelli, V., & Laurens, K. R. (2022). Cortisol Levels in Childhood Associated With Emergence of Attenuated Psychotic Symptoms in Early Adulthood. *Biological Psychiatry*, 91(2), 226–235. <https://doi.org/10.1016/j.biopsych.2021.08.009>
- Daalman, K., Boks, M. P. M., Diederens, K. M. J., De Weijer, A. D., Blom, J. D., Kahn, R. S., & Sommer, I. E. C. (2011). The Same or Different?: A Phenomenological Comparison of Auditory Verbal Hallucinations in Healthy and Psychotic Individuals. *The Journal of Clinical Psychiatry*, 72(03), 320–325. <https://doi.org/10.4088/JCP.09m05797yel>
- Daalman, K., Diederens, K. M. J., Derks, E. M., Van Lutterveld, R., Kahn, R. S., & Sommer, I. E. C. (2012). Childhood trauma and auditory verbal hallucinations. *Psychological Medicine*, 42(12), 2475–2484. <https://doi.org/10.1017/S0033291712000761>
- D’Agostino, A., Rossi Monti, M., & Starcevic, V. (2019). Psychotic symptoms in borderline personality disorder: An update. *Current Opinion in Psychiatry*, 32(1), 22–26. <https://doi.org/10.1097/YCO.0000000000000462>
- Dalglish, T., Black, M., Johnston, D., & Bevan, A. (2020). Transdiagnostic approaches to mental health problems: Current status and future directions. *Journal of Consulting and Clinical Psychology*, 88(3), 179–195. <https://doi.org/10.1037/ccp0000482>
- Daniel, C., Lovatt, A., & Mason, O. J. (2014). Psychotic-like experiences and their cognitive appraisal under short-term sensory deprivation. *Frontiers in Psychiatry*, 5, 106. <https://doi.org/10.3389/fpsy.2014.00106>
- Daniel, C., & Mason, O. J. (2015). Predicting psychotic-like experiences during sensory deprivation. *BioMed Research International*, 2015, 439379. <https://doi.org/10.1155/2015/439379>
- 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>
- David, A. S. (2010). Why we need more debate on whether psychotic symptoms lie on a continuum with normality. *Psychological Medicine*, 40(12), 1935–1942. <https://doi.org/10.1017/S0033291710000188>

- Davies, D. J., Teufel, C., & Fletcher, P. C. (2018). Anomalous Perceptions and Beliefs Are Associated With Shifts Toward Different Types of Prior Knowledge in Perceptual Inference. *Schizophrenia Bulletin*, 44(6), 1245–1253. <https://doi.org/10.1093/schbul/sbx177>
- De Weerth, C., Zijl, R. H., & Buitelaar, J. K. (2003). Development of cortisol circadian rhythm in infancy. *Early Human Development*, 73(1–2), 39–52. [https://doi.org/10.1016/S0378-3782\(03\)00074-4](https://doi.org/10.1016/S0378-3782(03)00074-4)
- Deckers, J. W. M., Lobbestael, J., Van Wingen, G. A., Kessels, R. P. C., Arntz, A., & Egger, J. I. M. (2015). The influence of stress on social cognition in patients with borderline personality disorder. *Psychoneuroendocrinology*, 52, 119–129. <https://doi.org/10.1016/j.psyneuen.2014.11.003>
- Dedovic, K., Renwick, R., Mahani, N. K., Engert, V., Lupien, S. J., & Pruessner, J. C. (2005). The Montreal Imaging Stress Task: Using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *Journal of Psychiatry & Neuroscience: JPN*, 30(5), 319–325.
- Dell’Osso, L., Cremone, I. M., Carpita, B., Dell’Oste, V., Muti, D., Massimetti, G., Barlati, S., Vita, A., Fagiolini, A., Carmassi, C., & Gesi, C. (2019). Rumination, post-traumatic stress disorder, and mood symptoms in borderline personality disorder. *Neuropsychiatric Disease and Treatment*, Volume 15, 1231–1238. <https://doi.org/10.2147/NDT.S198616>
- Demartini, B., Nisticò, V., Tedesco, R., Marzorati, A., Ferrucci, R., Priori, A., Gambini, O., & Caputo, G. B. (2021). Visual perception and dissociation during Mirror Gazing Test in patients with anorexia nervosa: A preliminary study. *Eating and Weight Disorders: EWD*, 26(5), 1541–1551. <https://doi.org/10.1007/s40519-020-00977-6>
- Dennis, B. K. (2014). Understanding Participant Experiences: Reflections of a Novice Research Participant. *International Journal of Qualitative Methods*, 13(1), 395–410. <https://doi.org/10.1177/160940691401300121>
- Deutsch, D. (2019). *Musical illusions and phantom words: How music and speech unlock mysteries of the brain*. Oxford University Press.

-
- Devinsky, O., Putnam, F., Grafman, J., Bromfield, E., & Theodore, W. H. (1989). Dissociative states and epilepsy. *Neurology*, 39(6), 835–840. <https://doi.org/10.1212/wnl.39.6.835>
- Distel, M. A., Carlier, A., Middeldorp, C. M., Derom, C. A., Lubke, G. H., & Boomsma, D. I. (2011). Borderline personality traits and adult attention-deficit hyperactivity disorder symptoms: A genetic analysis of comorbidity. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 156(7), 817–825. <https://doi.org/10.1002/ajmg.b.31226>
- Dokucu, M. E., & Cloninger, C. R. (2019). Personality disorders and physical comorbidities: A complex relationship. *Current Opinion in Psychiatry*, 32(5), 435–441. <https://doi.org/10.1097/YCO.0000000000000536>
- Dolan, R. J., Fink, G. R., Rolls, E., Booth, M., Holmes, A., Frackowiak, R. S. J., & Friston, K. J. (1997). How the brain learns to see objects and faces in an impoverished context. *Nature*, 389(6651), 596–599. <https://doi.org/10.1038/39309>
- Domes, G., Czieschnek, D., Weidler, F., Berger, C., Fast, K., & Herpertz, S. C. (2008). Recognition of Facial Affect in Borderline Personality Disorder. *Journal of Personality Disorders*, 22(2), 135–147. <https://doi.org/10.1521/pedi.2008.22.2.135>
- Domes, G., Schulze, L., & Herpertz, S. C. (2009). Emotion Recognition in Borderline Personality Disorder—A Review of the Literature. *Journal of Personality Disorders*, 23(1), 6–19. <https://doi.org/10.1521/pedi.2009.23.1.6>
- Donegan, N. H., Sanislow, C. A., Blumberg, H. P., Fulbright, R. K., Lacadie, C., Skudlarski, P., Gore, J. C., Olson, I. R., McGlashan, T. H., & Wexler, B. E. (2003). Amygdala hyperreactivity in borderline personality disorder: Implications for emotional dysregulation. *Biological Psychiatry*, 54(11), 1284–1293. [https://doi.org/10.1016/S0006-3223\(03\)00636-X](https://doi.org/10.1016/S0006-3223(03)00636-X)
- Duesenberg, M., Wolf, O. T., Metz, S., Roepke, S., Fleischer, J., Elias, V., Renneberg, B., Otte, C., & Wingefeld, K. (2019). Psychophysiological stress response and memory in borderline personality disorder. *European Journal of Psychotraumatology*, 10(1), 1568134. <https://doi.org/10.1080/20008198.2019.1568134>
- Dunn, E. C., Nishimi, K., Powers, A., & Bradley, B. (2017). Is developmental timing of trauma exposure associated with depressive and post-traumatic stress disorder

- symptoms in adulthood? *Journal of Psychiatric Research*, 84, 119–127.
<https://doi.org/10.1016/j.jpsychires.2016.09.004>
- Dyck, M., Habel, U., Slodczyk, J., Schlummer, J., Backes, V., Schneider, F., & Reske, M. (2009). Negative bias in fast emotion discrimination in borderline personality disorder. *Psychological Medicine*, 39(5), 855–864.
<https://doi.org/10.1017/S0033291708004273>
- Dziurkowska, E., & Wesolowski, M. (2021). Cortisol as a Biomarker of Mental Disorder Severity. *Journal of Clinical Medicine*, 10(21), 5204.
<https://doi.org/10.3390/jcm10215204>
- Ehrhart, I. C., Parker, P. E., Weidner, W. J., Dabney, J. M., Scott, J. B., & Haddy, F. J. (1975). Coronary vascular and myocardial responses to carotid body stimulation in the dog. *The American Journal of Physiology*, 229(3), 754–760.
<https://doi.org/10.1152/ajplegacy.1975.229.3.754>
- Ekino, S., Susa, M., Ninomiya, T., Imamura, K., & Kitamura, T. (2007). Minamata disease revisited: An update on the acute and chronic manifestations of methyl mercury poisoning. *Journal of the Neurological Sciences*, 262(1–2), 131–144.
<https://doi.org/10.1016/j.jns.2007.06.036>
- El-Gabalawy, R., Katz, L. Y., & Sareen, J. (2010). Comorbidity and Associated Severity of Borderline Personality Disorder and Physical Health Conditions in a Nationally Representative Sample. *Psychosomatic Medicine*, 72(7), 641–647.
<https://doi.org/10.1097/PSY.0b013e3181e10c7b>
- Ered, A., & Ellman, L. M. (2019). Specificity of Childhood Trauma Type and Attenuated Positive Symptoms in a Non-Clinical Sample. *Journal of Clinical Medicine*, 8(10), 1537. <https://doi.org/10.3390/jcm8101537>
- Fan, L., Klein, H., Bass, E., Springfield, C., & Pinkham, A. (2021). Amygdala hyperconnectivity in the paranoid state: A transdiagnostic study. *Journal of Psychiatric Research*, 138, 117–124.
<https://doi.org/10.1016/j.jpsychires.2021.03.049>
- Farnood, A., Johnston, B., & Mair, F. S. (2020). A mixed methods systematic review of the effects of patient online self-diagnosing in the ‘smart-phone society’ on the healthcare professional-patient relationship and medical authority. *BMC Medical*

- Informatics and Decision Making*, 20(1), 253. <https://doi.org/10.1186/s12911-020-01243-6>
- Feuchtersleben, E. (1845). *Lehrbuch der Ärztlichen Seelenkunde*. Austria: Gerold.
- First, M. B., Reed, G. M., Hyman, S. E., & Saxena, S. (2015). The development of the ICD-11 Clinical Descriptions and Diagnostic Guidelines for Mental and Behavioural Disorders. *World Psychiatry*, 14(1), 82–90. <https://doi.org/10.1002/wps.20189>
- Fisher, H. L., Jones, P. B., Fearon, P., Craig, T. K., Dazzan, P., Morgan, K., Hutchinson, G., Doody, G. A., McGuffin, P., Leff, J., Murray, R. M., & Morgan, C. (2010). The varying impact of type, timing and frequency of exposure to childhood adversity on its association with adult psychotic disorder. *Psychological Medicine*, 40(12), 1967–1978. <https://doi.org/10.1017/S0033291710000231>
- Fisher, H., Morgan, C., Dazzan, P., Craig, T. K., Morgan, K., Hutchinson, G., Jones, P. B., Doody, G. A., Pariante, C., McGuffin, P., Murray, R. M., Leff, J., & Fearon, P. (2009). Gender differences in the association between childhood abuse and psychosis. *British Journal of Psychiatry*, 194(4), 319–325. <https://doi.org/10.1192/bjp.bp.107.047985>
- Flasbeck, V., Popkirov, S., Ebert, A., & Brüne, M. (2020). Altered interoception in patients with borderline personality disorder: A study using heartbeat-evoked potentials. *Borderline Personality Disorder and Emotion Dysregulation*, 7(1), 24. <https://doi.org/10.1186/s40479-020-00139-1>
- Fleischhauer, V., Bruhn, J., Rasche, S., & Zaunseder, S. (2023). Photoplethysmography upon cold stress—Impact of measurement site and acquisition mode. *Frontiers in Physiology*, 14, 1127624. <https://doi.org/10.3389/fphys.2023.1127624>
- Fletcher, P. C., & Frith, C. D. (2009). Perceiving is believing: A Bayesian approach to explaining the positive symptoms of schizophrenia. *Nature Reviews Neuroscience*, 10(1), 48–58. <https://doi.org/10.1038/nrn2536>
- Flounders, M. W., González-García, C., Hardstone, R., & He, B. J. (2019). Neural dynamics of visual ambiguity resolution by perceptual prior. *eLife*, 8, e41861. <https://doi.org/10.7554/eLife.41861>
- 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>

- Fonseca-Pedrero, E., Paino, M., Santarén-Rosell, M., Lemos-Giráldez, S., & Muñiz, J. (2012). Psychometric properties of the Peters et al Delusions Inventory 21 in college students. *Comprehensive Psychiatry*, 53(6), 893–899. <https://doi.org/10.1016/j.comppsy.2012.01.007>
- Frankenburg, F. R., & Zanarini, M. C. (2006). Personality disorders and medical comorbidity. *Current Opinion in Psychiatry*, 19(4), 428–431. Scopus. <https://doi.org/10.1097/01.yco.0000228766.33356.44>
- Freeman, D. (2006). Delusions in the nonclinical population. *Current Psychiatry Reports*, 8(3), 191–204. <https://doi.org/10.1007/s11920-006-0023-1>
- Freeman, D. (2007). Suspicious minds: The psychology of persecutory delusions. *Clinical Psychology Review*, 27(4), 425–457. <https://doi.org/10.1016/j.cpr.2006.10.004>
- Frías, A. (2018). Positive psychotic symptoms in patients with borderline personality disorder: A valuable but still not completely understood clinical marker of the illness. *Australasian Psychiatry*, 26(3), 327–327. <https://doi.org/10.1177/1039856217749656>
- Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, 30(10), 1010–1016. <https://doi.org/10.1016/j.psyneuen.2005.04.006>
- Friston, K. (2005). A theory of cortical responses. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 360(1456), 815–836. <https://doi.org/10.1098/rstb.2005.1622>
- Friston, K., & Kiebel, S. (2009). Predictive coding under the free-energy principle. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364(1521), 1211–1221. <https://doi.org/10.1098/rstb.2008.0300>
- Furnes, D., Gjestad, R., Mehlum, L., Hodgekins, J., Kroken, R. A., Oedegaard, K., & Mellesdal, L. (2021). Borderline Personality Disorder: What Predicts Acute Psychiatric Readmissions? *Journal of Personality Disorders*, 35(4), 481–493. https://doi.org/10.1521/pedi_2019_33_459
- Garety, P. A., & Freeman, D. (1999). Cognitive approaches to delusions: A critical review of theories and evidence. *British Journal of Clinical Psychology*, 38(2), 113–154. <https://doi.org/10.1348/014466599162700>

- Garrison, J. R., Fernyhough, C., McCarthy-Jones, S., Simons, J. S., & Sommer, I. E. C. (2019). Paracingulate Sulcus Morphology and Hallucinations in Clinical and Nonclinical Groups. *Schizophrenia Bulletin*, 45(4), 733–741. <https://doi.org/10.1093/schbul/sby157>
- Gaudiano, B. A., & Zimmerman, M. (2009). The relationship between childhood trauma history and the psychotic subtype of major depression: Childhood trauma and psychotic depression. *Acta Psychiatrica Scandinavica*, 121(6), 462–470. <https://doi.org/10.1111/j.1600-0447.2009.01477.x>
- George, A., & Soloff, P. M. (1986). Schizotypal symptoms in patients with borderline personality disorders. *American Journal of Psychiatry*, 143(2), 212–215. <https://doi.org/10.1176/ajp.143.2.212>
- Ghisolfi, E. S., Margis, R., Becker, J., Zanardo, A. P., Strimitzer, I. M., & Lara, D. R. (2004). Impaired P50 sensory gating in post-traumatic stress disorder secondary to urban violence. *International Journal of Psychophysiology*, 51(3), 209–214. <https://doi.org/10.1016/j.ijpsycho.2003.09.002>
- Giannopoulou, I., Georgiades, S., Stefanou, M.-I., Spandidos, D., & Rizos, E. (2023). Links between trauma and psychosis (Review). *Experimental and Therapeutic Medicine*, 26(2), 386. <https://doi.org/10.3892/etm.2023.12085>
- Gibson, L. E., Alloy, L. B., & Ellman, L. M. (2016). Trauma and the psychosis spectrum: A review of symptom specificity and explanatory mechanisms. *Clinical Psychology Review*, 49, 92–105. <https://doi.org/10.1016/j.cpr.2016.08.003>
- Gillan, C. M., & Daw, N. D. (2016). Taking Psychiatry Research Online. *Neuron*, 91(1), 19–23. <https://doi.org/10.1016/j.neuron.2016.06.002>
- Gillan, C. M., Otto, A. R., Phelps, E. A., & Daw, N. D. (2015). Model-based learning protects against forming habits. *Cognitive, Affective & Behavioral Neuroscience*, 15(3), 523–536. <https://doi.org/10.3758/s13415-015-0347-6>
- Giourou, E., Skokou, M., Andrew, S. P., Alexopoulou, K., Gourzis, P., & Jelastopulu, E. (2018). Complex post-traumatic stress disorder: The need to consolidate a distinct clinical syndrome or to reevaluate features of psychiatric disorders following interpersonal trauma? *World Journal of Psychiatry*, 8(1), 12–19. <https://doi.org/10.5498/wjp.v8.i1.12>

- Girshkin, L., Matheson, S. L., Shepherd, A. M., & Green, M. J. (2014). Morning cortisol levels in schizophrenia and bipolar disorder: A meta-analysis. *Psychoneuroendocrinology*, 49, 187–206. <https://doi.org/10.1016/j.psyneuen.2014.07.013>
- Glaser, J.-P., Van Os, J., Thewissen, V., & Myin-Germeys, I. (2010). Psychotic reactivity in borderline personality disorder. *Acta Psychiatrica Scandinavica*, 121(2), 125–134. <https://doi.org/10.1111/j.1600-0447.2009.01427.x>
- Gloor, P., Olivier, A., Quesney, L. F., Andermann, F., & Horowitz, S. (1982). The role of the limbic system in experiential phenomena of temporal lobe epilepsy. *Annals of Neurology*, 12(2), 129–144. <https://doi.org/10.1002/ana.410120203>
- González-García, C., Flounders, M. W., Chang, R., Baria, A. T., & He, B. J. (2018). Content-specific activity in frontoparietal and default-mode networks during prior-guided visual perception. *eLife*, 7, e36068. <https://doi.org/10.7554/eLife.36068>
- González-Pinto, A., Van Os, J., Pérez De Heredia, J. L., Mosquera, F., Aldama, A., Lalaguna, B., Gutiérrez, M., & Micó, J. A. (2003). Age-dependence of Schneiderian psychotic symptoms in bipolar patients. *Schizophrenia Research*, 61(2–3), 157–162. [https://doi.org/10.1016/S0920-9964\(02\)00320-1](https://doi.org/10.1016/S0920-9964(02)00320-1)
- Government of Canada. (2015). *Electronic travel authorization: Description of application form fields*. Citizenship and Immigration Canada.
- Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., Smith, S. M., Dawson, D. S., Pulay, A. J., Pickering, R. P., & Ruan, W. J. (2008). Prevalence, Correlates, Disability, and Comorbidity of DSM-IV Borderline Personality Disorder: Results From the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *The Journal of Clinical Psychiatry*, 69(4), 533–545. <https://doi.org/10.4088/JCP.v69n0404>
- Gras, A., Amad, A., Thomas, P., & Jardri, R. (2014). Hallucinations et trouble de personnalité borderline: Une revue de littérature. *L'Encéphale*, 40(6), 431–438. <https://doi.org/10.1016/j.encep.2014.07.002>
- Gratz, K. L., Tull, M. T., & Gunderson, J. G. (2008). Preliminary data on the relationship between anxiety sensitivity and borderline personality disorder: The role of

- experiential avoidance. *Journal of Psychiatric Research*, 42(7), 550–559. <https://doi.org/10.1016/j.jpsychires.2007.05.011>
- Gratz, K. L., Weiss, N. H., McDermott, M. J., Dilillo, D., Messman-Moore, T., & Tull, M. T. (2017). Emotion Dysregulation Mediates the Relation Between Borderline Personality Disorder Symptoms and Later Physical Health Symptoms. *Journal of Personality Disorders*, 31(4), 433–448. https://doi.org/10.1521/pedi_2016_30_252
- Graumann, L., Duesenberg, M., Metz, S., Schulze, L., Wolf, O. T., Roepke, S., Otte, C., & Wingenfeld, K. (2021). Facial emotion recognition in borderline patients is unaffected by acute psychosocial stress. *Journal of Psychiatric Research*, 132, 131–135. <https://doi.org/10.1016/j.jpsychires.2020.10.007>
- Graumann, L., Heckerens, J. B., Duesenberg, M., Metz, S., Spitzer, C., Otte, C., Roepke, S., & Wingenfeld, K. (2023). Association between baseline dissociation levels and stress-induced state dissociation in patients with post-traumatic-stress disorder, borderline personality disorder, and major depressive disorder. *Borderline Personality Disorder and Emotion Dysregulation*, 10(1), 11. <https://doi.org/10.1186/s40479-023-00215-2>
- Griffin, J. D., & Fletcher, P. C. (2017). Predictive Processing, Source Monitoring, and Psychosis. *Annual Review of Clinical Psychology*, 13, 265–289. <https://doi.org/10.1146/annurev-clinpsy-032816-045145>
- Guillemin, M., Barnard, E., Allen, A., Stewart, P., Walker, H., Rosenthal, D., & Gillam, L. (2018). Do Research Participants Trust Researchers or Their Institution? *Journal of Empirical Research on Human Research Ethics*, 13(3), 285–294. <https://doi.org/10.1177/1556264618763253>
- Gunderson, J. G. (2007). Disturbed Relationships as a Phenotype for Borderline Personality Disorder. *American Journal of Psychiatry*, 164(11), 1637–1640. <https://doi.org/10.1176/appi.ajp.2007.07071125>
- Gunderson, J. G. (2009). Borderline Personality Disorder: Ontogeny of a Diagnosis. *American Journal of Psychiatry*, 166(5), 530–539. <https://doi.org/10.1176/appi.ajp.2009.08121825>
- Gunderson, J. G., Weinberg, I., Daversa, M. T., Kueppenbender, K. D., Zanarini, M. C., Shea, M. T., Skodol, A. E., Sanislow, C. A., Yen, S., Morey, L. C., Grilo, C. M.,

- McGlashan, T. H., Stout, R. L., & Dyck, I. (2006). Descriptive and Longitudinal Observations on the Relationship of Borderline Personality Disorder and Bipolar Disorder. *American Journal of Psychiatry*, 163(7), 1173–1178. <https://doi.org/10.1176/ajp.2006.163.7.1173>
- Haarsma, J., Fletcher, P. C., Griffin, J. D., Taverne, H. J., Ziauddeen, H., Spencer, T. J., Miller, C., Katthagen, T., Goodyer, I., Diederer, K. M. J., & Murray, G. K. (2021). Precision weighting of cortical unsigned prediction error signals benefits learning, is mediated by dopamine, and is impaired in psychosis. *Molecular Psychiatry*, 26(9), 5320–5333. <https://doi.org/10.1038/s41380-020-0803-8>
- Haarsma, J., Knolle, F., Griffin, J. D., Taverne, H., Mada, M., Goodyer, I. M., The Nspn Consortium, Fletcher, P. C., & Murray, G. K. (2020). Influence of prior beliefs on perception in early psychosis: Effects of illness stage and hierarchical level of belief. *Journal of Abnormal Psychology*, 129(6), 581–598. <https://doi.org/10.1037/abn0000494>
- Haijma, S. V., Van Haren, N., Cahn, W., Koolschijn, P. C. M. P., Hulshoff Pol, H. E., & Kahn, R. S. (2013). Brain Volumes in Schizophrenia: A Meta-Analysis in Over 18 000 Subjects. *Schizophrenia Bulletin*, 39(5), 1129–1138. <https://doi.org/10.1093/schbul/sbs118>
- Hair, J. F., Ringle, C. M., & Sarstedt, M. (2011). PLS-SEM: Indeed a Silver Bullet. *Journal of Marketing Theory and Practice*, 19(2), 139–152. <https://doi.org/10.2753/MTP1069-6679190202>
- Hamilton, H. K., Williams, T. J., Ventura, J., Jasperse, L. J., Owens, E. M., Miller, G. A., Subotnik, K. L., Nuechterlein, K. H., & Yee, C. M. (2018). Clinical and Cognitive Significance of Auditory Sensory Processing Deficits in Schizophrenia. *American Journal of Psychiatry*, 175(3), 275–283. <https://doi.org/10.1176/appi.ajp.2017.16111203>
- Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology*, 1, 293–319. <https://doi.org/10.1146/annurev.clinpsy.1.102803.143938>
- Hammersley, P., Dias, A., Todd, G., Bowen-Jones, K., Reilly, B., & Bentall, R. P. (2003). Childhood trauma and hallucinations in bipolar affective disorder: Preliminary

- investigation. *The British Journal of Psychiatry: The Journal of Mental Science*, 182, 543–547. <https://doi.org/10.1192/bjp.182.6.543>
- Hansson, G. K. (2005). Inflammation, atherosclerosis, and coronary artery disease. *The New England Journal of Medicine*, 352(16), 1685–1695. <https://doi.org/10.1056/NEJMra043430>
- Hara, K., Adachi, N., Akanuma, N., Ito, M., Okazaki, M., Matsubara, R., Adachi, T., Ishii, R., Kanemoto, K., Matsuura, M., Hara, E., Kato, M., & Onuma, T. (2015). Dissociative experiences in epilepsy: Effects of epilepsy-related factors on pathological dissociation. *Epilepsy & Behavior: E&B*, 44, 185–191. <https://doi.org/10.1016/j.yebeh.2014.12.018>
- Harris, C. L., Dinn, W. M., & Marcinkiewicz, J. A. (2002). Partial seizure-like symptoms in borderline personality disorder. *Epilepsy & Behavior*, 3(5), 433–438. [https://doi.org/10.1016/S1525-5050\(02\)00521-8](https://doi.org/10.1016/S1525-5050(02)00521-8)
- Hart, N., McGowan, J., Minati, L., & Critchley, H. D. (2013). Emotional Regulation and Bodily Sensation: Interoceptive Awareness Is Intact in Borderline Personality Disorder. *Journal of Personality Disorders*, 27(4), 506–518. https://doi.org/10.1521/pedi_2012_26_049
- Hegde, J., Thompson, S., & Kersten, D. (2010). Identifying faces in two-tone ('Mooney') images: A psychophysical and fMRI study. *Journal of Vision*, 7(9), 624–624. <https://doi.org/10.1167/7.9.624>
- Heins, M., Simons, C., Lataster, T., Pfeifer, S., Versmissen, D., Lardinois, M., Marcelis, M., Delespaul, P., Krabbendam, L., Van Os, J., Myin-Germeys, I., & for the GROUP project. (2011). Childhood Trauma and Psychosis: A Case-Control and Case-Sibling Comparison Across Different Levels of Genetic Liability, Psychopathology, and Type of Trauma. *American Journal of Psychiatry*, 168(12), 1286–1294. <https://doi.org/10.1176/appi.ajp.2011.10101531>
- Heinz, A., Voss, M., Lawrie, S. M., Mishara, A., Bauer, M., Gallinat, J., Juckel, G., Lang, U., Rapp, M., Falkai, P., Strik, W., Krystal, J., Abi-Dargham, A., & Galderisi, S. (2016). Shall we really say goodbye to first rank symptoms? *European Psychiatry*, 37, 8–13. <https://doi.org/10.1016/j.eurpsy.2016.04.010>

- Heinze, K., Lin, A., Nelson, B., Reniers, R. L. E. P., Upthegrove, R., Clarke, L., Roche, A., Lowrie, A., & Wood, S. J. (2018). The impact of psychotic experiences in the early stages of mental health problems in young people. *BMC Psychiatry*, 18(1), 214. <https://doi.org/10.1186/s12888-018-1767-y>
- Hengartner, M. P., Ajdacic-Gross, V., Rodgers, S., Müller, M., & Rössler, W. (2013). Childhood adversity in association with personality disorder dimensions: New findings in an old debate. *European Psychiatry: The Journal of the Association of European Psychiatrists*, 28(8), 476–482. <https://doi.org/10.1016/j.eurpsy.2013.04.004>
- Henriques-Calado, J., Pires, R., Paulino, M., Gama Marques, J., & Gonçalves, B. (2023). Psychotic spectrum features in borderline and bipolar disorders within the scope of the DSM-5 section III personality traits: A case control study. *Borderline Personality Disorder and Emotion Dysregulation*, 10(1), 2. <https://doi.org/10.1186/s40479-022-00205-w>
- Hepworth, C. R., Ashcroft, K., & Kingdon, D. (2013). Auditory hallucinations: A comparison of beliefs about voices in individuals with schizophrenia and borderline personality disorder. *Clinical Psychology & Psychotherapy*, 20(3), 239–245. <https://doi.org/10.1002/cpp.791>
- Herpertz, S. C., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., Thron, A., & Sass, H. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry*, 50(4), 292–298. [https://doi.org/10.1016/S0006-3223\(01\)01075-7](https://doi.org/10.1016/S0006-3223(01)01075-7)
- Hohwy, J. (2013). *The Predictive Mind*. Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780199682737.001.0001>
- Holowka, D. W., King, S., Saheb, D., Pukall, M., & Brunet, A. (2003). Childhood abuse and dissociative symptoms in adult schizophrenia. *Schizophrenia Research*, 60(1), 87–90. [https://doi.org/10.1016/S0920-9964\(02\)00296-7](https://doi.org/10.1016/S0920-9964(02)00296-7)
- Hotamisligil, G. S. (2006). Inflammation and metabolic disorders. *Nature*, 444(7121), 860–867. <https://doi.org/10.1038/nature05485>
- Howard, J., De Jesús-Romero, R., Peipert, A., Riley, T., Rutter, L. A., & Lorenzo-Luaces, L. (2021). The significance of anxiety symptoms in predicting psychosocial

- functioning across borderline personality traits. *PLOS ONE*, 16(1), e0245099. <https://doi.org/10.1371/journal.pone.0245099>
- Howes, O. D., & Kapur, S. (2009). The Dopamine Hypothesis of Schizophrenia: Version III--The Final Common Pathway. *Schizophrenia Bulletin*, 35(3), 549–562. <https://doi.org/10.1093/schbul/sbp006>
- Howes, O. D., Montgomery, A. J., Asselin, M.-C., Murray, R. M., Grasby, P. M., & McGuire, P. K. (2007). Molecular imaging studies of the striatal dopaminergic system in psychosis and predictions for the prodromal phase of psychosis. *British Journal of Psychiatry*, 191(S51), s13–s18. <https://doi.org/10.1192/bjp.191.51.s13>
- Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, 6(1), 1–55. <https://doi.org/10.1080/10705519909540118>
- Hudziak, J. J., Boffeli, T. J., Kreisman, J. J., Battaglia, M. M., Stanger, C., & Guze, S. B. (1996). Clinical study of the relation of borderline personality disorder to Briquet's syndrome (hysteria), somatization disorder, antisocial personality disorder, and substance abuse disorders. *The American Journal of Psychiatry*, 153(12), 1598–1606. <https://doi.org/10.1176/ajp.153.12.1598>
- Hynes, V. (2013). The trend toward self-diagnosis. *CMAJ: Canadian Medical Association Journal = Journal de l'Association Medicale Canadienne*, 185(3), E149-150. <https://doi.org/10.1503/cmaj.109-4383>
- Inyang, B., Gondal, F. J., Abah, G. A., Minnal Dhandapani, M., Manne, M., Khanna, M., Challa, S., Kabeil, A. S., & Mohammed, L. (2022). The Role of Childhood Trauma in Psychosis and Schizophrenia: A Systematic Review. *Cureus*. <https://doi.org/10.7759/cureus.21466>
- Ironside, M., Kumar, P., Kang, M.-S., & Pizzagalli, D. A. (2018). Brain mechanisms mediating effects of stress on reward sensitivity. *Current Opinion in Behavioral Sciences*, 22, 106–113. <https://doi.org/10.1016/j.cobeha.2018.01.016>
- Isop, L. (2011). *How do you hug a porcupine?* Simon & Schuster Books for Young Readers.

- Iudici, A., Quarato, M., & Neri, J. (2019). The Phenomenon of “Hearing Voices”: Not Just Psychotic Hallucinations—A Psychological Literature Review and a Reflection on Clinical and Social Health. *Community Mental Health Journal*, 55(5), 811–818. <https://doi.org/10.1007/s10597-018-0359-0>
- Janssen, I., Krabbendam, L., Bak, M., Hanssen, M., Vollebergh, W., Graaf, R., & Os, J. (2004). Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatrica Scandinavica*, 109(1), 38–45. <https://doi.org/10.1046/j.0001-690X.2003.00217.x>
- Jaremka, L. M., Lindgren, M. E., & Kiecolt-Glaser, J. K. (2013). SYNERGISTIC RELATIONSHIPS AMONG STRESS, DEPRESSION, AND TROUBLED RELATIONSHIPS: INSIGHTS FROM PSYCHONEUROIMMUNOLOGY: Review: Stress, Depression, Troubled Relationships, and Inflammation. *Depression and Anxiety*, 30(4), 288–296. <https://doi.org/10.1002/da.22078>
- Jaspers, K. (1913). *Allgemeine psychopathologie*. Springer-Verlag.
- Jiang, H. K., & Wang, J. Y. (1998). Diurnal melatonin and cortisol secretion profiles in medicated schizophrenic patients. *Journal of the Formosan Medical Association = Taiwan Yi Zhi*, 97(12), 830–837.
- Johns, L. C., Kompus, K., Connell, M., Humpston, C., Lincoln, T. M., Longden, E., Preti, A., Alderson-Day, B., Badcock, J. C., Cella, M., Fernyhough, C., McCarthy-Jones, S., Peters, E., Raballo, A., Scott, J., Siddi, S., Sommer, I. E., & Larøi, F. (2014). Auditory Verbal Hallucinations in Persons With and Without a Need for Care. *Schizophrenia Bulletin*, 40(Suppl_4), S255–S264. <https://doi.org/10.1093/schbul/sbu005>
- Johns, L. C., & Van Os, J. (2001). THE CONTINUITY OF PSYCHOTIC EXPERIENCES IN THE GENERAL POPULATION. *Clinical Psychology Review*, 21(8), 1125–1141. [https://doi.org/10.1016/S0272-7358\(01\)00103-9](https://doi.org/10.1016/S0272-7358(01)00103-9)
- Johnson, B. N., Lumley, M. A., Cheavens, J. S., & McKernan, L. C. (2020). Exploring the links among borderline personality disorder symptoms, trauma, and pain in patients with chronic pain disorders. *Journal of Psychosomatic Research*, 135, 110164. <https://doi.org/10.1016/j.jpsychores.2020.110164>

- Johnson, J. G., Cohen, P., Brown, J., Smailes, E. M., & Bernstein, D. P. (1999). Childhood maltreatment increases risk for personality disorders during early adulthood. *Archives of General Psychiatry*, 56(7), 600–606. <https://doi.org/10.1001/archpsyc.56.7.600>
- Jonas, J. M., & Pope, H. G. (1984). Psychosis in borderline personality disorder. *Psychiatric Developments*, 2(4), 295–308.
- Jones, S. R., & Fernyhough, C. (2007). Reliability of factorial structure of the Peters et al. Delusions inventory (PDI-21). *Personality and Individual Differences*, 43(4), 647–656. <https://doi.org/10.1016/j.paid.2007.01.018>
- Joynt, K. E., Whellan, D. J., & O'Connor, C. M. (2003). Depression and cardiovascular disease: Mechanisms of interaction. *Biological Psychiatry*, 54(3), 248–261. [https://doi.org/10.1016/s0006-3223\(03\)00568-7](https://doi.org/10.1016/s0006-3223(03)00568-7)
- Kapur, S. (2003). Psychosis as a State of Aberrant Salience: A Framework Linking Biology, Phenomenology, and Pharmacology in Schizophrenia. *American Journal of Psychiatry*, 160(1), 13–23. <https://doi.org/10.1176/appi.ajp.160.1.13>
- Kaymaz, N., & Van Os, J. (2010). Extended psychosis phenotype – yes: Single continuum – unlikely: A commentary on ‘Why we need more debate on whether psychotic symptoms lie on a continuum with normality’ by David (2010). *Psychological Medicine*, 40(12), 1963–1966. <https://doi.org/10.1017/S0033291710000358>
- Kegeles, L. S., Abi-Dargham, A., Frankle, W. G., Gil, R., Cooper, T. B., Slifstein, M., Hwang, D.-R., Huang, Y., Haber, S. N., & Laruelle, M. (2010). Increased Synaptic Dopamine Function in Associative Regions of the Striatum in Schizophrenia. *Archives of General Psychiatry*, 67(3), 231. <https://doi.org/10.1001/archgenpsychiatry.2010.10>
- Kelleher, I., Keeley, H., Corcoran, P., Ramsay, H., Wasserman, C., Carli, V., Sarchiapone, M., Hoven, C., Wasserman, D., & Cannon, M. (2013). Childhood trauma and psychosis in a prospective cohort study: Cause, effect, and directionality. *The American Journal of Psychiatry*, 170(7), 734–741. <https://doi.org/10.1176/appi.ajp.2012.12091169>
- Kelleher, I., Ramsay, H., & DeVlyder, J. (2017). Psychotic experiences and suicide attempt risk in common mental disorders and borderline personality disorder. *Acta Psychiatrica Scandinavica*, 135(3), 212–218. <https://doi.org/10.1111/acps.12693>

- Kelleher, I., Wigman, J. T. W., Harley, M., O'Hanlon, E., Coughlan, H., Rawdon, C., Murphy, J., Power, E., Higgins, N. M., & Cannon, M. (2015). Psychotic experiences in the population: Association with functioning and mental distress. *Schizophrenia Research*, 165(1), 9–14. <https://doi.org/10.1016/j.schres.2015.03.020>
- Kendler, K. S., & Mishara, A. (2019). The Prehistory of Schneider's First-Rank Symptoms: Texts From 1810 to 1932. *Schizophrenia Bulletin*, 45(5), 971–990. <https://doi.org/10.1093/schbul/sbz047>
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, 52(12), 1048–1060. <https://doi.org/10.1001/archpsyc.1995.03950240066012>
- Kingdon, D. G., Ashcroft, K., Bhandari, B., Gleeson, S., Warikoo, N., Symons, M., Taylor, L., Lucas, E., Mahendra, R., Ghosh, S., Mason, A., Badrakalimuthu, R., Hepworth, C., Read, J., & Mehta, R. (2010). Schizophrenia and Borderline Personality Disorder: Similarities and Differences in the Experience of Auditory Hallucinations, Paranoia, and Childhood Trauma. *Journal of Nervous & Mental Disease*, 198(6), 399–403. <https://doi.org/10.1097/NMD.0b013e3181e08c27>
- Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' – A Tool for Investigating Psychobiological Stress Responses in a Laboratory Setting. *Neuropsychobiology*, 28(1–2), 76–81. <https://doi.org/10.1159/000119004>
- Kirschbaum, C., Wolf, O. T., May, M., Wippich, W., & Hellhammer, D. H. (1996). Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sciences*, 58(17), 1475–1483. [https://doi.org/10.1016/0024-3205\(96\)00118-X](https://doi.org/10.1016/0024-3205(96)00118-X)
- Klein, P., Fairweather, A. K., & Lawn, S. (2022). Structural stigma and its impact on healthcare for borderline personality disorder: A scoping review. *International Journal of Mental Health Systems*, 16(1), 48. <https://doi.org/10.1186/s13033-022-00558-3>
- Kleindienst, N., Jungkunz, M., & Bohus, M. (2020). A proposed severity classification of borderline symptoms using the borderline symptom list (BSL-23). *Borderline*

- Personality Disorder and Emotion Dysregulation*, 7(1), 11. <https://doi.org/10.1186/s40479-020-00126-6>
- Kline, R. B. (2016). *Principles and practice of structural equation modeling* (Fourth edition). The Guilford Press.
- Knight, C., Stochl, J., Soneson, E., Russo, D. A., Jones, P. B., & Perez, J. (2020a). Revisiting CAPE-P15 cut-off values to increase sensitivity for detecting psychotic experiences in primary care. *Schizophrenia Research*, 216, 507–510. <https://doi.org/10.1016/j.schres.2019.11.051>
- Knight, C., Russo, D., Stochl, J., Jones, P. B., & Perez, J. (2020b). More sensitive identification of psychotic experiences in common mental disorder by primary mental healthcare services – effect on prevalence and recovery: Casting the net wider. *BJPsych Open*, 6(6), e136. <https://doi.org/10.1192/bjo.2020.120>
- Knight, C., Russo, D., Stochl, J., Croudace, T., Fowler, D., Grey, N., Reeve, N., Jones, P. B., & Perez, J. (2020c). Prevalence of and recovery from common mental disorder including psychotic experiences in the UK Primary Care Improving Access to Psychological Therapies (IAPT) Programme. *Journal of Affective Disorders*, 272, 84–90. <https://doi.org/10.1016/j.jad.2020.04.015>
- Knill, D. C., & Pouget, A. (2004). The Bayesian brain: The role of uncertainty in neural coding and computation. *Trends in Neurosciences*, 27(12), 712–719. <https://doi.org/10.1016/j.tins.2004.10.007>
- Knolle, F., Sterner, E., Moutoussis, M., Adams, R. A., Griffin, J. D., Haarsma, J., Taverne, H., Goodyer, I. M., Fletcher, P. C., & Murray, G. K. (2023). Action selection in early stages of psychosis: An active inference approach. *Journal of Psychiatry and Neuroscience*, 48(1), E78–E89. <https://doi.org/10.1503/jpn.220141>
- Krause-Utz, A. (2022). Dissociation, trauma, and borderline personality disorder. *Borderline Personality Disorder and Emotion Dysregulation*, 9(1), 14, s40479-022-00184-y. <https://doi.org/10.1186/s40479-022-00184-y>
- Krause-Utz, A., Frost, R., Chatzaki, E., Winter, D., Schmahl, C., & Elzinga, B. M. (2021). Dissociation in Borderline Personality Disorder: Recent Experimental, Neurobiological Studies, and Implications for Future Research and Treatment. *Current Psychiatry Reports*, 23(6), 37. <https://doi.org/10.1007/s11920-021-01246-8>

- Kreisman, J. J., & Straus, H. (1989). *I hate you—Don't leave me*. Avon Books.
- Kulaga, S. S., & Miller, C. W. T. (2021). Viral respiratory infections and psychosis: A review of the literature and the implications of COVID-19. *Neuroscience & Biobehavioral Reviews*, 127, 520–530. <https://doi.org/10.1016/j.neubiorev.2021.05.008>
- Kuyk, J., Spinhoven, P., Van Emde Boas, W., & Van Dyck, R. (1999). Dissociation in temporal lobe epilepsy and pseudo-epileptic seizure patients. *The Journal of Nervous and Mental Disease*, 187(12), 713–720. <https://doi.org/10.1097/00005053-199912000-00002>
- Lahey, B. B., Applegate, B., Hakes, J. K., Zald, D. H., Hariri, A. R., & Rathouz, P. J. (2012). Is there a general factor of prevalent psychopathology during adulthood? *Journal of Abnormal Psychology*, 121(4), 971–977. <https://doi.org/10.1037/a0028355>
- Lataster, T., Van Os, J., Drukker, M., Henquet, C., Feron, F., Gunther, N., & Myin-Germeys, I. (2006). Childhood victimisation and developmental expression of non-clinical delusional ideation and hallucinatory experiences: Victimization and non-clinical Psychotic experiences. *Social Psychiatry and Psychiatric Epidemiology*, 41(6), 423–428. <https://doi.org/10.1007/s00127-006-0060-4>
- Laurens, K. R., Hobbs, M. J., Sunderland, M., Green, M. J., & Mould, G. L. (2012). Psychotic-like experiences in a community sample of 8000 children aged 9 to 11 years: An item response theory analysis. *Psychological Medicine*, 42(7), 1495–1506. <https://doi.org/10.1017/S0033291711002108>
- Lawrie, S. M., Hall, J., McIntosh, A. M., Owens, D. G. C., & Johnstone, E. C. (2010). The ‘continuum of psychosis’: Scientifically unproven and clinically impractical. *British Journal of Psychiatry*, 197(6), 423–425. <https://doi.org/10.1192/bjp.bp.109.072827>
- Lee, C.-C., Chiang, H.-S., & Hsiao, M.-H. (2021). Effects of screen size and visual presentation on visual fatigue based on regional brain wave activity. *The Journal of Supercomputing*, 77(5), 4831–4851. <https://doi.org/10.1007/s11227-020-03458-w>
- LeGris, J., & Van Reekum, R. (2006). The Neuropsychological Correlates of Borderline Personality Disorder and Suicidal Behaviour. *The Canadian Journal of Psychiatry*, 51(3), 131–142. <https://doi.org/10.1177/070674370605100303>

- Leitman, D. I., Sehatpour, P., Higgins, B. A., Foxe, J. J., Silipo, G., & Javitt, D. C. (2010). Sensory Deficits and Distributed Hierarchical Dysfunction in Schizophrenia. *American Journal of Psychiatry*, 167(7), 818–827. <https://doi.org/10.1176/appi.ajp.2010.09030338>
- Levy, K. N., Edell, W. S., & McGlashan, T. H. (2007). Depressive Experiences in Inpatients with Borderline Personality Disorder. *Psychiatric Quarterly*, 78(2), 129–143. <https://doi.org/10.1007/s11126-006-9033-8>
- Li, S., Wang, C., Wang, W., Dong, H., Hou, P., & Tang, Y. (2008). Chronic mild stress impairs cognition in mice: From brain homeostasis to behavior. *Life Sciences*, 82(17–18), 934–942. <https://doi.org/10.1016/j.lfs.2008.02.010>
- Links, P. S., Steiner, M., & Mitton, J. (1989). Characteristics of Psychosis in Borderline Personality Disorder. *Psychopathology*, 22(4), 188–193. <https://doi.org/10.1159/000284595>
- Linscott, R. J., & Van Os, J. (2013). An updated and conservative systematic review and meta-analysis of epidemiological evidence on psychotic experiences in children and adults: On the pathway from proneness to persistence to dimensional expression across mental disorders. *Psychological Medicine*, 43(6), 1133–1149. <https://doi.org/10.1017/S0033291712001626>
- Loas, G., Pham-Scottez, A., Cailhol, L., Perez-Diaz, F., Corcos, M., & Speranza, M. (2013). Axis II comorbidity of borderline personality disorder in adolescents. *Psychopathology*, 46(3), 172–175. <https://doi.org/10.1159/000339530>
- Loewy, R. L., Corey, S., Amirfathi, F., Dabit, S., Fulford, D., Pearson, R., Hua, J. P. Y., Schlosser, D., Stuart, B. K., Mathalon, D. H., & Vinogradov, S. (2019). Childhood trauma and clinical high risk for psychosis. *Schizophrenia Research*, 205, 10–14. <https://doi.org/10.1016/j.schres.2018.05.003>
- Löffler, A., Foell, J., & Bekrater-Bodmann, R. (2018). Interoception and Its Interaction with Self, Other, and Emotion Processing: Implications for the Understanding of Psychosocial Deficits in Borderline Personality Disorder. *Current Psychiatry Reports*, 20(4), 28. <https://doi.org/10.1007/s11920-018-0890-2>

- Longden, E., Madill, A., & Waterman, M. G. (2012). Dissociation, trauma, and the role of lived experience: Toward a new conceptualization of voice hearing. *Psychological Bulletin*, 138(1), 28–76. <https://doi.org/10.1037/a0025995>
- Lopez, S. A., & Flagel, S. B. (2021). A proposed role for glucocorticoids in mediating dopamine-dependent cue-reward learning. *Stress*, 24(2), 154–167. <https://doi.org/10.1080/10253890.2020.1768240>
- Lowy, M. T., Gault, L., & Yamamoto, B. K. (1993). Rapid Communication: Adrenalectomy Attenuates Stress-Induced Elevations in Extracellular Glutamate Concentrations in the Hippocampus. *Journal of Neurochemistry*, 61(5), 1957–1960. <https://doi.org/10.1111/j.1471-4159.1993.tb09839.x>
- Lu, W., Mueser, K. T., Rosenberg, S. D., Yanos, P. T., & Mahmoud, N. (2017). Posttraumatic Reactions to Psychosis: A Qualitative Analysis. *Frontiers in Psychiatry*, 8, 129. <https://doi.org/10.3389/fpsyt.2017.00129>
- Ludmer, R., Dudai, Y., & Rubin, N. (2011). Uncovering Camouflage: Amygdala Activation Predicts Long-Term Memory of Induced Perceptual Insight. *Neuron*, 69(5), 1002–1014. <https://doi.org/10.1016/j.neuron.2011.02.013>
- Lupien, S. J., & Lepage, M. (2001). Stress, memory, and the hippocampus: Can't live with it, can't live without it. *Behavioural Brain Research*, 127(1–2), 137–158. [https://doi.org/10.1016/S0166-4328\(01\)00361-8](https://doi.org/10.1016/S0166-4328(01)00361-8)
- Lyssenko, L., Schmahl, C., Bockhacker, L., Vonderlin, R., Bohus, M., & Kleindienst, N. (2018). Dissociation in Psychiatric Disorders: A Meta-Analysis of Studies Using the Dissociative Experiences Scale. *American Journal of Psychiatry*, 175(1), 37–46. <https://doi.org/10.1176/appi.ajp.2017.17010025>
- MacCallum, R. C., Browne, M. W., & Sugawara, H. M. (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods*, 1(2), 130–149. <https://doi.org/10.1037/1082-989X.1.2.130>
- Madden, L. J., Flynn, C. T., Zandonatti, M. A., May, M., Parsons, L. H., Katner, S. N., Henriksen, S. J., & Fox, H. S. (2005). Modeling Human Methamphetamine Exposure in Nonhuman Primates: Chronic Dosing in the Rhesus Macaque Leads to Behavioral and Physiological Abnormalities. *Neuropsychopharmacology*, 30(2), 350–359. <https://doi.org/10.1038/sj.npp.1300575>

- Mak, A. D. P., & Lam, L. C. W. (2013). Neurocognitive profiles of people with borderline personality disorder: *Current Opinion in Psychiatry*, 26(1), 90–96. <https://doi.org/10.1097/YCO.0b013e32835b57a9>
- Markham, D. (2003). Attitudes towards patients with a diagnosis of ‘borderline personality disorder’: Social rejection and dangerousness. *Journal of Mental Health*, 12(6), 595–612. Scopus. <https://doi.org/10.1080/09638230310001627955>
- Martín-Blanco, A., Soler, J., Villalta, L., Feliu-Soler, A., Elices, M., Pérez, V., Arranz, M. J., Ferraz, L., Álvarez, E., & Pascual, J. C. (2014). Exploring the interaction between childhood maltreatment and temperamental traits on the severity of borderline personality disorder. *Comprehensive Psychiatry*, 55(2), 311–318. <https://doi.org/10.1016/j.comppsy.2013.08.026>
- Mason, O. J., Morgan, C. J. M., Stefanovic, A., & Curran, H. V. (2008). The Psychotomimetic States Inventory (PSI): Measuring psychotic-type experiences from ketamine and cannabis. *Schizophrenia Research*, 103(1–3), 138–142. <https://doi.org/10.1016/j.schres.2008.02.020>
- McAllister, T. W. (1998). Traumatic Brain Injury and Psychosis: What Is the Connection? *Seminars in Clinical Neuropsychiatry*, 3(3), 211–223.
- McGirr, A., Paris, J., Lesage, A., Phil., M., Renaud, J., & Turecki, G. (2007). Risk Factors for Suicide Completion in Borderline Personality Disorder: A Case-Control Study of Cluster B Comorbidity and Impulsive Aggression. *The Journal of Clinical Psychiatry*, 68(05), 721–729. <https://doi.org/10.4088/JCP.v68n0509>
- McGrath, B., & Dowling, M. (2012). Exploring Registered Psychiatric Nurses’ Responses towards Service Users with a Diagnosis of Borderline Personality Disorder. *Nursing Research and Practice*, 2012, 1–10. <https://doi.org/10.1155/2012/601918>
- McGrath, J. J., Lim, C. C. W., Plana-Ripoll, O., Holtz, Y., Agerbo, E., Momen, N. C., Mortensen, P. B., Pedersen, C. B., Abdulmalik, J., Aguilar-Gaxiola, S., Al-Hamzawi, A., Alonso, J., Bromet, E. J., Bruffaerts, R., Bunting, B., De Almeida, J. M. C., De Girolamo, G., De Vries, Y. A., Florescu, S., ... De Jonge, P. (2020). Comorbidity within mental disorders: A comprehensive analysis based on 145 990 survey respondents from 27 countries. *Epidemiology and Psychiatric Sciences*, 29, e153. <https://doi.org/10.1017/S2045796020000633>

- McGrath, J. J., Saha, S., Al-Hamzawi, A., Alonso, J., Bromet, E. J., Bruffaerts, R., Caldas-de-Almeida, J. M., Chiu, W. T., De Jonge, P., Fayyad, J., Florescu, S., Gureje, O., Haro, J. M., Hu, C., Kovess-Masfety, V., Lepine, J. P., Lim, C. C. W., Mora, M. E. M., Navarro-Mateu, F., ... Kessler, R. C. (2015). Psychotic Experiences in the General Population: A Cross-National Analysis Based on 31 261 Respondents From 18 Countries. *JAMA Psychiatry*, 72(7), 697. <https://doi.org/10.1001/jamapsychiatry.2015.0575>
- Meares, R., Gerull, F., Stevenson, J., & Korner, A. (2011). Is Self Disturbance the Core of Borderline Personality Disorder? An Outcome Study of Borderline Personality Factors. *Australian & New Zealand Journal of Psychiatry*, 45(3), 214–222. <https://doi.org/10.3109/00048674.2010.551280>
- Meijer, O. C., & De Kloet, E. R. (1998). Corticosterone and Serotonergic Neurotransmission in the Hippocampus: Functional Implications of Central Corticosteroid Receptor Diversity. *Critical ReviewsTM in Neurobiology*, 12(1–2), 1–20. <https://doi.org/10.1615/CritRevNeurobiol.v12.i1-2.10>
- Menon, M., Mizrahi, R., & Kapur, S. (2008). ‘Jumping to conclusions’ and delusions in psychosis: Relationship and response to treatment. *Schizophrenia Research*, 98(1–3), 225–231. <https://doi.org/10.1016/j.schres.2007.08.021>
- Merrett, Z., Rossell, S. L., & Castle, D. J. (2016). Comparing the experience of voices in borderline personality disorder with the experience of voices in a psychotic disorder: A systematic review. *Australian & New Zealand Journal of Psychiatry*, 50(7), 640–648. <https://doi.org/10.1177/0004867416632595>
- Meteran, H., Vindbjerg, E., Uldall, S. W., Glenthøj, B., Carlsson, J., & Oranje, B. (2019). Startle habituation, sensory, and sensorimotor gating in trauma-affected refugees with post-traumatic stress disorder. *Psychological Medicine*, 49(4), 581–589. <https://doi.org/10.1017/S003329171800123X>
- Millan, M. J., Agid, Y., Brüne, M., Bullmore, E. T., Carter, C. S., Clayton, N. S., Connor, R., Davis, S., Deakin, B., DeRubeis, R. J., Dubois, B., Geyer, M. A., Goodwin, G. M., Gorwood, P., Jay, T. M., Joëls, M., Mansuy, I. M., Meyer-Lindenberg, A., Murphy, D., ... Young, L. J. (2012). Cognitive dysfunction in psychiatric disorders:

- Characteristics, causes and the quest for improved therapy. *Nature Reviews Drug Discovery*, 11(2), 141–168. <https://doi.org/10.1038/nrd3628>
- Miller, F. T., Abrams, T., Dulit, R., & Fyer, M. (1993). Psychotic symptoms in patients with borderline personality disorder and concurrent Axis I disorder. *Hospital & Community Psychiatry*, 44(1), Article 1. psych.
- Minarikova, K., Prasko, J., Houdkova, M., Vanek, J., Kantor, K., Slepecky, M., Latalova, K., & Ociskova, M. (2022). Hallucinations and Other Psychotic Symptoms in Patients with Borderline Personality Disorder. *Neuropsychiatric Disease and Treatment*, Volume 18, 787–799. <https://doi.org/10.2147/NDT.S360013>
- Mittal, M., & Khan, S. (2010). Starvation Causes Acute Psychosis Due to Anterior Thalamic Infarction: *Southern Medical Journal*, 103(7), 701–703. <https://doi.org/10.1097/SMJ.0b013e3181e1e2f3>
- Moghaddam, B. (1993). Stress Preferentially Increases Extraneuronal Levels of Excitatory Amino Acids in the Prefrontal Cortex: Comparison to Hippocampus and Basal Ganglia. *Journal of Neurochemistry*, 60(5), 1650–1657. <https://doi.org/10.1111/j.1471-4159.1993.tb13387.x>
- Mondelli, V., Dazzan, P., Hepgul, N., Di Forti, M., Aas, M., D’Albenzio, A., Di Nicola, M., Fisher, H., Handley, R., Marques, T. R., Morgan, C., Navari, S., Taylor, H., Papadopoulos, A., Aitchison, K. J., Murray, R. M., & Pariante, C. M. (2010). Abnormal cortisol levels during the day and cortisol awakening response in first-episode psychosis: The role of stress and of antipsychotic treatment. *Schizophrenia Research*, 116(2–3), 234–242. <https://doi.org/10.1016/j.schres.2009.08.013>
- Monteleone, P., Tortorella, A., Scognamiglio, P., Serino, I., Monteleone, A. M., & Maj, M. (2012). The Acute Salivary Ghrelin Response to a Psychosocial Stress Is Enhanced in Symptomatic Patients with Bulimia Nervosa: A Pilot Study. *Neuropsychobiology*, 66(4), 230–236. <https://doi.org/10.1159/000341877>
- Moritz, S., Schilling, L., Wingenfeld, K., Köther, U., Wittekind, C., Terfehr, K., & Spitzer, C. (2011). Psychotic-like cognitive biases in borderline personality disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, 42(3), 349–354. <https://doi.org/10.1016/j.jbtep.2011.02.003>

- Moritz, S., Woodward, T. S., Whitman, J. C., & Cuttler, C. (2005). Confidence in Errors as a Possible Basis for Delusions in Schizophrenia. *Journal of Nervous & Mental Disease*, 193(1), 9–16. <https://doi.org/10.1097/01.nmd.0000149213.10692.00>
- Moscarelli, M. (2020). A major flaw in the diagnosis of schizophrenia: What happened to the Schneider's first rank symptoms. *Psychological Medicine*, 50(9), 1409–1417. <https://doi.org/10.1017/S0033291720001816>
- Muenzenmaier, K. H., Seixas, A. A., Schneeberger, A. R., Castille, D. M., Battaglia, J., & Link, B. G. (2015). Cumulative Effects of Stressful Childhood Experiences on Delusions and Hallucinations. *Journal of Trauma & Dissociation*, 16(4), 442–462. <https://doi.org/10.1080/15299732.2015.1018475>
- Murphy, F., Nasa, A., Cullinane, D., Raajakesary, K., Gazzaz, A., Sooknarine, V., Haines, M., Roman, E., Kelly, L., O'Neill, A., Cannon, M., & Roddy, D. W. (2022). Childhood Trauma, the HPA Axis and Psychiatric Illnesses: A Targeted Literature Synthesis. *Frontiers in Psychiatry*, 13, 748372. <https://doi.org/10.3389/fpsy.2022.748372>
- Murray, G. K., Corlett, P. R., Clark, L., Pessiglione, M., Blackwell, A. D., Honey, G., Jones, P. B., Bullmore, E. T., Robbins, T. W., & Fletcher, P. C. (2008). Substantia nigra/ventral tegmental reward prediction error disruption in psychosis. *Molecular Psychiatry*, 13(3), 267–276. <https://doi.org/10.1038/sj.mp.4002058>
- Mussgay, L., Klinkenberg, N., & Rüddel, H. (1999). Heart Beat Perception in Patients with Depressive, Somatoform, and Personality Disorders. *Journal of Psychophysiology*, 13(1), 27–36. <https://doi.org/10.1027//0269-8803.13.1.27>
- Nater, U., Lamarca, R., Florin, L., Moses, A., Langhans, W., Koller, M., & Ehlert, U. (2006). Stress-induced changes in human salivary alpha-amylase activity—Associations with adrenergic activity. *Psychoneuroendocrinology*, 31(1), 49–58. <https://doi.org/10.1016/j.psyneuen.2005.05.010>
- New, A. S., Hazlett, E. A., Buchsbaum, M. S., Goodman, M., Mitelman, S. A., Newmark, R., Trisdorfer, R., Haznedar, M. M., Koenigsberg, H. W., Flory, J., & Siever, L. J. (2007). Amygdala–Prefrontal Disconnection in Borderline Personality Disorder. *Neuropsychopharmacology*, 32(7), 1629–1640. <https://doi.org/10.1038/sj.npp.1301283>

- Neylan, T. C., Fletcher, D. J., Lenoci, M., McCallin, K., Weiss, D. S., Schoenfeld, F. B., Marmar, C. R., & Fein, G. (1999). Sensory gating in chronic post-traumatic stress disorder: Reduced auditory p50 suppression in combat veterans. *Biological Psychiatry*, 46(12), 1656–1664. [https://doi.org/10.1016/S0006-3223\(99\)00047-5](https://doi.org/10.1016/S0006-3223(99)00047-5)
- NHS Digital. (2023, January). Preparations for the implementation of ICD-11. *News*. <https://digital.nhs.uk/about-nhs-digital/essential-information-and-alerts-for-stakeholders/essential-information-for-technology-suppliers/preparations-for-the-implementation-of-icd-11>
- Niemantsverdriet, M. B. A., Slotema, C. W., Blom, J. D., Franken, I. H., Hoek, H. W., Sommer, I. E. C., & Van Der Gaag, M. (2017). Hallucinations in borderline personality disorder: Prevalence, characteristics and associations with comorbid symptoms and disorders. *Scientific Reports*, 7(1), 13920. <https://doi.org/10.1038/s41598-017-13108-6>
- Nishizono-Maher, A., Ikuta, N., Ogiso, Y., Moriya, N., Miyake, Y., & Minakawa, K. (1993). Psychotic symptoms in depression and borderline personality disorder. *Journal of Affective Disorders*, 28(4), 279–285. [https://doi.org/10.1016/0165-0327\(93\)90063-p](https://doi.org/10.1016/0165-0327(93)90063-p)
- Nuevo, R., Chatterji, S., Verdes, E., Naidoo, N., Arango, C., & Ayuso-Mateos, J. L. (2012). The Continuum of Psychotic Symptoms in the General Population: A Cross-national Study. *Schizophrenia Bulletin*, 38(3), 475–485. <https://doi.org/10.1093/schbul/sbq099>
- Nunes, P. M., Wenzel, A., Borges, K. T., Porto, C. R., Caminha, R. M., & De Oliveira, I. R. (2009). Volumes of the Hippocampus and Amygdala in Patients With Borderline Personality Disorder: A Meta-Analysis. *Journal of Personality Disorders*, 23(4), 333–345. <https://doi.org/10.1521/pedi.2009.23.4.333>
- Núñez, D., Godoy, M. I., Gaete, J., Faúndez, M. J., Campos, S., Fresno, A., & Spencer, R. (2021). The Community Assessment of Psychic Experiences-Positive scale (CAPE-P15) accurately classifies and differentiates psychotic experience levels in adolescents from the general population. *PLOS ONE*, 16(8), e0256686. <https://doi.org/10.1371/journal.pone.0256686>

- Okada, N., Fukunaga, M., Yamashita, F., Koshiyama, D., Yamamori, H., Ohi, K., Yasuda, Y., Fujimoto, M., Watanabe, Y., Yahata, N., Nemoto, K., Hibar, D. P., Van Erp, T. G. M., Fujino, H., Isobe, M., Isomura, S., Natsubori, T., Narita, H., Hashimoto, N., ... Hashimoto, R. (2016). Abnormal asymmetries in subcortical brain volume in schizophrenia. *Molecular Psychiatry*, 21(10), 1460–1466. <https://doi.org/10.1038/mp.2015.209>
- Oliva, F., Dalmotto, M., Pirfo, E., Furlan, P. M., & Picci, R. L. (2014). A comparison of thought and perception disorders in borderline personality disorder and schizophrenia: Psychotic experiences as a reaction to impaired social functioning. *BMC Psychiatry*, 14(1), 239. <https://doi.org/10.1186/s12888-014-0239-2>
- Pagura, J., Stein, M. B., Bolton, J. M., Cox, B. J., Grant, B., & Sareen, J. (2010). Comorbidity of borderline personality disorder and post-traumatic stress disorder in the U.S. population. *Journal of Psychiatric Research*, 44(16), 1190–1198. <https://doi.org/10.1016/j.jpsychires.2010.04.016>
- Pan, A., Keum, N., Okereke, O. I., Sun, Q., Kivimaki, M., Rubin, R. R., & Hu, F. B. (2012). Bidirectional association between depression and metabolic syndrome: A systematic review and meta-analysis of epidemiological studies. *Diabetes Care*, 35(5), 1171–1180. <https://doi.org/10.2337/dc11-2055>
- Pani, L., Porcella, A., & Gessa, G. L. (2000). The role of stress in the pathophysiology of the dopaminergic system. *Molecular Psychiatry*, 5(1), 14–21. <https://doi.org/10.1038/sj.mp.4000589>
- Paris, J. (2003). Personality Disorders Over Time: Precursors, Course and Outcome. *Journal of Personality Disorders*, 17(6), 479–488. <https://doi.org/10.1521/pedi.17.6.479.25360>
- Paris, J. (2005). Borderline personality disorder. *Canadian Medical Association Journal*, 172(12), 1579–1583. <https://doi.org/10.1503/cmaj.045281>
- Paris, J. (2019). Suicidality in Borderline Personality Disorder. *Medicina*, 55(6), 223. <https://doi.org/10.3390/medicina55060223>
- Paris, J., & Black, D. W. (2015). Borderline Personality Disorder and Bipolar Disorder: What is the Difference and Why Does it Matter? *Journal of Nervous & Mental Disease*, 203(1), 3–7. <https://doi.org/10.1097/NMD.0000000000000225>

- Parlar, M., Densmore, M., Hall, G. B., Frewen, P. A., Lanius, R. A., & McKinnon, M. C. (2017). Relation between patterns of intrinsic network connectivity, cognitive functioning, and symptom presentation in trauma-exposed patients with major depressive disorder. *Brain and Behavior*, 7(5), e00664. <https://doi.org/10.1002/brb3.664>
- Paulhus, D. L. (1984). Two-component models of socially desirable responding. *Journal of Personality and Social Psychology*, 46(3), 598–609. <https://doi.org/10.1037/0022-3514.46.3.598>
- Pearse, L. J., Dibben, C., Ziauddeen, H., Denman, C., & McKenna, P. J. (2014). A Study of Psychotic Symptoms in Borderline Personality Disorder. *Journal of Nervous & Mental Disease*, 202(5), 368–371. <https://doi.org/10.1097/NMD.0000000000000132>
- Pena-Garijo, J., Lacruz, M., Masanet, M. J., Palop-Grau, A., Plaza, R., Hernandez-Merino, A., Edo-Villamon, S., & Vallina, O. (2023). Specific facial emotion recognition deficits across the course of psychosis: A comparison of individuals with low-risk, high-risk, first-episode psychosis and multi-episode schizophrenia-spectrum disorders. *Psychiatry Research*, 320, 115029. <https://doi.org/10.1016/j.psychres.2022.115029>
- Peralta, V., & Cuesta, M. J. (1999). Diagnostic significance of Schneider's first-rank symptoms in schizophrenia: Comparative study between schizophrenic and non-schizophrenic psychotic disorders. *British Journal of Psychiatry*, 174(3), 243–248. <https://doi.org/10.1192/bjp.174.3.243>
- Peralta, V., & Cuesta, M. J. (2023). Schneider's first-rank symptoms have neither diagnostic value for schizophrenia nor higher clinical validity than other delusions and hallucinations in psychotic disorders. *Psychological Medicine*, 53(6), 2708–2711. <https://doi.org/10.1017/S0033291720003293>
- Perez, J., Russo, D. A., Stochl, J., Clarke, J., Martin, Z., Jassi, C., French, P., Fowler, D., & Jones, P. B. (2018). Common mental disorder including psychotic experiences: Trailblazing a new recovery pathway within the Improving Access to Psychological Therapies programme in England. *Early Intervention in Psychiatry*, 12(3), 497–504. <https://doi.org/10.1111/eip.12434>

- Perona-Garcelán, S., Carrascoso- López, F., García-Montes, J. M., Ductor-Recuerda, M. J., López Jiménez, A. M., Vallina-Fernández, O., Pérez-Álvarez, M., & Gómez-Gómez, M. T. (2012). Dissociative experiences as mediators between childhood trauma and auditory hallucinations: Dissociative Experiences, Childhood Trauma, and Auditory Hallucinations. *Journal of Traumatic Stress*, 25(3), 323–329. <https://doi.org/10.1002/jts.21693>
- Perona-Garcelán, S., García-Montes, J. M., Cuevas-Yust, C., Pérez-Álvarez, M., Ductor-Recuerda, M. J., Salas-Azcona, R., & Gómez-Gómez, M. T. (2010). A Preliminary Exploration of Trauma, Dissociation, and Positive Psychotic Symptoms in a Spanish Sample. *Journal of Trauma & Dissociation*, 11(3), 284–292. <https://doi.org/10.1080/15299731003786462>
- Peters, E., Joseph, S., Day, S., & Garety, P. (2004). Measuring Delusional Ideation: The 21-Item Peters et al. Delusions Inventory (PDI). *Schizophrenia Bulletin*, 30(4), 1005–1022. <https://doi.org/10.1093/oxfordjournals.schbul.a007116>
- Peters, E. R., Joseph, S. A., & Garety, P. A. (1999). Measurement of Delusional Ideation in the Normal Population: Introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin*, 25(3), 553–576. <https://doi.org/10.1093/oxfordjournals.schbul.a033401>
- Peters, E. R., Moritz, S., Schwannauer, M., Wiseman, Z., Greenwood, K. E., Scott, J., Beck, A. T., Donaldson, C., Hagen, R., Ross, K., Veckenstedt, R., Ison, R., Williams, S., Kuipers, E., & Garety, P. A. (2014). Cognitive Biases Questionnaire for Psychosis. *Schizophrenia Bulletin*, 40(2), 300–313. <https://doi.org/10.1093/schbul/sbs199>
- Petrowski, K., Herold, U., Joraschky, P., Wittchen, H.-U., & Kirschbaum, C. (2010). A striking pattern of cortisol non-responsiveness to psychosocial stress in patients with panic disorder with concurrent normal cortisol awakening responses. *Psychoneuroendocrinology*, 35(3), 414–421. <https://doi.org/10.1016/j.psyneuen.2009.08.003>
- Piacentini, M. F., Meeusen, R., Buyse, L., De Schutter, G., & De Meirleir, K. (2004). Hormonal responses during prolonged exercise are influenced by a selective DA/NA reuptake inhibitor. *British Journal of Sports Medicine*, 38(2), 129–133. <https://doi.org/10.1136/bjism.2002.000760>

- Polek, E., Neufeld, S. A. S., Wilkinson, P., Goodyer, I., St Clair, M., Prabhu, G., Dolan, R., Bullmore, E. T., Fonagy, P., Stochl, J., & Jones, P. B. (2020). How do the prevalence and relative risk of non-suicidal self-injury and suicidal thoughts vary across the population distribution of common mental distress (the p factor)? Observational analyses replicated in two independent UK cohorts of young people. *BMJ Open*, 10(5), e032494. <https://doi.org/10.1136/bmjopen-2019-032494>
- Pope, H. G. (1983). The Validity of DSM-III Borderline Personality Disorder: A Phenomenologic, Family History, Treatment Response, and Long-term Follow-up Study. *Archives of General Psychiatry*, 40(1), 23. <https://doi.org/10.1001/archpsyc.1983.01790010025003>
- Pope, H. G., Jonas, J. M., Hudson, J. I., Cohen, B. M., & Tohen, M. (1985). An empirical study of psychosis in borderline personality disorder. *American Journal of Psychiatry*, 142(11), 1285–1290. <https://doi.org/10.1176/ajp.142.11.1285>
- Porr, V. (2017). Real Life Consequences of Stigmatization, Misdiagnosis, Misunderstanding, and Mistreatment of Borderline Personality Disorder. *European Psychiatry*, 41(S1), S259–S260. <https://doi.org/10.1016/j.eurpsy.2017.02.065>
- Preti, A., Bonventre, E., Ledda, V., Petretto, D. R., & Masala, C. (2007). Hallucinatory Experiences, Delusional Thought Proneness, and Psychological Distress in a Nonclinical Population. *Journal of Nervous & Mental Disease*, 195(6), 484–491. <https://doi.org/10.1097/NMD.0b013e31802f205e>
- Pruessner, J. C., Champagne, F., Meaney, M. J., & Dagher, A. (2004). Dopamine Release in Response to a Psychological Stress in Humans and Its Relationship to Early Life Maternal Care: A Positron Emission Tomography Study Using [¹¹C]Raclopride. *The Journal of Neuroscience*, 24(11), 2825–2831. <https://doi.org/10.1523/JNEUROSCI.3422-03.2004>
- Quaedflieg, C. W. E. M., Meyer, T., & Smeets, T. (2013). The imaging Maastricht Acute Stress Test (iMAST): A neuroimaging compatible psychophysiological stressor: The imaging Maastricht Acute Stress Test (iMAST). *Psychophysiology*, 50(8), 758–766. <https://doi.org/10.1111/psyp.12058>

- Rafiq, S., Campodonico, C., & Varese, F. (2018). The relationship between childhood adversities and dissociation in severe mental illness: A meta-analytic review. *Acta Psychiatrica Scandinavica*, 138(6), 509–525. <https://doi.org/10.1111/acps.12969>
- Råheim, M., Magnussen, L. H., Sekse, R. J. T., Lunde, Å., Jacobsen, T., & Blystad, A. (2016). Researcher-researched relationship in qualitative research: Shifts in positions and researcher vulnerability. *International Journal of Qualitative Studies on Health and Well-Being*, 11, 30996. <https://doi.org/10.3402/qhw.v11.30996>
- Ramleth, R.-K., Groholt, B., Diep, L. M., Walby, F. A., & Mehlum, L. (2017). The impact of borderline personality disorder and sub-threshold borderline personality disorder on the course of self-reported and clinician-rated depression in self-harming adolescents. *Borderline Personality Disorder and Emotion Dysregulation*, 4(1), 22. <https://doi.org/10.1186/s40479-017-0073-5>
- Rausch, J., Gäbel, A., Nagy, K., Kleindienst, N., Herpertz, S. C., & Bertsch, K. (2015). Increased testosterone levels and cortisol awakening responses in patients with borderline personality disorder: Gender and trait aggressiveness matter. *Psychoneuroendocrinology*, 55, 116–127. <https://doi.org/10.1016/j.psyneuen.2015.02.002>
- Read, J., Agar, K., Argyle, N., & Aderhold, V. (2003). Sexual and physical abuse during childhood and adulthood as predictors of hallucinations, delusions and thought disorder. *Psychology and Psychotherapy: Theory, Research and Practice*, 76(1), 1–22. <https://doi.org/10.1348/14760830260569210>
- Read, J., Fosse, R., Moskowitz, A., & Perry, B. (2014). The traumagenic neurodevelopmental model of psychosis revisited. *Neuropsychiatry*, 4(1), 65–79. <https://doi.org/10.2217/npv.13.89>
- Read, J., Perry, B. D., Moskowitz, A., & Connolly, J. (2001). The Contribution of Early Traumatic Events to Schizophrenia in Some Patients: A Traumagenic Neurodevelopmental Model. *Psychiatry: Interpersonal and Biological Processes*, 64(4), 319–345. <https://doi.org/10.1521/psyc.64.4.319.18602>
- Read, J., Van Os, J., Morrison, A. P., & Ross, C. A. (2005). Childhood trauma, psychosis and schizophrenia: A literature review with theoretical and clinical implications. *Acta*

- Psychiatrica Scandinavica*, 112(5), 330–350. <https://doi.org/10.1111/j.1600-0447.2005.00634.x>
- Ringwald, W. R., Forbes, M. K., & Wright, A. G. C. (2021). Meta-analysis of structural evidence for the Hierarchical Taxonomy of Psychopathology (HiTOP) model. *Psychological Medicine*, 1–14. <https://doi.org/10.1017/S0033291721001902>
- Rivolta, D., Castellanos, N. P., Stawowsky, C., Helbling, S., Wibrall, M., Grützner, C., Koethe, D., Birkner, K., Kranaster, L., Enning, F., Singer, W., Leweke, F. M., & Uhlhaas, P. J. (2014). Source-Reconstruction of Event-Related Fields Reveals Hyperfunction and Hypofunction of Cortical Circuits in Antipsychotic-Naive, First-Episode Schizophrenia Patients during Mooney Face Processing. *The Journal of Neuroscience*, 34(17), 5909–5917. <https://doi.org/10.1523/JNEUROSCI.3752-13.2014>
- Rogers, D. C., Dittner, A. J., Rimes, K. A., & Chalder, T. (2017). Fatigue in an adult attention deficit hyperactivity disorder population: A trans-diagnostic approach. *The British Journal of Clinical Psychology*, 56(1), 33–52. <https://doi.org/10.1111/bjc.12119>
- Ross, C. A., Anderson, G., & Clark, P. (1994). Childhood abuse and the positive symptoms of schizophrenia. *Hospital & Community Psychiatry*, 45(5), 489–491.
- Ross, C. A., & Joshi, S. (1992). Paranormal Experiences in the General Population: *The Journal of Nervous and Mental Disease*, 180(6), 357–361. <https://doi.org/10.1097/00005053-199206000-00004>
- Roy-Byrne, P., Veitengruber, J. P., Bystritsky, A., Edlund, M. J., Sullivan, G., Craske, M. G., Welch, S. S., Rose, R., & Stein, M. B. (2009). Brief Intervention for Anxiety in Primary Care Patients. *The Journal of the American Board of Family Medicine*, 22(2), 175–186. <https://doi.org/10.3122/jabfm.2009.02.080078>
- Rozental, A., Sörman, K., Ojala, O., Jangard, S., El Alaoui, S., Månsson, K. N. T., Shahnava, S., Lundin, J., Forsström, D., Hedman-Lagerlöf, M., Lundgren, T., & Jayaram-Lindström, N. (2022). Mental health in individuals with self-reported psychiatric symptoms during the COVID-19 pandemic: Baseline data from a Swedish longitudinal cohort study. *Frontiers in Psychiatry*, 13, 933858. <https://doi.org/10.3389/fpsy.2022.933858>

- Ruggero, C. J., Zimmerman, M., Chelminski, I., & Young, D. (2010). Borderline personality disorder and the misdiagnosis of bipolar disorder. *Journal of Psychiatric Research*, 44(6), 405–408. <https://doi.org/10.1016/j.jpsychires.2009.09.011>
- Ruocco, A. C. (2005). The neuropsychology of borderline personality disorder: A meta-analysis and review. *Psychiatry Research*, 137(3), 191–202. <https://doi.org/10.1016/j.psychres.2005.07.004>
- Ruocco, A. C., Amirthavasagam, S., Choi-Kain, L. W., & McMain, S. F. (2013). Neural Correlates of Negative Emotionality in Borderline Personality Disorder: An Activation-Likelihood-Estimation Meta-Analysis. *Biological Psychiatry*, 73(2), 153–160. <https://doi.org/10.1016/j.biopsych.2012.07.014>
- Ruocco, A. C., Amirthavasagam, S., & Zakzanis, K. K. (2012). Amygdala and hippocampal volume reductions as candidate endophenotypes for borderline personality disorder: A meta-analysis of magnetic resonance imaging studies. *Psychiatry Research: Neuroimaging*, 201(3), 245–252. <https://doi.org/10.1016/j.pscychresns.2012.02.012>
- Rush, A. J., Trivedi, M. H., Ibrahim, H. M., Carmody, T. J., Arnow, B., Klein, D. N., Markowitz, J. C., Ninan, P. T., Kornstein, S., Manber, R., Thase, M. E., Kocsis, J. H., & Keller, M. B. (2003). The 16-Item quick inventory of depressive symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): A psychometric evaluation in patients with chronic major depression. *Biological Psychiatry*, 54(5), 573–583. [https://doi.org/10.1016/S0006-3223\(02\)01866-8](https://doi.org/10.1016/S0006-3223(02)01866-8)
- Rutter, L. A., Howard, J., Lakhan, P., Valdez, D., Bollen, J., & Lorenzo-Luaces, L. (2023). “I Haven’t Been Diagnosed, but I Should Be”—Insight Into Self-diagnoses of Common Mental Health Disorders: Cross-sectional Study. *JMIR Formative Research*, 7, e39206. <https://doi.org/10.2196/39206>
- Ryder, A. L., Azcarate, P. M., & Cohen, B. E. (2018). PTSD and Physical Health. *Current Psychiatry Reports*, 20(12), 116. <https://doi.org/10.1007/s11920-018-0977-9>
- Sachar, E. J. (1975). Evidence for neuroendocrine abnormalities in the major mental illnesses. *Research Publications - Association for Research in Nervous and Mental Disease*, 54, 347–358.

- Sala, M., Caverzasi, E., Lazzaretti, M., Morandotti, N., De Vidovich, G., Marraffini, E., Gambini, F., Isola, M., De Bona, M., Rambaldelli, G., d'Allio, G., Barale, F., Zappoli, F., & Brambilla, P. (2011). Dorsolateral prefrontal cortex and hippocampus sustain impulsivity and aggressiveness in borderline personality disorder. *Journal of Affective Disorders*, 131(1–3), 417–421. <https://doi.org/10.1016/j.jad.2010.11.036>
- Sanatinia, R., Wang, D., Tyrer, P., Tyrer, H., Crawford, M., Cooper, S., Loeberberg, G., & Barrett, B. (2016). Impact of personality status on the outcomes and cost of cognitive-behavioural therapy for health anxiety. *British Journal of Psychiatry*, 209(3), 244–250. <https://doi.org/10.1192/bjp.bp.115.173526>
- Sandi, C. (2013). Stress and cognition. *WIREs Cognitive Science*, 4(3), 245–261. <https://doi.org/10.1002/wcs.1222>
- Sanislow, C. A., Grilo, C. M., & McGlashan, T. H. (2000). Factor Analysis of the DSM-III-R Borderline Personality Disorder Criteria in Psychiatric Inpatients. *American Journal of Psychiatry*, 157(10), 1629–1633. <https://doi.org/10.1176/appi.ajp.157.10.1629>
- Sanislow, C. A., Morey, L. C., Grilo, C. M., Gunderson, J. G., Tracie Shea, M., Skodol, A. E., Stout, R. L., Zanarini, M. C., & McGlashan, T. H. (2002). Confirmatory factor analysis of DSM-IV borderline, schizotypal, avoidant and obsessive-compulsive personality disorders: Findings from the Collaborative Longitudinal Personality Disorders Study. *Acta Psychiatrica Scandinavica*, 105(1), 28–36. https://doi.org/10.1034/j.1600-0447.2002.0_479.x
- Sansone, R. A., & Sansone, L. A. (2012). Chronic pain syndromes and borderline personality. *Innovations in Clinical Neuroscience*, 9(1), 10–14.
- Scalabrini, A., Cavicchioli, M., Fossati, A., & Maffei, C. (2017). The extent of dissociation in borderline personality disorder: A meta-analytic review. *Journal of Trauma & Dissociation*, 18(4), 522–543. <https://doi.org/10.1080/15299732.2016.1240738>
- Schenk, L., & Bear, D. (1981). Multiple personality and related dissociative phenomena in patients with temporal lobe epilepsy. *The American Journal of Psychiatry*, 138(10), 1311–1316. <https://doi.org/10.1176/ajp.138.10.1311>
- Schiavone, F. L., McKinnon, M. C., & Lanius, R. A. (2018). Psychotic-Like Symptoms and the Temporal Lobe in Trauma-Related Disorders: Diagnosis, Treatment, and

- Assessment of Potential Malingering. *Chronic Stress*, 2, 247054701879704. <https://doi.org/10.1177/2470547018797046>
- Schmahl, C., Berne, K., Krause, A., Kleindienst, N., Valerius, G., Vermetten, E., & Bohus, M. (2009). Hippocampus and amygdala volumes in patients with borderline personality disorder with or without post-traumatic stress disorder. *Journal of Psychiatry & Neuroscience: JPN*, 34(4), 289–295.
- Schmitz, M., Müller, L. E., Schulz, A., Kleindienst, N., Herpertz, S. C., & Bertsch, K. (2020). Heart and brain: Cortical representation of cardiac signals is disturbed in borderline personality disorder, but unaffected by oxytocin administration. *Journal of Affective Disorders*, 264, 24–28. <https://doi.org/10.1016/j.jad.2019.11.139>
- Schneider, K. (1950). *Die psychopathischen Persönlichkeiten. [Psychopathic personalities.]* (pp. viii, 150). Franz Deuticke.
- Schneider, K. (1959). *Clinical psychopathology. (Trans. By M. W. Hamilton), 5th ed.* (pp. xvi, 173). Grune & Stratton.
- Schreiber, J. B., Nora, A., Stage, F. K., Barlow, E. A., & King, J. (2006). Reporting Structural Equation Modeling and Confirmatory Factor Analysis Results: A Review. *The Journal of Educational Research*, 99(6), 323–338. <https://doi.org/10.3200/JOER.99.6.323-338>
- Schreier, A., Wolke, D., Thomas, K., Horwood, J., Hollis, C., Gunnell, D., Lewis, G., Thompson, A., Zammit, S., Duffy, L., Salvi, G., & Harrison, G. (2009). Prospective Study of Peer Victimization in Childhood and Psychotic Symptoms in a Nonclinical Population at Age 12 Years. *Archives of General Psychiatry*, 66(5), 527. <https://doi.org/10.1001/archgenpsychiatry.2009.23>
- Schroeder, K., Fisher, H. L., & Schäfer, I. (2013). Psychotic symptoms in patients with borderline personality disorder: Prevalence and clinical management. *Current Opinion in Psychiatry*, 26(1), 113–119. <https://doi.org/10.1097/YCO.0b013e32835a2ae7>
- Schroeder, K., Schätzle, A., Kowohl, P., Leske, L., Huber, C., & Schäfer, I. (2018). Prävalenz und Phänomenologie fraglich psychotischer Symptome bei Borderline Persönlichkeitsstörungen: Assoziationen mit Suizidversuchen und Inanspruchnahme

- stationär-psychiatrischer Behandlung. *PPmP - Psychotherapie · Psychosomatik · Medizinische Psychologie*, 68(12), 516–524. <https://doi.org/10.1055/s-0043-124473>
- Schultz, H. E., & Hong, V. (2017). *Psychosis in borderline personality disorder: How assessment and treatment differs from a psychotic disorder*. 16, 24–29.
- Schulz, R., Beach, S. R., Ives, D. G., Martire, L. M., Ariyo, A. A., & Kop, W. J. (2000). Association between depression and mortality in older adults: The Cardiovascular Health Study. *Archives of Internal Medicine*, 160(12), 1761–1768. <https://doi.org/10.1001/archinte.160.12.1761>
- 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 Personality Disorder and Emotion Dysregulation*, 9(1), 35. <https://doi.org/10.1186/s40479-022-00206-9>
- Schulze, L., Domes, G., Köppen, D., & Herpertz, S. C. (2013). Enhanced Detection of Emotional Facial Expressions in Borderline Personality Disorder. *Psychopathology*, 46(4), 217–224. <https://doi.org/10.1159/000341730>
- Schulze, L., Schmahl, C., & Niedtfeld, I. (2016). Neural Correlates of Disturbed Emotion Processing in Borderline Personality Disorder: A Multimodal Meta-Analysis. *Biological Psychiatry*, 79(2), 97–106. <https://doi.org/10.1016/j.biopsych.2015.03.027>
- Seeman, P. (2010). Dopamine D2 Receptors as Treatment Targets in Schizophrenia. *Clinical Schizophrenia & Related Psychoses*, 4(1), 56–73. <https://doi.org/10.3371/CSRP.4.1.5>
- Selye, H. (1973). The evolution of the stress concept. *American Scientist*, 61(6), 692–699.
- Sevilla-Llewellyn-Jones, J., Camino, G., Russo, D. A., Painter, M., Montejo, A. L., Ochoa, S., Jones, P. B., & Perez, J. (2018). Clinically significant personality traits in individuals at high risk of developing psychosis. *Psychiatry Research*, 261, 498–503. <https://doi.org/10.1016/j.psychres.2018.01.027>
- Sharp, C., Ha, C., Carbone, C., Kim, S., Perry, K., Williams, L., & Fonagy, P. (2013). Hypermentalizing in Adolescent Inpatients: Treatment Effects and Association With Borderline Traits. *Journal of Personality Disorders*, 27(1), 3–18. <https://doi.org/10.1521/pedi.2013.27.1.3>

- Sharp, C., Pane, H., Ha, C., Venta, A., Patel, A. B., Sturek, J., & Fonagy, P. (2011). Theory of Mind and Emotion Regulation Difficulties in Adolescents With Borderline Traits. *Journal of the American Academy of Child & Adolescent Psychiatry*, 50(6), 563–573.e1. <https://doi.org/10.1016/j.jaac.2011.01.017>
- Sharp, C., Wright, A. G. C., Fowler, J. C., Frueh, B. C., Allen, J. G., Oldham, J., & Clark, L. A. (2015). The structure of personality pathology: Both general ('g') and specific ('s') factors? *Journal of Abnormal Psychology*, 124(2), 387–398. <https://doi.org/10.1037/abn0000033>
- Sheehy, M., Goldsmith, L., & Charles, E. (1980). A comparative study of borderline patients in a psychiatric outpatient clinic. *The American Journal of Psychiatry*, 137(11), 1374–1379. <https://doi.org/10.1176/ajp.137.11.1374>
- Shergill, S. S., Bays, P. M., Frith, C. D., & Wolpert, D. M. (2003). Two Eyes for an Eye: The Neuroscience of Force Escalation. *Science*, 301(5630), 187–187. <https://doi.org/10.1126/science.1085327>
- Shevlin, M., Murphy, J., Read, J., Mallett, J., Adamson, G., & Houston, J. E. (2011). Childhood adversity and hallucinations: A community-based study using the National Comorbidity Survey Replication. *Social Psychiatry and Psychiatric Epidemiology*, 46(12), 1203–1210. <https://doi.org/10.1007/s00127-010-0296-x>
- Siever, L. J., & Davis, K. L. (1991). A psychobiological perspective on the personality disorders. *American Journal of Psychiatry*, 148(12), 1647–1658. <https://doi.org/10.1176/ajp.148.12.1647>
- Skodol, A. E., Gunderson, J. G., Pfohl, B., Widiger, T. A., Livesley, W. J., & Siever, L. J. (2002). The borderline diagnosis I: Psychopathology, comorbidity, and personality structure. *Biological Psychiatry*, 51(12), 936–950. [https://doi.org/10.1016/S0006-3223\(02\)01324-0](https://doi.org/10.1016/S0006-3223(02)01324-0)
- Slotema, C. W., Bayrak, H., Linszen, M. M. J., Deen, M., & Sommer, I. E. C. (2019). Hallucinations in patients with borderline personality disorder: Characteristics, severity, and relationship with schizotypy and loneliness. *Acta Psychiatrica Scandinavica*, 139(5), 434–442. <https://doi.org/10.1111/acps.13012>
- Slotema, C. W., Blom, J. D., Niemantsverdriet, M. B. A., & Sommer, I. E. C. (2018). Auditory Verbal Hallucinations in Borderline Personality Disorder and the Efficacy

- of Antipsychotics: A Systematic Review. *Frontiers in Psychiatry*, 9, 347. <https://doi.org/10.3389/fpsyt.2018.00347>
- Slotema, C. W., Daalman, K., Blom, J. D., Dieren, K. M., Hoek, H. W., & Sommer, I. E. C. (2012). Auditory verbal hallucinations in patients with borderline personality disorder are similar to those in schizophrenia. *Psychological Medicine*, 42(9), 1873–1878. <https://doi.org/10.1017/S0033291712000165>
- Slotema, C. W., Niemantsverdriet, M. B. A., Blom, J. D., Van Der Gaag, M., Hoek, H. W., & Sommer, I. E. C. (2017). Suicidality and hospitalisation in patients with borderline personality disorder who experience auditory verbal hallucinations. *European Psychiatry*, 41(1), 47–52. <https://doi.org/10.1016/j.eurpsy.2016.10.003>
- Soares-Weiser, K., Maayan, N., Bergman, H., Davenport, C., Kirkham, A. J., Grabowski, S., & Adams, C. E. (2015). First rank symptoms for schizophrenia. *Cochrane Database of Systematic Reviews*. <https://doi.org/10.1002/14651858.CD010653.pub2>
- Sommer, I. E. (2010). The continuum hypothesis of psychosis: David's criticisms are timely: A commentary on 'Why we need more debate on whether psychotic symptoms lie on a continuum with normality' by David (). *Psychological Medicine*, 40(12), 1959–1961. <https://doi.org/10.1017/S0033291710000218>
- Sommer, I. E. C., Daalman, K., Rietkerk, T., Dieren, K. M., Bakker, S., Wijkstra, J., & Boks, M. P. M. (2010). Healthy individuals with auditory verbal hallucinations; who are they? Psychiatric assessments of a selected sample of 103 subjects. *Schizophrenia Bulletin*, 36(3), 633–641. <https://doi.org/10.1093/schbul/sbn130>
- Southward, M. W., Cheavens, J. S., & Cocco, E. F. (2023). Defining the *p*-factor: An empirical test of five leading theories. *Psychological Medicine*, 53(7), 2732–2743. <https://doi.org/10.1017/S0033291722001635>
- Southwick, S. M., Yehuda, R., & Giller, E. L. (1995). Psychological dimensions of depression in borderline personality disorder. *The American Journal of Psychiatry*, 152(5), 789–791. <https://doi.org/10.1176/ajp.152.5.789>
- Spauwen, J., Krabbendam, L., Lieb, R., Wittchen, H.-U., & Van Os, J. (2006). Impact of psychological trauma on the development of psychotic symptoms: Relationship with psychosis proneness. *British Journal of Psychiatry*, 188(6), 527–533. <https://doi.org/10.1192/bjp.bp.105.011346>

- Spearman, C. (1904). 'General Intelligence,' Objectively Determined and Measured. *The American Journal of Psychology*, 15(2), 201. <https://doi.org/10.2307/1412107>
- Spielberger, C. D. (1983). *State-Trait Anxiety Inventory for Adults* [dataset]. <https://doi.org/10.1037/t06496-000>
- Spitzer, C., Jelinek, L., Baumann, E., Benecke, C., & Schmidt, A. F. (2021). Negative self-conscious emotions in women with borderline personality disorder as assessed by an Implicit Association Test. *Personality Disorders*, 12(5), 456–465. <https://doi.org/10.1037/per0000467>
- Stanton, K. J., Denietolis, B., Goodwin, B. J., & Dvir, Y. (2020). Childhood Trauma and Psychosis. *Child and Adolescent Psychiatric Clinics of North America*, 29(1), 115–129. <https://doi.org/10.1016/j.chc.2019.08.004>
- Stefanis, N. C., Hanssen, M., Smirnis, N. K., Avramopoulos, D. A., Evdokimidis, I. K., Stefanis, C. N., Verdoux, H., & Van Os, J. (2002). Evidence that three dimensions of psychosis have a distribution in the general population. *Psychological Medicine*, 32(2), 347–358. <https://doi.org/10.1017/S0033291701005141>
- Stern, A. (1938). *Psychoanalytic investigation of and therapy in the borderline group of neuroses*. 7, 467–489.
- Stiglmayr, C. E., Ebner-Priemer, U. W., Bretz, J., Behm, R., Mohse, M., Lammers, C.-H., Anghelescu, I.-G., Schmahl, C., Schlotz, W., Kleindienst, N., & Bohus, M. (2008). Dissociative symptoms are positively related to stress in borderline personality disorder. *Acta Psychiatrica Scandinavica*, 0(0), 071120005752001-??? <https://doi.org/10.1111/j.1600-0447.2007.01126.x>
- Stiglmayr, C. E., Shapiro, D. A., Stieglitz, R. D., Limberger, M. F., & Bohus, M. (2001). Experience of aversive tension and dissociation in female patients with borderline personality disorder—A controlled study. *Journal of Psychiatric Research*, 35(2), 111–118. [https://doi.org/10.1016/S0022-3956\(01\)00012-7](https://doi.org/10.1016/S0022-3956(01)00012-7)
- Strachan, L. P., Paulik, G., Preece, D. A., & McEvoy, P. M. (2023). Pathways from trauma to unusual perceptual experiences: Modelling the roles of insecure attachment, negative affect, emotion regulation and dissociation. *Psychology and Psychotherapy: Theory, Research and Practice*, papt.12486. <https://doi.org/10.1111/papt.12486>

- Strauss, G. P., Raugh, I. M., Mittal, V. A., Gibb, B. E., & Coles, M. E. (2018). Bullying victimization and perpetration in a community sample of youth with psychotic like experiences. *Schizophrenia Research*, 195, 534–536. <https://doi.org/10.1016/j.schres.2017.08.056>
- Styron, T., Utter, L., & Davidson, L. (2017). The hearing voices network: Initial lessons and future directions for mental health professionals and Systems of Care. *Psychiatric Quarterly*, 88(4), 769–785. <https://doi.org/10.1007/s11126-017-9491-1>
- Suetani, S., Baker, A., Garner, K., Cosgrove, P., Mackay-Sim, M., Siskind, D., Murray, G. K., Scott, J. G., & Kesby, J. P. (2022). Impairments in goal-directed action and reversal learning in a proportion of individuals with psychosis. *Cognitive, Affective & Behavioral Neuroscience*, 22(6), 1390–1403. <https://doi.org/10.3758/s13415-022-01026-8>
- Sulzer, S. H. (2015). Does “difficult patient” status contribute to de facto demedicalization? The case of borderline personality disorder. *Social Science & Medicine*, 142, 82–89. <https://doi.org/10.1016/j.socscimed.2015.08.008>
- Sulzer, S. H., Jackson, G., & Yang, A. (2016). Borderline Personality Disorder and the Biomedical Mismatch. In B. L. Perry (Ed.), *Advances in Medical Sociology* (Vol. 17, pp. 263–290). Emerald Group Publishing Limited. <https://doi.org/10.1108/S1057-629020160000017011>
- Sun, P., Alvarez-Jimenez, M., Simpson, K., Lawrence, K., Peach, N., & Bendall, S. (2018). Does dissociation mediate the relationship between childhood trauma and hallucinations, delusions in first episode psychosis? *Comprehensive Psychiatry*, 84, 68–74. <https://doi.org/10.1016/j.comppsy.2018.04.004>
- Tabachnick, B. G., Fidell, L. S., & Ullman, J. B. (2019). *Using multivariate statistics* (Seventh edition). Pearson.
- Taminau, J., Lazar, C., Meganck, S., & Nowé, A. (2014). Comparison of Merging and Meta-Analysis as Alternative Approaches for Integrative Gene Expression Analysis. *ISRN Bioinformatics*, 2014, 1–7. <https://doi.org/10.1155/2014/345106>
- Tarullo, A. R., & Gunnar, M. R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behavior*, 50(4), 632–639. <https://doi.org/10.1016/j.yhbeh.2006.06.010>

- Teicher, M. H., Samson, J. A., Anderson, C. M., & Ohashi, K. (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience*, 17(10), 652–666. <https://doi.org/10.1038/nrn.2016.111>
- Teufel, C., Dakin, S. C., & Fletcher, P. C. (2018). Prior object-knowledge sharpens properties of early visual feature-detectors. *Scientific Reports*, 8(1), 10853. <https://doi.org/10.1038/s41598-018-28845-5>
- Teufel, C., Subramaniam, N., Dobler, V., Perez, J., Finnemann, J., Mehta, P. R., Goodyer, I. M., & Fletcher, P. C. (2015). Shift toward prior knowledge confers a perceptual advantage in early psychosis and psychosis prone healthy individuals. *Proceedings of the National Academy of Sciences*, 112(43), 13401–13406. <https://doi.org/10.1073/pnas.1503916112>
- Thoma, R. J., Hanlon, F. M., Moses, S. N., Edgar, J. C., Huang, M., Weisend, M. P., Irwin, J., Sherwood, A., Paulson, K., Bustillo, J., Adler, L. E., Miller, G. A., & Cañive, J. M. (2003). Lateralization of Auditory Sensory Gating and Neuropsychological Dysfunction in Schizophrenia. *American Journal of Psychiatry*, 160(9), 1595–1605. <https://doi.org/10.1176/appi.ajp.160.9.1595>
- Thompson, A., Marwaha, S., Nelson, B., Wood, S. J., McGorry, P. D., Yung, A. R., & Lin, A. (2016). Do affective or dissociative symptoms mediate the association between childhood sexual trauma and transition to psychosis in an ultra-high risk cohort? *Psychiatry Research*, 236, 182–185. <https://doi.org/10.1016/j.psychres.2016.01.017>
- Thompson, A., Nelson, B., Bechdolf, A., Chanen, A. M., Domingues, I., Mcdougall, E., & Yung, A. R. (2012). Borderline Personality Features And Development Of Psychosis In An ‘ultra High Risk’ (Uhr) Population: A Case Control Study. *Early Intervention in Psychiatry*, 6(3), 247–255. Scopus. <https://doi.org/10.1111/j.1751-7893.2012.00365.x>
- Thompson, A., Nelson, B., McNab, C., Simmons, M., Leicester, S., McGorry, P. D., Bechdolf, A., & Yung, A. R. (2010). Psychotic symptoms with sexual content in the “ultra high risk” for psychosis population: Frequency and association with sexual trauma. *Psychiatry Research*, 177(1–2), 84–91. <https://doi.org/10.1016/j.psychres.2010.02.011>

- Thornton, O. (2023). *Borderline Personality Disorder Diagnosis and Assessment: A Comprehensive Review of Criteria and Tools*. <https://doi.org/10.5281/ZENODO.7737910>
- Toh, W. L., Moseley, P., & Fernyhough, C. (2022). Hearing voices as a feature of typical and psychopathological experience. *Nature Reviews Psychology*, 1(2), 72–86. <https://doi.org/10.1038/s44159-021-00013-z>
- Trull, T. J., Distel, M. A., & Carpenter, R. W. (2011). DSM-5 Borderline Personality Disorder: At the Border Between a Dimensional and a Categorical View. *Current Psychiatry Reports*, 13(1), 43–49. <https://doi.org/10.1007/s11920-010-0170-2>
- Tsang, A., Bucci, S., Branitsky, A., Kaptan, S., Rafiq, S., Wong, S., Berry, K., & Varese, F. (2021). The relationship between appraisals of voices (auditory verbal hallucinations) and distress in voice-hearers with schizophrenia-spectrum diagnoses: A meta-analytic review. *Schizophrenia Research*, 230, 38–47. <https://doi.org/10.1016/j.schres.2021.02.013>
- Tschoeke, S., Steinert, T., Flammer, E., & Uhlmann, C. (2014). Similarities and differences in borderline personality disorder and schizophrenia with voice hearing. *The Journal of Nervous and Mental Disease*, 202(7), 544–549. <https://doi.org/10.1097/NMD.0000000000000159>
- Tuineag, M., Therman, S., Lindgren, M., Rouanet, M., Nahon, S., Bertrand, L., Saury, S., Renaud, S., Beaulieu, S., & Linnaranta, O. (2020). Dissociative symptoms as measured by the Cambridge Depersonalization Scale in patients with a bipolar disorder. *Journal of Affective Disorders*, 263, 187–192. <https://doi.org/10.1016/j.jad.2019.11.137>
- Tyrer, P., Mulder, R., Kim, Y.-R., & Crawford, M. J. (2019). The Development of the ICD-11 Classification of Personality Disorders: An Amalgam of Science, Pragmatism, and Politics. *Annual Review of Clinical Psychology*, 15(1), 481–502. <https://doi.org/10.1146/annurev-clinpsy-050718-095736>
- Vaessen, T., Reininghaus, U., Van Aabel, E., Beijer-Klippel, A., Steinhart, H., Myin-Germeys, I., & Waltz, J. (2023). Neural correlates of daily-life affective stress reactivity in early psychosis: A study combining functional MRI and experience

- sampling methodology. *Schizophrenia Research*, 255, 93–101. <https://doi.org/10.1016/j.schres.2023.03.038>
- Van Bork, R., Epskamp, S., Rhemtulla, M., Borsboom, D., & Van Der Maas, H. L. J. (2017). What is the p -factor of psychopathology? Some risks of general factor modeling. *Theory & Psychology*, 27(6), 759–773. <https://doi.org/10.1177/0959354317737185>
- Van Dael, F., Versmissen, D., Janssen, I., Myin-Germeys, I., Van Os, J., & Krabbendam, L. (2006). Data gathering: Biased in psychosis? *Schizophrenia Bulletin*, 32(2), 341–351. <https://doi.org/10.1093/schbul/sbj021>
- Van Dam, D. S., Van Der Ven, E., Velthorst, E., Selten, J. P., Morgan, C., & De Haan, L. (2012). Childhood bullying and the association with psychosis in non-clinical and clinical samples: A review and meta-analysis. *Psychological Medicine*, 42(12), 2463–2474. <https://doi.org/10.1017/S0033291712000360>
- Van Den Berg, D., Tolmeijer, E., Jongeneel, A., Staring, A. B. P., Palstra, E., Van Der Gaag, M., & Hardy, A. (2023). Voice phenomenology as a mirror of the past. *Psychological Medicine*, 53(7), 2954–2962. <https://doi.org/10.1017/S0033291721004955>
- Van Der Zwaard, R., & Polak, M. A. (2001). Pseudohallucinations: A pseudoconcept? A review of the validity of the concept, related to associate symptomatology. *Comprehensive Psychiatry*, 42(1), 42–50. <https://doi.org/10.1053/comp.2001.19752>
- Van Erp, T. G. M., Hibar, D. P., Rasmussen, J. M., Glahn, D. C., Pearlson, G. D., Andreassen, O. A., Agartz, I., Westlye, L. T., Haukvik, U. K., Dale, A. M., Melle, I., Hartberg, C. B., Gruber, O., Kraemer, B., Zilles, D., Donohoe, G., Kelly, S., McDonald, C., Morris, D. W., ... Turner, J. A. (2016). Subcortical brain volume abnormalities in 2028 individuals with schizophrenia and 2540 healthy controls via the ENIGMA consortium. *Molecular Psychiatry*, 21(4), 547–553. <https://doi.org/10.1038/mp.2015.63>
- Van Loon, A. M., Fahrenfort, J. J., Van Der Velde, B., Lirk, P. B., Vulink, N. C. C., Hollmann, M. W., Steven Scholte, H., & Lamme, V. A. F. (2016). NMDA Receptor Antagonist Ketamine Distorts Object Recognition by Reducing Feedback to Early

- Visual Cortex. *Cerebral Cortex*, 26(5), 1986–1996.
<https://doi.org/10.1093/cercor/bhv018>
- Van Os, J. (2003). Is there a continuum of psychotic experiences in the general population? *Epidemiology and Psychiatric Sciences*, 12(4), 242–252.
<https://doi.org/10.1017/S1121189X00003067>
- Varese, F., Barkus, E., & Bentall, R. P. (2012). Dissociation mediates the relationship between childhood trauma and hallucination-proneness. *Psychological Medicine*, 42(5), 1025–1036. <https://doi.org/10.1017/S0033291711001826>
- Velthorst, E., Nelson, B., O'Connor, K., Mossaheb, N., De Haan, L., Bruxner, A., Simmons, M. B., Yung, A. R., & Thompson, A. (2013). History of trauma and the association with baseline symptoms in an Ultra-High Risk for psychosis cohort. *Psychiatry Research*, 210(1), 75–81. <https://doi.org/10.1016/j.psychres.2013.06.007>
- Vonderlin, R., Kleindienst, N., Alpers, G. W., Bohus, M., Lyssenko, L., & Schmahl, C. (2018). Dissociation in victims of childhood abuse or neglect: A meta-analytic review. *Psychological Medicine*, 48(15), 2467–2476.
<https://doi.org/10.1017/S0033291718000740>
- Walker, E. F., Brennan, P. A., Esterberg, M., Brasfield, J., Pearce, B., & Compton, M. T. (2010). Longitudinal changes in cortisol secretion and conversion to psychosis in at-risk youth. *Journal of Abnormal Psychology*, 119(2), 401–408.
<https://doi.org/10.1037/a0018399>
- Walker, E. F., Trotman, H. D., Pearce, B. D., Addington, J., Cadenhead, K. S., Cornblatt, B. A., Heinssen, R., Mathalon, D. H., Perkins, D. O., Seidman, L. J., Tsuang, M. T., Cannon, T. D., McGlashan, T. H., & Woods, S. W. (2013). Cortisol Levels and Risk for Psychosis: Initial Findings from the North American Prodrome Longitudinal Study. *Biological Psychiatry*, 74(6), 410–417.
<https://doi.org/10.1016/j.biopsych.2013.02.016>
- Walter, M., Bureau, J.-F., Holmes, B. M., Bertha, E. A., Hollander, M., Wheelis, J., Brooks, N. H., & Lyons-Ruth, K. (2008). Cortisol response to interpersonal stress in young adults with borderline personality disorder: A pilot study. *European Psychiatry: The Journal of the Association of European Psychiatrists*, 23(3), 201–204. <https://doi.org/10.1016/j.eurpsy.2007.12.003>

- Wang, Y.-Y., Shi, H.-S., Liu, W.-H., Yan, C., Wang, Y., Chiu, C., So, S. H., Lui, S. S. Y., Cheung, E. F. C., & Chan, R. C. K. (2017). Invariance of factor structure of the 21-item Peters et al. Delusions Inventory (PDI-21) over time and across samples. *Psychiatry Research*, 254, 190–197. <https://doi.org/10.1016/j.psychres.2017.04.053>
- Waters, F., Chiu, V., Atkinson, A., & Blom, J. D. (2018). Severe Sleep Deprivation Causes Hallucinations and a Gradual Progression Toward Psychosis With Increasing Time Awake. *Frontiers in Psychiatry*, 9, 303. <https://doi.org/10.3389/fpsyt.2018.00303>
- Watts, J. (2019). Problems with the ICD-11 classification of personality disorder. *The Lancet Psychiatry*, 6(6), 461–463. [https://doi.org/10.1016/S2215-0366\(19\)30127-0](https://doi.org/10.1016/S2215-0366(19)30127-0)
- Weiner, L., Perroud, N., & Weibel, S. (2019). Attention Deficit Hyperactivity Disorder And Borderline Personality Disorder In Adults: A Review Of Their Links And Risks. *Neuropsychiatric Disease and Treatment*, 15, 3115–3129. <https://doi.org/10.2147/NDT.S192871>
- Westen, D. (1991). Cognitive-behavioral interventions in the psychoanalytic psychotherapy of borderline personality disorders. *Clinical Psychology Review*, 11(3), 211–230. [https://doi.org/10.1016/0272-7358\(91\)90101-Y](https://doi.org/10.1016/0272-7358(91)90101-Y)
- Westwater, M. L., Mancini, F., Shapleske, J., Serfontein, J., Ernst, M., Ziauddeen, H., & Fletcher, P. C. (2021). Dissociable hormonal profiles for psychopathology and stress in anorexia and bulimia nervosa. *Psychological Medicine*, 51(16), 2814–2824. <https://doi.org/10.1017/S0033291720001440>
- Whisman, M. A. (2010). Loneliness and the metabolic syndrome in a population-based sample of middle-aged and older adults. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, 29(5), 550–554. <https://doi.org/10.1037/a0020760>
- Whitfield, C. L., Dube, S. R., Felitti, V. J., & Anda, R. F. (2005). Adverse childhood experiences and hallucinations. *Child Abuse & Neglect*, 29(7), 797–810. <https://doi.org/10.1016/j.chiabu.2005.01.004>
- WHO ASSIST Working Group. (2002). The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST): Development, reliability and feasibility: ASSIST: development, reliability and feasibility. *Addiction*, 97(9), 1183–1194. <https://doi.org/10.1046/j.1360-0443.2002.00185.x>

- Wigman, J. T. W., Van Nierop, M., Vollebergh, W. A. M., Lieb, R., Beesdo-Baum, K., Wittchen, H.-U., & Van Os, J. (2012). Evidence That Psychotic Symptoms Are Prevalent in Disorders of Anxiety and Depression, Impacting on Illness Onset, Risk, and Severity—Implications for Diagnosis and Ultra-High Risk Research. *Schizophrenia Bulletin*, 38(2), 247–257. <https://doi.org/10.1093/schbul/sbr196>
- Wilton, R., Hansen, S., & Hall, E. (2017). Disabled people, medical inadmissibility, and the differential politics of immigration: Disability and medical inadmissibility. *The Canadian Geographer / Le Géographe Canadien*, 61(3), 389–400. <https://doi.org/10.1111/cag.12361>
- Wing, J. K., Cooper, J. E., Sartorius, N., Wing, J. K., & Cooper, J. E. (1975). *Measurement and classification of psychiatric symptoms: An instruction manual for the PSE and Catego program* (Reprinted). Cambridge Univ. Press.
- Wolf, E. J., Harrington, K. M., Clark, S. L., & Miller, M. W. (2013). Sample Size Requirements for Structural Equation Models: An Evaluation of Power, Bias, and Solution Propriety. *Educational and Psychological Measurement*, 73(6), 913–934. <https://doi.org/10.1177/0013164413495237>
- Wolfe, J., Kimerling, R., Brown, P. J., Chrestman, K. R., & Levin, K. (1996). *Life Stressor Checklist—Revised* [dataset]. <https://doi.org/10.1037/t04534-000>
- Wolke, D., Lereya, S. T., Fisher, H. L., Lewis, G., & Zammit, S. (2014). Bullying in elementary school and psychotic experiences at 18 years: A longitudinal, population-based cohort study. *Psychological Medicine*, 44(10), 2199–2211. <https://doi.org/10.1017/S0033291713002912>
- World Health Organization. (2016). *International statistical classification of diseases and related health problems* (10th ed.). <https://icd.who.int/browse10/2016/en>
- World Health Organization. (2019). *International statistical classification of diseases and related health problems* (11th ed.). <https://icd.who.int/>
- Yamamoto, T. (2022). The relationship between central fatigue and Attention Deficit/Hyperactivity Disorder of the inattentive type. *Neurochemical Research*, 47(9), 2890–2898. <https://doi.org/10.1007/s11064-022-03693-y>

- Yang, M., Coid, J., & Tyrer, P. (2010). Personality pathology recorded by severity: National survey. *British Journal of Psychiatry*, 197(3), 193–199. <https://doi.org/10.1192/bjp.bp.110.078956>
- Yee, L., Korner, A. J., McSwiggan, S., Meares, R. A., & Stevenson, J. (2005). Persistent hallucinosis in borderline personality disorder. *Comprehensive Psychiatry*, 46(2), 147–154. <https://doi.org/10.1016/j.comppsy.2004.07.032>
- Yen, S., Shea, M. T., Battle, C. L., Johnson, D. M., Zlotnick, C., Dolan-Sewell, R., Skodol, A. E., Grilo, C. M., Gunderson, J. G., Sanislow, C. A., Zanarini, M. C., Bender, D. S., Rettew, J. B., & McGLASHAN, T. H. (2002). TRAUMATIC EXPOSURE AND POSTTRAUMATIC STRESS DISORDER IN BORDERLINE, SCHIZOTYPAL, AVOIDANT, AND OBSESSIVE-COMPULSIVE PERSONALITY DISORDERS: FINDINGS FROM THE COLLABORATIVE LONGITUDINAL PERSONALITY DISORDERS STUDY: *The Journal of Nervous and Mental Disease*, 190(8), 510–518. <https://doi.org/10.1097/00005053-200208000-00003>
- Zachar, P., Krueger, R. F., & Kendler, K. S. (2016). Personality disorder in DSM-5: An oral history. *Psychological Medicine*, 46(1), 1–10. <https://doi.org/10.1017/S0033291715001543>
- Zakzanis, K. K., & Hansen, K. T. (1998). Dopamine D2 densities and the schizophrenic brain. *Schizophrenia Research*, 32(3), 201–206. [https://doi.org/10.1016/S0920-9964\(98\)00041-3](https://doi.org/10.1016/S0920-9964(98)00041-3)
- Zanarini, M. C., Frankenburg, F. R., Hennen, J., Reich, D. B., & Silk, K. R. (2006). Prediction of the 10-Year Course of Borderline Personality Disorder. *American Journal of Psychiatry*, 163(5), 827–832. <https://doi.org/10.1176/ajp.2006.163.5.827>
- Zanarini, M. C., Frankenburg, F. R., Wedig, M. M., & Fitzmaurice, G. M. (2013). Cognitive Experiences Reported by Patients With Borderline Personality Disorder and Axis II Comparison Subjects: A 16-Year Prospective Follow-Up Study. *American Journal of Psychiatry*, 170(6), 671–679. <https://doi.org/10.1176/appi.ajp.2013.13010055>
- Zanarini, M. C., Gunderson, J. G., & Frankenburg, F. R. (1990). Cognitive features of borderline personality disorder. *The American Journal of Psychiatry*, 147(1), 57–63. <https://doi.org/10.1176/ajp.147.1.57>

- Zanarini, M. C., Gunderson, J. G., Frankenburg, F. R., & Chauncey, D. L. (1989). The Revised Diagnostic Interview for Borderlines: Discriminating BPD from other Axis II Disorders. *Journal of Personality Disorders*, 3(1), 10–18. <https://doi.org/10.1521/pedi.1989.3.1.10>
- Zanarini, M. C., Vujanovic, A. A., Parachini, E. A., Boulanger, J. L., Frankenburg, F. R., & Hennen, J. (2003). A Screening Measure for BPD: The McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD). *Journal of Personality Disorders*, 17(6), 568–573. <https://doi.org/10.1521/pedi.17.6.568.25355>
- Zanarini, M. C., Williams, A. A., Lewis, R. E., Reich, R. B., Vera, S. C., Marino, M. F., Levin, A., Yong, L., & Frankenburg, F. R. (1997). Reported pathological childhood experiences associated with the development of borderline personality disorder. *The American Journal of Psychiatry*, 154(8), 1101–1106. <https://doi.org/10.1176/ajp.154.8.1101>
- Zetzsche, T., Frodl, T., Preuss, U. W., Schmitt, G., Seifert, D., Leinsinger, G., Born, C., Reiser, M., Möller, H.-J., & Meisenzahl, E. M. (2006). Amygdala Volume and Depressive Symptoms in Patients With Borderline Personality Disorder. *Biological Psychiatry*, 60(3), 302–310. <https://doi.org/10.1016/j.biopsych.2005.11.020>
- Zhang, X. Y., Zhou, D. F., Cao, L. Y., Wu, G. Y., & Shen, Y. C. (2005). Cortisol and Cytokines in Chronic and Treatment-Resistant Patients with Schizophrenia: Association with Psychopathology and Response to Antipsychotics. *Neuropsychopharmacology*, 30(8), 1532–1538. <https://doi.org/10.1038/sj.npp.1300756>
- Zimmerman, M., & Mattia, J. I. (1999). Axis I diagnostic comorbidity and borderline personality disorder. *Comprehensive Psychiatry*, 40(4), 245–252. [https://doi.org/10.1016/S0010-440X\(99\)90123-2](https://doi.org/10.1016/S0010-440X(99)90123-2)
- Zonnenberg, C., Niemantsverdriet, M. B. A., Blom, J. D., & Slotema, C. W. (2016). [Auditory verbal hallucinations in patients with borderline personality disorder]. *Tijdschrift Voor Psychiatrie*, 58(2), 122–129.

Appendix A Custom questionnaires

A.1 Screening questionnaires

A.1.1 Study 1

Participants were be asked to provide the following information:

1. Age (Textbox)
 - a. If < 18 or >65 → the session terminated → informed they are ineligible to take part.
 - b. If 18 or above and less than 66 → Q2
2. Gender
3. Have you been diagnosed with BPD?
 - a. If yes → potential BPD participant → MSI-BPD → Q4.
 - b. If no → Q4.
4. Do you identify with the diagnosis of BPD?
 - a. If yes:
 - i. If Q3 was No → Potential BPD participant → MSI-BPD → Q5.
 - ii. If Q3 was Yes → Q5 (*ascertains if individual identifies with BPD diagnosis*)
 - b. If no → potential control participant → Q5.
5. Are you currently diagnosed with any of the following conditions? Multi-choice: schizophrenia, bipolar disorder, schizoaffective disorder, delusional disorder (*Screening for additional diagnosis of psychotic disorder*)
 - a. If any are endorsed → ineligible
 - b. If none are endorsed → Q6 (*skip to Q7 for control participants*)

-
6. Are you current receiving or have previously received any of the following treatments of your BPD?
 - i. Psychological therapy?
 - a. If yes, please select: CBT, DBT, MBT, Trauma focussed CBT, Other
 - b. If no, next option
 - ii. Medication?
 - a. If yes, please select: antidepressants, antipsychotics, mood stabilisers, sedatives
 - b. If “antipsychotic” → Are you currently being treated with antipsychotic medication? (*ineligible for study but proceeding with screening*).
 7. Have you ever suffered with any of the following conditions? Multi-choice: depression, anxiety, OCD, PTSD, eating disorders, any other mental illness.
 - a. If control and “yes” → Have you been diagnosed with or needed to seek or take treatment for this condition → if yes, ineligible → Q8
 - b. If no → Q8
 8. Do you have a history of any of the following? (*For manual exclusion*)
 - a. A head injury
 - i. If yes, did you lose consciousness? For how long?
 - ii. Did you have a brain scan? If yes, did it show any injury?
 - iii. Did you require hospitalisation or treatment for the head injury?
 - b. Any neurological illness affecting the brain: e.g. stroke, multiple sclerosis, brain cancer
 - c. → Q9
 9. Psychosis screen (CAPE15)

A.1.2 Study 2

Participants were be asked to provide the following information:

1. Age (Textbox)
 - a. If <18 or >65 → the session terminated → informed they are ineligible to take part.
 - b. If 18 or above and less than 66 → Q2
2. Gender
3. What is your place of residence?
4. Have you been diagnosed with BPD?
 - a. If yes → BPD participant → Q5.
 - b. If no → Control participant → Q6
5. Have ever been diagnosed with any of the following conditions?
Multi-choice: depression, anxiety, OCD, PTSD, eating disorders, any other mental illness.
 - a. If OCD or PTSD and control participant → ineligible
6. Are you currently diagnosed with any of the following conditions? Multi-choice: schizophrenia, bipolar disorder, schizoaffective disorder, delusional disorder
(*Screening for additional diagnosis of psychotic disorder*)
 - a. If any are endorsed → ineligible
 - b. If none are endorsed → Q7
7. Do you have a history of any of the following? (*For manual exclusion*)
 - a. A head injury
 - i. If yes, did you lose consciousness? For how long?
 - ii. Did you have a brain scan? If yes, did it show any injury?
 - iii. Did you require hospitalisation or treatment for the head injury?
 - b. Any neurological illness affecting the brain: e.g. stroke, multiple sclerosis, brain cancer: If yes → ineligible
 - c. If no → Q8
8. Are you currently pregnant?
 - a. If yes → the session terminated → informed they are ineligible to take part

b. If no → Q9

9. Are you current receiving or have previously received any of the following treatments?

- i. Psychological therapy? (If yes, select: CBT, DBT, MBT, Trauma focussed CBT, Other)
- ii. Medication? (If yes, select: antidepressants, antipsychotics, mood stabilisers, sedatives)

If “antipsychotics” is selected → are you currently taking antipsychotic medication (this does not include antidepressants, mood stabilisers, or sedatives) → If yes: session terminated → informed they are ineligible to take part.

10. Psychosis screen (CAPE15)

Additional question for BPD participants (Displayed if Q3 = “Yes”)

11. At present are you receiving mental health care from (you can tick multiple options)

- i. My GP
- ii. Community mental health service
- iii. Community personality disorder service
- iv. Inpatient services- general
- v. Inpatient services- specialist personality disorder
- vi. Other: text box
- vii. None of the above

A.2 Psychotic experiences follow-up questionnaire

If participants endorsed symptoms on the PDI or CAPS, they were presented with a follow-up questionnaire, stating:

‘In the previous you indicated that you have experienced perceptual symptoms and/or unusual beliefs. We would like to better understand the patterns of these symptoms in different people.’

1. ‘Does anything appear to trigger or worsen these experiences?’

Yes/No and then options if yes (textbox options for more details)

Select all that apply:

- Stress
- Specific substances
- Specific medications
- Physical ill-health
- Dissociation
- Worsening anxiety
- Worsening mood
- In relation to your menstrual cycle (if applicable)
- Other (textbox)

Displayed if PDI symptoms endorsed:

‘‘You have said that you experience unusual beliefs (for example intense paranoia). Thinking about the symptom(s) that affect you the most, how frequently does this happen?:’

- Rarely or only under specific circumstances
- Occasionally (a few times a year perhaps)
- At least monthly

- At least weekly
 - At least daily (or continuously)
-
- How real do they feel? VAS 'Not at all' to 'Intensely real'
 - How much do you change how you live because of it? VAS 'Not at all' to 'make major changes to my life'.
 - After they stopped, do they still affect how you live? VAS 'Not at all' to 'made major changes to my life'.

Displayed if CAPS symptoms endorsed:

‘You have said that you experience perceptual symptoms (for example visual or auditory hallucinations. When they occur:’

- How long do they typically last?
 - A few minutes
 - A few hours
 - About a day
 - Up to a week
 - Up to a month
 - They are pretty much constant
- How real do they feel? VAS 'Not at all' to 'Intensely real'
- How much do you change how you live because of it? VAS 'Not at all' to 'make major changes to my life'.
- After they stopped, do they still affect how you live? VAS 'Not at all' to 'made major changes to my life.

Appendix B Phantom words task development

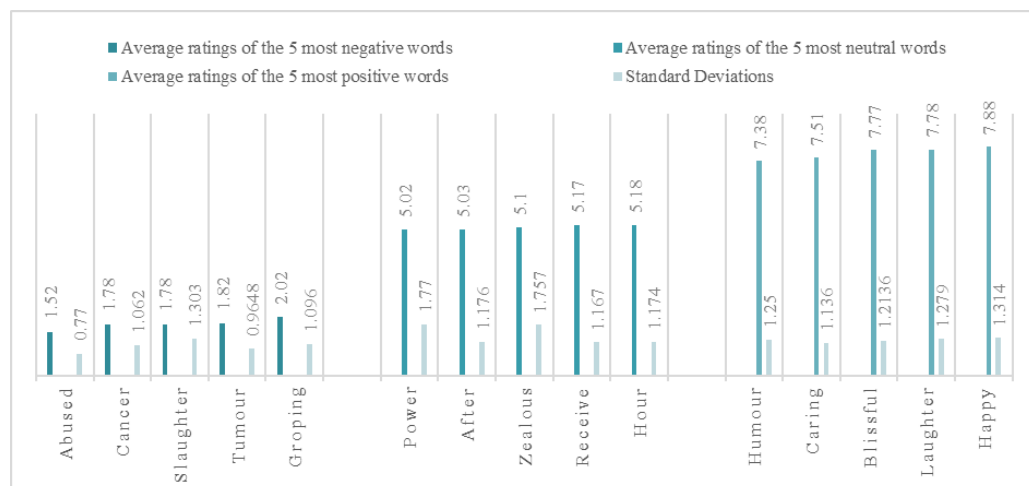
B.1 Demographics

Participant demographics of the emotional valence survey, and phantom word pilot studies one and two. m=male, f=female, d=diverse, y=yes, n=no

	Number of participants	Gender ratio (m:f:d)	Age mean(<i>sd</i>)	Age range (min:max)	Native speakers (y:n)
Emotional valence survey	162	25:127:10	42.2(12.5)	21-69	131:31
Phantom words - Pilot 1	31	7:22:2	33.35(8.9)	21-56	NA
Phantom words - Pilot 2	20	5:13:2	37.55(12.4)	20-57	NA

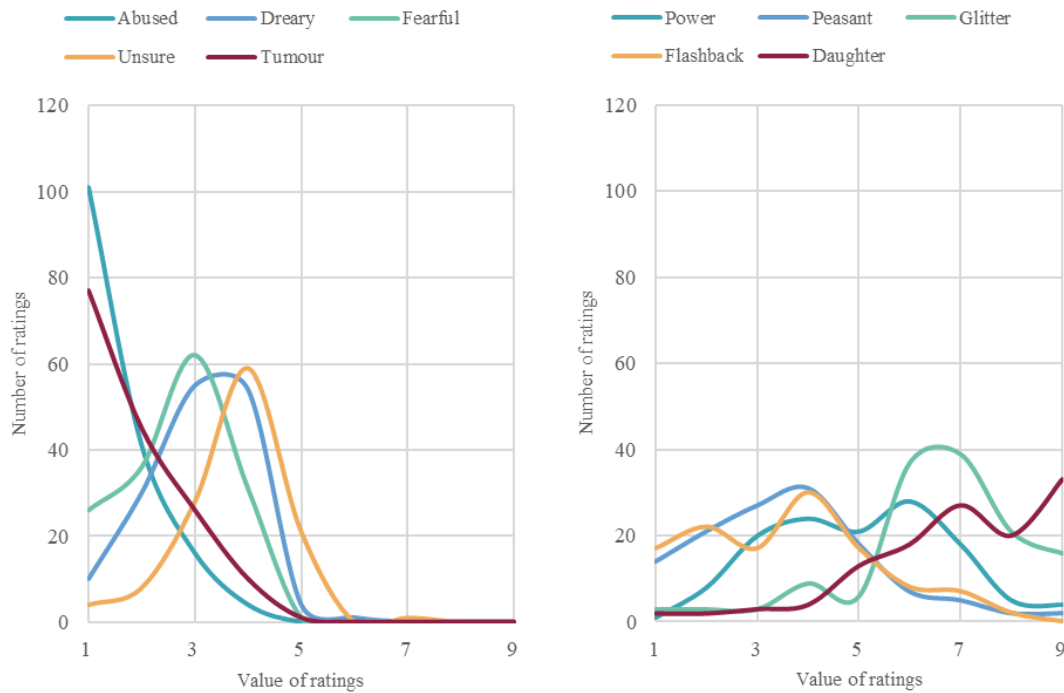
B.2 Emotional valence survey – example ratings

Average valence ratings and their standard deviations for the five most positively, neutrally, and negatively rated words.



B.3 Emotional valence survey – example distributions

Distribution of ratings for the 5 words with the highest consensus on their valence (i.e., lowest standard deviations: Abused=0.77; Tumour=0.96; Dreary=0.98; Unsure=0.99; Fearful=0.999) (left) and distribution of ratings for the 5 words with the lowest consensus on their valence (highest standard deviations: Daughter=1.88; Flashback=1.79; Glitter=1.77; Peasant=1.77; Power=1.77) (right).



B.4 Final phantom word pairs

Final selection of word pairs included in the phantom words task, with mean and standard deviation and sound-syllables of their respective stimuli.

*words added after initial emotional valence survey, mean and standard deviation displayed are of the Affective Norms for English language (Bradley and Lang, 1999)

**mean and standard deviation data inconclusive due to technical difficulties. The word was still included due to its reliable performance in both phantom word pilots.

Positive/Neutral	Mean	SD	Negative/Neutral	Mean	SD	Phantom word
Non-signal word pairs						
Curious*	6.08	1.63	Furious	2.77	1.34	Ur - Ious
Rightful	6.61	1.33	Spiteful	2.17	1.03	Ite - Full
Rightful	6.61	1.33	Frightful	3.10	1.31	Ite - Full
Rightful	6.61	1.33	Hateful	2.11	1.22	Ite - Full
Cupid	6.01	1.38	Stupid	2.79	1.34	U - Pid
Proudly	6.69	1.41	Loudly	4.08	1.37	Oud - Ly
Lucky	6.87	1.34	Yucky	3.29	1.21	Uh - Cky
Lucky	6.87	1.34	Ugly*	2.43	1.27	Uh - Cky
Kissing	6.98	1.35	Missing	2.94	1.21	Iss - Ing
Amused	7.05	1.08	Abused	1.52	0.77	Uh - Used
Pleasure	7.35	1.20	Pressure*	3.50	1.19	Peh - Jure
True signal word pairs						
Assure	6.44	1.32	Unsure	3.74	0.99	Ah - Sure
Humour	7.38	1.25	Tumour	1.61	0.86	Hugh - Mah
Rightful	5.96	1.55	Frightful	3.10	1.31	Right - Full
Eject	3.89	1.19	Reject	2.82	1.22	Ee - Ject
Arrayed	4.77	1.40	Afraid	2.70	1.17	A - rrayed
Armful*	5.50	1.79	Harmful	2.52	1.25	Arm - Full
Image**	N/A	N/A	Damage*	3.05	1.65	Im - age
Discuss	5.95	1.50	Disgust	2.51	1.23	Diss - Cuss
Pleasant	6.91	1.17	Peasant	3.64	1.77	Peh - Zent

Appendix C Order of tasks and questionnaires

C.1 Study 1

Order of questionnaire measures as presented to participants of Study 1 on the experiment platform Gorilla. The order was not counterbalanced between participants.

1. Information Sheet
2. Participant consent
3. Demographics
4. PDI (Peter's Delusions Inventory)
5. CAPS (Cardiff Anomalous Perceptions Scale - task version)
 - a. (Psychotic experiences follow-up custom addition)
6. DES-II (Dissociative Experiences Scale)
7. AQ-10 (Autism Spectrum Quotient)
8. CTQ (Childhood Trauma Questionnaire)
9. PCL-5 (PTSD checklist for DSM-V)
10. QIDS-SR16 (Quick inventory of depressive symptoms)
11. GAD-7 (Generalised anxiety disorder questionnaire)
12. Two-Tone Image Task
13. ASSIST (Alcohol, Smoking and Substance Involvement Screening Test)
14. Phantom Words Task
15. Debrief Sheet

C.2 Study 2

Order of questionnaire measures as presented to participants of Study 2. The order was not counterbalanced between participants.

* The emotion recognition task was included as an additional variable before and after stress. Due to technical difficulties analysis of the data is still pending and could not be considered within the scope of this dissertation.

** Additional measures included in collaborative research with Dr Jeremy Solly for into impulsivity and compulsivity in BPD, not intended for inclusion in thesis.

1. Stress Check-in
2. Demographics
3. Physical Health History
4. Current Treatment
5. CADSS (Dissociation)
6. PSI (Psychomimetic)
7. STAI (Anxiety)
8. Two-tone image Task
9. Go/No-Go Task**
10. Emotion Recognition Task*
11. Stress Check-in
12. Borderline Symptoms List
13. SUPPS-P (Impulsivity)**
14. CHI-T (Compulsivity)**
15. BDI (Depression)
16. CAPS + Custom (Hallucinations)
17. PDI + Custom (Delusions)
18. DEMO (Dissociation Trait)
19. Stress Check-in

Break

20. CTQ (Childhood Trauma)
21. LSC-R (Life Stressors)
22. PCL-5 (PTSD)
23. Stress Check-in
24. **Stress induction**
25. Stress Check-in
26. Go/No-Go Task**
27. Emotion Recognition Task*
28. Two-tone image Task
29. PSI
30. CADSS
31. Stress Check-in

Appendix D Additional statistics

D.1 CAPS symptom prevalence comparisons

D.1.1 Study 1 – BPD and Control

Comparison of prevalences of perceptual experiences in the BPD and control group of Study 1, for the nine CAPS symptom categories (a), other relevant symptom groups (as often reported in the literature) (b), and Schneider's first rank symptoms (FRS) of psychosis (c). Differences assessed using Pearson's Chi-squared test with Yates' continuity correction, p -values adjusted with Bonferroni correction for multiple comparison. Degrees of freedom of all $\chi^2_{df=1}$.

a)	relevant items	BPD (<i>n</i> =54)	Control (<i>n</i> =68)	χ^2	
Having a Nonshared Sensory Experience	13, 29, 31, 32	30 (56%)	2 (3%)	$\chi^2_{\text{M}}=41.506, p<.001$	
Unusual or Distorted Sensory Experience	5, 16, 25, 26, 30	36 (67%)	12 (18%)	$\chi^2_{\text{M}}=29.46, p<.001$	
Sensory Experience from Unexplained Source	4, 6, 8, 12, 14, 28	35 (65%)	13 (19%)	$\chi^2_{\text{M}}=25.562, p<.001$	
Distortion of Form (Own Body and World)	9, 10, 19, 22	35 (65%)	11 (16%)	$\chi^2_{\text{M}}=29.425, p<.001$	
Changes in Levels of Sensory Intensity	1, 18, 20, 21, 23	45 (83%)	23 (34%)	$\chi^2_{\text{M}}=29.356, p<.001$	
Sensory Flooding	15, 17	37 (69%)	6 (9%)	$\chi^2_{\text{M}}=45.751, p<.001$	
Thought Echo and Hearing Thoughts	3, 7	24 (44%)	5 (7%)	$\chi^2_{\text{M}}=21.637, p<.001$	
Temporal Lobe Abnormalities	2, 10, 24, 27	38 (70%)	16 (24%)	$\chi^2_{\text{M}}=26.094, p<.001$	
b)	Auditory Verbal Hallucinations	11,13, 28	21 (39%)	5 (7%)	$\chi^2_{\text{M}}=29.425, p<.001$
	Visual distortion	4, 26, 19	32 (59%)	6 (9%)	$\chi^2_{\text{M}}=34.504, p<.001$
	Visual Hallucination	31	19 (35%)	2 (3%)	$\chi^2_{\text{M}}=20.407, p<.001$
	Auditory distortion	16	16 (30%)	2 (3%)	$\chi^2_{\text{M}}=15.134, p<.001$
	Auditory non-verbal hallucination	6, 32	30 (56%)	5 (7%)	$\chi^2_{\text{M}}=32.918, p<.001$
c)	FRS	3, 5, 7, 11	39 (72%)	9 (13%)	$\chi^2_{\text{M}}=42.814, p<.001$

D.1.2 Study 2 – BPD and Control

Comparison of prevalences of perceptual experiences in the BPD and control group of Study 2, for the nine CAPS symptom categories (a), other relevant symptom groups (as often reported in the literature) (b), and Schneider's first rank symptoms (FRS) of psychosis (c). Differences assessed using Pearson's Chi-squared test with Yates' continuity correction, p -values adjusted with Bonferroni correction for multiple comparison. Degrees of freedom of all $\chi^2_M = 1$. *Chi-square approximation may be unreliable due to limited observations, p -value confirmed with Fisher's exact test for count data.

a)		relevant items	BPD (n=20)	Control (n=29)	χ^2
	Having a Nonshared Sensory Experience	13, 29, 31, 32	13 (65%)	1 (3%)	$\chi^2_M=19.061, p<.001$
	Unusual or Distorted Sensory Experience	5, 16, 25, 26, 30	12 (60%)	3 (10%)	$\chi^2_M=11.502, p<.001$
	Sensory Experience from Unexplained Source	4, 6, 8, 12, 14, 28	15 (75%)	3 (10%)	$\chi^2_M=18.6, p<.001$
	Distortion of Form (Own Body and World)	9, 10, 19, 22	12 (60%)	2 (7%)	$\chi^2_M=13.857, p<.001$
	Changes in Levels of Sensory Intensity	1, 18, 20, 21, 23	18 (90%)	6 (21%)	$\chi^2_M=20.066, p<.001$
	Sensory Flooding	15, 17	17 (85%)	4 (14%)	$\chi^2_M=30.455, p<.001$
	Thought Echo and Hearing Thoughts	3, 7	13 (65%)	1 (3%)	$\chi^2_M=19.061, p<.001$
	Temporal Lobe Abnormalities	2, 10, 24, 27	18 (90%)	4 (14%)	$\chi^2_M=24.791, p<.001$
	Auditory Verbal Hallucinations	11, 13, 28	11 (55%)	1 (3%)	$\chi^2_M=14.337, p<.001^*$
b)	Visual distortion	4, 26, 19	13 (65%)	0 (0%)	$\chi^2_M=22.431, p<.001$
	Visual Hallucination	31	10 (50%)	0 (0%)	$\chi^2_M=15.27, p<.001^*$
	Auditory distortion	16	5 (25%)	0 (0%)	$\chi^2_M=15.134, p<.001$
	Auditory non-verbal hallucination	6, 32	9 (45%)	2 (7%)	$\chi^2_M=5.576, p=.018^*$
c)	FRS	3, 5, 7, 11	16 (80%)	5 (17%)	$\chi^2_M=16.56, p<.001$

D.1.3 BPD groups of Studies 1 & 2

Comparison of prevalences of perceptual experiences in the BPD groups of studies one and two, for the nine CAPS symptom categories (a), other relevant symptom groups (as often reported in the literature) (b), and Schneider's first rank symptoms (FRS) of psychosis (c). Differences assessed using Pearson's Chi-squared test with Yates' continuity correction. Degrees of freedom of all $\chi^2_M = 1$. *Chi-square approximation may be unreliable due to limited observations, p -value confirmed with Fisher's exact test for count data. Original p -values shown in table. All p -values adjusted with Bonferroni correction for multiple comparison = 1.

a)		relevant items	BPD ($n=54$)	BPD ($n=20$)	χ^2
	Having a Nonshared Sensory Experience	13, 29, 31, 32	30 (56%)	13 (65%)	$\chi^2_M=0.217, p=.641$
	Unusual or Distorted Sensory Experience	5, 16, 25, 26, 30	36 (67%)	12 (60%)	$\chi^2_M=0.067, p=.795$
	Sensory Experience from Unexplained Source	4, 6, 8, 12, 14, 28	35 (65%)	15 (75%)	$\chi^2_M=0.304, p=.581$
	Distortion of Form (Own Body and World)	9, 10, 19, 22	35 (65%)	12 (60%)	$\chi^2_M=0.012, p=.912$
	Changes in Levels of Sensory Intensity	1, 18, 20, 21, 23	45 (83%)	18 (90%)	$\chi^2_M=0.121, p=.728^*$
	Sensory Flooding	15, 17	37 (69%)	17 (85%)	$\chi^2_M=1.261, p=.261$
	Thought Echo and Hearing Thoughts	3, 7	24 (44%)	13 (65%)	$\chi^2_M=1.713, p=.191$
	Temporal Lobe Abnormalities	2, 10, 24, 27	38 (70%)	18 (90%)	$\chi^2_M=2.082, p=.149$
	Auditory Verbal Hallucinations	11, 13, 28	21 (39%)	11 (55%)	$\chi^2_M=0.957, p=.328$
b)	Visual distortion	4, 26, 19	32 (59%)	13 (65%)	$\chi^2_M=0.033, p=.856$
	Visual Hallucination	31	19 (35%)	10 (50%)	$\chi^2_M=0.794, p=.373$
	Auditory distortion	16	16 (30%)	5 (25%)	$\chi^2_M=0.003, p=.954$
	Auditory non-verbal hallucination	6, 32	30 (56%)	9 (45%)	$\chi^2_M=0.298, p=.585$
c)	FRS	3, 5, 7, 11	39 (72%)	16 (80%)	$\chi^2_M=0.145, p=.703$

D.2 PDI symptom prevalence comparisons

D.2.1 Study 1 – BPD and Control

Comparison of the prevalences of delusional beliefs in the BPD and control groups of Study 1, for the seven most consistent of PDI symptom categories. Differences assessed using Pearson's Chi-squared test with Yates' continuity correction, p -values adjusted with Bonferroni correction for multiple comparison. Degrees of freedom of all $\chi^2_M = 1$.
*differences significant at $p < .01$

	BPD ($n=54$)	Control ($n=68$)	χ^2
*persecution (items 4, 5)	31 (57%)	8 (12%)	$\chi^2_M=26.771, p<.001$
*suspiciousness (items 1,3)	48 (89%)	40 (60%)	$\chi^2_M=12.08, p=.003$
*paranoid ideation (items 13, 15)	41 (76%)	24 (35%)	$\chi^2_M=18.262, p<.001$
religiosity (items 8, 11)	18 (33%)	17 (25%)	$\chi^2_M=0.655, p=1$
grandiosity (items 6, 7)	35 (65%)	36 (53%)	$\chi^2_M=0.011, p=1$
paranormal beliefs (items 9, 12)	29 (54%)	21 (31%)	$\chi^2_M=5.572, p<.128$
*thought disturbances (items 18, 20)	35 (65%)	5 (7%)	$\chi^2_M=42.527, p<.001$

D.2.2 Study 2 – BPD and Control

Comparison of the prevalences of delusional beliefs in the BPD and control groups of Study 2, for the seven most consistent of PDI symptom categories. Differences assessed using Pearson's Chi-squared test with Yates' continuity correction, p -values adjusted with Bonferroni correction for multiple comparison. Degrees of freedom of all $\chi^2_M = 1$.
*differences significant at $p < .05$

	BPD (<i>n</i> =20)	Control (<i>n</i> =29)	χ^2
persecution (items 4, 5)	12 (60%)	10 (34%)	$\chi^2_M=2.169, p<.090$
*suspiciousness (items 1,3)	17 (85%)	8 (28%)	$\chi^2_M=13.401, p=.002$
*paranoid ideation (items 13, 15)	16 (80%)	8 (28%)	$\chi^2_M=11, p=.006$
religiosity (items 8, 11)	3 (15%)	3 (10%)	$\chi^2_M=0.002, p=1^*$
grandiosity (items 6, 7)	11 (55%)	8 (28%)	$\chi^2_M=2.681, p=.711$
paranormal beliefs (items 9, 12)	9 (45%)	5 (17%)	$\chi^2_M=3.213, p=.512$
*thought disturbances (items 18, 20)	10 (50%)	3 (10%)	$\chi^2_M=7.623, p=.040$

D.2.3 BPD groups of Studies 1 & 2

Comparison of the prevalences of delusional beliefs in the BPD groups of studies one and two, for the seven most consistent of PDI symptom categories. Differences assessed using Pearson's Chi-squared test with Yates' continuity correction. Degrees of freedom of all $\chi^2_M=1$. Original *p*-values shown in table. All *p*-values adjusted with Bonferroni correction for multiple comparison = 1.

	BPD (<i>n</i> =54)	BPD (<i>n</i> =20)	χ^2
persecution (items 4, 5)	31 (57%)	12 (60%)	$\chi^2_M=0.000, p=1$
suspiciousness (items 1,3)	48 (89%)	17 (85%)	$\chi^2_M=0.003, p=.957$
paranoid ideation (items 13, 15)	41 (76%)	16 (80%)	$\chi^2_M=0.003, p=.953$
religiosity (items 8, 11)	18 (33%)	3 (15%)	$\chi^2_M=1.596, p=.207$
grandiosity (items 6, 7)	35 (65%)	11 (55%)	$\chi^2_M=0.253, p=.615$
paranormal beliefs (items 9, 12)	29 (54%)	9 (45%)	$\chi^2_M=0.110, p=.741$
thought disturbances (items 18, 20)	35 (65%)	10 (50%)	$\chi^2_M=0.794, p=.373$

Appendix E Structural equation model

E.1 SEM parameters

E.1.1 Measurement model

Key measures of model fit:

chisq	rmsea	cfi	aic	tli
5.746	0.000	1.000	1106.429	1.004

Model summary:

Estimator	ML
Optimization method	NLMINB
Number of model parameters	15
Number of observations	74

Model Test User Model:

Test statistic	5.746
Degrees of freedom	6
P-value (Chi-square)	0.452

Model Test Baseline Model:

Test statistic	181.252
Degrees of freedom	15
P-value	0.000

User Model versus Baseline Model:

Comparative Fit Index (CFI)	1.000
Tucker-Lewis Index (TLI)	1.004

Loglikelihood and Information Criteria:

Loglikelihood user model (H0)	-538.215
Loglikelihood unrestricted model (H1)	-535.342
Akaike (AIC)	1106.429
Bayesian (BIC)	1140.990
Sample-size adjusted Bayesian (SABIC)	1093.719

Root Mean Square Error of Approximation:

RMSEA	0.000
90 Percent confidence interval - lower	0.000
90 Percent confidence interval - upper	0.148
P-value H ₀ : RMSEA ≤ 0.050	0.566
P-value H ₀ : RMSEA ≥ 0.080	0.293

Standardized Root Mean Square Residual:

SRMR	0.037

Parameter Estimates:

Standard errors	Standard
Information	Expected
Information saturated (h1) model	Structured

Latent Variables:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
CMD =~						
Anxiety	1.000				0.870	0.882
Depression	0.914	0.132	6.917	0.000	0.795	0.806
Trauma =~						
CTQ.Score	1.000				0.493	0.496
PCL.Total.Scor	1.750	0.473	3.698	0.000	0.862	0.868
Psychosis =~						
CAPS.YesNo	1.000				0.780	0.785
PDI.YesNo	1.136	0.265	4.281	0.000	0.885	0.891

Covariances:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
CMD ~~						
Trauma	0.354	0.115	3.070	0.002	0.827	0.827
Psychosis	0.281	0.110	2.553	0.011	0.415	0.415
Trauma ~~						
Psychosis	0.199	0.084	2.374	0.018	0.519	0.519

Variances:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
.Anxiety	0.217	0.090	2.420	0.016	0.217	0.223
.Depression	0.341	0.089	3.851	0.000	0.341	0.351
.CTQ.Score	0.744	0.132	5.645	0.000	0.744	0.754
.PCL.Total.Scor	0.243	0.155	1.562	0.118	0.243	0.246
.CAPS.YesNo	0.379	0.143	2.650	0.008	0.379	0.384
.PDI.YesNo	0.203	0.169	1.197	0.231	0.203	0.205
CMD	0.756	0.176	4.290	0.000	1.000	1.000
Trauma	0.243	0.117	2.070	0.038	1.000	1.000
Psychosis	0.608	0.197	3.078	0.002	1.000	1.000

Correlations between model residuals:

	Anx.	Depr.	CTQ	PCL	CAPS	PDI
Anxiety	0.000					
Depression	0.000	0.000				
CTQ	-0.065	0.036	0.000			
PCL	0.011	-0.005	0.000	0.000		
CAPS	0.033	0.022	0.127	-0.037	0.000	
PDI	-0.006	-0.027	0.056	-0.004	0.000	0.000

Modification indices:

None.

E.1.2 Structural model**Key measures of model fit:**

chisq	rmsea	cfi	srmr	tli
10.212	0.043	0.994	0.035	0.987

Model summary:

Estimator	ML
Optimization method	NLMINB
Number of model parameters	19
Number of observations	74

Model Test User Model:

Test statistic	10.212
Degrees of freedom	9
P-value (Chi-square)	0.334

Model Test Baseline Model:

Test statistic	237.703
Degrees of freedom	21
P-value	0.000

User Model versus Baseline Model:

Comparative Fit Index (CFI)	0.994
Tucker-Lewis Index (TLI)	0.987

Loglikelihood and Information Criteria:

Loglikelihood user model (H0)	-616.210
Loglikelihood unrestricted model (H1)	-611.104
Akaike (AIC)	1270.419
Bayesian (BIC)	1314.197
Sample-size adjusted Bayesian (SABIC)	1254.320

Root Mean Square Error of Approximation:

RMSEA	0.043
90 Percent confidence interval - lower	0.000
90 Percent confidence interval - upper	0.142
P-value H ₀ : RMSEA ≤ 0.050	0.475
P-value H ₀ : RMSEA ≥ 0.080	0.341

Standardized Root Mean Square Residual:

SRMR 0.035

Parameter Estimates:

Standard errors
Information
Information saturated (h1) model

Standard
Expected
Structured

Latent Variables:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
CMD =~						
Anxiety	1.000				0.837	0.848
Depression	0.987	0.137	7.180	0.000	0.826	0.837
Trauma =~						
CTQ.Score	1.000				0.504	0.507
PCL.Total.Scor	1.674	0.430	3.892	0.000	0.843	0.849
Psychosis =~						
CAPS.YesNo	1.000				0.772	0.777
PDI.YesNo	1.159	0.183	6.343	0.000	0.894	0.900

Regressions:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
CMD ~						
Trauma	1.400	0.374	3.743	0.000	0.843	0.843
Psychosis ~						
Trauma (a)	0.515	0.579	0.890	0.374	0.336	0.336
Dissociation ~						
Trauma (b)	1.193	0.348	3.429	0.001	0.601	0.609
Psychosis ~						
Dissociatn (c)	0.461	0.122	3.768	0.000	0.597	0.589
CMD	-0.182	0.305	-0.594	0.552	-0.197	-0.197

Covariances:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
.CMD ~~						
.Dissociation	0.029	0.100	0.293	0.769	0.065	0.083

Variances:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
.Anxiety	0.273	0.085	3.210	0.001	0.273	0.280
.Depression	0.291	0.085	3.424	0.001	0.291	0.299
.CTQ.Score	0.733	0.130	5.638	0.000	0.733	0.743
.PCL.Total.Scor	0.275	0.144	1.911	0.056	0.275	0.279
.CAPS.YesNo	0.391	0.097	4.029	0.000	0.391	0.396
.PDI.YesNo	0.187	0.102	1.823	0.068	0.187	0.189
.Dissociation	0.612	0.133	4.582	0.000	0.612	0.629
.CMD	0.203	0.121	1.677	0.094	0.290	0.290
Trauma	0.254	0.119	2.133	0.033	1.000	1.000
.Psychosis	0.297	0.090	3.298	0.001	0.499	0.499

R-Square:

	Estimate
Anxiety	0.720
Depression	0.701
CTQ.Score	0.257
PCL.Total.Scor	0.721
CAPS.YesNo	0.604
PDI.YesNo	0.811
Dissociation	0.371
CMD	0.710
Psychosis	0.501

Defined Parameters:

	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
indirecttrauma	0.549	0.202	2.725	0.006	0.359	0.359
totaltrauma	1.010	0.293	3.444	0.001	0.956	0.947

Correlations between model residuals:

	Anx.	Depr.	CTQ	PCL	CAPS	PDI	Diss.
Anxiety	0.000						
Depression	0.000	0.000					
CTQ	-0.067	0.009	0.000				
PCL	0.037	-0.026	0.000	0.000			
CAPS	0.050	0.018	0.121	-0.032	0.000		
PDI	0.008	-0.038	0.044	-0.008	0.000	0.000	
Dissociation	-0.044	0.048	0.047	-0.011	0.004	-0.002	0.000

Modification indices:

	lhs	op	rhs	mi	epc	sepc.lv	sepc.all	sepc.nox
1	Anxiety	~~	PCL.Total.Score	4.41	0.176	0.176	0.642	0.642
2	Trauma	=~	Depression	3.87	-3.56	-1.79	-1.82	-1.82
3	Trauma	=~	Anxiety	3.87	3.60	1.82	1.84	1.84
4	Depression	~~	PCL.Total.Score	2.84	-0.140	-0.140	-0.494	-0.494
5	Anxiety	~~	CTQ.Score	1.98	-0.104	-0.104	-0.233	-0.233
6	Depression	~~	PDI.YesNo	1.46	-0.0627	-0.0627	-0.269	-0.269

E.2 SEM - Alternative model fit

Key measures of model fit:

chisq	rmsea	cfi	srmr	tli
5.756	0.000	1.000	0.034	1.027

Model summary:

Estimator	ML
Optimization method	NLMINB
Number of model parameters	20

Number of observations	74

Model Test User Model:

Test statistic	5.756
Degrees of freedom	8
P-value (Chi-square)	0.675

Model Test Baseline Model:

Test statistic	237.703
Degrees of freedom	21
P-value	0.000

User Model versus Baseline Model:

Comparative Fit Index (CFI)	1.000
Tucker-Lewis Index (TLI)	1.027

Loglikelihood and Information Criteria:

Loglikelihood user model (H0)	-613.981
Loglikelihood unrestricted model (H1)	-611.104
Akaike (AIC)	1267.963
Bayesian (BIC)	1314.044
Sample-size adjusted Bayesian (SABIC)	1251.017

Root Mean Square Error of Approximation:

RMSEA	0.000
90 Percent confidence interval - lower	0.000
90 Percent confidence interval - upper	0.108
P-value H ₀ : RMSEA ≤ 0.050	0.776
P-value H ₀ : RMSEA ≥ 0.080	0.121

Standardized Root Mean Square Residual:

SRMR	0.034

Parameter Estimates:

Standard errors	Standard

Information	Information saturated (h1) model			Expected Structured		
Latent Variables:						
	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
CMD =~						
Anxiety	1.000				0.748	0.760
Depression	1.228	0.224	5.482	0.000	0.919	0.932
Trauma =~						
CTQ.Score	1.000				0.539	0.542
PCL.Total.Scor	1.472	0.374	3.937	0.000	0.793	0.801
Psychosis =~						
CAPS.YesNo	1.000				0.770	0.776
PDI.YesNo	1.163	0.184	6.309	0.000	0.896	0.902
Regressions:						
	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
CMD ~						
Trauma	1.062	0.316	3.357	0.001	0.764	0.764
Psychosis ~						
Trauma (a)	0.354	0.376	0.943	0.346	0.248	0.248
Dissociation ~						
Trauma (b)	1.165	0.322	3.615	0.000	0.627	0.636
Psychosis ~						
Dissociatn (c)	0.479	0.125	3.821	0.000	0.622	0.613
CMD	-0.161	0.213	-0.754	0.451	-0.156	-0.156
Covariances:						
	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
.CMD ~~						
.Dissociation	0.045	0.084	0.534	0.593	0.093	0.122
.Anxiety ~~						
.PCL.Total.Scor	0.164	0.077	2.118	0.034	0.164	0.433
Variances:						
	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
.Anxiety	0.409	0.108	3.784	0.000	0.409	0.422
.Depression	0.128	0.129	0.994	0.320	0.128	0.132
.CTQ.Score	0.696	0.129	5.405	0.000	0.696	0.706
.PCL.Total.Scor	0.351	0.138	2.538	0.011	0.351	0.358
.CAPS.YesNo	0.393	0.097	4.030	0.000	0.393	0.398
.PDI.YesNo	0.184	0.103	1.783	0.075	0.184	0.187
.Dissociation	0.580	0.133	4.354	0.000	0.580	0.596
.CMD	0.233	0.090	2.579	0.010	0.416	0.416
Trauma	0.290	0.129	2.245	0.025	1.000	1.000
.Psychosis	0.302	0.089	3.389	0.001	0.509	0.509
R-Square:						
	Estimate					
Anxiety	0.578					
Depression	0.868					
CTQ.Score	0.294					
PCL.Total.Scor	0.642					
CAPS.YesNo	0.602					
PDI.YesNo	0.813					
Dissociation	0.404					
CMD	0.584					
Psychosis	0.491					
Defined Parameters:						
	Estimate	Std.Err	z-value	P(> z)	Std.lv	Std.all
indirecttrauma	0.558	0.201	2.772	0.006	0.390	0.390
totaltrauma	1.037	0.298	3.479	0.001	1.011	1.003

Appendix F Repeat measure ANOVA and pairwise comparisons of stress check-ins

F.1 Stress Ratings

ANOVA table:

Effect	DFn	DFd	F	p
1 Group	1.0	47.00	9.124	4.00e-03*
2 Checkpoint	4.2	197.29	17.428	1.13e-12*
3 Group x Checkpoint	4.2	197.29	0.429	7.97e-01*

Between-group pairwise comparisons for each check-point

Checkpoint	Test	group1	group2	n1	n2	p	p.adj
1 Stress 1	Rating BPD	CONTROL	CONTROL	20	29	0.0245	0.0245*
2 Stress 2	Rating BPD	CONTROL	CONTROL	20	29	0.00185	0.00185*
3 Stress 3	Rating BPD	CONTROL	CONTROL	20	29	0.0177	0.0177*
4 Stress 4	Rating BPD	CONTROL	CONTROL	20	29	0.0115	0.0115*
5 Stress 5	Rating BPD	CONTROL	CONTROL	20	29	0.071	0.071
6 Stress 6	Rating BPD	CONTROL	CONTROL	20	29	0.0306	0.0306*

Within-group pairwise comparisons of all check-point ratings

Group	Test	group1	group2	n1	n2	statistic	df	p.adj
1 BPD	Rating Stress	1	Stress 2	20	20	-0.224	19	1
2 BPD	Rating Stress	1	Stress 3	20	20	0.462	19	1
3 BPD	Rating Stress	1	Stress 4	20	20	-1.24	19	1
4 BPD	Rating Stress	1	Stress 5	20	20	-3.21	19	0.069
5 BPD	Rating Stress	1	Stress 6	20	20	0.809	19	1
6 BPD	Rating Stress	2	Stress 3	20	20	1	19	1
7 BPD	Rating Stress	2	Stress 4	20	20	-0.960	19	1
8 BPD	Rating Stress	2	Stress 5	20	20	-3.66	19	0.025*

9 BPD	Rating Stress 2 Stress 6	20	20	1.10	19	1
10 BPD	Rating Stress 3 Stress 4	20	20	-1.80	19	1
11 BPD	Rating Stress 3 Stress 5	20	20	-5.21	19	0.000748*
12 BPD	Rating Stress 3 Stress 6	20	20	0.462	19	1
13 BPD	Rating Stress 4 Stress 5	20	20	-2.18	19	0.631
14 BPD	Rating Stress 4 Stress 6	20	20	2.52	19	0.315
15 BPD	Rating Stress 5 Stress 6	20	20	4.22	19	0.007*
16 CONTROL	Rating Stress 1 Stress 2	29	29	1	28	1
17 CONTROL	Rating Stress 1 Stress 3	29	29	1.09	28	1
18 CONTROL	Rating Stress 1 Stress 4	29	29	-0.177	28	1
19 CONTROL	Rating Stress 1 Stress 5	29	29	-4.77	28	0.000782*
20 CONTROL	Rating Stress 1 Stress 6	29	29	0.849	28	1
21 CONTROL	Rating Stress 2 Stress 3	29	29	0.254	28	1
22 CONTROL	Rating Stress 2 Stress 4	29	29	-1.09	28	1
23 CONTROL	Rating Stress 2 Stress 5	29	29	-6.44	28	0.0000085*
24 CONTROL	Rating Stress 2 Stress 6	29	29	0	28	1
25 CONTROL	Rating Stress 3 Stress 4	29	29	-2.27	28	0.47
26 CONTROL	Rating Stress 3 Stress 5	29	29	-6.24	28	0.0000146*
27 CONTROL	Rating Stress 3 Stress 6	29	29	-0.297	28	1
28 CONTROL	Rating Stress 4 Stress 5	29	29	-4.38	28	0.002*
29 CONTROL	Rating Stress 4 Stress 6	29	29	1.15	28	1
30 CONTROL	Rating Stress 5 Stress 6	29	29	6.15	28	0.0000183*

F.2 Alertness Ratings

ANOVA table:

Effect	DFn	DFd	F	p
1 Group	1.00	47.00	1.545	0.220000
2 Checkpoint	4.07	191.37	5.789	0.000184*
3 Group x Checkpoint	4.07	191.37	0.685	0.605000

Between-group pairwise comparisons for each check-point

Checkpoint	Test	group1	group2	n1	n2	p	p.adj
1 Alert 1	Rating BPD	CONTROL		20	29	0.638	0.638
2 Alert 2	Rating BPD	CONTROL		20	29	0.153	0.153
3 Alert 3	Rating BPD	CONTROL		20	29	0.257	0.257
4 Alert 4	Rating BPD	CONTROL		20	29	0.37	0.37
5 Alert 5	Rating BPD	CONTROL		20	29	0.462	0.462
6 Alert 6	Rating BPD	CONTROL		20	29	0.124	0.124

Within-group pairwise comparisons of all check-point ratings

Group	Test	group 1	group2	n1	n2	statistic	df	p.adj
1 BPD	Rating	Alert 1	Alert 2	20	20	0.418	19	1
2 BPD	Rating	Alert 1	Alert 3	20	20	2.03	19	0.854
3 BPD	Rating	Alert 1	Alert 4	20	20	1	19	1
4 BPD	Rating	Alert 1	Alert 5	20	20	-0.645	19	1
5 BPD	Rating	Alert 1	Alert 6	20	20	2.07	19	0.792
6 BPD	Rating	Alert 2	Alert 3	20	20	1.67	19	1
7 BPD	Rating	Alert 2	Alert 4	20	20	0.809	19	1
8 BPD	Rating	Alert 2	Alert 5	20	20	-1.31	19	1
9 BPD	Rating	Alert 2	Alert 6	20	20	2.02	19	0.874
10 BPD	Rating	Alert 3	Alert 4	20	20	-0.567	19	1
11 BPD	Rating	Alert 3	Alert 5	20	20	-2.98	19	0.116
12 BPD	Rating	Alert 3	Alert 6	20	20	0.767	19	1
13 BPD	Rating	Alert 4	Alert 5	20	20	-1.83	19	1
14 BPD	Rating	Alert 4	Alert 6	20	20	1.31	19	1
15 BPD	Rating	Alert 5	Alert 6	20	20	2.33	19	0.462
16 CONTROL	Rating	Alert 1	Alert 2	29	29	-1.76	28	1
17 CONTROL	Rating	Alert 1	Alert 3	29	29	1	28	1
18 CONTROL	Rating	Alert 1	Alert 4	29	29	0.648	28	1
19 CONTROL	Rating	Alert 1	Alert 5	29	29	-1.98	28	0.867

20	CONTROL	Rating	Alert 1	Alert 6	29	29	1.16	28 1
21	CONTROL	Rating	Alert 2	Alert 3	29	29	3.64	28 0.016 *
22	CONTROL	Rating	Alert 2	Alert 4	29	29	3.02	28 0.079
23	CONTROL	Rating	Alert 2	Alert 5	29	29	-0.215	28 1
24	CONTROL	Rating	Alert 2	Alert 6	29	29	2.49	28 0.284
25	CONTROL	Rating	Alert 3	Alert 4	29	29	-0.328	28 1
26	CONTROL	Rating	Alert 3	Alert 5	29	29	-2.86	28 0.12
27	CONTROL	Rating	Alert 3	Alert 6	29	29	0	28 1
28	CONTROL	Rating	Alert 4	Alert 5	29	29	-1.95	28 0.925
29	CONTROL	Rating	Alert 4	Alert 6	29	29	0.205	28 1
30	CONTROL	Rating	Alert 5	Alert 6	29	29	3.27	28 0.043 *

F.3 Control Ratings

ANOVA table:

Effect	DFn	DFd	F	p
1 Group	1	47	19.032	6.98e-05*
2 Checkpoint	5	235	8.874	9.83e-08*
3 Group x Checkpoint	5	235	4.686	4.25e-04*

Between-group pairwise comparisons for each check-point

Checkpoint	Test	group1	group2	n1	n2	p	p.adj
1 Control	1	Rating BPD	CONTROL	20	29	0.14	0.14
2 Control	2	Rating BPD	CONTROL	20	29	0.000075	0.000075*
3 Control	3	Rating BPD	CONTROL	20	29	0.0000312	0.0000312*
4 Control	4	Rating BPD	CONTROL	20	29	0.0000269	0.0000269*
5 Control	5	Rating BPD	CONTROL	20	29	0.00181	0.00181*
6 Control	6	Rating BPD	CONTROL	20	29	0.000486	0.000486*

Within-group pairwise comparisons of all check-point ratings

Group	Test	group 1	group2	n1	n2	statistic	df	p.adj
1 BPD	Rating Control	1	Control 2	20	20	4.05	19	0.01*
2 BPD	Rating Control	1	Control 3	20	20	4.27	19	0.006*
3 BPD	Rating Control	1	Control 4	20	20	4.59	19	0.003*
4 BPD	Rating Control	1	Control 5	20	20	4.10	19	0.009*
5 BPD	Rating Control	1	Control 6	20	20	2.60	19	0.261
6 BPD	Rating Control	2	Control 3	20	20	0.698	19	1
7 BPD	Rating Control	2	Control 4	20	20	0.252	19	1
8 BPD	Rating Control	2	Control 5	20	20	0.400	19	1
9 BPD	Rating Control	2	Control 6	20	20	-1.71	19	1
10 BPD	Rating Control	3	Control 4	20	20	-0.271	19	1
11 BPD	Rating Control	3	Control 5	20	20	0	19	1
12 BPD	Rating Control	3	Control 6	20	20	-2.24	19	0.562
13 BPD	Rating Control	4	Control 5	20	20	0.237	19	1
14 BPD	Rating Control	4	Control 6	20	20	-2.27	19	0.526
15 BPD	Rating Control	5	Control 6	20	20	-2.24	19	0.562
16 CONTROL	Rating Control	1	Control 2	29	29	0.828	28	1
17 CONTROL	Rating Control	1	Control 3	29	29	0.626	28	1
18 CONTROL	Rating Control	1	Control 4	29	29	0.297	28	1
19 CONTROL	Rating Control	1	Control 5	29	29	2.07	28	0.713

20	CONTROL	Rating Control 1 Control 6	29	29	-0.701	28 1
21	CONTROL	Rating Control 2 Control 3	29	29	-0.254	28 1
22	CONTROL	Rating Control 2 Control 4	29	29	-0.528	28 1
23	CONTROL	Rating Control 2 Control 5	29	29	1.18	28 1
24	CONTROL	Rating Control 2 Control 6	29	29	-1.54	28 1
25	CONTROL	Rating Control 3 Control 4	29	29	-0.441	28 1
26	CONTROL	Rating Control 3 Control 5	29	29	1.65	28 1
27	CONTROL	Rating Control 3 Control 6	29	29	-1.68	28 1
28	CONTROL	Rating Control 4 Control 5	29	29	1.77	28 1
29	CONTROL	Rating Control 4 Control 6	29	29	-1	28 1
30	CONTROL	Rating Control 5 Control 6	29	29	-2.26	28 0.474

