The Biopsychosocial Model of Depersonalisation and Derealisation: An Updated Framework

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Abstract

Depersonalisation (DP) and derealisation (DR) are dissociative phenomena involving detachment from the self (DP) or the external world (DR). Although relatively common, they remain under-researched, with existing studies largely limited to small clinical samples. This thesis adopts a biopsychosocial framework to investigate their development, maintenance, and severity. Chapter 1 reviews literature on DPDR, focusing on adverse childhood experiences (ACEs) and associated biological processes. The definition of ACEs was broadened beyond abuse and neglect to include household instability, emphasising the need to assess both individual ACE types and cumulative ACE clusters. The importance of distinguishing between DP and DR, and of considering secondary factors influencing DPDR, is highlighted. Using participant data from the Avon Longitudinal Study of Parents and Children (ALSPAC; N = 7,906), Chapter 2 examines ACEs in the first 11 years as predictors of DP and DR at ages 12, 17, and 24. Potential mediators (attachment, depression, anxiety, and perseverative cognition) are tested. Longitudinal analyses suggest ACEs are associated with DPDR from adolescence through to adulthood. Chapter 3 explores biological mechanisms, assessing whether interleukin-6 (IL-6) and C-reactive protein (CRP) predict DP and DR and mediate ACE–DPDR links. Sex, ethnicity, and social position are also examined as confounders. Longitudinal analyses demonstrated that CRP predicted DR over time, and IL-6 predicted DP. Chapter 4 investigates maintenance processes through thematic analysis of 368 self-help forum posts, identifying reactivation and persistence themes. Chapter 5 evaluates whether DPDR severity follows a dose-response relationship with ACEs and compares trait rumination with hyper-reflexivity as potential cognitive processes associated with DPDR. Quantitative analyses further tested maintenance themes identified qualitatively. Taken together, findings indicated that DP and DR may have distinct aetiologies: DP is strongly predicted by diverse ACEs and childhood IL-6, while DR is linked to selective ACEs and CRP dysregulation over time. Anxiety, depression, perseverative cognition, and rumination did not mediate ACE–DPDR relationships, while hyper-reflexivity emerged as a key feature of DPDR. Maintenance of DPDR appears driven by dysregulation of core systems, cognitive triggers, and environmental reactivity, helping explain their persistence.

Thesis Format

This thesis adopts a hybrid structure that combines elements of both the traditional monograph (or 'tome') format and the ‘four-paper’ model. Specifically, while the overarching framework, including the general introduction and final discussion, is written in the style of a traditional monograph to provide conceptual coherence and a broader contextual narrative, the core empirical chapters are presented as standalone papers. Each study is structured in the format of a journal article, with its own brief introduction, methods, results, and discussion section. Therefore, repetition may appear in the introduction and discussion sections of each paper. Phrases have not been re-abbreviated. This approach allows for each empirical chapter to function independently while contributing to the overall theoretical and methodological development of the thesis. The final general discussion synthesises the findings across studies, highlighting their collective implications, strengths, and limitations, and proposes directions for future research.

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Abbreviations (A-Z)

ACEs = Adverse childhood experiences

AL = Allostatic load

ALSPAC = Avon Longitudinal Study of Parents and Children

ANS = Autonomic nervous system

CDC = Centres for Disease Control and Prevention

C-PTSD = Complex post-traumatic stress disorder

CRP = C-reactive protein

CSA = Childhood sexual abuse

DDD = Depersonalisation-derealisation disorder

DDs = Dissociative disorders

DiD = Difference-in-Differences

DID = Dissociative identity disorder

DP = Depersonalisation

DPDR = Depersonalisation and derealisation

D-PTSD = Dissociative post-traumatic stress disorder

DR = Derealisation

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

EA = Emotional abuse

EN = Emotional neglect

FND = Functional neurological disorder

HC = Healthy controls

HPA-axis = Hypothalamic-pituitary adrenal axis

IL-6 = Interleukin-6

IPV = Inter-parental violence

OCD = Obsessive-compulsive disorder

PA = Physical abuse

PC = Perseverative cognitions

PCH = Perseverative-cognition hypothesis

PMI = Parental mental illness

PN = Physical neglect

PSA = Parental substance abuse

PTSD = Post-traumatic stress disorder

rTMS = Repetitive transcranial magnetic stimulation

SSRIs = Selective serotonin reuptake inhibitors

TM = Trauma model

TNF-α = Tumor necrosis factor-alpha

TPJ = Temporoparietal junction

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Chapter 1: Introduction

1.1 Background to depersonalisation and derealisation

Depersonalisation (DP) and derealisation (DR; DPDR) are symptoms that disturb an individual's sense of self. DP is commonly referred to as feeling "disconnected from yourself", or feeling "like you're watching yourself from the outside" or are not "fully in your body" (Unreal, 2025). In contrast, DR refers to feeling as though "the world around you is strange, dreamlike, or unreal" or "like you're not fully part of the world" (Unreal, 2025). DPDR are dissociative experiences, cause disconnection to thoughts, memories, feelings, surroundings or identity. Other dissociative experiences include dissociative amnesia, identity alternation and identity confusion (American Psychiatric Association, 2013, Section II: Dissociative Disorders).

Dissociation is typically viewed along a continuum, ranging from everyday experiences like momentary absorption, such as zoning out, to more extreme forms, including dissociative disorders (DDs) such as depersonalisation-derealisation disorder (DDD), DPDR as part of dissociative post-traumatic stress disorder (D-PTSD), or dissociative identity disorder (DID). Within this spectrum, transient, mild symptoms are typical under certain circumstances (e.g., after traumatic events or periods of intense stress). Further, Holmes et al. (2005) propose that dissociation can be meaningfully divided into two distinct processes: detachment and compartmentalisation. Detachment refers to DPDR experiences, where cognitive and behavioural functioning remain largely intact. In contrast, compartmentalisation reflects a failure of integration between mental processes, such that information or functions that are ordinarily accessible to conscious awareness become temporarily unavailable or outside voluntary control (e.g., dissociative amnesia or functional neurological symptoms).

While not widely discussed, DPDR are commonly experienced in the general population (Aderibigbe et al.,2001; Ross, Joshi & Currie, 1991), and are a part of being human (Hunter, Sierra & David, 2004).

“Louise often feels like part of her is "acting." At the same time, "there is another part 'inside' that is not connecting with the me that is talking to you," she says. When the depersonalization is at its most intense, she feels like she just doesn't exist. These experiences leave her confused about who she really is, and quite often, she feels like an "actress" or simply, "a fake.” — Simeon & Abugel, 2006, p. 8

1.2 Epidemiology

DPDR are considered the third most common mental health symptom, following anxiety and depression (Simeon et al., 2003). The lifetime prevalence of DPDR in the general population has been estimated at 26–74%, with 31–66% reporting symptoms during traumatic events (Hunter, Sierra, & David, 2004). To put these figures into a UK context, given a total population of approximately 68.3 million, a lifetime prevalence of 26-74% suggests that between 17.7 million and 50.5 million individuals may experience DPDR at some point in their lives.

Further, DPDR prevalence is elevated in clinical samples (5–20% in outpatients; 17.5–41.9% in inpatients; Yang et al., 2023), and can be transdiagnostic symptoms of various psychopathology, such as panic disorder, depression, post-traumatic stress disorder (PTSD), and psychosis (Černis et al., 2025; Ellickson-Larew et al., 2020; Hallett et al., 2025; Hunter, Sierra and David, 2004). Additionally, DPDR also occurs across multiple mood and anxiety disorders (panic disorder, social phobia, bipolar I and II disorders), particularly in comorbid presentations (Simeon & Stein, 2025). Meta-analytic estimates indicate that approximately 38% of individuals with PTSD also meet criteria for D-PTSD (White et al., 2022). Elevated prevalence is also seen among individuals in recovery from addiction, where nearly one-quarter report severe DPDR and up to 44% experience symptoms when milder forms are included (Sirvent & Fernández, 2015).

When DPDR becomes persistent and unremitting, individuals may meet the criteria for DDD (Medford et al., 2005). Research on DDD remains in its early stages, and while prevalence estimates are uncertain due to lack of research and understanding, the current literature suggests that approximately 1-2% of the population may qualify for a diagnosis (Yang et al., 2023, Simeon & Stein, 2025). For context, the 2014

Adult Psychiatric Morbidity Survey estimated the prevalence of schizophrenia at 0.7% (Bebbington et al., 2016), obsessive-compulsive disorder (OCD) at 1.3%, and panic disorder at 0.6% (McManus et al., 2016).

Additionally, the prevalence of DDD ranges according to the population measured: with the highest rates observed in those with anxiety disorders (3–20%), other DDs (4–20%), schizophrenia (16%), and borderline personality disorder (17%) (Yang et al., 2023). Further, a study using a German sample found that 12% of adolescents experienced clinically significant DPDR (indicative of DDD), highlighting its relevance to early mental health (Michal et al., 2015). In context, among the estimated 8 million 10–19-year-olds in the UK, approximately 952,000 (12%) adolescents may experience clinically significant DPDR.

A diagnosis of DDD is often delayed, taking an average of 7-12 years (Baker et al., 2003; Michal et al., 2016), precluded by a lack of awareness among healthcare providers. This leaves individuals with DDD vulnerable to misdiagnosis and unproductive consultations, leading to ‘diagnostic slippage’, whereby an individual must diagnose themselves with the condition (Duran, 2021). While a correct diagnosis can be reassuring and even therapeutic (Medford et al., 2005), access to specialised psychiatric care is often limited, with many individuals relying on general practitioners who may be unfamiliar with the condition.

1.3 Functional and public health consequences of depersonalisation and derealisation

DDD symptoms are generally “invisible”; individuals can appear outwardly functional, but their internal experiences are fraught with challenges, and the sequelae associated with DPDR can be profoundly distressing for those affected (Hunter et al., 2017). For example, DDD causes substantial impairment in both interpersonal relationships and occupational functioning (American Psychological Association, 2013, p. 305). Further, individuals with DDD can experience a high level of catastrophic thoughts (Hunter et al., 2003), an increased stress response (Murphy, 2023), reduced phenomenal depth (the relatedness of one's self with one's mental processes, body, and the world; Gaebler et al., 2013), and increased traits of

psychoticism and negative affect (Fino et al., 2025). Further, individuals with DDD experience an inability to express themselves meaningfully due to alexithymia, a neuropsychological phenomenon where individuals find it difficult to articulate their emotions (Simeon et al., 2009). This can lead to individuals needing to use alternative language such as metaphors (Dilkes, 2024), contributing to their inability to meaningfully articulate. Externally, individuals with DDD can struggle to maintain jobs or socialise, create lasting relationships, or accomplish daily tasks, contributing to poor overall wellbeing (Simeon & Abugel, 2006) and low quality of life.

DDD is associated with other emotional-cognitive sequelae, such as poor immediate visual and verbal recall (Guralnik et al., 2007). Furthermore, DPDR are associated with poor interoceptive functioning (Sedeño et al., 2014), emotional detachment during threatening situations (Horn et al., 2020), as well as suppressed emotional reactivity in response to body-threats, even in individuals with sub-clinical DPDR (Dewe, Watson & Braithwaite, 2016). This suggests that individuals experiencing DDD are not only challenged by worse mental functioning but may also exhibit reduced awareness of their bodily signals and thus may be less perceptive to both internal and external threats.

The widespread prevalence and functional impairment associated with DDD contributes to the significant societal and economic burden of mental health, underscoring the urgent need for further research and intervention. DDs (such as DDD) are costly to society (Langeland et al., 2020), and are associated with long-term care, suicide risk, frequent hospitalisations, and significant disability. Additionally, individuals with DDD utilise healthcare services to a high degree but often find that existing mental health provisions do not meet their complex needs (Michal et al., 2016). DDs place a burden on family in terms of leisure, physical, mental, financial, and routine family interrelationship domains (Mohammad et al., 2023). Even though DDD is classified as a diagnosable DD by both the American Psychological Association (2013) and the World Health Organisation (2019), these symptoms remain significantly underrepresented in psychiatric research. The typical age of onset is age 16, and less than 5% of people experience the onset of symptoms after the age of 25 (American Psychological Association, 2013),

suggesting that this is a condition that young individuals are at a disproportionately higher risk for in comparison to older adults.

Overall, the evidence depicts DPDR as highly prevalent and distressing, severely impairing functioning, burdening families and social networks, and generating wider societal costs, all while disrupting core experiences of safety and wellbeing. Yet DPDR and DDD remain under-recognised and low on the agendas of both research and public health. Importantly, those affected continue to call for change, with individuals actively petitioning and lobbying governments to increase investment in understanding and addressing this debilitating disorder (UK Government & Parliament, 2023).

1.4 Precipitating and predisposing factors

1.4.1 Peritraumatic depersonalisation and derealisation

Peri-traumatic dissociation refers to dissociative symptoms that emerge during and immediately after exposure to extreme threat and are driven by fear expectancy and acute stress (McDonald et al., 2013; Schäfer, 2025). DPDR may arise within this peri-traumatic context, reflecting an intended protective dissociative response to acute threat. In some individuals, this response may persist beyond the resolution of the precipitating event, resembling an extended peri-traumatic state, which may explain the phenomenology of DPDR in the absence of trauma histories (Schäfer, 2025).

Two arguments exist as to whether peri-traumatic DPDR is an adaptive or non-adaptive mechanism in the face of trauma. Argument one is the hypothesis that peri-traumatic DPDR is adaptive, potentially protecting individuals from the overwhelming emotional impact of trauma, enabling them to continue functioning in the moment. For example, research with rescue paramedics suggests that DPDR allows them to operate on ‘autopilot’ under extreme stress, suppressing emotional responses until later. Once DPDR subsides, previously muted emotions such as fear, anger, or grief re-emerge and can be processed more appropriately (Pietkiewicz, Duszkiewicz, & Tomalski, 2023). Beyond this protective function, DPDR may also buffer against

longer-term psychiatric difficulties: individuals who reported dissociative symptoms during a traumatic event later experienced fewer psychiatric symptoms than those who did not (Shilony & Grossman, 1993).

However, argument two suggests that peri-traumatic dissociation (including DPDR) may precipitate development of PTSD in response to traumatic events, potentially due to disruption of normal information processing, leading to disruption of the trauma memory and inhibiting recovery (Ehlers & Clark, 2000). Meta-analyses indicate that retrospectively reported peri-traumatic dissociation is among the strongest predictors of later PTSD (Breh & Seidler, 2007; Ozer et al., 2008). Therefore, while DPDR is hypothesised as an adaptive function, it can also lead to mental health difficulties.

1.4.2 Drug-induced depersonalisation and derealisation

DPDR is commonly induced through drug use. For example, in a survey of 394 adults with DDD, approximately 50% of participants reported onset of symptoms following drug use, most commonly cannabis, hallucinogens, and ecstasy (Simeon et al., 2009). In a controlled analysis of 40 cases of DDD following illicit drug use, Medford et al. (2003) found that while those with drug-precipitated DPDR were younger and more often male, their symptom profiles, impairment, illness course, and treatment response were largely similar to DPDR cases not linked to a drug trigger.

1.4.3 Coronavirus

The coronavirus pandemic further highlighted the importance of researching DPDR, particularly in response to global events. Increased digital media use and heightened subjective distress during lockdown were associated with elevated DPDR (Ciaunica et al., 2022), and cases of DPDR were documented in the context of ‘COVID-19 psychosis’ (de Oca Rivas et al., 2020; Vepa et al., 2020). Healthcare workers, such as those in high-stress settings, developed post-traumatic stress symptoms during the pandemic (see d’Ettorre et al., 2021 for review), and a significant proportion report severe psychological distress, including DPDR (Majumder et al., 2021). Individual experiences of DPDR because of the coronavirus pandemic are a common feature of self-help online communities. For example, “It’s been two years of the pandemic, and my younger siblings’ mental health has deteriorated, and now has

DPDR. They don't know if reality is real and feels ethereal" (Reddit comment rephrased for the individuals' anonymity).

1.4.4 Adverse childhood experiences

A strong precipitating factor identified in DPDR is a history of adverse childhood experiences (ACEs), which are discussed at length below (see section 1.6 Adverse childhood experiences). This established ACE-DPDR link, coupled with a growing recognition of biological factors, highlights a critical research gap: investigating the potential mediating role of biological mechanisms between ACEs and DPDR. Furthermore, the factors that drive the transition from transient DPDR to chronic DDD, such as the role of hyper-reflexivity, remain poorly understood. Therefore, this thesis aims to elucidate these factors, and the following literature review builds the empirical basis for research question development. ACEs likely function as both a predisposing and precipitating factor in DPDR. As a predisposing factor, early adversity may confer enduring vulnerability through long-term alterations in stress responsivity, emotion regulation, and self-processing, increasing susceptibility to DPDR later in life. As a precipitating factor, ACEs may directly trigger DPDR when exposure is severe or cumulative, or when subsequent stressors activate these pre-existing vulnerabilities, promoting DPDR as an initially adaptive coping response. Together, this dual role may help explain the robust association between ACEs and DPDR, while also raising critical questions about the mechanisms that determine symptom persistence versus resolution.

1.5 Trauma model of depersonalisation and derealisation

The connection between traumatic experiences and dissociation has been noted since the 18th century (Janet, 1889). Janet noted that DPDR arises after extreme stress, and that initial DPDR responses to one stressor often predisposed individuals to be more easily triggered by subsequent stressors.

Over a century later, Dalenberg et al. (2012) evaluated the suitability of the Trauma Model (TM) for dissociation. The TM suggests that dissociation operates as a psychobiological mechanism, either as a transient state or enduring trait, which may shield individuals from the impact of traumatic or overwhelming experiences

(Dalenberg et al., 2012). It functions by shifting consciousness into altered states, thereby potentially shielding the individual from the overwhelming emotional and cognitive sequalae of the traumatic experience. Through this process, dissociation separates the traumatic material from ordinary awareness, preventing the full emotional and psychological meaning of the event from being fully processed or integrated (Boland, Verdiun & Ruiz, 2021). Thus, symptoms like amnesia, DP, DR, and identity fragmentation hypothetically allow a greater chance at survival. Additionally, the relationship between trauma and dissociation is temporal, where trauma precedes dissociative symptoms. Longitudinal research is lacking within DPDR specifically and therefore needs to be conducted to establish the role of the TM in DPDR.

The TM of DPDR is supported by observing the rates of DPDR in individuals who have experienced significant trauma, for instance ACEs (see 1.6 Adverse childhood experiences). For example, a study with refugees and asylum seekers trafficked to the UK experienced DPDR in relation to Complex PTSD (C-PTSD; Rees, 2025). Among individuals with subclinical psychotic symptoms and developmental trauma, DPDR alongside other dissociative experiences are a frequent and highly heterogeneous phenomena (Melegkovits et al., 2025).

This protective mechanism likely occurs in line with the Polyvagal Theory, developed by Stephen Porges (2011). This neurobiological model posits that the nervous system has three states in response to safety and threat. Firstly, the ventral vagal state, or the ‘safe and social’ state, is engaged when a person feels secure, supporting social engagement, connection, a feeling of calm, and physiological regulation. Secondly, the sympathetic state, or the ‘fight or flight’ state, is activated when a person senses danger, leading to increases in heart rate, respiration, and alertness, thereby preparing us to fight or flee. Thirdly, the dorsal vagal state, or the ‘shutdown / freeze’ state, is activated under extreme or inescapable threat, triggering numbing, dissociation, withdrawal, and energy conservation.

Therefore, DPDR may represent a symptom of the dorsal vagal state, activated in response to overwhelming sympathetic arousal. In this process, an individual transitions from a 'fight or flight' response to a 'freeze' state, leading to a profound

disconnection from themselves and their external environment to numb intense physiological activation.

ACEs, particularly those perpetrated by parents, are identified as a significant risk factor for DPDR, and the prolonged nature of inescapable stress in the household may contribute to this freeze. In this thesis, ACEs are the main social stressor.

Although trauma-based models of DPDR offer a compelling explanatory framework, they do not constitute a comprehensive account of DPDR aetiology. While a history of trauma is frequently associated with DPDR, it is neither necessary nor sufficient alone for the emergence of symptoms. Notably, up to 50% of individuals with DPDR do not report a recalled trauma history (Simeon et al., 2003), and in some samples trauma was identified as a contributing factor in as few as 14% of cases (Hunter et al., 2003). These findings suggest that trauma represents one of multiple developmental pathways to DPDR, rather than a singular or universal mechanism.

Further, predisposing genetic traits may determine whether individuals who experience trauma will develop dissociative symptoms, as evidenced by association between genes and dissociative symptoms (Wolf et al., 2015). Additionally, drug-induced DPDR are known phenomena (Sierra, 2008; Medford et al., 2003; Yildirim et al., 2024), with particularly potent induction through cannabis, psychedelics, and ecstasy (Simeon et al., 2009). However, a sole focus on substance use may be reductionist, and substance use may augment, rather than drive, underlying mechanisms (Michal, 2025).

1.6 Adverse childhood experiences

ACEs are “potentially traumatic events that can have negative lasting effects on health and well-being. This includes maltreatment and abuse as well as living in an environment that is harmful to their development” (Boullier & Blair, 2018). Cases of childhood abuse, neglect, and household instability can all serve as ACEs.

1.6.1 Sexual, physical and emotional abuse

The Centres for Disease Control and Prevention (CDC; 2024) defines childhood abuse as any form of mistreatment or harm that a child experiences prior to their 18th birthday. There are many forms of abuse, the most discussed being physical abuse (PA), sexual abuse (SA), and emotional abuse (EA). The CDC (2024) defines these forms of abuse as follows: PA is the intentional use of physical force that can result in physical injury. SA refers to any completed or attempted sexual acts or sexual contact with a child by a caregiver. EA refers to behaviours that harm a child's self-worth or emotional well-being.

1.6.2 Other types of adverse childhood experiences

Beyond PA, SA, and EA, other types of ACEs include physical neglect (PN) and emotional neglect (EN), and exposure to factors indicative of household instability. PN refers to “the failure to meet children’s physical needs, and including the failure to provide adequate nutrition, clothing, personal hygiene, supervision and medical attention” (Stoltenborgh et al., 2013). EN refers to “the failure to meet children’s emotional needs and includes for example the failure to provide adequate nurturance and affection, allowing children to witness domestic violence, to knowingly permit maladaptive behaviour by the child, the failure to seek care for emotional or behavioural problems, and the failure to provide adequate structure” (Stoltenborgh et al., 2013). Household instability includes witnessing inter-parental violence (IPV), living in a household with parental substance abuse (PSA) or parental mental illness (PMI), parental convictions, and parental divorces.

Evidence suggests that these 'other', non-traditional types of ACEs are equally as detrimental as 'traditional' ACEs, such as PA, SA, and EA (Gilbert et al., 2009), but are less researched.

1.6.3 Prevalence of adverse childhood experiences

A systematic review of self-reported child maltreatment across 34 countries revealed that child abuse is a widespread, global issue, with significant variation across regions and genders (Moody et al., 2018). Globally, the median prevalence of SA was 20% among girls and 14% among boys, with reported rates ranging from 1% to 44% for girls and 1% to 42% for boys. PA was even more prevalent, with median rates of 23% for girls and 25% for boys, and a broader range spanning 4% to 74% in

girls and 6% to 85% in boys. EA was also commonly reported, with a global median prevalence of 25% for girls and 16% for boys, ranging up to 67% and 60% respectively. Neglect showed the greatest variability across studies and was the least frequently assessed form of abuse, with a median global prevalence of 34% in girls and 28% in boys, but ranging from as low as 6% to as high as 78%. In the UK specifically, prevalence rates for PA range from 4% to 33%, while SA ranges from 1% to 28%. EA shows even greater variability, with estimates ranging from 4% to 67%. Neglect has been reported in 6% to 77% of participants.

The wide variation in international child maltreatment prevalence estimates is likely due to several methodological and contextual factors (Moody et al., 2018). Inconsistencies arise from differing definitions of abuse and neglect, varied measurement tools, and assessments over different time frames (e.g., lifetime vs. past year). Other contributing factors include sample characteristics, data collection methods, and cultural norms that affect the willingness to disclose experiences.

Regarding non-traditional ACEs, Pryce et al. (2017) analysed multiple data sources, including the 2014 Adult Psychiatric Morbidity Survey, the National Drug Treatment Monitoring Service, and the 2011 Census, and found that an estimated 189,000 to 208,000 children under 16 were living in households where at least one adult had alcohol dependence, suggestive of PSA. For PMI, prevalence rates are high, and appear to be increasing over time. Statistics from the Office for Health Improvement & Disparities show that emotional distress in mothers rose from 19% in 2014 – 2015 to 27% in 2019 – 2020, while emotional distress in fathers rose from 11% to 15% (2024). Census data demonstrates that approximately 7% of women and 3% of men aged 16 and over, equating to around 1.6 million women and 712,000 men, were estimated to have experienced domestic abuse within the past year (ONS Centre for Crime and Justice, 2024). In 2024 it was estimated that, in the UK, there were 2.5 million separated families, including approximately 4 million children. Further, this number appears to be growing, reflecting an increase in 100,000 separated families and 200,000 children compared to the previous year (Department for Work & Pensions, 2025). Data on the prevalence of parental convictions in the UK is limited, however data from the Ministry of Justice suggests that between 2021 and 2022, an estimated 192,912 children had a parent in prison (2024).

Whereas abuse-related ACEs involve direct exposure to harm, household instability ACEs may exert its effects indirectly, creating a chronically dysregulated environment where stress is persistently perceived rather than acutely encountered. This may lead to sustained physiological activation and engagement of stress-response systems. Household instability represents a chronic environmental stressor that may disrupt biological stress regulation. According to Bronfenbrenner's bioecological model (Bronfenbrenner, 1994), development occurs within multiple nested environmental systems, with the microsystem (immediate family environment) playing a critical role in shaping physiological and psychological health. Within this framework, unpredictable and chaotic home environments may dysregulate stress-response systems, leading to long-term alterations in neurobiological functioning.

In terms of the prevalence of cumulative ACEs, a study by Houtepen et al. (2020) using the Avon Longitudinal Study of Parents and Children (ALSPAC dataset found that 84% of participants reported at least one ACE, with 24% experiencing four or more by age 16. Similarly, Bellis et al. (2023) analysed data from eight cross-sectional surveys in England and Wales, revealing strong cumulative associations between ACEs and various health outcomes, highlighting that even experiencing just one ACE is associated with health problems. Prolonged and repeated exposure to stressors (Schneiderman et al., 2005) is linked to several mental and physical health conditions (Hughes et al., 2017; Kalmakis & Chandler, 2015; Norman et al., 2012). A dose-response relationship between ACEs and mental illness has been observed, indicating that children exposed to more detrimental conditions are more severely affected in their adulthood psychopathology (Norman et al., 2012). This thesis examines whether increased exposure to individual types of ACEs, as well as the cumulative number of ACEs, are associated with the odds and severity of DP and DR, with a particular focus on potential dose–response relationships.

To address limitations in previous research (including inconsistent definitions of abuse and neglect, varied measurement tools, reliance on lifetime self-reports, heterogeneous samples, differing data collection methods, and cultural variability), this thesis utilises data from the ALSPAC, a large, population-based cohort with both prospectively collected and retrospectively reported maltreatment measures. Within

the ALSPAC, information is obtained from both parents, including self-reports of their own behaviours and reports of their partner's behaviours (yielding two reports for maternal perpetration and two for partner perpetration). This multi-informant approach reduces the likelihood of missing or distorted data due to perpetrator underreporting and strengthens maltreatment ascertainment.

By examining both individual ACEs and cumulative ACE exposure, and analysing associations across multiple developmental time points, this thesis captures diverse trajectories of risk. Importantly, each form of abuse is analysed independently as well as within cumulative ACE models, allowing for more precise interpretation of their specific contributions to the development and maintenance of DPDR. This methodological approach addresses key weaknesses in prior work and enhances the internal validity and generalisability of the findings.

1.6.4 Psychological sequelae of traditional adverse childhood experiences

By comparing twin pairs discordant for ACE exposure, Daníelsdóttir et al. (2024) demonstrated that the increased risk of adult psychiatric outcomes among ACE-exposed individuals cannot be fully explained by shared environmental and genetic factors, with particularly robust effects observed for SA and cumulative adversity. This suggests that it is the role of ACE exposure, rather than genetics or environmental factors, that leads to psychiatric outcomes in adulthood. Further, findings suggest that this increased risk may be due to compromised neurocognitive and emotion regulation abilities following the interruption of normal development and neurobiological changes that occur after exposure to ACEs during sensitive periods of development (Dunn et al., 2017, see 1.9.1 Sensitive periods of development). Therefore, ACE exposure throughout childhood and the transition into adolescence is expected to impair neurocognitive functioning and emotion regulation, consequently leading to poorer mental health outcomes.

DPDR's established link to ACEs is evident across studies. For example, childhood interpersonal adversity is highly prevalent across dissociative presentations, including DPDR. In a study of participants with dissociative seizures, 52.6% reported SA and 61.5% reported PA, compared with 16.3% and 32.6% of healthy controls (HC), respectively, with higher overall trauma burden also observed (mean total

trauma score: 8.33 vs. 5.69; Pick et al., 2017). In DDD, EA showed the most pronounced elevation relative to controls (mean scores 228.7 vs. 81.2), while rates of PA (68.3% vs. 37.5%) and IPV (50.6% vs. 9.7%) showed trend-level differences (Simeon et al., 2001). Additionally, both clinical DPDR and non-clinical but impairing DPDR were characterised by elevated parental rejection and punishment compared with controls (Michal et al., 2009). Further, Wolfradt et al. (2003) linked authoritarian parenting to higher DPDR scores. Additionally, research demonstrates that, when compared to 40 HC, ten of 40 individuals who experienced interpersonal abuse had clinically significant DPDR (Aponte-Soto et al., 2019).

Interestingly, prior evidence suggested that exposure to interpersonal trauma increased DPDR, and interpersonal connectedness reduced them (Simeon et al., 2003), suggesting a dynamic relationship between ACEs and DPDR. Supporting this, Michal et al. (2007) and Ó Laoide, Egan, & Osborn (2018) both found a positive correlation between emotional maltreatment and DP severity.

Smiatek-Mazgaj et al. (2016) further exemplified the link between ACEs and DPDR in a larger sample of 2,582 women with neurotic or personality disorders, and reported predominantly small-to-moderate effects of ACEs on DPDR. Associations were typically in the range of OR ≈ 1.3–1.7 for parental separation, hostility, and lack of support, with larger effects observed for more severe or cumulative adversities (e.g. repeated academic failure or parental aggression during illness; ORs > 2). While this study highlights the importance of measuring a broader range of ACEs, its clinical sample limits generalisability. King et al. (2020) additionally demonstrated that PA significantly predicted D-PTSD symptoms (a main tenet of which is experiencing DPDR). Moreover, patients with functional neurological disorder (FND) experiencing SA also report higher DPDR levels (Dearden & Medford, 2017). Crucially, beyond being an outcome of trauma, DPDR may mediate the relationship between childhood abuse and severe outcomes such as psychotic-like experiences (O'Neill et al., 2021) and adolescent self-harm (Hoyos et al., 2019).

1.6.5 Psychological sequelae of non-traditional adverse childhood experiences

Although less studied than traditional ACEs, non-traditional ACEs have also been linked to various forms of psychopathology and, in certain contexts, to dissociative symptoms.

1.6.5.1 Parental substance abuse

The effect of PSA on children's development is well documented, revealing several associated difficulties. For example, Raitasalo and Holmila (2017) investigated the relationship between Finnish PSA and the somatic and psychological health of 0-6 year-old children. Findings suggested that maternal PSA increased the child's risk of psychiatric hospitalisations, with the highest risk if both parents were substance abusers. The authors suggested that this may be attributable to unsafe environments, chronic stress associated with PSA, and inadequate responsiveness to children's needs. This notion is supported by research identifying a twofold increased risk of experiencing SA and PA in children who experience PSA (Walsh, MacMillan & Jamieson, 2003), suggesting that those who experience PSA may be exposed to additional forms of maltreatment. Additionally, PSA has been shown to have increased negative effects on children compared to issues solely related to alcohol use disorder or tobacco use. Moreover, PSA displayed a significant longitudinal relationship to detrimental child well-being, suggesting that the effects of PSA on child-wellbeing persist over time (Kuppens et al., 2019).

1.6.5.2 Parental mental illness

Evidence suggests that children of parents with mental health issues have worse outcomes (Rutter & Quinton, 1984; Khoury, Kaur & Gonzalez, 2021) due to an increased level of discord, resulting in increased risk of persistent emotional and behavioural disturbances in children. Further, hostile parental behaviour due to PMI is demonstrated as a significant risk factor for child psychopathology (Rutter & Quinton, 1984). In terms of dissociation, a history of maternal trauma (Chu & DePrince, 2008) and maternal dysfunction (Draijer & Langeland, 1999) are related to dissociative symptoms.

1.6.5.3 Inter-parental violence

Childhood exposure to IPV is strongly associated with psychopathological outcomes. For example, when using a sample of 5029 children from the ALSPAC, witnessing IPV between 2 years and 12 years was associated with a 27% increased risk of depression at age 18 (Gondek et al., 2023). Additionally, longitudinal findings demonstrate that, in a sample of 1,152 low-income mothers with children between the ages of 10-14, increased exposure to IPV led to increased child externalising behaviours, resulting in severe parenting practices, and subsequently leading to further externalising behaviours, demonstrating a complex relationship between IPV and the impact on child psychopathology (Chung et al., 2021). Further, children of mothers who experience domestic violence were almost three times as likely to report behavioural problems compared to children of mothers with no history of IPV (Mottley et al., 2025), though this study may reflect cultural differences. The current literature suggests that IPV may lead to poor psychiatric outcomes for children who experience its occurrence.

The specific relationship between IPV and DPDR remains underexplored. One study did report that 27% of individuals with DDD had witnessed domestic violence, however this rate did not differ significantly from HC (Simeon et al., 1997). Further, a small, clinical sample size of 30 patients was used, ultimately limiting its generalisability. Other studies investigating dissociative experiences more broadly offer indirect insights to DPDR. For example, Rada (2002) demonstrated that familial verbal aggression significantly predicted adolescent dissociative experiences, while Draijer and Lengeland (1999) found that 30% of patients with dissociative symptoms reported witnessing IPV during childhood. However, the relationship between IPV and dissociative symptoms is somewhat inconsistent, as other research has produced mixed results regarding IPV's impact (Rafati, 2003). Overall, the existing literature indicates a need for comprehensive research exploring the relationship between IPV and DPDR.

1.6.5.4 Parental divorce

The relationship between parental divorce and DPDR has been investigated previously, yielding different results. In one study, after adjusting for other mental health problems, divorce remained a significant predictor of DPDR (Michal et al.,

2009), yet opposing findings demonstrated no relationship between parental divorce and DPDR (Lee et al., 2012). However, Lee et al. measured DPDR at age 36 only, rather than DPDR across earlier time points. Interestingly, previous research demonstrates that factors occurring before the divorce, as well as the divorce itself, predict poor mental health symptoms (Cherlin, Chase-Lansdale & McRae, 1998; Strohschein, 2005). This suggests that it is not solely the divorce causing poor mental health, but potentially the presence of marital distress, contributing to symptoms.

1.6.5.5 Parental conviction

In terms of longer-term parental imprisonment, a wide range of effects on children's emotional, psychological, and social wellbeing were documented in a review by Weaver and Nolan (2015), highlighting that children with an imprisoned parent are at heightened risk of emotional distress, behavioural difficulties, social stigma, and developmental disruption. The extent of these outcomes depends on contextual factors, including the character of the imprisoned parent prior to incarceration, the stability of care arrangements, and pre-existing vulnerabilities such as poverty, mental illness, or exposure to abuse. The review also notes that children often experience disenfranchised grief, where their emotional needs are overlooked or minimised, compounding the psychological toll. Financial strain is another common consequence, especially for families already living in disadvantaged circumstances.

Despite the existing evidence between non-traditional ACEs and DPDR, past research has not comprehensively investigated their impact on DPDR longitudinally and within general population samples. Therefore, this thesis addresses this research gap.

1.7 Psychological sequelae of the accumulation of adverse childhood experiences

The 'Accumulation Model' was first demonstrated by Felitti et al. (1998), who found that exposure to multiple ACEs led to progressively poorer health outcomes, with individuals experiencing several ACEs showing a 4–12 fold increased risk of depression, PTSD, substance abuse, and suicide attempts. Later, Anda et al. (2006)

utilised the same data to investigate the epidemiological and neurobiological impact of childhood trauma. They examined various outcomes as epidemiological evidence of trauma-related brain dysfunction, including mental health disturbances, somatic disturbances, substance abuse, impaired memory of childhood, perceived stress, reduced anger control, risk of intimate partner violence, and the number of comorbid outcomes. Participants' ACE score demonstrated a dose-response towards all categories of outcomes: individuals with four or more ACEs had a 2.5-fold increased risk of panic reactions, a 3.6-fold increased risk of depressed affect, a 2.4-fold increased risk of anxiety and a 2.7-fold increased risk of hallucinations. Additionally, ACE scores also displayed a dose-response towards the prevalence and risk of somatic health disturbance and sleep disturbance symptoms were increased 2.1-fold for people with 4+ ACEs.

Research demonstrating how trauma effects regions of brain development may explain how the accumulation of ACEs can lead to poorer mental health outcomes in adults. For example, research by Begemann et al., (2023) showed that in 554 participants, those reporting multiple forms of trauma, and across quartiles of cumulative trauma scores, had a pattern of more pronounced frontal grey matter reductions. Additionally, research by Rosada et al. (2021) found that childhood trauma severity correlated negatively with total brain volume. The cumulative exposure to multiple ACEs heightens the risk of prolonged and repeated trauma.

The specific influence of the Accumulation Model on DPDR's development and maintenance remains less investigated, however evidence does suggest that, in a sample of 298 children, those exposed to multiple forms of ACEs exhibited significantly increased DPDR compared to those who experienced a single ACE type, indicating a higher risk (De Silva, 2015). Additionally, Thomson & Jaque (2018) reported that, in multivariate analyses covarying age and gender, individuals with DDD endorsed more cumulative childhood adversity and more cumulative adult traumatic events than non-DDD peers, consistent with an accumulation effect. Considering DPDR as a hypothetical protective 'freeze' response, potentially a symptom of the dorsal vagal state in response to overwhelming sympathetic arousal, the prolonged stress from accumulated ACEs offers a compelling context for its persistence. Such chronic activation of stress responses can disrupt neurobiological

processes and emotion regulation (Teicher et al., 2016), potentially fostering a persistent reliance on DPDR as a coping mechanism. This mechanism can be observed in the cumulative impact of ACEs on measures of dissociation, rather than just DPDR specifically, whereby dissociation increases in a dose-response fashion as ACEs accumulate, through skills deficits, such as emotion regulation (Quiñones, 2022).

Recent research demonstrates that it is essential, when considering cumulative variables, that abuse versus household instability ACEs are separated as composite ACE scores (Fitzgerald & Bishop, 2024). This is due to apparent differences between abuse-related ACEs versus household instability ACEs on predicting adult mental health. Therefore, this thesis specifically investigates the dose-response relationship between the accumulation of abuse-related ACEs and neglect and household instability-related ACEs, on the increased odds and severity of of experiencing DPDR, addressing a critical gap in the existing literature.

1.8 Neurobiological model of depersonalisation and derealisation

1.8.1 Sierra & Berrios (1998)

Sierra and Berrios (1998) argue that DPDR is a biologically embedded defensive response to extreme anxiety, characterised by the simultaneous inhibition of emotional processing and heightened vigilant attention. Central to their model is excessive top-down control exerted by the medial, particularly left, prefrontal cortex over the amygdala and related limbic structures. This prefrontal overactivation suppresses emotional salience and dampens autonomic output, producing hypoemotionality and reduced subjective affect. As the amygdala normally “emotionally colours” perception and cognition, its inhibition disrupts affective tagging, leading to experiences of unreality, detachment, and diminished self-presence. Thus, DPDR is conceptualised not as a primary perceptual disturbance, but as a state of pathological frontal overcontrol that disconnects emotional systems from conscious experience while maintaining, or even enhancing, attentional vigilance.

This aligns with broader evidence indicating that dissociative conditions, or conditions that feature a high level of dissociation, are characterised by distinctive ANS biomarkers, such as borderline personality disorder (Austin et al, 2007), C-PTSD (Van der Kolk et al, 1996) and DDs (Farina et al, 2015; Reinders et al, 2006; Reinders et al, 2012). As summarised in Dalenberg's 2012 review supporting the TM, individuals experiencing dissociation exhibit distinct biomarkers that differentiate them from those without dissociative symptoms. Within DPDR specifically, research identified that DPDR was observed within several physical health conditions, such as temporal lobe epilepsy (see Cassady & Baslet, 2023 for review), cerebrovascular disease, postencephalitic states, migraine and head trauma (Sierra & Berrios, 1998). Additionally, individuals with DPDR show neurobiological differences and distinctive neural activation/connectivity patterns relative to individuals without DPDR (Murphy, 2023; Roydeva et al., 2021; Ketay et al., 2014; Sierk et al., 2018).

1.8.2 Biological approaches to depersonalisation and derealisation treatment

Further evidence from pharmacological treatment approaches offers important insights into potential biological mechanisms underlying the disorder. A recent systematic review identified studies involving selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, opioid antagonists, anticonvulsants, benzodiazepines, and antipsychotics, with most compounds tested in small-scale trials or case series (Wang et al., 2024). Findings suggested that certain neurobiological systems may play a role in DPDR: for example, naloxone demonstrated potential efficacy in a placebo-controlled study, pointing toward involvement of the opioid system. Other agents, including SSRIs and anticonvulsants, showed mixed or limited effects, which may reflect the heterogeneity of DPDR or the involvement of multiple interacting neurobiological pathways. Together, this body of work suggests that DPDR is unlikely to be explained by a single neurochemical mechanism and underscores the importance of further research into its biological underpinnings.

Alternative neurobiological interventions also provide valuable insight into potential mechanisms involved in DPDR. For example, ANS modulation through mindful breathing has been shown to enhance arousal regulation in individuals with DDD (Michal et al., 2013). Similarly, repetitive transcranial magnetic stimulation (rTMS)

has offered insights into the cortical regions implicated in DDD symptom expression: stimulation of the right prefrontal cortex increased electrodermal activity (Jay et al., 2014), suggesting a role for prefrontal regulation of arousal, while stimulation of the right temporoparietal junction (TPJ) produced significant reductions in symptom severity. In a clinical trial targeting the TPJ, half of participants improved after three weeks, with an average 68% reduction in symptoms after nine weeks (Mantovani et al., 2011). These findings point toward the prefrontal cortex and TPJ as key nodes in the neurocircuitry of DPDR and suggest that neuromodulation of these regions may have therapeutic potential.

While this thesis does not focus on neurobiological treatments, evidence for their impact supports a neurobiological basis for DPDR, underscoring the importance of investigating broader physiological mechanisms.

1.8.3 Stress activation in depersonalisation and derealisation

Research into ANS function in DPDR, including a recent systematic review (Millman et al., 2024), reveals a complex and variable pattern. Despite these variations, studies consistently point to a severance between subjective emotional experience and objective physiological reactivity, alongside atypical temporal response patterns and disrupted physiological downregulation under cognitive load (Michal et al., 2013; Sierra et al., 2002; Giesbrecht et al., 2010; Schulz et al., 2015; Lemche et al., 2016).

1.8.4 Hypothalamic-pituitary-adrenal axis

People experiencing DPDR exhibit a unique, more pronounced cortisol response following acute stress induction than other dissociative states (Giesbrecht et al., 2007). Studies using the dexamethasone suppression test indicate hypothalamic-pituitary-adrenal axis (HPA-axis) dysregulation in DDD: compared with HC, individuals with DDD show reduced cortisol suppression to low-dose dexamethasone, and the degree of non-suppression correlates with DPDR severity (Simeon et al., 2001). Subsequent work has replicated/extended these HPA-axis findings in dissociative-spectrum samples, again demonstrating impaired suppression relative to controls (Simeon et al., 2007). In healthy individuals, dexamethasone suppresses cortisol production; in DDD, this suppression is attenuated, consistent with HPA-axis dysregulation. Further, elevated basal cortisol

levels have also been observed in DDD compared to depressed individuals, suggesting distinct HPA-axis functioning (Stanton et al., 2001). Importantly, HPA-axis alterations are not confined to clinical samples: in healthy undergraduates, higher trait DPDR is associated with heightened cortisol responses to acute psychosocial stress, consistent with a dimensional (dose-response) relationship between DPDR and stress reactivity (Giesbrecht et al., 2007).

This body of evidence provides compelling support for a multifaceted neurobiological dysfunction in DPDR. Although this thesis did not directly investigate specific autonomic markers or HPA-axis dysregulation, their established relationship to chronic inflammation highlights them as potential measures underpinning DPDR. Current evidence of inflammation and DPDR is discussed below.

1.9 Bringing together adverse childhood experiences, biological processes, and depersonalisation and derealisation

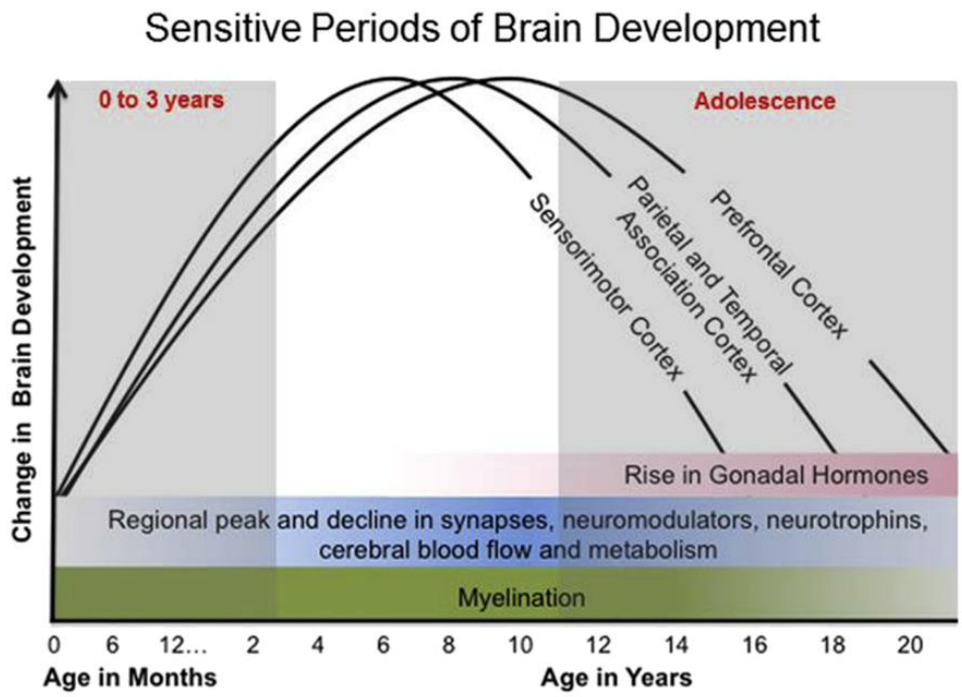
The relationship between ACEs, biological processes, and DPDR can be understood through the following hypothetical framework: Exposure to ACEs may disrupt normative development by exposing the child to chronically elevated stress levels during sensitive periods of development. This can result in atypical maturation of stress-regulation systems, including blunted or exaggerated autonomic arousal, altered HPA-axis responsivity, and persistent low-grade inflammation. Such biological disruptions may increase vulnerability to dissociative mechanisms, including DPDR. Thus, individuals who experience ACEs in childhood may be more likely to develop DPDR, with this relationship mediated by the long-term dysregulation of stress-responsive biological systems.

1.9.1 Sensitive periods of development

Sensitive periods are specific developmental windows whereby experience has a uniquely strong and lasting effect on brain development, shaping or altering certain capacities (Knudsen, 2004). Therefore, these sensitive periods are essential for learning and behaviour, preceded by several complex neurobiological processes.

Pre-natal and post-natal developmental periods are critical in brain development (see figure 1 below). Prenatally, prolific neurogenesis occurs, with the brain's billions of neurons produced by mid-gestation (Howland et al., 2017). Within the first year post-natal, the brain grows to approximately 70% of its full adult size following rapid increases in dendrites, axons and synapses (van Dyck & Morrow, 2017). Pre-adolescence is a period of brain growth and development. By the age of 6, the brain has quadrupled in size from birth (Dobbing & Sands, 1973). Grey matter increases 13% from years 6 to 9 (Courchesne et al., 2000). According to Gee and Casey's (2015) model of sensitive periods in brain development (see figure 1), childhood, particularly before age 12, represents a critical window during which adversity can significantly shape the maturation of neural systems involved in stress regulation.

Figure 1: Model of sensitive periods of brain development



Gee & Casey (2015) - Model of sensitive periods of brain development where substantial changes in brain development occur. The ages shaded in grey are periods of rapid change and may provide increased opportunity for adaptive behavioural changes. These are additionally vulnerable periods to the effects of stress on a developing child.

During sensitive periods of development, children undergo key behavioural and cognitive milestones, including the emergence of emotion regulation, a core skill for adaptive functioning across the lifespan (Cole, Lougheed, & Ram, 2018; Kopp &

Neufeld, 2003; Silvers, 2022). Poor caregiving during these periods can disrupt brain development, impair emotion regulation, and increase vulnerability to psychopathology (Zeanah et al., 2008). Coping is considered a subset of emotion regulation, activated specifically under stress (Eisenberg et al., 2010), and may be particularly susceptible to disruption when stress-response systems are altered early in life.

Longitudinal neuroimaging studies show that early maternal support is linked to healthier hippocampal development and improved coping in adolescence (Luby et al., 2016). Conversely, childhood maltreatment, especially in middle childhood, has been associated with elevated emotional dysregulation in adulthood (Dunn et al., 2018). These findings are especially relevant to DPDR, which has been conceptualised as a maladaptive coping mechanism, initially protective against overwhelming stress but maintained through persistent emotional dysregulation (Hunter et al., 2017).

Therefore, ACEs are likely to disrupt typical stress system development, or alter existing systems, given that sensitive periods are windows of increased neuroplasticity and occur during development (Gabard-Durnam and McLaughlin, 2020). Consequently, ACEs occurring between early childhood and early adolescence represent a high-risk period for atypical stress system development, as these ages span multiple sensitive periods. Of particular interest is the effect on the inflammatory response, which is influenced by several disrupted stress systems, including the ANS and HPA-axis.

1.9.2 Sensitive periods and nervous system development

The ANS matures through foetal life and into childhood and has sustained vulnerability to conditions that can alter its normal development (Mulkey and Plessis, 2019). Overwhelming stress (such as ACEs) during these sensitive periods of neuroplasticity may lead to such alterations of stress systems and adversely impact brain development (De Bellis et al., 1999) and thus experience of stressors later in life. Research consistently demonstrates a relationship between ACEs and measures of ANS function, including increased startle reactivity, reduced heart-rate variability,

and increased physiological responses measured by galvanic skin response (Jovanovic et al., 2009; Stone et al., 2018; Sigrist et al., 2021; Orr et al., 1998).

Regulatory mechanisms of the HPA-axis are an essential component in the translation of ACEs to mental ill health across the lifespan. Cortisol level alterations are considered a primary factor (Kuhlman et al, 2015). ACEs have significant effects on the HPA-axis as it is sensitive to stress throughout development (Kuhlman et al, 2015) and altered cortisol secretion is shown to be a biomarker of childhood stress (Lemieux & Coe, 1995; Kuhlman et al, 2015). Additionally, total trauma exposure is associated with altered reactivity to and recovery from stress (Bosch et al, 2012; Kuhlman et al, 2015). There is however confounding evidence for the Accumulation Model, with recent research suggesting that the number of adversities experienced was not associated with basal or reactive cortisol secretion (Raymond et al, 2021).

Inflammation is increasingly viewed as a stable and integrative biomarker of chronic stress, offering an advantage over more reactive indicators like autonomic arousal and cortisol by capturing longer-term immune system activation. Importantly, chronic inflammation also reflects underlying HPA-axis dysfunction, as prolonged or dysregulated cortisol activity can impair immune regulation and promote a pro-inflammatory state. While inflammatory markers such as interleukin-6 (IL-6) and C-reactive protein (CRP) are nonspecific and influenced by contextual factors, they offer insight into the biological embedding of early adversity, particularly when interpreted alongside ACEs.

1.9.3 Chronic inflammation

In this thesis, “chronic stress” refers to experiences in which a stressor persists in a child’s environment over an extended period, such as ongoing parental maltreatment or household instability. Stress may also be considered chronic when the perceived threat endures long after the original stimulus has passed, for example, the lingering sense of danger following PA (Miller, Chen & Parker, 2011). It is worth noting that chronic stress will inevitably vary per individual in terms of its duration and intensity (Segerstrom & Miller, 2004). However, defining the transition from acute to chronic stress is important, as the latter is associated with more detrimental health outcomes,

including systemic inflammation and psychopathology (Cohen et al., 2007; Rohleder, 2019).

A substantial body of evidence supports chronic inflammation as the way in which chronic stressors get ‘under the skin’, for example through the biological embedding model (Hertzman, 1999). Through the biological embedding model, exposure to chronic psychosocial stressors early in life can become ‘programmed’ into the body’s cells, leading to pro-inflammatory tendencies such as exaggerated cytokine responses to challenges, and decreased sensitivity to inhibitory hormonal signals (Miller, Chen & Parker, 2011). Examples of chronic stress as a precursor to systemic inflammation are evidenced in research concerning a lower financial social position (Brydon et al., 2004; Steptoe et al., 2002; Miller, Chen & Parker), lower subjective social status (Derry et al., 2013), lack of home ownership and/or low parental education (Miller & Chen, 2007).

Inflammation can be measured by indicators of systemic inflammation, including Tumor Necrosis Factor-alpha (TNF- α), IL-6 and CRP (Menzel et al., 2021). This thesis focuses on IL-6 and CRP. IL-6 is a multifunctional cytokine that is produced by various cell types in response to infections, tissue injuries, and other inflammatory stimuli. Beyond its role in acute inflammation, IL-6 has been implicated in the pathogenesis of chronic inflammatory conditions, such as rheumatoid arthritis and cardiovascular diseases. Its ability to cross the blood-brain barrier (Banks et al., 1994) and influence neuroinflammatory processes (Wright et al., 2006) links elevated IL-6 to neuropsychiatric conditions, including depression (Mac Giollabhui et al., 2021) and schizophrenia (Lv et al., 2024). CRP is a widely used biomarker in the study of systemic inflammation. Elevated CRP levels are associated with an increased risk of developing various chronic diseases (Dhingra et al., 2007), including cardiovascular diseases and diabetes. In neuroinflammation, CRP serves as a valuable marker for understanding the link between peripheral inflammation and central nervous system alterations. Elevated CRP levels are implicated in various psychiatric conditions (Orsolini et al., 2023), including DDD (Zheng et al., 2024), indicating its potential role in the pathophysiology of these symptoms. Crucially, the precise influence of IL-6 on DPDR remains to be fully elucidated.

1.9.4 Adverse childhood experiences and chronic inflammation

ACEs are a form of chronic stress evidenced to biologically embed pro-inflammatory tendencies in an individual, resulting in systemic immune suppression (e.g. impaired cellular immunity), chronic basal inflammation, and heightened inflammatory reactivity (Berens, Jensen & Nelson, 2017). Several studies have linked ACEs to immune dysfunction (Steel et al., 2020; D'Elia et al., 2018; Wong et al., 2022; Schrepf, Markon & Lutgendorf, 2017). Additionally, both CRP and IL-6 have been found to correlate significantly with indicators of dysfunctional households (Kim et al., 2019; Iob, Lacey & Steptoe, 2020; Dempster et al., 2023). There is further longitudinal support for the notion that ACEs predict immune dysfunction, as evidence suggests that ACEs, including cumulative ACEs, occurring from birth to 8 years are associated with higher levels of IL-6 and CRP at age 10 and CRP at age 15, suggesting longitudinal impacts after the events take place (Slopen et al., 2013).

Additionally, the effect of ACEs on the immune response may be strongest when the child is also experiencing mental health problems. For example, among 12-year-old children, the most pronounced immune dysregulation, indicated by elevated CRP levels, was observed in those who experienced both maltreatment and depressive symptoms. Children exposed to only maltreatment or only depression showed less immune activation, suggesting a compounding effect of co-occurring adversity and mood disturbance (Danese et al., 2011).

1.9.5 Chronic inflammation, psychopathology, depersonalisation and derealisation

As both the inflammatory theory of psychopathology and the study of DPDR remain relatively novel, research directly linking inflammatory markers to DPDR is scarce. To date, only one study has examined CRP in relation to DPDR (Zheng et al., 2024), and none have explored associations with IL-6. Given this gap, it is necessary to draw on related fields to guide understanding. This includes reviewing evidence on inflammation in broader psychopathology.

The role of inflammation in mental health has received growing attention, with evidence linking inflammatory markers to various psychiatric conditions (Lee & Giuliani, 2019; Müller et al., 2015). These effects are thought to arise from inflammation-related disruptions in neural circuits and neurotransmitter systems,

including dopaminergic and serotonergic pathways (Capuron & Miller, 2004; Felger & Treadway, 2017). For instance, elevated inflammatory markers like IL-6 and CRP are frequently observed in individuals with depression, even independent of infection, and are associated with symptoms such as fatigue, anhedonia, and cognitive difficulties, often termed "sickness behaviours" (Dowlati et al., 2010; Chamberlain et al., 2019; Maes et al., 2012). This robust evidence for inflammation's role in the pathophysiology of major psychiatric disorders provides a strong theoretical and empirical basis for investigating its involvement in DPDR.

CRP has been implicated in dissociative symptoms. For example, individuals diagnosed with the D-PTSD have been found to have higher levels of CRP than those with the standard PTSD (Jarkas et al., 2021). Additionally, research has demonstrated that CRP predicts overall dissociation severity in a hierarchical linear regression model, independent of abuse, PTSD, depression and emotion dysregulation (Powers et al., 2019), suggesting a direct relationship.

Bizik et al. (2011) further investigated the relationship between dissociation and inflammation by examining TNF-α levels in a sample of inpatients with unipolar depression. They discovered a significant, negative correlation between TNF-α levels and participant dissociative experiences scale (DES) scores. This study was later replicated with a slightly larger sample, including additional measures for dissociative symptoms, and yielded consistent results. The findings indicated a significant negative association between TNF-α levels and both dissociative and somatoform dissociative symptoms (Bizik et al., 2014). This is relevant to IL-6 and CRP, as TNF-α acts as an early pro-inflammatory cytokine, stimulating the production of IL-6, which then promotes the synthesis of CRP in the liver during the acute phase response (Giovannini et al., 2011).

Additionally, past research has revealed a link between dissociative symptoms and elevated IL-6 cytokine levels. For example, Bob et al. (2010) demonstrated a relationship between IL-6 cytokines and somatoform dissociative symptoms, suggesting that elevated IL-6 might be associated with more severe somatoform dissociative symptoms. However, no significant correlation was found between IL-6 levels and DES scores, suggesting symptom specific inflammatory markers.

However, while these findings between inflammation and dissociative symptoms are promising, the limited size of the clinical samples in these studies restricts their generalisability to broader populations, underscoring the need for larger-scale studies.

Research looking at the relationship between inflammatory markers and DPDR specifically is preliminary, yet promising. A recent study by Zheng et al., (2024) investigated the potential relationship between inflammatory markers and individuals with DDD. By analysing serum samples from 30 DDD patients and 32 HC, the researchers identified several dysregulated proteins that may serve as biomarkers for the disorder. Specifically, the study found that levels of CRP, complement C1q subcomponent subunit B (C1QB), apolipoprotein A-IV (APOA4), and alpha-1-antichymotrypsin (SERPINA3) were significantly altered in DDD patients. CRP and C1QB were downregulated, while SERPINA3 was upregulated. These results suggest that dysregulation of these immune-related proteins may reflect an underlying biological mechanism in individuals with DDD, potentially involving chronic low-grade inflammation. However, the use of small, clinical sample sizes limits the generalisability outside of such cases, which is particularly important within DPDR research, as receiving an official diagnosis can take between 7-12 years (Baker et al., 2003; Michal et al., 2016). Therefore, several individuals with DDD likely go undiagnosed, and research using a general population sample will yield larger sample sizes of individuals with DPDR, allowing for more robust conclusions to be made.

1.10 Secondary factors impacting depersonalisation and derealisation

The relationship between ACEs and DPDR may be shaped by additional factors such as attachment style, anxiety, depression, perseverative cognitions (PC) and hyper-reflexivity. These factors are investigated in this thesis for two main reasons. Firstly, their inclusion allows the delineation of potential independent associations between ACEs on DPDR, isolating direct effects from possible mediation. Secondly, given the frequent comorbidity of DPDR with conditions like anxiety and depression, examining

these factors helps clarify whether DPDR functions as a standalone condition or as a symptom of other disorders.

1.10.1 Attachment

Attachment style is linked to various types of psychopathologies (Mikulincer & Shaver, 2012) and has a unique relationship with dissociative experiences (Liotti, 1992). Attachment theory was developed to understand the considerable distress that can evolve due to parenting practices. Based on quality of parental caregiving, children can develop one of four different types of attachment: secure, anxious, avoidant, and disorganised (Benoit, 2004). When caregivers are sensitive to the needs of a child, they are more likely to develop a secure attachment style. However, when caregivers are insensitive in a rejecting or inconsistent manner, they may develop an insecure attachment style. Those with insensitive-rejecting caregivers tend to develop an avoidant attachment, minimising emotional expression in the presence of the caregiver, and those with insensitive-inconsistent caregivers tend to develop an anxious attachment, maximising their distress signals to elicit caregiving (Ijzendoorn, Schuengel & Bakermans-Kranenburg, 1999). When infants experience atypical caregiving, such as frightening behaviours or a caregiver who is frightened or dissociated, they may develop a disorganised attachment (Abrams, Rifkin & Hesse, 2006; Madigan et al., 2006; Schuengel, Bakermans-Kranenburg & van Ijzendoorn, 1999). In the Strange Situation experiment, infants with a disorganised attachment style display conflicting or undirected behaviours, such as freezing (Main & Solomon, 1990), and disorganised attachment functions as a dissociative process itself, and further predisposes individuals to react to later life trauma via dissociative mechanisms (Liotti, 2006).

Other forms of insecure attachment are demonstrated to have an impact on dissociative mechanisms, too. For example, an anxious attachment style is significantly correlated with dissociative experiences in female college students (Calamari & Pini, 2003). Importantly, early life insecure attachment style is a longitudinal risk factor for dissociation, including DPDR, at ages 17 and 19 (Carlson, 1998). Furthermore, the combination of traumatic experiences and insecure attachment styles further exacerbates these dissociative tendencies (Sandberg,

2010; Gušić et al., 2016; Kong et al., 2018; Subocz, 2022), suggesting that individuals with this combination may be the worst afflicted.

Recent studies underscore the significant role of attachment styles and emotional regulation in the development of DPDR specifically. Anxious and avoidant attachment are associated with disturbed self-awareness and DPDR in individuals with psychosis, when compared to unaffected siblings and HC (de With, de Haan & Schirmbeck, 2023). Additionally, research demonstrates the role of DPDR as a mediating factor between insecure attachment and psychological distress, suggesting DPDR contributes to the effect of insecure attachment on poor mental health outcomes (O'Rourke & Egan, 2023). Although existing studies on the relationship between insecure attachment style, DPDR, and other psychopathologies provide a valuable foundation, the complex interplay between ACEs, insecure attachment, and DPDR warrants further investigation. Specifically, more research is needed to disentangle whether insecure attachment mediates the relationship between ACEs and DPDR, or if it acts as an independent contributor to the development of DPDR.

1.10.2 Perseverative cognitions

Once an individual experiences DPDR, even transiently, the response can become persistent, highly distressing, and occur frequently without an identifiable present stressor. Hunter et al. (2003) conceptualised this through a cognitive-behavioural lens, proposing that symptom persistence arises from a negative feedback loop. In this loop, the catastrophic appraisal of normally transient DPDR (e.g., misinterpreting them as signs of severe mental illness or brain dysfunction) escalates anxiety, which in turn exacerbates DPDR. Furthermore, maintaining the disorder are cognitive and behavioural reactions, such as avoidance, "safety behaviours," and cognitive biases, which increase symptom awareness, amplify the perceived threat, and prevent the disconfirmation of catastrophic interpretations.

The Perseverative-Cognition Hypothesis (PCH; Brosschot, Gerin & Thayer, 2006) posits that PC can lead to disruptions in usual physiological functions. The PCH utilises “perseverative cognition” as an umbrella term incorporating cognitions that

share a common theme: they tend to be repetitive thoughts regarding a stressor and engage the mind into trying to control outcomes related to the stressor through thinking. Consequently, in developing the PCH, the authors reviewed research concerning worry, rumination, and obsessing, defining PC as “the repeated or chronic activation of the cognitive representation of one or more psychological stressors”.

Research notes PC in DPDR (Hunter et al., 2003; Medford et al., 2005; Roth, 1959; Sierra et al., 2012; Torch, 1978; Vannikov-Lugassi et al., 2021). Medford, Sierra, and David summarised DPDR in the New Oxford Textbook of Psychiatry (Geddes, Andreasen & Gelder, 2012), noting that ruminations about being on the verge of a ‘mental breakdown’ are common. These concerns manifest as obsessional ruminations and self-monitoring, for example thinking “there is something going wrong with my brain” or “I’m losing control” (Hunter, Salkovskis and David, 2014), which perpetuates DPDR in a vicious cycle. Further, individuals susceptible to DDD are noted to have a ‘harm-avoidant temperament’ (American Psychiatric Association, 2013), and patient fears of ‘going crazy’ or having irreversible brain damage, along with extreme ruminations and obsessional preoccupations, are diagnostic features. Therefore, DPDR can be observed through a cognitive-behavioural framework.

Despite the prevalence of PC in DDD, little empirical work has examined its specific impact; existing studies are small and often focus on general PC rather than DDD-related processes (Vannikov-Lugassi et al., 2021; Quigley, Warren & Townsend, 2024). Broader evidence, however, links PC to dissociative symptoms using larger samples. Three studies found PC to be strongly and consistently associated with more severe dissociation across clinical and non-clinical groups, even after controlling for anxiety and neuroticism (Watson, Wu & Cutshall, 2004). Associations were strongest between obsessive–compulsive symptoms involving repetitive thinking (e.g., obsessing, checking) and detachment-related dissociation (DPDR and obliviousness), while links with milder dissociative traits or anxiety-related OCD symptoms were weaker or absent. This points to a potentially specific relationship between PC and dissociative detachment.

1.10.3 Hyper-reflexivity

The phenomenon of hyper-reflexivity, characterised by excessive self-focused attention on mental processes and symptoms, aligns closely with DDD. Jaspers (1997) notably posited this heightened self-focus as an integral phenomenological feature of DDD. Individuals experiencing hyper-reflexivity may become overly focused on their own thinking patterns, leading to a heightened self-consciousness and introspection. Hyper-reflexivity is widely researched under a myriad of names, like metacognition and self-focussed attention (Pérez-Álvarez, 2008).

Hyper-reflexivity has been shown to characterise mental health conditions such as psychosis and schizophrenia (Sass & Feyaerts, 2024), however, a growing body of evidence suggests that it is a common feature within DPDR too (Ciaunica, 2021; Cinaunica, 2022). Additionally, hyper-reflexivity has been proposed to hold causal precedence in the development of psychopathology, insofar as heightened self-focused attention precedes symptom onset, predicts vulnerability following stress, can be experimentally induced to exacerbate symptoms, and is associated with symptom reduction when attention is therapeutically reoriented outward (Pérez-Álvarez, 2008). Importantly, hyper-reflexivity is not conceptualised as the original cause of distress, but as the process through which life difficulties are transformed into psychopathology.

In prior qualitative research, Ciaunica (2021, 2022) observed that individuals often report experiencing hyper-reflexivity and are aware of how it manifests in their daily lives. For instance, one participant vividly illustrated this by stating, “Sometimes I’m so preoccupied with the way I walk or the way I present myself, I don’t realize what’s happening around me, for example if someone appeared next to me” (Ciaunica, 2021). Additionally, individuals may engage in compulsive self-monitoring of their internal states, as demonstrated by another participant reflecting: “how do I feel now?”, “’Who am I?”, suggesting a pattern of rumination and over-intellectualisation of their thoughts (Ciaunica et al., 2022).

Despite qualitative findings and anecdotal reports of hyper-reflexivity in DPDR, no quantitative research has confirmed a relationship between hyper-reflexivity and DPDR.

1.10.4 Depression

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) notes that DPDR can emerge during depressive episodes, contributing to the overall experience of the disorder (American Psychiatric Association, 2013). In fact, 85% of patients with DDD have also been demonstrated to have a depressive disorder, indicating a strong overlap (Michal et al., 2016). However, once depressive systems cease, DPDR can persist (Simeon, 2004), suggesting it can exist outside of depression. This significant comorbidity underscores the importance of distinguishing whether DPDR is a primary symptom or secondary to another condition like depression, and proper diagnosis and treatment often require addressing both conditions simultaneously to achieve optimal outcomes (Hunter, Charlton, & David, 2017; Medford et al., 2005).

Additionally, DDD can have a unique clinical profile with specific phenomenological features, not entirely explained by comorbid conditions like anxiety and depression (Simeon et al., 1997), such as emotional numbing and a sense of watching themselves outside their body (Baker et al., 2003). Additionally, Michal et al. (2011), through a large-scale study, identified that DPDR is independently associated with impairments in mental and physical health beyond the impacts of anxiety and depression.

A longitudinal study by Michal et al (2024) provides additional insight into the complex interplay between DPDR and depression and highlights the necessity of delineating the relationship between depression and DPDR. Over a five-year period, this study examined the relationship between DPDR and depression in a representative population-based sample. The findings revealed that DPDR is a significant prognostic factor for the course and severity of depression, and individuals with co-occurring DPDR experienced more severe impairments across various life domains, including social integration, physical health, and mental health. They also faced a higher risk of chronic depression and had lower remission rates compared to those with depression alone. Notably, only 7% of depressed individuals with DPDR achieved remission at the five-year follow-up, compared to 16% of those with depression only, highlighting a substantial impact of these symptoms on the trajectory of depression.

1.10.5 Anxiety

Some of the earliest published work on DPDR highlighted the presence of anxiety. For example, Oberndorf (1934, cited in Jacobson, 1959) suggested that DPDR may be a specific defence against anxiety, a “phenomenon of death closely related to the ‘playing dead’ defence used by animals when in great danger”, and Roth (1959) highlighted the presence of anxiety symptoms in individuals with DDD and introduced the term "phobic-anxiety depersonalisation syndrome" to describe a distinct anxiety disorder, characterised primarily by DPDR and agoraphobia. Later research utilising more valid research frameworks additionally supported Roth’s “phobic-anxiety depersonalisation syndrome”, suggesting a positive correlational relationship between anxiety and DPDR (Trueman, 1984). Additionally, Brauer, Harrow and Tucker (1970) found that DPDR occurs more in younger individuals who are frequently anxious and posited that DPDR emerges as symptoms rather than as a standalone condition. A systematic review of the prevalence of DPDR, conducted by Hunter, Sierra and David (2004), identified DPDR as a common feature of anxiety disorders, particularly in cases where anxiety levels are extremely high, or in the context of life-threatening situations. Further, a recent systematic review identified that up to 20% of individuals with an anxiety disorder have comorbid DDD (Yang et al., 2023). Also, individuals with DDD exhibit elevated anxiety, anger, and negative affect compared to HC, with anxiety emerging as the strongest predictor of DPDR and pathological dissociation (Simeon et al., 2003).

However, where relationships between anxiety and DPDR have been identified, some results lack expected linearity. For example, Sierra et al. (2012) compared ‘high’ and ‘low’ severity in 291 DPDR patients, finding a small but significant relationship only with ‘low’ DPDR, and no meaningful contribution of anxiety to overall clinical features. Additionally, a recent epidemiological study by Simeon and Stein (2025) found that while 21.2% of individuals with clinically significant DPDR also had mood or anxiety disorders, only 3% of all people with mood or anxiety disorders exhibited clinically significant DPDR. Specifically, clinically significant DPDR was present in 0% of individuals with generalised anxiety disorder, 5% in social anxiety disorder, and 6% in panic disorder, further supporting the distinct nature of DPDR.

The literature exhibits conflicting findings, with some studies suggesting that anxiety may be overemphasised in the context of DPDR, while others demonstrate a strong comorbidity between anxiety and DPDR. A key gap in the research is the lack of robust studies employing larger, non-clinical samples to clarify this relationship.

1.11 Maintenance of depersonalisation and derealisation

DDD develops when DPDR become more frequent and pervasive, moving beyond transient experiences (American Psychiatric Association, 2013). Yet there is surprisingly little formal research exploring what contributes to ongoing episodes of DPDR after the initial precipitating factor has passed. In clinical settings, therapeutic manuals and practitioner reports offer anecdotal insight into common triggers for recurrence. These include overthinking about DDD, physical symptoms of anxiety, worrying, harsh lighting (especially fluorescent), social environments, reading about psychosis, being hungover, tiredness (Kennedy et al., 2013), and even extreme positive emotions like intense joy (Simeon & Abugel, 2006) can trigger episodes of DPDR.

Additionally, several sources support the relevance of these stated triggers. For example, existential thinking (Ciaunica et al., 2023), rumination (Fortuna & Golonka, 2024; Vannikov-Lugassi et al., 2021; Uysal et al., 2025), and hyper-reflexivity (Ciaunica, 2021; Ciaunica, 2022). Environmental contributors such as certain lighting conditions (Baker et al., 2003), flashing lights, pulsing music, and prolonged visual fixation (Leonard et al., 1999) have also been noted. Panic symptoms (Miller et al., 1994; Lickel et al., 2008), virtual reality immersion (Peckmann et al., 2022), increased screen time, social isolation (Ciaunica, 2022), and interpersonal difficulties (Ciaunica, 2023) further add to this picture. Mirror exposure (Brake et al., 2025; Caputo et al., 2021; Shin et al., 2018), academic pressure (Schweden et al., 2018), sensory overload (Harricharan et al., 2017), sleep disruption (Arora et al., 2020; Menicucci et al., 2022; van Heugten-van der Kloet et al., 2015), and dysregulated autonomic functioning (Horn et al., 2020) have also been implicated. Taken together, these factors are likely to play a role in the maintenance and potential worsening of DPDR over time.

The cognitive-behavioural model provides a possible explanation of symptom maintenance, and an experimental study comparing patients with DDD, anxiety patients, and HC, found that individuals with DDD made fewer normalising attributions for ambiguous symptoms and reported more catastrophic health-related appraisals (Hunter, Salkovskis & David, 2014). Crucially, when their attention was shifted onto cognitively demanding tasks, DDD patients reported a reduction in symptom severity, whereas anxiety patients and controls experienced the opposite effect. These findings support the idea that maladaptive appraisals and symptom-focused attention contribute to the development and persistence of DDD.

Additionally, a qualitative examination of what leads to the reoccurrence of dissociative symptoms offers insight. Černis et al. (2020) interviewed individuals with non-affective psychosis, who self-reported four key subjective triggers sustaining their dissociative symptoms, including: (1) both immediate and longstanding stress, (2) exhaustion, (3) cognitive dwelling, and (4) underlying beliefs about emotion and stress. While these findings align with symptom patterns seen in DPDR and offer useful insight, their relevance is limited by the sample, as the study was completed exclusively with individuals with non-affective psychosis. Thus, further research demonstrating what leads to the reoccurrence and maintenance of DPDR in a general population sample is required to better understand its chronicity and to support those affected.

1.12 Depersonalisation and derealisation as separate constructs

Historically, DP and DR have often been treated as a single phenomenon. Early psychiatric descriptions and diagnostic manuals, including DSM-II through DSM-IV, used DP as an umbrella term, with DR regarded as a secondary feature rather than a distinct experience. Yet as early as 1935, British physicians Mapother and Mayer-Gross highlighted important distinctions between these experiences. Their separation gained formal recognition decades later in the International Classification of Diseases (ICD-10, 1992), which introduced the category of depersonalisation–derealisation syndrome. The DSM-5 subsequently adopted this distinction, renaming the disorder DDD. This change signifies a move towards understanding DP and DR as distinct,

though often co-occurring, entities, reflecting a growing clarity in distinguishing these two facets of dissociation.

DP and DR are usually measured together as a single construct; however, they are distinct in their phenomenology and can occur independently of the other (Aderibigbe, Bloch & Walker, 2001). Additionally, it is commonly believed that DP is the more common symptom, however the epidemiology within the ALSPAC dataset demonstrates that at 17 and 24 years of age the prevalence of DR was higher than the prevalence of DP (this is discussed further in Chapter 2).

DP and DR may also have distinct neurobiological mechanisms (Sierra et al., 2002). Additionally, other evidence indicates that DP may result from frontal lobe misfiring, whereas DR is more closely linked to temporal lobe dysfunction, as seen in postictal states (Hollander et al., 1992). Yet, additional studies propose shared left frontotemporal activation in both phenomena (Heydrich et al., 2019), suggesting overlapping but not identical neural mechanisms. Overall, while findings remain mixed, emerging data support the view that DP and DR may reflect partially distinct neurobiological processes.

In view of this data, there is sufficient evidence to suggest that DR can occur independently of DP, and vice versa. Additionally, there is neurobiological evidence to suggest unique biomarkers of each experience. However, there is clearly a lack of research into these symptoms separately. This thesis addressed this by investigating independent trajectories of ACEs on DP and DR over time, as well as inflammatory markers to elucidate potential independent biological mechanisms.

1.13 Limitations of existing research on adverse childhood experiences and depersonalisation and derealisation

While the existing literature invariably suggests that ACEs are related to the development of DPDR, it is important to note that these studies suffer from one or more of six areas of weaknesses: (1) Not separating how ACEs may differentially effect DP and DR, assuming that their impact is universal across both symptoms (as discussed above). (2) Utilising narrow definitions of ACEs that do not encapsulate the

full spectrum of potential events that can lead to psychopathology. (3) Utilisation of retrospective reports in adulthood of ACEs, where a significant amount of time has passed since occurrence. (4) Reliance on cross-sectional designs in the existing literature limits insight into the temporal ordering of ACEs and DPDR, underscoring the need for longitudinal approaches to examine developmental associations over time (5) Past DPDR research has trended towards utilising clinical samples, for example inpatients or individuals receiving therapy, reducing generalisability to the general population where the majority of DPDR exists. (6) There is generally the use of small sample sizes, reflecting the clinical nature of research. (7) Prior research has primarily operationalised ACEs as abuse-related ('traditional' ACEs) when looking at their relationship to DPDR. However, previous research demonstrates that an array of non-traditional ACEs, such as indicators of household instability, are linked to psychopathology.

Retrospective reports of ACEs are vulnerable to a substantial rate of false negatives and substantial measurement error, especially when events happened at a very young age (Hardt and Rutter, 2004), reducing the reliability of findings. Small sample sizes can allow false conclusions to be drawn (Faber and Fonseca, 2014) and a focus on clinical patients limit the generalisability of results, which is particularly problematic in DPDR research as it is astoundingly difficult to obtain a clinical diagnosis, and participants with a diagnosis may over-represent individuals with highly severe symptoms and/or those who are already receiving treatment.

1.14 Research aims and overview of thesis

1.14.1 Research aims

The DPDR community faces significant challenges, including limited research recognition and a lack of comprehensive understanding regarding its prevalence, aetiology, and factors contributing to maintenance and severity of DDD. Addressing DPDR as a public health concern requires robust research to determine predisposing factors and prevalence, ultimately guiding the refinement and enhancement of treatment interventions.

This thesis aimed to fill these gaps by adopting a comprehensive biopsychosocial framework that could elucidate the onset, maintenance, and severity of DPDR. This thesis' integrated large-scale, longitudinal data from a general population sample, as well as qualitative insights from an online sample, and data from sub-clinical and clinical recruits.

Specifically, this thesis:

1. Examined a potential role of psychosocial factors in DPDR onset by investigating if ACEs were a key social factor. The study further explored whether attachment style, depression, anxiety, and perseverative cognition mediated the relationship between ACEs and DPDR, when controlling for sex.
2. Investigated biological mechanisms underpinning DPDR, focusing on low-grade systemic inflammation as a biomarker of chronic stress. This included assessing whether inflammatory markers independently predicted DPDR and whether inflammation mediated the ACE-DPDR pathway.
3. Identified factors contributing to the maintenance of DPDR through qualitative analysis of forum data, followed by quantitative validation in individuals meeting clinical criteria for DDD.
4. Assessed cognitive contributors to DPDR severity by examining perseverative cognition (measured via rumination) and hyper-reflexivity as independent predictors. Additionally, this study investigated the mediating effect of rumination on the relationship between ACEs and DPDR prevalence was explored.

By integrating biological, psychological, and social dimensions, this thesis provides a holistic biopsychosocial model, advancing understanding of DPDR development and chronicity. The findings clarified DPDR risk factors, distinguishing between DP and DR symptoms as separate constructs, and identified coping mechanisms with implications for improved treatment strategies.

1.14.2 Overview of thesis

To achieve the aims of providing a comprehensive, biopsychosocial framework for DPDR, the thesis proceeded through four integrated empirical chapters, moving from etiological risk factors to maintenance mechanisms:

Chapter 2: This chapter initiated empirical investigations by exploring whether negative environments during development were potential primary risk factors for the odds of experiencing DP and DR over time at ages 12, 17 and 24. This study used data from 7,603 ALSPAC participants. By separating DP from DR, this study investigated potential differences in ACE-related aetiology, while also assessing whether key secondary factors (attachment style, depression, anxiety, and perseverative cognitions) mediated the significant relationships between ACEs and DPDR.

Chapter 3: This chapter shifted the focus to biological mechanisms and investigated inflammatory markers as a potential physiological pathway linking ACEs to DPDR. Using available CRP data (N = 5,127) and IL-6 data (N = 2,560) from the ALSPAC, this study investigated whether CRP and IL-6 independently predicted the odds of experiencing DP and DR at ages 12, 17 and 24, while controlling for key covariates. Where possible, the role of IL-6 or CRP as potential mediators in the ACE–DP and ACE–DR relationship was tested.

Chapter 4: Moving from aetiology to maintenance, Chapter 4 identified possible factors that contributed to the maintenance of DPDR, and examined what led to symptom persistence beyond the cessation of the original stressor. A thematic analysis of 367 self-help forum posts identified common subjective symptom triggers and overarching themes that users felt led to symptom reactivation, offering rich, qualitative insight into the cognitive and emotional processes that sustained DPDR and may have led to frequent DPDR, and DDD.

Chapter 5: The final chapter utilised a survey-based design, including 124 participants from the general population who met the clinical threshold for DDD. A dose-response relationship between single ACE frequency and types of cumulative ACEs and DPDR severity was investigated, as well as whether general rumination mediated this relationship. Crucially, independent roles of general rumination versus hyper-reflexivity in predicting DPDR severity is assessed. Finally, validation of several maintenance factors discovered qualitatively in Chapter 4 was sought by integrating them into the quantitative survey, and additionally collecting self-reported data on the effectiveness of common calming strategies

Chapter 2: Adverse Childhood Experiences and the Development of Depersonalisation and Derealisation: An Updated View Using Large-Scale Data and Longitudinal Analysis

Abstract

Background: Depersonalisation and derealisation are dissociative symptoms that remain understudied longitudinally and in relation to adverse childhood experiences (ACEs). This study investigates how distinct ACE types are associated with the development of depersonalisation and derealisation over time.

Methods: Using data from the Avon Longitudinal Study of Parents and Children (N = 7,906), generalised linear mixed models examined whether abuse, neglect, and household instability ACEs predicted increased or decreased odds of depersonalisation and derealisation symptoms at ages 12, 17, and 24 when controlling for covariates.

Results: ACEs showed stronger associations with depersonalisation than derealisation. Only parental substance abuse predicted increased odds of both depersonalisation and derealisation. Four other ACEs (emotional abuse and neglect, inter-parental violence, and parental divorce) predicted increased odds of depersonalisation, while parental mental illness and divorce predicted decreased odds of derealisation. Cumulative adversity predicted depersonalisation but not derealisation. The effects of ACEs emerged during adolescence and often persisted into young adulthood.

Conclusion: This is the first longitudinal study distinguishing associations between ACEs and depersonalisation and derealisation, highlighting abuse, neglect, and household instability as key predictors of depersonalisation, with derealisation linked to household instability. The findings support the need for targeted family interventions and suggest differing developmental pathways for depersonalisation and derealisation.

2.1 Introduction

2.1.1 Understanding depersonalisation, derealisation and their relationship with adverse childhood experiences

DP and DR are dissociative symptoms that remain under-researched, despite their frequency in the general population (Hunter, Sierra, & David, 2004), and suggestions that they are the third most common mental health symptom following depression and anxiety (Simeon et al., 2003). DP refers to “the experience of unreality, detachment or being an observer of one’s thoughts, feelings, sensations, body or actions. This can include alterations in perceptions, such as time distortion, a sense of an unreal or absent self, and emotional or physical numbing” (Hallett et al., 2025). Additionally, DR refers to “experiences of unreality or detachment with respect to one’s surroundings. This can include individuals or objects experienced as unreal, dreamlike, lifeless, foggy or visually distorted” (Hallett et al., 2025).

DP and DR can significantly disrupt daily activities, relationships, and overall well-being, underscoring their importance in mental health research and treatment (Simeon & Abugel, 2006). Yet, due to limited understanding from healthcare professionals and the public, there are inadequate resources for treatment. Thus, individuals can develop chronic experiences of DPDR, potentially leading to DDD. Further, a lack of research in this area has led to an average of 7-12 years to accurately diagnose DDD (Baker et al., 2003; Michal et al., 2016). Estimates suggest that 1-2% of the population (Sierra et al., 2000; Yang et al., 2022) may qualify for a diagnosis of DDD, yet experts fear the prevalence may be far higher. For context, according to the 2014 Adult Psychiatric Morbidity Survey, the prevalence of schizophrenia is estimated at 0.7% (Bebbington et al., 2016), OCD is estimated at 1.3% and panic disorder is estimated at 0.6% (McManus et al., 2016), yet these rare disorders receive far more research attention in comparison to DDD.

Dissociation is viewed as a coping response, whether temporary or lasting, potentially protecting individuals from the impact of traumatic or overwhelming experiences (Dalenberg et al., 2012), combining heightened alertness with a suppression of emotional reactivity (Sierra & Berrios, 1998). For example, research demonstrates that rescue paramedics state DPDR allows them to operate on

‘autopilot’ under extreme stress, suppressing emotional responses until later when they can be addressed appropriately (Pietkiewicz, Duszkiewicz, & Tomalski, 2023). Further, DPDR may potentially prevent psychopathology associated with the traumatic event (Shilony & Grossman, 1993). Therefore, when adequately functioning as a transient protective mechanism, DPDR can be adaptive.

As well as acute, in-the-moment stressors, DPDR is related to trauma history (Dalenberg et al., 2012). For example, research demonstrates that up to 54% of adults who experience DPDR have a experienced interpersonal abuse (Yang et al., 2022), and that 38% of PTSD patients experience high degrees of DPDR, qualifying for the dissociative subtype of PTSD (D-PTSD; White et al., 2022). Given the intimate relationship between past trauma and DPDR, naturally ACEs likely exhibit a relationship to DPDR.

ACEs are defined as highly stressful and potentially traumatic events occurring during childhood or adolescence, including maltreatment, abuse, and environments non-conducive to healthy development (Boullier & Blair, 2018). Global self-report data suggest high rates of maltreatment, with around 20% of girls and 14% of boys experiencing SA, 23–25% PA, 16–25% EA, and 28–34% neglect (Moody et al., 2018). In the ALSPAC, 84% reported at least one ACE and 24% reported four or more by age 16 (Houtepen et al., 2020). Both single and cumulative ACEs are linked to poorer mental and physical health (Amos et al., 2023), with a clear dose-response pattern showing greater exposure predicts worse outcomes (Norman et al., 2012; Hughes et al., 2017; Kalmakis & Chandler, 2015).

DPDR’s link to ACEs has been observed previously and may act as both a predisposing and precipitating factor (see 1.4.4 Adverse childhood experiences). In summary, children who endured PA or SA, EA, neglect, or interpersonal trauma during childhood frequently present with pathological DPDR later in life (King et al., 2020; Michal et al., 2007, 2009; Simeon et al, 2001; Simeon et al., 2008). Simeon et al. (2001) demonstrated a strong correlation between childhood interpersonal trauma and DDD. The study assessed trauma across six domains and found significantly higher total trauma scores in individuals with DDD compared to controls, with EA (often from both parents) emerging as a key predictor of the disorder (although the

sample sizes were relatively small [N = 75]). Supporting research is observed: Michal et al. (2007) found a positive correlation between emotional maltreatment and DPDR. Later, the authors conducted another study using a larger dataset (N = 1,287) and identified that that childhood adversities were independently associated with DPDR when controlling for anxiety and depression (2009). Further, Ó Laoide, Egan, & Osborn (2018) observed that childhood emotional maltreatment was linked to higher levels of DPDR in adulthood; and Wolfradt et al. (2003) found that exposure to authoritarian parenting styles in childhood is associated with higher DPDR scores.

Related research further supports the notion that ACEs are related to DPDR. For example, research by King et al. (2020) examined how childhood maltreatment type and severity is linked to D-PTSD. Among 106 women with PTSD, multivariate regression analysis showed that childhood EA and PA significantly predicted D-PTSD symptoms, highlighting the role of these ACEs in the development of D-PTSD. Additionally, patients with FND, a condition often associated with DPDR, who had experienced SA report higher levels of DPDR (Dearden & Medford, 2017), and somatoform dissociation is evidenced to mediate the relationship between SA and experiencing dissociative seizures. Furthermore, DPDR may not only be an outcome of ACEs, but also act as a mediator between ACEs and more severe psychological conditions, such as psychotic-like experiences in adulthood (O'Neill et al., 2021) and self-harming behaviours in adolescents (Hoyos et al., 2019), underlining its potential for extreme harm.

While these results suggest that ACEs are related to DPDR, the evidence should be interpreted considering methodological weaknesses. Previous research has relied on retrospective reports of ACEs, which are vulnerable to a substantial rate of false negatives and substantial measurement error (Hardt and Rutter, 2004). Additionally, use of small sample sizes may lead to false conclusions to be drawn (Faber and Fonseca, 2014), and clinical samples may limit generalisability of results, an issue in DPDR research due to prolonged difficulty to attain a diagnosis (7-12 years; Baker et al., 2003; Michal et al., 2016). Further, clinical patients may disproportionately reflect increased severity, those already engaged in treatment, and those from higher socio-economic backgrounds with access to private specialised services. Finally, lack of longitudinal studies prevents a better estimation of cause and effect of ACEs on

DPDR, highlighting the need for use of larger, prospective longitudinal studies with individuals from the general population.

The ALSPAC dataset provides a rare opportunity to address all of these methodological weaknesses, as it is the only child-based, prospective, longitudinal dataset within the UK to also provide variables on ACEs and DPDR.

2.1.2 Secondary factors impacting depersonalisation and derealisation

In addition to ACEs, several mental health symptoms are commonly associated with DPDR, leading to conclusions that DPDR are transdiagnostic (meaning they are a feature of several other mental health disorders; Černis et al., 2025). Yet, a debate in the literature as to whether DPDR can also be considered a standalone condition, or is sequelae of other mental health disorders, exists. Given that attachment, anxiety, depression, and PC are associated with both ACEs and DPDR, this thesis seeks to clarify whether these secondary experiences mediate the relationship between ACEs and DPDR, or whether ACEs exert a direct effect on DPDR.

2.1.2.1 Attachment

Attachment status is a critical area of research in understanding DPDR, as insecure attachment in childhood has been linked to a heightened vulnerability to DDs (Liotti, 1992, Calamari & Pini, 2003). Additionally, the combination of traumatic experiences and insecure attachment styles further exacerbates these dissociative tendencies (Sandberg, 2010; Gušić et al., 2016; Kong et al., 2018; Subocz, 2022). This includes both pathological and ‘normative’ forms of DPDR and dissociation (Simeon & Knutelska, 2022). Longitudinal studies confirm that insecure attachment in childhood is a significant risk factor for dissociation, including DPDR, in late adolescence and early adulthood, as observed at ages 17 and 19 (Carlson, 1998).

2.1.2.2 Perseverative cognitions

A cognitive-behavioural model of DDD, developed by Hunter et al. (2003), suggests that DPDR starts as a transient experience, is misappraised as catastrophic, and leads to distressing thoughts like “something is wrong with my brain” or “I’m losing control.” Individuals prone to DDD often have a harm-avoidant temperament

(American Psychiatric Association, 2013), unlike others who perceive their symptoms as unusual but not catastrophic and trust they will remit. The model posits that DPDR is maintained by negative cognitions that increase attention to symptoms, rehashing the event, ultimately leading to DDD.

Related, research has consistently noted rumination and existential questioning in DPDR (Hunter et al., 2014; Medford et al., 2005; Roth, 1959; Sierra et al., 2012; Torch, 1978; Vannikov-Lugassi et al., 2021), and the DSM-5 (American Psychiatric Association, 2013) note that extreme ruminations and obsessional preoccupations are diagnostic features. Recent research supports the association between rumination and DPDR in a general population sample, demonstrating increased reporting of negatively coded PC items were significantly associated with DPDR, after controlling for general distress (Quigley, Warren and Townsend, 2024). However, after controlling for the shared variance between clinical variables, PC was no longer significantly associated with DPDR, suggesting that it may not represent the sole symptom domain contributing to DPDR.

2.1.2.3 Depression

DPDR frequently occurs in individuals with depression, suggesting it may function as a symptom within the broader depressive disorder. The DSM-5 notes that DPDR can emerge during depressive episodes, contributing to the overall experience of the disorder (American Psychiatric Association, 2013). In fact, 85% of patients with DDD may also have a depressive disorder, indicating a strong overlap (Michal et al., 2016). This highlights the need to distinguish primary from secondary DPDR, and to address comorbidities in diagnosis and treatment (Hunter, Charlton & David, 2017; Medford et al., 2005). Additionally, a five-year longitudinal study by Michal et al. (2011) found that DPDR predicted poorer outcomes for individuals with depression, with great functional and health-related impairments, including higher risk of chronic depression, and lower remission rates. Only 7% of depressed individuals with DPDR achieved remission, compared to 16% with depression alone, underscoring the significance of DPDR on the depression trajectory.

However, research also indicates that DPDR can stand alone as a condition (Sierra et al., 2002; Michal et al., 2011), suggesting it is not simply a function of depression.

Michal et al. (2011) highlighted the distinct nature of DPDR in individuals without anxiety and depression, suggesting that DPDR contributes independently to mental ill health. Simeon et al. (1997) further emphasised that DDD has a unique clinical profile not entirely explained by comorbid conditions like depression or anxiety, a perspective supported by Baker et al. (2003), who demonstrated that individuals with DDD report emotional numbing and a sense of watching themselves from outside their body, experiences not solely attributable to depressive symptoms.

2.1.2.4 Anxiety

Systematic reviews regarding the prevalence of DPDR demonstrate it as a common feature in anxiety disorders (Yang et al., 2023), particularly in cases where anxiety levels are extremely high, or in the context of life-threatening situations (Hunter, Sierra and David, 2004). However, while increased rates of DPDR are noted in connection with anxiety disorders (20%; Yang et al., 2023), the association appears most specifically to panic disorder. Panic disorder is characterised by high levels of physical symptoms: shortness of breath, heart palpitations, feeling faint, tingling and nausea (National Health Service, 2023). Thus, DPDR is prevalent in scenarios characterised by extensive stress system activation, rather than solely through the vaguely defined ‘anxiety’, which may be less acutely physical than panic disorder, with a higher emphasis on cognitive symptoms such as uncontrollable worries (Mind, 2021). Of note, Chapter 4 of the this thesis demonstrated that recurrence of DPDR is highly linked to stimuli that an individual finds highly stressful. Thus, the relationship between DPDR and ‘anxiety’, may better be framed as a relationship between DPDR and increased stress system activation.

Where associations between anxiety and DPDR are demonstrated, results are inconsistent. In a study with 291 DDD patients, Sierra et al. (2012) reported a small association between anxiety and low DPDR, but no link with high DPDR, and regression analyses demonstrated that anxiety did not meaningfully contribute to core DPDR features. They concluded that anxiety may be over-emphasised in DPDR research, suggesting DPDR as a distinct condition. More recently, Simeon and Stein (2025) found that over 20% of individuals with clinically significant DPDR had a mood or anxiety disorder, yet an average of only 3% of those with mood or anxiety disorders reported clinically significant DPDR: 0% in GAD, 5% in social anxiety, and

6% in panic disorder. These findings suggest that while DPDR often co-occurs with anxiety, the overlap is limited and asymmetrical.

While these secondary factors are linked to DPDR, the evidence remains limited by small samples and a lack of non-clinical populations. Crucially, no existing research has longitudinally compared these factors within the same dataset to clarify which secondary factor exerts the strongest influence on DPDR over time. However, while the present study tests these factors within the context of ACEs, the findings may not be uniform with those who have not experienced ACEs.

2.1.3 Depersonalisation and derealisation as separate constructs

Although often measured together, DP and DR are distinct phenomena that can occur independently of each other (Aderibigbe, Bloch & Walker, 2001). DR prevalence can exceed DP (for example at ages 17 and 24 in the ALSPAC cohort; see section 2.2.2.1 Depersonalisation and derealisation variables), despite opinion that DP is the predominant symptom. Very little research has separated DP and DR, especially within the context of ACEs. To the authors' knowledge, only one study has attempted to do so, utilising a clinical cohort of FND patients (Dearden & Medford, 2017). Therefore, this study aims to address this research gap by examining how a large range of ACEs may predict divergent trajectories of DP and DR.

2.1.4 Exploratory Hypotheses

2.1.4.1 Total number of adverse childhood experiences

This study explored the potential longitudinal relationships between the accumulation of ACEs and the odds of experiencing DP and DR at ages 12, 17, and 24, when controlling for sex.

2.1.4.2 Types of adverse childhood experiences

This study examined the relationship between repeated exposure to individual ACE types (PA, EA, EN, PN, PSA, IPV, PMI, parental divorce, and parental conviction), captured using multiple time points. Each ACE was assessed along a continuum of

frequency or severity to reflect a graded impact of exposure. The dose-response association between repeated exposure to an individual ACE and the odds of experiencing DP and DR at ages 12, 17, and 24, when controlling for sex, was tested.

No specific directional hypotheses were made, allowing for the possibility that different types of ACEs may have unique or varying effects on DP and DR over time.

2.1.4.3 Secondary factors influencing depersonalisation and derealisation

Finally, this study explored how attachment, anxiety, depression, and PC, influenced the relationship between ACEs and DP and DR at ages 12, 17, and 24.

2.1.5 The present study

This study seeks to extend and improve on prior research on ACEs and DPDR by using a large, non-clinical dataset of parents and children from the ALSPAC, incorporating prospective reports of ACEs (apart from EN and PN, which are retrospective reports) to enhance data reliability. This study's approach included longitudinal analysis to better model outcomes over time, allowing a comprehensive understanding of developmental trajectories. Additionally, DP and DR were separated to examine whether different types of ACEs have distinct effects on these symptoms.

2.2 Methods

2.2.1 Participants

ALSPAC, often referred to as the "Children of the 90s" study, is a long-term health research project that began in the early 1990s. Based in the Avon area of England, the study initially recruited over 14,000 pregnant women who were due to give birth between April 1991 and December 1992. Annually, data is collected and can be categorised into four categories: (1) mother-reported (data is collected from the mother, regarding the mother), (2) partner-reported (data is collected from the partner, regarding the partner), (3) child-based (data is collected by the primary

caregiver, regarding the child) and (4) child-completed (data is collected by the child, regarding the child). Data is collected through a combination of questionnaires, clinical assessments, biological samples, and linkage to health records (www.alspac.bris.ac.uk).

The current study utilised 7906 (50.53%) child participants from the ALSPAC cohort. These individuals had completed the psychosis-like symptoms (PLIKS) interview at ages 12 (mean age 12.9, range = 12.5-13.3 years) and/or 17 (mean age 17.8 years, range = 16.25-20.1 years) and/or 24 (mean age 24 years, range = 22.4-24.4 years). From this cohort, 2542 had complete cases, 2365 had completed the PLIKS interview two out of 3 times, and 2993 had completed the PLIKS interview once. See Table 1 for a summary of the final sample characteristics.

Table 1: Chapter 2 demographic information

Demographic Category	**N (%)**
Sex	
Male	3722 (47.3%)
Female	4148 (52.7%)
Ethnicity	
White	6617 (83.7%)
Not white	294 (3.7%)
Missing	959 (12.1%)
Social Class - Mother	
I	426 (5.4%)
II	2124 (26.9%)
III (non-manual)	2530 (32%)
III (manual)	410 (5.2%)
IV	492 (6.2%)
V	88 (1.1%)
Armed Forces	2 (0%)
Missing	1798 (22.7%)
Social Class - Father	
I	830 (10.5%)
II	2327 (29.4%)
III (non-manual)	767 (9.7%)
III (manual)	1812 (22.9%)
IV	540 (6.8%)

V	156 (2%)
Armed Forces	8 (0.1%)
Missing	1430 (18.1%)

2.2.2 Measures

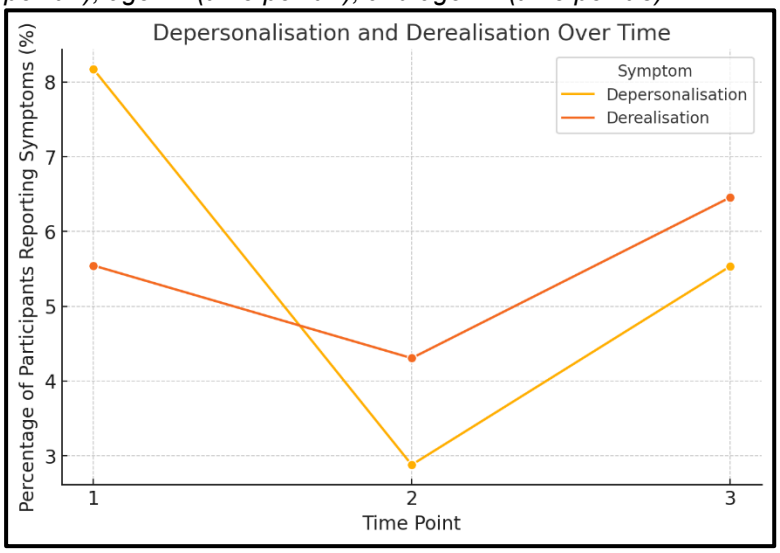
2.2.2.1 Depersonalisation and derealisation variables

At ages 12, 17, and 24, individuals were asked if they had “ever felt that the world was unreal, that things around them were like a stage set” (DR) or “ever felt that they were not a real person, not part of the living world” (DP). These questions were part of the psychosis-like symptoms (PLIKS) interview. Responses of 'frequently' or 'sometimes' were coded as having experienced DP or DR. These variables were set up as binary values (0 = No, 1 = Yes). See frequencies of reported DPDR in table 2 below: McNemar’s test demonstrated significantly higher proportions of individuals reporting DP scores at age 12, but DR at age 17 and 24 (see table 2 and figure 2, below).

Table 2: Depersonalisation and derealisation frequencies across ages

	DP – Count (%)		**DR – Count (%)**		**McNemar Test**		
	No	**Yes**	**No**	**Yes**	**Mcnemar’s χ^2**	**Df**	**p**
12 years	6245 (92)	537 (8)	6390 (94)	391 (6)	34.467	1	**<.001**
17 years	4554 (97)	139 (3)	4487 (96)	207 (4)	23.626	1	**<.001**
24 years	3747 (96)	140 (4)	3700 (95)	188 (5)	10.42	1	**.001**

Figure 2: Depersonalisation and derealisation percentage prevalence comparisons at age 12 (time point 1), age 17 (time point 2), and age 24 (time point 3)



2.2.2.2 *ACE variables*

Most ACE variables were based on prospective reports from both mothers and father figures at various ages. However, certain variables, such as SA, IPV and parental divorce were exclusively reported by mothers at different ages (see Table 3 below). EN and PN were reported retrospectively by the child at age 22 and 23, respectively, indicating whether they had experienced these ACEs. For each year that the ACE was measured, the participant received a binary score (0 = did not experience, 1 = did experience), and then all each binary score for each ACE was summed, creating a graded score for each ACE type. To see all of the items used to create the ACE variables, see Supplementary Excel, Sheet 1. For further detail on the construction of the ACE variables, see Appendix 1.

Table 3: Adverse childhood experiences measured and ages

		Age (years)																							
		8 months		1		2		3		4		5		6		7		8		9		10		11		22	23
		MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	MR	PR	CR	CR
ACE	EA	XX	XX	XX		XX	XX	XX	XX			XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX		
	PA	XX	XX	XX	XX	XX	XX	XX	XX			XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX	XX		
	SA			X		X		X		X		X		X				X									
	EN																									X	
	PN																										X
	PSA	X	X	X	X	X	X	X	X			X	X	X	X	X	X	X	X	X	X	X	X	X	X		
	PMI	X	X	X	X	X	X	X	X			X	X	X	X	X	X	X	X	X	X	X	X	X	X		
	IPV	X	X	X	X	X		X	X			X	X	X	X	X	X	X	X	X	X	X	X	X	X		
	Divorce	X	X	X	X	X	X	X	X			X		X	X	X	X	X	X	X	X	X	X	X	X		
	Convictions	X	X	X	X	X	X	X	X			X	X	X	X	X	X	X	X	X	X	X	X	X	X		

Abbreviations: MR = Mother-reported; PR = Partner-reported; CR = Child-reported; ACE = Adverse Childhood Experience; EA = Emotional abuse; PA = Physical abuse; SA = Sexual abuse; EN = Emotional neglect; PN = Physical neglect; PSA = Parental substance abuse; PMI = Parental mental illness; IPV = Inter-parental violence
X = Reported on themselves only
XX = Reported on themselves and the alternative measured partner

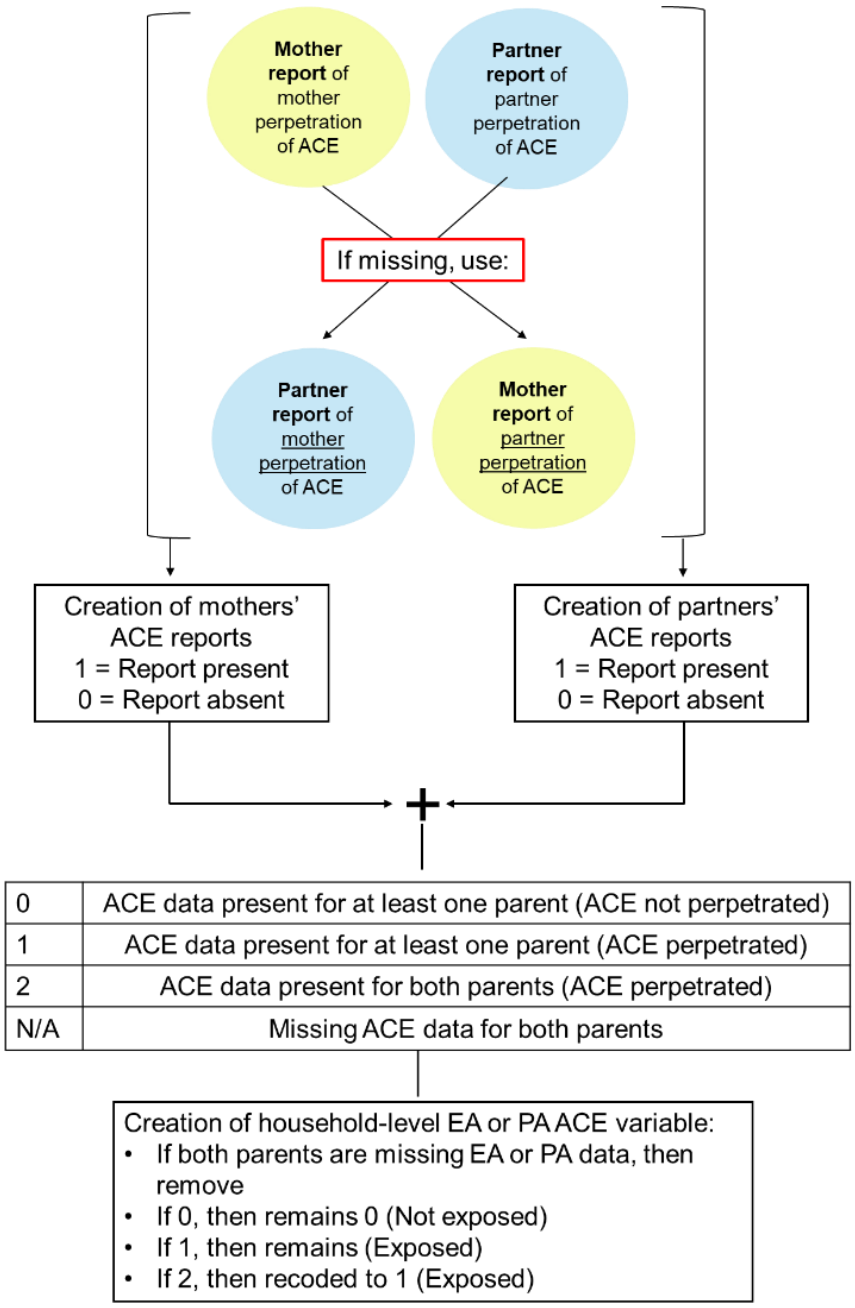
In regard to the missing data process for EA and PA, information was collected from both self-reports (mother on mother, partner on partner) and cross-reports (mother on partner, partner on mother). Self-reports were treated as the primary data source. Where self-reports were missing, the corresponding cross-report from the other parent was substituted (e.g., if the mother's self-report was missing, the partner's report of the mother was used). For further detail on the missing data process, see Appendix 1.

After deriving mother and partner reports, these were combined into a household-level variable to indicate whether the child was exposed to EA or PA. Specifically:

- If both parents' data were missing, the participant was removed for that ACE type.
- If both reports indicated no perpetration, the ACE was coded as absent (0).
- If either parent was reported as a perpetrator, the ACE was coded as present (1).
- If both parents were reported as perpetrators, the ACE was coded as present (1).

This approach, visualised in figure 3 below, ensured that participants were only excluded where no information was available, and that the EA and PA variables captured any exposure within the household.

Figure 3: Emotional and physical abuse missing data process



Each single ACE type was a cumulative variable based on the number of years that the parents endorsed each abuse type. For example, if parents reported perpetrating EA five of the eight timepoints measured (each time point reflecting whether EA was perpetrated over the past year), they would receive a score of five. EN and PN, which were based on a single retrospective report each, were simple binary measures.

To create the accumulation ACE variables, individual ACEs were summed. Two cumulative ACE variables were derived: (1) abuse-related ACEs (EA, PA, and SA), and (2) neglect and household instability-related ACEs (EN, PN, PSA, PMI, IPV, parental conviction, and divorce).

2.2.2.3 Attachment

Attachment style in childhood was determined based on observed behaviours during caregiver reunions at ages 1 and 2. Children who moved or pushed away their caregiver upon reunion were classified as 'insecure' attachment, while those who cuddled their caregiver were classified as 'secure'. To create a single childhood attachment measure, data from both ages were combined, ensuring that missing values were accounted for while maintaining a clear distinction between secure and insecure attachment.

2.2.2.4 Anxiety, depression, and perseverative cognitions

The study child's anxiety was assessed at ages 10, 17 and 24, depression at ages 10, 17 and 24, and PC at age 7, 17 and 24, using self-reported questionnaires and clinical assessments. Further details on the measures used are provided in the Supplementary Excel, Sheet 2.

2.3 Model Building

The objective of this study was to investigate if a history of ACEs interacted with time to significantly impact the odds of experiencing DP and DR symptoms across three developmental stages: ages 12, 17, and 24, while controlling for sex. Analysis was conducted using generalised linear mixed models (GLMM), incorporating various covariates (childhood attachment style, anxiety, depression and PC) over several models to understand their influence on the relationship between ACEs and DPDR.

To comprehensively assess the relationship between ACEs and DPDR, both partially and fully adjusted models were tested. Partially adjusted models examined the longitudinal associations between ACEs and DP and DR, while controlling for sex (to control for sex differences in the perpetration of ACEs; model 1), sex and attachment (model 2), sex, attachment, and anxiety (model 3), sex, attachment, and depression

(model 4), sex, attachment, and PC (model 4), and the final adjusted model included: sex, attachment, anxiety, depression, and PC. This allowed us to determine whether any single covariate uniquely has potential influence on the relationship. The fully adjusted model is the analysis presented in this study, while results from the partially adjusted models are provided in Appendix 2.

2.4 Data analysis

Data analysis was carried out using R Studio Version 2021.09.0, Build 351. For data organisation, 'base' packages were used, as well as dplyr (Wickham, 2015) and tidyverse (Wickham et al., 2019). For data analysis, the package glmmTMB (Brooks et al., 2017) was utilised in order to run GLMMs. Data were visualised using ggplot2 (Wickham, 2016). Analysis was performed in line with the models outlined above. All ACE variables other than PN and EN (which were binary) underwent square root transformation due to the data being skewed.

As analyses were pre-specified as exploratory, no formal adjustment for multiple comparisons was applied. Instead, results were interpreted as preliminary, with greater weight placed on effect sizes, confidence intervals, and the consistency of associations across nested model specifications and outcomes. Given the correlated nature of ACE indicators and the non-independence of the model set, strict family-wise error correction was not considered optimal. Replication in independent datasets is necessary to confirm the observed patterns.

2.5 Results

The results are split up into three sections. First, longitudinal associations between each of the single 'traditional' ACEs and DP and DR (EA, EN, PA, and PN) are tested. Second, the longitudinal associations between each of the single 'non-traditional' ACEs and DP and DR (PSA, IPV, PMI, parental divorce, and parental convictions) are tested. Third, the longitudinal associations between the accumulation of abuse-related ACEs, and the accumulation of neglect and household instability-related ACEs were tested. Of note, the individual ACE of SA was not

included due to extremely small reporting by parents, however it was included in the cumulative abuse-related ACE score.

Of note, in the GLMM framework used here, the baseline DPDR score (measured at age 12) functions as the reference point against which later outcomes are compared. This means that the reported odds at ages 17 and 24 represent changes in the odds of experiencing symptoms relative to age 12. Thus, when the text refers to 'in relation to baseline scores', the results of ages 17 and 24 in comparison to the baseline time point (age 12) are being described, rather than separate cross-sectional estimates at each age.

2.5.1 Do abuse and neglect ACEs predict the odds of experiencing depersonalisation and derealisation?

For DP, EA (ranging from 0–8 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 4; figure 4). However, EA significantly predicted an increase in the odds of experiencing DP at age 17 relative to baseline (125% increased odds per event; see table 4; figure 4), and at age 24 (97% increased odds per event; see table 4; figure 4). In addition, when holding other variables constant, anxiety significantly predicted the odds of DP at each time point (OR = 5.64, 95% CI: 2.75–11.71, p < .001; 464% increased odds), as did depression (OR = 2.69, 95% CI: 1.15–6.23, p = .022; 168% increased odds). PC did not significantly predict DP (OR = 0.61, 95% CI: 0.24–1.54, p = .296). Neither sex (OR = 1.15, 95% CI: 0.63–2.10, p = .647) nor early life attachment style (OR = 1.13, 95% CI: 0.61–2.08, p = .699) significantly predicted increased odds of experiencing DP.

For DR, EA did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 4; figure 5). At age 17, EA was associated with a non-significant increase in odds relative to baseline (see table 4; figure 5). At age 24, EA did not significantly predict DR compared with baseline (see table 4; figure 5). In addition, when holding other variables constant, anxiety significantly predicted the odds of DR at each time point (OR = 2.95, 95% CI: 1.63–5.31, p < .001; 195% increased odds), as did depression (OR = 2.34, 95% CI: 1.21–4.57, p = .012;

134% increased odds). PC did not significantly predict DR (OR = 1.11, 95% CI: 0.53–2.32, p = .793). Neither sex (OR = 1.16, 95% CI: 0.66–2.05, p = .610) nor early life attachment style (OR = 1.02, 95% CI: 0.57–1.80, p = .954) significantly predicted increased odds of experiencing DR.

For DP, EN (ranging from 0-1 events [did not ever experience = 0 / did ever experience = 1), did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 4; figure 4). However, EN significantly predicted an increase in the odds of experiencing DP at age 17 (290% increased odds; see table 4; figure 4) and age 24 (868% increased odds; see table 4; figure 4) relative to baseline. In addition, when holding other variables constant, anxiety significantly predicted the odds of DP at each time point (OR = 2.29, 95% CI: 1.49 – 3.56, p < .001; 129% increased odds), but depression (OR = 1.72, 95% CI: 0.72 – 4.10, p = 224) and PC (OR = 0.78, 95% CI: 0.28 – 2.18, p = 637) did not. Neither sex (OR = 1.02, 95% CI: 0.50 – 2.10, p = .952) nor early life attachment style (OR = 1.17, 95% CI: 0.58 – 2.39, p = .652) significantly predicted increased odds of experiencing DP.

For DR, EN did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 4; figure 5). Additionally, EN did not significantly predict changing odds of experiencing DP at age 17 or age 24 relative to baseline (see table 4; figure 5). In addition, when holding other variables constant, anxiety significantly predicted the odds of DR at each time point (OR = 2.16, 95% CI: 1.54 – 3.03, p < .001; 116% increased odds), but depression (OR = 1.68, 95% CI: 0.83 – 3.42, p = .149) and PC (OR = 1.03, 95% CI: 0.45 – 2.39, p = .937) did not. Neither sex (OR = 0.97, 95% CI: 0.50 – 1.92, p = .936) nor early life attachment style (OR = 1.08, 95% CI: 0.56 – 2.10, p = .813) significantly predicted increased odds of experiencing DR.

For DP, PA (ranging from 0–8 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 4; figure 4). At age 17, PA was associated with a non-significant increase in odds relative to baseline (see table 4; figure 4). At age 24, PA did not significantly predict DP compared with baseline (see table 4). When holding other variables constant, anxiety

significantly predicted the odds of DP (OR = 1.79, 95% CI: 1.09–2.92, p = .021; 79% increased odds), while depression (OR = 1.99, 95% CI: 0.74–5.31, p = .172) and PC (OR = 0.42, 95% CI: 0.13–1.32, p = .140) were not significant predictors. Neither sex (OR = 0.95, 95% CI: 0.46–1.97, p = .900) nor early life attachment style (OR = 1.09, 95% CI: 0.53–2.29, p = .805) significantly predicted increased odds of experiencing DP.

For DR, PA did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 4; figure 5). At age 17 and age 24 (see table 4; figure 5), PA did not significantly predict DR compared with baseline. In addition, when holding other variables constant, anxiety significantly predicted the odds of DR at each time point (OR = 1.94, 95% CI: 1.38–2.69, p < .001; 94% increased odds), but depression (OR = 2.03, 95% CI: 0.95 – 4.35, p = .067) and PC (OR = 1.16, 95% CI: 0.49–2.75, p = .730) did not significantly predict DR. Neither sex (OR = 0.86, 95% CI: 0.44–1.72, p = .677) nor early life attachment style (OR = 0.99, 95% CI: 0.49–1.97, p = .973) significantly predicted increased odds of experiencing DR.

For DP, PN (ranging from 0-1 events [did not ever experience = 0 / did ever experience = 1), did not significantly predict the odds of experiencing DP at age 12, the baseline time point (see table 4; figure 4). Further, PN did not predict DP at age 17 or age 24 in comparison to baseline (see table 4; figure 4). When holding other variables constant, anxiety significantly predicted the odds of DP (OR = 8.09, 95% CI: 3.63 – 18.17, p <.001; 709% increased odds), but depression (OR = 2.39, 95% CI: 0.94 – 5.99, p = .066) and PC (OR = 0.54, 95% CI: 0.19 – 1.57, p = .260) did not. Neither sex (OR = 0.99, 95% CI: 0.47 – 2.05, p = .976) nor early life attachment style (OR = 1.28, 95% CI: 0.63 – 2.64, p = .499) significantly predicted increased odds of experiencing DP.

For DR, PN did not significantly predict the odds of experiencing DR at age 12, the baseline time point (see table 4; figure 5). However, PN did not predict DR at age 17 or age 24 in comparison to baseline (table 4; figure 5). When holding other variables constant, anxiety significantly predicted the odds of DP (OR = 3.86, 95% CI: 1.99 – 7.46, p <.001; 286% increased odds), but depression (OR = 1.73, 95% CI: 0.81 –

3.71, p = .153) and PC did not (OR = 0.54, 95% CI: 0.19 – 1.57, p = .487) Neither sex (OR = 1.07, 95% CI: 0.54 – 2.16, p = .840) not early life attachment style (OR = 1.13, 95% CI: 0.57 – 2.20, p = .737) significantly predicted increased odds of experiencing DR.

Table 4: Fully adjusted longitudinal associations between abuse and neglect ACEs and depersonalisation and derealisation

		Depersonalisation			Derealisation		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
Emotional Abuse	12	0.89	0.60 – 1.34	**.572**	1.03	0.72 – 1.49	.860
	17	2.25	1.36 – 3.67	**.002****	1.27	0.80 – 2.01	.309
	24	1.97	1.17 – 3.32	**.011***	0.83	0.44 – 1.58	.568
Physical Abuse	12	0.78	0.27 – 2.20	.633	1.20	0.55 – 2.61	.645
	17	3.22	0.87 - 11.82	.080	0.72	0.23 – 2.27	.577
	24	1.49	0.29 – 7.68	.632	1.23	0.40 – 3.78	.715
Emotional Neglect	12	0.90	0.41 – 1.99	.792	0.88	0.39 – 1.99	.756
	17	3.90	1.19 – 12.94	**.025***	1.16	0.42 – 3.25	.775
	24	9.68	2.89 – 32.46	**<.001*****	1.63	0.53 – 4.95	.393
Physical Neglect	12	0.98	0.17 – 5.59	.984	1.27	0.24 – 6.62	.774
	17	1.72	0.09 – 31.82	.719	0.28	0.02 – 4.02	.346
	24	6.55	0.43 - 100.48	.176	4.53	0.48 – 42.52	.189

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

Figure 4: Longitudinal odds ratios of depersonalisation for emotional abuse, physical abuse, emotional neglect, and physical neglect at ages 12, 17 and 24

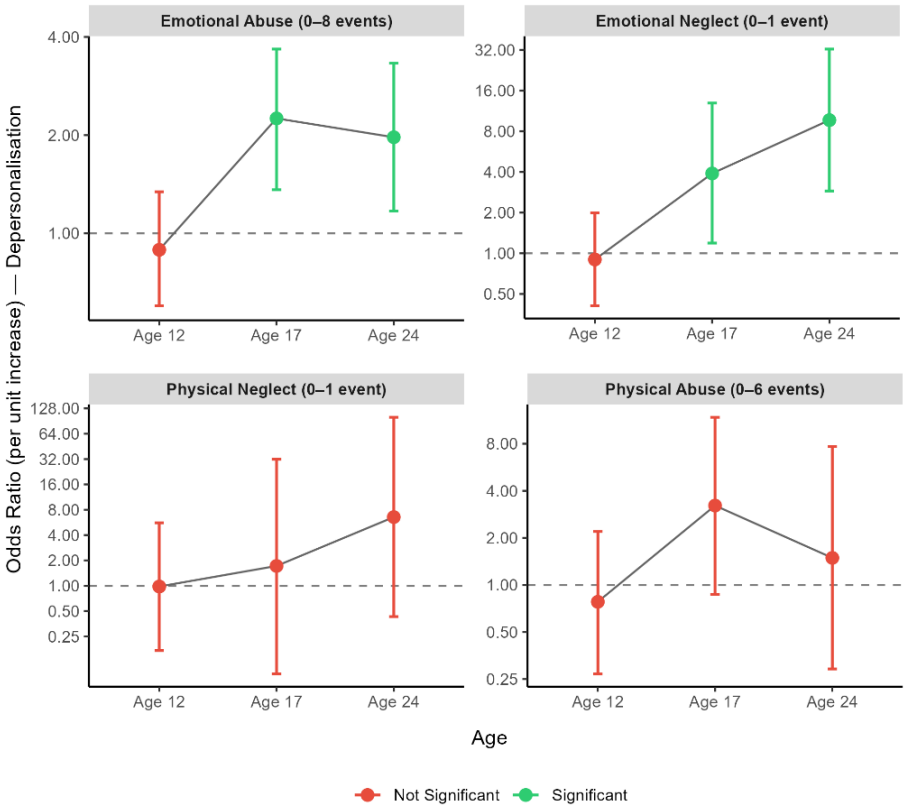
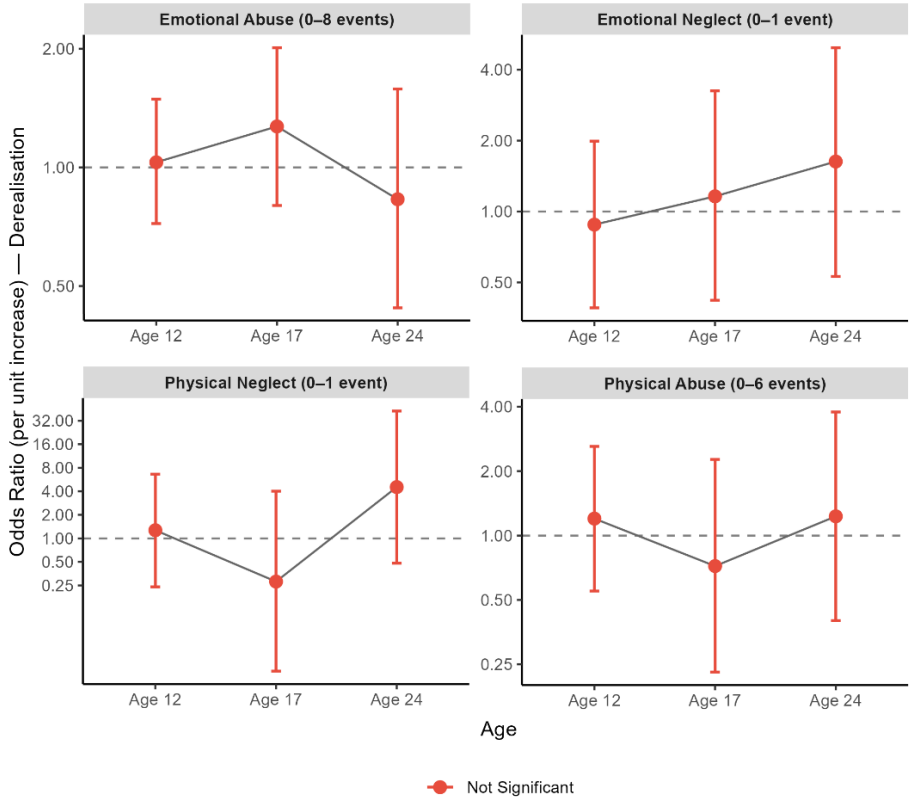


Figure 5: Longitudinal odds ratios of derealisation for emotional abuse, physical abuse, emotional neglect, and physical neglect at ages 12, 17 and 24



2.5.2 Do household instability ACEs predict the odds of experiencing depersonalisation and derealisation?

For DP, PSA (ranging from 0–8 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 5; figure 6). At age 17, PSA did not significantly predict increased odds of DP in relation to baseline levels (see table 5; figure 6). However, at age 24, PSA significantly predicted increased odds of DP compared with baseline (60% increased odds per year of exposure; see table 5; figure 6). In addition, when holding other variables constant, anxiety significantly predicted the odds of DP at each time point (OR = 5.15, 95% CI: 2.49–10.65, p < .001; 415% increased odds), as did depression (OR = 2.72, 95% CI: 1.17–6.30, p = .019; 172% increased odds). PC were not a significant predictor (OR = 0.62, 95% CI: 0.25–1.59, p = .321). Neither sex (OR = 1.15, 95% CI: 0.64–2.10, p = .637) nor early life attachment style (OR = 1.12, 95% CI: 0.61–2.05, p = .718) significantly predicted increased odds of experiencing DP.

For DR, PSA not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 5; figure 7). At age 17, PSA was not significantly associated with DR prevalence in relation to baseline levels (see table 5; figure 7). By age 24, however, PSA significantly predicted increased odds of experiencing DR relative to baseline (37% increased odds per year of exposure; see table 5; figure 7). In addition, when holding other variables constant, anxiety significantly predicted the odds of DR at each time point (OR = 2.97, 95% CI: 1.63–5.37, p < .001; 197% increased odds), as did depression (OR = 2.29, 95% CI: 1.19–4.44, p = .014; 129% increased odds). PC were not a significant predictor (OR = 1.12, 95% CI: 0.54–2.36, p = .757). Neither sex (OR = 1.16, 95% CI: 0.66–2.05, p = .593) nor early life attachment style (OR = 1.00, 95% CI: 0.56–1.79, p = .990) significantly predicted increased odds of DR.

For DP, IPV *(*ranging from 0–8 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 5; figure 6). However, IPV significantly predicted an increase in the odds of experiencing DP at age 17 in relation to baseline levels (238% increased odds per year of exposure; see table 5; figure 6). By contrast, IPV did not significantly predict increased odds of experiencing DP at age 24 compared with baseline (see table 5; figure 6). In addition,

when holding other variables constant, anxiety significantly predicted the odds of DP at each time point (OR = 5.21, 95% CI: 2.51–10.81, p < .001; 421% increased odds), as did depression (OR = 3.03, 95% CI: 1.31–7.03, p = .010; 203% increased odds). PC, however, were not a significant predictor (OR = 0.55, 95% CI: 0.22–1.38, p = .201). Neither sex (OR = 1.15, 95% CI: 0.63–2.10, p = .641) nor early life attachment style (OR = 1.13, 95% CI: 0.61–2.10, p = .691) significantly predicted increased odds of experiencing DP.

For DR, IPV did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 5; figure 7). Additionally, IPV did not significantly predict changing odds of DR at age 17 (see table 5; figure 7) or age 24 compared with baseline (see table 5; figure 7) in relation to baseline. In addition, when holding other variables constant, anxiety significantly predicted the odds of DR at each time point (OR = 2.95, 95% CI: 1.63–5.31, p < .001; 195% increased odds), as did depression (OR = 2.46, 95% CI: 1.27–4.81, p = .008; 146% increased odds). PC did not significantly predict DR (OR = 1.09, 95% CI: 0.52–2.29, p = .813). Neither sex (OR = 1.16, 95% CI: 0.66–2.03, p = .613) nor early life attachment style (OR = 1.02, 95% CI: 0.57–1.80, p = .959) significantly predicted increased odds of experiencing DR.

For DP, PMI (ranging from 0-8 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 5; figure 6). Additionally, PMI did not predict DP at age 17 or at age 24 compared with baseline DP (see table 5; figure 6). In addition, when holding other variables constant, anxiety significantly predicted increased odds of experiencing DP at each time point (OR = 1.73, 95% CI: 1.05 – 2.86, p = .031; 73% increased odds). However, PC (OR = 0.40, 95% CI: 0.13 – 1.25, p = 113) and depression (OR = 2.01, 95% CI: 0.76 – 5.37, p = .162) were not significant predictors. Additionally, neither sex (OR = 0.95, 95% CI: 0.46 – 1.97, p = .890) nor early life attachment style (OR = 1.11, 95% CI: 0.53 – 2.32, p = .788) significantly predicted increased odds of experiencing DP.

For DR, PMI did not significantly predict an increase in the odds of experiencing DR prevalence at age 12, the baseline time point (see table 5; figure 7). Additionally, at age 17, PMI did not predict DR in relation to baseline (see table 5; figure 7), but PMI

did predict DR at age 24 (47% decrease in odds per year of exposure; see table 5; figure 7) relative to baseline. In addition, when holding other variables constant, anxiety significantly predicted DR (OR = 1.92, 95% CI: 1.36 – 2.67, p = <.001, 92% increase in odds). However, PC (OR = 1.14, 95% CI 0.48 – 2.69, p = .772) and depression (OR = 2.14, 95% CI: 1.00 – 4.57, p = .051) did not predict DR. Neither sex (OR = 0.86, 95% CI: 0.44 – 1.72, p = .679) nor early life attachment style (OR = 0.98, 95% CI: 0.49 – 1.95, p = .956) significantly predicted increased odds of experiencing DR.

For DP*,* parental divorce, ranging from 0-5 events, did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 5; figure 6). However, parental divorce did predict DP at age 17 (180% increased odds per year of exposure; see table 5; figure 6) and at age 24 (249% increased odds per year of exposure; see table 5; figure 6) in comparison to baseline. In addition, when holding other variables constant, anxiety (OR = 1.94, 95% CI: 1.28 – 2.92, p = .002; 94% increased odds) and depression (OR = 3.25, 95% CI: 1.41 – 7.54, p = .006; 225% increased odds) significantly predicted increased odds of DP, but PC did not (OR = 0.58, 95% CI: 0.22 – 1.54, p = 275). Additionally, neither sex (OR = 1.11, 95% CI: 0.61 – 1.99, p = .744) nor early life attachment style (OR = 1.12, 95% CI: 0.61 – 2.05, p = 723) significantly predicted increased odds of experiencing DP.

For DR, parental divorce did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 5; figure 7). Additionally, parental divorce did predict DP at age 17 (71% decreased odds per year of exposure; see table 5; figure 7) in comparison to baseline, but did not at age 24 (see table 5; figure 7). Additionally, when holding each other variable constant, anxiety (OR = 1.90, 95% CI: 1.39 – 2.59, p = <.001; 90% increased odds) and depression (OR = 1.20, 95% CI: 1.13 – 4.35, p = .020; 20% increased odds) significantly predicted DR, but PC did not (OR = 0.97, 95% CI: 0.45 – 2.08, p = 935). Neither sex (OR = 1.11, 95% CI: 0.62 – 1.95, p = 744) nor early life attachment (OR = 0.98, 95% CI: 0.55 – 1.77, p = 957) significantly predicted DR.

For DP, parental convictions (ranging from 0-3 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 5;

figure 6). Additionally, parental conviction did not predict changing odds of DP at age 17 or at age 24 (see table 5; figure 6) in comparison to baseline. Additionally, when holding other variables constant, anxiety (OR = 1.95, 95% CI: 1.30 – 2.95, p = .001; 95% increased odds) and depression (OR = 3.46, 95% CI: 1.48 – 8.09, p = .004) predicted DP, but PC did not (OR = 0.60, 95% CI: 0.23 – 1.57, p = .294). Additionally, neither sex (OR = 1.12, 95% CI: 0.61 – 2.01, p = .723) nor early life attachment style (OR = 1.12, 95% CI: 0.61 – 2.05, p = .718) significantly predicted increased odds of experiencing DP.

For DR, parental convictions did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 5; figure 7). Additionally, parental conviction did not predict changing odds of DP at age 17 or at age 24 (see table 5; figure 7) in comparison to baseline. Additionally, when holding other variables constant, anxiety (OR = 1.92, 95% CI: 1.41 – 2.59, p = <.001; 92% increased odds) and depression (OR = 2.25, 95% CI: 1.15 – 4.39, p = .018; 125% increased odds) predicted DR, but PC did not (OR = 0.90, 95% CI: 0.42 – 1.92, p = .779). Additionally, neither sex (OR = 1.11, 95% CI: 0.62 – 1.97, p = .734) nor early life attachment style (OR = 0.99, 95% CI: 0.55 – 1.77, p = .962) significantly predicted increased odds of experiencing DR.

Table 5: Fully adjusted, longitudinal associations between household instability ACEs and depersonalisation and derealisation trajectories

		Depersonalisation			**Derealisation**		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
PSA	12	1.06	0.87 – 1.30	.582	0.99	0.80 – 1.22	.941
	17	0.87	0.64 – 1.29	.385	1.20	0.94 – 1.52	.149
	24	1.60	1.16 – 2.20	**.004****	1.37	1.03 – 1.82	**.029***
IPV	12	0.81	0.48–1.38	. 444	0.85	0.51–1.43	.544
	17	3.38	1.79–6.42	**<.001*****	1.65	0.91–3.00	.096
	24	1.67	0.71–3.94	.241	0.87	0.40–1.86	.714
PMI	12	0.95	0.71 – 1.27	.719	1.09	0.94 – 1.45	.502
	17	1.42	0.95 – 2.14	.090	0.91	0.64 – 1.30	.581
	24	1.43	0.91 – 2.27	.117	0.53	0.30 – 0.94	**.032***
Parental Divorce	12	0.85	0.45 – 1.63	.620	1.01	0.52 – 1.94	.979
	17	2.80	1.12 - 7.03	**.028***	0.29	0.10 – 0.84	**.023***
	24	3.49	1.19 – 10.18	**.023***	0.40	0.11 – 1.39	.149
Parental Conviction	12	0.78	0.24 – 2.56	.686	0.88	0.28 – 2.75	.820
	17	0.18	0.02 – 1.95	.160	0.39	0.07 – 2.18	.283
	24	0.42	0.03 – 6.49	.538	3.60	0.84 – 15.49	.084

*Abbreviations: Parental Substance Abuse (PSA), Inter-parental Violence (IPV), Parental Mental Illness (PMI); *** < .001, ** < .01, * <. 05*

Figure 6: Longitudinal odds ratios of depersonalisation for parental substance abuse, inter-parental violence, parental mental illness, parental divorce and parental convictions at ages 12, 17 and 24

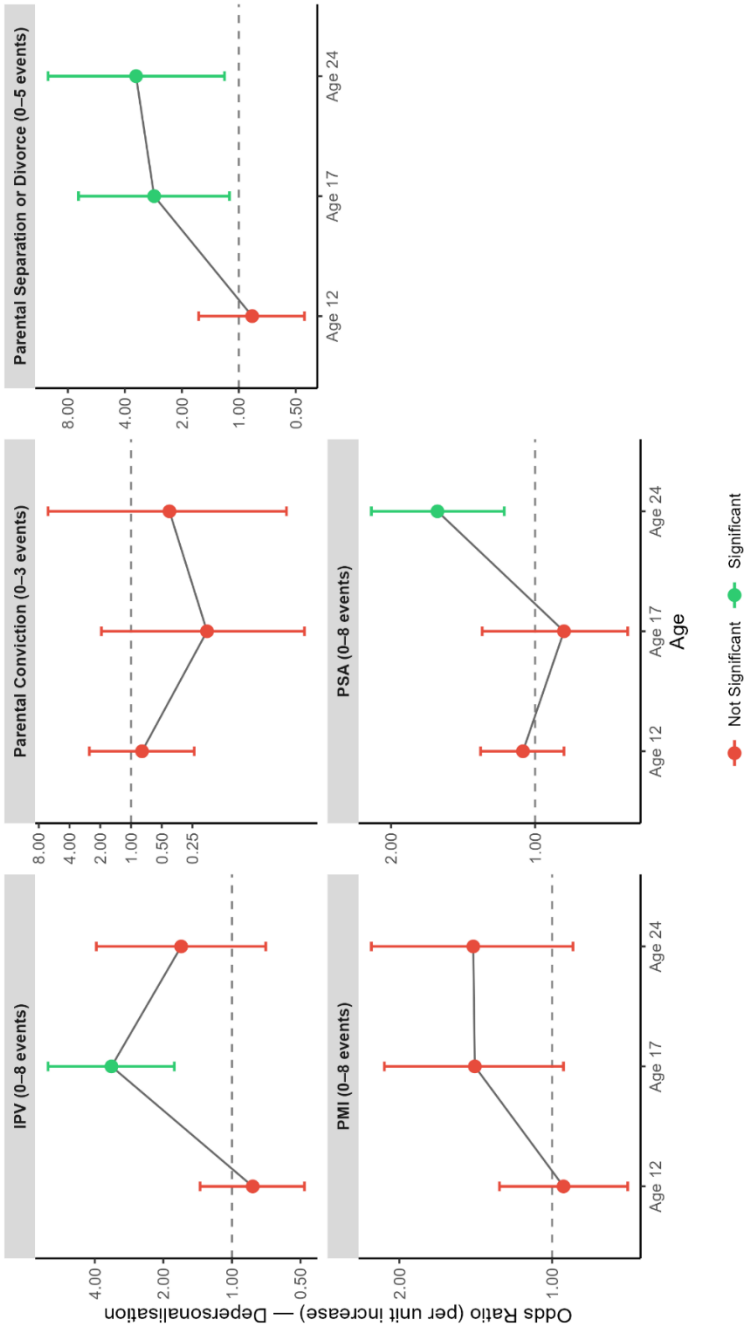
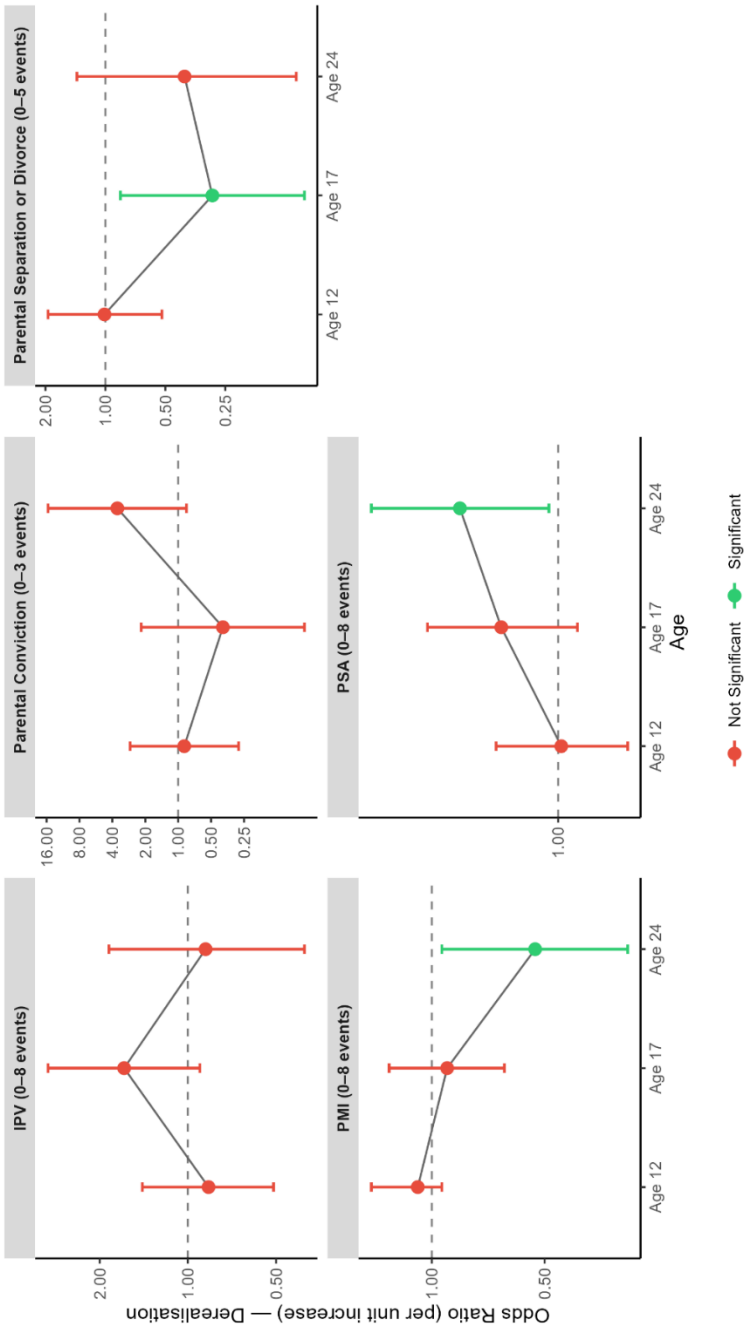


Figure 7: Longitudinal odds ratios of derealisation for parental substance abuse, inter-parental violence, parental mental illness, parental divorce and parental convictions at ages 12, 17 and 24



2.4.3 Do the accumulation of ACEs predict the odds of experiencing depersonalisation and derealisation?

For DP, abuse-related ACEs (ranging from 0-12 events) did not significantly predict the odds of experiencing DP prevalence at age 12, the baseline time point (see table 6; figure 8). However, Abuse-only ACEs did predict increased odds of experiencing

DP at age 17 (110% increased odds per unit increase in abuse-related ACE exposure; see table 6; figure 8) and age 24 (67% increased odds per unit increase in abuse-related exposure; see table 6; figure 8) in relation to baseline. Additionally, when holding other variables constant, anxiety predicted increased odds of DP (OR = 1.73, 95% CI: 1.06 – 2.86, p = .029; 73% increased odds), however depression (OR = 2.05; 95% CI: 0.76 – 5.59, p = 152) and PC (OR = 0.42, 95% CI: 0.13 – 1.34, p = .143) did not. Additionally, neither sex (OR = 0.95, 95% CI: 0.46 – 1.99, p = .900) nor early life attachment style (OR = 1.08, 95% CI: 0.52 – 2.29, p = .824) significantly predicted increased odds of DP.

For DR, abuse-related ACEs did not significantly predict the odds of experiencing DR prevalence at age 12, the baseline time point (see table 6; figure 9). Additionally, abuse-related ACEs did not predict increased odds of experiencing DR at age or 24 in relation to baseline (see table 6; figure 9). Additionally, when holding other variables constant, anxiety predicted increased odds of DR (OR = 1.94, 95% CI: 1.39 – 2.69, p <.001; 94% increased odds), however depression (OR = 1.97, 95% CI: 0.92 – 4.22, p = .078) and PC (OR = 1.17, 95% CI: 0.50 – 2.75, p = .721) did not. Additionally, neither sex (OR = 0.86, 95% CI: 0.44 – 1.72, p = 675) nor early life attachment style (OR = 0.99, 95% CI: 0.50 – 1.97, p = .976) significantly predicted increased odds of DR.

For DP, neglect and household instability-related ACEs, ranging from 0-18 events, did not significantly predict the odds of experiencing DP at age 12, the baseline time point (see table 6; figure 8). Additionally, Neglect-related ACEs did not significantly predict increased odds of DP at age 17 (see table 6; figure 8) in relation to baseline, but did significantly predict increased odds at age 24 (34% increased odds per unit increase of neglect and household instability-related ACEs; see table 6; figure 8). In addition, when holding all other variables constant, anxiety (OR = 4.76, 95% CI: 2.25 – 10.18, p < .001; 376% increased odds) and depression (OR = 3.74, 95% CI: 1.57 – 9.03, p = .003; 274% increased odds) significantly predicted increased odds of DP, but PC (OR = 0.57, 95% CI: 0.21 – 1.52, p = .266) did not. Additionally, neither sex (OR = 1.15, 95% CI: 0.63 – 2.14, p = .644) nor early life attachment style (OR = 1.14, 95% CI: 0.61 – 2.14, p = .677) significantly predicted increased odds of DP.

For DR, neglect and household instability-related ACEs did not significantly predict the odds of experiencing DR at age 12, the baseline time point (see table 6; figure 9). Additionally, neglect and household instability-related ACEs did not significantly predict increased odds of DR at age 17 or age 24 in relation to baseline (see table 6; figure 9). In addition, when holding all other variables constant, anxiety (OR = 2.83, 95% CI: 1.54 – 5.16, p < .001) and depression (OR = 2.67, 95% CI: 1.35 – 5.26, p = .005) significantly predicted increased odds of DP, but PC (OR = 1.16, 95% CI: 0.53 – 2.56, p = .706) did not. Additionally, neither sex (OR = 1.17, 95% CI: 0.66 – 2.10, p = .579) nor early life attachment style (OR = 1.01, 95% CI: 0.56 – 1.82, p = 964) significantly predicted increased odds of DR.

Table 6: Fully adjusted, longitudinal associations between cumulative abuse-related ACEs and neglect and household instability-related ACEs on depersonalisation and derealisation trajectories

		Depersonalisation			**Derealisation**		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
Abuse ACEs	12	0.82	0.55 – 1.21	.314	1.06	0.77 – 1.45	.725
	17	2.10	1.35 – 3.29	**.001****	1.01	0.66 – 1.55	.951
	24	1.67	1.04 – 2.69	**.035***	0.94	0.58 – 1.55	.823
Neglect & Household Instability ACEs	12	1.01	0.89 – 1.14	.916	1.03	0.91 – 1.16	.677
	17	1.21	0.99 – 1.46	.065	1.05	0.90 – 1.23	.527
	24	1.34	1.06 – 1.68	**.012***	0.99	0.80 – 1.22	.944

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

Figure 8: Longitudinal odds ratios of depersonalisation for abuse-related ACEs and neglect and household instability-related ACEs at ages 12, 17 and 24

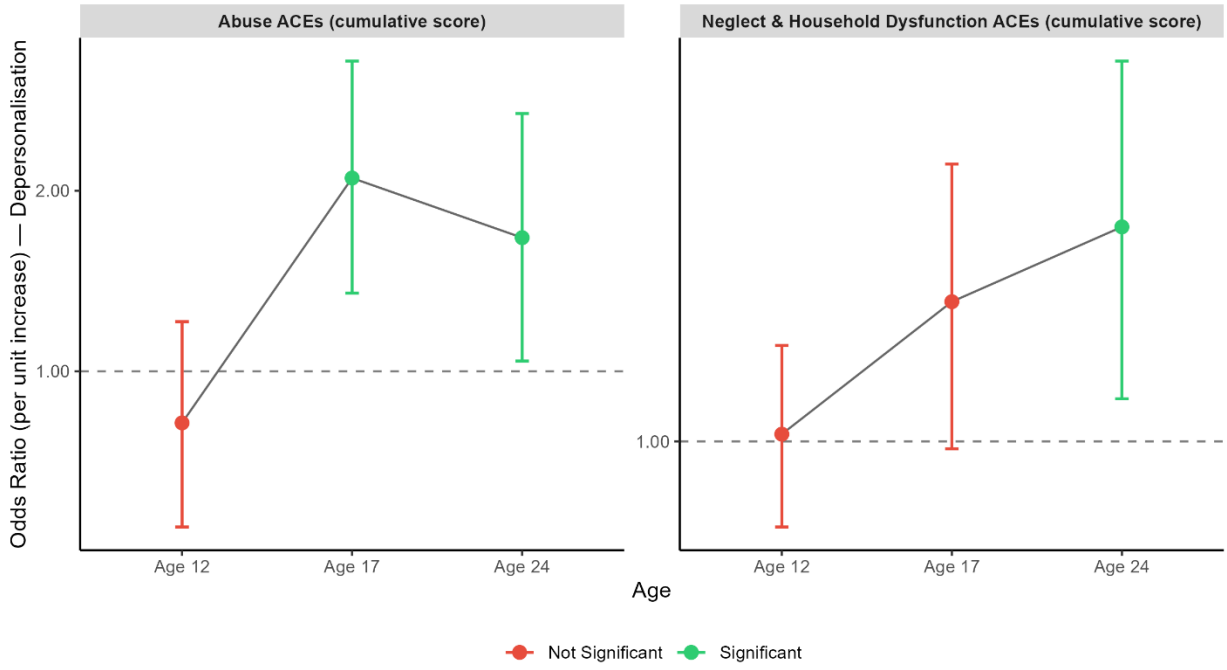
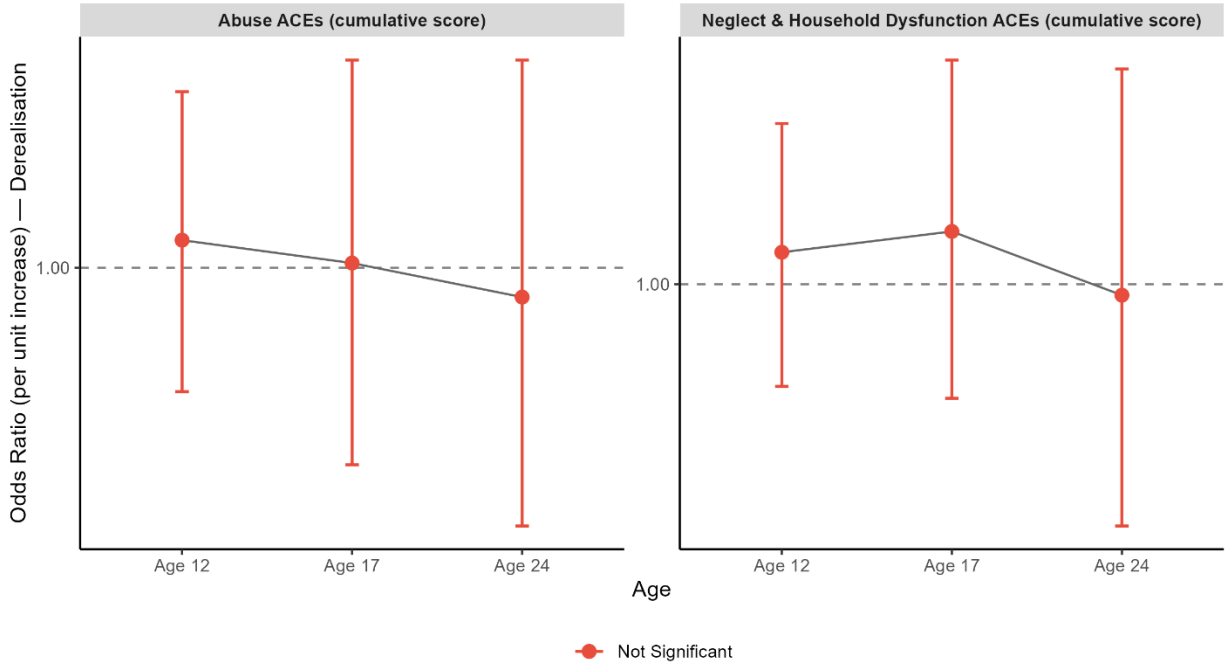


Figure 9: Longitudinal odds ratios of derealisation for abuse-related ACEs and neglect and household instability-related ACEs at ages 12, 17 and 24



2.6 Discussion

2.6.1 Results summary

To the authors' knowledge, this is the first study to use a large, prospective, population-based cohort, and longitudinal analysis to examine DP and DR as separate constructs, and to identify the differing associations of single and cumulative ACEs and DPDR. Additionally, secondary factors implicated in DPDR were controlled for.

This study used a large, longitudinal cohort to build on previous clinical observations linking EA and EN to DPDR, demonstrating that these associations are specific to DP, and not DR. Additionally, IPV predicted DP at age 17, but not at age 24, and parental divorce predicted DP at ages 17 and 24. In contrast, parental divorce and PMI were found to interact with DR in complex, and at times unanticipated ways, highlighting the potential influence of unmeasured or nuanced contextual factors. Of all ACEs measured, only experiencing PSA predicted increased odds of experiencing both DP and DR, suggesting that PSA acts as a distinct risk factor for both DP and DR. PA, PN and parental convictions were not significantly associated with increased or decreased odds of DP or DR at any age.

This research further revealed that, within larger cumulative ACE-type scores, neglect and household instability-related ACEs are significantly associated with increased odds of DP symptoms at age 24, while abuse-related ACEs are linked to increased odds of DP symptoms at ages 17 and 24, relative to age 12. Neither cumulative ACE-type predict DR at any age.

Additionally, the findings demonstrated that anxiety was a significant, independent predictor of both DP and DR in every model examined, PC were not significant in any model and depression displayed selective, context-dependent effects, emerging as an independent predictor in a subset of models. Formal mediation analyses were not conducted; however, ACE effects observed in minimally adjusted models generally persisted after adjustment for these secondary factors, arguing against complete mediation analysis by anxiety, depression, or PC (see Appendix 2). Together, these results indicated that anxiety is the most robust correlate of DP and DR, depression

exerts variable effects depending on context, and PC does not account for additional variance.

2.6.2 Emotional abuse, emotional neglect and depersonalisation

The present study demonstrated that EA and EN significantly predicted increased odds of experiencing DP at both age 17 and age 24, in relation to baseline DP scores. While the association between EA/EN and DP is well-established in existing literature (Ó Laoide et al., 2018; Simeon et al., 2001; Thomson & Jaque, 2018; Ural et al., 2015), this research represents the first and largest study to longitudinally model their relationship to DP prevalence across key developmental stages within a non-clinical sample, significantly enhancing the generalisability of these findings.

Examining the specific trajectories for EA, the highest odds of experiencing DP were observed at age 17, followed by a slight decline at age 24. This decline suggests that some individuals may experience a resolution of DP by age 24. Conversely, for EN, the odds of experiencing DP at age 24 were notably higher than at age 17, with both points still reflecting a significantly increased odds compared to baseline.

Taken together, these findings suggest that exposure to EA and/or EN may lead to sustained, increased odds of experiencing DP, indicating potential chronicity, and reinforce previous demonstrations that EA and EN have a particularly intimate relationship with DP (see section 1.6.4 Psychological sequelae of traditional adverse childhood experiences for a detailed review).

These longitudinal patterns may reflect the complex interplay of social, relational, and biological changes occurring after EA and EN. The escalating odds of experiencing DP between ages 17 and 24 when predicted by EN could reflect the influence of romantic relationships in early adulthood. Research demonstrates that childhood EN profoundly impacts social dysfunction in adulthood, such as insecure attachment representations (Müller et al., 2019). Crucially, within this pathway, oxytocin may mediate the relationship between EN and insecure attachment (Müller et al., 2019) suggesting early neglect may disrupt fundamental biological systems involved in social bonding via the oxytocin system. Furthermore, the GG-allele of the oxytocin

receptor gene rs53576, which is associated with less efficient oxytocin signalling, acts as a significant combined risk factor for DP symptoms when in conjunction with an insecure (unresolved) attachment status (Reiner et al., 2016). This may, hypothetically, suggest a complex interplay where EN during childhood might affect oxytocin-mediated relational capacities, which, coupled with specific genetic vulnerabilities in the oxytocin system, could collectively contribute to increased odds of DP. While simply speculative, this may be of interest in future DPDR research, which suffers from limited biological investigation.

Beyond relational dynamics, the transition from adolescence to early adulthood (such as ages 17 to 24) is marked by increasing environmental stressors that may interact with vulnerabilities established by EN. This period commonly involves navigating newfound independence, entering higher education (Ribeiro et al., 2018) or the workforce (Sawang & Newton, 2018), and confronting associated work-related or financial strains (for example, student loan debts; Zhan, 2022). All these factors are well-established triggers for increased stress, and in some cases, are directly linked to DP (Schweden et al., 2018).

In contrast to EN, the association between EA and DP should be interpreted as increased odds per unit increase (EA range is 0-8 events, whereas EN is simple binary). Despite a slight decline in these per-unit odds from age 17 to 24, the findings suggest sustained, increased odds of DP at both 17 and 24 when compared to baseline scores. This indicates potential for DP chronicity when predicted by EA. The association between EA and DP is established, however this research's novelty lies in the method of association delineation: a GLMM allowed us to understand the enduring relationship over time, modelling independent level trajectories.

This chronicity could reflect biological embedding, whereby childhood maltreatment can lead to lasting biological variations, and can result in psychopathology (Berens, Jensen & Nelson III, 2017). A key consequence of biological embedding is stress system dysfunction, characterised by alterations in hormonal stress regulation (like cortisol levels), changes in DNA methylation patterns influencing gene expression, and dysregulation within the stress-immune network, significantly increasing vulnerability (Kumsta, 2023). Evidence suggests that EA can lead to biological

embedding-related stress system dysfunction, for example DNA methylation age acceleration (Tang et al., 2020) and DNA methylation-mediated embedding within the HPA-axis (Hecker et al., 2016). DP is consistently associated with EA (as discussed) and stress system dysfunction (review see Millman et al., 2024; Giesbrecht et al, 2007; Simeon, Knutelska, & Nelson, 2003; Simeon et al., 2001; 2007; Stanton et al., 2001), and thus DP chronicity may reflect a level of biological embedding. Early evidence suggests a relationship between inflammatory markers, a robust assessment of how adversity 'gets under the skin', and DPDR (Zheng et al., 2024).

This section summarises the distinct longitudinal associations between EN and EA and DP risk across adolescence and early adulthood. EN showed an increasing association with DP into early adulthood, suggesting continued exacerbation by stressors, while EA was linked to a persistently high odds of DP, suggesting deeper biological embedding. These findings emphasise a need to build on theoretical links between chronic stress, biological embedding, which is addressed in Chapter 3.

2.6.3 Parental substance abuse, depersonalisation and derealisation

Our findings demonstrate that PSA was the only ACE to predict increasing odds of both DP and DR at age 24. The association between PSA and DPDR is scarcely explored in the literature, and to the authors' knowledge, this study represents the first to longitudinally investigate this relationship. While PSA is a well-established risk factor for a variety of other mental health outcomes (see McGovern et al., 2023 for review), the present findings provide novel insights into how early-life exposure to PSA is associated with DP and DR long-term, suggesting a shared aetiology and a similar degree of influence on these distinct dissociative experiences. These findings suggests that DPDR do not manifest immediately in response to PSA but develop later in life.

The impact of PSA on development is well documented, with evidence consistently linking it to a range of difficulties. For example, Raitasalo & Holmila (2017) looked at the connection between Finnish PSA and 0–6-year-old children's somatic and psychological health, demonstrating that PSA increased a child's risk of hospitalisations for psychiatric disorders, and this risk was higher if both parents were

substance abusers. Raitasalo & Holmila suggest this may be linked to unsafe environments associated with substance abuse; for example, enduring stress associated with PSA and non-adequate responding to children's needs. This notion is supported by research identifying that rates of PA and SA were significantly higher in an environment of PSA, with a twofold increased risk (Walsh, MacMillan & Jamieson, 2003).

Research shows that PSA can impede parenting and the provision of a nurturing environment (Barnard and McKeganey, 2004). Children of parents with substance abuse problems are exposed to a range of associated sequelae, and can be acutely aware of PSA, while also experiencing anxiety, insecurity and emotional turmoil, and some children try to protect their parents and minimise the impact (Fraser, McIntyre and Manby, 2009). Overall, PSA can create an enduring, highly stressful environment for a child, and despite young ages, a child is aware of the impact that a parent is having on them. This cognisance likely contributes to the traumatic nature of PSA.

PSA can result in 'parentification,' a developmentally inappropriate process in which children take on caregiving responsibilities for parents or siblings (Tedgård et al., 2019). This may extend to a 'reversal of dependence needs,' where the child is effectively required to parent the parent (Lander, Howsare & Byrne, 2013). Parentified children employ dissociative strategies, highlighting themes like the creation of a 'false self' and profound emotional disengagement to manage overwhelming early experiences (Schorr & Goldner, 2023). For example: 'Actually, my experience was like I was leaving myself outside, serving others, not thinking my thoughts, not feeling my feelings' (Schorr & Goldner, 2023, p. 12).

This process of detachment aligns with the TM of dissociation, whereby 'dissociation is a phylogenetically important aspect of the psychobiological response to threat and danger that allows for automatisation of behaviour, analgesia, depersonalisation, and isolation of catastrophic experiences to enhance survival during and in the aftermath of these events' (Dalenberg et al., 2012).

The delayed emergence of DP and DR at age 24, rather than at 17, could reflect a ‘re-triggering’ of early-life dissociative processes in early adulthood, like patterns observed with EN. PSA and neglect are closely linked (Murphy et al., 1991; Kelleher et al., 1994; Kepple, 2018), and the unpredictable, emotionally dysregulating environment of childhood PSA may create a latent vulnerability to DPDR. However, these symptoms may remain suppressed or less overt during adolescence. As individuals transition into early adulthood, the demands of independence, intimate relationships, and entry into higher education or the workforce may reactivate this vulnerability, leading to the later onset of DPDR.

A significant pathway contributing to the delayed onset of DPDR may involve the intergenerational transmission of substance use, a widely documented phenomenon that often manifests in emerging adulthood (Sheridan, 1995; Myhra & Wieling, 2014; Henry et al., 2018; Gomis-Pomares et al., 2023) between ages 18 to 24 (Knight, Menard & Simmons, 2014). Therefore, this period is a high-risk window for recreational drug use, a known trigger for DPDR (Medford et al., 2003; Simeon et al., 2004, 2009; Madden & Einhorn, 2018). For some individuals, early vulnerability from childhood PSA may be reactivated and intensified by substance use in early adulthood, creating a pharmacological pathway for the onset of DPDR.

Drug-induced symptoms could reflect the concordance of both DP and DR in relation to PSA. Drugs known to induce DPDR, such as hallucinogens and cannabis, exert acute neurobiological effects that globally disrupt perception, self-awareness, and consciousness (Millière, 2017; Pujol et al., 2014; Zaytseva et al., 2019). When interacting with childhood trauma such as experiencing PSA, these direct pharmacological actions could induce a more pervasive and less specific sense of unreality, affecting both the internal experience of self (DP) and the external perception of reality (DR).

In summary, these findings indicate that PSA represents a distinct and powerful risk factor for the later emergence of both DP and DR. The delayed onset of symptoms until early adulthood suggests that PSA establishes an enduring vulnerability to DPDR, which may remain latent through adolescence before being reactivated by new developmental challenges or substance use in early adulthood. Importantly, the

convergence of DP and DR outcomes highlights that PSA may operate through broad dissociative processes rather than pathways specific to one symptom domain, underscoring its significance in shaping long-term DPDR risk.

2.6.4 Inter-parental violence and depersonalisation

The present study identifies that, in relation to baseline DP scores, IPV predicts increased odds of experiencing DP at age 17. Conversely, these increased odds were no longer significant by age 24. This longitudinal pattern suggests that the association between IPV and DP appears to peak in mid-adolescence and diminish in early adulthood. Such a trajectory may indicate a potential for resolution of IPV-related DP, suggesting more favourable outcomes and reduced chronicity over time.

Prior evidence supports the significance of witnessing IPV on development and mental health (for review, see Pringley, 2017). A review by Howell et al. (2016) highlighted that exposure to IPV across varying age-spans (0-2, 3-6, 6-12 years) lead to age-specific and common disruptions across multiple developmental domains, including psychological functioning, where internalising symptoms were a common theme. This study extends these findings of internalising symptoms in the context of IPV by demonstrating that witnessing childhood IPV between 0-12 years increased the odds of experiencing DP, however it is acknowledged that this design does not allow for stratification of specific age exposures.

Prior research consistently links childhood IPV exposure to heightened PTSD symptomatology, a condition often accompanied by dissociative symptoms such as DP. Studies in young children have shown increased PTSD diagnoses and related symptoms, including hyperarousal and new fears, following exposure to violence against a caregiver, pointing to stress system dysfunction (Scheeringa & Zeanah, 1995). These effects appear dose-dependent (Bogat et al., 2006) and are amplified when IPV co-occurs with other traumatic events, with exposed children more likely to develop PTSD and internalising symptoms (Graham-Bermann et al., 2012). Evidence also connects IPV to dissociation more broadly (Draijer & Langeland, 1999; Teicher et al., 2006; Rada, 2002). Previous research has noted a prevalence of witnessing domestic violence in childhood in patients with DDD (27%), but this was not

significantly higher than HC (Simeon et al., 1997). However, due to a small and clinical only sample size of 30, this is unlikely to reflect the population at large. To the authors' knowledge, this study is the first time IPV has been modelled as a predictive function of later DP.

The heightened dissociative symptoms in relation to IPV may stem from its overt and threatening nature. Exposure to IPV is a well-established risk factor for PTSD symptoms in children, with chronic exposure creating a pervasive sense of fear, emotional dysregulation, and hypervigilance (Margolin & Vickerman, 2007). Unlike isolated traumatic events, IPV is chronic and often unpredictable, preventing the child from achieving a sense of safety. Margolin & Vickerman (2007) also highlight common disruptions in attachment security, which can result in disorganised attachment patterns (which was not directly measured in the current study), as caregivers in IPV contexts can be both sources of safety and threat. This instability may increase vulnerability to dissociation, as children learn to psychologically detach (as per the TM) from their distressing reality when caregivers fail to provide emotional regulation.

The observed non-significance of IPV predicting DP at age 24, subsequent to its peak at age 17, suggests that DP may resolve later in life. This potential resolution could be attributed to a change in environmental context, as individuals at age 24 are likely no longer living in the parental household (for example, in 2021, 50% of children had moved out of their family home by age 24; Pereira, 2024), thereby removing them from the immediate source of the stressor.

Furthermore, the transition to early adulthood may facilitate the development of more secure attachments in new relationships (Arriaga & Kumashiro, 2019; Keren & Mayseless, 2013; Umemura et al., 2015). This process could potentially mediate the reduction of DP by addressing underlying disorganised attachment patterns. Crucially, the attachment style controlled for in the current study was derived from behaviours observed at ages 1 and 2. Therefore, the body of literature concerning the relationship between adult attachment style and DPDR must be considered a separate construct from the early childhood measure utilised here.

In summary, childhood IPV predicted elevated DP in mid-adolescence, with effects peaking at 17 and diminishing by early adulthood. This pattern suggests that IPV-related DP may be time-limited, with resolution potentially supported by removal from the parental home and the formation of more secure adult relationships. IPV did not predict DR at any age.

2.6.5 Parental mental illness and unexpected derealisation trajectories

The present study demonstrated that PMI predicted decreased odds of DR at age 24 in comparison to age 12. This is an unusual finding, as it is expected that PMI could lead to an increase of symptoms in line with previous evidence (Rutter & Quinton, 1984; Khoury, Kaur & Gonzalez, 2021; Chu & DePrince, 2008; Draijer & Langeland, 1999). For example, individuals experiencing PMI may inherit challenges in self-regulation (Bridgett et al., 2015) or transmission of mental health problems (Gomis-Pomares, Villanueva & Prado-Gascó, 2023). Furthermore, given that DR is a transdiagnostic phenomenon, there could even be a potential for direct intergenerational transmission of DR through inherited DDD, or transdiagnostically experiencing symptoms through inherited anxiety, depression or psychosis (Černis et al., 2025), although this was not tested here. Overall, this unexpected finding raises important questions about specific contextual factors influencing these results.

This finding could reflect unmeasured social support and protective mechanisms. For instance, parents with milder mental health symptoms can still provide a stable home environment (Kristensen et al., 2024). Even in cases of more severe mental illness, protective strategies (such as robust coping skills developed by the child or the presence of strong external support systems) may buffer children from psychological distress (van der Ende et al., 2016). In households with multiple caregivers, the presence of another emotionally available adult may buffer children from the full impact of a parent's mental health difficulties. For example, involvement from a grandmother or other sources of social support can act as protective factors, reducing the risk of externalising behaviours even in the context of poor parenting (Barnett et al., 2011; Parkes & Sweeting, 2018). Similarly, the effect of early maternal psychological distress on child internalising and externalising behaviour was shown to be buffered by social support (Heberle et al., 2014). This reliance on additional

social support may be particularly salient in families where a parent experiences mental health issues, potentially leading to a greater presence of protective external resources for the child.

Alternatively, this inverse relationship could be an artefact of under-reporting of PMI within the ALSPAC dataset, given its reliance on self-report measures. Social desirability bias or stigma might lead parents to under-disclose symptoms, thus masking a true positive association between PMI and dissociative symptoms in offspring.

In summary, PMI did not predict DP at any age but unexpectedly predicted reduced odds of DR at age 24. This finding may reflect unmeasured protective factors, such as positive parenting by mentally ill parents, additional caregiver involvement, and wider social support, or it may be an artefact of under-reporting within the ALSPAC dataset. Together, these possibilities highlight the importance of examining both protective mechanisms and measurement limitations to clarify the true association between parental mental illness and dissociative outcomes.

2.6.6 Parental divorce, depersonalisation and unexpected derealisation trajectories

Our findings demonstrated that parental divorce predicted distinct and divergent longitudinal trajectories for both DP and DR in relation to baseline scores. Specifically, parental divorce predicted increased odds of DP at both age 17 and age 24, suggesting an increasing risk over time. In contrast, parental divorce significantly predicted decreased odds of DR at age 17, but there was no significant association at age 24.

The literature on parental divorce and dissociation is mixed. Some studies report that parental divorce predicts both clinical and non-clinical DP (Michal et al., 2009). Others, however, found no association with later DPDR (Lee et al., 2012), though this study assessed symptoms at age 36, when additional confounding factors may be present. Earlier work also reported no predictive relationship between parental separation and dissociation (Draijer & Langeland, 1999), but its relevance may be

limited given changing social norms around divorce. Overall, the evidence remains inconclusive, underscoring the value of the present study's longitudinal design with DPDR assessment at ages 12, 17, and 24.

The present findings may help explain inconsistencies in prior research on parental divorce and dissociation. It was observed that parental divorce increased odds of DP but decreased odds of DR; thus, when DP and DR are combined into a single DPDR measure, these opposing effects may counteract each other. This variance within a composite outcome could obscure the true associations, giving the impression of no effect. Therefore, by failing to separate DP and DR in their investigation, previous research may have underestimated the relationship between parental divorce on DPDR.

The observed increasing odds of DP from age 17 to 24 in the context of parental divorce warrant closer examination. While analyses controlled for attachment style, this was measured between ages 1 and 2. Therefore, for a significant proportion of the cohort, parental divorce would transpire after the initial assessment of attachment style. Given that ACE measurements were between 8 months and 12 years, this would allow ample time for these profound family changes to influence the development of later attachment styles and relational capacities through mediating processes throughout childhood and adolescence. Example of such processes are reduced coping (Zhang & Labouvie-Vief, 2004) or a history of depression or low self-esteem (Cozzarelli et al., 2003). Therefore, a plausible mechanism could be through the dissociative consequences of disorganised attachment.

The parental divorce variable also captures potential inconsistencies in parenting presence and practices, particularly where multiple divorces occurred. Such instability may involve parenting conflicts, shifting living arrangements, inconsistent rules across households, and fluctuating emotional availability as parents manage their own distress. Chronic exposure to this unpredictable and emotionally turbulent environment can profoundly affect a child's developing nervous system (Shonkoff et al., 2012).

Amato's (2000) Divorce-Stress-Adjustment Perspective provides a useful framework for understanding the divergent trajectories of DP and DR. This model conceptualises marital dissolution not as a single event but as a prolonged process beginning with pre-divorce conflict and extending well beyond legal separation. For children, this uncoupling introduces multiple stressors, such as reduced parental support and control, loss of contact with one parent, ongoing conflict, economic decline, and other disruptions. Such chronic and cumulative stressors, particularly the decline in reliable parental presence and support, closely parallel experiences linked to DP, such as EN. By contrast, the decreased odds of DR at age 17 may also be interpreted through this framework. Children in high-conflict homes are often exposed to persistent hostility and unpredictability prior to divorce. This environment may elicit DR as a coping mechanism, blunting the external world to manage ongoing threat. In these cases, the divorce itself could paradoxically provide relief by reducing direct exposure to inter-parental conflict. For some individuals, the resolution of intense pre-divorce hostility may therefore diminish a key trigger for DR.

However, it is critical to acknowledge that a reduction in DR does not equate to an absence of DP. While the external world may become less "unreal" for some after the immediate conflict subsides, the aforementioned stressors associated with the divorce process itself (e.g., parental decline, loss of contact, economic strain) persist and could continue to fuel DP. Thus, the divergent patterns highlight the nuanced ways in which specific facets of the divorce experience may affect different dimensions of dissociation.

In summary, parental divorce predicted divergent trajectories: increased risk of DP into early adulthood but reduced odds of DR in mid-adolescence. This pattern suggests that while divorce may relieve DR by reducing immediate conflict, ongoing instability and loss continue to foster vulnerability to DP. These findings emphasise the need to examine DP and DR separately to capture their distinct developmental pathways.

2.6.7 ACE-type clusters and depersonalisation

This study contributes novel findings to a growing body of research that investigates whether there is an association between cumulative risk by ACE-type clusters and dissociative symptoms. It was demonstrated that, while controlling for sex, early life attachment style, anxiety, depression, and PC, experiencing abuse-related ACEs significantly increased the risk of DP at both age 17 and age 24 in relation to baseline. However, the highest risk for abuse-related DP was observed at age 17 (OR = 2.10), with a reduction (remaining significant) by age 24 (OR = 1.67), suggesting that early adolescence may be a particularly potent time point for the manifestation of abuse's effects on DP. In contrast, neglect and household instability-related ACEs significantly predicted increased risk of DP specifically at age 24 in relation to baseline, with no increased risk observed at age 17.

Beyond these temporal differences, the magnitude of risk associated with ACE-type clusters also varied. Neglect and household instability–related ACEs ranged from 0–18 events. At age 24, each additional event was associated with a 1.34-fold increase in the odds of DP, such that individuals exposed to 18 events had approximately 194-fold higher odds compared to those with no events. Abuse-related ACEs, despite a narrower range of 0–12 events, consistently demonstrated a stronger association with DP. For individuals exposed to multiple abuse-related events, the odds of DP escalated markedly; for example, exposure to 12 abuse-related events corresponded to an estimated ~7,360-fold increase in odds at age 17 (calculated as 2.10^12). This pattern suggests that, although the neglect/household instability cluster encompasses a greater number of potential events, abuse-related ACEs confer a substantially higher relative risk, particularly in early adolescence. These findings underscore that a profound relationship between abuse and DP development may exist.

2.6.8 Secondary factors

The present study showed minimal differences between initial models (adjusted only for sex) and fully adjusted models (including early-life attachment, anxiety, depression, and PC), indicating that DPDR can be associated directly with ACEs, rather than solely as a secondary symptom of other disorders. This supports

considering DPDR as both standalone phenomena and a transdiagnostic symptom (Černis et al., 2025), occurring independently or in comorbidity with other psychopathology.

Across all models, anxiety was a consistent, independent predictor of both DP and DR: in every single-ACE model and cumulative-ACE clusters, higher anxiety was associated with higher odds of DP and DR. By contrast, depression showed selective associations, significant in a subset of models only, while PC were not a significant predictor of DP or DR in any model.

Further, our findings showed that adjustment for early-life attachment style neither attenuated the relationship between ACEs and DPDR, nor independently predicted symptoms in multivariate models. This contrasts with prior research linking disorganised attachment to DPDR (Simeon & Knutelska, 2022). A likely explanation is that disorganised attachment was not explicitly measured: attachment was classified as secure or insecure from measures observing avoidance, resistance, and comfort-seeking behaviours in the child, which does not capture disorganised attachment's full complexity. Timing may also be relevant: the attachment measure was taken in infancy (ages 1–2), whereas much existing research uses adult self-report questionnaires (Calamari & Pini, 2003; Simeon & Knutelska, 2022; Kong et al., 2018; Gušić et al., 2016). Attachment is also dynamic and may shift with age (Zhang & Labouvie-Vief, 2004). Foundational studies (e.g., Ogawa et al., 1997; Pasquini et al., 2002; Liotti, 2006) often relied on proxy or observer measures, further complicating interpretation. Future work should employ gold-standard procedures, such as the Strange Situation Procedure (Ainsworth et al., 1978) or the Attachment Q-Sort (Waters & Deane, 1985), alongside validated DP measures (e.g., CDS).

Additionally, the mediating potential of PC, such as rumination, has been observed in other mental health conditions, including anxiety, depression (Kim et al., 2017), and PTSD, where it predicts symptom development and persistence (Moulds et al., 2020). This is particularly relevant for DPDR given its comorbidity with PTSD. However, these findings suggest that PC is not a strong mediator between ACEs and DPDR, nor does it function as an independent predictor of DPDR, a notion supported

in other studies showing increased DPDR with increased rumination (Vannikov-Lugassi et al., 2021).

Overall, these findings suggest that anxiety does not mediate the relationship between ACEs and DPDR, indicating that DPDR can arise independently of anxiety. However, the fact that anxiety independently predicted DP and DR in all contexts raises the possibility of a causal role. Anxiety is therefore best conceptualised as an independent predictor of DPDR. Future longitudinal research examining bidirectional effects (e.g., DPDR predicting later anxiety) is needed to determine whether anxiety also represents a secondary consequence of DPDR. Taken together, this supports the view that DPDR can emerge independently of anxiety, while also allowing for alternative pathways in which anxiety contributes to its development and persistence. In line with Černis et al. (2025), these findings reinforce the framing of DPDR as a transdiagnostic symptom, particularly in its relationship with anxiety.

Finally, the present study found that depression did not significantly attenuate the relationship between ACEs and DPDR, reinforcing the notion that DPDR are distinct phenomena, rather than simply a symptom of depressive disorders. This aligns with research demonstrating that DPDR can persist independently of depressive episodes (Simeon, 2004; Michal et al., 2011). Additionally, the findings demonstrated that depression only emerged as an independent predictor of DPDR in certain contexts of controlling for certain ACEs. A recent meta-analysis by Černis et al. (2025) identified that the literature on DPDR and depression is inconsistent and underdeveloped. While some studies suggest that DPDR predicts or mediates depressive symptoms, others find no relationship, or conversely, that depression mediates DPDR. The lack of consensus, coupled with limited exploration of mechanisms, suggests that the relationship remains poorly understood.

2.6.9 Strengths and limitations

This study possesses several key strengths that significantly contribute to its novel findings, advancing DPDR research.

The use of the ALSPAC dataset enabled ACE variables to be derived from prospectively collected data, reducing biases typical of retrospective reports, which are prone to false negatives and measurement error (Hardt & Rutter, 2004). Parental self-reports of EA and PA, often affected by social desirability bias, were cross verified across both parents, providing a more accurate account and minimising memory lapses. Additionally, the availability of dual parent reports on other measures of ACEs allowed a concrete solution to missing data problems. These methods strengthen the validity and reliability of the findings.

This study's large, population-based sample offers far greater representativeness than the small clinical samples typically used in DPDR research, which often capture only diagnosed cases, overlooking the many undiagnosed or untreated individuals. By addressing this sampling bias, the current findings both corroborate earlier clinical observations demonstrating the EA and EN have a close relationship to DP (but not DR) and highlight where they may not generalise to the wider population, for example evidence that PA is associated with DPDR.

Third, the study's comprehensive approach involved examining a wide range of ACEs, including often-overlooked categories such as neglect and household instability, rather than focusing solely on more overt forms of abuse. This study's results suggest that these specific factors may be more significant in contributing to DPDR risk, thereby offering new and important directions for future research and informing clinical practice, particularly within psychodynamic therapy.

Despite its strengths, this study has limitations that warrant consideration.

First, the use of a secondary dataset inevitably constrained the choice of measures, creating potential operationalisation issues and underpowered phenotypes that may have increased the risk of Type II errors. This study's PC variable may have been underpowered, particularly at baseline where it relied on uneven OCD prevalence. In addition, the operationalisation of PC likely did not fully reflect disorder-specific ruminations thought to perpetuate DPDR. Future work should refine measurement of PC to better test their contribution as either comorbidity or maintenance factors, which is addressed in Chapter 5 by using a validating measure of rumination (a core

PC type). Additionally, the operationalisation of attachment style could have been improved through more granular, developmentally sensitive measures to better capture its potential mediating or moderating role.

Second, EN and PN were assessed retrospectively at age 22 and 23, respectively, introducing possible recall bias, particularly for early childhood experiences (Hardt & Rutter, 2004). While this may have reduced measurement precision, the findings are consistent with broader evidence linking EN to long-term DPDR, suggesting validity despite these challenges.

Third, although this study examined a range of abuse, neglect, and household instability ACEs, other forms of adversity such as bullying or community violence were not captured. Bullying is demonstrated to be an important measure of childhood adversity, demonstrating similar impact to ACEs in terms of child internalising and externalising problems (Trompeter et al., 2023). Additionally, community violence is an emerging measure of ACEs (Lopez-Tamayo et al., 2022). Future work incorporating these broader experiences will help to clarify whether the present study's observed associations extend to additional ACE types.

Fourth, DPDR was measured dichotomously, which limited the ability to capture symptom severity and phenomenological nuance. Such are the challenges of secondary data, and chapter 5 aimed to address this by using a validated, clinical scale measuring DDD, providing richer symptom variability and subscale measurement, including four different dimensions of DDD.

Fifth, ethnicity and social position could not be included as a covariate due to limitations in the available data. In the ALSPAC, ethnicity is categorised as just white or non-white and had a substantial missing data problem, leading to a highly oversampled dataset of white individuals. Additionally, including ethnicity would have required removing over 11% of the sample, undermining statistical power. Given limited prior evidence linking ethnicity directly to DPDR, this was judged a necessary compromise. While this study's measure of social position was more diverse than measures of ethnicity, the missing data problem persisted: if individuals with missing data were removed, 18.1% of the dataset would be removed if focussing on father

social position, or 22.7% of the sample if focussing on mother social position. Therefore, given the lack of evidence that ethnicity and social position are associated with DPDR, they were not included in analysis.

2.6.10 Clinical implications

The 2021 Census shows that 29.1% of people in England and Wales are under 25 (Gov UK, 2023), yet access to timely mental health care remains a major challenge, with 270,300 children and young people waiting for support in 2022–2023 alone (Children's Commissioner, 2024). Since 50% of mental health disorders emerge by age 14 and 75% by age 24 (Kessler et al., 2005), early intervention is essential to reduce the long-term impact of ACEs. Treatment options for DDD remain limited; a systematic review of 40 studies found no clear treatment pathway due to low-quality research and modest efficacy across interventions (Wang et al., 2024). While EMDR is commonly used for trauma, its application to dissociative symptoms often requires significant adaptations, such as extended preparation phases, structured techniques like tapping, and phased work to prevent re-traumatisation (Mind, 2023; Shapiro & Brown, 2019; Mosquera, 2021). EMDR has also shown reduced effectiveness for those with high levels of DPDR or complex trauma (Bae et al., 2016). Alternative therapies like Lifespan Integration (LI), which emphasises gentle processing and identity integration, show promise for dissociative presentations (Bahans, 2024; Thorpe, 2021), but are not yet widely available and require further evaluation.

Lengthy NHS wait times further exacerbate access issues, with reports of CAMHS delays extending up to 49 weeks (South West London & St George's NHS Trust, 2022), making timely treatment difficult for young people with trauma-related dissociation. Multimodal approaches may hold greater promise: combining pharmacotherapy (e.g., lamotrigine with SSRIs), neuromodulation (e.g., rTMS), and psychotherapy such as CBT has shown moderate success, with CBT alone helping 29% of participants no longer meet DDD criteria after 13 sessions (Wang et al., 2024). Childhood maltreatment complicates recovery and underscores the need for trauma-specific adaptations (Lippard & Nemeroff, 2020). Trauma-focused CBT has been shown to help children and adolescents with ACEs (de Arellano et al., 2014), and family-based models, such as those trialled in the U.S. (e.g., 'Parents Under

Pressure', 'Focus on Families'), have demonstrated strong improvements in parenting, family functioning, and child outcomes (Calhoun et al., 2015). In the UK, while provision remains limited, initiatives like Adfam, NHS addiction services, and programs such as 'Strengthening Families, Strengthening Communities' offer important avenues for support. Early, integrated, and family-focused responses remain critical to preventing long-term DPDR.

2.6.11 Conclusion

The present study concludes that DP and DR may have distinct ACE-related antecedents, despite past literature conflating these experiences and assuming them aetiologically equal. The results identified that alongside being phenomenologically distinct, they can occur independently of each other, with occasions where DR is more common than DP, despite beliefs that DR is solely a feature of DP. The results demonstrate the importance of separating these two dissociative states.

Additionally, it was identified that past research on ACEs remains applicable to DP to an extent, as it was identified several instances where ACEs predicted increased odds of DP over time. However, the relationship between ACEs and DR was not so straightforward. Increased levels of PMI and parental divorce reflected reductions in DR symptoms, suggesting complex relationships that may reflect unmeasured variables such as external social support or resolution of exposure to inter-parental conflict through divorce. PSA significantly predicted both DP and DR, suggesting PSA as a unique risk factor for both experiences.

Additionally, early life attachment style, anxiety, depression and PC did not mediate the relationship between ACEs and DPDR, yet anxiety was a consistent independent predictor of DP and DR, highlighting its independent role as a predictor of these experiences. Depression was less consistent, demonstrating a context specific association only, while sex, early life attachment and PC did not independently predict DP or DR in any models. The operationalisation of attachment and PC in the current study may have led to associations being underpowered, and therefore their roles remain inconclusive.

Chapter 3: Interleukin-6 predicts depersonalisation and C-reactive protein predicts derealisation: A longitudinal study using the Avon Longitudinal Study of Parents and Children dataset

Abstract

Background: Depersonalisation and derealisation are dissociative symptoms marked by detachment from self and environment. Research links them to adverse childhood experiences (ACEs) and neurobiological mechanisms, recently including inflammation. This study investigates whether inflammatory markers interleukin-6 (IL-6) and C-reactive protein (CRP) predict depersonalisation and derealisation symptoms longitudinally and explores their role between ACEs, depersonalisation and derealisation.

Methods: Data from the Avon Longitudinal Study of Parents and Children were analysed using generalised linear mixed models. IL-6 at age 9 (N = 2,560) and CRP at ages 9, 15, and 24 (N = 5,127) were tested for associations with depersonalisation and derealisation symptoms at ages 12, 17, and 24, adjusting for sex, ethnicity, and social position. Mediation by IL-6 and CRP in ACE-DPDR pathways was explored where possible.

Results: Elevated IL-6 at age 9 predicted increased odds of depersonalisation symptoms at age 24, while CRP was associated with derealisation symptoms at ages 12 and 24, showing complex temporal patterns. Among ACEs, only inter-parental violence and accumulation of neglect and household instability-ACEs emerged as potential candidates for mediation analysis but showed no mediating effect.

Conclusions: Findings support distinct inflammatory profiles for depersonalisation and derealisation and highlight inflammatory instability as a potential mechanism in derealisation symptom persistence. This study was the first longitudinal analysis of inflammation in depersonalisation and derealisation in a large population sample, emphasising the need to distinguish depersonalisation and derealisation in neurobiological research and could inform immune-targeted interventions, however further research is necessary.

3.1 Introduction

3.1.1 What are depersonalisation and derealisation?

DP and DR are dissociative symptoms characterised by profound alterations in self-perception and perception of the external world. DP involves feelings of detachment from one's thoughts, emotions, body, or actions, often accompanied by perceptual distortions, a disrupted sense of time, emotional numbing, and a diminished sense of self (American Psychiatric Association, 2013). DR, in contrast, refers to an altered perception of the external environment, where surroundings may appear unreal, dreamlike, or visually distorted (American Psychiatric Association, 2013). These symptoms can be functional and frequently manifest in response to traumatic stress but can also become persistent and therefore dysfunctional (Simeon et al., 2008). Further, peri-traumatic DPDR can predict PTSD development (Ehlers & Clark, 2000; Seidler, 2007; Ozer et al., 2008). If symptoms do not remit, individuals may be diagnosed with DDD, which is considered 'uncommon' (approximately 1-2% of the population may qualify for a diagnosis; Hunter, Sierra & David, 2004; Yang et al., 2022). However, given that up to 74% of people experience DPDR at some point in their lives (Hunter, Sierra, & David, 2004), this diagnosis rate likely underestimate the true scale of the problem.

Considered the third most common mental health symptom following anxiety and depression (Simeon et al., 2003), DPDR are a commonly experienced transiently and are considered a normal part of human experience (Hunter, Sierra, and David, 2004). DPDR are related to trauma, such as ACEs, which may act as predisposing and precipitating factors (see 1.4.4 Adverse childhood experiences). Additionally, several biomarkers are associated with DPDR, such as markers of autonomic dysfunction and HPA-axis dysfunction (see sections 1.9.5 and 1.8.2 for a detailed review).

3.1.2 Neurobiological underpinnings of depersonalisation and derealisation

DPDR have been associated with several areas of brain dysfunction, for example, suppression of the limbic system in response to emotional stimuli (Sierra, 2008). This response supports DPDR's role as a protective function, allowing individuals to maintain adaptive behaviour in situations that would typically induce anxiety by dampening emotional responses. However, in individuals who experience DPDR, this

function would be triggered extremely frequently, therefore suggesting long-term adaptations that likely extends to several other areas, such as stress response. Therefore, what was first a temporary adaptive function becomes a permanent maladaptive function.

Stress system dysfunction in individuals with DPDR is empirically supported by findings suggesting dysregulation within the ANS, HPA-axis and immunological functions. ANS dysfunction features in dissociative-and-related mental health conditions (Austin et al., 2007; Van der Kolk et al, 1996, Farina et al., 2015; Reinders et al., 2006; Reinders et al., 2012), measured through several methods, but most notably an elevated skin conductance response (Giesbrecht et al., 2008; Giesbrecht et al., 2010) and heartbeat-evoked potentials (Schulz et al., 2015). Additionally, HPA-axis dysfunction is observed in individuals with DPDR, measured through the cortisol response but results vary and are often contradictory (Giesbrecht et al., 2007; Simeon et al., 2001, 2007; Stanton et al., 2001). In addition, this research outdated, and there has been a recent shift in focus to the role of inflammatory markers, such as IL-6 and CRP, as a biomarker of prolonged stress-system activation (Zheng et al., 2024; Powers et al., 2019; Bizik et al., 2011; Jarkas et al., 2021; Bob et al., 2010).

As yet, there is only one published paper on the role of inflammatory markers in DPDR. A recent study by Zheng et al (2024) investigated the potential relationship between inflammatory markers and individuals with DDD. They identified, when comparing results for 30 DDD patients to 32 HC, that levels of CRP were significantly downregulated in DDD patients. The fact that there is only one published study on the association between inflammatory markers and DPDR is evidence that this area of science is novel.

The relationship between inflammation and general dissociation has a wider reach and demonstrates insight for DPDR by linking inflammatory markers to dissociative outcomes (Powers et al., 2019, Bizik et al., 2011, Jarkas et al., 2021; Bob et al., 2010). For example, evidence suggests that, even when controlling for childhood trauma, PTSD, depression and emotion dysregulation, CRP significantly predicts dissociative symptom severity (Powers et al., 2019). Further, dissociation may be a ‘sickness behaviour’, as individuals with inflammation-related conditions are

significantly more likely to exhibit dissociative symptoms compared to HC (Ferrarese et al., 2021).

IL-6 and CRP are also biological markers that can also offer insight into the biological embedding of early adversity (Berens et al., 2017). For example, lack of home ownership or low parental education, low social position and self-reported childhood trauma, are significantly associated, and can predict, increased concentrations of IL-6 and CRP (Miller & Chen, 2007; Packard et al., 2011; Carpenter et al., 2010; Lin et al., 2016).

Additionally, the ALSPAC dataset is a rich source of data for both psychopathology and inflammatory markers. Previous research has effectively utilised the ALSPAC dataset to investigate the relationship between IL-6 and CRP and depression (Khandaker et al., 2014; Osimo et al., 2020); psychosis (Perry et al., 2019; 2021; Donnelly et al., 2022) and anxiety (Mongan et al., 2023; Khandaker et al., 2016). Therefore, use of the ALSPAC dataset in the current study is a reliable source of inflammation data that is well poised for use in the first longitudinal study of the relationship between inflammatory markers and DP and DR. Additionally, the consistent success of using CRP and IL-6 within multiple empirical studies demonstrates its versatility and applicability to investigating several different psychopathologies.

Further, there is evidence of symptom specificity regarding the relationship between inflammatory markers and symptoms, for example in depression. Raison and Miller (2015) proposed that inflammation may be linked to specific depressive symptoms rather than depression as a whole - a notion later supported by findings that inflammatory markers were associated with fatigue, low energy, sleep disturbances, and appetite changes (Jokela, Virtanen, & Batty, 2016). Subsequent research using large-scale datasets has further refined this understanding (Milaneschi et al., 2021; Frank et al., 2021), supporting the symptom-specific relationship between inflammation and mental health using large sample sizes. Further, in bipolar disorder (BD), inflammation appears to play a state-dependent role. Post-mortem and genetic studies have identified increased pro-inflammatory and reduced anti-inflammatory markers in the frontal cortex of BD patients (Barbosa et al., 2014). IL-6 has emerged

as a consistently elevated marker across mood states, with inflammation intensifying during manic and depressive episodes. CRP levels are also significantly higher in BD, particularly during manic and euthymic phases, suggesting symptom-specific immune profiles (Dargél et al., 2015). Interestingly, dissociation is highly associated with BD, potentially suggesting that inflammatory markers seen in BD may be seen in dissociation and DPDR, too (Mula et al., 2009; Steardo et al., 2021). As DPDR is made up of two main symptoms (DP and/or DR), separating them is essential to see whether inflammatory markers are symptom specific in DPDR.

Beyond absolute levels of inflammatory markers, increasing attention is being paid to the effects of biological instability on psychopathology. For example, the role of inflammatory variability may serve as a marker for allostatic overload (McCrory et al., 2023; Finlay et al., 2022), particularly relevant for symptoms linked to chronic stress, such as DP and DR. Allostatic load (AL) is critical to measure given its link to worse psychological outcomes. For example, there is evidence to suggest that AL is positively associated with increased risks of depression, anxiety, and suicide (Gou et al, 2025). Despite its high risks, once AL is identified as an issue, can be used as a treatment target and thus a tool to increase overall wellbeing (Fava et al., 2023). Therefore, as inflammation is a biomarker of system overload, the presence of low-grade inflammation serves as an indicator of AL.

Despite growing insights into the inflammatory mechanisms underlying DP and DR, research remains limited by small, clinical sample sizes, limiting generalisability and power. Given the prolonged time to diagnosis (Baker et al., 2003) and the likelihood that many individuals with DP and DR symptoms remain undiagnosed, larger population-based studies are necessary to better capture the full spectrum of individuals experiencing DP and DR.

The aim of the current study was to address these limitations in the literature and investigate the effect of inflammatory markers on DP and DR symptoms in a large prospective cohort study in the general population, thereby providing crucial novel insights into the biological mechanisms of sub-clinical DDD.

3.1.3 The present study

This study seeks to build on previous research demonstrating a relationship between inflammatory markers and DPDR using longitudinal data from a large general population sample. Additionally, the role of IL-6 was investigated, which has not yet been used as an inflammatory marker of interest for DPDR.

Further, this was the first study to observe whether inflammatory markers serve as a mediator between ACEs and DPDR. Additionally, previous research has identified that inflammatory markers may be symptom specific, and previous research additionally demonstrates that DP and DR may have separate biological aetiology. Therefore, this was the first study to investigate the effects of different inflammatory markers on the two distinct phenomena of DPDR.

3.1.4 Research questions

It should be noted that there was only availability of IL-6 data at age 9, whereas there was availability of CRP data at ages 9, 15 and 24. These discrepancies are reflected in the hypotheses and are discussed in detail in the methods section.

3.1.4.1 Long-term Effects of IL-6

RQ1 (Depersonalisation): Do higher levels of IL-6 at age 9 predict greater odds of experiencing depersonalisation symptoms at ages 12, 17, and/or 24, after adjusting for sex, ethnicity, and social position?
RQ2 (Derealisation): Do higher levels of IL-6 at age 9 predict greater odds of experiencing derealisation symptoms at ages 12, 17, and/or 24, after adjusting for sex, ethnicity, and social position?

3.1.4.2 Medium-term Effects of CRP

RQ3 (Depersonalisation): Do higher CRP levels at ages 9, 15, and 24 predict increased odds of experiencing depersonalisation symptoms at age 12, 17, and 24, respectively, after accounting for sex, ethnicity, and social position?
RQ4 (Derealisation): Do higher CRP levels at ages 9, 15, and 24 predict increased odds of experiencing derealisation symptoms at age 12, 17, and 24, respectively, after accounting for sex, ethnicity, and social position?

3.1.4.3 Mediation of ACEs' Effects on Depersonalisation and Derealisation

a) By IL-6

RQ5 (IL-6 & Depersonalisation): Does Interleukin-6 at age 9 mediate the relationship between exposure to ACEs (both individual* and cumulative** between 8 months and 9 years) and increased odds of experiencing depersonalisation symptoms at ages 12, 17, and/or 24?

RQ6 (IL-6 & Derealisation): Does Interleukin-6 at age 9 mediate the relationship between exposure to ACEs (both individual* and cumulative** between 8 months and 9 years) and increased odds of experiencing derealisation symptoms at ages 12, 17, and/or 24?

b) By CRP

RQ7 (CRP & Depersonalisation): Does C-reactive protein at ages 9, 15, and/or 24 mediate the relationship between exposure to ACEs (both individual* and cumulative** between 8 months and 12 years) and increased odds of experiencing depersonalisation symptoms at ages 12, 17, and/or 24?

RQ8 (CRP & Derealisation): Does C-reactive protein at ages 9, 15, and/or 24 mediate the relationship between exposure to ACEs (both individual* and cumulative** between 8 months and 12 years) and increased odds of experiencing derealisation symptoms at ages 12, 17, and/or 24?

*Individual ACEs: Physical Abuse, Emotional Abuse, Physical Neglect, Emotional Neglect, Witnessing Inter-parental Violence, Parental Substance Abuse, Parental Mental Illness, Parental Divorce, Parental Conviction

**Accumulated ACEs: Abuse-related ACEs (Sexual Abuse, Physical Abuse and Emotional Abuse), Neglect and Household Instability ACEs (Physical Neglect, Emotional Neglect, Witnessing Inter-parental Violence, Parental Substance Abuse, Parental Mental Illness, Parental Divorce, Parental Conviction)

3.2 Methods

3.2.1 Dataset

Participant data from the ALSPAC were used. The ALSPAC dataset is a long-term research project that began in the 1990's and recruited over 14,000 pregnant mothers in the Avon area of England between April 1991 and December 1992. The ALSPAC collects data annually, and data can be categorised as: mother-reported, partner-reported, child-based, and child-completed. Data is obtained through questionnaire, clinical assessments, biological samples and linkage to health records (www.alspac.bris.ac.uk).

3.2.2 Participants

Initially, the current study had 7906 study child participants from ALSPAC who had completed the Psychosis-Like Symptoms (also known as PLIKS) interview at least once. However, as CRP and IL-6 were measured at varying ages and frequencies, attrition impacted these variables differently. The decision was made to create and run analysis using two different datasets, based on the number of individuals who had measurement data for DPDR, ACEs and inflammatory markers (Dataset 1: IL-6 [N = 2,560], Dataset 2: CRP [N = 5,127]), in order to retain the maximum sample size possible (demographic information in table 7).

Table 7: Chapter 3 demographic Information

Demographics	CRP Dataset	IL-6 Dataset
	N (%)	
Sex		
Male	2438 (47.55)	1208 (47.19)
Female	2689 (42.45)	1352 (52.81)
Ethnicity		
White	4620 (90.11)	2182 (85.23)
Non-white	181 (3.53)	92 (3.60)
Missing	326 (6.36)	286 (11.17)
Registrar General's Social Class Scale - Father's employment		
I (Professional	590 (11.51)	305 (11.91)

II (Managerial / Technical)	1663 (32.44)	804 (31.41)
III – N (Skilled non-manual occupations)	568 (11.08)	292 (11.41)
III – M (Skilled manual occupations)	1229 (23.97)	567 (22.15)
IV – Partly-skilled occupations	354 (6.91)	137 (5.35)
V – Unskilled occupations	92 (1.79)	43 (1.68)
Missing	631 (12.30)	412 (16.09)

3.2.3 Measures

3.2.3.1 Depersonalisation and derealisation

Participants completed the PLIKS interview at ages 12 (mean age 12.9, range = 12.5-13.3 years) and/or 17 (mean age 17.8 years, range = 16.25-20.1 years) and/or 24 (mean age 24 years, range = 22.4-24.4 years). At each measurement point, participants were asked if they “ever felt that the world was unreal, that things around them were like a stage set” (DR) or “ever felt that they were not a real person, not part of the living world” (DP). Responses of 'frequently' or 'sometimes' were coded as experienced DP or DR. These variables were set up as binary values (0 = No, 1 = Yes). Further information can be seen in Appendix 3.

3.2.3.2 Adverse childhood experiences

Participants had prospective measurements of ACEs (see Appendix 1 for a detailed explanation) allowing us to observe the cumulative effect of single ACE types from the age of 8 months to 9 years. In the previous chapter, ACEs were measured up to age 11, however, given this study’s biological measures were first taken from age 9, ACE value creation was capped at 9 years, ensuring that the influence of events that happened after the age of 9 were not accounted for. Additionally, ACEs were grouped into: (1) abuse-related ACEs (EA, PA, and SA) and (2) neglect and household instability (EN, PN, PSA, PMI, IPV, divorce, and conviction) cumulative variables. This grouping enabled comparison between traditionally examined ACEs (e.g., abuse) and less traditionally examined ones (e.g., neglect and instability).

To reduce bias associated with retrospective reporting of abuse, EA and PA were assessed using prospective parent reports. At each time point, mothers and partners

reported on both self- and partner-perpetrated abuse. If data from one parent were missing, the other parent's report was used. In cases of disagreement, endorsement of abuse was prioritised, under the assumption that self-reports may underrepresent abusive behaviour (see Chapter 2, Section 2.2.2.2 for a detailed overview).

3.2.3.3 Biological measures at age 9

The Focus@9 non-fasting blood samples were assayed in 2008 after a median of 7.5 years in storage with no previous freeze–thaw cycles during this period. All assay coefficients of variation were <5%. Interleukin 6 was measured by enzyme-linked immunosorbent assay (the IL-6 Immunoassay kit was manufactured and supplied by R&D Systems Europe Ltd), and high-sensitivity CRP was measured by automated particle-enhanced immunoturbidimetric assay (The manufacturer and supplier of these kits is Roche Diagnostics GmbH). The IL-6 analysis (ELISA) was carried out by Dr Lynne Cherry and Miss Pauline Watt of the Metabolic Medicine Group, of the Department of Vascular Biochemistry, BHF GCRC, Glasgow University. A Multiskan Ascent Plate reader and Ascent Software were used for the calculation of results. IL6 was measured twice at 9 years old in different labs. (IL6_pgml and IL6_log2). The variable IL6_pgml was measured using Picogram/milliliter scale (the variable used in the present study). IL-6 levels at age 9 in the total sample ranged from 0.1 to 13.9 pg/mL. Individuals with recent infection within the past two weeks were excluded from the study. CRP levels at age 9 in the sample initially ranged from 0.1mg/L – 67.44mg/L, and individuals with CRP levels of >10mg/L were excluded from the study. IL-6 and CRP were log-transformed.

High-sensitivity CRP assays were employed for CRP measurements at each age. This methodology is necessary to detect the low levels of CRP that are reflective of low-grade, systemic inflammation, which serves as a biomarker of chronic stress. For ease of readability and flow throughout the remainder of the results and discussion chapters, high-sensitivity CRP is referred to as CRP.

3.2.3.4 Biological measures at age 15

TeenFocus 3 (biological measures for age 15) participants were asked to fast overnight (for those attending in the morning) or for a minimum of 6 hours for those

attending after lunch. Blood samples were immediately spun and frozen at –80oC. Measurements were assayed in batches 3-12 months after sampling with no previous freeze-thaw cycles during this period. All assays were completed at Professor Naveed Sattar's laboratory at the University of Glasgow. CRP was measured by automated particle-enhanced immunoturbidimetric assay (the manufacturer and supplier of these kits is Roche Diagnostics GmbH).

CRP levels at age 15 in the sample initially ranged from 0.7mg/L – 72.55mg/L, and individuals with CRP levels of >10mg/L were excluded from the study. CRP values were log-transformed.

3.2.3.5 Biological measures at age 24

CRP were measured by particle enhanced immunoturbidimetric assay using the Cardiac C-Reactive Protein (Latex) High Sensitive kit (the manufacturer and supplier of these kits is Roche Diagnostics GmbH). CRP levels at age 24 in the sample initially ranged from 0.1mg/L – 14.93mg/L, and individuals with CRP levels of >10mg/L were excluded from the study. CRP values were log-transformed.

3.3 Model building

The primary outcomes of this study are DP and DR symptoms, assessed at ages 12, 17, and 24, with both items coded as binary (0 = Did not experience, 1 = Experienced) to indicate the presence of symptoms. To analyse these repeated binary outcomes, generalised linear mixed models (GLMMs) were employed, specifically utilising a binomial distribution with a logit link function. This approach allows us to account for the non-independence of observations within the same individual over time by including participant IDs as random intercepts. While missingness may not be completely random in this cohort, GLMMs provide a flexible and appropriate framework for analysing unbalanced longitudinal data under plausible assumptions about the missing data mechanism.

Analyses were conducted using two separate datasets to accommodate the differing availability of inflammatory marker data and retain the maximum possible sample size. Dataset 1 focussed on IL-6 levels measured at age 9 (N = 2,560), investigating

the long-term association with DP and DR outcomes across all three developmental stages. Dataset 2 examined CRP levels, measured at ages 9, 15, and 24 (N = 5,127), and their medium-term effects on DP and DR at subsequent time points.

For all models, common confounding variables were adjusted for, including sex, ethnicity, and social position as fixed effects. While inclusion of ethnicity and social position led to the dataset being reduced due to missing data, these are important to include as they are commonly associated with inflammatory markers and are therefore significant confounding variables.

To investigate whether inflammatory markers interact with time to predict the odds of DP and DR, this study's GLMMs included an interaction term between the specific inflammatory marker (IL-6 or CRP) and time (age), allowing us to assess how the marker's association with DPDR may change across developmental stages. Furthermore, to explore the mediating role of IL-6 and CRP in the relationship between ACEs and later DP and DR symptoms, a three-step GLMM approach was used. First, the association between ACEs (individual types and cumulative groupings) and DP and DR were established (see Appendix 3). Second, the association between ACEs and the mediator (IL-6 or CRP; see Appendix 3) was established. Finally, the mediator was added to the ACE-DPDR model: if the association between ACEs and DPDR is attenuated (reduced or becomes non-significant) while the association between the mediator and DPDR remains significant, candidates were indicated for formal mediation analysis.

3.4 Data analysis

Data were analysed using RStudio (R version 3.3.4). The following R packages were used for data organisation, statistical analysis, and visualisation: dplyr (Wickham, 2015), glmmTMB (Brooks et al., 2017), and ggplot2 (Wickham, 2016). Analysis was conducted using GLMMs covariates included sex, ethnicity, and social position, to adjust for potential confounding effects.

3.5 Results

3.5.1 Longitudinal association between interleukin-6 and depersonalisation and derealisation symptoms

This section details the results of the GLMMs and the subsequent mediation analysis. The primary goal was twofold: first, to determine if a direct relationship exists between the inflammatory marker IL-6, and DP and/or DR, and second, if that relationship exists, to test if IL-6 mediates the known link between ACEs and DPDR (as established in Chapter 2).

For mediation analysis to be statistically viable, the ACEs must predict both the DPDR outcomes and the inflammatory marker. Unfortunately, across all tested ACEs and inflammatory markers, only two candidates met these criteria for potential mediation: IPV and neglect/household instability-related ACEs.

Therefore, given this study's exploratory nature, the main chapter is focused on the direct effect of IL-6 on DP and DR and the attempted mediation analysis. Relationships that did not meet the criteria for mediation are excluded from the primary discussion. However, all other statistics are reported in Appendix 3.

Each analysis was performed twice: (1) the unadjusted model, and (2) the adjusted model, which controls for sex, ethnicity and social position which are all commonly associated with elevated inflammation (Szanton, Gill & Allen, 2005; Gruenewald et al., 2012; Petrovic et al., 2016; Moore et al., 2021).

Unadjusted model statistics for the association between IL-6 at age 9 and DP and DR at ages 12, 17 and 24 can be seen in table 8 below.

In the adjusted model, IL-6 at 9 years was not significantly associated with DP at age 12 (see table 8). Further, in relation to DP scores at age 12, IL-6 did not significantly predict increased odds of experiencing DP at age 17 (see table 8 below). However, higher IL-6 levels at age 9 was associated with an increased odds of experiencing DP at age 24, in comparison to baseline (see table 8). The adjusted odds ratios (aOR) indicated a 52% increase in the odds of experiencing DP, supporting a dose-

dependent relationship between early-life IL-6 and later DP experiences. Sex (aOR = 1.01, 95% CI: 0.52 – 1.94, p = .986), social position (aOR = 0.95, 95% CI: 0.74 – 1.23, p = .693) and ethnicity (aOR = 1.67, 95% CI: 0.35 – 7.77, p = .521) were not significant predictors of DP, and did not attenuate the relationship between IL-6 and DP.

Further, in the adjusted model, IL-6 did not exhibit a significant relationship to DR at age 12 (see table 8). Further, in relation to DR at age 12, IL-6 did not predict a significant increase or decrease in the odds of experiencing DR at age 17 or age 24 in comparison to baseline (see table 8). Sex (aOR = 1.12, 95% CI: 0.60 – 2.05, p = .733), social position (aOR = 0.87, 95% CI: 0.68 – 1.11, p = .254) and ethnicity (aOR = 1.84, 95% CI: 0.45 – 7.61, p = .393) were not significant predictors of DR, and did not change the relationship between IL-6 and DR.

Table 8: Unadjusted and adjusted longitudinal associations between interleukin-6 and depersonalisation and derealisation trajectories

		Unadjusted model			**Adjusted model**		
	Age	*OR*	*CI*	*P*	*aOR*	*CI*	*P*
Depersonalisation	12	0.98	0.79 – 1.22	.881	0.97	0.76 – 1.25	.819
	17	0.96	0.68 – 1.35	.801	0.95	0.66 – 1.38	.800
	24	1.45	1.12 – 1.90	**.005****	1.52	1.14 – 2.03	**.004****
Derealisation	12	0.99	0.79 – 0.99	.958	0.99	0.77 – 1.27	.939
	17	0.98	0.73 – 1.31	.884	1.03	0.76 – 1.38	.864
	24	1.01	0.76 – 1.35	.923	1.03	0.76 – 1.38	.867

3.5.2 Association between C-reactive protein and depersonalisation and derealisation symptoms

CRP was measured at age 9 (corresponding to DPDR measured at age 12), age 15 (corresponding to DPDR at age 17), and age 24 (measured a few months before DPDR assessment at age 24).This design offers a methodological advantage: whereas the IL-6 analysis examines the association between a single childhood inflammatory measurement and later mental health, the CRP analysis allows us to explore how changes in inflammation over time may be associated with changes in DPDR. Thus, the CRP results can be interpreted as reflecting potential dynamic,

time-varying relationships between inflammation and mental health outcomes, rather than only long-term effects of childhood inflammation.

The unadjusted model statistics for the association between CRP at ages 9, 15 and 24 and the odds of experiencing DP and DR at ages 12, 17 and 24 can be seen in table 9 below.

In the adjusted model, no significant relationship was observed between CRP at 9 and DP at age 12 (see table 9). Additionally, in relation to DP at age 12, CRP at age 15 did not predict significant increases or decreases in the odds of experiencing DP at age 17 (see table 9) and CRP at age 24 did not predict increased odds of DP at age 24 (see table 9). Sex (aOR = 1.08, 95% CI: 0.53 – 2.20, p = .821), social position (aOR = 0.97, 95% CI: 0.73 – 1.28, p = .831) and ethnicity (aOR = 1.31, 95% CI: 0.22 – 8.01, p = .766) were not significant predictors of DP and did not change the lack of relationships observed in the unadjusted model.

In contrast, a significant relationship was observed between CRP at age 9 and DR at age 12 (see table 9), indicating a 43% increase in the odds of experiencing DR. Further, the relationship between CRP at age 15 was and DR at age 17 was not significantly different from the baseline relationship (see table 9). However, CRP at age 24 significantly predicted decreased odds of experiencing DR at age 24 in comparison to the baseline relationship (see table 9), indicating a 55% decrease in the odds of experiencing DR. Sex (aOR = 1.07, 95% CI: 0.55 – 2.12, p = 832), social position (aOR = 0.92, 95% CI: 0.71 – 1.20, p = .542), and ethnicity (aOR = 1.11, 95% CI: 1.18 – 6.96, p = .911) were not significant predictors and did not impact the relationships between CRP and DR to a significant degree.

Table 9: Unadjusted and adjusted longitudinal associations between C-reactive protein and depersonalisation and derealisation trajectories

		Unadjusted model			Adjusted model		
	Age	*OR*	*CI*	*P*	*aOR*	*CI*	*P*
Depersonalisation	12	1.00	0.77 – 1.31	.974	0.99	0.74 – 1.32	.959
	17	0.89	0.51 – 1.57	.688	1.02	0.55 – 1.88	.960
	24	0.85	0.49 – 1.46	.554	0.83	0.46 – 1.51	.536
Derealisation	12	1.45	1.13 – 1.88	**.004****	1.43	1.09 – 1.90	**.010***
	17	0.78	0.50 – 1.22	.277	0.74	0.45 – 1.21	.233
	24	0.51	0.32 – 0.79	**.002****	0.45	0.27 – 0.72	**<.001*****

3.5.3 Relationships between ACEs, inflammatory markers, depersonalisation and derealisation

As previously discussed, IPV predicted IL-6 at age 9 (B = 0.24, 95% CI: 0.09 – 0.38, p = .002) and DP at age 24 (OR = 3.07, 95% CI: 1.42 – 6.62, p = .004). Additionally, IL-6 at age 9 predicted DP at age 24 (OR = 1.45, 95% CI: 1.12 – 1.90, p = .005). To investigate if IL-6 played a mediating role in the relationship between IPV and DP, IL-6 was added into the model. However, adjusting for IL-6 at age 9 did not attenuate the relationship between childhood IPV exposure and DP at age 24, which remained significant (OR = 1.94, 95% CI: 0.95 – 3.03, p = <0.001).

Further, neglect and household instability-related ACEs predicted IL-6 at age 9 (B = 0.12, 95% CI: 0.05 – 0.19, p <.001) and DP at age 24 (OR = 1.36, 95% CI: 1.12 – 1.65, p = .002). Again, IL-6 at age 9 predicted DP at age 24 (OR = 1.45, 95% CI: 1.12 – 1.90, p = .005). To investigate if IL-6 played a mediating role, it was added into the model, but did not significantly alter the relationship between neglect and household instability-related ACEs and DP at age 24 (OR = 1.36, 95% CI: 1.12 – 1.67, p = .002).

To see all other analyses between ACEs, DPDR and inflammatory markers, see Appendix 3.

3.5.4 Post-hoc results

To evaluate the potential association between inflammatory instability and changes in DR symptoms over time, a Difference-in-Differences (DiD) analysis was conducted. This method was chosen to estimate the isolated effect of significant CRP fluctuations, used here as a proxy for unstable systemic inflammation, on the emergence of DR symptoms between age 17 and age 24.

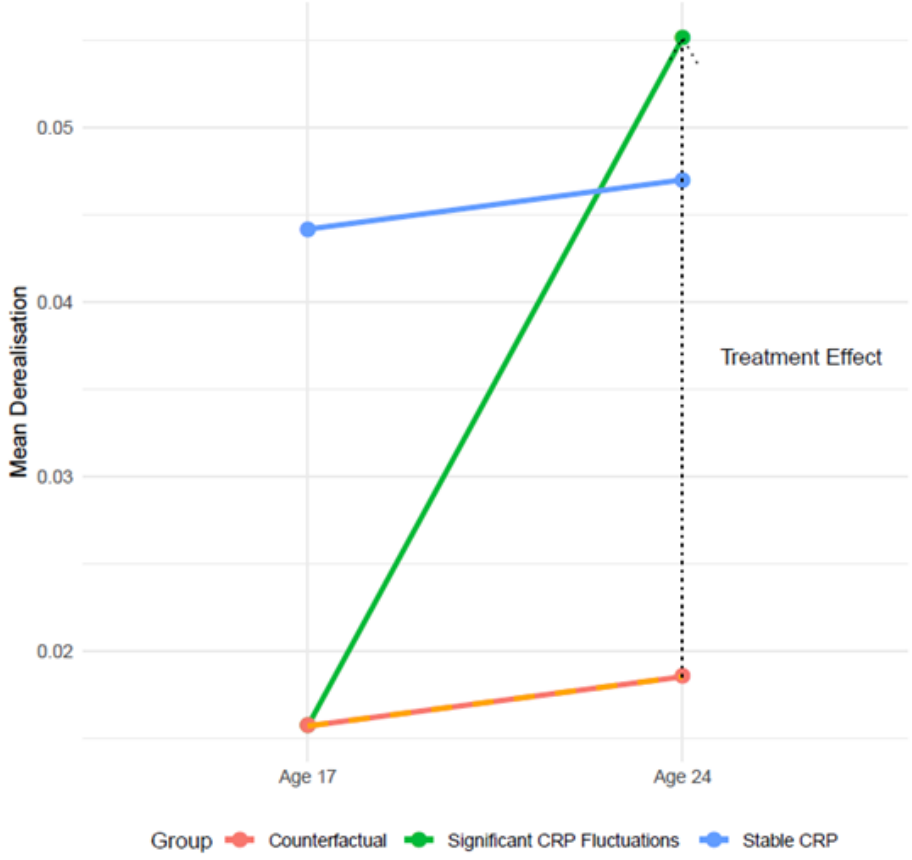
The DiD approach allows for comparison of change in outcomes across two groups over time: a ‘treatment’ group (participants with significant fluctuations in CRP levels) and a control group (participants with stable CRP levels). This design controls for both baseline differences between groups and for shared time-related changes that could influence all participants, thus enabling stronger causal inference from observational data.

For the unadjusted model, the DiD interaction term, representing the isolated ‘treatment’ effect of CRP change over time, was statistically significant (OR = 14.59, 95% CI: 1.12 – 190.57, p = .041). This OR of 14.59 indicates that individuals with significant CRP fluctuations had 14.5 times higher odds of experiencing DR symptoms at age 24, compared to what would be expected based on temporal trends in the stable CRP group. However, the confidence intervals were wide, and therefore, the interpretation of this result should be approached with caution. The wide confidence intervals suggest a high degree of uncertainty around the estimate, and while the point estimate indicates a substantial effect, the true effect could vary widely. These large intervals may indicate that further research with larger sample sizes or refined measures of CRP fluctuation is needed to better understand the relationship between CRP fluctuations and DR symptoms at age 24.

For the adjusted model, the DiD interaction term was statistically significant (aOR = 39.68, 95% CI: 2.39–665.14, p = .010). However, the confidence intervals are extremely wide, indicating a high degree of uncertainty around the effect estimate, likely due to small sample sizes or sparse outcome data in some covariate subgroups. Therefore, this adjusted model cannot be interpreted as providing a precise or stable estimate of the treatment effect, and the findings should be considered exploratory.

Figure 10 illustrates the model-predicted probabilities across groups and time. The counterfactual trajectory for the CRP fluctuation group (dashed line) represents the expected outcome had this group followed the same trend as the stable CRP group. The vertical arrow marks the estimated DiD effect, capturing the divergence due to CRP instability.

Figure 10: Difference-in-Differences analysis of CRP fluctuations and changes in the odds of derealisation from age 17 to 24



3.6 Discussion

3.6.1 Discussion outline

This section interprets the findings of this study, which utilised the ALSPAC dataset to investigate whether a longitudinal relationship between inflammatory markers and DPDR. The interpretation is organised around the study's research questions. First, the novel results are interpretated, namely, the symptom-specific associations

between IL-6 and DP and CRP and DR, within the context of the established neurobiological model of DPDR. Crucially, it highlights how the use of a large, longitudinal, population-based cohort provides novel insights into these biological mechanisms in sub-clinical DPDR, extending previous research that was limited to small, clinical samples. Subsequently, the observed relationships in terms of biological embedding, exploring how ACEs may influence subsequent inflammation (IL-6 and CRP), which in turn, may impact the later emergence of DP and DR symptoms, even when a formal mediation is not established, is discussed. The novel post-hoc finding regarding CRP instability and its potential relevance to the concept of AL is then discussed. Finally, this study's limitations and practical and theoretical implications are discussed, suggesting guidance for future research into the neurobiological mechanisms underlying DPDR.

3.6.2 Research Questions 1 and 2

Research questions 1 and 2 aimed to identify whether higher levels of IL-6 at age 9 would predict increased odds of DP and DR symptoms at ages 12, 17, and/or 24, after adjusting for sex, ethnicity, and social position. The findings demonstrated that that IL-6 at age 9 did not significantly predict increased odds of experiencing DR at any age (see table 8). However, the results demonstrated that IL-6 at age 9 predicted 52% increased odds of experiencing DP at age 24, but not earlier, suggesting a biological incubation period where the developmental impact of early immune activation may remain latent. This interpretation would align with evidence suggesting that early danger exposure and inflammatory processes exert delayed effects on young adult behaviour and health outcome (Beach et al., 2015). Further, this latency is consistent with the Neuro-immune Network (NIN) model (Nusslock & Miller, 2016), positing that ACEs can trigger persistent changes in peripheral inflammation. As individuals mature, these biological shifts interact with typical life stressors, potentially triggering the eventual expression of underlying vulnerabilities like DP.

Within this framework, this study's findings align with previous studies demonstrating similar relationships between childhood IL-6 and a range of adult psychopathology. Specifically, elevated childhood IL-6 levels have been identified as a predictor for various mental health outcomes, including depression (Chu et al., 2019), hypomania

(Hayes et al., 2017), and later depressive and psychotic symptoms in young adulthood (Perry et al., 2021).

A contextual explanation of this latent emergence of DP may be the transition into “emerging adulthood”, a distinct and significantly challenging developmental period identified by Arnett (2000). This stage is marked by major demographic shifts (e.g., independent living, university enrolment) and subjective distinctions (e.g., identity exploration, navigating the 'in-between' state). While emerging adulthood offers substantial opportunities for personal growth, the nature of these concurrent life changes can elicit unprecedented stress. This increased stress may serve as the environmental trigger necessary for the expression of latent childhood developmental anomalies, such as low-grade inflammation, ultimately increasing the risk of negative young adult outcomes, including the emergence of DP.

Viewing the emergence of DP through the NIN framework provides several promising avenues for treatment. Evidence suggests that cultivating a healthy NIN through interventions such as healthy sleep practices, physical activity, and promoting a healthy gut microbiome may mitigate the long-term negative impact of early immune activation in adulthood (Bower et al., 2019).

3.6.3 Research Questions 3 and 4

Research Questions 3 and 4 aimed to explore the relationship between CRP levels at three developmental stages (ages 9, 15, and 24) and the subsequent odds of experiencing DP and DR (at ages 12, 17 and 24) respectively, while controlling for key covariates (sex, ethnicity, and social position).

The findings clearly indicated a symptom-specific separation between the two outcomes. CRP did not predict DP at any developmental stage (see Table 9), whereas CRP predicted DR, albeit in varying ways, at ages 12 and 24, but not at age 17. CRP at age 9 significantly predicted 43% increased odds of experiencing DR at age 12, and CRP at age 24 significantly predicted 55% decreased odds of experiencing DR at age 24, relative to baseline. These results highlight the

importance of distinguishing DP and DR, suggesting that only DR is associated with the CRP, at specific developmental stages.

The relationship between static CRP levels and DR appeared complex, suggesting a possible shift in inflammatory vulnerability across the lifespan. Post-hoc DiD analyses revealed that individuals with significant CRP fluctuations had a substantial increase in the odds of DR symptoms at age 24, representing a 449% increase in odds, compared to those with stable CRP levels, who had no statistically significant change, suggesting a more consistent symptom trajectory for this control group. These results suggest that instability in inflammatory signalling, rather than its direction or magnitude, may be more closely associated with DR exacerbation. However, the very wide confidence intervals surrounding these large effect estimates indicate substantial statistical uncertainty, likely reflecting small subgroup sizes.

Nevertheless, this exploratory pattern warrants interpretation within frameworks that account for physiological variability rather than causal inference and should be examined further in larger samples. Notably, it aligns with AL models, which emphasise fluctuating physiological stress responses over mean inflammatory levels.

The only previous study that investigated the relationship between CRP and DDD identified a downregulation of CRP in individuals with DDD compared to HC (Zheng et al., 2024). However, in comparison to the present study, CRP downregulation in the Zheng et al. cohort was only significantly correlated with the component of ‘emotional numbing’, and not with ‘unreality of surroundings’ (which is reflective of DR). These combined findings strongly suggest that CRP's relevance is limited to specific dissociative features rather than the disorder, powerfully supporting the notion that symptom specificity is a crucial distinction in DPDR research.

The difference in the associated symptom (DR in the current study vs. emotional numbing in Zheng et al., 2024) may stem from methodological differences, such as the use of small clinical samples in the prior work. Furthermore, the present study's reliance on specific measures of DP and DR, rather than including additional components like anomalous subjective recall and emotional numbing (Sierra et al., 2005), means that DR variance may have inadvertently absorbed the variance

typically explained by emotional numbing. Nevertheless, the results unequivocally demonstrate a clear, symptom-specific relationship between CRP and the DR component of the dissociative continuum.

These findings indicate that instability of inflammatory signalling, rather than direction, suggest that chronic or fluctuating inflammation could be a meaningful contributor to the persistence of DR in early adulthood, potentially reflecting a relationship between AL and DR. As a reminder, AL refers to the dysregulation of the body's stress response system. While ACEs were not candidates for testing mediation between stressful life events and DPDR, there are several other stress-related childhood phenomena that were not utilised as a covariate but could have significant impact on both inflammatory markers and DPDR. Importantly, AL can arise from subjective experiences rather than objective; for example, while evidence consistently links AL to social position (Szanton, Gill & Allen, 2005; Gruenewald et al., 2012), age, race/ethnicity and gender (Petrovic et al., 2016; Moore et al., 2021), evidence also suggests that perceived social status (e.g. the person's sense of control, placement in the community rank hierarchy, perception of inequality in the workplace) is associated with higher levels of AL (Seeman et al., 2014). While in the present study social position was operationalised through paternal employment status, providing little intel regarding the participants' own subjective social status, and therefore, an individual's self-perception may explain the relationship between biological dysregulation and DR.

Additionally, several linked risk factors for increased AL were not measured. For example, neighbourhood poverty, independent of household poverty, is demonstrated as a significant predictor of AL (Schulz et al., 2012), mediated by self-reported neighbourhood environmental stress. This suggests that objective neighbourhood poverty may lead to AL, but again, subjective perceptions also influence the relationship. Additionally, the heritability of AL is estimated to 30% (Petrovic et al., 2016), suggesting that genetic makeup may confound attempts to decipher its environmental determinants. The treatment implications of DR emerging through AL is discussed.

Previous research identifying downregulation of CRP correlating to positive DDD symptoms (Zheng et al., 2024) may suggest a trend in the CRP-DPDR literature specifically, as within the larger dissociation literature there is a trend towards increased inflammatory markers and dissociative symptoms (Power et al., 2019; Bizik et al., 2011, Jarkas et al., 2021; Bob et al., 2010), and the present study identified that increased early life IL-6 predicted DP suggesting upregulation may be unique to CRP. However, this is highly speculative, as the current study did not have longitudinal data for IL-6.

3.6.4 Research questions 5 and 6

Research questions 5 and 6 aimed to explore whether IL-6 mediated the relationship between ACEs (both individual and cumulative) occurring between 8 months and 9 years and DP and DR at ages 12, 17 and 24, after adjusting for sex, ethnicity and social position. Our results demonstrated mixed findings. Crucially, when the analysis from Chapter 2 was rerun within the smaller IL-6 dataset, some existing significant relationships did not remain significant (see Appendix 3).

For mediation to be statistically viable, the ACE predictor needed to be significantly associated with both the outcome and the proposed mediator (IL-6). EN, PA, PSA, and abuse-related ACEs did not predict increases in IL-6 at age 9. Consequently, only pathways involving IPV and neglect and household instability-related ACEs met the criteria for testing IL-6 mediation. When IL-6 was entered into these models, the relationships between both IPV and neglect/household instability-related ACEs and DP remained significant, suggesting that while these specific ACEs are linked to both immune system activation and DP, IL-6 did not appear to mediate them.

For DR, the required condition for mediation was absent, as IL-6 at age 9 did not significantly predict increased or decreased odds of DR at any age. The absence of a significant link between IL-6 and DR symptoms therefore precluded the possibility of addressing Research Question 6 entirely. Collectively, these findings for both outcomes suggest that IL-6 may not serve as a mediator between childhood ACEs and later DPDR.

These findings indicate that IL-6 may play a more independent role in the development of DP, rather than acting as a direct mechanism by which early adversity (specifically IPV) leads to later dissociation. Alternatively, the relationship between IPV and DP may be more directly explained by other biological or psychological mediators, such as HPA-axis dysregulation, emotion regulation difficulties, or neural alterations (Sturge-Apple et al., 2012; Samuelson et al., 2012; Tomoda et al., 2012).

Collectively, these results highlight the complexity of biological embedding and underscore the value of directly testing mediation within a comprehensive model. Assuming that a biological marker is automatically associated with both the exposure and the outcome based on prior literature carries a significant risk of model misspecification.

3.6.5 Research questions 7 and 8

Research questions 7 and 8 aimed to explore whether CRP at ages 9, 15, and/or 24 mediated the relationship between exposure to ACEs (individual and cumulative) and increased odds of experiencing DP at 12, 17 and/or 24 (research question 7) and derealisation at 12, 17 and/or 24 (research question 8).

Our analysis revealed a clear result: CRP did not significantly predict DP at any timepoint. Since a significant link between the mediator (CRP) and the outcome (DP) is a necessary condition, this finding precluded the possibility of conducting a mediation analysis for Research Question 7. These results suggest that CRP does not serve as a relevant biological pathway in the development of DP. This reinforces the notion that there are fundamental aetiological differences between these dissociative phenomena, which was consistently observed throughout this study.

However, while CRP at age 9 predicted increased odds of DR at age 12 and CRP at age 24 predicted decreased odds of DR at age 24, the necessary conditions for a direct ACE-CRP-DR mediation pathway were not met, as the required relationships were fragmented across the causal chain. For example, the ACEs that predicted the inflammatory marker did not align with the ACEs that predicted the outcome: only EN

predicted CRP at age 24, yet the corresponding outcome (DR at age 24) was predicted by different adversities (IPV and parental divorce). Similarly, only PSA predicted CRP at age 12, but this was not paired with an aligned outcome predictor.

Because these necessary mutual relationships, where a single ACE predicted both the required inflammatory marker and the dissociative symptom in the same model, were absent, mediation analysis could not be conducted. These findings suggest that while early adversity and inflammation may independently contribute to changing odds of DR, there is no evidence from the current study that CRP functions as a singular, mechanistic pathway linking early adversity to later DR.

3.6.6 Neurobiological model of depersonalisation and derealisation

The present study provides compelling support for the neurobiological model of DPDR. This model, initially proposed by Sierra and Berrios (1998), suggested that DPDR emerge from dysfunction in brain systems responsible for emotional processing, particularly suppression of limbic activity in response to stress (see 1.8 Neurobiological model of depersonalisation and derealisation). Further studies identified that individuals with DPDR have demonstrated reduced physiological reactivity to emotional stimuli (Millman et al., 2024). This view was extended by providing longitudinal evidence that alterations in stress-related physiological systems, specifically the immune response (via IL-6 and CRP), may contribute significantly to the onset and maintenance of symptoms.

Crucially, this study significantly advances the existing neurobiological literature by using a large, population-based cohort and a longitudinal design. By capturing IL-6 during childhood and systematically tracking its relationship with DP across multiple developmental time points, the findings offer stronger evidence of temporal precedence and enhance the generalisability of the results compared to previously available cross-sectional or smaller clinical studies. This longitudinal approach was particularly valuable for the CRP analysis, as it permitted the use of advanced methods to identify inflammatory fluctuations across development: a finding that would have been inaccessible in a static, cross-sectional design.

3.6.7 Neurobiological disparities in depersonalisation and derealisation

The contrasting results for childhood IL-6 predicting DP and CRP fluctuations predicting DR provide powerful empirical support for a divergence in the biological pathways underlying these two dissociative phenomena. This supports the notion that DP and DR, although often co-occurring, possess distinct neurobiological profiles and aetiological mechanisms (Sierra et al., 2002). For example, DP may involve frontal cortex hyperactivation or inhibition of limbic areas like the amygdala, while DR may involve disruption in temporal-parietal regions or dysfunction in multisensory processing (Murphy, 2023). While the current study did not directly assess brain areas, evidence demonstrates that peripheral inflammation can impact neural activity and reduce functional connectivity in brain networks implicated in mood disorders, such as the Default Mode Network (DMN) and Salience/Ventral Attention Network (Schmitz et al., 2025; Aruldass et al., 2021). Therefore, within the context of this study's findings, it is plausible that increased levels of inflammatory markers may impact brain areas responsible for DP and DR, and that the differential associations observed (e.g., IL-6 with DP, CRP with DR) may reflect distinct inflammatory markers preferentially influencing different neural circuits underlying these unique dissociative phenomena.

Building on this, given that DP is associated with disruptions in self-referential processing, one possible interpretation of the current findings is that early-life IL-6 exposure may influence DMN development and function, leading to long-term impairments in self-awareness and identity processing. Supporting this, a study investigating the neural basis of DP symptoms in depression found that reduced functional connectivity between the extrastriate body area (EBA), a region involved in the visual perception of the body, and the DMN predicted higher DP symptoms (Paul et al., 2019). While speculative, these findings suggest a potential theoretical link between peripheral inflammation, its downstream neuroinflammatory consequences, DMN-EBA connectivity, and the emergence of DP symptoms.

3.6.8 Allostatic load

The current findings could also be understood in the context of an AL framework. AL is defined as 'the cost of chronic exposure to fluctuating or heightened neural or neuroendocrine response resulting from repeated or chronic environmental challenge

that an individual reacts to as being particularly stressful' (McEwen & Stellar, 1993). Although establishing a significant mediation pathway between ACEs, inflammation, and DPDR was not possible in this cohort, the theoretical and empirical convergence remains compelling.

Individuals experiencing DPDR tend to have a history of chronic stress (Hunter, Sierra & David, 2004; Simeon, 2004), particularly marked by exposure to ACEs and often manifesting in conditions like the D-PTSD. Individuals with DDs and a history of childhood maltreatment are noted to have smaller amygdala and hippocampal volumes (Vermetten et al., 2006), areas of the brain highly associated with chronic stress and AL (Danese & McEwen, 2012). Therefore, the presence of DPDR in the cohort, coupled with the observed associations between low-grade systemic inflammation and both DP and DR, and the evidence pointing to immune dysregulation in their prevalence and potential maintenance, collectively suggest a state of 'allostatic overload' (McEwen, 2004).

3.6.9 Immune habituation or cessation of stressors

As discussed, CRP and DR revealed a reversal in the association between CRP and DR symptoms over time. Initially this may appear counterintuitive, as prior research demonstrates that increased CRP typically leads to an increase in psychiatric symptoms (Lee & Giuliani, 2019; Osimo, 2018; Osimo et al., 2019). However, these results may reflect immunosuppression because of long-term, chronic stress, which can suppress or dysregulate innate and adaptive immune responses (Dhabhar, 2014), with the body becoming less reactive to chronic stress and inflammation (Barthel et al., 2025).

This framework would explain why lower CRP at age 24, in comparison to higher CRP at age 12, was demonstrated. The immune system may have adapted or become less responsive due to repeated stress exposure. Even if inflammation stabilises or decreases over time, DR may persist, suggesting that while the body's response to stress may no longer be as acute, the psychological impact of prior stressors may endure.

Alternatively, this reduction in CRP could reflect the cessation of psychosocial stressors, yet DR persists. This is consistent with the cognitive-behavioural model of DPDR (Hunter et al., 2003), positing DPDR as an adaptive response to stress, where the individual's symptoms are an attempt to cope with overwhelming stress. However, as the individual continues to experience catastrophic interpretations of these symptoms, they maintain attention to DPDR. This reinforced attention and focus on symptoms can lead to recurrent episodes of DPDR, even after the initial stressor has subsided or the stress response (inflammation) has decreased.

3.6.10 Strengths and limitations

This study offers several key strengths that bolster the robustness and novelty of the findings.

First, a paramount strength lies in the use of a large, population-based longitudinal dataset. This extensive cohort not only provides statistical power but also significantly enhances the generalisability of these findings to a broader population, minimising the risk of selection bias often inherent in smaller or clinically ascertained samples. The longitudinal design is crucial, enabling robust tracking of inflammatory markers and dissociative symptoms across critical developmental stages, from childhood through early adulthood. Furthermore, the repeated measurement of CRP at ages 9, 15, and 24 represents a significant methodological advantage. This multi-time-point assessment allowed for dynamic identification of inflammatory fluctuations and trajectories over a substantial period, rather than relying solely on static, single time-point levels. This nuanced approach more accurately reflects the physiological instability and dynamic nature of systemic inflammation, which is arguably more pertinent to chronic health outcomes than isolated measurements.

Second, the detailed temporal data further facilitated the innovative, quasi-experimental application of a DiD model. This analytical framework provided a novel test of the hypothesis that CRP instability, rather than merely chronically elevated levels, is associated with the emergence or progression of DR. While this method offers a powerful framework for examining the temporal interplay between inflammatory dynamics and symptom presentation, the wide confidence intervals

associated with the resulting estimates require a cautious interpretation. The insights generated by this analysis are thus best understood as preliminary and exploratory, pointing toward a potentially relevant mechanism that warrants robust replication in larger samples.

Third, a notable contribution of this study is its position as one of the first to meticulously examine DP and DR as distinct outcomes within a neurobiological framework. By treating these experiences separately, and using objective biomarker data, this study explored potential differences in their etiological pathways. This approach offers a critical refinement that may help to explain and resolve inconsistencies prevalent in the existing literature, paving the way for more targeted aetiological investigations.

Finally, the integration of well-established theoretical frameworks, such as AL, significantly strengthens the interpretation of this study’s findings. By grounding demonstrated empirical observations in these robust models of chronic stress physiology, a coherent mechanistic basis is provided for understanding how prolonged or repeated exposure to stress, leading to allostatic overload, might contribute to inflammatory dysregulation and subsequent dissociative symptomatology. This theoretical grounding not only enhances the explanatory power of the results but also reinforces the study's contribution to the broader understanding of mind-body connections in psychopathology.

Despite these considerable strengths, this study also has important methodological limitations that warrant candid discussion.

First, while CRP was comprehensively assessed at three critical time points, IL-6 was only measured at age 9. This single time-point measurement severely limited the ability to assess the longitudinal inflammatory dynamics for this crucial cytokine. This disparity in temporal data collection between CRP and IL-6 restricted the capacity for a comparative longitudinal analysis of two key inflammatory markers, thus hindering a more complete understanding of diverse inflammatory pathways to DPDR.

Second, the reliance on binary measures of DPDR, while ensuring consistency across different time points, inherently constrained the granularity of outcome assessments. These binary indicators did not capture the continuous spectrum of symptom severity, frequency, or duration. Consequently, this limitation restricted the ability to assess precise dose-response relationships between inflammatory markers and DPDR, or to detect subtle, subthreshold symptom changes that may nonetheless be clinically significant or indicative of early pathophysiological processes.

Third, although analyses identified compelling associations between inflammatory markers and changes in DPDR, a critical limitation is that this study cannot definitively establish causality. The observed relationships could be bidirectional, meaning inflammation might directly contribute to symptom emergence, or conversely, DPDR itself (or an upstream common factor like chronic stress or early adversity) might drive inflammatory changes. Without experimental manipulation or more advanced causal inference designs that account for unmeasured confounders, the precise causal direction remains a complex challenge for observational studies.

Finally, it is particularly important to consider the potential impact of these methodological limitations on mediation analyses. While the overall cohort was substantial, the sample size available for robust longitudinal inflammatory analyses, especially for investigating mediation pathways, was comparatively reduced due to the inherently limited availability of complete inflammatory data at all required time points for every participant. This unfortunately diminished dataset size limited statistical power, specifically for detecting indirect or mediated effects, potentially leading to Type II errors where true underlying relationships or complex pathways might exist but were not detected as statistically significant. Therefore, the absence of statistically significant mediation effects in the tested models should not be definitively interpreted as a complete lack of a biological embedding pathway linking early adversity, inflammation, and DPDR. It is entirely plausible that with a larger, more complete, and comprehensively measured longitudinal dataset, certain mediation effects, or more nuanced indirect pathways, could be revealed. This underscores the pressing need for future research utilising larger, comprehensively phenotyped, and consistently measured longitudinal cohorts to more fully elucidate

the complex and intricate interplay between early adversity, inflammatory processes, and the development of dissociative symptoms.

3.6.11 Clinical implications and future directions

In the first instance, research needs to prioritise the investigation of inflammatory markers, or a collection of AL indices, in the relationship to DPDR. This study is a significant advancement in the DPDR research space and provides long-awaited potential for symptom management. While trauma-focused treatments remain the first-line approach for DPDR, this study suggests that immune-modulating strategies may be beneficial, particularly for patients with elevated IL-6 or CRP fluctuations. Interventions targeting systemic inflammation (such as omega-3 supplementation, probiotics, exercise, and cytokine-targeting therapies) could be explored as adjunctive treatments in DPDR, particularly in individuals showing inflammatory dysregulation.

Future research should examine the potential for anti-inflammatory treatments to have a beneficial impact on prevalence of DPDR. Previous research demonstrates that CRP level can help to guide antidepressant treatment for individuals with depression. Specifically, individuals with CRP levels <1 mg/L had better outcomes with monotherapy, and patients with >1 mg/L had better outcomes with a bupropion-SSRI combination (Jha et al., 2017). Following this logic, CRP-stratified antidepressant use could eventually be applied to DPDR, where individuals at a later age, or those with elevated or unstable CRP profiles might benefit more from treatments that target norepinephrine or dopamine systems, such as bupropion, noradrenergic antidepressants or anti-inflammatory agents like TNF-α antagonists. Conversely, those with low, stable CRP might respond better to traditional SSRIs or non-pharmacological approaches.

Additionally, while exploratory and limited by small sample sizes, the results from the DiD analysis suggested that the instability itself was the prevalent risk factor in continuation of DR symptoms at age 24 in relation to age 12. While these are by no means definitive findings, they do implicate that further research into the instability of CRP could be revolutionary in understanding DR, which has long been neglected yet may be more prevalent than DP, particularly at older ages (as discussed in Chapter

2). A valuable avenue for future research involves conducting longitudinal studies that track CRP levels over time to identify individuals with significant inflammatory fluctuations, which may reflect increased AL and physiological stress. These individuals could then be assigned to interventions designed to stabilise inflammation and reduce allostatic burden, such as pharmacological treatments or lifestyle modifications. Physiological stability could be operationalised as reduced variability in CRP levels over time. Investigating whether achieving such stability leads to reductions in DPDR could provide important insights into the role of chronic inflammation and stress-related processes in the development of DPDR.

3.6.12 Conclusion

This study provides compelling longitudinal evidence, suggestive that DP and DR may have distinct inflammatory antecedents, reinforcing the necessity to distinguish between these phenomena.

Our findings reveal a clear divergence in biological pathways: early IL-6 predicted the later emergence of DP symptoms in adulthood (at age 24), suggesting that DP may be rooted in a stable, developmental vulnerability stemming from early immune activation. In contrast, DR symptoms were linked to CRP through a complex, shifting relationship. The data suggested that the instability of inflammation over time, rather than just consistently high levels, was the key factor. This pattern aligns with the AL framework, which accounts for the systemic "wear and tear" caused by a chronically stressed and dysregulated body.

While attempts to formally test a mechanistic pathway where childhood stress is related to inflammation, which in turn is related to DPDR, were largely precluded, the results strongly reinforce the idea that conflating DP and DR risks obscuring their unique developmental trajectories. The observed biological divergence offers a powerful hypothesis that different inflammatory pathways may preferentially influence the distinct neural circuits underlying the perception of self (DP) and external reality (DR).

Chapter 4: What Triggers Recurrent Episodes of Depersonalisation and Derealisation? A Qualitative Analysis of Online Forum Data

Abstract

Background: Depersonalisation (DP) and derealisation (DR) are dissociative symptoms that can significantly impair functioning. DPDR often emerges following high stress situations, yet symptoms can persist following cessation of stressors. No research to date has investigated the factors leading to persistence of symptoms.

Purpose: To identify and categorise subjective triggers maintaining DPDR episodes using thematic analysis of online forum data.

Methods: Posts were extracted from two online forums, Reddit and DP Self Help, focusing on user discussions about subjective DPDR triggers. A total of 368 posts from 288 participants were analysed using inductive thematic analysis to identify recurring patterns of subjective triggers.

Results: Thematic analysis revealed three primary categories for subjective triggers of recurrent DPDR episodes: Dysregulation of Core Systems, Environmental Reactivity, and Cognitive Triggers. Dysregulation of core systems included stress activation, social disconnection, and sleep disturbances. Stress activation emerged as the most frequently reported trigger, and appeared to mediate the relationship between other triggers and DPDR onset. Cognitive triggers centred on philosophical-existential thinking and hyper-reflexive thoughts, which were characterised by distressing self-reflection and overthinking. Environmental reactivity included exposure to mirrors, specific lighting conditions, and screen exposure.

Conclusions: This study highlights the importance of managing stress as a key factor in reducing the frequency and severity of DPDR episodes. Findings emphasise the need for tailored interventions addressing physiological, environmental, and cognitive triggers to improve outcomes for individuals experiencing recurrent DPDR. These results provide new insights into DPDR triggers, paving the way for future research and clinical practice improvements.

4.1 Introduction

"There are many more people like us, dealing with depersonalisation, than we first thought. It upsets me that there is not any awareness around this condition" – Reddit user (reworded for anonymity)

4.1.1 Background to depersonalisation and derealisation

DPDR are dissociative symptoms, a group of symptoms related to disruptions in consciousness, identity, memory, perception, or awareness (American Psychiatric Association, 2013). DP involves experiences of unreality, including detachment, being an observer of one's thoughts, feelings, sensations, body or actions, and DR involves experiences of unreality regarding an individuals' surroundings (Hallett et al., 2025). Lifetime prevalence rates range from 26% to 74% (Hunter, Sierra, & David, 2004).

Despite this and the fact that DDD is one of the four DDs, recognised by the American Psychological Association and the World Health Organization, these symptoms are significantly underrepresented in psychiatric research. The functional consequences of DDD are severe, impairing daily functioning (American Psychiatric Association, 2013). Yet, it takes an average of 7-12 years to accurately diagnose DDD (Baker et al., 2003; Michal et al., 2016). Despite DPDR being under-studied, the prevalence within both clinical and general populations is high (Aderibigbe et al., 2001; Ross, Joshi & Currie, 1991) and are estimated to be the third most common mental health symptom, following depression and anxiety (Simeon et al., 2003). Additionally, DPDR can also serve as a mediator, linking childhood abuse and more severe psychological outcomes, such as psychotic-like experiences in adults (O'Neill et al., 2021) and deliberate self-harm in adolescents (Hoyos et al., 2019). Individuals experiencing DPDR utilise mental health care to a high degree, yet often find that existing mental health provisions do not meet their complex needs (Michal et al., 2016; Hunter, Charlton and David, 2017; Hansard, 2019).

4.1.2 Precipitants of depersonalisation and derealisation

The TM of dissociation (Dalenberg et al., 2012) suggests that dissociation serves a regulatory function in response to fear or other extreme emotions, with measurable

biological correlates. Within this framework, it is important to distinguish between predisposing factors, which increase long-term vulnerability to developing DPDR/DDD, and precipitating factors, which act proximally to trigger symptom onset. ACEs may operate as both: early trauma can confer enduring psychobiological vulnerability, while acute traumatic or stress-related events may precipitate dissociative symptoms in the shorter term.

Additionally, DPDR occurring at the time of a traumatic event ranges between 31% and 66% of individuals (Hunter, Sierra, & David, 2004), and is associated with forms of trauma at various ages. For example, those who had experienced interpersonal trauma in adulthood (Yang et al., 2023), or abuse in childhood (King et al., 2020; Michal et al., 2007,2009; Simeon, 2001; Simeon et al., 2008). Moreover, DPDR can be symptoms of various mental health conditions, such as panic disorder, depression and PTSD (Hunter, Sierra and David, 2004). DPDR can also occur in healthy people under conditions of stress or fatigue (Simeon, 2004; Trueman, 1984), for example, during the Covid-19 pandemic (Ciaunica et al., 2022). DPDR commonly occurs alongside drug use (such as cannabis, hallucinogens, ecstasy and ketamine) or extended periods of extreme stress or abuse, however, occasionally can have no obvious trigger.

4.1.3 Maintenance of depersonalisation and derealisation symptoms

Sensitivity to recurrent episodes distinguishes individuals who experience DPDR transiently from those who develop chronic symptoms (DDD). Chronicity arises when initially transient episodes evolve into a persistent source of distress, marked by increasing frequency and pervasive impact, making them no longer classifiable as transient. Despite this, very little formal research has investigated factors leading to recurrent DPDR episodes after initial triggers (such as a traumatic incident) subside, however, some factors are reported as part of therapeutic manuals, likely based on themes gathered by clinicians in clinical settings from the first-hand accounts of patients. An example is within the book “Cognitive Behavioural Approaches to the Understanding and Treatment of Dissociation” (Kennedy et al., 2013), whereby the authors state that triggers of a DPDR state may be due to thinking about DDD, physical anxiety, worrying, fluorescent lights, social situations, reading about psychosis, being ‘hungover’ and tiredness. Additionally, within the book “Feeling

Unreal: Depersonalisation Disorder and the Loss of the Self" (Simeon and Abugel, 2006), the authors state that even overwhelming joy can trigger DPDR.

Černis et al. (2020) explored factors that sustain dissociation through interviews with 12 individuals. Using deductive thematic analysis, they identified four subjective triggers: (1) stress (proximal and distal), (2) fatigue, (3) dwelling on thoughts and feelings, and (4) predisposing beliefs about emotion and stress. These findings offer valuable insights into the chronicity of dissociative symptoms, likely applicable to DPDR due to shared symptomatology. However, as the study focused on individuals with non-affective psychosis, further research specifically targeting DPDR is needed to better serve this underserved community.

While not explicitly investigating maintenance of DPDR, research demonstrates various factors as significantly associated with DPDR experiences, potentially supporting anecdotal reports. These include existential thinking (Ciaunica et al., 2023), rumination (Fortuna & Golonka, 2024; Vannikov-Lugassi et al., 2021; Hunter et al., 2023; Uysal, Ağaç & Konkan, 2025), hyper-reflexivity (Ciaunica, 2021; Cinaunica, 2022), specific environmental lighting (Baker et al., 2003), flashing lights, pulsing music, sustained visual fixation (Leonard, Telch & Harrington, 1999), panic (Miller et al., 1994; Lickel et al., 2008), virtual reality (VR) experiences (Peckmann et al., 2022), increased screen use, social isolation (Ciaunica, 2022) and social challenges (Ciaunica, 2023), mirror exposure, including mirror exposure methods to induce dissociation (Brake et al., 2025; Caputo et al., 2021; Shin et al., 2019), academic stress (Schweden et al., 2018), struggles with sensory overload (Harricharan et al., 2017), sleep issues (Arora et al., 2020; Menicucci et al., 2022; van Heugten-van der Kloet et al., 2015) and dysfunctional autonomic responses (for reviews see Horn et al., 2020). Therefore, factors of this nature are expected to contribute to maintenance of DPDR.

4.1.4 Forum use in the depersonalisation and derealisation community

As DPDR is under-researched and not widely discussed, those who experience it are prone to seek out information online and congregate within online communities globally to share information and support one another. Forums, or online discussion boards, are digital platforms where users exchange information, ask questions, and

discuss various topics asynchronously. Typically, forums are organised into categories or threads based on specific topics or themes, where users can post messages and replies, initiate discussions or contributing to existing ones. Forums can cover a wide range of subjects, including hobbies, health, and education, and can act as support groups, and are valuable resources for information exchange, community building, problem-solving, and advice giving. Many forums also have moderators who ensure a respectful and safe environment for all participants. Searching on forums is commonplace for the DPDR community as they persist in their goal for reducing symptoms and provide each other with a source of hope and understanding through such forums.

Individuals tend to meet on two websites: Reddit (https://www.reddit.com/) and DP Self Help (https://www.dpselfhelp.com/). Reddit is an extremely popular social-networking site, with over 57 million daily users. As of 2024, there are two sub-Reddits dedicated to the discussion of DDD, as well as transient experiences of DPDR. Reddit may hold the largest community of DPDR sufferers. DP Self Help is an online community created with the purpose of helping individuals to manage their symptoms and feel connected. Since its inception in 2004, DP Self Help has 39.4K members and is a thriving community. Similarly to Reddit, DP Self Help has specific forums dedicated to various topics around DDD.

The use of online forum data for research is increasing and spans multiple disciplines and methodological approaches (Proferes et al., 2021). The utilisation of this rich, publicly available data minimises participant bias common in traditional research methods, such as social desirability (Bispo Júnior, 2022). This data describes a substantial cohort explaining what triggers the maintenance of DPDR, presenting a unique opportunity to integrate the global lived experience of DPDR into shaping research inquiries. This grassroots input enhances the cultural sensitivity and ecological validity of investigations in psychological research.

Recent DPDR research successfully used an online forum data framework to investigate the lived experience of symptoms. For example, exploring how individuals with DPDR communicate their experiences through metaphor, revealing language

and imagery that extend beyond formal diagnostic criteria (Dilkes, 2024), and identifying themes of what is discussed on DPDR forums (Fury, 2023).

4.2 Research question

To elucidate the maintaining factors of DPDR, the present exploratory study drew on qualitative data from online DPDR fora to examine one central question: which subjectively perceived experiences are reported by individuals as triggering recurrent episodes of DPDR?

4.3 Methods

4.3.1 Data extraction

The process for acquiring the dataset is outlined in detail in Dilkes (2024). In brief, posts were extracted from the subReddit r/dpdr via the Reddit pushshift.io Application Programming Interface in June 2022, and from dpselfhelp.com via the Scrapy web scraping framework in August 2022. The extracted Reddit data consisted of 205,419 posts made by 27,503 participants, the extracted dpselfhelp.com data consisted of 281,128 posts from the sub-forums “Discussion” (~178 K posts), “The Daily Forum” (~40 K posts) and “Introduce Yourself” (~26 K posts), made by 12,472 participants.

400 posts in total were selected at random based on including one or more of the following words: “trigger”, “triggered”, “triggers”, “induces”, “induced”, “induce”, “set off” and “generate”. 32 posts were discarded as their content alone did not provide sufficient context to the discussion of topics, and/or did not contain any relevant information to the topic being studied. Therefore, 368 posts were included in the thematic analysis.

The term "triggers" to describe stressors is preferred for this study. While the term "stressors" adequately captures events that disrupt physical and psychological harmony, it lacks the broad recognition of the term "triggers", which is more familiar in contemporary society and to internet users. Therefore, when analysing text from natural language sources, the term "trigger" serves as a crucial artefact in modern

research, referring in mental health to stimuli or events that provoke or exacerbate symptoms of a mental health condition or disorder.

4.3.2 Participants

Of these 368 posts, the analysis included 288 participants, some contributing several posts within the forums regarding individuals' subjective DPDR triggers. The number of posts made by participants ranged from 1 to 6. Therefore, data was analysed related to each participant (N = 288) rather than to each post (N = 368) to ensure that all themes were being identified as part of a larger network of individuals endorsing them, rather than one individual posting several times about the same topic, creating the appearance of multiple endorsements.

4.3.3 Qualitative analysis

Inductive thematic analysis followed Braun and Clarke's (2006) framework, including (1) repeated familiarisation with the data through close reading of all posts, (2) systematic generation of initial codes capturing meaningful features of the data relevant to subjective DPDR triggers, (3) collating codes into candidate themes, (4) reviewing themes in relation to both the coded extracts and the full dataset, (5) refining and defining themes to ensure internal coherence and clear distinctions between themes, and (6) producing the final thematic structure. Themes were derived directly from the data to capture participants' lived experiences of their DPDR triggers. Thematic analysis allows themes to emerge directly from text-based data, making it well-suited to the rich insights available in online forum posts.

Coding and theme development were conducted by the primary researcher. To enhance analytical rigour and credibility, emerging codes and themes were discussed with supervisory team members at multiple stages of the analysis. Supervisors provided critical feedback on theme boundaries, coherence, and interpretation, which informed iterative refinement of the coding framework.

The qualitative analysis was conducted by the primary researcher, who has an academic background in psychology and prior research experience in dissociation and trauma-related processes. The researcher also brings lived experience of dissociative symptoms, which enhanced empathetic engagement with the data, but

also required ongoing reflexive attention to potential assumptions or over-identification with participants' accounts. This background informed sensitivity to participants' descriptions of DPDR. Reflexivity was maintained throughout the analytic process through iterative engagement with the data, critical reflection on analytic decisions, and regular discussion of emerging themes with the supervisory team. This process aimed to ensure that themes remained grounded in participants' accounts while acknowledging the interpretative role of the researcher.

4.3.4 Important ethical considerations for internet-mediated research (IMR)

The use of IMR presents significant ethical challenges, particularly concerning the blurred lines between public and private spaces online. The British Psychological Society (BPS) guidelines (2021) provide a framework to address these issues, centred on four core principles.

4.3.4.1 Respect for autonomy, privacy, and dignity

Researchers must carefully distinguish between public and private online spaces, as data in "public" forums may still carry an expectation of privacy. When data status is unclear, researchers must prioritise user privacy, often through anonymisation and a careful assessment of potential harm. Using public data without explicit consent may be acceptable, but only when justified by scientific value and with robust confidentiality safeguards. This study respects the DPDR community's autonomy by responding to their demand for more research. Data from public but perceived as semi-private forums are handled with care: no direct quotes or identifying information are used, and posts are only paraphrased to maintain confidentiality.

4.3.4.2 Scientific integrity

IMR can limit a researcher's ability to gather crucial demographic data, affecting the rigor and generalisability of the research. Additionally, there is a risk of disseminating inaccurate or misleading information, especially when using complex AI or machine learning models (AI was not used, which directly mitigates this risk). Researchers must be transparent about these limitations and ensure their findings are derived from sound methodologies to avoid causing harm through invalid conclusions. This project's scientific value lies in its goal to generate user-informed insights to address

a research gap. The limitations of IMR, such as the lack of demographic data, are openly acknowledged and considered when interpreting results.

4.3.4.3 Social responsibility

Researchers have a duty to avoid disrupting online communities and to be mindful of users' perceptions of privacy. They must also recognise that relying solely on IMR may exclude individuals without internet access, thereby reinforcing existing inequalities in research representation. The use of algorithms to infer personal information must be handled with care to avoid creating unfair or biased outcomes, particularly for marginalised groups (algorithms were not used, which mitigates this risk). This research is socially responsible as it aims to amplify the voices of a marginalised DPDR population without disrupting their online communities. The use of online data is justified by the fact that it is the most accessible platform for capturing real-world narratives from this group.

4.3.4.4 Maximising responsibility and minimising harm

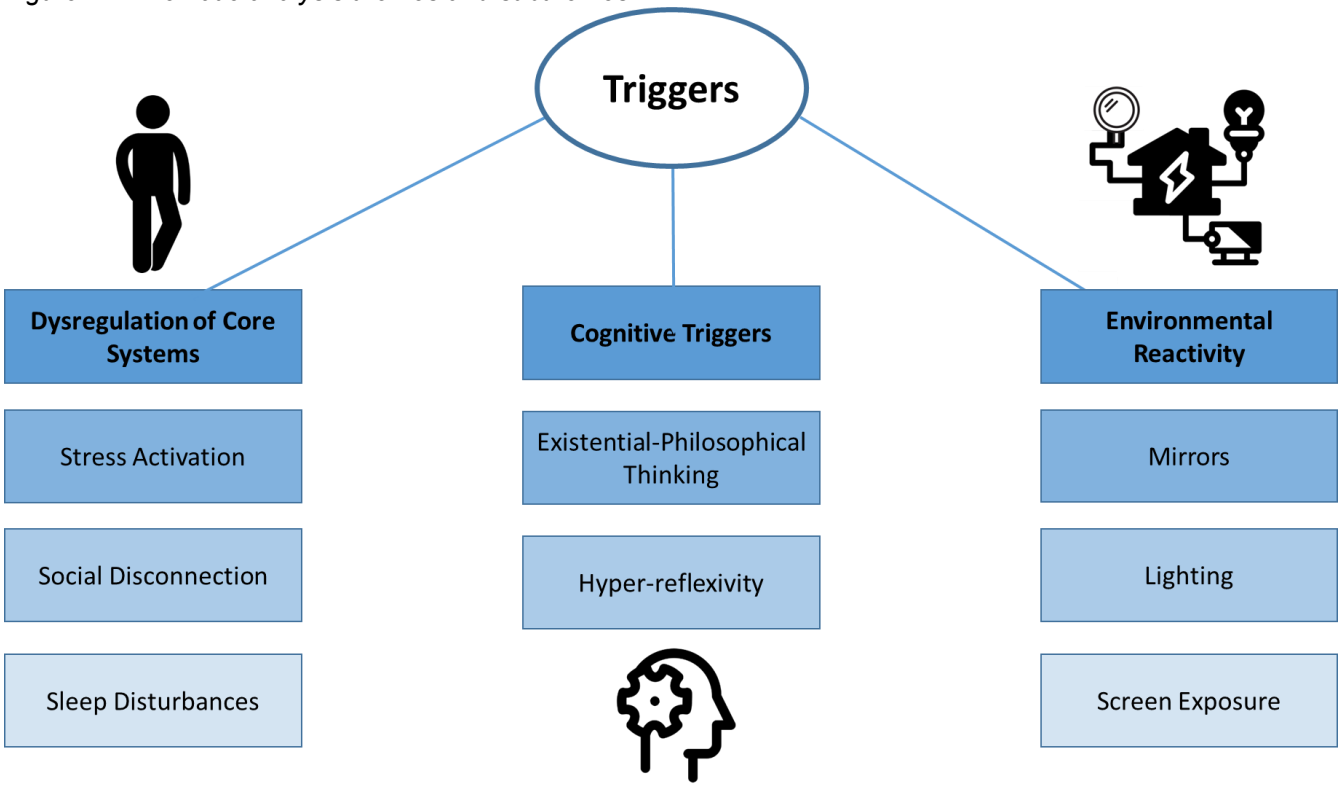
The potential for harm is a key consideration. Research must have clear scientific value, and ethical safeguards (like consent and anonymity) must be proportional to the potential risks and benefits. Researchers must acknowledge that IMR reduces control over participant identity and age, which may increase risk. For sensitive topics, it's crucial to evaluate if IMR is appropriate at all. A key mitigation strategy is to avoid publishing traceable quotes or identifying information by using paraphrasing and anonymisation. In the present study, the potential benefit of informing future interventions is high. Risks, such as not being able to verify participant identity or age, are mitigated by using anonymised, non-sensitive, and paraphrased content.

4.4 Results

The following sections present the identified themes and the subthemes within them. They are explained using excerpts of text from users of the subReddit r/dpdr and the DPSelfHelp forum. To protect the identity of participants and prevent their specific posts from being easily searched and traced online (a risk given the unique 'internet speak' and idiosyncratic phrasing common in forum settings) all direct excerpts presented have been lightly reworded while retaining the identical core message,

content, and emotional tone. An overarching summary of themes and subthemes are presented in figure 11.

Figure 11: Thematic analysis themes and subthemes



4.4.1 Dysregulation of core systems

The theme ‘dysregulation of core systems’ emerged, including the subthemes ‘stress activation, ‘social disconnection’ and ‘disrupted sleep patterns’, demonstrating how an imbalance in homeostasis could trigger the participants’ episodes of DPDR. A regulated stress system, social connection with others, and adequate sleep are all essential to well-being, and can therefore be considered a cluster of core systems for health. The subtheme of ‘stress activation’ was the most frequently endorsed subtheme.

4.4.1.1 Stress activation

Stress activation is classified as the elicitation of an intensified stress response, for example when experiencing distress and anxiety. This stress activation suggests stress system dysfunction. 65 participants (18%) stated that stress activation triggered a recurrent episode of DPDR.

Forum users described stress as a sensitive trigger for their DPDR onset: “even the slightest amount of stress triggers me” (User 7), and symptom severity: "they seem to get worse when im more stressed.” (User 114),

Furthermore, additional indicators of stress activation were noted as subjective triggers: “It usually starts from having a panic attack” (User 254), “My triggers for depersonalization are usually due to fatigue, stress or anxiousness” (User 290).

To recover from a DPDR episode, users noted that it is helpful to gain control over anxiety and stress to reduce symptoms: “When my anxiety is severe, I often start to depersonalise. Learning to manage anxiety and recognise triggers has helped me ground myself and cope better in stressful situations” (User 263). Additionally, similar strategies were used to reduce episode length: “The most helpful advice is to stay calm, as stress and anxiety can make the experience last longer” (User 289).

Additionally, stress activation is noted as a mediator between alternative subjective triggers and experiencing DPDR. For example, within social situations: “I sometimes experience depersonalisation in social situations, where I feel stressed or unsafe” (User 239). Additional examples of the mediating role of stress activation are provided throughout the results section.

4.4.1.2 Social disconnection

17 participants (5%) stated that types of social disconnection, namely social isolation and social vulnerability, could lead to recurrent DPDR.

The feeling of disconnect can emerge when there's minimal human contact or prolonged periods spent alone: "When I spend the whole day alone without interacting with anyone, I sometimes depersonalise - it feels as though my brain forgets my body exists" (User 233).

Additionally, individuals identify a temptation to isolate themselves, but that socialising may help their symptoms in the long run: "Spending all my time indoors has led to more depersonalisation episodes. Although returning to normal life might

initially trigger some symptoms, being outside and around others ultimately feels beneficial for mental health" (User 177).

DPDR episodes manifested during social events or gatherings, particularly in crowded or socially demanding situations: “During episodes at social events, I often feel as though I’m walking on clouds” (User 200), and "Episodes usually happen at parties or gatherings, especially when I’m surrounded by too many people or when I have to speak" (User 188).

Additionally, social anxiety contributed to individuals’ subjective DPDR triggers: "Social anxiety sometimes triggers my depersonalisation" (User 190), and "Fear of going to class and drama can trigger my depersonalisation and derealisation" (User 246).

4.4.1.3 Disrupted sleep patterns

13 participants (4%) stated that disruption in sleep patterns, namely sleeping too little, or achieving low quality sleep, were noted subjective triggers of recurrent episodes of DPDR.

Individuals describe feeling tired or not getting enough sleep led to their DPDR onset: “Lack of sleep is a massive trigger." (User 197), and "Getting enough sleep is important, as being sleepless often triggers my depersonalisation" (User 1).

Posts also suggested a temporal relationship between DPDR and potential disruption in circadian rhythms, for example: "I’ve experienced depersonalisation for over two years, after it was triggered by working night shifts" (User 128), and "My symptoms tend to get worse at night" (User 30).

Interestingly, DR was noted as linked to poor sleep quality, and in turn, individuals felt DP was exacerbated: “Derealisation disrupts my sleep, and as a result my depersonalisation is worse when I wake up” (User 189).

Sleep disturbances appeared as a potential mediator between stress activation and DPDR: “The worst episodes happen when I’m highly stressed and haven’t had

enough sleep” (User 69), but also how stress activation mediated the relationship between sleep disturbance and DPDR: "When I’m tired, it can trigger a panic attack, which then leads to stronger dissociation and feeling as though I might faint" (User 201).

4.4.2 Cognitive triggers

The second theme that emerged was ‘Cognitive Triggers’. This theme encompassed two similar types of cognitions that are unwanted and distressing: ‘Existential-Philosophical thoughts’ and ‘Hyper-reflexivity’. This theme revolves around individuals’ thoughts and cognitions. These subthemes appear linked, often experienced simultaneously. It emerged that certain environmental conditions may be conducive to such thoughts and experiences.

4.4.2.1 Existential-philosophical thoughts

51 participants (14%) stated that existential-philosophical thoughts subjectively triggered a recurrent episode of DPDR. Individuals may experience DPDR when reflecting on deep existential or philosophical ideas, such as life-after-death, determinism, or the nature of consciousness.

Exploring abstract concepts or philosophical theories may also trigger episodes of DPDR, for example: “My depersonalisation was first triggered in 2014, after spending a lot of time thinking about physics, neuroscience, philosophy, and spiritual ideas such as Buddhism and Eckhart Tolle’s The Power of Now” (User 265), and “Whenever my thoughts turn to existential questions about the meaning of life, I find them very triggering, probably because they feel unanswerable” (User 202).

Certain settings may serve as a catalyst for existential-philosophical questioning, leading to DPDR: “Late at night, when everything is quiet and dark, I start to feel completely alone, which then brings on existential thoughts and a sense of crisis" (User 25).

Participants highlight the association with existential-philosophical thoughts and obsessive-type thinking: “My episodes are usually triggered by overthinking, where I

begin questioning my existence - wondering what I truly am beyond this human skin and bones” (User 218), and "It feels linked to existential OCD or anxiety, and since developing derealisation, these thoughts have become very triggering" (User 202), and "My symptoms are triggered more severely when I over-focus on certain things, especially my own existence and consciousness, or when I get caught in negative thought spirals" (User 266).

4.4.2.2 Hyper-reflexivity

16 participants (4%) stated that hyper-reflexivity (ruminative reflecting on DPDR episodes and the self) triggered DPDR. This section highlights the ways in which excessive contemplation and intense focus on DPDR exacerbate symptoms.

Individuals recognise that focusing excessively on their thoughts or feelings can trigger DPDR: "When I focus too much on my feelings or thoughts, I become anxious and can almost trigger another episode" (User 287), and "It only happens when I think too hard about it, and I wonder if in the future I’ll be able to think about it without triggering an episode" (User 272), and “A common feature of DPDR is constant rumination about the symptoms themselves” (User 47).

Additionally, there was an awareness that becoming entrenched in thoughts leads to further disconnection from their body: "When I focus too much, it feels like I exist entirely in my head and not enough in my body or surroundings." (User 266).

Individuals identified that tackling these thoughts can help to avoid episodes: “Training my brain to focus on things other than the symptoms of DPDR has been really helpful” (User 47) and "For some people, an important part of recovery is avoiding content or posts related to DPDR, so they can focus their attention elsewhere" (User 27).

4.4.3 Environmental reactivity

The final theme, "Environmental Reactivity", highlights external factors that trigger DPDR, such as exposure to mirrors, various types of lighting and screen exposure.

Their pervasive presence in daily life makes them difficult to avoid, increasing susceptibility to DPDR episodes and contributing to distress and impairment.

4.4.3.1 Mirrors

29 participants (8%) stated that looking at their reflection in the mirror triggered DPDR.

Users noted that looking in the mirror triggered their symptoms, sometimes to an extremely high degree: “One of my triggers is looking at my reflection in the mirror, which can lead to terrifying depersonalisation episodes” – (User 77).

Mirrors trigger first ever episodes of DPDR, and mirrors became a trigger of recurrent episodes: “The first time I experienced depersonalisation and derealisation was when looking in the bathroom mirror. I didn’t recognise myself at all, and it was utterly terrifying, but I went back to normal. But now I look at myself and I don’t know who the hell I am” (User 157)

Users noted that mirrors can engender existential-philosophical thoughts: “Mirrors trigger the feeling that the reflection isn’t really me, but a fake image pretending to be human, and that I’m the only one who can see it. This happens to me all the time” (User 184).

It was noted that even when observing someone else looking in the mirror, for example in a video game, it was highly triggering: “While playing a video game where the character looked into a mirror, I found the experience extremely unsettling and it triggered my symptoms" – (User 33).

Users noted that avoiding mirrors was a coping technique when providing advice: "This might sound extreme, but mirrors trigger my DPDR so badly that I try to stay away from them altogether” (User 33),

4.4.3.2 Lighting

27 participants (7%) stated that specific lighting could trigger episodes of DPDR.

Lighting was discussed in the general sense, such as: "The lighting in a room and the time of day consistently trigger this state for me" (User 41), and "One of the first things I noticed with DPDR was that light always feels too bright. Overcast days are the worst, and nighttime with streetlights can also make it really bad" (User 258).

Individuals discussed sensitivity to intense lighting, particularly fluorescent or strobe lighting: "Bright or strobe lights can trigger my symptoms. When it happens, everything feels fake and as though it isn't really happening" (User 125), and "I still hate fluorescent lights, they trigger me big time!" (User 12).

Users offered suggestions to manage light sensitivity related to DPDR, for example use of sunglasses: "Try sunglasses, they mellow down all the lighting." (User 144), and "Wearing sunglasses for some reason helps loads" – (User 200). However, other users noted the benefit of exposing themselves to light, despite the DPDR that may initially arise: "Bright lights and natural daylight can trigger my DPDR, but avoiding daylight makes things worse, in the long run, being exposed to it helps." (User 103).

4.4.3.3 Screen exposure

13 participants (4%) stated that screen exposure triggered recurrent episodes of DPDR.

Some examples were relatively general, for example: "Devices and screens tend to make it a load worse" (User 256). However, they also became more specific, citing computer use and playing video games as triggers: "After playing a video game, I dissociated so strongly that it felt like being in an alternate reality, with no emotional attachment and feeling weirded out by my own existence" (User 185), and "I feel like my only major trigger is using the computer" (User 114).

Users identified that a reduction could prevent triggering DPDR, or worsening of symptoms: "What can I do to cope until I get proper help? Should I avoid looking at screens?" (User 157).

4.5 Discussion

This exploratory thematic analysis of online forum data aimed to bridge a gap in DPDR research by examining the factors that trigger recurring DPDR episodes. Three overarching themes: ‘Dysregulation of Core Systems’, ‘Cognitive Triggers’ and ‘Environmental Reactivity’ were identified, and a total of 8 subthemes of the subjective triggers of recurrent episodes of DPDR (refer to figure 11). The discussion delves into these themes, exploring theoretical foundations and linking them to prior findings, to discuss how they answer the research question.

4.5.1 Dysregulation of core systems

Stress activation is a common trigger for DPDR, consistent with evidence of nervous system dysfunction, including atypical stress responses and altered cortical representation (Giesbrecht et al., 2010; Lemche et al., 2008; Owens et al., 2015; Schulz et al., 2015; Sierra et al., 2002; Horn et al., 2020; Millman et al., 2024).

Additionally, stress activation emerged as a central mediator, linking other subjective triggers like sleep deprivation, hyper-reflexivity and screen use, which is predictable given the relationship between stress activation and each of these subjective triggers. For example, the impact of stress system dysfunction on sleep homeostasis (see Martire et al., 2020 for review), maintenance of stress activation due to rumination (Capobianco, Morris & Wells, 2018), and distress in visual cortical and/or circadian systems from screen light exposure (Höhn et al., 2021). Further examples are discussed throughout.

A second subtheme was social disconnection. This included going without socialising for too long, but also being in social environments, with users additionally reporting stress as a subsequent outcome of socialising, which resulted in DPDR.

Ciaunica (2023) identified key social challenges among participants with DPDR, who demonstrated difficulties in communication, including self-expression, alexithymia, and conveying emotions. Many described unconventional social responses, such as avoidance, exaggerated behaviours, or adapting to fit in. Participants also reported difficulties with disconnection and loneliness, social cues and perceiving familiar

people as lifeless. The complex social difficulties encountered by those with DPDR are likely to cause stress, both when there is too little or too much social exposure, which may be the mechanism by which social interaction triggers DPDR. However, empirical research on the underlying mechanisms is needed to explore the role of social disconnection and DPDR.

Further, the thematic analysis identified sleep disturbances as a DPDR trigger. Achieving too little sleep, or low-quality sleep, were identified as triggering or worsening DPDR. Participants also noted that DPDR itself can cause poor quality sleep, in turn causing fatigue, ultimately creating a reciprocal cycle of poor sleep and DPDR. These findings support numerous previous studies that have established a strong link between dissociative phenomena and various sleep-related experiences (e.g., Arora et al., 2020; van der Kloet et al., 2012; Watson, 2001; Poerio et al., 2016) and extend them by suggesting a specific maintenance role of sleep on DPDR. Individuals experiencing DPDR describe it using sleep-related metaphors, likening their experiences to being in a dream-like state (Poerio et al., 2016). The dream-like quality inherent in DPDR has been attributed to the overlap between consciousness during sleep and wakefulness, resulting in a blurred boundary between these states (Arora et al., 2020). Therefore, the feeling of ‘sleepiness’ because of too little sleep or poor sleep quality may create feelings akin to DPDR, potentially leading to the triggering of episodes.

Those who engage in daytime naps or exhibit lower sleep efficiency tend to report significantly higher levels of DPDR compared to the general population (Arora et al., 2020). Notably, research has shown that sleep deprivation and DPDR are not just anecdotally, but causally linked (Menicucci et al., 2022; van Heugten-van der Kloet et al., 2015). Additionally, heightened levels of dissociation have been linked to self-reported sleep disturbances, suggesting a bidirectional relationship (Giesbrecht & Merckelbach, 2004). These results support those of the current findings, indicating a clear link between sleep and recurrent DPDR.

4.5.2 Cognitive triggers

Existential-philosophical thoughts include engaging in, or being exposed to, stimuli of an existential-philosophical nature (e.g., questions that by their nature are unanswerable) was seen to be a common subtheme of participants' subjective DPDR triggers. These existential-philosophical thoughts were noted to be intrusive and scary.

Types of existential turmoil have been noted in individuals with DPDR traits. For example, individuals with DPDR experience "greater existential concerns about others and the world", as well as towards themselves (Ciaunica, 2023). An example illustrating this states: "I feel that the universe has a special meaning and if I discover this meaning I will stop to exist. Sometimes I think people do not exist and even doubt about my existence at all, like if I were in a dream" (Ciaunica, 2023).

A relationship between dissociative symptoms and OCD symptoms may exist, demonstrated by their comorbidity (e.g., Belli et al., 2012; 2014). Within the existential-philosophical thoughts subtheme, users stated that thoughts trigger anxiety and subsequently trigger DPDR. They were able to associate these types of thoughts with obsessive–compulsive existential type, a subtype of OCD characterised by intrusive and repetitive thinking about unanswerable questions (Fukuda, Tamelini & Messas, 2023). Hyper-reflexivity mirrored these processes, where users identified their excessive focus as the cause of their anxiety and trigger for their DPDR.

Soffer-Dudek (2023) proposed several different models of the causal mechanisms that could explain the relationship between dissociative and obsessive-compulsive symptoms, one of which purports that OCD causes inward-directed attention and repetition, which causes dissociative experiences. It is therefore plausible that inward-directed attention of DPDR (for example hyper-reflexivity) could lead to DPDR, leading then to uncontrollable existential thoughts. Examples of such existential concerns in the present study was reported by individuals: "Lately I've been battling constant questions about what makes me who I am. I feel like I should know myself - and in one sense I do - but at the same time I feel as though I don't" (User 202), and "Conversations about topics like free will, the soul, or life after death

can trigger my depersonalisation. When it happens, I feel detached from myself, as though I’m operating my body like machinery from the back of my head” (User 15).

The second subtheme identified was experiencing hyper-reflexivity, which refers to an excessive or heightened awareness of one's own thoughts and sensations in relation to DPDR. Individuals experiencing hyper-reflexivity may become overly focused on their own thinking patterns and experiences of self, leading to a heightened self-consciousness and introspection. The concept of hyper-reflexivity (an intensified, self-focused awareness of self and self-processes) is a key characteristic in certain mental health disorders, notably including psychosis and schizophrenia (Feyaerts, Nelson & Sass, 2025). Importantly, there is a growing consensus that this phenomenon is also a common and relevant feature within DPDR (Ciaunica, 2021; Ciaunica, 2022). Beyond its role as a symptom, hyper-reflexivity is theorised to hold causal priority in psychiatric conditions (Álvarez, 2008), suggesting that its presence may not merely be symptomatic but may actively precipitate the onset of DP or DR experiences.

In previous qualitative research, Ciaunica (2021, 2022) noted that individuals frequently report hyper-reflexivity and are conscious of the way that it manifests in their lives. For example, one participant exemplified this well, stating “Sometimes I’m so preoccupied with the way I walk or the way I present myself, I don’t realize what’s happening around me, for example if someone appeared next to me” (Ciaunica, 2021). Individuals may also engage in obsessional self-checking of internal states, for example one individual stating: “how do I feel now?”, “’Who am I?”, suggesting rumination and over-intellectualisation of thoughts (Ciaunica et al., 2022). This was seen in the present study, with hyper-reflexive thoughts triggering symptoms: "Wondering whether the symptoms will ever go away can itself be a trigger" (User 129), "It only happens when I think too hard about it, and I wonder if one day I’ll be able to think about it without triggering an episode" (User 272).

In this study, individuals identified coping strategies related to this hyper-reflexivity around DPDR: “I’ve found it really helpful to train my mind to focus on things other than the symptoms of DPDR” (User 47), " For some people, a big part of recovery is avoiding posts, videos, or art related to DPDR, so they can focus their attention

elsewhere" (User 27), and “The main goal is to shift your mind away from DPDR and the existential thoughts that come with it. Recovery starts when you notice those thoughts becoming less frequent, and over time, everything begins to look and feel normal again” (User 4). Individuals were able to identify the maladaptive nature of hyper-reflexivity and subsequently develop positive strategies to reduce this thinking style and engage more wholly with the world around them.

Various studies have consistently documented experiences of rumination and broader existential questioning among individuals with DPDR (e.g., Hunter et al., 2003; Roth, 1959; Sierra et al., 2012; Torch, 1978; Vannikov-Lugassi et al., 2021). For example, distressing episodes of detachment lead to repetitive, intrusive thoughts questioning both inward and outward aspects of existence, thereby perpetuating self-monitoring (Medford et al., 2005). In this sense, the two subthemes of hyper-reflexivity and existential thinking identified in the thematic analysis interact and reinforce one another.

4.5.3 Environmental reactivity

The first environmental reactivity trigger identified was exposure to mirrors. Users discussed that simply looking in the mirror could trigger episodes of DPDR, and that there was overlap with other subthemes, such as sleep, where mirrors featured in dreams, and existential thoughts (a cognitive trigger), which users said could be triggered by looking in mirrors and in turn triggered DPDR episodes.

Previous research has identified links between DPDR and mirror exposure. Mirror-gazing protocols are well-documented to induce symptoms of DPDR and dissociative identity in otherwise healthy participants (for review see Caputo et al., 2021; Shin et al., 2018), and individuals with DDs tend to avoid confrontation with their own faces in a mirror. When confronted, they experienced more subjective stress and acute dissociation than HC (Schäflein et al., 2018), aligning with the present findings indicating that individuals experiencing DPDR find mirrors triggering, leading to confrontations that elicit DPDR.

The second environmental reactivity subtheme that emerged was exposure to lighting. There appeared to be a particular aversion toward brighter lights, for example those in a hospital setting or at supermarkets, as well as fluorescent and strobe lights. Additionally, individuals were able to identify strategies to prevent the negative effects of lighting on their symptoms.

Previous investigations into the impact of lighting on DPDR episodes is relatively limited. However, Baker et al (2003) identified environmental lighting as contributing to symptom exacerbation among specific participants. Experimental studies have shown causal links through deliberate manipulations of lighting conditions to evoke states of DPDR. For example, Lickel et al (2008) employed various lighting techniques, including lightbulb-staring, exposure to strobe lights, and combinations of strobe light exposure with hyperventilation to trigger episodes of DPDR, contrasting them against established methods like dot-staring and mirror-gazing exercises known to elicit DPDR. They found that while lightbulb-staring failed to induce significant DP, strobe lights in a dark environment led to heightened DR compared to mirror-gazing. The combination of hyperventilation with strobe light exposure resulted in notably greater DPDR compared to dot-staring. This suggests that, as indicated in the present study, specific lighting can induce recurrent DPDR.

The mechanisms by which this induction occurs may be a type of sensory overload to the visual cortical system because of certain types of lighting. For example, fluorescent lighting exacerbates discomfort and anxiety levels significantly more in individuals with anxiety disorders when compared to HC (Korshid et al., 2021). Anxiety is closely related to stress, a physiological trigger of DPDR, and therefore there could be a mediating relationship of stress between lighting environments and DPDR. Visual input containing flicker, high levels of contrast and specific luminance structures is known to cause hyperexcitation of visual cortical regions alongside self-reported visual discomfort, particularly in people with photosensitive migraine, epilepsy and other neurological conditions marked by visual stress (e.g., Wilkins, 2018). It has recently been proposed that cortical hyperexcitation extends beyond the visual system to play a role in prefrontal cortical inhibition of autonomic responses, which is dysfunctional in those with symptoms of DPDR (Joshi et al., 2024).

The third environmental reactivity subtheme was the connection between screen use and the onset of DPDR episodes. For instance, screen exposure, particularly in activities like video gaming, emerged as a common trigger. Users subjectively recognised the potential link between screen use and DPDR, noting that reducing screen time could facilitate recovery. These findings align with prior research indicating a link between screen activities and DPDR. During the Covid-19 pandemic, increased engagement in digital media through online social meetings coincided with heightened DPDR feelings (Ciaunica et al., 2022). Studies also suggest a correlation between internet gaming disorder (characterised by high amounts of screen time) and increased dissociative symptoms (De Pasquale et al., 2018; Kandeğer et al., 2021), suggesting a relationship between the two phenomena. Similarly, scores on the DES were found to positively correlate with hours per week spent on the internet, again suggesting a relationship between screen use and dissociative symptoms (Bernardi & Pallanti, 2009).

While causality remains uncertain, the existing literature aligns with this study's findings and implies that prolonged screen exposure may trigger recurring DPDR. There are various evidenced mechanisms by which screen use could trigger DPDR, such as through stress and anxiety (Vahedi & Saiphoo, 2018) or by light-emitting diode impacting circadian physiology, preventing sleepiness (Cajochen et al., 2011) and preventing adequate sleep, in turn increasing susceptibility to DPDR.

4.5.4 Strengths and limitations

Utilising forum posts as a qualitative research tool presents both strengths and limitations. On the one hand, it offers access to diverse perspectives and experiences, providing valuable insights into naturalistic behaviour within an online context. The existing DPDR literature tends to utilise clinical samples biasing research findings towards individuals who are high severity, such as inpatients, or from higher socio-economic groups, who have been able to afford private mental health care. Clinical samples are not representative of the community at large; thus forum data allows samples of a large community of sufferers who have not yet been able to receive a diagnosis. In this study, steps were taken to mitigate personal details of individuals being identified by removing usernames and utilising only

snippets of posts in texts. Ethical considerations related to privacy and consent must always be carefully navigated. This can be challenging to achieve and special care must be taken when designing such research. Nevertheless, doing so can provide extremely valuable insights into phenomena where formal empirical research is still outstanding. For the question addressed here, “what triggers recurring episodes of DPDR?”, forum data provided a rich tapestry from which an answer could be extracted: recurring episodes of DPDR may be elicited by dysfunctions in core systems, cognitive triggers and environmental reactivity.

Limitations must also be considered. Firstly, the demographic composition of online forum data may introduce sampling bias, limiting the generalisability of findings to broader populations. Statistics show that these types of websites are used predominantly by people within the ages of 18-34 years (58%) and slightly more often by men than women (57%), therefore this inadvertently may result in over representation of young adults and men. Additionally, self-selection bias may skew results, as individuals who participate in fora discussions may have unique personality characteristics or life circumstances, for example, lack of a real-world community to turn to. While online forums provide anonymity, which can elicit honest responses, revealing personal experiences that may not be shared in other environments, the anonymity provided by these forums also raises concerns about the authenticity and validity of the information shared, as users may provide inaccurate or exaggerated accounts of their experiences. Additionally, the interactive nature of forums introduces the risk of network confounding or "contagion." Since users read and respond to one another, thematic endorsement may reflect peer influence and social learning within the online community rather than purely independent, internal experiences of the disorder.

4.5.5 Research implications

These findings reveal a nuanced understanding of DPDR triggers and coping mechanisms, suggesting that while certain triggers can exacerbate episodes, individuals can become aware of them and employ various strategies to mitigate their impact. This implies that DPDR triggers may be manageable through effective coping strategies, and this may help to avoid transient episodes forming into DDD. Furthermore, the research highlights the episodic nature of DPDR, indicating that its

occurrence fluctuates depending on diverse factors including environmental, cognitive, and overall system states. Unlike past literature, this study emphasises the importance of addressing ongoing symptoms and preventing future episodes. By recognising the dynamic interplay between triggers and coping mechanisms, interventions can be tailored to better manage DPDR and enhance overall well-being.

Based on pre-existing therapies and studies of effectiveness, the following could be useful for individuals looking to target specific triggers such as stress reduction and cognitive triggers. Managing stress activation, the most endorsed trigger of DPDR, could include implementing routines aimed to lower stress. One area that has seen success in DPDR individuals is that of mindfulness-based cognitive therapy, which in one clinical case study saw an individual's DDD totally resolved (Mishra et al., 2022), and research utilising a mix of clinical and general population participants found that there was an inverse correlation between DPDR and mindfulness (Michal et al., 2007). Additionally, research has demonstrated effectiveness of brief cognitive-behavioural group intervention of psychoeducation and interoceptive exposure exercises on individuals experiencing DPDR following acute anxiety (Schweden et al., 2020).

Individuals triggered by existential-philosophical thoughts and hyper-reflexivity related to DPDR experiences would also benefit from mindfulness-based therapies. Additionally, psychoeducation by first-line health professionals, such as General Practitioners (GPs) surrounding the theorised function of DPDR would be useful for individuals experiencing worries about their symptoms. Describing the commonality of such experiences and helping individuals to reframe experiences as a temporary discomfort rather than interpret them as catastrophic events would likely reduce symptoms. In line with Hunter et al's (2003) cognitive-behavioural model of DPDR, prevention of rumination on symptoms should prevent development of chronicity. Additionally, development of online resources to be advertised by the National Health Service or popular mental health charities such as Mind would allow access for individuals who lack access to GP services. Additionally, individuals triggered by existential-philosophical thoughts may benefit from elements of exposure and response prevention therapy due to its significant overlap with OCD existential type,

ideally through counselling services. However, online resources could be sufficient on platforms such as Silvercloud, the NHS's digital mental health provider to provide expert-led exercises to reduce symptoms.

Other triggers experienced by individuals, such as screen use and sleep, would likely benefit from accessible 'self-care' techniques such as establishing a consistent schedule, avoiding screens before bed and having dedicated screen-time hours to prevent overuse, and create a calming night-time routine to improve sleep quality. Other triggers related to environmental reactivity would benefit from strategies at making these experiences more comfortable for individuals to mitigate development of avoidance behaviours. For example, wearing sunglasses when lighting is uncomfortable, while also employing techniques to promote autonomic relaxation. Further research on therapeutic advancements for such triggers is a priority.

4.5.6 Conclusion

This study explored the real-world experiences of individuals with recurrent DPDR, revealing a range of internal and external factors perceived to contribute to symptom recurrence. By analysing naturally occurring discussions within an online forum, the research captured nuanced insights into how everyday situations may trigger or intensify DPDR episodes. These findings extend existing knowledge by utilising non-clinical, community-based narratives that may highlight symptom patterns often overlooked in traditional clinical contexts.

Although the sample was drawn from an online forum and is not representative of all individuals with DPDR, the consistency of key themes and the diversity of experiences shared suggest the findings offer meaningful insights. They are likely to be generalisable to others experiencing recurrent DPDR, particularly those who seek mental health support or information online, engage in peer-support communities, or are navigating symptoms outside formal treatment settings. These insights may also inform clinicians and researchers working with individuals in the early stages of DPDR or those not currently engaged in clinical care.

The results underscore the importance of recognising the variability and subjectivity of DPDR triggers and suggest that tailored, person-centred approaches may be critical in both clinical intervention and public education. Furthermore, the study highlights the potential value of online platforms as spaces for both early identification and support, particularly for individuals who may not be actively seeking treatment. Future research should continue to explore how these derived triggers interact with individual vulnerabilities, and how awareness of such triggers might inform preventative strategies or therapeutic interventions aimed at reducing the frequency and impact of DPDR episodes.

Chapter 5: Investigating Dose-Response Patterns and Cognitive Risk Factors in Depersonalisation and Derealisation

Abstract

Background: This study investigated associations between adverse childhood experiences (ACEs) and the severity and symptom dimensions of depersonalisation-derealisation disorder (DDD). Building on prior research that identified diagnostic risks, dose-response relationships for both single and cumulative ACE exposures were examined. Cognitive vulnerabilities, specifically trait rumination and hyper-reflexivity, were assessed as potential mediators and predictors of DDD severity. Additionally, empirically derived maintenance triggers, distress, avoidance behaviours, and coping strategies were evaluated in relation to symptom severity.

Methods: One hundred and twenty-four young adults with clinically significant DPDR completed validated measures of ACEs, DPDR, rumination, hyper-reflexivity, and trigger sensitivity via an online survey. Quantitative analyses, including correlational and regression models, were conducted.

Results: Findings demonstrated significant dose-response relationships between ACEs and DDD symptom severity. Trait rumination mediated the link between parental divorce/separation frequency and DDD severity. Hyper-reflexivity emerged as a robust cognitive predictor of DDD severity, mediating the relationship between rumination and symptoms. DDD severity was meaningfully associated with increased sensitivity to potential triggers and showed more variable relationships with distress and avoidance of triggers.

Conclusion: This study clarifies mechanisms underlying DPDR, highlighting the profound and dose-dependent association with ACEs, and the possible central role of hyper-reflexivity in symptom severity. These findings critically position hyper-reflexivity as a primary cognitive vulnerability, paving the way for mechanism-informed psychological interventions that specifically target and modulate self-focused attention to alleviate the distressing symptoms of DDD.

5.1 Introduction

5.1.1 Background to depersonalisation and derealisation

DP and DR are symptoms of distress that are strikingly under researched despite their prominence in clinical and general populations (Aderibigbe et al, 2001; Ross, Joshi & Currie, 1991). DP and DR are characterised by strong feelings of detachment from one's body and self (DP) and from one's environment (DR), which can be extremely distressing to individuals (Hunter et al., 2017). These symptoms are common during or following acute stress, for example feeling 'time slowing down' or 'in a dream' after a moment of calamity. Individuals suffering from DDD are experiencing those same phenomena, only instead of fleeting states, they are experiencing it for long periods of time, sometimes weeks, months or years, depending on the individual circumstances. DP and DR are usually discussed together; however, DR is simply a feature of DP, alongside, emotional numbing and anomalous subjective recall (Sierra et al., 2005).

Considered the third most common mental health symptom (Simeon et al., 2003), DPDR is experienced transiently and is considered a normal feature of human experience (Hunter, Sierra and David, 2004). Existing on a spectrum of severity, as the symptoms become more intense and regular, they become problematic. This could be likened to the transition from a low mood in response to a sad event, to a consistently low mood indicative of major depression. Like depression, DPDR are 'invisible', meaning that individuals can maintain external appearances (e.g. hold down a job, socialise with friends), yet their internal experience is fraught (e.g. feeling disconnected from the world/their body, inability to put their experiences into words).

5.1.2 Dose-responses of adverse childhood experiences

Pathological dissociation (including DPDR) is widely conceptualised as a response to antecedent traumatic stress or severe psychological adversity, often serving as an indicator of a trauma history (Dalenberg et al., 2012). DPDR holds established links to ACEs (see Chapter 2 for a detailed discussion), which may act as precipitating and predisposing factors to DPDR development. ACEs include maltreatment, abuse, and

unsafe environments non-conducive to healthy development (Boullier & Blair, 2018; see Chapter 2 for a detailed review).

A "dose-response relationship" is a fundamental concept in toxicology, pharmacology, epidemiology, and public health, describing how the level of exposure (the ‘dose’) to a substance, factor, or event relates to the magnitude of the outcome (the ‘response’). Chapter 2 partially tested a dose-response, whereby increasing years of exposure to ACEs related to increased odds (in some cases) of DP, and DR (in less cases), however it could not be observed how ACEs related to increased severity of DPDR. Traditionally in adversity research, a dose-response refers to the number of distinct adversities experienced (Felliti et al., 1998). However, in the present study, a ‘dose-response’ of ACEs is conceptualised in three ways.

5.1.2.1 Frequency of single ACEs

Firstly, dose-response is conceptualised by examining how increasing exposure to a single ACE type over time, such as repeated EA, relates to greater DPDR symptom severity. While prior research often relies on subjective ratings of abuse severity, this can overlook the impact of chronic or repeat adversity. By quantifying cumulative instances of specific ACEs, both the unique association between each ACE type and how symptomatology escalates with frequency can be assessed. Importantly, ACEs rarely occur in isolation, and their strong interrelatedness (Dong et al., 2004) necessitates controlling for co-occurring adversities to isolate the distinct contribution of each ACE to DPDR (which is addressed by subsequently creating a multivariate risk model, described in the methods). This cumulative approach aligns with Herman’s 1992 framework of C-PTSD, which highlights the critical role of prolonged trauma in shaping trauma responses. Thus, even when analysing single ACE types, the focus remains on how increasing exposure intensifies DPDR severity.

Previous research demonstrates the importance of considering the frequency and duration (suggestive of chronicity) of ACEs in DDD. Simeon et al. (2001) examined 49 individuals with DDD and 26 HC, assessing the impact of six types of childhood adversity - separation or loss, PN, EA, PA, IPV, and SA. They calculated total trauma scores by multiplying duration, frequency, and severity for each adversity and compared the impact of total trauma scores over severity scores. Total trauma

scores were significantly higher in the DDD group compared to controls (mean 420.29 vs. 194.62). Comparing total trauma scores to severity alone showed that, while both models were significant, total scores, reflecting the repetition and chronicity of trauma, better predicted DDD diagnosis ($\chi^2 = 27.55$, 90% classification accuracy) than severity alone ($\chi^2 = 25.23$, 76% accuracy). Likewise, total EA scores ($\chi^2 = 17.95$, $p < .01$) outperformed severity alone ($\chi^2 = 15.23$, $p < .05$) in predicting DDD diagnosis. These results underscore the importance of assessing trauma frequency and duration, not just severity, in understanding DDD.

In a study involving a Latino/Hispanic population, ACE scores were found to be strongly and positively correlated with DPDR severity as measured by the Cambridge Depersonalisation Scale (CDS), with a Pearson's correlation coefficient of 0.68 ($p < .001$; Aponte-Soto et al., 2019). Importantly, after controlling for depression, individuals with mild victimisation exhibited a statistically significant increase in CDS scores, showing an average elevation of 29.4 points compared to those with no reported history of abuse or victimisation ($p < .01$). Moreover, participants who experienced frequent victimisation demonstrated even greater symptom severity, with CDS scores increasing by 46.7 points relative to non-exposed individuals ($p < .001$). These findings underscore the critical role that the frequency of ACE exposure plays in exacerbating the severity of DPDR.

5.1.2.2 ACE category count

Secondly, the accumulation of distinct ACE types is examined, which was termed ACE category count. This represents the traditional dose-response approach and reflects the number of different categories of adversity an individual has been exposed to. Each ACE type is counted once, regardless of how often it occurred. For example, if a participant only experienced the death of a family member, they would receive a score of 1. If they experienced SA, PA, parental separation or divorce, death of a family member, severe illness in childhood, and another upheaval, they would receive the maximum score of 6. This measure therefore captures the breadth of adversity, emphasising the diversity of categories experienced.

The importance of this type of cumulative ACE exposure was first demonstrated in the pioneering ACEs study by Felitti et al. (1998), who demonstrated that exposure to a greater number of ACE types was associated with a graded increase in risk for a range of mental health outcomes, with a 4–12 fold increase in depression and substance use among those exposed to four or more ACEs. Additionally, another pioneering study demonstrated that the highest ACE count had the highest link to depression and substance use in those who experienced four or more ACEs (Turner & Lloyd, 1995).

These results have been replicated across several mental health symptoms. For example, psychosis-risk (comprising 59,975 participants, Flinn et al., 2025), demonstrating that those experiencing five or more ACEs had 546% increased odds of developing psychosis symptoms compared to 76% for those with one exposure. Additionally, in BD, three or more ACEs was linked to significantly earlier onset of BD, significantly longer duration of BD, and significantly more depressive and manic/hypomanic episodes (Sala et al., 2014). Additionally, for experiences of delusions and hallucinations, each additional ACE experienced was related to a 1.20 increase incidence rate for hallucinations and 1.19 increased incidence rate for delusions (Muenzenmaier et al., 2015). Interestingly, dissociative symptoms mediated the relationship between ACEs and hallucinations, and results demonstrated that each increased ACE was associated with a 1.03 ($p < .01$) increased incidence rate for dissociative symptoms, and therefore a 23% increased risk of dissociative symptoms for individuals who experienced seven ACEs, suggesting a dose-response relationship between ACEs and dissociative symptoms.

Accumulating evidence indicates a clear dose-response relationship between ACEs and distress-related dissociative symptoms. Higher numbers of ACEs disrupt critical developmental processes, such as attachment formation and emotional regulation, leading to increased vulnerability to pathological dissociation in later life (Quiñones, 2022). Addressing this gap is critical to advancing understanding of DPDR’s aetiology and informing targeted interventions.

5.1.2.3 Total ACE exposure

Thirdly, total ACE exposure was examined by summing the frequency of all individual ACE events reported by each participant, thereby accounting for both the repetition of single adversities and the co-occurrence of multiple types. For instance, an individual experiencing repeated PA would accumulate a higher total score than someone with a single instance, and ongoing stressors such as multiple bereavements or persistent parental substance use would further increase this cumulative burden. This approach offers a more nuanced and detailed measure of cumulative adversity, reflecting overall trauma load. It aligns with C-PTSD frameworks emphasising the impact of chronic, repeated trauma (Herman, 1992) and extends Felitti's foundational dose-response model through incorporation of both the frequency and multiplicity of ACE exposures.

A recent meta-analysis of 14,000 participants identified a dose-response relationship between ACEs and the risk of developing depression, specifically examining the impact of different types and frequencies of ACEs (Tan & Mao, 2023). Additionally, in first-episode psychosis research, for each additional adversity-type reported, the risk of psychosis increased by 2.5 times in patients (Trauelsen et al., 2015), emphasising how the impact of cumulative ACEs, including the co-occurrence of different types of ACEs, impacts psychosis risk to a high degree. In women, the impact may be even more pronounced, as research demonstrated that women who experienced multiple forms of victimisation were 5.7 times more likely to report depression and 4.2 times more likely to report psychosis symptoms (Kennedy et al., 2015). In addition to broad psychopathology, there is evidence of a dose-response between ACEs and specific symptomatology, for example maladaptive metacognitive beliefs (Horváth et al., 2024), such as the tendency to focus attention on thought processes, which is highly relevant to DDD.

5.1.3 Cognitive factors

5.1.3.1 Trait rumination

Trait rumination is a habitual cognitive style characterised by repetitive and passive focus on one's distress, its possible causes, and its consequences (Nolen-

Hoeksema, Wisco, & Lyubomirsky, 2008). A systematic review of 18 studies demonstrated that in individuals exposed to adversity, rumination was associated with worse mental health outcomes, including severe psychiatric symptoms, depression, dysphoria, suicidal ideation, and post-traumatic stress symptoms (Mansueto et al., 2021). Additionally, rumination has been demonstrated as a significant mediator between ACEs and depression (Kim et al., 2017), features of borderline personality disorder (Zielinski, Borders & Giancola, 2015), and lower mental wellbeing (Bırni et al., 2025).

Studies consistently indicate that rumination functions as a key cognitive mechanism linking childhood trauma to a range of mental health outcomes. For example, rumination mediates the relationship between childhood adversity and depressive symptoms, with specific subtypes such as brooding significantly explaining the impact of EA on depressive mood (Raes & Hermans, 2008; Dehghan et al., 2024). This mediating role extends to complex disorders, including schizophrenia, where rumination bridges childhood trauma and depressive symptomatology (Fang et al., 2022). Additionally, rumination partially mediates the effects of childhood trauma on cognitive and emotional regulation strategies such as cognitive defusion, acceptance, and suppression (Erduran Tekin & Sirin, 2023). A meta-analysis encompassing 215 studies further supports these findings, revealing medium to large effect sizes for rumination as a mediator between childhood adversity and psychopathology (Miu et al., 2022). Notably, lower trait rumination appears protective, mediating reduced risk of psychopathology following ACE exposure (Fritz et al., 2018). Together, these findings suggest that rumination is a significant (though not exclusive) pathway through which ACEs influence mental health, underscoring its potential relevance to DPDR.

In terms of the impact of rumination on DPDR, the cognitive-behavioural model of DPDR posits that DDD is linked to the anxiety disorders, including panic disorder (Hunter et al., 2003). Cognitive-behavioural models of anxiety disorders like panic disorder or health anxiety emphasise that it is the ‘catastrophic misinterpretation’ of symptoms that lead to the development of the condition. Rumination plays a key role in the anxiety disorders. For example, a meta-analysis of 179 correlational studies (N = 52,760) and 37 clinical group comparison (clinically diagnosed: N = 2,059, HC: N =

3, 879) studies demonstrated that anxiety symptoms had significant, independent effects on overall rumination and emotion-driven rumination (Olatunji, Naragon-Gainey & Wolitzky-Taylor, 2013). The cognitive-behavioural model of DPDR implicates ruminative thinking as a key aspect of the condition, whereby individuals ruminate on symptoms to attempt to understand what the cause of these symptoms may be (for example, thinking they may have brain damage, or schizophrenia). In addition, Vannikov-Lugassi et al. (2021) demonstrated that rumination and DPDR levels increased and decreased simultaneously in a population of 49 outpatients at a mental health clinic, with rumination in one month predicting DP the following month, suggesting a temporal relationship. While valuable, their study did not examine the specific content of ruminations. They measured general ruminative tendencies regarding to ruminative style, such as depressive ruminations, reflective ruminations or brooding. More recently, a study of 277 individuals from the general population suggested that DPDR were significantly associated with rumination (measured through PC; Quigley et al., 2024).

Overall, the literature provides a strong basis for hypothesising that rumination may mediate the relationship between ACEs and DPDR, given the consistent associations between these constructs. This represents an important research gap. In Chapter 2, a potential mediating role was explored, using a broad construct of perseverative thinking; however, as rumination can take several forms, some of which may be adaptive (for example, positive rumination can influence the course of depression; Li, Starr, & Hershenberg, 2017), it was necessary to employ validated psychometric tools that specifically capture its maladaptive aspects. Furthermore, findings from Chapter 4 suggested that rumination about existential concerns and self-related symptoms was associated with increases in DPDR episodes. While these processes overlap with rumination, the underlying construct is more accurately characterised within hyper-reflexivity.

5.1.3.2 Hyper-reflexivity

In contrast to trait rumination, hyper-reflexivity is best understood as a state that arises in direct response to symptoms. It involves an excessive or heightened awareness of ordinarily tacit processes, including one's own thoughts, sensations, and perceptions. This manifests as intense monitoring and analysis of these

experiences, fostering increased self-consciousness and symptom-focused introspection (Sass & Parnas, 2003), and is highly characteristic of DPDR. Whereas rumination tends to focus on unproductive thoughts, hyper-reflexivity involves a heightened, sometimes alienating, focus on one's own thoughts, sensations, or presence "in the moment", for example, becoming vividly aware of one's movements, facial expressions, or the act of existing itself. This heightened awareness often feels automatic and can create a sense of detachment or perceptual distortion.

Findings from Chapter 4 suggested that rumination about existential concerns and self-related symptoms was associated with increases in DPDR episodes. While these repetitive cognitive processes overlap with general rumination, the underlying construct is more accurately characterised as hyper-reflexivity because the self-focus is uniquely heightened, alienating, and automatically intrusive in DPDR.

Hyper-reflexivity is best conceptualised as a state-based phenomenon (a direct and intrusive consequence of DPDR) whereas rumination is typically understood as a broader, trait-based phenomenon characterised by voluntary, goal-directed negative thinking. Thus, temporally, rumination is better positioned as a mediator of the ACE–DPDR relationship as it captures the general, negative cognitive style; hyper-reflexivity is more appropriately understood as a downstream consequence or the immediate phenomenological manifestation of the DPDR state.

This downstream effect, however, may create a feedback loop: The earliest depictions of DPDR, like those by Roth (1959), identified this self-scrutiny as obsessive thinking towards the DDD symptoms themselves. While prominently linked to conditions such as psychosis and schizophrenia (Sass & Feyaerts, 2024), evidence highlights its common presence in DPDR (Ciaunica, 2021; Ciaunica, 2022). Furthermore, hyper-reflexivity is argued to hold potential causal priority for mental health conditions (Pérez-Álvarez, 2008), suggesting that once triggered, it can function not only as a characteristic symptom but also as a contributing, self-perpetuating factor in the disorder's maintenance.

This causal priority is commonly observed within DPDR, which begins as a transient symptom, but catastrophic misinterpretations of symptoms (e.g., as indicative of

severe mental or physical illness) and subsequent intensified self-monitoring lead to development of further symptoms and chronicity (Hunter et al., 2023). These misappraisals can lead to distressing cognitions such as “There is something going wrong with my brain” or “I’m losing control” (Hunter, Salkovskis and David, 2014). This is supported by research demonstrating that distressing experiences of detachment lead to recurring thoughts of inward and outward existential questioning about the nature of reality and the self that become repetitive and intrusive, leading to the maintenance of self-monitoring (Medford et al, 2005). The work of Hunter, Salkovskis and David (2014) is particularly relevant: patients with DDD made fewer normalising attributions and more catastrophic appraisals of symptoms than controls, and symptom severity decreased when attention was diverted away from these appraisals. This provides direct experimental evidence that hyper-reflexivity plays a central role in the persistence of DDD. Therefore, it could be argued that hyper-reflexivity, rather than rumination, is the process through which DDD increases in severity. Rumination may be a predisposed precursor to hyper-reflexivity, through which rumination on DDD becomes hyper-reflexive.

Qualitative investigations, notably by Ciaunica (2021, 2022), have revealed that individuals with DPDR frequently report hyper-reflexivity and are explicitly conscious of its impact. One participant aptly conveyed this, noting: "Sometimes I’m so preoccupied with the way I walk or the way I present myself, I don’t realise what’s happening around me, for example if someone appeared next to me" (Ciaunica, 2021). Additionally, research by Černis et al. (2020) offers valuable insights into factors that initiated, maintained, and resolved dissociative episodes, which lend support to the hypothesis of hyper-reflexivity as a significant factor. Through interviews with 12 individuals who had significant past and present dissociative experiences, the authors identified four key precipitants, one of which was dwelling on thoughts and feelings related to their dissociative experiences. This finding is particularly suggestive of hyper-reflexivity, where an excessive focus on internal mental processes contributes to the persistence of symptoms.

Overall, the literature demonstrates that hyper-reflexivity is a cognitive response to DPDR that contributes to symptom exacerbation and maintenance, thereby promoting chronicity. However, a significant gap in the current research is the lack of

exploration into the specific content of hyper-reflexive thoughts and their bidirectional relationship with DPDR. Building on this, it can be hypothesised that ACEs may foster a predisposition toward rumination, which acts as a risk factor for developing DDD as these ruminative thoughts become hyper-reflexive in nature.

5.1.4 Triggers, distress, avoidance and coping

Further, in Chapter 4, maintenance triggers for DPDR were identified through qualitative accounts from individuals with chronic DPDR revealed eight recurring triggers thought to maintain or exacerbate symptoms: stress activation, social disconnection, sleep disturbances, philosophical-existential thinking, hyper-reflexivity, mirror exposure, types of lighting, and screen exposure. These findings provided a foundation for theorising how such triggers might contribute to the persistence of DPDR and allowed us to generate specific hypotheses regarding the subjective experience of these factors.

It was speculated that exposure to these triggers would vary in terms of how likely they are to elicit DPDR, how distressing they are perceived to be, and whether individuals tend to avoid them. In addition, reports of helpful calming strategies that offered further insight into potential symptom relief were identified, leading us to explore perceived effectiveness across a range of commonly endorsed techniques.

5.2 Aims

The present study investigates associations between ACEs and DPDR symptom severity and whether trait rumination mediates the relationships. Additionally, given the literature on hyper-reflexivity, the current study wanted to assess its role within DPDR, though it is acknowledged as likely downstream sequelae of DPDR, and therefore temporally it would be inappropriate to include it as a mediator between ACEs and DPDR. Additionally, this study assessed the role of empirically identified triggers, distress, avoidance behaviours, and coping strategies in relation to DPDR severity. This approach seeks to clarify pathways influencing the maintenance and heterogeneity of DPDR.

1. To identify whether a dose-response relationship exists between single ACE frequencies and DPDR symptom severity.
2. To identify whether a dose-response relationship exists between cumulative ACEs and DPDR symptom severity.
3. To identify whether ACEs (single ACE frequencies and cumulative ACEs) positively correlate to DPDR subscale severity.
4. To investigate whether trait rumination mediates the relationship between ACEs and DPDR symptom severity.
5. To explore the relationship between hyper-reflexivity and DPDR, and to determine if hyper-reflexivity acts as a more specific and influential cognitive predictor of DPDR than trait rumination.
6. To assess whether DPDR severity correlates with triggers identified through lived experience analysis of forum posts regarding DPDR likelihood, DPDR-related distress and avoidance of triggers.
7. To assess whether DPDR severity correlates with perceived effectiveness of coping strategies.

5.3 Methods

5.3.1 Study design

This chapter addressed key gaps in the thesis by quantitatively examining how ACEs relate to DPDR severity, extending beyond the odds of experiencing symptoms. It also investigates specific cognitive vulnerabilities: trait rumination, and, notably hyper-reflexivity, a prominent maintenance factor identified qualitatively in Chapter 4. Additionally, the study validates empirically derived triggers, enhancing understanding of factors perceived to sustain DPDR. Collectively, these analyses aim to clarify mechanisms underlying DPDR's chronic course and inform targeted interventions.

5.3.2 Participants

A total of 250 young adults aged 18–30 were recruited via opportunity sampling. However, 126 responses were excluded due to incomplete data or indications that the survey had not been completed appropriately (e.g., patterned responding or

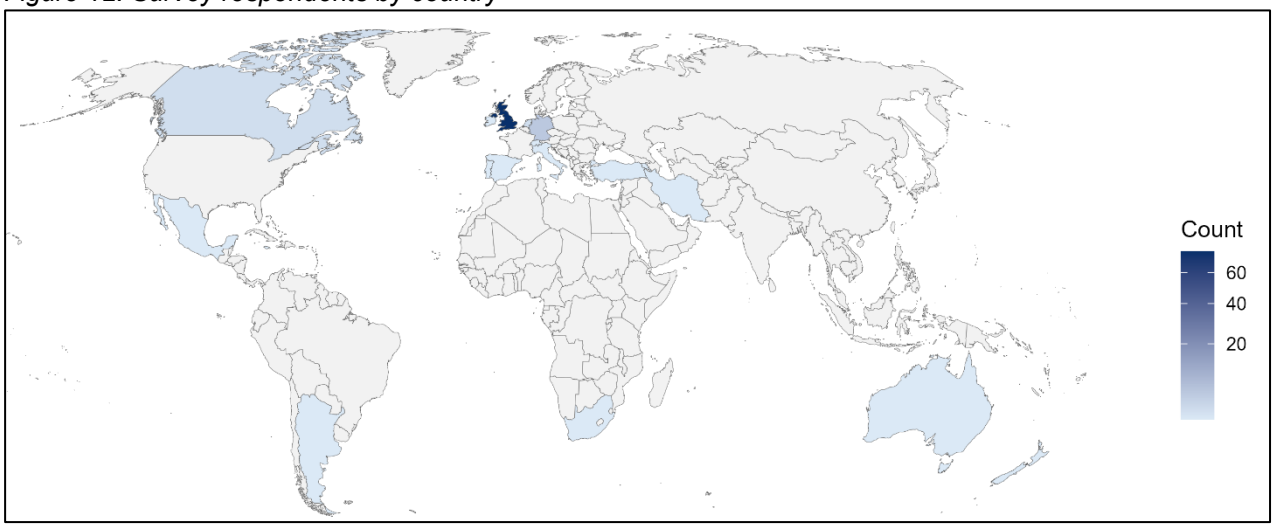
implausible response times), recent diagnosis of a neurological or medical cognition (e.g. epilepsy, traumatic brain injury) and recreational drug use (excluding moderate and below alcohol consumption) in the past month. Therefore, the final sample was 124 participants (see table 10 for demographic information). Participants were primarily recruited through social media platforms (e.g., Twitter, Instagram) and through the Unreal UK website, the UK's national charity for DPDR. This recruitment strategy aimed to access individuals with lived experience of DPDR, including those outside of formal clinical pathways. Participants were recruited globally (see figure 12), with the highest concentration in the UK.

Table 10: Demographic information of respondents

Demographics	**Count & Percentage**
Sex at birth	
Male	32 (26%)
Female	89 (74%)
Ages	
18-21	36 (30%)
22-25	41 (34%)
26-30	44 (36%)
Ethnicity	
Asian	9 (8%)
Black	4 (3%)
Mixed	7 (6%)
White	97 (80%)
Other ethnic group	4 (3%)
Gender identity	
Female	73 (60%)
Male	34 (28%)
Non-binary	10 (8%)
Transgender	1 (1%)
Different gender identity	3 (3%)
Social position (occupation of main household earner)	
Professional / managerial	77 (64%)
Clerical / intermediate	6 (5%)
Skilled trades / technical	9 (7%)
Semi-routine	9 (7%)
Routine	7 (6%)
Other economic status	7 (6%)
Not applicable / don't know / prefer not to say	6 (5%)
Sexual orientation	
Heterosexual	54 (45%)
Gay	2 (2%)
Lesbian	7 (6%)
Bisexual/Pansexual	39 (32%)

Asexual	10 (8%)
Questioning	9 (7%)

Figure 12: Survey respondents by country



5.3.3 Ethics statement

Ethical approval for this study was granted by the University of Essex Ethics Sub Committee 1 (Application: ETH2223-0190). The study was conducted in accordance with the Declaration of Helsinki. All participants provided informed consent, and participation was voluntary and anonymous.

5.3.4 Measures

5.3.4.1 Depersonalisation-derealisation symptoms

DPDR was assessed using the 29-item Cambridge Depersonalisation Scale (CDS; Sierra & Berrios, 2000), which measures their frequency (0 = never; 1 = rarely; 2 = often; 3 = very often; 4 = all the time) and duration ([on average it lasts]: 1 = few seconds; 2 = few minutes; 3 = few hours; 4 = about a day; 5 = more than a day; 6 = more than a week). Scores are summed and higher scores denote higher symptoms. Participants scoring above 70 were classified as experiencing clinically significant DPDR and were subsequently invited to complete the remainder of the survey. The

CDS items were additionally grouped into four empirically informed subscales (Sierra et al., 2005; see Table 11). See table 12 for internal consistency.

Table 11: Cambridge Depersonalisation Scale subscales

Subscale	CDS items	Max score	Symptom focus	Example statements
Anomalous body experience	3, 5, 8, 11, 15, 20, 23, 24, 27	90	Complaints related to changes in body experience	"Body feels as if it didn't belong to self"
Emotional numbing	7, 9, 10, 18, 25, 28	60	Different aspects of attenuated emotional experiencing	"Flavour of meals no longer gives a feeling of pleasure or distaste"
Anomalous subjective recall	14, 16, 17, 19, 21	50	Complaints involving recall of autobiographical events, and of being unable to evoke visual images	"Feeling detached from personal memories, as if one had not been involved in them"
Alienation from surroundings	1, 2, 5, 13	40	Derealisation, being cut-off from the world	"Feeling unreal or cut-off from the world"

Table 12: Internal consistency of measures

Subscale / Scale	Cronbach's α	95% CI	Interpretation
Cambridge depersonalisation scale	.96	.95 – .97	Excellent
Anomalous body experiences subscale	.92	.89 – .93	Excellent
Emotional numbing subscale	.90	.86 – .92	Excellent
Alienation from Surroundings	.86	.82 – .90	Good
Anomalous subjective recall	.86	.81 – .89	Good
Repetitive thoughts	.92	.90 – .94	Excellent

Intrusive rumination	.95	.94 – .96	Excellent

Cronbach's α values ≥ .90 are generally considered excellent, values between .80–.89 good, and values between .70–.79 acceptable (Nunnally & Bernstein, 1994). All subscales and the total scale demonstrated good to excellent internal consistency in the present sample, indicating robust reliability.

5.3.4.2 Trait rumination

The Repetitive Thoughts subscale of the Ruminative Thought Style Questionnaire (RTSQ; Brinker & Dozois, 2009) measured traditional concepts of rumination (e.g., "I can't stop thinking about some things", Tanner et al., 2013). Participants rated each statement on a 7-point Likert scale, indicating the extent to which the statement reflected them (1 = not at all descriptive of me; 7 = very descriptive of me). See table 12 for internal consistency.

5.3.4.3 Hyper-reflexivity

To assess hyper-reflexivity (e.g., ruminative thought specifically related to DPDR episodes), participants completed the Event-Related Rumination Inventory (ERRI; Cann et al., 2011). The ERRI comprises two subscales: intrusive rumination (e.g., involuntary re-experiencing) and deliberate rumination (e.g., purposeful contemplation), however only the intrusive rumination subscale was used in the present study, used to measure hyper-reflexivity in relation to DPDR experiences. The deliberate rumination subscale was not used. Participants were instructed to reflect on their most recent episode of DP or DR: "After an experience like the one you reported, people sometimes, but not always, find themselves having thoughts about their experience even though they don't try to think about it. Indicate for the following items how often, if at all, you had the experiences described during the weeks immediately after the event". Participants then used a 4-point Likert scale ranging from: 0 = not at all, 1 = rarely, 2 = sometimes, and 3 = often. They answered questions such as "Reminders of the event brought back thoughts about my experience." See table 12 for internal consistency.

5.3.4.4 Adverse childhood experiences

Childhood adversity was assessed using the Childhood Traumatic Events Scale (CTES; Pennebaker & Susman, 1988). This self-report instrument evaluates exposure to six categories of traumatic experiences occurring before the age of 17: (1) death of someone close, (2) significant conflict or separation between parents, (3)

sexual trauma, (4) experiences of physical violence, (5) severe illness or injury, and (6) other major disruptive events. For each category, participants indicated the number of occasions (one, two, three, four, or five+) each ACE occured. Following prior research, scores were summed to create a cumulative index of childhood adversity. As the CTES assesses distinct categories of events, rather than a single latent construct, internal consistency indices such as Cronbach's α are not appropriate and therefore were not calculated.

5.3.4.5 Health information

Individuals were asked to whether they had ever been diagnosed with a mental health condition, and if so, what they were. Individuals were subsequently coded as having diagnoses other than a dissociative disorder (1), or no mental health condition (0). DDs other than DDD were not excluded, as this would over control for the high level of DPDR common in other dissociative conditions. Following this, individuals were asked if they took any regular medications for mental health conditions, had ever been diagnosed with temporal lobe epilepsy and/or migraine without aura (exclusionary criteria) or a substance use disorder (exclusionary criteria). See full questions in Appendix 5.

5.3.4.6 Trigger sensitivity

Trigger scenarios were assessed using a bespoke measure developed for the current study. Scenarios were generated from the Chapter 4 thematic analysis and represent commonly reported potential triggers of DPDR recurrence. Individuals were asked "If you experience derealisation and/or depersonalisation chronically, think about whether these things would make your symptoms worse. We would also like to understand, if you did experience derealisation and/or depersonalisation or a change in severity of symptoms, how distressed you might feel in that scenario, and how likely (if at all) you would be to avoid those types of situations in the future."

Examples of triggers were: "Philosophical or existential thoughts. For example, thinking about the meaning of life, whether humans have free will, where do we go when we die?" or "Your reflection. For example, you look in the mirror as usual and you realise that you are the person staring back at you" (for all measured, see Appendix 5).

Participants rated on a 100-point visual analogue scale: (a) how likely they believed each scenario would trigger their symptoms, (b) the expected distress associated with the experience, and (c) the extent to which they typically avoided the scenario. Participants were also given the opportunity to provide their own triggering scenarios; they were asked to “Please state the trigger and then state, out of 10, the likelihood of experiencing depersonalisation and/or derealisation, the level of distress and the likelihood of future avoidance in brackets. For example: Being on a boat (5/10, 8/10, 4/10)”. As this was a novel, exploratory instrument intended to capture the breadth of lived experience, internal consistency indices (e.g., Cronbach’s α) were not calculated.

5.3.4.7 Calming factors

Participants were also presented with scenarios identified as helpful or calming in managing DPDR (see Chapter 4). Individuals were asked “to think about some of the things that in the past, or you believe would presently, help you to 'come out' of a depersonalisation and/or derealisation state”, and gave them examples such as: “Mindfulness exercises. For example, in the present moment, focussing on things you can see, hear, smell, taste and touch”, or “Exercise. For example, taking a 5–10-minute run” (for all measured, see Appendix 1). As with the trigger scenarios, participants rated the likelihood that each scenario would reduce their DPDR.

5.3.4.8 Lived experience component

As an optional final section, participants were invited to provide a written description of how DP or DR affects them. This open-ended data provided qualitative insight into the subjective impact and maintenance of DPDR: “Is there anything else you would like to add regarding your experiences of derealisation and depersonalisation? For example, are there any particular situations that you feel would bring on an episode of depersonalisation or derealisation only?” See Appendix 5.

5.3.5 Procedure

The questionnaire was set up on Qualtrics (Qualtrics, Provo, UT). Individuals completed the 29-item CDS. Following this, they reported on specific ACEs, the

number of times experienced and the ages experienced. If individuals scored 70 or more on the CDS, thus considered to have clinically significant DPDR (Sierra & Berrios, 2000), were taking to the following section of the study. If not, the study ended there. Where the study continued, participants then completed the RTSQ, followed by the ERRI. Individuals were then taken to the triggers section of the questionnaire and then taken to the calming factors section of the questionnaire. Participants were also given the option to provide their lived experience of DPDR. Finally, at the end of the survey, participants were provided resources should they have become distressed during the questionnaire. For more information, please see Appendix 1. Participants who completed the survey were put in a draw to receive one of five £20 Amazon vouchers.

5.4 Data analysis

All analyses were conducted using RStudio (Version 4.3.3). The following packages were used for data organisation, analysis, and visualisation: base, dplyr (Wickham, 2015), haven (Wickham et al., 2019), and ggplot2 (Wickham, 2016). Correlational analyses were first undertaken to examine associations between DPDR (CDS scores) and cognitive factors: trait rumination (RTSQ total) and hyper-reflexivity (ERRI total). Additionally, correlations were run between single ACE frequencies to observe the level of inter-correlation between them.

To investigate the relationship between ACEs and total CDS scores, multiple linear regression analyses were conducted for all ACE variables. When examining ACEs in relation to CDS subscales, Spearman's rho correlations were run as ACE data was positively skewed.

Each regression followed a two-step hierarchical procedure. Step 1 examined the association between ACEs and DPDR outcomes, controlling for sex and the presence of non-dissociative psychopathology. Step 2 included trait rumination. This approach evaluated the mediating role of rumination in the ACE-DPDR relationship. If no significant relationship was found between the ACE being examined and CDS scores in Step 1, then Step 2 was not conducted. To account for multiple comparisons, a Bonferroni correction was applied by dividing the nominal alpha level

(0.05) by the number of analyses conducted to create a significance threshold. Any p-values over the calculated threshold were considered non-significant.

As discussed in the introduction, one aim of the study was to observe a dose-response in three ways: (1) Single ACE frequencies, including a multivariate risk model*, (2) ACE category count, where for each ACE type, participants were coded as either 1 = ever experienced or 0 = never experienced, and the binary indicators were summed, and (3) total ACE exposure, where the total frequencies across all single ACEs were summed. Dose-responses were interpreted through CDS score increases per unit increase in the ACE variable, which can be observed through the beta coefficients (β) in the regression tables.

*The multivariate risk model includes all ACE types simultaneously, allowing us to account for shared variance between adversities. In contrast, the single ACE models examine each adversity in isolation, capturing its total predictive association with DPDR without statistical competition from other ACEs. This distinction is critical: an ACE may appear non-significant in the combined model not because it lacks relevance, but because its effects overlap with those of other adversities.

ACEs often co-occur and are moderately correlated, meaning one may absorb the predictive variance of another in a multivariate model. By first testing each ACE independently, their overall association with the outcome was confirmed prior to determining their unique contributions in the multivariate model. Since inter-correlations were only moderate, the attenuation of effects wasn't solely due to multicollinearity. This approach provided complementary insights into both overall adversity associations and specific, unique risks.

5.5 Results

The results are split into several sections. Firstly, the relationship between all ACE types and CDS scores are tested through correlations and subsequent multiple regression analyses. Secondly, the association between ACEs and CDS subscales (anomalous body experience, emotional numbing, anomalous subjective recall and

alienation from surroundings) are tested through correlations. Following this, the role of rumination as a mediator between the ACEs-CDS relationship was tested.

Following this, the relationship between rumination and DPDR was investigated, and subsequently whether hyper-reflexivity mediated the relationship through regression analyses. Additionally, whether DPDR predicted hyper-reflexivity was tested through regression analyses.

Finally, how CDS scores correlated to DPDR triggers was investigated, specifically: how likely each trigger is to elicit DPDR, the level of distress associated with each trigger, and the likelihood of avoiding the trigger in the future. Additionally, whether CDS scores negatively correlated to proposed calming factors was investigated.

5.5.1 Exploratory analyses

This section examined the associations between CDS Scores, RTSQ Scores, and ERRI Scores and ACEs. The correlation matrices (see Table 13 and 14) displays the strength and direction of these associations. Correlations were run to identify whether basic relationships existed between measures that were tested via regression models.

Table 13: Correlations between Cambridge Depersonalisation Scale scores, Ruminative Thought Style Questionnaire subscale score and the Event-related Rumination Inventory scores

Scales	1.	2.	3.
1. DPDR (CDS Total)	.		
2. Trait rumination (RTSQ Total)	Rs = .216, **p = .010**	.	
3. Hyper-reflexivity (ERRI Total)	Rs = .389, **p <.001**	Rs = .369, **p < .001**	.

Table 14: Correlations between adverse childhood experiences frequencies

ACEs	**1**	**2**	**3**	**4**	**5**
1. Death of Loved One	.				
2. Abuse/Assault	rs = -.042, p = .651	.			
3. Major Upheaval	rs = .096, p = .295	rs = 0.381, **p < .001**	.		

4. Parental Divorce	rs = .106, p = .247	rs = .389, **p < .001**	rs = .353, **p < .001**	.	
5. Serious Illness/Injury	rs = .097, p = .290	rs = .271, **p < .001**	rs = .272, **p < .001**	rs = .346, **p < .001**	.
6. Sexual abuse	rs = .113, p = 219	rs = .365, **p < .001**	rs = .174, **p < .001**	rs = .345, **p < .001**	rs = .331, **p < .001**

ERRI scores exhibited moderate correlations with CDS scores, while RTSQ scores were more weakly correlated with CDS scores. A significant, positive correlation was observed between trait rumination and hyper-reflexivity, indicating an overlap between these constructs (see table 13). Correlations met the p-value correction for multiple comparisons (p = .05 / 3 = .016).

Additionally, table 14 demonstrates that ACEs were highly correlated with each other, other than the death of a loved one. All other ACEs correlations met the p-value correction for multiple comparisons (p = .05 / 15 = .003).

5.5.2 Frequency of single ACE type events and depersonalisation and derealisation

in this section the relationship between ACEs frequency and DPDR is discussed. The mediating role of rumination on the relationship between single ACE frequency and DPDR are examined in Section 5.5.6 (Mediating Role of Rumination on ACEs and DPDR Severity).

Initial models, adjusted for sex and non-dissociative psychopathology, indicated several single ACE frequencies were significant predictors of DPDR in a dose-response fashion. SA emerged as a robust predictor (see table 15), accounting for 5.7% of the variance. For every unit increase in SA frequency, CDS total scores increased by 4.5%. Serious illness or injury in childhood was also significant (see table 16), accounting for 3.1% of the variance, but the overall model did not reach statistical significance (p = .085). Each additional unit of childhood illness or injury was associated with a 3.5% increase in CDS scores. Parental divorce or separation showed a weaker yet significant relationship with CDS scores (see Table 17), accounting for 1.9% of the variance. Again, the model was not statistically significant

overall (p = .154). Here, each unit increase was associated with a 2.75% increase in CDS scores. Finally, major upheavals during childhood demonstrated the strongest effect (B = 9.34, SE = 2.81, p < .001; see Table 18), accounting for 6.8% of the variance (ΔR^2 = .068, p = .010). Each additional upheaval was associated with a 3.2% increase in CDS scores. Death of a loved one and PA did not independently, significantly predict DPDR severity; see Appendix 4.

Table 15: Hierarchical linear regression examining the association between sexual abuse frequency and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
Sexual abuse frequency	13.25	4.32	**.004**	12.07	4.28	**.006**
Sex at birth	-2.97	12.65	.815	-3.35	12.43	.788
Non-dissociative psychopathology	9.97	11.43	.385	7.33	11.29	.518
Trait rumination	–	–	–	2.29	1.01	**.026**

ΔR^2 = .057 for Step 1 (p = .020), ΔR^2 = .032 for Step 2 (p = .005)

Table 16: Hierarchical linear regression examining the association between serious illness/injury frequency and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
Serious illness/injury frequency	9.93	4.04	**.016**	8.41	4.04	**.040**
Sex at birth	-9.06	12.73	.478	-8.82	12.53	.483
Non-dissociative psychopathology	7.31	11.56	.528	4.95	11.43	.666
Trait rumination	–	–	–	2.27	1.04	**.030**

ΔR^2 = .031 for Step 1 (p = .085), ΔR^2 = .030 for Step 2 (p = .023)

Table 17: Hierarchical linear regression examining the association between parental divorce and/or separation events and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
Parental divorce and/or separation	7.97	3.71	**.034**	7.97	3.71	.083
Sex at birth	-9.86	12.83	.444	-9.43	12.83	.456
Non-dissociative psychopathology	7.58	11.63	.516	5.13	11.63	.657
Trait rumination	–	–	–	2.31	1.13	**.028**

ΔR^2 = .019 for Step 1 (p = .154), ΔR^2 = .031 for Step 2 (p = .039)

Table 18: Hierarchical linear regression examining the association between major upheaval frequency and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
Major upheaval frequency	9.34	2.81	**.001**	8.51	2.79	**.003**
Sex at birth	-13.3	12.58	.292	-12.7	12.38	.304
Non-dissociative psychopathology	7.78	11.33	.494	5.40	11.20	.630
Trait rumination	–	–	–	2.23	1.01	**.029**

Δ R^2 = .068 for Step 1 (p = .010), Δ R^2 = .030 for Step 2 (p = .003).

Next, a multivariate risk model was tested, demonstrating that, when controlling for sex at birth and non-dissociative psychopathology, both frequencies of major upheaval and SA event were significant, independent predictors of increasing DPDR scores (see table 19). The total model explained 11.2% of the variance (p = .006).

Table 19: Hierarchical linear regression examining the association between ACEs while controlling for shared variance and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
Death of a loved one	4.40	4.50	.330	4.77	4.46	.287
Abuse / assault	-1.63	3.72	.663	-0.70	3.72	.852
Major upheaval	7.35	2.96	**.015**	6.82	2.95	**.023**
Parental divorce	1.98	4.14	.634	1.00	4.14	.809
Serious illness / injury	5.46	4.34	.211	4.59	4.33	.292
Sexual abuse	10.56	4.42	**.019**	9.81	4.40	**.028**
Sex at birth	-10.18	12.46	.416	-9.81	12.35	.429
Non-dissociative psychopathology	10.01	11.11	.370	7.89	11.08	.478
Trait rumination	-	-	-	1.79	1.02	.083

Δ R^2 = .112 for Step 1 (p = .006), Δ R^2 = .016 for Step 2 (p = .003)

5.5.3 ACE category count

Next, analysis to observe how simple ACE category count predicted CDS scores was completed. ACE category count ranged from 0 to 6. The mediating role of rumination

on the relationship between ACE category count and DPDR are examined in Section 5.5.6 (Mediating Role of Rumination on ACEs and DPDR Severity).

The baseline model, adjusted for sex and non-dissociative psychopathology, indicated that ACE category count was a significant predictor of overall CDS scores (see table 20), with each additional ACE experienced was associated with a 16.38-point increase, or 6% increase, in CDS scores. This suggests that for individuals experiencing a maximum of 6 ACEs, there is a 34% increase in CDS scores. Model 1 explained 16.1% of the variance ($p < .001$).

Table 20: Hierarchical linear regression examining the association between ACE category count and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
ACE category count	16.38	3.27	**< .001**	15.38	3.27	**< .001**
Sex at birth	-7.56	3.27	.524	-7.53	11.68	.521
Non-dissociative psychopathology	9.84	10.7	.363	7.64	10.68	.476
Trait rumination	–	–	–	1.95	0.96	**.045**

$\Delta R^2 = .161$ for Step 1 ($p < .001$), $\Delta R^2 = .022$ for Step 2 ($p < .001$)

5.5.4 Total ACE exposure

Beyond examining individual and multiple ACEs, it is crucial to understand the combined effect of both the variety and frequency/severity of adversities. Therefore, to capture the most comprehensive 'dose' of childhood adversity, the relationship between total ACE exposure and CDS scores was investigated. This score, ranging from 0 to 21, reflects the overall exposure of different ACE types and frequencies. The mediating role of rumination on the relationship between total ACE exposure and DPDR are examined in Section 5.5.6 (Mediating Role of Rumination on ACEs and DPDR Severity).

The baseline model, adjusted for sex and non-dissociative psychopathology indicated that the total ACE exposure was a significant predictor of DPDR, and for

each occurrence of ACE there was a 4.18-point increase, or 1.5% increase, in DPDR score (see table 21). Therefore, scoring the maximum of 21 ACE exposures would correspond to a 30% increase in CDS scores. Model 1 was statistically significant, explaining 8.9% of the variance.

Table 21: Hierarchical linear regression examining the association between total ACE events and DPDR symptom severity

Predictor	Model 1			Model 2		
	β	SE	p	β	SE	p
Total ACE exposure	4.18	1.12	**< .001**	3.81	1.12	**< .001**
Sex at Birth	-12.57	12.40	.313	-12.10	12.23	.325
Non-dissociative psychopathology	7.18	11.21	.523	5.00	11.10	.653
Trait rumination	–	–	–	2.09	1.00	**.039**

Δ R^2 = .089 for Step 1 (p = .003), Δ R^2 = .025 for Step 2 (p = .001)

Table 22 presents the proportion of variance in DPDR explained by different models assessing ACEs, allowing for an easy comparison. Among individual ACE types, major upheaval (6.8%, p = .010) and SA (5.7%, p = .020) accounted for the most variance in DPDR, while other individual ACEs, such as death of a loved one or PA, explained minimal or non-significant variance.

Table 22: Variance explained by individual and cumulative ACE models in predicting CDS scores

Model Specification	Model Variance Explained	p
Sexual abuse	5.7%	**.020**
Death of a loved one	-0.6%	.510
Major upheaval	6.8%	**.010**
Parental divorce	1.9%	.154
Serious illness / injury	3.1%	.085
Physical abuse	0.4%	.324
Multivariate risk model	11.2%	**.006**
ACE category count	16.1%	**<.001**
Total ACE exposure	8.9%	**.003**

When examining broader models, the ACE category count emerged as the strongest predictor, explaining 16.1% of the variance in DPDR ($p < .001$), suggesting that experiencing a large diversity of ACEs, regardless of the frequency of each, has the greatest explanatory power of CDS scores. The multivariate risk model accounted for 11.2% of the variance ($p = .006$), while the total ACE exposure model explained 8.9% ($p = .003$).

Overall, these results indicate that cumulative measures of adversity (particularly the number of different ACE types experienced) provide a more robust explanation of DPDR symptom variability than any single ACE type alone.

5.5.5 Total Cambridge Depersonalisation Scale subscale scores

In addition to overall CDS scores, analysis was undertaken to identify relationships between ACEs and CDS subscales. Due to the large number of associations, and the fact that this was not the primary goal of the study, regression analyses were not undertaken. Further, as there were a high number of correlations, several significant relationships did not survive following correction for multiple comparisons (.05 / 24 = p = .002). The analysis revealed varied patterns of association (see Table 23).

Following corrections for multiple comparisons, death of a family member or friend, parental divorce and/or separation, physical violence, serious illness or injury frequencies were not significantly associated with any of the CDS subscales (see Table 23).

Table 23: Spearman's rho correlations coefficients and corresponding significance levels for the associations between ACEs and CDS Subscales

	Anomalous body experience	**Emotional numbing**	**Anomalous subjective recall**	**Alienation from surroundings (DR)**
Death of a family and/or friend	rs = .122, p = .184	rs = .040, p = .665	rs = .198, p = .029	rs = -.021, p = .822
Parental divorce and/or separation	rs = .194, p = .033	rs = .165, p = .071	rs = .152, p = .095	rs = .190, p = .037
Sexual abuse	rs = .277, p = .002	rs = .358, **p < .001**	rs = .278, p = .002	rs = .157, p = .084
Physical violence	rs = .163, p = .073	rs = .164, p = .072	rs = .12, p = .192	rs = .075, p = .412

Serious illness or injury	rs = .211, p = .02	rs = .228, p = .012	rs = .231, p = .011	rs = .052, p = .572
Experienced major upheaval	rs = .325, **p < .001**	rs = .190, p = .037	rs = .292, **p = .001**	rs = .243, p = .007
Total ACE exposure	rs = .308, **p < .001**	rs = .221, p = .015	rs = .317, **p < .001**	rs = .195, p = .032
ACE category count	rs = .388, **p < .001**	rs = .331, **p < .001**	rs = .390, **p < .001**	rs = .231, p = .011

*Significant correlations are bolded

SA was significantly associated with emotional numbing only, and major upheavals in childhood was associated with anomalous body experience and anomalous subjective recall only.

For cumulative measures, ACE category count was significantly associated with the anomalous body experience, emotional numbing, and anomalous subjective recall, whereas total ACE exposure was only associated with anomalous subjective recall and anomalous subjective recall.

No ACEs were significantly associated with the alienation from surroundings subscale.

5.5.6 Mediating role of rumination on ACEs and depersonalisation-derealisation severity

To understand the role of rumination in the relationship between ACEs and DPDR, each analysis was conducted twice: an initial model unadjusted for rumination (as described above) and a second model adjusted for rumination. Rumination did mediate the relationship between parental divorce and/or separation (see table 17), however did not mediate the relationship between SA (see table 15), serious illness or injury in childhood (see table 16), or experiencing major upheavals (see table 18).

In the multivariate risk model, where all ACE frequency variables are inserted into the model to control for shared variance, both major upheaval and SA remained

significant predictors when controlling for trait rumination. Additionally, trait rumination was not a significant predictor of DPDR in this model (see Table 19).

In terms of ACE category count (where each ACEs were coded as either 1 = ever experienced or 0 = never experienced, and the binary indicators were summed for each individual), when controlling for trait rumination, ACE category count remained a significant predictor, and trait rumination also emerged as a significant predictor, contributing an additional 2.2% of the variance, but did not attenuate the relationship between ACE category count and DPDR (see Table 20).

In terms of total ACE exposure (where the total frequencies across all single ACEs were summed), when controlling for trait rumination, total ACE exposure remained a significant predictor, and trait rumination also emerged as a significant predictor, contributing an additional 2.5% of the variance, but did not attenuate the relationship between total ACE exposure and DPDR (see Table 21).

5.5.7 How does hyper-reflexivity relate to depersonalisation-derealisation, and vice versa?

To further delineate cognitive mechanisms involved in DPDR, the relationship between trait rumination and DPDR was investigated, and whether the relationship could be accounted for by hyper-reflexivity, which is conceptualised as a downstream, consequence-based phenomenon that represents a more specific and intense form of self-focused rumination in DPDR. Regression models were utilised to compare the variance explained when including trait rumination versus hyper-reflexivity in predicting DPDR (see table 24).

Table 24: Multiple regression analyses examining whether trait rumination and hyper-reflexivity attenuate the association between ACEs and CDS scores, controlling for sex and psychopathology

Predictor	Model 1			Model 2			Model 3		
	β	SE	p	β	SE	p	β	SE	p
Trait Rumination	2.64	1.03	**.012**	-	-	-	1.01	1.13	.373
Hyper-reflexivity	-	-	-	2.63	0.60	**< .001**	2.41	0.65	**< .001**
Sex at Birth	-7.77	12.70	.542	-5.2	11.71	.655	-5.09	11.72	.665

Non-dissociative psychopathology	4.82	11.59	.678	-0.93	10.65	.931	-1.73	10.70	.872

Model 1: $\Delta R^2 = .034$, ($p = .069$), Model 2: $\Delta R^2 = .129$ ($p < .001$), $\Delta R^2 = .128$, ($p < .001$)

The results demonstrated a weak but significant relationship between trait rumination and DPDR severity, after adjusting for sex and non-dissociative psychopathology. For each unit increase in RTSQ scores, there was a 2.64 unit increase in CDS scores, or a 0.91% increase. Possible RTSQ scores range from 4 to 28 points, and therefore, individuals who scored 28 had a 25.5% increase in CDS scores. However, the model explained only 3.4% of the variance and was not statistically significant overall (see Table 24), suggesting that while trait rumination may be associated with increased DPDR severity, it is not a particularly strong or consistent predictor in isolation. This aligns with the previous ACE models, where trait rumination did not appear particularly strong, never reaching $p \leq .01$.

Further, the results demonstrated a significant relationship between hyper-reflexivity and DPDR while adjusting for sex and non-dissociative psychopathology (B = 2.63, SE = 0.60, $p < .001$; see Table 24), explaining 12.9% of the variance in CDS scores. Additionally, 12% of individuals experienced the highest level of hyper-reflexivity (see figure 13). Individuals at the highest level (30) would experience an approximate 79-point (27%) increase (e.g., 30 x B(2.63) = 79 points) in CDS scores.

When including both trait rumination and hyper-reflexivity into a combined model and adjusting for sex at birth and non-dissociative psychopathology, the relationship between trait rumination and CDS scores was fully attenuated, and hyper-reflexivity was a strong independent predictor of DPDR severity (see Table 24). Additionally, the combined model performed worse than the hyper-reflexivity only model, suggesting that hyper-reflexivity accounts for the variance previously attributed to trait rumination. These findings are consistent with the correlations that demonstrated hyper-reflexivity to be more strongly associated with CDS scores than trait rumination (see Table 13).

In addition to hyper-reflexivity predicting DPDR, DPDR also predicted hyper-reflexivity when controlling for sex at birth and non-dissociative psychopathology. Overall, the model was significant, explaining 13.1% of the variance in hyper-reflexivity scores (see Table 25). The model suggests that for every point increase in CDS scores, there is a 0.06-point increase in hyper-reflexivity scores. Individuals who experienced the highest CDS score (290) would have a 17.4-point increase in hyper-reflexivity scores (e.g., 290 x B(0.06) = 17.4 points), equivalent to a 58% increase in hyper-reflexivity scores.

Figure 13: Histogram of hyper-reflexivity scores

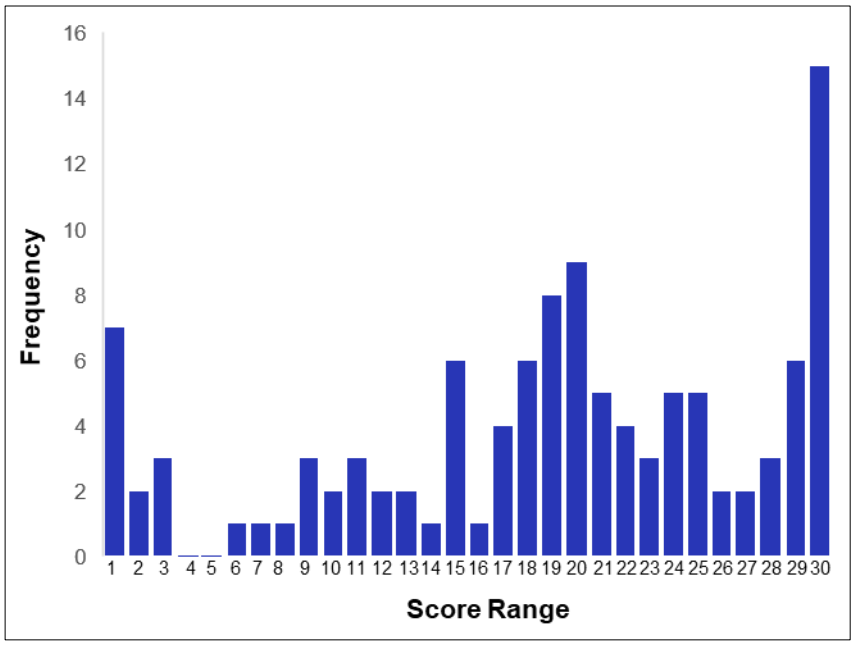


Table 25: Multiple regression examining the association between CDS scores and hyper-reflexivity scores, controlling for sex and psychopathology

Predictor	β	SE	p
CDS score	0.06	0.01	**< .001**
Sex at Birth	0.90	1.73	.603
Non-dissociative psychopathology	-0.64	1.58	.687

ΔR^2 = .131, (p < .001)

5.5.8 Triggers and coping mechanisms

To deepen the understanding of the maintenance factors identified in Chapter 4, the present study assessed their nuanced association on DPDR severity on individuals with DPDR by correlating three key trigger-related outcomes with the CDS scores: (1) increased perceived likelihood of triggering symptoms, (2) increased likelihood to experience distress related to that trigger, (3) increased perceived likelihood of avoiding DPDR-related triggers.

Participants rated each trigger on a 0–100 likelihood scale (where 0 was 'not likely' and 100 was 'highly likely'). Due to non-normal variable distributions, Spearman's rho correlations were computed between these perception scores and participants' CDS total scores. Given the multiple comparisons, a conservative significance level was set at $p \leq .001$.

Higher DPDR symptom severity was consistently and significantly associated with a higher perceived likelihood of being triggered by a wide range of stimuli, including: reflection, multiple mirrors, self-staring, fluorescent lighting, stressful situations, flashing lights, hand focus, rumination on past episodes, screen exposure, insufficient sleep, and excessive sleep (see Table 26).

Higher symptom severity also correlated with an increased likelihood of experiencing distress related to triggers such as reflection, multiple mirrors, self-staring, fluorescent lighting, looking at their hands, past episode rumination, screen exposure, and sleep deprivation (see Table 26).

Symptom severity was significantly correlated with the avoidance of self-staring (see Table 26).

Table 26: Spearman's rho correlation coefficients and corresponding significance levels for the associations between CDS total scores and maintenance triggers.

Maintenance Triggers	Likelihood of Triggering DPDR and CDS Totals	Likelihood of Triggering Distress related to DPDR and CDS Totals	Likelihood of Avoiding These Triggers and CDS Totals
Stressful Situations	rs = .389, **p < .001**	rs = .251, p = .010	rs = .153, p = .120

Philosophical Thoughts	rs = .071, p = .474	rs = .088, p = .373	rs = -.002, p = .988
Reflection	rs = .518, **p < .001**	rs = .474, **p < .001**	rs = .299, p = .002
Multiple Mirrors	rs = .572, **p < .001**	rs = .477, **p < .001**	rs = .284, p = .003
Staring at Self	rs = .447, **p < .001**	rs = .445, **p < .001**	rs = .311, **p = .001**
Fluorescent Lights	rs = .371, **p < .001**	rs = .357, **p < .001**	rs = .180, p = .066
Flashing Lights	rs = .330, **p < .001**	rs = .283, p = .004	rs = .210, p = .032
Hands	rs = .454, **p < .001**	rs = .434, **p < .001**	rs = .238, p = .015
Thinking of past episodes	rs = .344, **p < .001**	rs = .362, **p < .001**	rs = .167, p = .089
Screen	rs = .400, **p < .001**	rs = .346, **p < .001**	rs = .143, p = .145
Lack of Sleep	rs = .347, **p < .001**	rs = .347, **p < .001**	rs = .133, p = .175
Too much sleep	rs = .281, **p < .001**	rs = .284, p = .003	rs = .127, p = .198

Significant correlations are bolded

Additionally, how individuals' perceptions of coping efficacy related to overall DPDR severity was explored. A Spearman's rho correlation examined associations between perceived coping strategy effectiveness and CDS scores, chosen due to non-normal data distribution. Multiple comparisons adjustment set significance at $p < .005$ (0.05/10). No coping strategies were significantly correlated with DPDR severity (see Table 27).

Table 27: Spearman's rho correlation coefficients and corresponding significance levels for the associations between CDS total scores and active coping strategy efficacy

Coping Strategy	CDS Scores
Mindfulness	rs = -.275, p = .005
Exercise	rs = -.261, p = .008
Practicing Acceptance	rs = -.237, p = .015
Meditation	rs = -.207, p = .036
Distraction	rs = -.198, p = .045
Reframing Thoughts	rs = -.157, p = .114
Talking It Out	rs = -.148, p = .137
Grounding Techniques	rs = -.136, p = .170
Journalling	rs = -.059, p = .553
Progress Muscle Relaxation	rs = -.051, p = .609

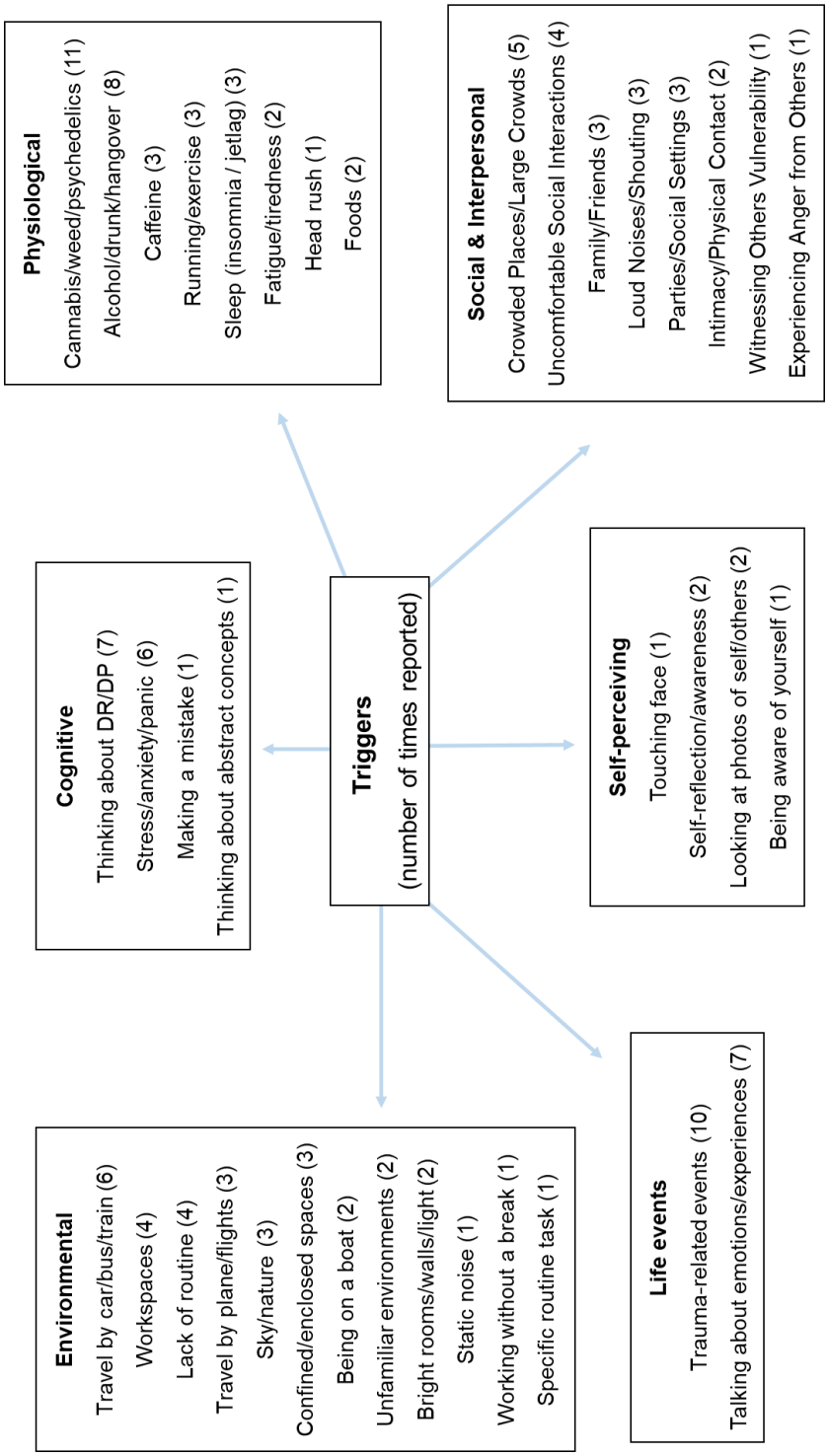
5.5.9 Participant reported triggers

Participants reported additional challenging triggers, some previously identified and others new. These triggers were grouped thematically, with the frequency that they were mentioned (demonstrated in Figure 14). Due to inconsistent ratings of trigger severity, no analysis was conducted on their likelihood to elicit DPDR, associated distress, or avoidance.

5.5.10 Lived experience of depersonalisation and derealisation

Participants were given the optional opportunity to provide additional details about their DPDR experience, detailed further in the Appendix 5. From 55 responses, five themes emerged: (1) *chronicity and persistence of DPDR*, often lasting years and resistant to treatment; (2) *fluctuations in severity and triggers*, including stress and environmental factors; (3) *DPDR as a response to trauma or stress*, though some reported no clear cause; (4) *coping mechanisms* ranging from mindfulness and distraction to reliance on relationships, with mixed success; and (5) *existential fear and intrusive cognition*, which intensified symptoms and led to avoidance (see figure 14).

Figure 14: Triggers reported by participants (number of times reported)



5.6 Discussion

5.6.1 Results summary

The present study examined the relationship between ACEs and DPDR, focusing on dose-response models, a role for cognitive processes, as well as validation of ecologically derived triggers and coping strategies. Several significant findings emerged. SA and major upheavals showed the strongest independent associations with symptom severity over the past 6 months. A cumulative risk model (ACE category count), based on the number of ACEs types experienced, was the most robust predictor of symptom severity, followed by the total cumulative burden (total ACE exposure), which was followed by burden from repeated exposure to a single ACE. Trait rumination only mediated the relationship between parental separation and/or divorce and CDS scores.

Both trait rumination and hyper-reflexivity predicted CDS scores, however when entered into the model together, only hyper-reflexivity over the past two weeks (centred on a recent DPDR episode) independently predicted CDS scores, highlighting its greater explanatory power and potential causal role.

Finally, CDS scores were significantly, positively correlated with likelihood to be triggered by several scenarios, additionally CDS scores were significantly associated with distress-related to the triggering scenario, whereas only self-staring was significantly associated with increased likelihood to trigger avoidance of that scenario. While no coping strategies survived corrections multiple comparisons, mindfulness and exercise are notable in terms of statistical strength and are therefore discussed.

5.6.2 Sexual abuse and depersonalisation-derealisation scores

SA emerged as a particularly strong predictor, as shown through multiple regression analyses (controlling for sex and other psychopathology), with each additional event associated with a 13.25-point (4.5%) increase in DPDR scores. Individuals reporting 5 or more SA events showed a 66.25-point (22.85%) increase in CDS scores compared to those with no such history. This represents the first time in this thesis

that SA has been directly linked to DPDR, a relationship not explored in Study 1 or 2 due to limited parental reporting of SA. These findings confirm both the predictive role of SA and the presence of a dose-response effect.

Our findings support the established link between SA and DPDR within dissociative responses. Prior evidence demonstrates a clearer SA-DPDR link. A study of young adult women found that SA was associated with DPDR (Lassri et al., 2023). Crucially, this association was moderated by self-concept clarity (having a positive and consistent self-perception and identity coherence, a core failure in individuals with DPDR): SA predicted elevated DPDR only among individuals with low self-concept clarity. This suggests SA does not uniformly lead to DPDR; rather, individual differences in self-concept shape vulnerability. Further evidence highlights indirect pathways between SA and DPDR. For instance, in individuals with borderline personality disorder, DR was one of the strongest predictors of SA history (Ogata et al., 1990). SA has also been linked to DPDR in patients with FND (Dearden & Medford, 2017), and somatoform dissociation mediates the relationship between SA and experiencing dissociative seizures (Pick et al., 2017). Moreover, DPDR has been shown to mediate the association between SA and broader psychopathology, including psychosis-like experiences (O'Neill, Maguire & Shevlin, 2021), PTSD symptoms (Kratzer et al., 2018; Vang et al., 2018), and even sexual re-victimisation in adulthood (Krause-Utz et al., 2021).

Our findings extend existing knowledge by showing that in a general population sample, controlling for sex and other mental health conditions, SA significantly predicts DPDR severity with a dose-response effect. While trait rumination did not mediate the SA-DPDR link, it independently contributed to DPDR severity, suggesting an association with DPDR, but not explaining the pathway from SA to DPDR. Collectively, these the current findings and those demonstrated previously reinforce a strong SA-DPDR link.

5.6.3 Parental divorce/separation, childhood illness/injury, major upheavals, and depersonalisation-derealisation scores

Parental divorce/separation showed dose-response relationships with DPDR. Each additional parental divorce/separation predicted a 7.97-point increase in DPDR severity ($p = .034$), equating to a 39.85-point difference between those with five or more events and those with none. This extends prior work on parental divorce/separation, where findings have been mixed, some showing a significant prediction of DPDR (Michal et al., 2009), and others finding no association (Lee et al., 2012). In view of a dose-response interpretation, previous mixed results may stem from not quantifying event frequency (e.g., Lee et al., 2012 used a binary measure). This finding reinforces the results from Chapter 2, that parental divorce significantly predicts increased odds of experiencing DP at ages 17 and 24.

Trait rumination uniquely mediated the modest association ($p = .034$) between parental divorce/separation and DPDR, suggesting a potential mechanism linking parental separation to symptom severity. This novel finding is supported by broader literature showing rumination mediates relationships between household chaos (likely associated with repeated separation/divorce) and outcomes like anxiety and physical symptoms (Deng et al., 2025). These initial mediation effects should be interpreted cautiously due to the modest initial association, but they inform an understanding of how rumination impacts DPDR severity following parental separation. Further research is necessary to clarify these pathways.

Notably, this is the first study to examine childhood illness/injury and major upheavals in relation to DPDR: each illness/injury event predicted a 9.93-point increase ($p = .016$), and each upheaval event a 9.34-point increase ($p < .001$), equating to 49.65- and 46.7-point differences at the highest exposure levels. In both models, the addition of trait rumination did not mediate the relationship, suggesting that rumination does not play a role.

A limitation in the variable 'experiencing some other major upheaval' is acknowledged, which permitted participants to indicate an unlisted ACE. Because the specific nature of these events was not collected, the precise experiences represented by this variable remain unknown. Despite this methodological constraint,

the strong association between this variable and DPDR is significant. It suggests the measure may reflect the impact of unmeasured, non-traditionally captured adversity (such as EA or EN, which are known strong predictors), or ACEs not discussed in the thesis. This limitation is addressed further in the discussion.

Additionally, while childhood illness or injury may appear outside the traditional abuse / neglect / dysfunction framework of ACEs, it can overlap meaningfully. Recurrent illness or injury in childhood may reflect PN, for instance when caregivers fail to provide adequate supervision or timely medical care. Given the lack of association between PA and DPDR in this and earlier chapters, the authors do not speculate on abuse as a driver here but instead highlight the potential roles of neglect or illness itself.

Emerging evidence suggests biological pathways may also be important, with DPDR potentially reflecting a form of “sickness behaviour” like that observed in depression (Maes et al., 2012; see also Chapter 3). Thus, the link between childhood illness/injury and DPDR may arise through multiple pathways, warranting further research into the biological contributions to dissociation.

These findings help elucidate mixed findings on parental separation/divorce by demonstrating a dose-response association with DPDR severity. It also indicates a selective cognitive pathway: trait rumination mediated the separation/divorce-DPDR link (on a modest base effect) but did not mediate associations for childhood illness/injury or major upheavals. Illness/injury showed a dose-response relationship with DPDR, yet mechanisms remain uncertain.

5.6.4 Multivariate risk model and depersonalisation-derealisation

A multivariate risk model with all ACE types explained 11.2% of DPDR symptom variance. SA and major upheavals were the only significant predictors, explaining 6.8% and 5.7% respectively, showing their robust, independent effects even when accounting for other adversities. This contrasts with individual ACE analyses, where SA, major upheavals, parental divorce/separation, and childhood illness/injury were

all significant predictors. These results suggest SA and major upheavals have the most consistent relationship with DPDR within cumulative adversity.

5.6.5 ACE category count & total ACE exposure on depersonalisation and derealisation symptoms

A significant dose-response relationship for both ACE category count and total ACE exposure with DPDR severity was confirmed. The ACE category count (range 0–6) was the stronger predictor: each additional type of ACE predicted a 16.38-point increase in CDS scores, explaining 16.1% of the variance in adjusted models.

Although a relationship was also found for total ACE exposure (range 0–21), the per-event effect was smaller (4.18-point increase; 8.9% variance explained). These findings suggest that adversity diversity is a more potent risk factor for DPDR than sheer frequency of exposure. These findings may however suggest that total ACE exposure could be predictive of ‘more severe’ dissociative responses. For example, Daniels et al. (2024) demonstrated a dose-response relationship between childhood maltreatment and dissociative symptoms, with individuals in the higher severity groups (including most with DID, considered the most severe dissociative disorder) reporting both greater severity and multiplicity of maltreatment.

Even so, both ACE category count and total ACE events predict higher CDS scores, but breadth of ACE types explains more variance (16.1% vs 8.9%) and has a larger per-unit effect. This suggests that exposure to multiple distinct types of adversity is a stronger driver of DPDR severity than repeated instances of a single type.

5.6.6 Non-significant findings

The ACE of “death of a friend or family in childhood” did not significantly predict DPDR, consistent with prior research showing separation or loss was not linked to symptom severity and was more common in non-DDD groups (Simeon et al., 2001). This distinction likely reflects that unlike abuse, neglect, or illness, bereavement does not typically involve interpersonal betrayal or chronic danger. The pattern aligns with broader trauma literature showing interpersonal traumas are more strongly

associated with PTSD than non-interpersonal events (Charuvastra & Cloitre, 2003). For example, a meta-analysis found PTSD rates were higher following interpersonal trauma (25.2%) versus non-interpersonal trauma (9.7%), with stronger effects in girls (Alisic et al., 2014).

The finding that PA did not significantly predict DPDR severity reinforces results from Chapter 2, where it also failed to predict DPDR incidence. Prior research similarly shows that PA does not independently predict DPDR (Simeon et al., 2001). Some have suggested this reflects the severity of PA leading to more severe dissociative responses beyond DPDR (Simeon et al., 2004; Vonderlin et al., 2018). However, this explanation is not fully supported here, as SA (another severe ACE) emerged as one of the strongest predictors of DPDR. This suggests that DPDR can be associated with more severe forms of ACEs. Therefore, PA may simply be a poor predictor of DPDR. However, one possibility is the lack of parental specificity in the current measure: PA could have been perpetrated by non-parental figures (e.g., peers, strangers). Yet, even in Chapter 2, where parental perpetration was explicitly measured prospectively, no relationship was observed.

5.6.7 Correlations between ACEs and Cambridge Depersonalisation Scale subscales

To the authors' knowledge, only one study has previously investigated the association between SA and CDS subscale scores (Dearden & Medford, 2017) in patients with FND, but no other studies have looked at ACEs and CDS subscales. Therefore, this is the first study investigating the associations between an array of ACEs and CDS subscales in a general population sample (without a diagnosis of FND).

This investigation is crucial because, as identified in Chapters 2 and 3, DP and DR may have unique aetiologies. Therefore, this study extends this thesis' previous findings by utilising a more comprehensive measure of DPDR (e.g., the CDS) which allowed for the examination of DPDR subscales: anomalous body experiences, emotional numbing, anomalous subjective recall, and alienation from surroundings.

The analysis revealed varied patterns of association between ACEs and CDS subscales: SA was significantly, positively correlated with emotional numbing and major upheavals in childhood was significantly, positively correlated with anomalous body experience and anomalous subjective recall. No other single ACEs demonstrated correlations with CDS subscale scores.

In terms of cumulative ACE scores, ACE category counts were significantly correlated with anomalous body experience, emotional numbing and anomalous subjective recall, and total ACE events were significantly correlated with anomalous body experience and anomalous subjective recall.

Interestingly, neither single ACEs nor cumulative ACE scores were significantly associated with alienation from surroundings, a core feature of DR. This aligns with findings from Chapter 2, which showed that ACEs were comparatively weak predictors of DR relative to DP. Together, these results reinforce the hypothesis that DP and DR have distinct aetiological pathways, with DP being strongly predicted by ACE exposure, whereas DR appears less directly linked to early adversity.

SA was significantly correlated with emotional numbing. Although emotional numbing is recognised as a central feature of DPDR, its specific association with SA has received little attention outside of clinical groups. Previous work identified elevated emotional numbing in SA survivors with FND (Dearden & Medford, 2017). This study's findings extend this evidence by showing that the SA-emotional numbing association is also present among individuals without FND, highlighting a broader relevance of this pathway. Moreover, the results support and refine Frewen et al. (2015), who identified a "Severe Dissociative" PTSD subgroup characterised by high dissociation and SA, including elevated CDS emotional numbing scores. By showing that repeated SA events, not just subjective severity, predict higher emotional numbing, the potential role of the cumulative impact of chronic trauma is highlighted.

While correlations between SA and anomalous subjective experience and anomalous subjective recall did not survive correction for multiple comparisons, the results did demonstrate that a relationship likely exists. Related evidence suggests that SA disrupts autobiographical memory, particularly when abuse is prolonged, involves

betrayal, or elicits dissociative coping (Wolf & Nochajski, 2022). Large-scale studies confirm this link: SA doubled the prevalence of autobiographical memory loss in women and increased it 1.5-fold in men, with a dose–response effect (Edwards et al., 2001). SA has also been linked to memory flashbacks (Priebe et al., 2013).

By contrast, SA was not associated with DR in the present sample, diverging from prior studies (e.g., Dearden & Medford, 2017; Bradley et al., 2019). The discrepancy may reflect methodological differences: whereas previous studies often used group comparisons, the current study's approach focused on linear regressions. It is possible that group-based methods would have identified an association, but this study's findings suggest that SA does not predict DR in a cumulative framework.

5.6.8 Trait rumination

The literature widely acknowledges rumination as a significant mediator in the relationship between ACEs and the development of various mental health symptoms (Kim et al., 2017; Zielinski, Borders & Giancola, 2015; Bırni et al., 2025; Mansueto et al., 2021; Dehghan Manshadi, Neshat-Doost and Jobson, 2024; Raes & Hermans, 2008; Fang et al., 2023) and cognitive processes (Erduran Tekin & Sirin, 2023). The current study's findings, however, consistently demonstrated that there was very little mediating value of trait rumination on the relationship between ACEs (SA, childhood illness and/or injury, and major upheavals, ACE category count and total ACE exposure) and DPDR, a stark contrast to this pattern. Additionally, while trait rumination sometimes emerged as an independent predictor, its model contribution was a modest percentage of additional variance (ranging from approximately 1.6% to 3.2%). Rumination did mediate the relationship between parental separation and/or divorce and DPDR, however the initial relationship was modest (as discussed above).

The lack of mediation through rumination suggests that it does not serve as a primary cognitive pathway linking ACEs to DPDR. This may be due to the use of a subscale specifically capturing negative ruminative tendencies rather than a full measure that also assesses positive or potentially adaptive rumination traits, which may partly account for the divergence from previous findings. Nevertheless, given that negative

rumination is typically the dimension most strongly implicated in psychopathology, measurement differences alone are unlikely to fully explain the lack of mediation observed here. These findings point toward a potentially different pathway for DPDR, where the association with ACEs on symptom severity may be more direct or mediated by other, more dissociation-specific mechanisms not captured by rumination, for example, other types of dysfunctional coping strategies (e.g. “I’ve been refusing to believe that it has happened”; Lam & Fung, 2024, p. 3).

5.6.9 Hyper-reflexivity

Prior evidence has identified hyper-reflexivity as a core feature in schizophrenia (Sass, 2003; Feyaerts, Nelson & Sass, 2025; Fernández & Bliss, 2016), dissociation (Černis, 2022), and DPDR (Ciaunica, 2021; 2022). However, despite longstanding theoretical emphasis on hyper-reflexivity as an excessive self-awareness disrupting pre-reflective experience in self-disorders (Sass et al., 2011; Englebert et al., 2018; Sass et al., 2018), empirical quantification within DPDR has been lacking. This study makes a novel contribution by providing rare quantitative support for hyper-reflexivity’s direct relevance to the phenomenology and maintenance of DPDR.

In contrast to trait rumination, (which showed a weak but statistically significant relationship to CDS scores, hyper-reflexivity emerged as a more potent and specific factor. To the authors’ knowledge, this is the first quantitative analysis directly examining hyper-reflexivity in relation to DPDR severity, and it demonstrated a robust effect, accounting for 12.9% of the variance in CDS scores (compared to 3.4% by trait rumination). Additionally, hyper-reflexivity was associated with a substantial estimated increase in symptom severity (79 CDS points, where the CDS ranges from 0 to 290, CDS clinical cut-off is ≥70), highlighting hyper-reflexivity as a central cognitive mechanism in the persistence of DPDR. Notably, 12% of individuals in the sample reported the highest levels of hyper-reflexivity, and the mean score for hyper-reflexivity was 19 out of 30, suggesting a relatively high average. Additionally, 18% of individuals scored within 0-10, 37% scored within 11-20, and 45% scored within 21-30 points. This may point towards hyper-reflexivity as a potential hallmark feature of the disorder.

While trait rumination showed a weak association with DPDR severity, this effect was attenuated once hyper-reflexivity was included in the model. Hyper-reflexivity remained a strong and specific predictor, accounting for variance previously attributed to rumination. This suggests that rather than rumination broadly, it is the hyper-reflexive form of self-focused monitoring that plays a central role in maintaining and exacerbating DPDR. Additionally, the further finding that DPDR predicts hyper-reflexivity is pivotal, identifying it not just as a symptom but as a potentially crucial maintenance mechanism that fuels the chronicity of DPDR through a detrimental feedback loop, consistent with the cognitive-behavioural model of DPDR (Hunter et al., 2003).

Given that hyper-reflexivity in this study was measured in individuals who had already met the threshold for clinically significant DPDR and were asked to reflect on the past two weeks, this study's findings suggest that hyper-reflexivity emerges as a psychological consequence of DPDR. This aligns with cognitive-behavioural models emphasising a reciprocal cycle, whereby catastrophic appraisals of early symptoms (e.g., "I don't feel real, something must be wrong with my brain") intensify self-focus, which in turn deepens detachment (Hunter et al., 2003). Prior research links DPDR with rumination, self-consciousness, and perseverative cognition (Vannikov-Lugassi et al., 2021; Fortuna et al., 2024; García et al., 2024; Quigley, Warren, & Townsend, 2024). This study's findings extend this work by suggesting that it is specifically hyper-reflexivity around DPDR, rather than rumination broadly, that represents the most potent perpetuating factor, highlighting the unique role of this intensified self-focus in maintaining the DPDR state.

Mechanisms through which hyper-reflexivity may exacerbate DPDR can be illuminated by parallels with other conditions. In body dysmorphic disorder, excessive self-focused attention on distorted body image maintains symptoms through rumination and safety behaviours (Veale, 2004). In social anxiety, heightened self-focus amplifies the sense of being observed (Canvin, Janecka, & Clarke, 2016). In DPDR, this manifests as intense monitoring of internal states or the experience of detachment itself, paradoxically reinforcing feelings of unreality and emotional numbness. Contemporary predictive processing models extend this account, suggesting that hyper-reflexivity reflects the system's attempt to explain altered

perceptual engagement through maladaptive hypotheses (e.g., “I am an embodied perceiver, but I am not in control of my perception”; Ciaunica et al., 2022). This positions hyper-reflexivity not only because of DPDR but as an active cognitive mechanism perpetuating its persistence and distress.

5.6.10 Perceived triggers, associated distress and avoidance behaviours

This study provides novel and valuable insights into the dynamic interplay between previously identified subjective triggers of DPDR, associated distress, and avoidance behaviours. The findings of the current study indicated that DPDR severity was associated with increased likelihood of being triggered by a range of stimuli, including stressful situations, reflection, multiple mirrors, self-staring, fluorescent lighting, flashing lights, looking at their hands, thinking of past episodes, extended screen time, and dysregulation of sleep. These pervasive associations suggest that as DPDR increases, so too does general sensitivity to both internal and external triggers, likely exacerbating re-triggering and symptom maintenance. This pattern reinforces past empirical and theoretical work on the role of hyper-reflexivity, lighting, mirror exposure, sleep, stress activation, and screen time (Ciaunica, 2021; 2022; Baker et al., 2003; Caputo et al., 2020, Leonard et al., 1999; Arora et al., 2020; Menicucci et al., 2022; van Heugten-van der Kloet et al., 2015; Horn et al., 2020).

DPDR severity was also significantly, positively associated with an increased likelihood of experiencing distress related to these triggers. The triggers most strongly tied to distress were fundamentally related to selfhood and bodily awareness (reflection, multiple mirrors, self-staring, and looking at one's hands). These specific stimuli are highly associated with hyper-reflexivity through excessive self-focused attention (Pérez-Álvarez, 2008). The fact that distress was also triggered by looking at one's hands suggests the issue is not with the mirror, but with exposure to part of the physical self that compels self-scrutiny. This finding aligns directly with the current study’s multiple regression analyses showing that increasing hyper-reflexivity independently predicts increasing CDS scores, confirming that intrusive self-monitoring plays a role in DPDR severity.

Despite the widespread reported likelihood of triggering and associated distress, the measure of avoidance showed limited significance. Only the avoidance of self-staring

was significantly, positively associated with overall DPDR severity, suggesting a behavioural paradox: although the likelihood of being triggered and distressed is high, this does not translate into widespread avoidance. However, the specificity of avoidance to self-staring reinforces the unique threat posed by explicit self-observation (Schäflein et al., 2018). Therefore, given its mutual indication of high trigger likelihood and high distress, mirror exposure and self-staring may represent important targets for clinical practice, adapting principles of exposure therapy successful for extreme mirror aversion in other disorders (e.g., body dysmorphic disorder; Griffen, Naumann & Hildebrandt, 2018).

5.6.11 Perceived efficacy of coping strategies

Our analysis of coping strategies aimed to determine if perceived coping effectiveness was negatively associated with DPDR symptom severity. Following correction for multiple comparisons, none of the assessed coping strategies were significantly negatively correlated with CDS scores.

Nevertheless, strategies like mindfulness and exercise showed promising trends toward a negative association, suggesting that individuals who utilise and perceive these active, adaptive mechanisms as more effective tended to report lower levels of DPDR. These trends align with literature showing mindfulness reduces dissociative tendencies (D'Antoni et al., 2021) and exercise builds resilience in trauma-related conditions (Hefferon, Grealy & Mutrie, 2009). Therefore, despite the non-significant findings, these results underscore the need for future research into targeted interventions enhancing mindfulness and exercise skills for individuals prone to DPDR.

5.6.12 Strengths, limitations and future research

Several strengths characterise the present study. Firstly, the use of a dose-response framework provided important insights into the relationship between adversity and DPDR. By focusing on frequency rather than only severity, the study demonstrated that frequent exposure to ACEs is associated with worse outcomes, aligning with disorders due to chronic trauma, such as D-PTSD or C-PTSD, where DPDR is a hallmark symptom.

Second, the study extended beyond “traditional ACEs” to examine alternative forms of adversity, such as childhood illness and death of a loved one. This helped demonstrate that DPDR may not arise solely from abuse, but rather from broader stress exposures affecting physiological and psychological systems.

Third, this is the first study to quantitatively investigate hyper-reflexivity in relation to DPDR. While previous research has examined catastrophising and self-focused attention (Hunter et al., 2014), the current study makes a novel contribution by linking these processes theoretically to hyper-reflexivity, thereby advancing conceptual clarity in this domain.

Fourth, this study’s recruitment strategy enhanced representativeness by including individuals who met a clinical threshold for DDD without requiring a formal diagnosis. Given that an accurate diagnosis typically takes 7–12 years to obtain, and that access may be limited by socio-economic factors, this approach ensured inclusion of individuals more reflective of the broader DPDR community.

Nevertheless, the study has several limitations. Although the measurement of hyper-reflexivity addressed important cognitive processes, it primarily captured state responses rather than trait-like tendencies. Furthermore, the temporal ordering of measures (hyper-reflexivity over the past two weeks versus DPDR over the past six months) was not optimal for establishing causal inference. While it is not assumed that hyper-reflexivity causes DPDR as it is a state in response to symptoms, a longitudinal approach would have allowed better estimations of cause and effect. A stronger design would resemble that of Vannikov-Lugassi et al. (2021), who employed a longitudinal approach to examine rumination and DPDR over time, but with refinements to better capture DPDR-specific ruminations. Ecological momentary assessment could track state hyper-reflexivity and DPDR within the same temporal window, with each moment’s rumination coded as DPDR-focused vs. non-DPDR, enabling within-person cross-lagged analyses to test directionality. This would clarify whether DPDR-specific rumination is a mechanism linking ACEs to DPDR

Second, although the study investigated major upheavals as an ACE category (one of the strongest predictors of CDS scores) participants were not asked to specify the

nature of these events. The term is broad and could encompass diverse circumstances (e.g., loss of housing, forced relocation, or unsafe environments). Collecting more detailed descriptions would have enabled a deeper understanding of stressors outside of the ACE categories included. Moreover, although prior chapters examined ACEs such as EA, PMI, and PSA, these were not included here. Incorporating such ACEs alongside scale outcomes would have allowed a more direct comparison of their contributions to DPDR severity beyond the binary measures used previously.

Additionally, future studies would benefit from a more comprehensive approach to ACE measurement, allowing participants to specify the nature of major upheavals, but also capturing the severity and duration (e.g., several events in close succession versus chronic exposure) of adversities in addition to the frequency that the ACE was experienced. This would enable the development of a more nuanced, cumulative ACE index akin to that proposed by Simeon et al. (2001). In addition, longitudinal designs that follow individuals across childhood and into adulthood could clarify how diverse adversities, and their cumulative burden, shape trajectories of DPDR symptom severity over time.

Finally, limitations also apply to the coping findings. Relying solely on self-reported perceived effectiveness introduces potential biases, as subjective ratings may not reflect actual strategy use or objective efficacy. The cross-sectional design further prevented causal conclusions: it remains unclear whether effective coping reduces DPDR, whether lower DPDR facilitates better coping, or whether unmeasured third variables influence both. In addition, the study did not quantify the frequency or consistency of coping engagement, precluding differentiation between perceived effectiveness and actual application. Future longitudinal research incorporating objective measures of coping engagement will be essential to clarify these causal pathways.

5.6.13 Conclusion

The current study provides novel insights into how ACEs and cognitive factors (trait rumination and hyper-reflexivity) increase DPDR severity. The findings underscore that both frequency of single ACE types and cumulative ACE types can significantly

predict DPDR in a dose-response fashion. This extends prior work demonstrating that ACEs are strong risk factors for DPDR. ACE category count and total ACE exposure emerged as the strongest predictors, supporting models of complex trauma where multiplicity and chronicity of adversity drive the most severe dissociative outcomes.

Crucially, this research highlights hyper-reflexivity as a key cognitive mechanism implicated in DPDR maintenance. In the combined model, trait rumination was not significantly association with DPDR, and hyper-reflexivity demonstrated a strong dose-response relationship, accounting for a substantial portion of the variance in DPDR severity. Its robust predictive power, coupled with the attenuation of trait rumination's effect, positions hyper-reflexivity as a more proximal and influential cognitive contributor to DPDR. This aligns with qualitative and theoretical conceptualisations that describe hyper-reflexivity as an excessive self-focused monitoring, exacerbating feelings of unreality and detachment. The prevalence of high hyper-reflexivity levels in this sample further suggests its central role in the experience of many individuals with DPDR.

In conclusion, this research underscores the profound association between ACEs and DPDR and critically identifies hyper-reflexivity as a distinct and powerful cognitive predictor of symptom severity. These insights pave the way for more targeted and effective psychological interventions that specifically address and modulate self-focused attention and hyper-reflexive processes, ultimately aiming to alleviate the distressing symptoms of DDD.

Chapter 6: Discussion

6.1 Discussion summary

This thesis embarked on a comprehensive investigation into the complex and often misunderstood phenomena of DPDR. While primarily employing a longitudinal design to uncover distinct aetiological pathways of DP and DR within a large, population-based cohort, this research additionally used qualitative methods. This formed a significant part of this thesis, providing rich, lived-experience insights that complemented and informed the subsequent cross-sectional analyses. These cross-sectional results elucidated the role of hyper-reflexivity and investigated the relationship between SA and DPDR. Additionally, DPDR moved beyond a singular conceptualisation and towards understanding DP and DR as separate experiences with distinct aetiologies.

The preceding chapters presented the detailed methodologies and empirical findings from four interconnected studies. This discussion synthesises these findings, beginning by contrasting the thesis aims against what was found. Subsequently, the discussion delves into the insights gleamed from each study in more detail, focusing on: (1) the differential association between various ACEs and DP and DR, including the significant role of household instability-ACEs and the non-predictive nature of PA and PN in a general population sample; (2) the complex, independent associations between inflammatory markers (IL-6 and CRP) and DP and DR, and their lack of mediation between ACEs and DPDR; (3) the identification of Dysregulation of Core Systems, Environmental Reactivity, Cognitive Triggers and the mediating role of stress activation in DPDR maintenance; and (4) the significant role of SA in DPDR, how a dose-response relationships between ACEs and DPDR exists, the critical contribution of hyper-reflexivity as a powerful cognitive mechanism driving symptom persistence, and validation of the subjective maintenance triggers identified through qualitative analysis.

Finally, these integrated findings are used to propose a refined biopsychosocial model of DP and DR. The discussion concludes by outlining the key implications for future research and clinical practice, emphasising the necessity for distinct

conceptualisations of DP and DR and the development of targeted, multi-faceted interventions.

6.2 Research aims and what was found

This thesis aimed to investigate the role of ACEs in DP and DR onset (while controlling for sex differences) and explore the mediating roles of attachment style, depression, anxiety, and PC.

In Chapter 2, ACEs were identified as strong predictors of DP, with the odds of DP increasing over time predicted by EA, EN, PSA, IPV, parental divorce, as well as cumulative ACEs (abuse-related and neglect and household instability-related). The pattern for DR was more complex: PSA predicted increased odds of DR, whereas PMI and parental divorce predicted decreased odds.

Secondary factors (anxiety, depression, PC) did not mediate the significant ACE-DPDR relationships. However, anxiety was confirmed as a strong, direct predictor of both DP and DR in all models.

Further dose-response analyses conducted in Chapter 5 confirmed that SA, major upheavals, childhood illnesses/injuries, and parental separation/divorce all significantly predicted DPDR severity. Additionally, similarly to chapter 2, it was observed that ACEs were not strongly associated with DR.

Additionally, this thesis aimed to investigate low-grade systemic inflammation (via inflammatory markers) and its potential mediating role in the ACE-DPDR pathway. Inflammatory markers did not mediate the significant ACE-DPDR relationships. But, the analysis did reveal unique biological correlates for the phenomena: IL-6 was a significant predictor of DP but not DR, while CRP was a significant predictor of DR but not DP. Post-hoc analysis suggested that CRP instability may be the driving factor in the prediction of DR.

Further, this thesis sought to identify factors contributing to the maintenance of DPDR through qualitative analysis of forum data, followed by quantitative validation.

Findings from Chapter 4 identified that, following qualitative analysis, a comprehensive range of participant identified stimuli triggered recurrent episodes of DPDR. These included: stress activation, social disconnection and sleep issues (dysregulation of core systems), mirror exposure, specific lighting and screen use (environmental reactivity), and philosophical-existential thinking and hyper-reflexivity (cognitive triggers). Quantitative analysis in Chapter 5 confirmed that DPDR severity was positively correlated to heightened general sensitivity to several of these stimuli (as well as other measured triggers identified through the literature, including looking at hands and too much sleep).

Further, individuals with increased DPDR severity were more likely to experience distress related to self-staring, looking in the mirror, being exposed to multiple mirrors, looking at their hands, fluorescent lighting, thinking of past episodes (hyper-reflexivity), screen exposure and lack of sleep. Increased DPDR severity was significantly associated with avoidance of self-staring.

Finally, this thesis aimed to assess whether the PC of rumination and hyper-reflexivity were independent predictors of DPDR and explore rumination's mediating effect in the ACE-DPDR pathway. Results demonstrated that rumination was a relatively weak predictor of DPDR and generally failed to mediate the ACE-DPDR relationships. In sharp contrast, hyper-reflexivity was confirmed as a much stronger and specific predictor of DPDR severity, fully accounting for the variance previously attributed to rumination. This establishes hyper-reflexivity as the primary, proximal cognitive driver of the disorder. Although, it should be noted that due to the temporal relationship between hyper-reflexivity and DPDR, the mediating role of hyper-reflexivity was not tested.

The further sections go through each study in more detail, highlighting the key findings and implications.

6.3 Chapter 2

Our first study examined the longitudinal association between ACEs and DPDR at ages 12, 17, and 24, using data from the ALSPAC dataset. GLMMs were used to

assess the role of both single ACE types and cumulative categories, which included: abuse (EA, PA and SA) and neglect and household instability (which included: EN, PN, PSA, IPV, PMI, parental separation/divorce, and parental conviction).

The study sought to extend previous research by using a large, non-clinical dataset, incorporating prospective reports of ACEs, and distinguishing between DP and DR to see if different types of ACEs had distinct associations with these symptoms. Additionally, this study aimed to elucidate any attenuating or mediating roles of early life attachment style, anxiety, depression, and PC on the relationship between each ACE-type, DP, and DR.

6.3.1 Disaggregating depersonalisation and derealisation

Historically, DP and DR have frequently been conflated in their study, with an assumption among many researchers that they represent variations of a single dissociative process, sharing common underlying mechanisms. This has not been exactly proposed, but rather there has been a lack of differentiation between DP and DR in existing research, although existing research has demonstrated prevalence distinctions (DP = 19.1% vs. DR = 14.4%; Aderibigbe, Bloch & Walker, 2001; also see section 2.2.2.1 Depersonalisation and derealisation variables).

In this study, upon disaggregating DP and DR, clear differences emerged, reinforcing the need to consider them as distinct constructs. DP was more prevalent in early adolescence, while DR became more prominent in later life, suggesting developmentally distinct pathways. Furthermore, the predictors of DP and DR varied, further supporting the notion that these symptoms may arise through separate aetiological mechanisms.

Our research provides a compelling update, providing robust evidence that DP and DR are indeed distinct experiences with mostly independent aetiological pathways. The findings unequivocally demonstrate that DP is more strongly linked to specific forms of ACEs, as well as the accumulation ACE types.

6.3.2 Replication of established ACE-depersonalisation-derealisation relationships

The association between EN and DP showed a distinct longitudinal pattern, with escalating odds of experiencing DP between ages 17 and 24 (as a reminder, EN was a binary measure, and therefore the OR of 3.90 at age 17 and 9.68 at age 24 represents the maximum effect possible in the sample). This suggests that EN's effects on DP may become more pronounced as individuals navigate complex social and environmental stressors in early adulthood (Ribeiro et al., 2018; Sawang & Newton, 2018; Zhan, 2022; Schweden et al., 2018), potentially linked to relational vulnerabilities and disruptions in the oxytocin system (Müller et al., 2019; Reiner et al., 2016). Existing literature also supports a specific association between EN and DP during this period (Ó Laoide, Egan & Osborn, 2018; Wright, Crawford & Del Castillo, 2009).

Additionally, EA was associated with a sustained high likelihood of experiencing DP across later time points, although the effect was slightly reduced at age 24 (OR = 1.97) in comparison to age 17 (OR = 2.25). However, these differences are relatively small, and suggest a stable chronicity over time, in comparison to EN, where the odds escalated over time. This stable chronicity potentially reflects a deeper biological embedding of adversity (Berens, Jensen & Nelson III, 2017; Kumsta, 2023; Tang, Howe & Houtepen, 2020), whereby stress system dysfunction is considered stable because the embedded changes create a new "set point". Therefore, embedding of EA could lead to long-term stress system dysfunction which is consistently linked to DP (Millman et al., 2024; Giesbrecht et al, 2007; Simeon et al., 2001, 2008; Stanton et al., 2001).

EA and EN were robustly associated with DP, whereas PA and PN were not, suggesting prior research identifying a link between these ACEs and DP may have disproportionately focused on high-severity cases or specific populations, rather than reflecting a generalisable trend. Most past research utilises a clinical sample, which is both high-severity and specific populations. For example, a recent meta-analysis of experimental methods in DPDR research included only individuals with a diagnosis with DDD, rather than general population samples with DPDR (Millman et al., 2024). However, the replication of the EA-EN-DP relationship in a large, general population sample underscores its broad relevance in DPDR. Conversely, EA, EN, PA, or PN

did not predict DR. This suggests that these types of ACEs are specifically associated with DP, rather than DR, which would not have been identified without previously disaggregating DP and DR.

6.3.3 Household instability and depersonalisation and derealisation

This research expands on existing literature by broadening the definition of ACEs to include reflections of household instability. Previous research has emphasised childhood trauma, particularly abuse and neglect, as primary drivers of DPDR (Yang et al., 2023; King et al., 2020; Michal et al., 2007, 2009; Simeon, 2001; Simeon et al., 2003; Ó Laoide, Egan, & Osborn, 2018; Smiatek-Mazgaj et al., 2016; Frau & Corrigan; 2025). While unstable household environments have been discussed in PTSD (for review, see Ye et al., 2023), suicidality (for review, see Pu et al., 2025), and other psychopathology, as well as poor social and functional outcomes (Pitkänen et al., 2025), they are not widely investigated as risk factors for DPDR.

This research gap was addressed by demonstrating that household instability (e.g., PSA, parental divorce, PMI, and IPV) are strong predictors of changing odds of experiencing DP and/or DR, more so than PA or PN. This suggests that environmental unpredictability may be a key driver of DPDR, shifting the focus from solely acute trauma (for example, SA, PA and EA, EN or PN) towards, in addition, chronic stressors such as household instability. Additionally, these findings support the TM, which suggests that DPDR is a defence mechanism against traumatic experiences (Dalenberg et al., 2012).

IPV predicted DP at age 17 but the relationship was no longer significant at age 24. Each additional year of exposure increased the odds of experiencing DP at age 17 by 238%. However, this relationship weakened by age 24, becoming a non-significant 67% increase in odds in relation to baseline. This suggests that DP may be directly linked to ongoing exposure to IPV during adolescence but dissipate once the individual is no longer living in the parental home. Evidence of remission of mental health issues upon cessation of conflict can be seen (El-Sheikh et al., 2019), suggesting that should an individual leave a household, their symptoms may remit. This finding suggests that DP associated with witnessing IPV may be

environmentally contingent, ceasing on leaving the household, rather than enduring across the lifespan.

Parental divorce, ranging from 0-5 years, was an interesting case, where it was linked to a significant decrease in the likelihood of experiencing DR at age 17 (71% reduction in the odds per unit increase), but resolving by age 24, suggesting it might draw individuals toward DP instead. For each year increase, the odds of experiencing DP at age 17 increased the odds per event by 180%, and by 249% by age 24.

6.3.4 Parental substance abuse, depersonalisation and derealisation

A key finding was that PSA was associated with increased odds of experiencing both DP and DR at age 24, but not earlier. PSA was the only ACE that predicted odds of both DP and DR. Each additional year of PSA increased the odds of experiencing DP and DR at age 24 by 60% and 37%, respectively. The identification of a delayed association between PSA, DP, and DR could indicate two explanations: (1) a delayed incubation effect, where early-life exposure to PSA leads to chronic stress that manifests in DP and DR later in adulthood, and (2) a secondary effect through substance use, where individuals exposed to PSA are more likely to use substances themselves, triggering DP through drug experiences, a well-documented phenomenon.

This incubation effect is demonstrated in phenomena related to DPDR. For example, the DSM-5 diagnostic criteria for PTSD specifies a PTSD with delayed expression (American Psychiatric Association, 2013). This delayed onset may result from a network of underlying neurobiological processes (including inflammation) potentially influencing the likelihood to develop prodromal symptoms preceding the onset of a complete presentation of PTSD, which can last for decades (Smid, Lind & Bond, 2022). As a key aspect of PTSD, It is therefore possible that individuals experiencing PSA during childhood may experience a delayed expression. In these cases, a reliance on specific environmental or social experiences may lead to the expression of DPDR, in line with the diathesis-stress model (e.g. a combination of a pre-existing vulnerability [diathesis] and stressful life events [stress] is necessary for a disorder to emerge; Goforth, Pham & Carlson, 2011). Exposure to drugs could present itself as a

risk factor to experience DPDR in several ways (within the context of PSA). Firstly, individuals who experienced PSA may have pre-existing diathesis towards DPDR due to environmental challenges early in life and therefore drugs may be the stress that expresses the DPDR. Additionally, exposure to PSA early in life may lead to a normalisation of drug use, leading to a higher likelihood to adopt alcohol and drug taking behaviours, which could lead to the development of DP and DR, particularly later in life when these types of behaviours are more likely to occur (e.g., in 2024, ~971,000 16-24 year olds had used drugs in the past year; Office for National Statistics, 2024).

The finding that PSA is the sole ACE predicting both increased odds of experiencing both DP and DR, at the same age, is striking. This specificity necessitates further investigation to elucidate the mechanisms by which PSA uniquely contributes to the risk profile for both dissociative symptoms.

6.3.5 Unexpected findings: parental mental illness, parental divorce and derealisation

Particularly novel and counterintuitive findings were that increases in PMI were associated with decreased odds of experiencing DR. While seemingly paradoxical, this may reflect the protective mechanisms that exist in families where a parent has a mental illness. Children in these households may receive additional support from extended family members, such as grandparents, buffering them from adversity (Barnett et al., 2011; Parkes & Sweeting, 2018). Additionally, mentally ill parents may engage in overprotective or hyper vigilant parenting strategies, increasing child safety behaviours that reduce overall stress exposure (van der Ende et al., 2016). If this hypothesis is correct, it would suggest that certain forms of adversity can indirectly foster resilience through external support networks, which has been demonstrated (Abate et al., 2024). This finding invites further investigation into the role of familial compensation in buffering against DPDR.

Parental divorce had divergent effects on DP and DR. Exposure to parental divorce increased DP prevalence, suggesting that disruptions in parental relationships may contribute to dissociative tendencies through attachment mechanisms or stress responses. However, parental divorce decreased DR symptoms - a counterintuitive

finding. This indicates that children living in household with high levels of conflict may experience chronic relational distress (such as feeling ‘caught’ between two parents’; Amato & Afifi, 2006), potentially contributing to DR. The idea that DR may develop as a response to an unrelenting, high-conflict environment, rather than as a direct consequence of parental divorce, introduces a new perspective on how familial stability influences DR. The Divorce-Stress-Adjustment Perspective (Amato, 2000) offers a compelling framework for understanding the divergent trajectories of DP and DR in relation to parental divorce. This model views marital dissolution as a prolonged process, not a single event, leading to chronic and cumulative stressors for children. These stressors, particularly the perceived decline in reliable parental presence and support, strongly resonate with experiences known to foster DP, such as EN. Conversely, the initial decreased odds of DR at age 17 might be explained by the same perspective: children in high-conflict homes prior to divorce are exposed to chronic hostility, which could trigger a DR state as a coping mechanism. In such cases, the actual marital dissolution could paradoxically bring relief from this immediate, acute conflict, thereby alleviating a primary trigger for DR.

6.3.6 The accumulation of ACEs and depersonalisation

There was a significant association between cumulative abuse-related ACEs and DP that remained elevated over time, with persistent DP symptoms at ages 17 and 24, suggesting that both the frequency of specific adversities and the presence of multiple types of adversity contribute to chronic DP. Additionally, neglect and household instability ACEs was not a significant predictor of DP at age 17, but emerged as a significant predictor at age 24, indicating that DP may become apparent later in life due to interactions with the environment, essentially ‘expressing’ the incubated effect of neglect and household instability related ACEs at age 24.

6.3.7 The role of covariates in depersonalisation and derealisation prevalence

Overall, mental health covariates had minimal attenuating effects on the observed relationships. Across all models, anxiety was a consistent, independent predictor of both DP and DR, whereas depression showed varied associations. In contrast, PC did not predict DP or DR in any model. These findings shed light on whether DPDR is a standalone symptom or as a symptom of other mental health conditions by identifying that it could be both. One, DPDR as a symptom of other conditions (e.g.

Panic Disorder, PTSD; Hunter et al., 2003), but also, DDD as its own disorder, arising in the context of ACEs (and likely other stressful stimuli).

6.3.8 Implications

Due to the presence of ACEs in DPDR, trauma-specific adaptations are required (Lippard & Nemeroff, 2020), for example, trauma-focused CBT (de Arellano et al., 2014), LI, which emphasises gentle processing and identity integration, show promise for dissociative presentations (Bahans, 2024; Thorpe, 2021), but are not yet widely available and require further evaluation. Multimodal approaches may hold greater promise for addressing DPDR in relation to ACEs: combining pharmacotherapy, neuromodulation, and psychotherapy (Wang et al., 2024). Early intervention is essential to reduce the long-term impact of ACEs.

Given the significant association between household instability and DPDR, prevention efforts must shift focus from solely addressing severe abuse to a broader consideration of the family environment. Early, integrated, and family-focused interventions, such as those offered by organisations like Adfam and NHS addiction services, could be critical to preventing long-term DPDR. Further, these findings suggest that public health and clinical screening should specifically focus on emotional forms of adversity when assessing the risk and outcomes for DP, given that EN may lead to escalation of odds over time, whereas EA may lead to chronically high yet stable DP over time.

EA is linked to emotion dysregulation, which mediates the relationship between EA and posttraumatic symptoms (Burns, Jackson & Harding., 2010) and prevents positive treatment outcomes (Mennin, 2006). Additionally, overall dissociation is correlated with maladaptive domains of emotion regulation (Lam & Fung, 2024), such as disengagement and aversive PC (Cavicchioli et al., 2021). To manage DPDR as psychological sequelae of EA, emotion regulation strategies may be a significant approach. In terms of DID and complex DDs, an online educational programme regarding skills to improve emotion regulation improved symptoms (Brand et al., 2019), although DPDR is a less complex dissociative phenomena and may lead to differential outcomes. Interventions such as mindfulness-based emotion regulation strategies (Raugh, Berglund & Strauss, 2025) may be a good treatment option, given

DPDR's positive response to other mindfulness-based approaches. Additionally, addressing maladaptive emotion beliefs in relation to emotion regulation problems may decrease use of maladaptive emotion regulation strategies such as expressive suppression, ignoring, rumination and catastrophising (Johnston et al., 2025).

Additionally, given the findings that DP and DR may have ACE-related aetiological differences, these findings necessitate a significant paradigm shift in future research and clinical practice. It is imperative that future studies disaggregate DP and DR in their analyses, moving beyond treating them as a single construct to explore their unique neurobiological underpinnings, cognitive mechanisms, and developmental trajectories. From a clinical perspective, this distinction is paramount: interventions may need to be specifically tailored to target the different underlying mechanisms depending on whether an individual primarily experiences DP, DR, or a combination of both. This could lead to more precise diagnostic formulations and the development of highly effective, mechanism-specific therapeutic strategies, ultimately improving outcomes for individuals suffering from these often-debilitating conditions.

6.4 Chapter 3

This section extends the investigation of ACEs and DPDR by examining the potential mediating role of systemic inflammation. Prior research has suggested an association between chronic inflammation and DPDR (Zheng et al., 2024), but these findings were limited by small sample sizes and clinical populations, making them prone to bias. To address these limitations, this study used a large-scale, population-based dataset to assess the relationship between inflammatory markers and DPDR, with the goal of providing more robust and generalisable evidence.

6.4.1 The role of inflammatory markers in depersonalisation and derealisation

This study examined two key inflammatory markers, IL-6 and CRP. IL-6 was only available at age 9, allowing for an analysis of childhood inflammation as a predictor of DP and DR at ages 12, 17, and 24. In contrast, CRP was measured at multiple time points (ages 9, 15, and 24), enabling an examination of the longitudinal effects of CRP fluctuations on DP and DR across development.

It was found that only a subset of ACEs predicted inflammation, and the effects varied by timepoint and biomarker. Cross-sectionally (ACEs up to age 9, IL-6 at age 9), IPV, parental divorce, neglect, household instability-related ACEs, and total ACE exposure were associated with elevated IL-6. Later, at age 24, PMI and parental divorce were associated with higher CRP, while EN was linked to lower CRP levels.

Despite strong theoretical links between ACEs and inflammation (for review, see Soares et al., 2021), findings from Chapter 3 did not support inflammation (IL-6, CRP) as a mediator between ACEs and DPDR. This suggests that while inflammation may play a role in DPDR, it is not the primary biological mechanism linking early environmental instability to DPDR. Therefore, household instability may exert its influence through alternative routes, such as autonomic arousal or HPA-axis dysfunction, which, while established predictors of DPDR (Giesbrecht et al., 2007; Simeon et al., 2001; Simeon et al., 2007; Simeon, Knutelska, & Nelson, 2003; Stanton et al., 2001), have not yet been investigated as mediators between household instability and DPDR.

6.4.2 A shift in perspective: inflammation as an independent driver of depersonalisation and derealisation

Given the absence of mediation, analysis focussed on the direct role of inflammation in DP and DR. The results pointed to distinct roles for IL-6 and CRP, suggesting that these markers act as independent biological mechanisms rather than mediators of childhood adversity.

IL-6 predicted DP at age 24, despite showing no significant association at age 17, indicating that childhood inflammation may exert long-term effects that emerge only in early adulthood. This finding resonates with broader psychiatric research linking early-life inflammation to later vulnerability to conditions such as depression and psychosis (Khandaker et al., 2014). Importantly, IL-6 was not associated with DR at any age, suggesting that it may represent a DP-specific mechanism.

The relationship between CRP and DR, in contrast, followed an opposite pattern to IL-6. Higher CRP levels at age 9 predicted increased DR at age 12, suggesting that elevated inflammation in childhood may be an early precursor to DR. At age 24, a

reduction in CRP levels relative to baseline was associated with increased DR prevalence. This unexpected finding suggests that instability in CRP levels over time, rather than consistently high or low levels, may contribute to DR symptom development. Notably, CRP was not associated with DP at any age, reinforcing the idea that CRP is specifically linked to DR, whereas IL-6 is related to DP.

6.4.3 Positioning C - reactive protein findings in existing research

While CRP is linked to other mental health conditions, its role in dissociation is mixed. Zhang et al. (2024) identified a link between CRP downregulation and increased overall DPDR in a sample similar to ours, though their specific correlation with DR was non-significant, possibly due to limited power. Conversely, Power et al. (2019) found higher CRP significantly predicted dissociation severity (measured using the dissociative experiences scale) while controlling for childhood trauma, PTSD, depression and emotion dysregulation. However, Power et al's findings used an older, demographically and clinically distinct sample (Type 2 diabetic African American women), suggesting that age and adiposity could explain these discrepancies (Wyczalkowska-Tomasik et al., 2015; Stanimirovic et al., 2022), whereas Zhang et al.'s findings utilised a Chinese sample.

Our research significantly contributes to this understanding by demonstrating that it may be CRP instability, rather than simply consistently high or low levels, that predicts DR. This novel finding suggests that dysregulated immune responses, rather than chronic inflammation alone, may be linked to DR, further indicating that DR may have distinct biological underpinnings from DP, supporting their partial independence. The observed temporal pattern of CRP and DR is particularly intriguing: an initial finding that increased CRP predicts DR aligns with transdiagnostic inflammatory models in psychiatry (Thylur & Goldsmith, 2022), yet a later transition to what appears to be immunosuppressive mechanisms predicting CRP at age 24 was unexpected, suggesting complex, potentially non-linear, mechanisms underlying the association between CRP and DR across development.

6.4.4 Implications

Together, these findings posit a novel perspective: systemic inflammation may represent a primary risk factor for DPDR, rather than simply a downstream

consequence of childhood adversity. By demonstrating distinct associations of IL-6 with DP and CRP with DR, this study provides the first longitudinal evidence that DP and DR may be differentiated at the biological level. While prior work has suggested potential neurobiological distinctions (Sierra et al., 2002; Hollander et al., 1992; Heydrich et al., 2019), these results extend the literature by showing that specific inflammatory pathways may differentially contribute to DP and DR. This strongly supports the need to disaggregate DP and DR in both research and clinical settings.

The findings suggest that inflammatory processes may operate independently of ACEs, running in parallel with psychosocial risk. This has significant clinical implications: it may help to explain why some individuals present with treatment-resistant DPDR even in the absence of identifiable (and treatable) trauma histories. Recognising that ACEs are not the sole pathway into dissociation broadens both conceptualisation and intervention strategies.

Clinically, these insights open the door to novel therapeutic approaches. If inflammatory dysregulation contributes to DPDR onset or maintenance, then interventions aimed at restoring immune homeostasis could represent new treatment avenues. This may include pharmacological anti-inflammatory strategies, as well as behavioural or lifestyle-based interventions designed to stabilise immune function, reduce systemic stress, and promote physiological equilibrium. In the long term, a biological signature of DPDR could also aid diagnosis, addressing current challenges such as alexithymia that obscure symptom recognition.

For DR specifically, the association with CRP fluctuations highlights the possibility that dynamic inflammatory instability, rather than chronically elevated inflammation alone, may disrupt perceptual and self-awareness processes. Future research should test whether autoimmune or stress-related immune responses contribute to these perceptual disturbances, and whether stabilising inflammatory rhythms could mitigate DR symptoms.

6.5 Chapter 4

Chapter 4 aimed to identify the key subjective triggers that lead to recurrent DPDR episodes and examine how these triggers interact with predisposing risk factors. Firstly, a crucial distinction must be made between aetiology and maintenance. While individuals with a history of ACEs and stress system dysfunction may be predisposed to experiencing DPDR, these factors alone do not necessarily lead to chronicity. Rather, DPDR often begins as a transient or fleeting state, which, through repeated activation, may become more frequent and ultimately chronic (Hunter et al., 2003).

To illustrate this concept, it is useful to draw a parallel with anxiety disorders, which are well understood within mental health research. Many individuals experience fleeting episodes of anxiety, yet this does not equate to an anxiety disorder. However, if an individual has experienced ACEs and has a dysfunctional stress response, they may become increasingly prone to interpreting specific environmental stimuli as anxiety-provoking. Over time, if these anxiety reactions occur frequently and in response to various triggers, the individual may develop and be diagnosed with generalised anxiety disorder (e.g. symptoms present for six months; American Psychological Association, 2013). A similar process appears to underlie DPDR. Individuals may initially experience DPDR as an isolated or momentary dissociative episode, but due to predisposing factors and recurrent exposure to specific triggers, DPDR may become self-reinforcing and chronic (as described in Hunter et al's 2003 cognitive-behavioural model).

Despite this, the majority of DPDR research has focused on its causes, often treating it as a disorder that emerges once and remains static, rather than considering the factors that prolong and sustain symptoms over time. However, as with GAD, the recurrence and persistence of DPDR are not inevitable but appear to be linked to identifiable triggers. These triggers, which may cause the recurrence and endurance of DPDR, represent an underexplored yet crucial aspect of DPDR phenomenology.

6.5.1 Investigating subjective depersonalisation and derealisation triggers through a lived experience perspective

To systematically identify the most reported subjective triggers of recurrent DPDR episodes, this study adopted a lived experience approach. Given that individuals with

DPDR frequently engage in online forums and communities, these naturalistic settings were used to extract meaningful insights. Online platforms provide accessible, real-world narratives of DPDR experiences, reducing potential biases that can arise in structured clinical interviews or experimental settings. This approach also aligns with broader trends in mental health research, where digital ethnography and online qualitative methods have become increasingly valuable tools for understanding lived experience in DPDR (Fury, 2023), and other areas of health research (for example, Almeida et al., 2025; Fusar-Polli et al., 2025).

The findings revealed that individuals consistently described a range of subjective triggers, which could be categorised into three overarching themes: Dysregulation of Core Systems, Environmental Reactivity, and Cognitive Triggers. While some of these triggers may appear unusual or unexpected initially, they align highly with the phenomenological experience of DPDR. For example, the act of looking in a mirror is an ordinary experience for individuals without DPDR, yet for those with DPDR, it reinforces the sensation of self-alienation or unfamiliarity with one's own reflection (for review see Caputo et al., 2021). This experience, in turn, may lead to distress, reactivating DPDR, illustrating how seemingly mundane stimuli can perpetuate the cycle.

Many of the subjective triggers identified have been previously noted anecdotally by clinicians and referenced in various clinical guides (Kennedy et al., 2013; Simeon & Abugel, 2006). Further, some specific triggers have been previously investigated in DPDR research (see 1.11 Maintenance of depersonalisation and derealisation). However, until now, these observations had not been systematically analysed or categorised, nor had they been examined from a lived experience perspective.

6.5.2 The role of stress as a mediator of depersonalisation and derealisation triggers

One of the most noteworthy findings was the consistent association between subjective DPDR triggers and stress activation. Many individuals reported that when confronted with a trigger, they experienced an immediate physiological or emotional reaction, such as feeling "freaked out" or distressed, which was then followed by the onset of DPDR. This suggests that stress activation may serve as a mediating factor

between exposure to triggers and the re-emergence of DPDR. This supports the hypothesis that heightened stress reactivity may play a central role in DPDR maintenance, reinforcing the link between stress system dysfunction and the disorder's persistence. These findings align with Chapter 3, which demonstrated that low-grade inflammation (a biomarker of chronic stress) was significantly associated with both DP and DR.

6.5.3 Important ethical considerations for internet-mediated research (IMR)

IMR presents ethical challenges, particularly regarding the privacy of online data, which blurs the lines between public and private. The British Psychological Society (BPS) guidelines for IMR are based on four key principles:

1. Privacy: Researchers must carefully assess users' expectations of privacy, even in public forums. Anonymising data and obtaining consent are crucial.
2. Scientific Integrity: The lack of demographic data in IMR can affect research rigor. Researchers must be transparent about these limitations and avoid misleading conclusions, especially when using complex data analysis techniques.
3. Social Responsibility: Researchers must avoid disrupting online communities and acknowledge that IMR may exclude individuals without digital access, potentially reinforcing inequalities.
4. Maximising Responsibility and Minimising Harm: Research must have clear scientific value, with ethical safeguards proportional to risks. Mitigating harm involves carefully anonymising data and paraphrasing content to prevent re-identification.

This project's ethical approach directly addresses the BPS guidelines. It respects the DPDR community's privacy by carefully handling data from online forums, using only paraphrased, anonymised content. The study's scientific value lies in its response to the community's demand for more research, with limitations (like the absence of demographic data) being openly acknowledged. The work is socially responsible by amplifying the voices of a marginalised population without disrupting their online spaces. Finally, potential risks are minimised by ensuring all shared content is untraceable and non-sensitive.

6.5.4 Implications

This research filled a crucial gap in the literature: which subjectively perceived experiences are reported by individuals as triggering recurrent episodes of DPDR? This new understanding allows an initial understanding of the diversity of DPDR triggers, and paves the way for further quantitative research, with potential to further treatment avenues.

A holistic and multifaceted approach is needed for treating and preventing chronic DPDR. Treatment should go beyond trauma-focused therapy to address a variety of ongoing, everyday triggers. Interventions targeting the dysregulation of core systems must address fundamental unmet needs. A variety of targeted interventions should be used to overcome the specific factors inhibiting these basic needs.

Individuals can aim to reduce DPDR by addressing stress activation in the moment (in response to other triggers) and work towards long-term homeostasis. In the first instance, the following strategies may provide relief: progressive relaxation, breathing exercises, and mindfulness strategies, e.g. meditation (Hazlett-Stevens & Fruzzetti, 2021). Additionally, as identified in Chapter 5, exercise was deemed a useful coping mechanism, and is evidenced to impact stress and autonomic arousal (for review, see: Paludo, Ferraz & Medeiros, 2024; Martins-Pinge, 2010; de-Abreu et al., 2019).

For longer term stress management, SSRIs may be effective, though results are mixed (Fiani et al., 2023). These mixed results could explain why some individuals with DPDR respond to antidepressants, and others do not (Halder, Kundu & Ray, 2023; Simeon, 2004; Simeon et al., 2004). Further, lamotrigine, an occasionally effective treatment for DDD (Alphy et al., 2024; Zheng et al., 2024), can reduce stress activation through glutamate modulation (Costa & Vale, 2023). Additionally, lamotrigine as an adjunct to SSRIs is also a promising treatment for DDD (Zheng et al., 2024), and it can be hypothesised that stress activation modulation may be partially responsible for the efficacy.

For sleep, implementing sleep hygiene practices can systematically address behaviours that prevent adequate rest. For individuals with long-standing insomnia, CBT-I is an effective route to address these deeper issues through sleep

consolidation, stimulus control, cognitive restructuring, sleep hygiene, and relaxation techniques (Rossman, 2019).

Addressing social isolation is critical, as a multitude of factors can inhibit socialising, such as co-occurring social anxiety, which is associated with having fewer confidants and reduced time with close friends (Hur et al., 2019). Social anxiety can worsen mood, yet individuals with social anxiety actually derive greater benefits from their close companions such as lower levels of anxiety, depression and negative affect (Hur et al., 2019), suggesting that targeting social anxiety could be an effective avenue for decreasing DPDR through improved wellbeing, such as through psychotherapy, an effective treatment for social anxiety with several different modalities (de Ponti et al., 2024).

Environmental reactivity can be a potent source of triggers for individuals, as they are often unavoidable (e.g., mirror exposure, certain lighting, and screens). Daily, practical changes for individuals include self-care measures to mitigate the impact of these environmental triggers. For example, limiting extensive screen use or using blue-light glasses for digital eyestrain (Coles-Brennan, Sulley & Young, 2019), particularly for individuals who are exposed to screens for long periods (e.g. university students, Chen et al., 2024). Additionally, blue-light glasses can be an effective intervention to improve insomnia, and thus may provide a dual-action intervention, targeting both eyestrain from screens and targeting insomnia (Hester et al., 2021). Additionally, wearing sunglasses can help manage the impact of bright, straining environments, such as those found in supermarkets or hospitals, or on sunny days.

In relation to cognitive triggers, Chapter 5 evidenced that hyper-reflexivity has a profound association with DPDR, and Chapter 4 evidenced that hyper-reflexivity is linked to stress activation. Since a key therapeutic goal in managing DPDR is restoring physiological homeostasis by targeting stress activation, psychoeducation can help individuals understand its reciprocal relationship with DPDR and its link to hyper-reflexivity. Mindfulness-based cognitive therapies may then reduce hyper-reflexive thoughts that heighten arousal. Brief cognitive-behavioural group therapy, shown to reduce DPDR in students with acute test anxiety (Schweden et al., 2020),

incorporates psychoeducation on catastrophic attributions and interoceptive exposure to test whether changing DPDR appraisal alters symptom persistence. These findings suggest this approach can effectively address hyper-reflexivity while linking DPDR reduction to anxiety-related stress activation.

6.6 Chapter 5

The final study (Chapter 5) provided a multidimensional analysis of DPDR in young adults, providing novel insights by: (1) testing dose-response models for ACEs; (2) testing correlations between ACE frequencies and CDS subscales (anomalous body experience, emotional numbing, anomalous subjective experience, and unreality of surroundings); (3) clarifying the role of cognitive mechanisms (trait rumination and hyper-reflexivity) in DPDR severity; and (4) validating qualitative findings from Chapter 4 by investigating the perceived impact of the subjective, empirically derived triggers and coping strategies on current DPDR severity.

6.6.1 Dose-response relationships

Unlike previous binary assessments, this study used the CDS, allowing an investigation into whether increasing frequency of ACEs led to increasing DPDR severity (a dose-response relationship). This was achieved using four methods: frequency of single ACE types (doses ranged from 0-5), a multivariate risk model, ACE category count, and total ACE exposure.

6.6.1.1 Single ACE types

When examining the dose-response relationship for individual ACE types, SA demonstrated the most substantial effect, with each event predicting a 13.25-point increase in CDS scores (a maximum possible increase of 66.25 points). Following this, serious illness/injuries in childhood (9.93-point increase per event), major upheavals in childhood (9.34-point increase per event), and parental divorce (7.97-point increase per event) were all significant single predictors of CDS scores. Crucially, while the overall models for SA and major upheavals were significant, the overall models for both serious illness/injuries in childhood and parental separation/divorce were not statistically significant, despite the significant relationship between each variable within the model. Additionally, in the multivariate risk model

(controlling simultaneously for shared variance between single ACEs), only SA and major upheavals were significant predictors of CDS scores.

6.6.1.2 ACE category count and total ACE exposure

Analyses of ACE category count (ranging from 0 to 6) demonstrated a highly potent dose-response effect. Each different type of ACE experienced was associated with a 16.38-point increase in CDS scores. This cumulative effect indicates that individuals who experienced all six types of ACEs would have a maximum predicted CDS score increase of 98.28 points. This maximum score is 28.28 points above the clinical cut-off for DPDR (70), starkly illustrating that the multiplicity of ACEs is strongly predictive of severe DPDR outcomes.

In contrast, analysis of total ACE exposure (the aggregate number of individual events experienced, from 0 to 21) was associated with a comparatively smaller 4.18-point increase per event. This total count predicted a maximum increase of 87.78 CDS points for those experiencing 21 events. While a strong association, this finding reinforces that the variety of trauma (ACE category count) is a more potent predictor of DPDR severity than the sheer frequency of individual adverse events (Total ACE exposure).

ACE category count and total ACE exposure capture different aspects of an individual's adversity history. The ACE category count focuses on the diversity of experiences, quantifying the number of different types of ACEs faced. In contrast, total ACE exposure is a less granular aggregate measure of the sheer frequency of individual events, counting both repeated instances of the same ACE and different ACE types. While this second measure's primary limitation is its inability to distinguish chronic repetition from diverse exposure, its highest scores likely represent the most severe cases of both high frequency and high multiplicity, but this comprises only a specific subset of individuals.

An alternative interpretation for why total ACE exposure (the sheer number of events) was a weaker predictor of DPDR severity than the category count relates to the spectrum view of dissociation. It is plausible that high-frequency, chronic exposure to adversity is primarily linked to the development of more severe dissociative reactions,

such as dissociative amnesia or characteristics of DID, that are not fully captured by the CDS. In this context, DPDR represents a milder presentation, likely developing in response to less chronic or complex ACE exposure. This is consistent with prior research identifying that DID is associated with the greatest severity and multiplicity of childhood maltreatment (Daniels et al., 2024), suggesting that the effect of high-frequency trauma exposure may manifest as dissociative symptoms beyond the scope of this study.

Despite the differences in predictive strength, both cumulative measures (ACE category count and total ACE exposure) confirm that increased exposure to adversity intensifies DPDR.

6.6.2 Associations between ACE frequencies and Cambridge Depersonalisation Scale subscale scores

This study provides the first investigation into the associations between an array of ACEs and the specific symptom dimensions of DPDR (CDS subscales) in a non-clinical population. The analysis revealed varied patterns of association between ACE exposure and DPDR dimensions. When examining single ACE types, SA was significantly and positively correlated with the emotional numbing subscale. This finding is highly significant, as it extends evidence from clinical groups (Dearden & Medford, 2017) to a community sample, highlighting that the SA-emotional numbing pathway is broadly relevant and supports models defining a "Severe Dissociative" phenotype (individuals who reported the highest PTSD symptom severity but also endorsed frequent DPDR; Frewen et al., 2015). Furthermore, major upheavals in childhood were significantly correlated with both anomalous body experience and anomalous subjective recall, yet due to the lack of specification of what ‘major upheavals’ entails, it cannot be compared to past literature.

ACE category count was significantly correlated with anomalous body experience, emotional numbing, and anomalous subjective recall, while total ACE events correlated with the two anomalous experience subscales. This once again reinforces ACE category count as more concretely associated with DPDR.

Significantly, no single ACEs or cumulative ACEs were significantly associated with alienation from surroundings, the subscale describing DR, aligning with results from Chapter 2, indicating that ACEs are comparatively weak predictors of DR relative to DP.

Taken together, these results reinforce the hypothesis that DP is more strongly predicted by the exposure and multiplicity of ACEs, whereas DR appears less directly linked to early adversity and may operate via distinct biopsychosocial pathways.

6.6.3 The role of cognitive factors in depersonalisation and derealisation

While prior studies have suggested an association between trait rumination and DPDR (Vannikov-Lugassi et al., 2021; Quigley, Warren & Townsend, 2024), the current findings indicate that this relationship is better accounted for by hyper-reflexivity.

In isolation, trait rumination showed only a weak dose-response relationship with DPDR and explained a modest 3.4% of the variance in symptoms. Crucially, it also failed to play a significant role in mediating the relationship between ACEs and DPDR severity.

Further, when both trait rumination and hyper-reflexivity were entered into a combined model, the association between trait rumination and DPDR was fully attenuated, whereas hyper-reflexivity remained a strong and significant independent predictor. This distinction is theoretically meaningful: the model including only hyper-reflexivity significantly outperformed the combined model, explaining 12.9% of the variance in DPDR compared to 11.3%. Overall, the results point toward rumination being a weak predictor of DPDR in isolation.

While rumination is a stable, transdiagnostic trait, hyper-reflexivity more directly captures the symptom-driven content characteristic of DPDR: the excessive self-awareness and altered experiential perspective. The difference in model performance suggests that the variance in symptoms previously attributed to general, trait-like rumination is likely due to an unmeasured overlap with this more specific mechanism. Since past research did not identify the content of ruminations (e.g.,

Vannikov-Lugassi et al., 2021), the present results suggest that earlier findings identifying rumination as predictive of DPDR may have inadvertently measured the effects of hyper-reflexivity, which is critically linked to DPDR severity.

These findings provide further empirical support to qualitative conceptualisations of hyper-reflexivity as a facet of DPDR (Ciaunica, 2021, 2022), while also supporting the cognitive-behavioural of DPDR (Hunter et al., 2003), which posits that symptom maintenance is driven by catastrophic misinterpretation and subsequent self-monitoring. The results of Chapter 4 thus suggest that hyper-reflexivity acts as the specific mechanism that drives this catastrophising and self-monitoring over rumination.

6.6.4 Verifying qualitative findings quantitatively

This study found that DPDR severity was associated with increased sensitivity to a range of maintenance triggers subjectively reported by participants in Chapter 4. Crucially, the likelihood of experiencing distress was highest for selfhood triggers (reflection, hands, self-staring), potentially suggesting that these triggers lead to an intensified self-awareness characteristic of hyper-reflexivity (Pérez-Álvarez, 2008), potentially suggesting an intimate relationship.

Additionally, a behavioural paradox was observed: while the likelihood of triggering DPDR and related distress was high, avoidance was only significantly associated with self-staring. This suggests self-staring poses a unique threat that necessitates an active behavioural response, reinforcing the need for clinical targets like exposure therapy, which has seen success in other conditions (e.g., body dysmorphic disorder, Griffen, Naumann & Hildebrandt, 2018).

Additionally, Chapter 5 found no significant negative correlations between the perceived effectiveness of any coping strategy and lower DPDR severity. Nevertheless, strategies like mindfulness and exercise showed promising negative associations, and the results underscore the need for future research into targeted towards these interventions, which have shown efficacy in dissociative and similar conditions (D’Antoni et al., 2021; Hefferon, Grealy & Mutrie, 2009).

Additionally, the perception of coping strategies as efficacious were negatively associated with CDS scores, suggesting a role of self-efficacy in DPDR severity. These results may have inadvertently indicated that the self-efficacy, rather than the outcome of the coping mechanism, may be the critical variable associated with lower CDS scores. This interpretation is supported by established findings that high treatment self-efficacy predicts greater symptom reduction in anxiety (Schønning & Nordgreen, 2021) and that clinically affected adolescents with DPDR report lower self-efficacy (Michal et al., 2015). Given that diagnostic delays may further erode autonomy and self-efficacy (Baker et al., 2003), these results suggest that building self-efficacy alongside targeted interventions like mindfulness and exercise is a credible research area.

6.6.5 Implications

This chapter provided several important findings: the dose-response analysis confirmed that SA was the largest risk factor for DPDR severity (out of the measured ACEs), and that trauma multiplicity is a significantly stronger predictor of DPDR severity than total ACE event frequency. Additionally, correlational findings suggested that DP is more strongly associated with ACEs, whereas DR was not correlated with any ACE category, supporting the previous findings suggesting distinct aetiology. Additionally, hyper-reflexivity was identified as a significant factor driving DPDR severity and potentially maintenance, potentially refining the cognitive-behavioural model of DPDR (Hunter et al., 2003).

DPDR severity was associated with increased general sensitivity to triggers, with the highest distress linked to selfhood triggers (e.g., self-staring, looking at hands). This confirms the role of hyper-reflexivity as the core mechanism of distress generation. Furthermore, this internal distress translated into a specific behavioural finding: individuals with higher CDS scores were more likely to avoid self-staring, reinforcing its status as a uniquely high-risk trigger and a potent target for intervention.

6.7 A revised biopsychosocial model of depersonalisation and derealisation

This section integrates the findings from this thesis, presenting a cohesive model that accounts for the complex interplay of biological, psychological, and social factors underlying DPDR. This model posits that DPDR emerges from dysregulation of biological stress systems, triggered by stressors, such as ACEs (but not exclusively). The critical factor is a stressors' capacity to disrupt the functioning of systems such as the immune response, but also the ANS and HPA-axis (as addressed in previous research). This dysregulation creates a physiological environment conducive to DPDR.

Furthermore, the model suggests that hyper-reflexivity is a particularly potent factor; a likely cognitive consequence of stress system dysfunction, and highly prevalent within DPDR, acting as a maintenance mechanism, intensifying self-monitoring and perpetuating DPDR. Additionally, other maintenance triggers include stress activation, sleep disturbances, social isolation, mirror exposure, lighting types, and existential-philosophical thoughts. Each trigger was linked, to some extent, with stress activation, suggesting that it could mediate the relationship between triggers and DPDR.

Beyond abuse and neglect, it is proposed that other factors also contribute to stress system dysregulation, including household instability (e.g., PSA, divorce, IPV, as explored in Chapter 2), the presence of stress system dysfunction biomarkers (Chapter 3) potentially linked to unmeasured unsafe environments (such as neighbourhood safety or bullying), the persistence of stress system activation in relation to DPDR (Chapter 4), and experiences of SA, serious illness or major upheavals in childhood (Chapter 5). This aligns with the understanding that DPDR is part of the broad spectrum of traumatic responses, and this model includes the belief that further stressors, such as community violence (as seen in comorbidity with C-PTSD; Maercker et al., 2022) or natural disasters (Pietkiewicz, Duszkiewicz & Tomalski, 2023), could also contribute, as it is the impact of stress on the body that creates the environment for DPDR to arise.

Our findings complement existing evidence suggesting that adult stress can equally cause DPDR. This model suggests that factors causing stress system dysregulation at any age is at its root. For instance, both ACEs and adult traumatic events predict D-PTSD, while ACEs are a stronger predictor of C-PTSD (Frewen, Zhu, & Lanius, 2019). Both D-PTSD and C-PTSD include dissociative elements. While the model does not privilege any single developmental period, it recognises childhood as a particularly vulnerable time due to heightened brain plasticity and sensitive periods of neurodevelopment (see 1.9.1 Sensitive periods of development). As such, early life events may have a more potent and enduring impact on stress system regulation and subsequent self-representation. However, this model also emphasises that significant stressors in adolescence or adulthood can result in DPDR if they fundamentally alter biological stress system function (such as in D-PTSD; White et al., 2022).

While the study of genetic or epigenetic predispositions in the development of pathological DPDR was outside of the scope of this thesis, they are additional factors that are important to DPDR. Not all individuals who experience stressful life events and subsequent dysregulated stress systems experience pathological, or even transient, DPDR. This suggests a genetic predisposition, which aligns with the diathesis-stress model. Evidence from the first genome-wide association study (GWAS) on DPDR found 10 suggestive single nucleotide polymorphisms (SNPs) associated with symptom severity (Wolf et al., 2014). Notably, SNP rs263232, located in the ADCY8 gene on chromosome 8, is linked to emotion regulation and memory, and this association remained significant even after controlling for PTSD severity. Furthermore, individuals who develop pathological DPDR likely have a predisposition toward neuroticism (Fino et al., 2024) which could foster hyper-reflexivity during DPDR episodes. This hyper-reflexivity, in turn, may further activate the dysregulated stress system, perpetuating the disorder.

Another central feature of this model is that DPDR may persist well beyond the original stressor due to continued biological stress system activation. Processes like hyper-reflexivity, maintenance triggers, and unresolved physiological arousal can lock the nervous system in a feedback loop. Thus, inflammation, cortisol, and stress dysfunction may act not only as triggers but also as maintenance mechanisms that prolong or intensify symptoms.

Additionally, the physical manifestation of this dysregulated stress system can be observed by biomarkers of stress, predicting DPDR both cross-sectionally and longitudinally. In the present thesis, inflammatory markers were significantly associated with DP or DR, depending on the marker measured. Interestingly, these inflammatory markers were predicted by only a few ACEs, and where mediation analysis was possible, they did not mediate the ACE-DPDR relationship. Yet, low-grade inflammation is a marker of chronic stress, suggesting alternative stress sources may have caused inflammation in the sample used in Chapter 3: for example anxiety or depression, which were evidenced to predict DPDR exclusively of ACEs (as demonstrated in Chapter 2), and are also evidenced as related to inflammatory markers (for review, see Szabo, Burns & Lantrip, 2022; Mac Giollabhui et al., 2021; Costello et al., 2019), and are associated with physiological stress symptoms (Black et al., 2015; Alvares et al., 2016 Ottaviani et al., 2016). Although not the primary scope of this thesis, the findings did suggest a potential hypothesis for mediation through mental health symptoms and DPDR. Additionally, social stressors not measured in the present study, but implicated for both inflammation and DPDR, may have an effect, for example childhood bullying (Campbell & Morrison, 2007; Copeland et al., 2014). These findings strengthen the argument that DPDR is associated with observable biomarkers, and that ACEs are not the sole stressor implicated.

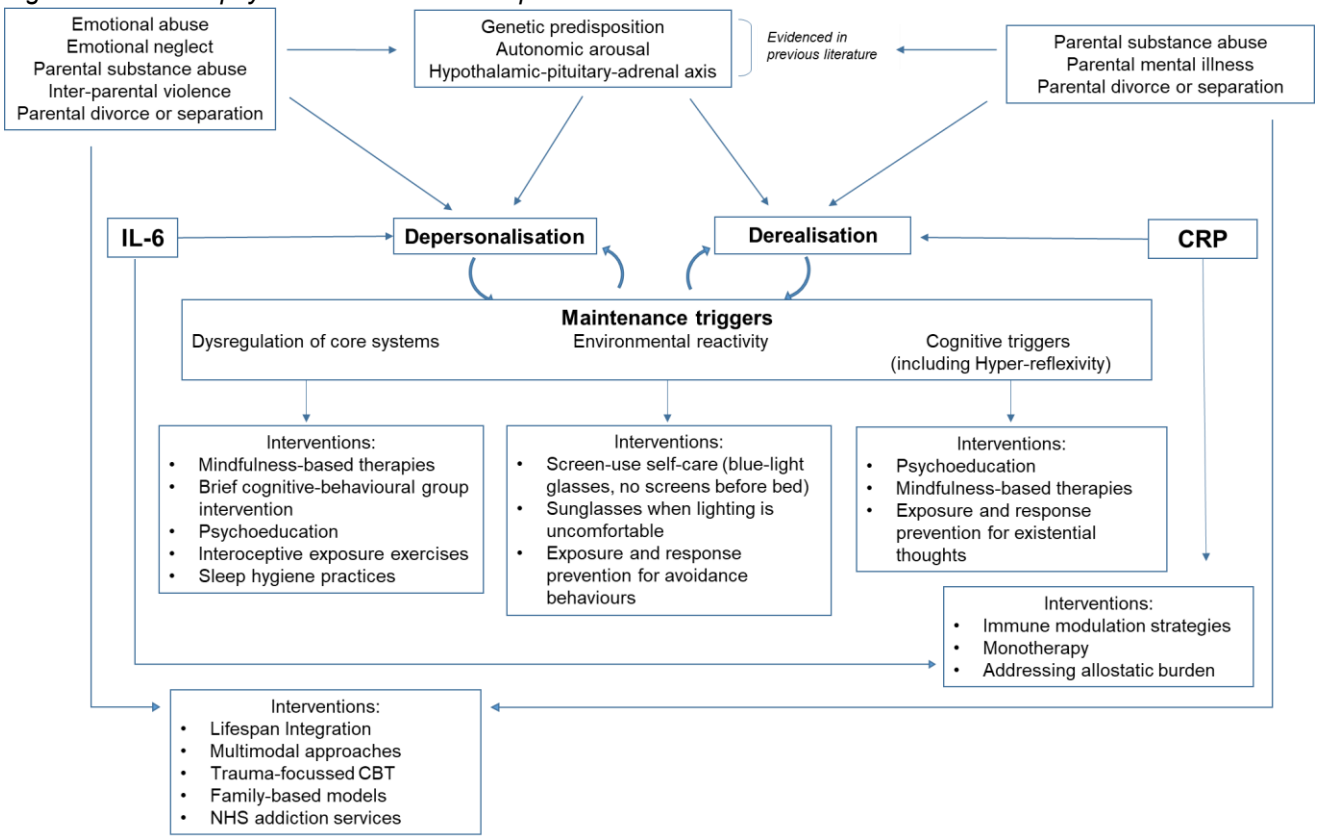
This hypothesis also recognises that DP and DR are not a single unified construct. While they frequently co-occur and share functional consequences, they may have distinct aetiological pathways, both in terms of stressor nature and underlying biological mechanisms. In this thesis, DP was associated with a larger variation of ACEs, including abuse, neglect, household instability, and cumulative variables. Conversely, DR was associated with fewer ACEs and no cumulative variables. Additionally, DP was significantly associated with childhood IL-6 but not CRP over time, whereas DR was associated with CRP over time but not childhood IL-6.

Finally, the hypothesis suggests that targeting stress system dysfunction is essential for recovery. Interventions that reduce inflammation, regulate stress (e.g., vagal nerve stimulation, grounding techniques), or normalise cortisol rhythms may offer

relief, especially when paired with trauma-informed psychotherapeutic approaches. This highlights the potential for biopsychosocial treatment frameworks addressing both the biological and experiential dimensions of DPDR.

Taken together, these findings support the biopsychosocial model of DP and DR (see figure 15), reframing them as the product of stress-system dysregulation interacting with stressful life experiences and cognitive vulnerabilities. This integrative perspective offers a foundation for future research and clinical innovation.

Figure 15: The biopsychosocial model of depersonalisation and derealisation



6.8 Intervention approaches

6.8.1 Psychological and trauma-focussed interventions

Trauma remains a significant risk factor in the development of DPDR, and trauma-focused approaches are widely considered first-line treatments. EMDR can be applied with modifications for dissociative presentations, such as prolonged preparation, phased exposure, and grounding techniques to prevent destabilisation

(Shapiro & Brown, 2019; Mosquera, 2021). However, EMDR demonstrates reduced efficacy in patients with severe dissociation or complex trauma histories (Bae et al., 2016), highlighting the need for alternative modalities.

Emerging therapies such as LI emphasise gentle processing of traumatic material and integration of fragmented autobiographical memory, with preliminary findings suggesting improved outcomes for individuals with dissociation (Bahans, 2024; Thorpe, 2021). Trauma-focused CBT has also been adapted for younger populations with ACEs, showing significant reductions in dissociative and trauma-related symptoms (de Arellano et al., 2014). Multimodal and family-based interventions (Calhoun et al., 2015) demonstrate potential to buffer long-term dissociative outcomes, particularly important given evidence that childhood maltreatment complicates recovery trajectories (Lippard & Nemeroff, 2020).

Psychological therapies for DPDR should emphasise regulation of stress, addressing maladaptive cognitions, and improving coping. CBT shows moderate efficacy, with 29% of participants no longer meeting DDD diagnostic criteria following a course of therapy (Wang et al., 2024). Adapted CBT focusing on psychoeducation, interoceptive exposure, and addressing hyper-reflexivity aligns with Hunter et al.'s (2003) cognitive-behavioural model and may reduce chronic symptoms (Schweden et al., 2020).

Mindfulness-based interventions are promising, given evidence of an inverse association between DPDR and mindfulness (Michal et al., 2007). Case evidence demonstrates complete remission of DDD symptoms with mindfulness-based cognitive therapy (Mishra et al., 2022). Mechanistically, such approaches may reduce hyper-reflexivity, disrupting the cognitive loops that reinforce DPDR (Vancappel et al., 2021). Additionally, psychoeducation by may normalise transient experiences, reducing catastrophic interpretations (Hunter et al., 2003).

6.8.2 Biological interventions

Although pharmacological treatment remains inconclusive, research offers important insights into the neurobiological underpinnings of DPDR. A systematic review (Wang et al., 2024) identified modest effects across SSRIs, tricyclic antidepressants, opioid

antagonists, anticonvulsants, and antipsychotics. Naloxone, for instance, demonstrated efficacy in placebo-controlled trials, implicating the opioid system in symptom regulation. Similarly, lamotrigine in combination with SSRIs has shown partial benefit in treatment-resistant cases. However, evidence relied on small samples. Novel avenues involve immune-modulating strategies, given this thesis' findings linking inflammatory dysregulation to DPDR. However, further research into the inflammatory processes surrounding DPDR are needed.

6.8.3 Environmental and lifestyle interventions

Given the role of physiological stress, lifestyle-based modifications constitute important adjunctive interventions. Exercise demonstrates robust effects in trauma recovery, resilience-building, and post-traumatic growth (Nilsson et al., 2019; Hefferon, Grealy & Mutrie, 2009). While not yet systematically trialled in DPDR, exercise may mitigate symptom severity through improved arousal regulation and inflammatory stabilisation (Harte, Vujanovic & Potter, 2015; Ley, Barrio & Koch, 2018).

Lifestyle changes such as sleep hygiene (e.g., limiting screen exposure before bedtime), paced screen use, and light adjustments (e.g., sunglasses for sensitivity) combined with autonomic relaxation strategies, may help individuals. Integration of digital tools, such as NHS-approved mindfulness platforms (e.g., Silvercloud), can expand accessibility, particularly given lengthy NHS wait times for specialist support (South West London & St George's NHS Trust, 2022).

6.9 Limitations

In Chapters 2 and 3, the primary measure of cumulative ACEs was the total number of ACE events experienced, stratified into two broad clusters: abuse-related ACEs, and neglect/household instability ACEs. While this distinction was intended to reflect differences in severity, with abuse representing the more severe end of the spectrum, the approach still implicitly assumes that all ACEs within each cluster contribute equally to harm. This is a limitation, as the models captured frequency of exposure across years rather than the severity or qualitative associated with different ACE types. A more nuanced approach, such as latent class analysis, could have identified

naturally occurring patterns of ACE clustering, allowing us to determine which combinations of adversities were most strongly associated with DP or DR.

Additionally, while CRP was assessed longitudinally, IL-6 was only measured once (at age 9), which severely constrained the ability to examine its inflammatory trajectories across development. Future studies should incorporate repeated, dense measures of IL-6 to clarify its longitudinal contribution to DPDR. To build on the DiD finding, larger cohorts with denser biomarker sampling are needed to clarify whether CRP instability at particular developmental windows disproportionately contributes to DP risk. Additionally, advanced causal inference methods are necessary to disentangle the bidirectional nature of the relationship between inflammation and DPDR.

In Chapters 2 and 3, the use of binary measures of DP and DR as outcome variables limited capacity to capture symptom severity, frequency, or duration, thereby restricting the exploration of subtle symptom changes or dose-response relationships. This was addressed in Chapter 5 but using a much smaller sample and less varied ACEs.

Because the cumulative ACE measures summed total exposures (Chapters 2, 3, and 5), the chronicity of exposure could not be captured, meaning distinguishment between continuous year-on-year exposure and intermittent episodes over the span was not possible. This limits the precision with which the persistence of adversity can be assessed. Nevertheless, the approach still provided a robust dose–response estimate by quantifying the overall frequency of exposure, capturing the cumulative burden of adversity on DPDR.

While severity of adversity is often prioritised in measurement, chronicity and intermittency may be equally, if not more, important to capture when considering developmental impact. Severity typically reflects a singular time point, giving little indication of how adversity unfolds across the life course. In contrast, chronic exposure may affect multiple sensitive periods of development, exerting a cumulative influence on neurobiological and psychosocial systems as they emerge. Similarly, intermittent exposure, though not constant, can disrupt several distinct

developmental windows, leaving repeated imprints over time. Both chronic and intermittent adversity therefore provide richer insight into developmental pathways to DPDR than severity alone, as they reflect how adversity persists or re-emerges during periods of heightened vulnerability.

In Chapters 2, 3, and 5, ACEs were modelled but did not include adult adverse experiences. This is important because childhood adversity increases later revictimisation: in a national survey, exposure to one abuse type doubled risk of past-year physical assault and tripled risk of IPV or sexual violence; multiple abuse types raised risks roughly 3× (physical assault), 6× (IPV), and 7× (sexual violence) (Butler, Quigg & Bellis, 2020). Revictimisation also differs by gender and race/ethnicity: women face higher sexual/physical revictimisation, and Black and Hispanic individuals have higher rates than White individuals (Widom, Czaja & Dutton, 2008). Accordingly, not adjusting for adult revictimisation and sociodemographic factors surrounding them may confound ACE-DPDR associations at ages 17 and 24.

A significant limitation was the inability to estimate a single, integrated model spanning ACEs, inflammatory markers, cognitive processes, and DPDR outcomes. This was primarily due to reliance on a secondary dataset for ACE and biomarker variables (with pre-set constructs and schedules), and because the trigger/maintenance findings came from qualitative work that couldn't be integrated into the earlier quantitative analyses. Consequently, the inferences relied on converging evidence across complementary models, rather than one unified framework. Future research requires purpose-built longitudinal designs with harmonised measures and concurrent, dense sampling to test relative effects and mediation in a unified model.

6.10 Strengths and Contributions

This thesis makes several substantive contributions. First, the study is among the first to examine DP and DR as distinct outcomes within a biopsychosocial framework, using ACEs, comorbid symptoms and objective biomarkers to identify potentially divergent etiological pathways. This approach helps to clarify inconsistencies in the literature and supports more targeted investigation of DP and DR as separate

experiences. Second, the work employs methodological triangulation: longitudinal cohort analyses, cross-sectional modelling, and qualitative thematic analysis.

Biologically, the thesis identifies differentiated inflammatory patterns (DP associated with IL-6; DR with CRP instability), offering a novel account of distinct pathways. Analytically, the use of robust models (e.g., GLMMs), multiple-testing control, and a DiD approach for inflammatory instability strengthens inference from observational data. Additionally, these findings advance existing theory by showing that hyper-reflexivity explains variance beyond trait rumination and likely sustains symptoms in line with catastrophic feedback models.

The qualitative component extends maintenance accounts beyond the classic loop, mapping real-world triggers and situating them within stress/autonomic processes, thereby explaining the episodic, context-sensitive nature of DPDR.

Throughout, the thesis demonstrates measurement transparency, enhancing reproducibility. Looking forward, the thesis sets a clear agenda for mechanism-informed, multimodal care. A principal hope is to drive the integration of these findings into precision therapeutic protocols. This involves explicitly disaggregating DP from DR in assessment, using inflammation screening to guide pharmacological decisions (following further research confirming the role of inflammation in DPDR), and treating hyper-reflexivity as a core, targetable mechanism. Specifically, this could include adapting principles from existential OCD treatment, such as exposure and response prevention, to address hyper-reflexive thoughts and associated avoidance behaviours, potentially through controlled mirror use. While trauma-focused CBT must remain the first-line intervention, incorporating these mechanistic targets will pave the way for a new generation of personalised, effective care for dissociative experiences. Further, lifespan integration is a promising avenue for treatment but is yet to be widely rolled out in the NHS.

The longitudinal nature of the cohort provided substantial statistical power and unique temporal insights into psychobiological pathways. The design enabled the robust tracking of ACEs across childhood (from 8 months to 12 years), as well as

DPDR and secondary factors (such as attachment, anxiety, and depression) across key developmental stages.

Crucially, the repeated assessment of CRP at three distinct time points (ages 9, 15, and 24) was particularly valuable. This provided the empirical basis for the dynamic identification of inflammatory fluctuations and facilitated the novel application of a quasi-experimental DiD model to test the specific association between inflammatory instability and DR.

Focusing on general population cohorts rather than clinic samples strengthens the real-world applicability of the findings. It avoids biases that are common in treatment-seeking populations and captures the full range of individuals experiencing DPDR, including subclinical DPDR. Clinical samples may reflect higher symptom severity and access to specialised care, which can be skewed toward people with greater resources, such as higher-income households. As a result, the prevalence and variability estimates are more realistic, and the links between ACEs, inflammation, and cognitive processes are more likely to reflect the population, rather than artefacts of clinical sampling.

6.11 Conclusion

This thesis offers a systematic, multifaceted account of DPDR, integrating ACEs, inflammatory processes, cognitive styles and maintenance factors. By combining longitudinal, quantitative, and qualitative approaches, this thesis advances understanding of DPDR's complex aetiology and chronic, often relapsing course.

Across chapters, DP and DR emerged as related yet separate phenomena. ACEs predicted DP more robustly and straightforwardly than DR, for which associations were more complex. Biological findings reinforced this differentiation: DP tracked with IL-6, while DR aligned with CRP, suggesting distinct inflammatory pathways.

Evidence for classical "biological embedding" via inflammatory mediation was limited. Few ACEs co-predicted both DP and DR and IL-6 and CRP, and where preconditions were met, mediation paths were null. Therefore, in this cohort,

inflammation appeared to operate largely in parallel to psychosocial risk rather than as a conduit linking ACEs to symptoms. Two implications follow: first, alternative biomarkers may better capture biological embedding in terms of DPDR; second, unmeasured ACEs (e.g., bullying, community violence) may show stronger biosocial coupling. Notwithstanding these constraints, the observed pattern - DP with IL-6 and DR with CRP - highlights biologically plausible routes to symptom expression that merit follow-up.

The qualitative study identified maintenance factors beyond the classic catastrophic feedback loop. It catalogued diverse, context-sensitive subjective triggers (dysregulation of core systems, environmental reactivity, and cognitive triggers) that often relate to stress, episodically re-activating symptoms. This dynamic view challenges static explanations and underscores that factors interact to retrigger DPDR over time.

Cognitively, trait rumination predicted DPDR, but its apparent association was largely absorbed by hyper-reflexivity. In practice, general rumination may foster catastrophic interpretations that amplify hyper-reflexivity, sustaining stress physiology and prolonging symptoms (coherent with the PCH and Hunter et al.'s cognitive-behavioural model) yet hyper-reflexivity itself appears the more disorder-specific driver of persistence.

Clinically, these findings could help explain treatment resistance when interventions target a single level (pharmacological or psychological) without addressing trauma profiles, inflammatory vulnerability, and DPDR-specific cognitions. Multimodal formulations that (1) disaggregate DP from DR, (2) consider trauma history, (3) consider inflammatory risk (including variability), and (4) assess hyper-reflexivity, may improve care pathways.

In terms of future directions, longitudinal studies should be conducted, including the following parameters: (1) sampling IL-6 and CRP more densely to test timing and variability effects, (2) extending ACE measurement to type, frequency, severity, and developmental timing, and (3) including measures of hyper-reflexivity.

In sum, this thesis clarifies DP and DR as separate constructs with distinct psychosocial and biological correlates, identifying hyper-reflexivity as a key cognitive mechanism, and models DPDR as a dynamic, multi-systemic condition shaped across development.

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Appendices

Appendix 1: ACE Construction Supplementary Information

To operationalize adverse childhood experiences (ACEs), binary variables were constructed at multiple developmental time points: 8 months, 1 year, 2 years, 3 years, 5 years, 6 years, 9 years, and 11 years. These variables were derived from parental self-report questionnaires completed by mothers and, where available, their partners.

1. Domains Included

At each time point, indicators were created to reflect whether a child was exposed to any of the following ACE domains:

1. Parental emotional abuse
2. Parental physical abuse
3. Emotional neglect*
4. Physical neglect*
5. Sexual abuse (included only in Abuse-related ACE scores due to extremely small samples)
6. Parental substance use (including cannabis, amphetamines, heroin/cocaine, alcohol dependence, or general addiction)
7. Parental mental illness or suicidality (including suicide attempts, antidepressant use, schizophrenia, anorexia, bulimia)
8. Interparental violence (IPV)
9. Parental divorce
10. Parental criminal conviction

**Emotional neglect was measured retrospectively by individuals at age 22, and physical neglect at age 24.*

Each domain was binary-coded per year of measurement (1 = exposure, 0 = no exposure that year).

2. Handling Missing Data and Dual-Informant Reporting of Perpetration

To enhance the accuracy and completeness of abuse exposure data, a dual-informant approach was employed, utilising responses from both mothers and partners. Each parent was asked to report on their perpetration of ACEs. If there was missing data for one parent, then it could beestablished whether there was any household-ACE perpetration based on the alternative parents' report. If both parents had missing data, then the participant was removed. If both parents reported as having perpetrated, then this variable was recoded to 1. If one parent reported as having perpetrated, and the other parent did not perpetrate, then they were assigned a value of 1, which indicated it being present by at least one parent in the household.

For emotional abuse and physical abuse, which was more likely to have a high level of missing not at random data, each parent was asked to report on (1) Their own perpetration of abuse (self-report), and (2) their partner's perpetration of abuse (partner-report). This resulted in four potential data points for PA and EA:

1. Mother-reported mother perpetration
2. Mother-reported partner perpetration
3. Partner-reported partner perpetration
4. Partner-reported mother perpetration

These sources were synthesized into a single binary indicator for each ACE domain (e.g., parental emotional abuse) using the following rules:

- If both parents provided data, and at least one reported abuse (whether self- or partner-perpetrated), the variable was coded as 1 (abuse present).
- If both reported and both denied any abuse, the variable was coded as 0 (abuse absent).
- If only one parent provided data but the other parents' data was missing, the value from that parent (either their self-report or their report of the partner) was used.
- If both parents' data were missing, the value was set to missing (NA) and removed.

Importantly, in instances of discordant reporting - where, for example, a parent denied perpetrating abuse (e.g., mother-reported "no"), but their partner reported that abuse had occurred (e.g., partner-reported "yes" about the mother) - the report indicating abuse was prioritised. This decision was made to reduce the likelihood of under-reporting due to social desirability bias or intentional minimisation of abusive behaviour by the perpetrator.

This strategy allowed for more robust identification of abuse by integrating cross-informant validation, while also minimizing data loss due to non-response or disagreement.

3. Constructing Composite ACE Scores

As there were binary outcomes for each time point that an ACE was measured, a cumulative ACE score for each ACE type was calculated by summing the ACE-indicators from 8 months to 11 years. When constructing cumulative ACE scores for clusters of ACEs, the cumulative ACE scores for each ACE type were summed. Sexual abuse was only included in cumulative ACE scores, rather than analysed individually, due to very low frequencies in the sample.

Therefore, Abuse-related ACEs were calculated by summing the cumulative ACE scores for EA, PA and SA. Neglect and household dysfunction-related ACEs were calculated by summing the cumulative ACE scores for EN, PN, PSA, PMI, IPV, convictions and divorces.

Importantly, the variables recorded at age 9 reflect adversity occurring “over the last 3 years”, effectively capturing exposure between ages 7 to 9. Therefore, this developmental window is treated as a single time frame, rather than three separate yearly scores.

4. Variable Notes

To streamline the analyses and account for overlapping constructs, some indicators were grouped:

- Parental drug use included cannabis, amphetamines, heroin/cocaine, and alcohol-related variables.
- Parental mental health encompassed antidepressant use, suicide attempts, schizophrenia, anorexia, and bulimia.
- Each domain was scored as 1 if any component was present.

Appendix 2: Chapter 2 Supplementary Information

To understand how the relationship between ACEs and DPDR change according to the inclusion of covariates, undertaken analyses controlling initially for (1) sex (our most basic model), and then (2) inclusion of early life attachment style, and then (3), depression, anxiety and perseverative cognitions (PC), which are demonstrated fully in the main thesis.

1. Adjustment for sex in the association between ACEs and DPDRUnlike other models whereby the inclusion of covariates was to observe how it affected the relationship between ACEs and DPDR, the inclusion of sex was only to control for sex differences that may emerge in the perpetration of ACEs, which was not the focus of this thesis. Nevertheless, the results demonstrated that sex was not a strong predictor of DPDR, and did not predict DP or DR in any model ($p < .05$).

Chapter 2 Supplementary Table 1: Relationships of Emotional Abuse, Physical Abuse, Emotional Neglect and Physical Neglect on the odds of experiencing DP and DR, when controlling for sex

		Depersonalisation			Derealisation		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
Emotional Abuse	12	0.92	0.62 – 1.36	.676	1.05	0.73 – 1.52	.801
	17	2.39	1.49 – 3.81	**<.001**	1.32	0.85 – 2.05	.211
	24	1.95	1.16 – 3.25	**.012**	0.81	0.43 – 1.54	.522
Physical Abuse	12	0.76	0.27 – 2.14	.599	1.23	0.59 – 2.61	.579
	17	3.32	0.93 – 11.94	.065	0.69	0.21 – 2.20	.530
	24	1.68	0.34 – 8.42	.526	1.19	0.39 – 3.60	.767
Emotional Neglect	12	0.90	0.41 – 1.97	.782	0.88	0.38 – 2.01	.763
	17	5.87	1.86 – 18.54	**.003**	1.46	0.53 – 3.98	.464
	24	10.49	3.16 – 34.81	**<.001**	1.68	0.56 – 5.05	.355
Physical Neglect	12	1.00	0.19 – 5.21	.999	1.23	0.23 – 6.55	.810
	17	1.36	0.07 – 25.03	.833	0.20	0.01 – 3.10	.252
	24	6.55	0.46 – 91.84	.164	4.48	0.51 – 39.25	.176

Chapter 2 Supplementary Table 2: Relationships of Parental Substance Abuse, Inter-parental Violence, Parental Mental Illness, Parental Divorce and Parental Convictions on the odds of experiencing DP and DR, when controlling for sex

		Depersonalisation			Derealisation		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
PSA	12	1.06	0.87 – 1.28	.576	0.99	0.80 – 1.22	.916
	17	0.86	0.63 – 1.17	.341	1.19	0.93 – 1.52	.159
	24	1.75	1.30 – 2.39	**<.001**	1.43	1.09 – 1.90	**.010**

IPV	12	0.79	0.47 – 1.34	.381	0.85	0.51 – 1.43	.553
	17	3.60	1.95 – 6.62	**<.001**	1.70	0.94 – 3.03	.077
	24	1.97	0.88 – 4.44	.099	0.92	0.43 – 1.99	.844
PMI	12	0.95	0.71 – 1.28	.750	1.11	0.84 – 1.46	.468
	17	1.51	1.01 – 2.25	**.043**	0.97	0.68 – 1.38	.860
	24	1.46	0.92 – 2.29	.102	0.53	0.30 – 0.94	**.030**
Parental Divorce	12	0.89	0.47 – 1.67	.706	1.05	0.54 – 2.01	.889
	17	2.72	1.07 – 6.96	**.036**	0.25	0.09 – 0.72	**.010**
	24	3.60	1.25 – 10.28	**.018**	0.42	0.12 – 1.48	.175
Parental Conviction	12	0.82	0.25 – 2.64	.733	0.86	0.27 – 2.75	.798
	17	0.24	0.02 – 2.75	.252	0.43	0.08 – 2.27	.318
	24	0.70	0.04 – 11.02	.798	3.67	0.89 – 15.18	.073

Chapter 2 Supplementary Table 3: Relationships of Abuse-ACEs and Neglect and Household Dysfunction-ACEs on the odds of experiencing DP and DR, when controlling for sex

		Depersonalisation			**Derealisation**		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
Abuse ACEs	12	0.83	0.57 – 1.21	.335	1.08	0.80 – 1.46	.630
	17	2.09	1.36 – 3.22	**<.001**	1.03	0.68 – 1.55	.883
	24	1.67	1.04 – 2.67	**.034**	0.92	0.55 – 1.51	.728
Neglect & Household Dysfunction ACEs	12	1.01	0.90 – 1.15	.832	1.03	0.91 – 1.16	.626
	17	1.26	1.04 – 1.54	**.019**	1.07	0.92 – 1.26	.352
	24	1.43	1.15 – 1.79	**.001**	1.05	0.86 – 1.28	.613

2. Adjustment for early life attachment style on the relationship between ACEs and DPDR

The results demonstrated that, in all models, early life attachment was did not significantly mediate the relationship between ACEs, DP and DR, not did it independently demonstrate a significant relationship to DP or DR at any age ($p > .05$).

Chapter 2 Supplementary Table 4: Relationships of Emotional Abuse, Physical Abuse, Emotional Neglect and Physical Neglect on the odds of experiencing DP and DR, when controlling for sex and attachment

		Depersonalisation			**Derealisation**		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
Emotional Abuse	12	0.92	0.62 – 1.36	.681	1.05	0.73 – 1.52	.800
	17	2.39	1.49 – 3.82	**<.001**	1.32	0.85 – 2.05	.211
	24	1.95	1.16 – 3.25	**.012**	0.81	0.43 – 1.54	.522

Physical Abuse	12	0.76	0.27 – 2.14	.600	1.23	0.59 – 2.61	.579
	17	3.32	0.92 – 11.94	.065	0.69	0.21 – 2.20	.530
	24	1.68	0.34 – 8.42	.527	1.19	0.39 – 3.60	.768
Emotional Neglect	12	0.90	0.41 – 1.97	.787	0.88	0.38 – 2.01	.765
	17	5.87	1.86 – 18.36	**.003**	1.46	0.54 – 3.98	.464
	24	10.49	3.16 – 34.81	**<.001**	1.68	0.56 – 5.05	.354
Physical Neglect	12	1.00	0.19 – 5.21	.998	1.22	0.23 – 6.55	.811
	17	1.36	0.07 – 25.03	.834	0.20	0.01 – 3.10	.252
	24	6.55	0.47 – 91.84	.164	4.48	0.51 – 39.25	.175

Chapter 2 Supplementary Table 5: Relationships of Parental Substance Abuse, Inter-parental Violence, Parental Mental Illness, Parental Divorce and Parental Convictions on the odds of experiencing DP and DR, when controlling for sex and attachment

		Depersonalisation			**Derealisation**		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
PSA	12	1.05	0.87 – 1.28	.588	0.99	0.80 – 1.22	.914
	17	0.86	0.63 – 1.17	.340	1.19	0.93 – 1.52	.159
	24	1.75	1.30 – 2.39	**<.001**	1.43	1.09 – 1.90	**.010**
IPV	12	0.80	0.47 – 1.34	.387	0.85	0.51 – 1.43	.555
	17	3.60	1.95 – 6.62	**<.001**	1.70	0.94 – 3.04	.077
	24	1.97	0.88 – 4.44	.099	0.92	0.43 – 1.99	.844
PMI	12	0.95	0.71 – 1.28	.747	1.11	0.84 – 1.46	.468
	17	1.51	1.01 – 2.25	**.043**	0.97	0.68 – 1.38	.860
	24	1.46	0.93 – 2.29	.102	0.53	0.30 – 0.94	**.030**
Parental Divorce	12	0.89	0.47 – 1.67	.708	1.05	0.54 – 2.01	.888
	17	2.72	1.07 – 6.96	**.036**	0.25	0.09 – 0.72	**.010**
	24	3.56	1.25 – 10.28	**.018**	0.42	0.12 – 1.48	.175
Parental Conviction	12	0.81	0.25 – 2.64	.728	0.86	0.27 – 2.75	.797
	17	0.24	0.02 – 2.75	.252	0.43	0.08 – 2.27	.318
	24	0.70	0.04 – 11.02	.798	3.67	0.89 – 15.18	.073

Chapter 2 Supplementary Table 6: Relationships of Abuse-ACEs and Neglect and Household Dysfunction-ACEs on the odds of experiencing DP and DR, when controlling for sex and attachment

		Depersonalisation			**Derealisation**		
ACE	Age	OR	95% CI	P value	OR	95% CI	P-value
Abuse ACEs	12	0.83	0.57 – 1.21	.337	1.07	0.80 – 1.46	.629
	17	2.10	1.36 – 3.22	**<.001**	1.03	0.68 – 1.55	.884
	24	1.67	1.04 – 2.67	**.034**	0.91	0.55 - 1.51	.727
	12	1.01	0.90 – 1.15	.837	1.03	0.91 – 1.16	.628
	17	1.26	1.04 – 1.54	**.019**	1.07	0.92 – 1.26	.352

Neglect & Household Dysfunction ACEs	24	1.43	1.15 – 1.79	**.001**	1.05	0.86 – 1.28	.612

Appendix 3: Chapter 3 Supplementary Information

To address research questions 5–8, preliminary analyses was first conducted to establish the relationships between (1) each ACE type and the inflammatory markers, (2) each ACE type and DPDR, and (3) the inflammatory markers and DPDR (4 are in the main thesis). While the associations between ACE types and DPDR were already examined in Chapter 2, the analyses in Chapter 3 required smaller subsamples, as biological measures were not available for all participants. Further, the number of available samples for IL-6 was smaller than for CRP (as discussed in section 3.2.4 Model Building in Chapter 3), necessitating that the analyses be carried out in separate datasets.

Because IL-6 and CRP were measured at age 9, our previously defined ACE measures (spanning 8 months to 11 years) could not be used. Instead, ACE exposure was recalculated up to age 9, meaning that the number of possible years of exposure differed from earlier analyses.

1. ACEs as predictors of inflammatory markers

Our supplementary analyses indicated that witnessing inter-parental violence (IPV), parental mental illness (PMI), parental divorce, and neglect-related ACEs (measured between 8 months and 9 years) were significantly associated with elevated IL-6 levels (see Supplementary Table 2). No other ACEs showed significant associations with IL-6 at this age.

For CRP, only emotional neglect (EN) at age 24 and parental substance abuse (PSA) at baseline predicted elevated CRP levels (see Supplementary Table 1).

All analyses included sex, social position, and ethnicity as covariates. While not of direct interest to our research questions, these results are reported briefly below for transparency.

The longitudinal effects of ACE types on CRP are demonstrated in table Supplementary Table 1.

The impact of covariates for all models in both datasets are listed below.

In the EA → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, $p < .001$) and social position (OR = 1.03, 95% CI: 1.01–1.05, $p = .007$) significantly predicted CRP, but ethnicity did not ($p > .05$). In the EA → IL-6 model, sex (B = 0.21, 95% CI: 0.09–0.33, $p < .001$) significantly predicted IL-6, but social position and ethnicity did not ($p > .05$).

In the PA → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, $p < .001$) and social position (OR = 1.03, 95% CI: 1.01–1.05, $p = .007$) significantly predicted CRP, but ethnicity did not ($p > .05$). In the PA → IL-6 model, sex (B = 0.21, 95% CI: 0.09–0.32, $p < .001$) significantly predicted IL-6, but social position and ethnicity did not ($p > .05$).

In the EN → CRP model, sex (OR = 1.32, 95% CI: 1.22–1.42, p < .001) and social position (OR = 1.04, 95% CI: 1.01–1.07, p = .005) significantly predicted CRP, but ethnicity did not (p > .05). In the EN → IL-6 model, sex (B = 0.20, 95% CI: 0.02–0.38, p = .034) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the PN → CRP model, sex (OR = 1.39, 95% CI: 1.28–1.49, p < .001) and social position (OR = 1.03, 95% CI: 1.00–1.06, p = .025) significantly predicted CRP, but ethnicity did not (p > .05). In the PN → IL-6 model, sex (B = 0.23, 95% CI: 0.06–0.40, p = .007) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the PSA → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.00–1.05, p = .006) significantly predicted CRP, but ethnicity did not (p > .05). In the PSA → IL-6 model, sex (B = 0.13, 95% CI: 0.08–0.19, p < .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the PMI → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.00–1.05, p = .007) significantly predicted CRP, but ethnicity did not (p > .05). In the PMI → IL-6 model, sex (B = 0.15, 95% CI: 0.04–0.25, p = .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the IPV → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.01–1.05, p = .008) significantly predicted CRP, but ethnicity did not (p > .05). In the IPV → IL-6 model, sex (B = 0.21, 95% CI: 0.09–0.33, p = .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the Conviction → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.01–1.05, p = .007) significantly predicted CRP, but ethnicity did not (p > .05). In the Conviction → IL-6 model, sex (B = 0.21, 95% CI: 0.09–0.32, p < .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the Divorce → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.01–1.05, p = .007) significantly predicted CRP, but ethnicity did not (p > .05). In the Divorce → IL-6 model, sex (B = 0.20, 95% CI: 0.09–0.32, p < .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the Neglect and Household Dysfunction-related ACEs → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.01–1.05, p = .005) significantly predicted CRP, but ethnicity did not (p > .05). In the Neglect-ACEs → IL-6 model, sex (B = 0.21, 95% CI: 0.09–0.33, p < .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

In the Abuse-related ACEs → CRP model, sex (OR = 1.35, 95% CI: 1.28–1.43, p < .001) and social position (OR = 1.03, 95% CI: 1.01–1.05, p = .007) significantly predicted CRP, but ethnicity did not (p > .05). In the Abuse-ACEs → IL-6 model, sex (B = 0.21, 95% CI: 0.09–0.33, p < .001) significantly predicted IL-6, but social position and ethnicity did not (p > .05).

2. ACEs as predictors of DPDR

Our supplementary analysis indicated that, when controlling for sex, social position, and ethnicity, emotional abuse (EA) predicted increased odds of DP at ages 17 and 24 in the hsCRP dataset, but not in the IL-6 dataset. However, in the IL-6 dataset, EA predicted lower odds of DR at age 24, but no relationship was identified in the CRP dataset. Sex, ethnicity and social position were not significant (p > .05).

Further, physical abuse (PA) predicted increased odds of DP at ages 17 and 24 in the hsCRP dataset, but only age 24 in the IL-6 dataset. PA did not predict changing odds of DR in either dataset. Sex, ethnicity and social position were not significant (p > .05).

Physical neglect (PN) did not predict changing odds of DP or DR at any age in either dataset. Sex, ethnicity and social position were not significant (p > .05).

EN predicted increased odds of DP at ages 17 and 24 in both datasets, and did not predict changing odds of DR. Sex, ethnicity and social position were not significant (p > .05).

Similarly, PSA predicted increased odds of DP at age 24 in both datasets, but did not predict changing odds of DR. Sex, ethnicity and social position were not significant (p > .05).

PMI predicted increased odds of DP at age 24 in the hsCRP dataset, but not in the IL-6 dataset. Conversely, PMI predicted decreased odds of DR at age 24 in the IL-6 dataste, but not in the hsCRP dataset. Sex, ethnicity and social position were not significant (p > .05).

Interparental-violence (IPV) predicted increased odds of both DP and DR at age 24 in both datasets. Sex, ethnicity and social position were not significant (p > .05).

Parental convictions predicted increased odds of DP at age 24 in the hsCRP dataset, but not in the IL-6 dataset. Parental convictions did not predict changing odds of DR in either dataset. Sex, ethnicity and social position were not significant (p > .05).

Parental divorce predicted decreased odds of DR at age 17 in both datasets, but did not predict changing odds of DP in either. Sex, ethnicity and social position were not significant (p > .05).

Abuse-related ACEs predicted increased odds of DP at ages 17 and 24 in both datasets, but only predicted decreased odds of DR at age 24 in the IL-6 dataset. Sex, ethnicity and social position were not significant (p > .05).

Finally, Neglect and household dysfunction related ACEs predicted increased odds of DP at age 24 in both datasets, but did not predict changing odds of DR in either datasets. Sex, ethnicity and social position were not significant (p > .05).

Chapter 3 Supplementary Table 1: ACEs as Predictors of high-sensitivity C-reactive Protein, Depersonalisation and Derealisation (CRP Dataset)

		hsCRP			**Depersonalisation**			**Derealisation**		
		Gaussian (Scale outcomes)			**Logit (Binary outcomes)**			**Logit (Binary outcomes)**		
		B	*CI*	*P*	*OR*	*CI*	*P*	*OR*	*CI*	*P*
Emotional Abuse	12	0.01	-0.04 – 0.06	.623	0.72	0.41 - 1.27	.257	1.19	0.79 - 1.79	.421
	17	0.03	-0.04 – 0.10	.439	2.12	1.03 - 4.35	**.042***	0.83	0.42 - 1.62	.574
	24	-0.00	-0.07 – 0.07	.982	2.69	1.51 - 4.86	**<.001*****	0.59	0.26 – 1.31	.195
Physical Abuse	12	0.08	-0.01 – 0.18	.076	0.42	0.11 – 1.60	.203	1.31	0.62 - 2.75	.483
	17	-0.02	-0.15 – 0.12	.804	4.53	1.19 - 17.29	**.027***	0.50	0.14 - 1.77	.285
	24	-0.12	-0.26 – 0.02	.104	5.26	1.11 – 25.03	**.037***	0.55	0.16 - 1.92	.345
Physical Neglect	12	-0.13	-0.39 – 0.14	.354	1.08	0.12 – 9.39	.946	0.28	0.01 – 6.30	.423
	17	0.28	-0.10– 0.65	.145	4.71	0.20 – 111.05	.338	0.58	0.02 – 20.91	.763
	24	0.06	-0.34 – 0.46	.768	5.26	0.35 – 79.04	.231	23.34	1.23 – 441.42	**.035**
Emotional Neglect	12	0.04	-0.09 – 0.16	.564	0.88	0.33 – 2.36	.800	1.11	0.41 – 3.00	.837
	17	0.10	-0.06 – 0.27	.208	6.43	1.57 – 26.58	**.010***	0.83	0.21 – 3.25	.790
	24	-0.17	-0.33 - -0.01	**.038***	9.74	2.61 – 36.23	**<.001*****	1.34	0.39 – 4.57	.642
Parental Substance Abuse	12	-0.03	-0.06 – 0.01	**.023***	1.07	0.84 – 1.35	.576	1.00	0.77 – 1.30	.996
	17	0.02	-0.03 – 0.06	.451	1.05	0.76 – 1.45	.764	1.12	0.94 – 1.51	.448
	24	-0.00	-0.05 – 0.04	.872	1.36	1.05 – 1.79	**.021***	1.27	0.96 – 1.67	.093
Parental Mental Illness	12	-0.02	-0.05 – 0.01	.174	0.95	0.71 – 1.28	.730	1.12	0.84 – 1.46	.442
	17	0.05	0.00 – 0.09	.060	1.44	0.94 – 2.23	.094	1.13	0.76 – 1.65	.554
	24	0.04	0.01 – 0.08	.104	1.63	1.14 – 2.34	**.008****	0.81	0.53 – 1.23	.319
Inter-parental Violence	12	0.00	-0.05 – 0.06	.881	0.68	0.34 – 1.38	.284	0.59	0.28 – 1.22	.158
	17	0.01	-0.08 – 0.09	.860	1.90	0.84 – 4.31	.126	2.12	0.97 – 4.57	.058
	24	-0.01	-0.09 – 0.07	.852	2.97	1.35 – 6.55	**.007****	2.51	1.16 – 3.37	**.019***
Conviction	12	0.03	-0.11 – 1.68	.675	0.41	0.07 – 2.54	.335	1.41	0.43 – 4.62	.581
	17	0.05	-0.15 – 2.41	.643	2.23	0.13 – 38.86	.583	0.34	0.05 – 2.41	.278
	24	-0.19	-0.39 - -7.72	.050	9.87	1.43 – 68.03	**.020***	0.95	0.18 – 5.05	.953
Divorce	12	-0.00	-0.08 – 0.08	.975	0.93	0.42 – 2.08	.864	1.32	0.64 – 2.75	.454

	17	-0.02	-0.14 – 0.11	.777	1.01	0.20 – 5.05	.993	0.12	0.03 – 0.59	**.009****
	24	0.01	-0.11 – 0.14	.837	2.46	0.71 – 8.50	.155	0.19	0.04 – 1.02	.053
Abuse-ACEs	12	0.02	-0.02 – 0.06	.287	0.68	0.42 – 1.11	.118	1.15	0.85 – 1.54	.363
	17	0.01	-0.04 – 0.07	.661	2.08	1.20 – 3.60	**.010***	0.77	0.45 – 1.31	.342
	24	-0.02	-0.07 – 0.04	.507	2.39	1.45 – 3.94	**<.001*****	0.66	0.37 – 1.16	.153
Neglect-ACEs	12	-0.02	-0.03 – 0.00	.058	0.98	0.84 – 1.15	.846	1.02	0.88 – 1.20	.749
	17	0.02	-0.01 – 0.04	.130	1.22	1.00 – 1.49	.055	1.07	0.89 – 1.31	.470
	24	0.00	-0.02 – 0.03	.912	1.49	1.25 – 1.79	**<.001*****	1.13	0.93 – 1.35	.210

Chapter 3 Supplementary Table 2: ACEs as Predictors of Interleukin-6, Depersonalisation and Derealisation (IL-6 Dataset)

	Interleukin-6 (Cross-sectional)				**Depersonalisation**			**Derealisation**		
	Gaussian (Scale outcomes)				**Logit (Binary outcomes)**			**Logit (Binary outcomes)**		
	B	*CI*	*P*	Ages	*OR*	*CI*	*P*	*OR*	*CI*	*P*
Emotional Abuse	0.09	-0.05 – 0.22	.206	12	0.91	0.55 – 1.51	.718	1.22	0.80 – 1.86	.356
				17	1.75	0.95 – 3.22	.071	0.81	0.45 – 1.45	.483
				24	1.77	0.99 – 3.16	.053	0.39	0.17 – 0.91	**.028***
Physical Abuse	0.20	-0.01 – 0.41	.061	12	0.45	0.13 – 1.51	.192	1.70	0.89 – 3.25	.112
				17	3.25	0.96 – 11.02	.058	0.45	0.19 – 1.09	.077
				24	5.00	1.42 – 17.64	**.012***	0.53	0.21 – 1.30	.162
Physical Neglect	-0.01	-0.43– 0.40	.959	12	1.47	0.20 – 10.70	.701	0.61	0.05 – 7.24	.693
				17	2.82	0.17 – 46.99	.470	0.48	0.03 – 9.12	.622
				24	2.18	0.18 – 26.31	.541	10.84	1.00 – 116.75	.050
Emotional Neglect	0.06	-0.14 – 0.26	.541	12	0.92	0.36 – 2.32	.861	0.89	0.33 – 2.41	.822
				17	4.80	1.36 – 16.95	**.015***	2.21	0.69 – 7.10	.183
				24	6.19	1.82 – 21.12	**.004****	1.29	0.40 – 4.18	.678
Parental Substance Abuse	0.04	-0.06 – 0.14	.401	12	1.05	0.84 – 1.34	.662	0.97	0.76 – 1.26	.842
				17	1.12	0.82 – 1.52	.478	0.99	1.68 – 1.28	.063
				24	1.34	1.02 – 1.75	**.035***	1.27	0.97 – 1.65	.078
Parental Mental Illness	0.15	0.04 – 0.25	**.005****	12	0.91	0.63 – 1.32	.626	1.25	0.92 – 1.70	.154
				17	1.62	1.00 – 2.61	.051	0.95	0.64 – 1.42	.823
				24	1.55	1.00 – 2.41	.052	0.58	0.37 – 0.92	**.021***
Inter-parental Violence	0.24	0.09 – 0.38	**.002****	12	0.64	0.31 – 1.31	.220	0.71	0.36 – 1.35	.291
				17	1.34	0.58 – 3.03	.495	1.65	0.82 – 3.32	.163
				24	3.07	1.42 – 6.62	**.004****	2.36	1.23 – 4.53	**.010***
Conviction	0.07	-0.19 – 0.33	.606	12	0.94	0.23 – 3.90	.930	1.99	0.63 – 6.30	.244
				17	0.24	0.02 – 3.35	.288	0.09	0.01 – 1.00	.050
				24	1.72	0.17 – 16.78	.645	0.11	0.01 – 1.16	.067
Divorce	0.20	0.01 – 0.38	**.040***	12	0.80	0.30 – 2.12	.657	1.02	0.42 – 2.51	.970

				17	1.25	0.25 – 6.30	.789	0.16	0.03 – 0.84	**.031***
				24	0.05	2.80 – 0.37	.334	0.44	0.08 – 2.44	.346
Abuse-ACEs	0.09	-0.03 – 0.20	.141	12	0.84	0.55 – 1.26	.387	1.21	0.91 – 1.60	.188
				17	1.63	1.01 – 2.61	**.044***	0.76	0.51 – 1.14	.186
				24	1.75	1.12 – 2.75	**.015***	0.60	0.37 – 0.98	**.041***
Neglect-ACEs	0.12	0.05 – 0.19	**<.001*****	12	0.98	0.83 – 1.16	.789	1.05	0.89 – 1.23	.576
				17	1.19	0.95 – 1.48	.122	1.11	0.91 – 1.34	.293
				24	1.36	1.12 – 1.65	**.002****	1.06	0.88 – 1.27	.557

Appendix 4: Chapter 5 Supplementary Information

This supplementary section provides additional analyses examining the associations between specific ACEs and DPDR symptom severity. While the main text of Chapter 5 presents the core findings, these supplementary results offer further transparency and detail on individual ACE types that were not the primary focus of the chapter.

For each ACE type, hierarchical regression models were conducted, adjusting for sex at birth and the presence of non-dissociative mental health diagnoses in baseline models. Where relevant, cognitive mechanisms such as general rumination and hyper-reflexivity were entered in subsequent steps to assess their potential contribution to variance in DPDR scores.

The following sections present results for selected ACE types, including (1) death of a friend or family member, and (2) physical abuse or assault. Full regression tables are provided for each model to allow readers to evaluate the strength, direction, and significance of these associations, as well as the incremental variance explained at each modelling step.

1. Death of Friend or Family

The baseline model indicated that ACE death of a family member/friend was not a significant predictor of the dependent variable. Similarly, sex at birth and having a mental health diagnosis other than dissociation were not significantly associated with the outcome. The overall model was not statistically significant, indicating that the predictors did not meaningfully explain variation in DPDR scores. See table X below for statistics.

Chapter 5 Supplementary Table 1: Hierarchical Linear Regression Examining the Association Between Death of a Family Member and/or Friend and DPDR Symptom Severity

Predictor	β	Std. Error	p-value
Death of a Family Member/Friend	5.98	4.72	0.208
Sex at Birth	-8.82	12.98	0.498
Non-dissociative psychopathology	8.05	11.78	0.496

Δ R^2 = -0.006 for Step 1 (p = .510)

2. Parental Divorce or Separation

The baseline model, adjusted for sex and non-dissociative psychopathology indicated that physical abuse was not a significant predictor of DPDR (p = 0.098), and for each occurrence of physical abuse, there was a 5.5-point increase in DPDR scores. The overall model was not statistically significant (p = .324), explaining 0.4% of the variance.

Chapter 5 Supplementary Table 3: Hierarchical Linear Regression Examining the Association Between Physical Abuse/Assault Events and DPDR Symptom Severity

Predictor	B	Std. Error	p-value
Physical Abuse/Assault Events	5.53	3.32	0.098
Sex at Birth	-7.84	12.89	0.544
Non-dissociative psychopathology	6.74	11.73	0.566

Δ R^2 = .004 for Step 1 (p = .324)

Appendix 5: Chapter 5 Survey

Depersonalisation Scoping Study - University of Essex

Survey Flow

Standard: Introduction (4 Questions)
Standard: Demographics (7 Questions)
Block: Derealisation and Depersonalisation (2 Questions)

EmbeddedData
Score = ${gr://SC_4Oq5cUWVmzKQPLE/Score}

Branch: New Branch
If
If Score Is Less Than 70

Block: Thinking Style (1 Question)
Block: Childhood Experiences (18 Questions)
Block: End of Survey (4 Questions)

EndSurvey: Advanced

Standard: Childhood Experiences (18 Questions)
Standard: Thinking Style (1 Question)
Standard: Thinking Style (1 Question)
Standard: Potential Triggers (15 Questions)
Standard: Health Information (5 Questions)
Standard: Potential Calming Activities (12 Questions)
Standard: Personal Experience (1 Question)
Standard: End of Survey (4 Questions)

EndSurvey:

Page Break

Start of Block: Introduction

Q90 Investigating Experiences of Depersonalisation and Derealisation

Q39 If you are a young person aged between 18 and 30 years, I would like to invite you to participate in this study. You should only participate if you want to; choosing not to take part will not disadvantage you in any way. Before you decide whether you want to take part, it is important for you to read the participant information sheet and discuss its contents with others if you wish. Email Evelyn Dilkes on ed21531@essex.ac.uk if there is anything that is not clear or you would like more information. This survey is about depersonalisation and derealisation and some experiences that can be associated with it. Depersonalisation is the experience of feeling detached from your mind or your body, and/or feeling as though you are watching yourself from the outside. Derealisation is the feeling of the world around you being unreal and/or feeling detached from your surroundings. In this survey you will be presented with statements that are sometimes experienced when someone is going through depersonalisation and/or derealisation and you will be asked to indicate how frequently you identify with the statement, and, if you do experience it, you will be asked how long the experience lasts. This survey will ask you about the following areas:

- Demographic information (age, where you live, ethnicity, gender, social position, sexual orientation)
- Adverse childhood experiences
- Depersonalisation and derealisation
- Thinking styles
- Potential triggers of depersonalisation and derealisation
- Information about your health
- Potential calming activities to reduce depersonalisation and derealisation
- The option to provide us with more information about your experiences of depersonalisation and derealisation, which is optional.

At the end of the survey, you will be given the option to provide your name and email address, which will be used to contact you if you win a £20 Amazon voucher, and also you will be able to opt-in to be contacted to participate in a lab experiment where you would be paid £50.

This questionnaire should take between 20-30 minutes.

Page Break

Q91 Statement of consent

By completing this online study you are consenting to the following:

I am 18 years or older

I agree to participate in the research project 'Investigating Experiences of Depersonalisation and Derealisation' being carried out by Evelyn Dilkes

This agreement has been given voluntarily and without coercion

I have been given full information about the study and contact details of the researchers

I have read and understood the information provided above

Risk statement:

I agree to have my anonymised data shared on publicly accessible repositories

I agree to be contacted in the future by the researchers

I have had the opportunity to ask questions about the research and my participation in it

By selecting 'Agree' below, you are acknowledging the above and are happy to participate within this study.

Q92 I agree to these terms:

- ○ Agree (1)
- ○ Disagree (2)

Skip To: End of Survey If Q92 = 2

End of Block: Introduction

Start of Block: Demographics

Q76 Age

How old are you? (1)

▼ 18 (1) ... 30 (13)

Q77 Ethnicity

What is your ethnicity? (1)

▼ Asian or Asian British (Indian, Pakistani, Bangladeshi, Chinese, Any other Asian background) (1) ... Other ethnic group (Arab, Any other ethnic group) (5)

Q78 Gender

What is your gender identity? (1)

▼ Female (1) ... Different identity (5)

Q82 Sex at birth

What sex were you assigned at birth? (1)

▼ Male (1) ... Female (2)

Q80 Social position

What was the occupation of your main household earner when you were about aged 14? (1)

▼ Modern professional such as: teacher, nurse, physiotherapist, social worker, musician, police officer (sergeant or above), software designer. (1) ... I prefer not to say (13)

Q81 Sexual orientation

What is your sexual orientation? (1)

▼ Heterosexual (1) ... Questioning (6)

Q74 Where do you live?

_____

End of Block: Demographics

Start of Block: Derealisation and Depersonalisation

Q1 PLEASE READ INSTRUCTIONS CAREFULLY: This questionnaire describes strange and 'funny' experiences that normal people may have in their daily life. We are interested in their: (a) frequency, i.e. how often you have had these experiences over the last six months and (b) their approximate duration (if you selected 'Never' in the frequency column, then select 'Not applicable' in the duration column). For each question, please select the answers that suit you best. If you are not sure, give your best guess.

Q2

	Frequency	Duration

Out of the blue, I feel strange, as if I were not real or as if I were cut off from the world. (1)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
What I see looks 'flat' or 'lifeless', as if I were looking at a picture. (2)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
Parts of my body feel as if they didn't belong to me. (3)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I have found myself not being frightened at all in situations which normally I would find frightening or distressing. (4)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
My favourite activities are no longer enjoyable. (5)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
Whilst doing something I have the feeling of being a "detached observer" of myself. (6)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
The flavour of meals no longer gives me a feeling of pleasure or distaste. (7)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
My body feels very light, as if it were floating on air. (8)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
When I weep or laugh, I do not seem to feel any emotions at all. (9)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I have the feeling of not having any thoughts at all, so that when I speak it feels as if my words were being uttered by an 'automaton'. (10)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
Familiar voices (including my own) sound remote and unreal. (11)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I have the feeling that my hands or my feet have become larger or smaller. (12)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
My surroundings feel detached or unreal, as if there was a veil between me and the outside world. (13)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
It seems as if things that I have recently done had taken place a long time ago. For example anything which I have done this morning feels as if it were done weeks ago. (14)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)

Whilst fully awake I have "visions" in which I can see myself outside, as if I were looking my image in a mirror. (15)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I feel detached from memories of things that have happened to me - as if I had not been involved in them. (16)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
When in a new situation, it feels as if I have been through it before. (17)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
Out of the blue, I find myself not feeling any affection towards my family and close friends. (18)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
Objects around me seem to look smaller or further away. (19)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I cannot feel properly the objects that I touch with my hands for, it feels as if it were not me who were touching it. (20)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I do not seem able to picture things in my mind, for example, the face of a close friend or a familiar place. (21)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
When a part of my body hurts, I feel so detached from the pain that if feels as if it were 'somebody else's pain.' (22)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I have the feeling of being outside my body. (23)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
When I move it doesn't feel as if I were in charge of the movements, so that I feel 'automatic' and mechanical as if I were a 'robot'. (24)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
The smell of things no longer gives me a feeling of pleasure or dislike. (25)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I feel so detached from my thoughts that they seem to have a 'life' of their own. (26)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I have to touch myself to make sure that I have a body or a real existence. (27)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)
I seem to have lost some bodily sensations (e.g. of hunger and thirst) so that when I eat or drink, it feels an automatic routine. (28)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)

Previously familiar places look unfamiliar, as if I had never seen them before. (29)	▼ Never (1 ... All the time (4)	▼ Few seconds (1 ... Not applicable (0)

End of Block: Derealisation and Depersonalisation

Start of Block: Thinking Style

Q123 For each of the items below, please rate how well the item describes you.

	1 - Not at all (1)	2 (2)	3 (3)	4 - Neutral (4)	5 (5)	6 (6)	7 - Very well (7)
I find that my mind goes over things again and again. (1)	○	○	○	○	○	○	○
When I have a problem, it will gnaw on my mind for a long time. (2)	○	○	○	○	○	○	○
I find that some thoughts come to my mind over and over throughout the day. (3)	○	○	○	○	○	○	○
I can't stop thinking about some things. (4)	○	○	○	○	○	○	○

End of Block: Thinking Style

Start of Block: Childhood Experiences

Q43 Prior to the age of 18, did you experience a death of a very close friend or family member?

- Yes (1)
- No (0)
- Prefer not to say (2)

Display this question:

If Q43 = 1

Q49 (OPTIONAL) How old were you? If you cannot remember specifically, give your best guess. If this happened at multiple ages, please select each age it happened.

- 0-5 years (1)
- 6-10 years (2)
- 11-15 years (3)
- 16-18 years (4)

Display this question:

If Q43 = 1

Q61 (OPTIONAL) How many times did this happen?

- One occasion (1)
- Two occasions (2)
- Three occasions (3)
- Four occasions (4)
- 5+ occasions (5)

Page Break

Q44 Prior to the age of 18, was there a major upheaval between your parents (such as divorce, separation)?

- Yes (1)
- No (0)
- Prefer not to say (2)

Display this question:

If Q44 = 1

Q56 (OPTIONAL) How old were you? If you cannot remember specifically, give your best guess. If this happened at multiple ages, please select each age it happened.

- [ ] 0-5 years (1)
- [ ] 6-10 years (2)
- [ ] 11-15 years (3)
- [ ] 16-18 years (4)

Display this question:

If Q44 = 1

Q62 (OPTIONAL) How many times did this happen?

- ○ One occasion (1)
- ○ Two occasions (2)
- ○ Three occasions (3)
- ○ Four occasions (4)
- ○ 5+ occasions (5)

X→

Q45 Prior to the age of 18, did you have a traumatic sexual experience (raped, molested, etc.)?

- ○ Yes (1)
- ○ No (0)
- ○ Prefer not to say (2)

Display this question:

If Q45 = 1

Q57 (OPTIONAL) How old were you? If you cannot remember specifically, give your best guess. If this happened at multiple ages, please select each age it happened.

- ☐ 0-5 years (1)
- ☐ 6-10 years (2)
- ☐ 11-15 years (3)
- ☐ 16-18 years (4)

Display this question:

If Q45 = 1

Q63 (OPTIONAL) How many times did this happen?

- ○ One occasion (1)
- ○ Two occasions (2)
- ○ Three occasions (3)
- ○ Four occasions (4)
- ○ 5+ occasions (5)

X→

Q46 Prior to the age of 18, were you the victim of violence (child abuse, mugged or assaulted -- other than sexual)?

- ○ Yes (1)
- ○ No (0)
- ○ Prefer not to say (2)

Display this question:

If Q46 = 1

Q58 (OPTIONAL) How old were you? If you cannot remember specifically, give your best guess. If this happened at multiple ages, please select each age it happened.

- ☐ 0-5 years (1)
- ☐ 6-10 years (2)
- ☐ 11-15 years (3)
- ☐ 16-18 years (4)

Display this question:

If Q46 = 1

Q64 (OPTIONAL) How many times did this happen?

- One occasion (1)
- Two occasions (2)
- Three occasions (3)
- Four occasions (4)
- 5+ occasions (5)

X→

Q47 Prior to the age of 18, were you extremely ill or injured?

- Yes (1)
- No (0)
- Prefer not to say (2)

Display this question:

If Q47 = 1

Q59 (OPTIONAL) How old were you? If you cannot remember specifically, give your best guess. If this happened at multiple ages, please select each age it happened.

- [ ] 0-5 years (1)
- [ ] 6-10 years (2)
- [ ] 11-15 years (3)
- [ ] 16-18 years (4)

Display this question:

If Q47 = 1

Q65 (OPTIONAL) How many times did this happen?

- ○ One occasion (1)
- ○ Two occasions (2)
- ○ Three occasions (3)
- ○ Four occasions (4)
- ○ 5+ occasions (5)

X→

Q48 Prior to the age of 18, did you experience any other major upheaval that you think may have shaped your life or personality significantly?

- ○ Yes (1)
- ○ No (0)
- ○ Prefer not to say (2)

Display this question:

If Q48 = 1

Q60 (OPTIONAL) How old were you? If you cannot remember specifically, give your best guess. If this happened at multiple ages, please select each age it happened.

- [ ] 0-5 years (1)
- [ ] 6-10 years (2)
- [ ] 11-15 years (3)
- [ ] 16-18 years (4)

Display this question:

If Q48 = 1

Q66 (OPTIONAL) How many times did this happen?

- ○ One occasion (1)
- ○ Two occasions (2)
- ○ Three occasions (3)
- ○ Four occasions (4)
- ○ 5+ occasions (5)

End of Block: Childhood Experiences

Start of Block: End of Survey

Q83 Thank you for your participation in this study.

You are reminded that by submitting a completed version of this study you are agreeing to participate in this research. If you would like to see the participant information sheet again, you can access it here: participant information sheet

If you have any queries, please contact me via email (ed21531@essex.ac.uk).

Q84 If you would like to be entered to win a £20 Amazon voucher, please leave your name and email address below so we can contact you (there are 5 up for grabs!)

_____

Q85 Would you like to opt-in to be contacted for participation in a lab study where you will be paid £50? To opt-in, please leave your email address below.

_____

Q147 Resources

Unreal UK is a charity reaching out to people with lived experiences of depersonalisation and derealisation. Unreal UK provide a page of helpful resources for people who experience depersonalisation and derealisation. You can access this page here: https://www.unrealuk.org/resources

No Panic is a UK charity helping people with experiences that commonly co-occur with depersonalisation and derealisation, such as panic and anxiety. You can find relevant resources here: https://nopanic.org.uk/unreality/. If you are experiencing distressing levels of anxiety or panic following this questionnaire, then you can call the No Panic helpline (0300 772 9844). The helpline is

run by trained volunteers who will help you to calm down and talk you through exercises to help you.

If you would prefer not to talk but want some mental health support, you could text SHOUT to 85258. Shout offers a confidential 24/7 text service providing support if you are in crisis and need immediate help.

To talk about anything that is upsetting you, you can contact Samaritans 24 hours a day, 365 days a year. You can call 116 123 (free from any phone), email jo@samaritans.org or visit some branches in person. You can also call the Samaritans Welsh Language Line on 0808 164 0123 (7pm–11pm every day).

End of Block: End of Survey

Start of Block: Thinking Style

X→

Q129 After experiencing something like derealisation (the feeling of the world around you being unreal and/or feeling detached from your surroundings) and/or depersonalisation (the experience of feeling detached from your mind or your body, and/or feeling as though you are watching yourself from the outside), people sometimes, but not always, find themselves having thoughts about their

experience even though they don't try to think about it. Indicate for the following items how often, if at all, you had the experiences described during the weeks immediately after the event.

	Not at all (0)	Rarely (1)	Sometimes (2)	Often (3)
I thought about the event when I did not mean to. (1)	○	○	○	○
Thoughts about the event came to mind and I could not stop thinking about them. (2)	○	○	○	○
Thoughts about the event distracted me or kept me from being able to concentrate. (3)	○	○	○	○
I could not keep images or thoughts about the event from entering my mind. (4)	○	○	○	○
Thoughts, memories, or images of the event came to mind even when I did not want them. (5)	○	○	○	○
Thoughts about the event caused me to relive my experience. (6)	○	○	○	○
Reminders of the event brought back thoughts about my experience. (7)	○	○	○	○
I found myself automatically thinking about what had happened. (8)	○	○	○	○
Other things kept leading me to think about my experience. (9)	○	○	○	○
I tried not to think about the event, but could not keep the thoughts from my mind. (10)	○	○	○	○

End of Block: Thinking Style

Start of Block: Potential Triggers

Q93 The next section encourages you to think about some of the things that you believe would trigger an experience of derealisation and/or depersonalisation. If you experience derealisation and/or depersonalisation chronically, think about whether these things would make your symptoms worse. This could be based on your past experiences, or you could imagine that if presented with it, it could trigger or make worse your derealisation and/or depersonalisation. We would also like to understand, if you did experience derealisation and/or depersonalisation or a change in severity of symptoms, how distressed you might feel in that scenario, and how likely (if at all) you would be to avoid those types of situations in the future. As a reminder, derealisation is the feeling of the world around you being unreal and/or feeling detached from your surroundings and depersonalisation is the experience of feeling detached from your mind or your body, and/or feeling as though you are watching yourself from the outside. Please be reminded that you have the right to withdraw and stop this questionnaire, especially if you become distressed.

Q113 Stressful, overwhelming situations. For example, intense anxiety before giving a presentation.

	0 10 20 30 40 50 60 70 80 90 100
Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

Q114 Philosophical or existential thoughts. For example, thinking about the meaning of life, whether humans have free will, where do we go when we die?

0 10 20 30 40 50 60 70 80 90 100

Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

Q112 Your reflection. For example, you look in the mirror as usual and you realise that you are the person staring back at you.

0 10 20 30 40 50 60 70 80 90 100

Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

Q111 Multiple mirrors within a confined space. For example, you enter a dressing room and there are large mirrors on every wall and the door.

0 10 20 30 40 50 60 70 80 90 100

Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

Q110 Staring at your yourself for a long period. For example, you are looking in the mirror, fixing your hair for an event, and you have been looking at yourself for 5-10 minutes straight.

	0 10 20 30 40 50 60 70 80 90 100
Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

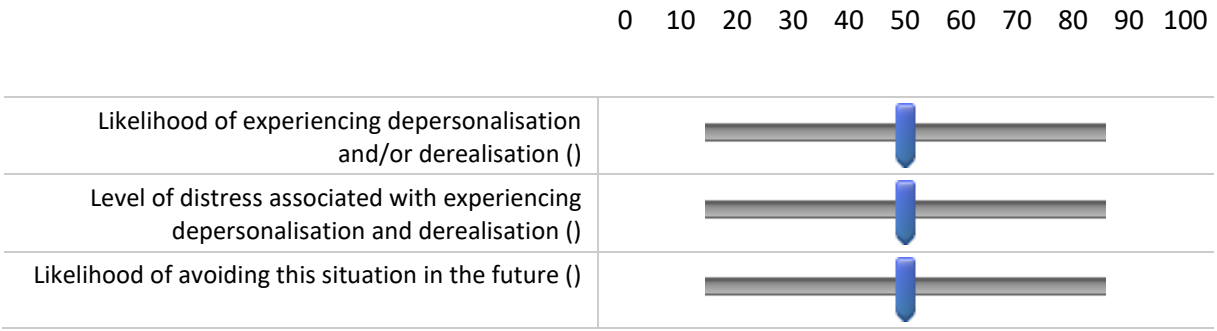
Q109 Intense fluorescent lights. For example, lighting within a hospital or supermarket.

	0 10 20 30 40 50 60 70 80 90 100
Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

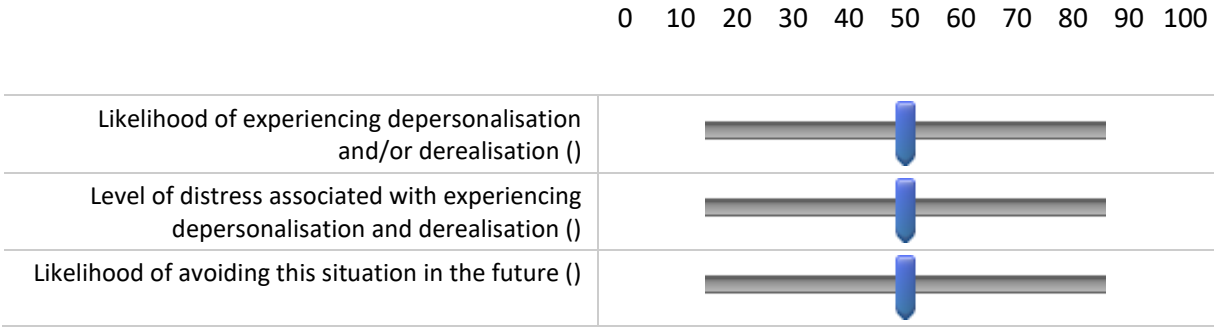
Q115 Bright flashing lights. For example, a strobe light in a dark room.

	0 10 20 30 40 50 60 70 80 90 100
Likelihood of experiencing depersonalisation and/or derealisation ()	
Level of distress associated with experiencing depersonalisation and derealisation ()	
Likelihood of avoiding this situation in the future ()	

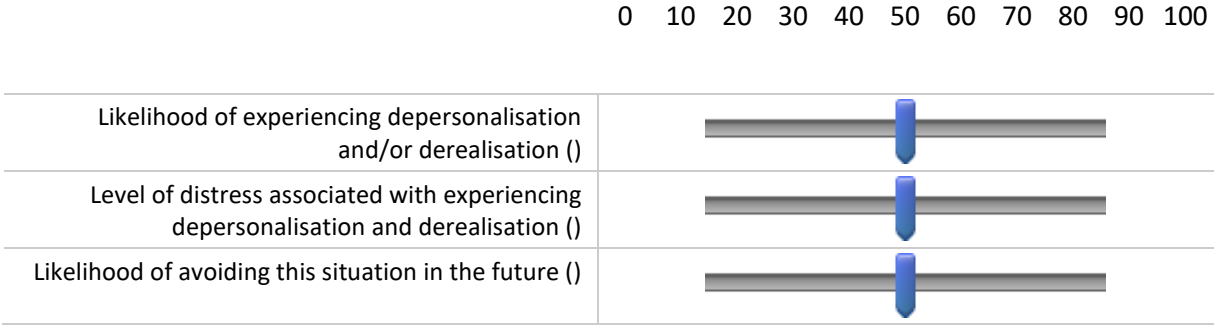
Q116 Looking at your hands. For example, you look at your hands and think about the fact that they belong to you.



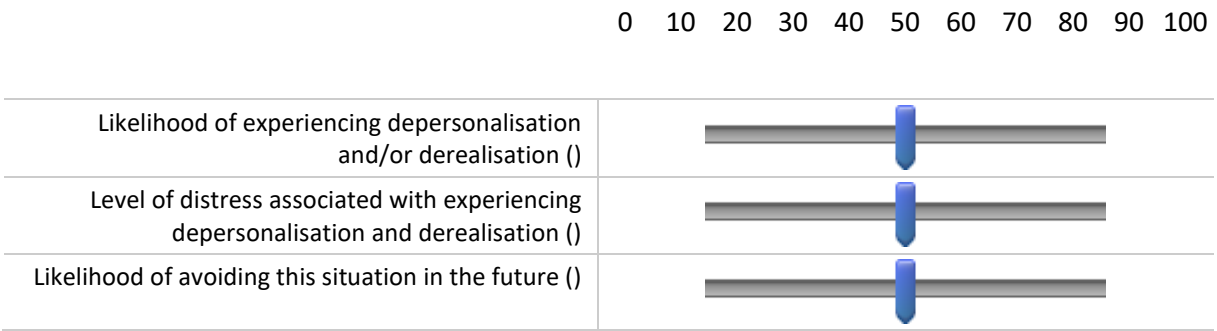
Q117 Thinking about a past episode of feeling depersonalisation and/or derealisation.



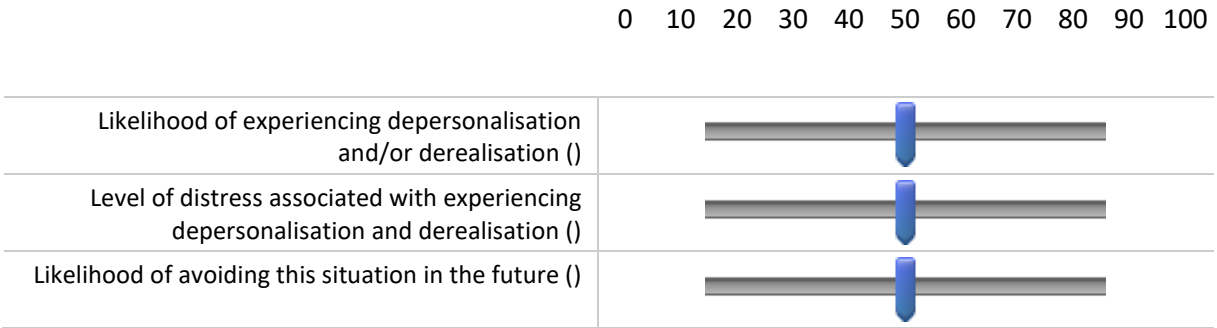
Q118 Staring at a screen for extended periods. For example, you have been looking at your computer or laptop screen for a couple of hours and you quickly look away at something else and realise you have become detached.



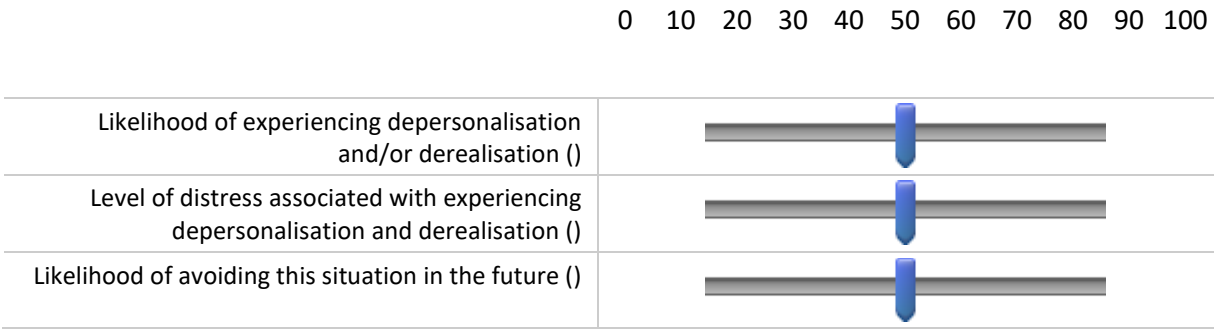
Q119 Sensory overload. For example, being in a scenario where there are bright flashing lights, loud music, many people and is hot.



Q120 Lack of sleep. For example, you usually get 8 hours of sleep per night, however you only managed to get 4 hours.



Q121 Sleeping too much. For example, you usually get 8 hours of sleep per night, however you overslept significantly and slept for 12 hours.



Q133 Other triggers: Please state the trigger and then state, out of 10, the likelihood of experiencing depersonalisation and/or derealisation, the level of distress and the likelihood of future avoidance in brackets. For example: Being on a boat (5/10, 8/10, 4/10).

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End of Block: Potential Triggers

Start of Block: Health Information

X→

Q67 Have you ever been diagnosed with a mental health condition?

- ○ No (0)
- ○ Yes (1)

Display this question:

If Q67 = 1

Q69 What mental health condition(s) were you diagnosed with?

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Display this question:

If Q67 = 1

Q70 Do you take any prescribed medications for your diagnosed mental health condition(s)?

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Q71 Have you ever been diagnosed with temporal lobe epilepsy and/or migraines with or without aura?

- No (1)
- Yes (2)

Q72 Have you ever been diagnosed with, or believe you would qualify for, a substance use disorder (a mental health condition leading to a person's inability to control their use of substances such as legal or illegal drugs, alcohol, or medications)?

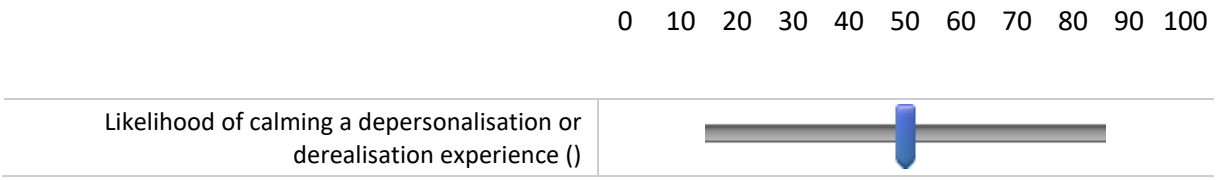
- No (1)
- Yes (2)

End of Block: Health Information

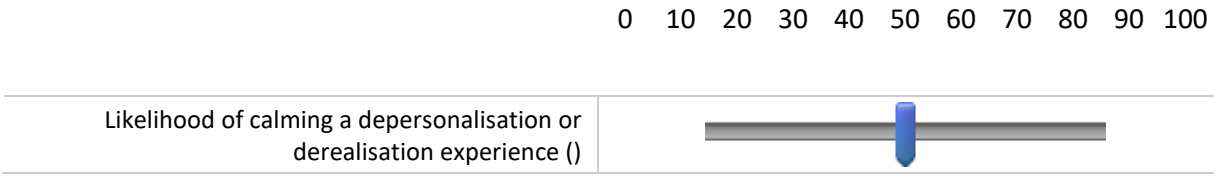
Start of Block: Potential Calming Activities

Q135 The next section encourages you to think about some of the things that in the past, or you believe would presently, help you to 'come out' of a depersonalisation and/or derealisation state.

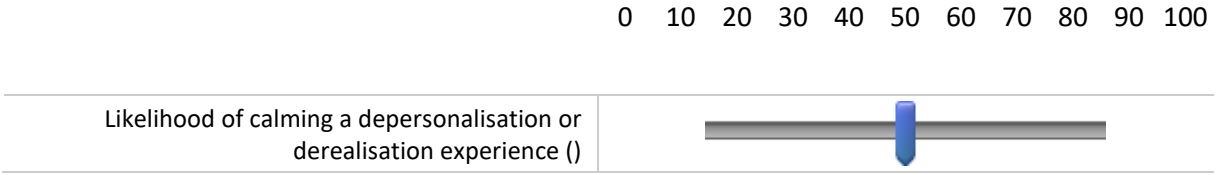
Q136 Journalling. For example, writing down your experiences in the present moment.



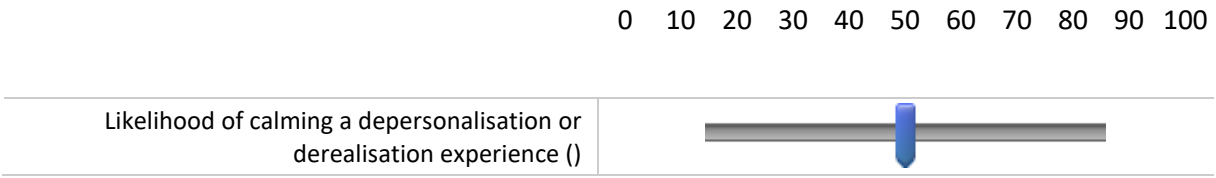
Q137 Mindfulness exercises. For example, in the present moment, focussing on things you can see, hear, smell, taste and touch.



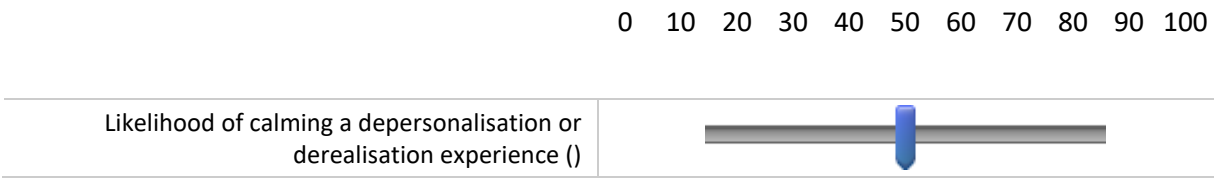
Q138 Meditation. For example, sitting for 5-10 minutes and focussing on your breathing.



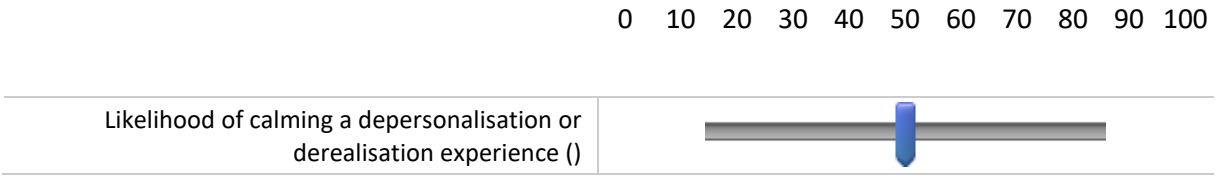
Q139 Practicing acceptance of your sensations. For example, making a conscious effort to accept what you cannot control in the situation, and taking action on what you can control.



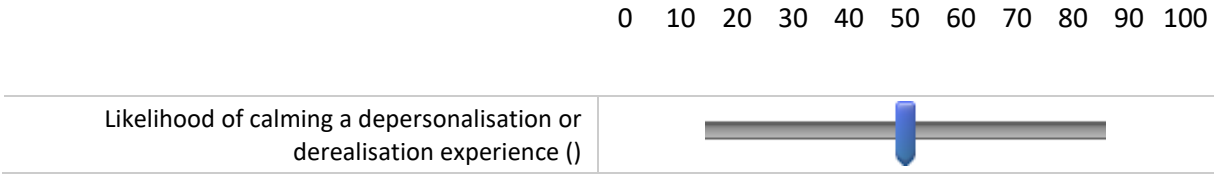
Q140 Simply talking about your experience with another. For example, spending 5-10 minutes describing your experiences.



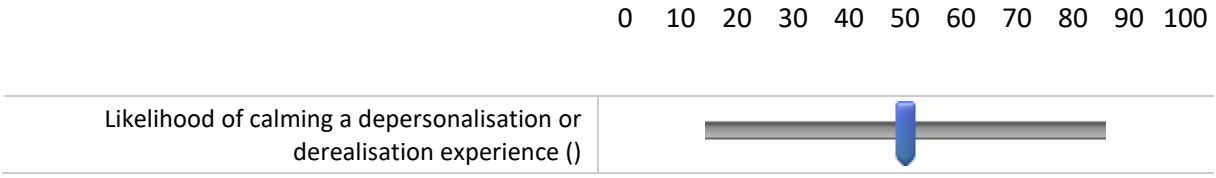
Q141 Grounding techniques. For example, focussing on your feet on the floor and the weight of your body.



Q142 Distraction activities. For example, focussing intently on an audiobook or a podcast.



Q143 Exercise. For example, taking a 5-10 minute run.



Q144 Progressive muscle relaxation. For example, tensing and relaxing the muscles in your body to produce a calm sensation.

	0 10 20 30 40 50 60 70 80 90 100
Likelihood of calming a depersonalisation or derealisation experience ()	

Q145 Reframing your thoughts. For example, seeing your sensations as a way your body is trying to keep you safe, rather than as a danger to you.

	0 10 20 30 40 50 60 70 80 90 100
Likelihood of calming a depersonalisation or derealisation experience ()	

Q146 Other calming activities: Please state the activity and then state, out of 10, the likelihood of becoming it bringing you out of the depersonalisation and/or derealisation experience. For example: Calling a friend (8/10).

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End of Block: Potential Calming Activities

Start of Block: Personal Experience

Q132 OPTIONAL: Is there anything else you would like to add regarding your experiences of derealisation and depersonalisation? For example, are there any particular situations that you feel would bring on an episode of depersonalisation or derealisation only?

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End of Block: Personal Experience