

**An Exploration of the Relationship Between Ineffective Modes of Mentalisation and
Difficulties Related to Borderline Personality Disorder: A Network Approach**

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Abstract

Background: Borderline personality disorder (BPD) is a complex mental health problem whose comprehension, classification and aetiology is still debated. The mentalisation-based understanding of BPD emphasises volatile interpersonal functioning, which has been suggested to originate from ineffective modes of mentalisation. However, it is not yet fully understood how ineffective mentalising relates to specific difficulties associated with BPD. Network theory provides a novel tool to measure the assumption that individual but interlinked mental difficulties ('symptoms') form 'disorders' via the self-perpetuating interactions between them. Therefore, ineffective modes of mentalisation can be included in the network of BPD-related difficulties to explore their role in the activation and maintenance of BPD.

Objective: The purpose of this study was to evaluate the pairwise interactions between ineffective modes of mentalisation and BPD-related difficulties and compare these between groups of people with (clinical group) and without (community group) a BPD diagnosis via a network analysis approach.

Methods: A cross-sectional secondary data analysis using network analysis (Mixed Graph Model) was conducted in 575 people (350 in the clinical group, 225 in the community group) to assess the relationship between ineffective modes of mentalisation (hypermentalisation, hypomentalisation and lack of mentalisation) as measured with the MASC and self-reported BPD-related difficulties. Group differences were investigated in the whole sample via moderation network analysis with group membership employed as moderator variable. Significant and robust interactions were followed up within each group separately.

Results: The moderation network analysis confirmed the presence of significant associations between ineffective modes of mentalisation and specific interpersonal BPD-related difficulties that were moderated by the presence of BPD diagnosis. The pattern of association between mentalising and interpersonal difficulties differed greatly between groups.

Conclusion: The findings provide novel insights into the relationship between hypermentalisation and hypomentalisation and BPD-related problems. Implications for clinical practice, theory development, and future research are discussed.

Chapter One: Introduction

Borderline Personality Disorder

Borderline personality disorder (BPD) is a psychiatric disorder associated with significant dysfunction in multiple domains of daily functioning, affecting an individual's emotional life, cognition and behavioural patterns (American Psychiatric Association, 2013). BPD is marked by a pervasive pattern of dysregulation and instability in emotions, interpersonal relationships, impulse control and identity (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013) contains nine clinical symptoms, of which at least five must be met to meet the diagnostic threshold for BPD. These diagnostic criteria include fear of abandonment, unstable and intense interpersonal relationships, identity disturbance, impulsivity, recurrent suicidal or self-harming behaviour, affective instability, chronic feelings of emptiness, anger outbursts and periods of dissociation or paranoid ideation.

BPD is the most commonly diagnosed personality disorder (PD) associated with complex presentations in modern clinical practice (Loranger, Janca, & Sartorius, 1997). In the UK, the prevalence rate reaches approximately 1.6% in the general population, 10-30% of the psychiatric outpatient and 20% of the inpatient populations (Chapman, Jamil, & Fleisher, 2021; National Institute for Health and Care Excellence, 2007). These rates are similar in the US (Grant et al., 2008; Torgersen, 2005). The suicide rate in people with BPD is up to 10% (Paris, 2019), which is 50% more common than in the general population and is reported to be particularly high for young women (American Psychiatric Association Practice Guidelines, 2001; Gunderson, Herpertz, Skodol, Torgersen, & Zanarini, 2018). Despite 78%-99% of individuals experiencing shorter or longer periods of remission over time (Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012), BPD is associated with persistent and severe social and functional impairment. For instance, high levels of BPD-related difficulties predict lower academic attainment and fewer qualifications (Winograd, Cohen, & Chen, 2008). Only one quarter of diagnosed people are in full-time employment and almost half continue to receive disability payments 10 years after diagnosis (Gunderson et al., 2011). Relatives and carers of individuals with BPD report increased levels of subjective and objective burden, alongside feelings of grief and increased mental health problems (Bailey & Grenyer, 2013). Furthermore, direct and indirect treatment costs associated with BPD are estimated to be high. The economic cost of the care for people with BPD was found to be €16,852 per service

user (SU) per annum in a Dutch (van Asselt, Dirksen, Arntz, & Severens, 2007) and €11,817 per SU in a German study (Bode, Vogel, Walker, & Kröger, 2017). These findings suggest that the disorder remains burdensome on individuals, their families and the public for a long time after the initial diagnosis.

The Aetiology of BPD. Extensive research has resulted in several theories about the aetiology of the borderline personality structure. These generally consider both genetic or neurobiological dispositions, alongside trauma and adverse environmental factors (Trimboli & Marshall, 2020). Nevertheless, the construct of borderline personality has undergone considerable change since it was first used by Adolph Stern in 1938 (Gunderson & Links, 2008; Stern, 1938). The classical psychoanalytic paradigm used the term borderline for a specific form of personality organisation, which then shifted in accordance with the changes within the field of psychiatry, and led to the term to be understood first as a syndrome and then, as a disorder (Gunderson & Links, 2008). It is important to note early on that the terminology of BPD has become increasingly controversial and even pejorative, with Herman (1992, p.123) describing it as a term that mental health professionals use “as a little more than a sophisticated insult”. While it is possible that, with time, the term will be abandoned, retained or revised by professionals, for now it is widely used and accepted in mental health services. For this reason, neutrality over the terminology will be maintained in the present study, which aims to examine the experiences and difficulties associated with the current aetiological understanding of BPD. As Ratcliffe and Bortolan (2020) highlight, if the terminology were no longer acknowledged, it would not discredit the form of experience itself nor the questions the current study aims to answer in relation to it.

In line with the evolution of the borderline construct, the current aetiological understanding considers various factors that might contribute to the complexity of the phenomenon. These include psychological factors, such as early childhood traumas (e.g. sexual, physical, emotional abuse) and invalidating home environment; biological factors, such as dysregulation of neurochemical synthesis and transmission (e.g. oxytocin, MAOA NMDA) or organic brain diseases; and sociological factors, such as social and systemic impacts on the individual (e.g. gender bias, racism, institutional power differences) (Cameron, Calderwood, & McMurphy, 2019; Stepp, Lazarus, & Byrd, 2016). These factors are assumed to interact and lead to psychological vulnerabilities (e.g. emotional dysregulation or reduced mentalisation capacity) and maladaptive behavioural patterns, such as self-harm (Johnson & Vanwoerden, 2021). These are difficulties that individuals who receive BPD diagnosis often struggle with.

The Mentalisation Framework of BPD. One of the most important aetiological frameworks is the mentalisation-based developmental model (Bateman & Fonagy, 2004b; Fonagy, 1991; Fonagy, Gergely, Jurist, & Target, 2002). Mentalisation is a broad concept, incorporating social-cognitive functions, such as theory of mind, emotion recognition and reflective capacities (Ha, Sharp, Ensink, Fonagy, & Cirino, 2013). It has been defined as a developmentally acquired, internal imaginary capacity, which allows the individual to understand and interpret their own and other people's overt actions and behaviours as the expression of underlying mental states (such as needs, desires or beliefs; Fonagy et al., 2002). Mentalisation incorporates the implicit and explicit processes that enable people to make sense of social situations and understand social cues in various relational contexts (Fonagy et al., 2002; Mitchell, 2006). It is therefore argued to be of fundamental importance for effective interpersonal functioning and the ability to form coherent narratives about relationships (Bateman & Fonagy, 2010; Daubney & Bateman, 2015). BPD has been conceptualised as a disorder of self-other relatedness, especially since difficulties with interpersonal functioning such as relationship instability and excessive fear of abandonment are core phenomenological features (Bender & Skodol, 2007; Sharp & Fonagy, 2008b). Consequently, the mentalisation framework is highly relevant towards the understanding of BPD (and other personality disorders).

This aetiological framework of BPD assumes that diversion from the normal developmental pathway occurs as the result of early traumatic environmental effects such as relational traumas and attachment disruptions (Choi-Kain & Gunderson, 2008; Fonagy et al., 2002). These hinder the child's experience of being 'held in mind' and reflected upon as an intentional being (Fonagy et al., 2002). In usual development, the ability to mentalise is thought to result from the sensitive attunement and reflective functioning capacities (referring to the operationalised referent to mentalising capacities) of the primary caregiver in the context of a secure attachment relationship (Bowlby, 1969), which allows the child to "find himself in the other" (Fonagy et al., 2002, p.348). Gergely and Watson (1996) highlight that emotional self-awareness and one's sense of identity evolve through the parent's affective mirroring of the child's internal states. This is further facilitated by the marked and contingent parental exhibition of ostensive communication cues, such as eye contact or infant directed speech. The process promotes secure attachment bonding between the child and their caregiver, which leads to the safe opportunity for the child to make assumptions of the caregiver's mental state, marking an important phase in their socioemotional development (Bowlby, 1988; Sharp & Fonagy, 2008a). However, maltreatment, abuse and parental neglect

lead to insecure attachment relationships, reduced affective mirroring and a less sensitive, infant-attuned environment, which result in the disruption in or diversion from the usual development of mentalising capacities (Bowlby, 1969; Fonagy & Bateman, 2007; Gergely & Watson, 1996).

A large body of empirical studies have evidenced the relationship between borderline symptomatology and mentalisation impairments both in adults and in adolescents (Roepke, Vater, Preißler, Heekeren, & Dziobek, 2013; Carla Sharp et al., 2011), while the link between poor mentalisation abilities, interpersonal problems, impulsiveness and emotional dysregulation has also been demonstrated (Berenson et al., 2018; De Meulemeester, Vansteelandt, Luyten, & Lowyck, 2018; Euler et al., 2021). Mentalisation-based therapy (MBT) is an intervention that focuses on the repairment of mentalisation capacities developed by Bateman and Fonagy (2004a) to treat BPD specifically. Evidence from several systematic reviews evaluating randomised control trials suggest that MBT can effectively reduce BPD-related difficulties, with some studies indicating that the achieved changes stem directly from the improvement of mentalising capacities (Malda-Castillo, Browne, & Perez-Algorta, 2019; Vogt & Norman, 2019; Volkert, Hauschild, & Taubner, 2019). The distorted identification of one's own and others' mind states and the misunderstanding of social cues that have been evidenced in clients with a borderline personality organisation (Schaffer, Barak, & Rassevsky, 2013) is hypothesized to directly affect all other core difficulties related to borderline states, such as problems with emotion regulation, impulsivity, unstable self-representation etc (Fonagy et al., 2002).

Ineffective Modes of Mentalisation. According to the mentalisation model, the behaviour of people with severe psychological difficulties is dominated by non-reflective internal working models in complex and emotionally charged interpersonal situations, particularly in attachment relationships (Bowlby, 1982; Fonagy et al., 2002). As mentioned, the model hypothesizes that the early development of mentalisation capacities was hindered. Therefore, as the integration between various social-cognitive modalities might be hindered, people with BPD may be disadvantaged in providing accurate predictions and inferences in emotionally charged situations (Sharp & Vanwoerden, 2015). The individual might thus attempt to understand social cues and interpersonal processes with ineffective mentalisation techniques that lack the integration of controlled/explicit and automatic/implicit socio-cognitive reasonings (Fonagy et al., 2002; Sharp & Vanwoerden, 2015). This relates to various pre-mentalistic states which may lead to hypermentalisation, hypomentalisation and a lack of mentalisation.

Hypermentalisation. Individuals with complex traumas might retreat to a so-called pretend mode, which is a pre-mentalistic state characterised by the disconnection between the individual's internal world and their outer reality (Swenson & Choi-Kain, 2015). This enables the individual to escape from the intolerable feelings and worries of external reality and enter into a fantasy narrative about the self and others (Duschinsky & Foster, 2021; Fonagy, 1995). This state might result in an alternative (and presumably ineffective) mentalising strategy about others' internal states called hypermentalising. Hypermentalising is defined as the excessive attribution of mental states to others, without observable data to justify it (Frith, 2004; C. Sharp et al., 2011). Compared to people who engage in accurate mentalising processes and show more certainty about their own mental states than about others', individuals who hypermentalise exhibit opposite patterns in showing elevated levels of certainty about others' mental states rather than their own (Müller, Wendt, & Zimmermann, 2021; C. Sharp et al., 2011). The excessive attribution of internal states to others has been understood as a defence mechanism aimed at protecting the self against the emotional impact of anticipated harm (Fonagy, Target, & Gergely, 2000). Hypermentalising is also referred to as "pseudomentalising", as it may look like mentalising but lacks some of its essential features (Allen, Fonagy, & Bateman, 2008), likely leading to the misinterpretation of social situation (Sharp et al., 2013).

Hypermentalisation has been associated with borderline traits, particularly in adolescents, where it is the most commonly used alternative mentalising strategy detected in SUs (Sharp et al., 2013; C. Sharp et al., 2011; Somma et al., 2019), especially in girls (Akca, Wall, & Sharp, 2021). Adults with a BPD diagnosis or borderline traits have also been shown to resort to hypermentalising more frequently than community controls or people with other PDs (Fossati, Borroni, Dziobek, Fonagy, & Somma, 2018; Kvarstein et al., 2020; Normann-Eide et al., 2020; Vaskinn et al., 2015); however, excessive mentalisation in adults was associated with symptom distress and the severity of PD pathology in general rather than BPD features specifically (Normann-Eide et al., 2020). Therefore it may be a less specific feature of BPD in adults than in adolescents.

Hypomentalisation. Another pre-mentalistic state is referred to as the psychic equivalence mode, where the individual experiences their feelings and fantasies as truthful reality and not as mental states that represent external reality (Fonagy et al., 2002). This mode has been linked to hypomentalising, which is defined as a reduced mentalizing capacity stemming from lack of engagement in social cognition and from misattribution or incorrect inference of mental states (Dziobek et al., 2006; Fonagy et al., 2016). This may manifest as

misconstruing social situations or lacking subtle relational understanding (Kvarstein et al., 2020). Hypomentalisation has been related to high levels of uncertainty about self and other's mental states due to the inability to consider complex models about mental states (Badoud et al., 2018), which might lead to simplistic narratives of others' behaviour (Müller et al., 2021)

Hypomentalisation was found to be positively correlated with BPD features and emotional dysregulation in adolescents (Vahidi, Ghanbari, & Behzadpoor, 2021). Goueli, Nasreldin, Madbouly, Dziobek, and Farouk (2020) found positive correlation between hypomentalisation and the severity of BPD symptoms and emptiness specifically. Fossati et al. (2018) demonstrated that hypomentalisation was correlated with the presence of a PD diagnosis in an adult outpatient sample, while De Meulemeester et al. (2018) showed that long-term psychoanalytic treatment of adults with BPD decreased hypomentalisation significantly, which in turn was strongly associated with the decrease of symptomatic distress over time. Similarly, Kvarstein et al. (2020) found that in people with a BPD diagnosis, increased hypomentalisation was associated with more comorbid PD traits, complex childhood trauma history and PTSD, as well as with fewer good clinical outcomes after receiving MBT.

Lack of mentalisation. In certain situations (e.g. when BPD is coupled with depression or other mood disorders), mentalisation might not only be reduced and insufficient, but it might be lacking completely (Luyten & Fonagy, 2015). The total lack of mentalising could be understood as an extreme end of the psychic equivalence mode, associated with drawing inferences purely based on physical causations rather than the understanding of one's own or others' internal states (Dziobek et al., 2006; Fonagy et al., 2002). While several of the aforementioned studies failed to detect direct relationships, Fossati et al. (2018) found that in an adult outpatient sample, lack of mentalisation was correlated with both interview-based and self-reported measures of BPD and emotion dysregulation.

The Network Approach

Theoretical background. Similar to physical diseases, mental disorders have historically been conceptualized in the context of a latent variable approach. This assumes that symptoms of a disorder cluster and covary along one or more underlying latent variable(s), which may also be the disorder itself (Borsboom, 2017a). According to this approach, the observable co-occurrence of symptoms stems from these latent variables (Borsboom & Cramer, 2013). For instance, the core symptoms of BPD (e.g. fear of

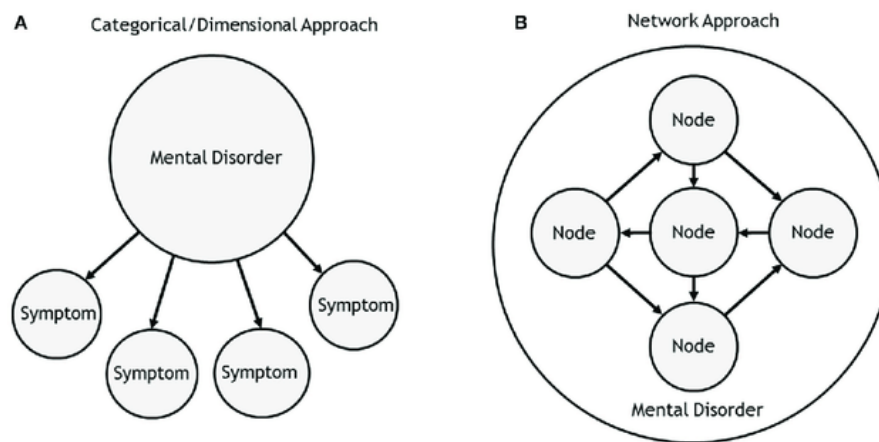
abandonment, rapid mood changes) share variance and occur together solely due to the latent problem itself, namely BPD. Theoretically, this also suggests that treating or removing the disease entity (e.g. BPD) leads to the disappearance of the various symptoms and difficulties experienced by the person. Although this latent variable approach has proven to be fairly beneficial in understanding and treating physical diseases in Western medicine, there is less consistent evidence for the validity and utility of this approach in mental disorders (Borsboom, 2017b; Cramer & Borsboom, 2015). Cramer and Borsboom (2015) suggest that this framework promotes a reductionist scientific focus within psychological empirical research. A particular problem that they highlight is that despite the extensive amount of research aiming to find a small number of possible latent root causes (e.g. brain abnormalities, genes or specific environmental risk factors), the yielded evidence seems to be inconsistent, often lacking e.g. specificity (a common cause might be implicated in the aetiology of different mental health disorders) or omnipresence (the common cause cannot be detected in many people with the same condition), generally explaining only a small portion of the variance in mental health disorders. This suggest that latent variables either “do not exist or else are very hard to find” (Cramer & Borsboom, 2015; p. 5). While this challenge subsequently inspired many researchers to look even harder and deeper into the common causes of co-occurring symptoms (e.g. by improving research methodologies or using better research equipment), the theoretical and empirical relevance of the primary challenges of this approach have faded into the background, until recently (Cramer & Borsboom, 2015).

Over the last decade an alternative approach to conceptualizing psychopathology has emerged: the theory of networks (Borsboom, 2017b; Humphry & McGrane, 2010). Contending that the limits of reductionism have been reached, this approach has recently been adopted in several scientific disciplines, emphasising complexity, interconnectedness and ultimately, the importance of systems rather than single latent variables (Barabási, 2012). Instead of viewing mental disorders as isolated latent entities formed by clusters of covarying symptoms, it is hypothesized that the complex network of symptoms directly cause and influence each other, leading to the emergence of the disorder (Borsboom, 2017b; Borsboom & Cramer, 2013; Cramer & Borsboom, 2015). In other words, the network theory of psychopathology assumes that interlinked difficulties provide the very essence and source of mental problems due to the causal and meaningful interaction found between them. For instance, an excessive fear of abandonment might lead to hyperarousal and misinterpretations of other people’s motives and cues, which might generate frequent interpersonal conflicts. In turn, the instability in social relationships might maintain or exacerbate fear of abandonment,

since frequent social conflicts likely result in further experiences of abandonment. Thus, symptoms reinforce and feed back to each other, creating loops and vicious cycles until the symptoms' activation becomes persistent in the network, which is when a mental health problem emerges clinically (Borsboom, 2017b).

Figure 1.

Latent variable approach (A) vs network theory approach (B) to psychological disorders (Jones, Heeren, & McNally, 2017)



Applying network theory to understanding psychological problems. If the aetiology of psychopathology is assumed to be the consequence of symptom-symptom interaction, the pattern of connections between symptoms can be structured into a network, which is characterized by a specific architecture (Cramer & Borsboom, 2015). In this network, the chosen variables i.e. symptoms or experienced difficulties are defined as nodes, and the set of connections between them are the described as edges (these terminologies are used interchangeably in the followings). Node selection is theory-driven by the inclusion of substantive rather than methodological aspects (Borsboom et al., 2021) that may consist from wide range of variables, such as psychological difficulties, personality traits or environmental risk factors (Fonseca-Pedrero, 2018). Nodes are connected by edges if the particular symptoms have a direct activating effect on each other (Borsboom, 2017b), since edges represent pairwise conditional correlations between two variables, whilst conditioning on all other variables in the network (Borsboom et al., 2021). The set of integrated techniques and procedures that entails the estimation of network parameters (e.g. nodes and edges) is called psychometric network analysis (Borsboom et al., 2021).

Causal interconnectedness between the symptoms may be inferred by an interventionist point of view (Pearl, 2000; Woodward, 2003), whereby activation in a symptom raises the probability of activation in another (X is causing Y if changing X generates change in Y, while holding other variables fixed). However, this is not an exclusive process, aiming at identifying sole causes, but rather a step to understand which one factors (X) contribute to causing another one (Y), knowing that other factors (Z) can also contribute to the cause of Y.

The network's state (e.g. level and structure of activation between the symptoms) is of particular interest for understanding psychopathology (Robinaugh, Hoekstra, Toner, & Borsboom, 2020). Individual symptoms may be triggered by events (e.g. loss of loved one/abuse) from the external field, i.e. outside of the network (Borsboom, 2017b). Depending on the network's state, the activation spreads and interconnected difficulties are stimulated. Highly connected networks therefore facilitate and accelerate the activation of nodes to a greater extent than less connected networks (Borsboom, 2017a). Once a network is induced, it can turn into an independent, self-sustaining entity over time, preserving its internal activation in the absence of the original external trigger (Borsboom, 2017b). Hence, the symptom-symptom relationships are sufficient to maintain an elevated symptom activation, which can be understood as a harmful equilibrium state, or in other words, a mental health disorder (Robinaugh et al., 2020). Further characteristics of the network include the phenomenon of symptom clustering based on the presence or strength of connectivity between the nodes, leading to aggregated groups of symptoms in the network that activate each other to a greater extent (Borsboom, 2017a).

In order to enrich our understanding of mental health disorders, it has been recommended to include additional variables, i.e. nodes, which vary at an individual level but might play causal roles in disorders (Jones et al., 2017). These include relevant cognitive, biological or social variables (e.g. metacognitive beliefs or social cognitive factors).

Links to clinical practice. Historically, psychopathology research has avoided drawing causal interpretations of symptom constellations and interpreting them as potential maintenance factors. However, the understanding of intercausal cycles has long been a routine and necessary component of clinical practice, especially when formulating service users' difficulties and developing their treatment plans (Johnstone & Dallos, 2014; Kim & Ahn, 2002). Cognitive Behavioural Therapies (CBT) for instance stress the importance of symptom maintenance or maladaptive cycles, in which the problematic behaviours are reinforced by their own consequences, as they often lead to the confirmation of negative core

beliefs that further fuel the initial problematic behaviour (Josefowitz & Myran, 2017). Similarly, while systemic therapies shifted their focus from intrapsychic factors to interpersonal ones, several early cybernetics ideas (e.g. feedback loops, harmful equilibriums and the notion of circular causality) are consistent with the theoretical underpinnings of the network theory approach (Dallos & Draper, 2015). Therefore, the application of network theory in the empirical study of psychopathology may improve alignment between research and clinical practice.

Centrality versus causality. The application of network theory in empirical research has been shown to be useful and has pioneered insights into several domains of psychopathology, such as exploring comorbidity between mental health disorders or predicting their pathogenesis (Fried et al., 2017). Naturally, scrutinizing the importance of specific nodes, i.e. symptoms, in the causality of a disorder has also become a core interest (Fried et al., 2017). It is argued that the influence of specific nodes on the network varies based on their connectivity with other nodes (Borsboom, 2017b). Centrality indices are used to assess the importance of specific nodes in the network, by relating the positions of individual nodes to potential dynamics of the system (Borsboom et al., 2021; Costantini et al., 2015; Kolaczyk, 2009). For instance, a symptom with high centrality indices may present as a greater risk factor in the maintenance and development of the disorder than a peripheral node (van Bork, van Borkulo, Waldorp, Cramer, & Borsboom, 2018). By measuring the unique role that nodes play in a network, centrality indices could help to identify effective treatment targets. Since they shed light on those nodes with stronger activation of other symptoms, targeting them could potentially induce a sequence of symptom deactivation across the network (Fried et al., 2017).

However, centrality indices must be interpreted cautiously. Central symptoms do not necessarily correspond to those with the highest clinical importance or to the most effective intervention target (Fried et al., 2018). For instance, Fried et al. (2018) emphasize that a high centrality value of a node could stem from a symptom sitting at the endpoint of a causal chain. The temporal quality of such a causal chain might remain hidden in a cross-sectional design, resulting in incorrect causal inferences about the specific difficulty. It is also possible that a node with high centrality features in several feedback loops with other symptoms. If this is the case, an intervention primarily targeting the specific node might have limited success due to the reoccurring triggering effect of the other symptoms. Since cross-sectional studies do not make it possible to demonstrate whether a high-centrality node is the source or

the recipient of the activation, treatments focusing only on centrality are unlikely to succeed (McNally, 2021).

Furthermore, several recent studies additionally caution against the use of centrality indices as substitutes for causal inferences and treatment targets because the relationship found between the symptoms in cross-sectional partial correlation network models (a common network method applied for network theory) might stem from disregarded common causes or indirect causal relationships (Borsboom et al., 2021; Dablander & Hinne, 2019; von Klipstein, Borsboom, & Arntz, 2021). Although the network theory approach seems to outperform traditional confirmatory factor models (which is the primary underpinning statistical approach for discovering latent variables) on different measures of cognitive performance (Kan, van der Maas, & Levine, 2019), some researchers have proposed a combination of conceptualisations by re-introducing latent or mediating variables as complimentary models to the network models in network methodologies (Dablander & Hinne, 2019; Kan et al., 2019). These hybrid statistical models could potentially include the common external causes (e.g. trauma) in the model as well, which might underpin different mental health problems and subsequently generate further ones, until self-maintaining networks of difficulties take shape (Epskamp, Rhemtulla, & Borsboom, 2017; Fried & Cramer, 2017; Fried et al., 2018).

Nevertheless, some research has provided promising findings about the importance of centrality measures (McNally, 2021). For example, change in high-centrality nodes of a grief network has been related to change in other grief-related difficulties as well (Robinaugh, Millner, & McNally, 2016). A longitudinal study showed evidence that high-centrality nodes of a depression network were predictive of an episode of major depression disorder within a 6-year follow-up (Boschloo, van Borkulo, Borsboom, & Schoevers, 2016). Similarly, (Elliott, Jones, & Schmidt, 2019) found that high-centrality symptoms predict both clinical impairment and failure to recover in a group of patients receiving treatment for anorexia nervosa. The debate about which causal inferences can be statistically interpreted from a network model is ongoing. However, a reasonable suggestion by Borsboom et al. (2021) is that researchers should interpret the statistical structure of the networks only in the context of evidence-based assumptions and strong network theories.

The Relevance of Network Theory for BPD

The number of studies using network analysis increased in recent years. Robinaugh et al. (2020) identified 363 psychology-related articles, with the majority being empirical

studies. Network studies have explored a wide range of mental health difficulties across the lifespan, including several studies examining the network structure of various PDs and their comorbidities (McNally, 2021). Yet, the classification of a “borderline” personality construct faces several challenges and remains debated. Network analysis might be a useful way to address some of these.

Impact of individual symptoms. The current diagnostic systems for BPD have been challenged by an array of criticisms. These generally focus on the lack of consideration of empirical findings about the nature and significance of BPD and PD traits more broadly (Johnson & Levy, 2019). Firstly, subclinical representations of BPD are hardly recognized, even though evidence suggests that meeting one of the nine criteria of the *DSM-5* could already be associated with significant functional impairments (Ellison, Rosenstein, Chelminski, Dalrymple, & Zimmerman, 2016). Secondly, although causal symptom-symptom interactions in general are considered as part of the diagnostic criteria for specific mental health problems in the *DSM-V*, this is not the case for PDs (American Psychiatric Association, 2013; Borsboom, 2017b). Instead, the categorical approach of the *DSM-5* PD criteria assumes the existence of an underlying condition with symptoms loading uniquely onto the relevant PDs with identical contribution (Contreras, Nieto, Valiente, Espinosa, & Vazquez, 2019). However, specific problems seem to cross-load to several PD diagnoses rather than just the one they are assigned to (especially in the case of BPD; Hawkins et al., 2014) and are proved to have different contributions to the final diagnosis as individual criteria and combination of criteria (Cooper, Balsis, & Zimmerman, 2010). This suggests that not every PD criterion contributes equally to the latent concept, and that accounting for the specificity of the endorsed criteria provides important information.

The network approach on the other hand emphasizes the shortcomings of interpreting symptoms interchangeably, proposing that their position in and connectivity across the network influences their potential for activating other symptoms (Cramer, Waldorp, Van Der Maas, & Borsboom, 2010). This is underpinned by the notion that individual symptoms are likely to differ in terms of their connectivity and their impact on maintaining the problem (Cramer et al., 2010). By focusing on individual symptoms rather than the presence or absence of the disorder, this approach allows for the disentanglement of the individual contributions of specific BPD symptoms towards the maintenance of the problem (Bringmann & Eronen, 2018).

Categorical versus dimensional understanding of BPD. Another challenge of current diagnostic systems involves the debate around conceptualizing BPD (and PDs in

general) as a categorical or a dimensional construct. The *DSM-5* determines the diagnosis of BPD categorically: a diagnosis is only made if at least five of the nine defined criteria are present alongside significant levels of distress or impairment. However, the validity of this has been criticised since it does not seem to account for the heterogeneity reported by clinicians (Wardenaar & de Jonge, 2013). In fact, a total of 256 distinct symptom combinations is possible according to the *DSM-5* BPD criteria (Busch, Balsis, Morey, & Oltmanns, 2016). Additionally, the clinical manifestation of BPD is further influenced by the presence of other diverse and differentially severe comorbidities (Meehan, Clarkin, & Lenzenweger, 2018).

In light of this, the recently published *International Classification of Diseases, 11th Revision (ICD-11; World Health Organization, 2020)* has shifted towards a dimensional, transdiagnostic understanding of PDs (Johnson & Vanwoerden, 2021). It removed specific PD types and narrowed the diagnosis of PD into one general disorder, with differing levels of severity. However, this approach has been argued to be too reductionist and potentially stigmatizing; not to mention that it also seems to lack valid scientific evidence (Herpertz et al., 2017). This dimensional construct provides an alternative to the use of arbitrary cut-offs found in traditional categorical constructs. However, it still assumes the existence of an underpinning latent yet discrete disorder which exists on a continuum (Borsboom, 2008; Solomon, Haaga, & Arnow, 2001). Thus, the dimensional understanding of mental health problems still promotes the separation of people who have a diagnosis of mental health disorder from the general population (Borsboom, 2008).

Contrary to the categorical and dimensional perspectives, the network approach applies a causal system perspective, suggesting that experienced symptoms do not define the construct of BPD but instead drive the relationship between each other. This integrates the categorical and dimensional views by first identifying if a system of BPD-related difficulties has been activated (akin to categorical view) and then measuring the severity of this system activation (akin to dimensional view; Borsboom, 2008). Preliminary evidence supports this notion by suggesting that network analysis can capture the differences and transitions between “healthy”, clear states and “unhealthy”, disordered states, by examining the impact of individual symptoms and their connectivity on the activation of the system (Fried et al., 2017; Scheffer et al., 2009; van de Leemput et al., 2013).

The network approach also promotes a transdiagnostic perspective. Certain symptoms that are connected and therefore receive and send out effects to more than one disorders may be identified as “bridge symptoms” or transdiagnostic symptoms between different

diagnoses, leading to symptom activation across disorders and subsequently to the observable phenomena of comorbidity (Cramer et al., 2010). This perspective may thus help to explain the heterogeneity of symptoms within and the comorbidity between in mental health disorders (such as depression; Fried, 2015) by better capturing the complexity that characterises severe mental health difficulties (Borsboom & Cramer, 2013; Boschloo et al., 2015). The theory emphasises a dynamic rather than a static approach, examining interactions and changes rather than structures of covariance and resulting latent variables (Bringmann & Eronen, 2018). This fluidity may be particularly relevant for conceptualising BPD, as people who receive this diagnosis typically to experience frequent and volatile emotional fluctuations, which often trigger further emotional processes and behavioural reactions with destabilising effects (Ratcliffe & Bortolan, 2020).

Adding Mentalisation to the Network of BPD-related Difficulties

The above has primarily focused on applying network theory towards understanding BPD in the context of its current diagnostic criteria. However, the network approach advocates for the inclusion of any internal or external factors (e.g. biological, cognitive, behavioural, psychological) which potentially explain the causal activation of symptoms associated with the observed phenomena (Borsboom, 2017b). It views psychological problems in an intrinsically complex manner, impacted by intrapsychic and environmental factors as well (Blanco et al., 2019; Borsboom, Cramer, & Kalis, 2019).

While various factors are likely to influence the network of BPD-related features, the concept of mentalisation might be of particular relevance. Baker (1995) suggested that the content of mental states plays a central role in relating symptoms to each other. Borsboom et al. (2019) also emphasized that an individual's beliefs might affect the activation of network connections. As described in detail above, mentalisation is an integrative process requiring cognitive and affective components, through which accurate (or inaccurate) interpretations of the social world and the mental state of the self and others are formed (Choi-Kain & Gunderson, 2008; Fonagy & Luyten, 2009). Due to the significance of mentalisation in interpersonal relationships and social situations, ineffective or inadequate mentalising could trigger heightened emotional arousal and unhelpful automatic thought processes. In an effort to regulate emotions and maintain a (false) sense of stability, this may then lead to a disorganised self and destructive and impulsive behaviours (Fonagy & Bateman, 2007; Laurensen et al., 2014). Thus, it may be difficult to establish the role of mentalisation as it may be obscured by the volatility in mood, self-image and interpersonal relationships

(Ratcliffe & Bortolan, 2020). However, network analysis could be an effective tool to identify the underpinning mentalisation-related drivers of people's thoughts and actions, not only in terms of the symptom covariation but also in terms of the possible activation loops (Borsboom et al., 2019).

In recent years, an increasing number of studies applied a network analysis approach to understanding the development and maintenance of BPD-related difficulties, incorporating various cognitive, behavioural and emotional factors that might contribute to the activation of the network. However, to the author's best knowledge, no systematic summary of these studies currently exists. The following therefore provides a systematic literature review of network analysis studies that aims to answer the following research questions:

- 1) What BPD-related difficulties have been identified in feedback loops and network connections in community versus clinical samples?
- 2) What additional cognitive, behavioural and emotional factors have been studied in the network of BPD-related difficulties?
- 3) Has any study incorporated mentalisation in the network of BPD-related difficulties?

It is important to distinguish that the first question specifically focuses on studies that include validated measurements of BPD as a construct only, assessing difficulties proven to be related to the phenomenological experience of people having a BPD diagnosis. Contrastingly, the second question refers to any additional mental health-related difficulty or other factors (e.g. cognitive or behavioural) that might activate and maintain the system of BPD-related traits.

Systematic Literature Review

Search strategy. Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses ([PRISMA]; Moher, Liberati, Tetzlaff, & Altman, 2009) guidelines, a systematic search of peer-reviewed literature was conducted on electronic databases including Scopus, Web of Science, PsycINFO, CINAHL, MEDLINE, PsycARTICLES, ScienceDirect and PubMed between the 3rd and 18th of September 2021. The applied search strategies used terms related to BPD and network theory literature, while Boolean operators were employed to increase search sensitivity. Titles, abstracts and keywords of articles were searched for the following terms: (borderline personality disorder OR bpd OR emotionally unstable personality disorder OR eupd) AND (network analysis OR network theory OR network model OR network perspective OR network modelling OR network

structure OR network approach OR network design). A manual search on the bibliography and citations of relevant papers was also conducted.

Eligibility criteria. Studies using network analysis in relation to BPD symptomatology were identified. Based on the recommendations for systematic reviews of network analysis studies (Malgaroli, Calderon, & Bonanno, 2021), titles and abstracts were screened against the following inclusion criteria:

- 1) Empirical study
- 2) Measures BPD-related symptoms
- 3) Includes network analysis of BPD-related difficulties
- 4) Uses cross-sectional study design
- 5) Peer-reviewed
- 6) English text is available

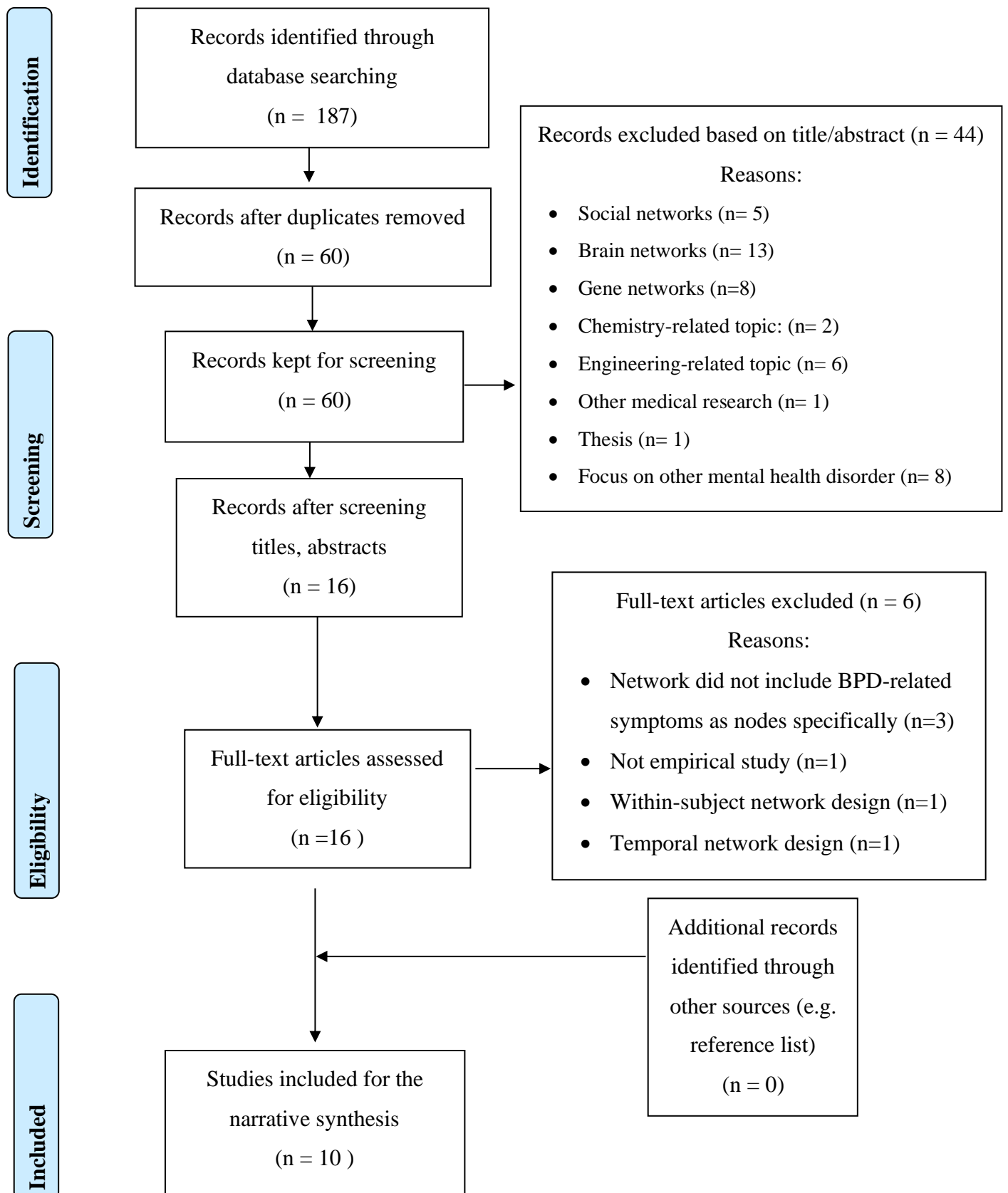
Articles and their network models were screened against the following exclusion criteria:

- 1) Conference extracts, dissertations
- 2) Used social networks
- 3) Used brain networks
- 4) Used gene networks
- 5) Used a longitudinal or within-subjects network design
- 6) Not an empirical study
- 7) Network does not include BPD-related difficulties specifically

Study selection. Figure 2 provides the flow chart for the study selection process. The database searches yielded 187 records in total. After removing duplicates 60 papers remained. Screening of titles and abstracts left 16 relevant studies. Reference lists and citations of the 16 papers were hand searched, but no further eligible studies were identified. Full texts of the 16 studies were then screened against the eligibility criteria, leading to a total of 10 studies to be selected for analyses.

Data extraction. Data was extracted and recorded into a pre-defined Excel table (columns correspond to those in Table 1). Data analysis was conducted according to the recommended structure for narrative synthesis, which includes the following stages: theory development about the interventions, preliminary synthesis of findings, investigation of relationships in the data and evaluation of robustness of the synthesis (Popay et al., 2006). However, since the current narrative synthesis focuses on studies that include network analyses and therefore a network of associations, the current review focuses on the assessment of correlation between nodes, rather than interventions.

Figure 2.
PRISMA flow chart of search strategy



Study characteristics. Relevant characteristics and demographics of the 10 cross-sectional studies are summarised in Table 1. All network studies were published in the last five years (between 2016 and 2021) and were conducted in various countries: United States (n=2), the Netherlands (n=2), Iran (n=1), Belgium (n=1), Australia (n=1), Austria (n=1), Italy (n=1) and Hungary (n=1). The reported age of participants ranged from 12 to 87 years, although De Paoli, Fuller-Tyszkiewicz, Huang, and Krug (2020) and Southward and Cheavens (2018) did not report this. Based on available data, it appears as though only three studies included participants below the age of 18 (Buelens, Costantini, Luyckx, & Claes, 2020; De Paoli et al., 2020; Rivnyák, Pohárnok, Péley, & Láng, 2021). Female participants were overrepresented by 4%-75% in seven of the ten studies. The ethnicity of participants was only reported in four studies (Buelens et al., 2020; De Paoli et al., 2020; Peckham et al., 2020; Southward & Cheavens, 2018), with most of them indicating a majority (from 44% to 88%) of Caucasian participants of Belgian nationality. Two studies used data that was collected as part of larger projects (Buelens et al., 2020; Knefel, Tran, & Lueger-Schuster, 2016), while another study analysed data pooled from several small studies (von Klipstein et al., 2021). Participants were recruited in community settings (n=3), clinical settings (n=3) or both (n=4).

The studies used a variety of tools to capture BPD-related difficulties. Two studies (Esmailian, Dehghani, Koster, & Hoorelbeke, 2019; Southward & Cheavens, 2018) used the self-report Personality Assessment Inventory - Borderline Personality Feature Scale (PAI-BOR), while two others included the nine BPD diagnostic criteria of the Structured Clinical Interview for Diagnostic and Statistical Manual-Fourth Edition Axis II Disorders ([SCID-II]; Knefel et al., 2016; Köhne & Isvoranu, 2021). Other studies opted for the self-report Borderline Personality Disorder Checklist ([BPDCL]; Richetin, Preti, Costantini, & De Panfilis, 2017), the McLean Screening Instrument for Borderline Personality Disorder ([MSI-BPD]; Peckham et al., 2020), and the Borderline Personality Disorder Severity Index, 4th version ([BPDSI-IV]; von Klipstein et al., 2021). Of the studies including adolescents and young people, two employed the self-report Borderline Personality Features Scale for Children ([BPFSC-11]; Buelens et al., 2020; Rivnyák et al., 2021), whilst the third used the self-report Borderline Personality Questionnaire ([BPQ]; De Paoli et al., 2020).

Table 1.
Main characteristics of the 10 included network analysis studies of the systematic literature review.

Publication	Sample size (N)	Population ^a	Age M (range or SD) ^b	Female/male ratio (%)	BPD features ^c	Additional factors	Other measures ^d	Key findings
Knefel et al. (2016)	219	Community	57.95 (29–87)	40/60	SCID-II	PTSD, Complex PTSD	ICD-TQ	<ul style="list-style-type: none"> • Strong within-disorder connection for PTSD and Complex PTSD but not for BPD; • BPD is weakly connected to PTSD and Complex PTSD; • Most central transdiagnostic symptoms: re-experiencing and dissociation
Richetin et al. (2017)	Community: 1317	Mixed (community and clinical, examined separately)	Non-clinical: 22.56 (17–65)	Non-clinical: 74/26	BPDCL	-	-	<ul style="list-style-type: none"> • The general structure of the networks was very similar between the clinical and non-clinical population; • Most central symptoms: affective instability, identity problems and efforts to avoid abandonment
	Clinical: 96		Clinical: 37.75 (18–66)	Clinical: 60/40				
Southward and Cheavens (2018)	4,386	Mixed (community and clinical, not examined separately)	22.44 (8.15)	61/39	PAI-BOR	Emotion regulation difficulties; interpersonal difficulties	DERS; IIP	<ul style="list-style-type: none"> • The most central difficulties in the network for people with more severe BPD traits: loneliness, recklessness/impulsivity and intense moods; while for people with less severe BPD traits: identity difficulties and problems with socializing; • Networks of BPD traits were not different across genders; • Bridge symptoms between emotion dysregulation and interpersonal difficulties for people with severe BPD traits: nonacceptance of one's emotions; while for people with less severe BPD traits: chronic emptiness
Esmailian et al. (2019)	706	Community	19.48 (18–24)	59/41	PAI-BOR	Early maladaptive schemas	YSQ-SF	<ul style="list-style-type: none"> • Identity problems were connected with abandonment, insufficient self-control, dependence/incompetence, and vulnerability to harm/illness cognitive schemas; • Interpersonal problems were associated with mistrust/abuse and abandonment schemas • Self-harm was connected to emotional deprivation and failure schemas

Table 1 (Continued)

Publication	Sample size (<i>N</i>)	Population ^a	Age M (range or SD) ^b	Female/male ratio (%)	BPD features ^c	Additional factors	Other measures ^d	Key findings
Buelens et al. (2020)	347	Community	15.05 (12-20)	78/22	BPFSC-11	Non-suicidal self-injury (NSSI)	Survey questions based on DSM-V criteria for Non-suicidal Self-injury Disorder (NSSI-D)	<ul style="list-style-type: none"> • NSSI is distinct but closely related to BPD in adolescents; • Interconnectedness between the two disorders did not differ across genders; • Most important bridge symptoms between BPD and NSSI: loneliness, impulsivity, separation anxiety, frequent thinking about NSSI, and negative affect prior to NSSI
De Paoli et al. (2020)	753	Mixed (community and clinical, not examined separately)	22.36	82/18	BPQ	Eating disorder (ED); Attachment style; Emotion dysregulation; Theory of mind (ToM); Emotion recognition; Rejection sensitivity	EDI-3; ECR-R; DERS; RME; Ekman 60 Faces Test; RSQ	<ul style="list-style-type: none"> • BPD and ED symptoms were distinct constructs, connected through some transdiagnostic symptoms; • Most central element of the network: abandonment, emotion dysregulation; • ToM and emotion recognition had very few connections in the network
Peckham et al. (2020)	5212	Clinical	34.4 (17-78)	54/46	MSI-BPD	-	-	<ul style="list-style-type: none"> • Network of BPD symptoms differed between people aged above and below 46; • In older participants the connection between NSSI/suicide and emptiness was weaker, while the connection between anger and interpersonal difficulties was stronger
Köhne and Isvoranu (2021)	376	Clinical	34.27 (18-72)	33/67	SCID-II	Major depression (MD)	BDI-II	<ul style="list-style-type: none"> • Transdiagnostic links found between symptoms of MD and BPD; • Strongest links found between sadness reported in MD and emptiness reported in BPD
Rivnyák et al. (2021)	169	Mixed (community and clinical, not examined separately)	15.38 (12-18)	48/52	BPFSC-11	Identity diffusion	AIDA	<ul style="list-style-type: none"> • Most central difficulty: identity diffusion. All BPD features were positively correlated with identity diffusion

Table 1 (Continued)

Publication	Sample size (N)	Population ^a	Age M (range or SD) ^b	Female/male ratio (%)	BPD features ^c	Additional factors	Other measures ^d	Key findings
von Klipstein et al. (2021)	683	Clinical	32.28 (18-61)	87/13	BPDSI-IV	-	-	<ul style="list-style-type: none"> • Most central feature: affective instability

Note. - = data not reported. BPD=Borderline personality disorder. PTSD=Post-traumatic stress disorder.

^aCommunity=people in the community who filled out a BPD-related questionnaire but were not SUs in mental health services, clinical population=mental health SUs; ^bM=mean, SD=standard deviation; ^c SCID-II= Structured Clinical Interview for DSM-IV, BPDCL= Borderline Personality Disorder Checklist, PAI-BOR= Personality Assessment Inventory - Borderline Personality Feature Scale, BPFSC-11= Borderline Personality Features Scale for Children-11; BPQ= Borderline Personality Questionnaire; MSI-BPD= McLean Screening Instrument for Borderline Personality Disorder; BPDSI-IV= Borderline Personality Disorder Severity Index, 4th version, ^d ICD-TQ= ICD-11 Trauma Questionnaire, DERS=Difficulties in Emotion Regulation Scale, IPP=Inventory of Interpersonal Problems, YSQ-SF=Young Schema Questionnaire-Short Form, EDI-3=Eating Disorder Inventory 3, ECR-R=Experiences in Close Relationships-Revised, RME=Reading the Mind in the Eyes, RSQ=Relationship Scales Questionnaire, BDI-II=Beck Depression Inventory-II, AIDA=Assessment of Identity Development in Adolescence.

Methodological quality assessment. Several validated methodological quality assessment tools were considered to review these non-randomised quantitative studies, including e.g. the Effective Public Health Practice Project tool (Armijo-Olivo et al., 2012; Thomas, Ciliska, Dobbins, & Micucci, 2004) or the Newcastle-Ottawa scale (Bae, 2016). However, none of the commonly used assessment tools cover the specificities of network analysis. To the author's knowledge, there are only two systematic literature reviews published so far that evaluate studies, which examine psychological problems using network analysis (Contreras et al., 2019; Malgaroli et al., 2021). Neither of these used a validated methodological quality assessment tool, and to the author's knowledge there is no such instrument currently available. However, the review by Malgaroli et al. (2021) suggested a comprehensive structure for reviewing the quality of network components. This was followed in the current study, although it must be noted that global appraisal scores (typically found in validated quality assessment tools) were not assigned. The synthesis of results was done according to best practice documentation standards for reporting on network models, as suggested by Burger et al. (2020).

Categories. Table 2 summarizes the extracted characteristics of the networks generated in each study. The columns show the categories used for evaluating these. The first section outlines general characteristics of the estimated networks. This includes the following categories:

- 1) *Model N*: total number of cross-sectional network models generated.
- 2) *Centrality*: centrality indices measure the importance of a node in the network (Borsboom et al., 2021). The most common centrality indices are node strength, closeness, betweenness, expected influence and predictability (Malgaroli et al., 2021).
- 3) *Symptom connectivity*: refers to overall symptom connectivity, i.e. the edges in a network model, and can be measured by global strength, density values or the sum of absolute edge weights (Burger et al., 2020).
- 4) *Group differences*: comparisons across groups.

The second section of Table 2 outlines the standards for estimating networks:

- 1) *Parameters*: an increased number of parameters leads to increasingly computationally demanding networks, so large sample sizes are needed for accurate estimations (Fried & Cramer, 2017; Malgaroli et al., 2021). Parameters for each paper were calculated based on the equation model provided by Fried and Cramer (2017), which was also used for evaluating the accuracy of results in relation to sample size. As the rule of

thumb, Fried and Cramer (2017) suggest at least three participants per parameter are required for accurate estimations.

- 2) *Correlation*: the type of correlation provided as input (e.g. covariance matrix), which the model builds on (Epskamp & Fried, 2018). For categorical and binary variables polychoric and tetrachoric are recommended, while for continuous items Pearson and Spearman correlations are often applied (Malgaroli et al., 2021).
- 3) *Analytic model*: the type of network model employed. Gaussian Graphical Models (GGM) are conducted when the data is continuous and normally distributed, while Ising Models are applied for binary data (Epskamp, Kruis, & Marsman, 2017). Mixed Graphical Models (MGM) are used for estimating different type of data together i.e. categorical and continuous (Haslbeck & Waldorp, 2020). The Fused Graphical Lasso (FGL) model is used for comparing networks (Costantini et al., 2019).
- 4) *Regularization*: the regularization technique used for the overestimation of false positive associations in the network. Graphical least absolute shrinkage and selection operator (GLASSO) is a commonly applied regularization technique (Babyak, 2004; Friedman, Hastie, & Tibshirani, 2008), which uses a tuning parameter called lambda (λ). If using FGL models, the regularization can be extended with another tuning parameter called lambda2 (λ_2 ; Richetin et al., 2017).
- 5) *Selection of tuning parameter*: The optimal selection of tuning parameter is often evaluated according to the Extended Bayesian Information Criterion (EBIC; Chen & Chen, 2008). This has been shown to estimate the true network structure accurately by utilising a hypertuning parameter called gamma (γ), which is usually set between the values of 0.1 and 0.5 (Epskamp & Fried, 2018; Foygel & Drton, 2010).
- 6) *Missing data*: reporting and handling missing data is important when conducting network analysis (Burger et al., 2020). Missing data is often handled with a multiple imputation method or by the full information maximum likelihood (FIML) method, which estimates missing values of the population based on the available data. (Cham, Reshetnyak, Rosenfeld, & Breitbart, 2017).

The third section of Table 2 outlines the stability indices that were used in network studies:

- 1) *Centrality stability*: estimation of the stability of centrality indices reported, often conducted with a case-dropping bootstrapping method and quantified by a correlation stability coefficient (CS; Epskamp, Borsboom, & Fried, 2018).
- 2) *Edge accuracy*: use of confidence intervals (e.g. 95% CI) to assess accuracy of edges in the network, i.e. the edge-weight estimates (Hevey, 2018).

The last section of Table 2 refers to the reproducibility of studies.

- 1) *Open Data*: whether the researchers shared their dataset (or parts of it), the covariance matrices that they based their network on or the R-code used for the study (Malgaroli et al., 2021).
- 2) *Open Access*: refers to the free accessibility of papers via open-access peer-reviewed journals (Gold) or the authors' personal website/online repository (Green; Malgaroli et al., 2021).

Table 2.*Network characteristics and quality measurements*

	Characteristics				Estimation				Stability			Reproducibility		
	Model N	Centrality	Symptom connectivity	Group differences	Parameters	Correlation	Analytic model ^a	Regularization ^b	Selection of tuning parameter ^c	Missing data ^d	Centrality stability ^e	Edge accuracy ^f	Open data	Open Access
Knefel et al. (2016)	2	Node strength, closeness, betweenness	Density	-	496	Polychoric	GGM	GLASSO	EBIC ($\gamma=0.1$)	FIML	Case-drop bootstrapping	95% CI	No	No
Richetin et al. (2017)	2	Node strength, closeness, betweenness	-	Community student vs. clinical samples	36	-	FGL	GLASSO + λ_2	EBIC	-	-	-	Dataset	Gold
Southward and Cheavens (2018)	5	Node strength, expected influence, closeness, betweenness	Global strength across group	Low BPD vs. high BPD with and without emotion dysregulation and interpersonal problems; women vs. men;	276, 105	-	GGM, FGL	GLASSO	EBIC ($\gamma=0.5$)	Multiple imputation	Case-drop bootstrapping, CS coefficients	-	Code	Green
Esmailian et al. (2019)	1	Node strength, closeness, betweenness, predictability	-	-	253	-	GGM	GLASSO	EBIC ($\gamma=0.5$)	No missing data	Case-drop bootstrapping, CS coefficients	95% CI	Matrix	Green
Buelens et al. (2020)	3	Node strength, expected influence	Global strength across gender	Female vs. male	253	-	GGM	GLASSO	EBIC ($\gamma=0.25$)	Multiple imputation	Case-drop bootstrapping, CS coefficients	95% CI	Matrix	Gold
De Paoli et al. (2020)	1	Node strength, closeness, betweenness	-	-	153	-	GGM	GLASSO	-	-	Case-drop bootstrapping, CS coefficients	-	Matrix	Green

Table 2. (Continued)

	Characteristics						Estimation			Stability		Reproducibility		
	Model N	Centrality	Symptom connectivity	Group differences	Parameters	Correlation	Analytic model	Regularization	Selection of tuning parameter	Missing data	Centrality stability	Edge accuracy ^f	Open data	Open Access ^g
Peckham et al. (2020)	2	Expected influence	Global expected influence	Younger vs older people	45	Pearson	GGM, Ising	GLASSO	EBIC ($\gamma=0.5$)	Multiple imputation	Case-drop bootstrapping, CS coefficients	95% CI	No	No
Köhne and Isvoranu (2021)	1	-	-	-	513	-	GGM	GLASSO	EBIC ($\gamma=0.5$)	No missing data	-	95% CI	No	Gold
Rivnyák et al. (2021)	1	Betweenness, closeness	-	Community vs clinical	66	-	GGM	GLASSO	EBIC	-	-	-	Dataset	Gold
von Klipstein et al. (2021)	1	Node strength, closeness, betweenness	-	-	36	Pearson	GGM	GLASSO	EBIC ($\gamma=0.5$)	-	-	-	Code	Gold

Note. - = data not reported. BPD=Borderline Personality Disorder.

^aGGM=Gaussian Graphical Model, Ising=Ising Model, FGL=Fuesd Graphical Lasso; ^bGLASSO= Graphical least absolute shrinkage and selection operator, λ_2 =Lamda tuning parameter; ^cEBIC= Extended Bayesian Information Criterion, γ =Gamma hypertuning parameter; ^dFIML= full information maximum likelihood; ^eCS=correlation stability; ^f95% confidence intervals; ^g Gold=Gold open access, Green= Green open access.

Results of quality assessment. The results of the quality assessment will only be discussed regarding the second, third and fourth section of Table 2, as these are directly associated with quality assessment.

Network estimation. The number parameters were compared with the sample sizes based on the rule of thumb of Fried and Cramer (2017). Four studies were found to have less than optimal sample sizes (Buelens et al., 2020; Esmailian et al., 2019; Knefel et al., 2016; Rivnýák et al., 2021). Furthermore, only three of the studies reported the type of correlation applied for the estimation of network models (Knefel et al., 2016; Peckham et al., 2020; von Klipstein et al., 2021). In general, when psychological network studies used GLASSO models and EBIC selection, they typically used a polychoric correlation matrix as the input for their partial correlations, but the poor documentation of the type of correlation used across the current studies is important to note as it means that this can only be assumed (Epskamp & Fried, 2018).

Regarding the analytic method, most studies used GGM models, except from Richetin et al. (2017) and Southward and Cheavens (2018), who chose FGL models, and Peckham et al. (2020), who chose the Ising model. All studies used GLASSO regularisation technique. However, justification of the model selection was rarely reported, especially in relation to the data type used for the models. Similarly, the value of the GLASSO tuning parameter (λ) was almost never reported, with the exception of Richetin et al. (2017), who documented it alongside the λ_2 value used for the FGL model. While the λ can be informative, the more important information is the value of the EBIC tuning parameter (γ), as this indicates the number of edges removed in the effort of reducing false positive edges (Type 1 error) and retaining only the edges that represent the true network structure (Burger et al., 2020; Epskamp & Fried, 2018). Two studies did not report the value of the γ in their studies (Richetin et al., 2017; Rivnýák et al., 2021). Most studies opted for a γ -value of 0.5, which is a rather cautious and parsimonious value (Esmailian et al., 2019; Köhne & Isvoranu, 2021) that ensures fewer edges are retained in order to maintain higher specificity (rate of true-negatives) than sensitivity (rate of true-positives). This stricter approach can however lead to some true edges not being identified (Type 2 error; Epskamp & Fried, 2018; Foygel & Drton, 2010). Only two studies chose lower γ -values (Buelens et al., 2020; Knefel et al., 2016), and these therefore should be considered as more exploratory, with the ability to discover further potentially true positive edges but a higher risk of identifying false positive edges (Epskamp & Fried, 2018). Lastly, four studies reported how missing data was handled; three used multiple imputation (Buelens et al., 2020; Peckham et al., 2020; Southward & Cheavens, 2018) and

one used the FIML method (Knefel et al., 2016). A further two studies reported that no data was missing (Esmailian et al., 2019; Köhne & Isvoranu, 2021), while the rest of the studies did not report on missing data at all.

Network stability. Of those studies that examined centrality indices, three did not conduct case-drop bootstrapping stability analysis for these values (Richetin et al., 2017; Rivnyák et al., 2021; von Klipstein et al., 2021). Furthermore, only half of the studies reported edge-weight accuracy (De Paoli et al., 2020; Richetin et al., 2017; Southward & Cheavens, 2018; von Klipstein et al., 2021), meaning that symptom connectivity should be interpreted cautiously in the rest of the studies.

Reproducibility. Transparency is essential for replicability and integrity of research. Three studies did not provide open access to their data or correlation matrices (Knefel et al., 2016; Köhne & Isvoranu, 2021; Peckham et al., 2020). Southward and Cheavens (2018) and von Klipstein et al. (2021) shared their R analysis script but not their data. Overall, eight studies made their papers freely available: five with Gold open access, and three with Green open access. This left only two studies that provided no free access to their research (Knefel et al., 2016; Peckham et al., 2020).

Synthesis of systematic review findings. The following paragraphs summarise the narrative results of the systematic review in the order of the research questions.

Q1: Networks of BPD-related difficulties. Four studies conducted a network analysis that only included difficulties that are related to the borderline phenomena (Peckham et al., 2020; Richetin et al., 2017; Southward & Cheavens, 2018; von Klipstein et al., 2021).

Richetin et al. (2017) compared the network structure of BPD-related difficulties between a community and a clinical sample; however, their clinical sample had several limitations. The clinical sample size was small, allowing only tentative interpretation of the findings.

Furthermore, the individuals did not have a BPD diagnosis prior to participating in the study. Instead, SUs were assessed with a DSM-oriented clinical assessment tool that evaluated their experiences of BPD-related difficulties in the month before their hospital admission (but they were all included regardless of the severity of problems). Over 85% of people in this sample had more than one psychiatric diagnosis. Southward and Cheavens (2018) used a considerably larger but mixed sample, recruiting people who did and did not seek psychological treatments and comparing them based on high and low levels of BPD traits. The sample used by Peckham et al. (2020) involved the first large clinical sample.

Participants with a range of psychiatric diagnoses were included if they met the “threshold” for BPD on a self-report questionnaire, meaning this sample was rather transdiagnostic in

nature. The clinical sample of von Klipstein et al. (2021) however only included participants with a primary diagnosis of BPD, while also appearing to be large enough to show robust connections.

In terms of the findings (see Table 1), Richetin et al. (2017) and Southward and Cheavens (2018) examined differences in the networks of people with less and more severe BPD-related difficulties. Richetin et al. (2017) found that the general network structure of the community and clinical samples (corresponding to samples of people with low and high BPD-related difficulties) was similar. Affective instability, identity disturbance and fear of abandonment were central nodes in both samples. Southward and Cheavens (2018) found partially overlapping results between the networks of people with low and high BPD-related difficulties (and their whole sample), identifying affective instability and mood disturbance as central nodes in both samples. Additionally, in the people with low BPD-related difficulties, emptiness and unhappiness were also central nodes, while for people with high BPD-related difficulties, loneliness and recklessness/impulsivity showed high centrality. There were no gender differences in the networks. In line with both previous studies, Peckham et al. (2020) also found that affective instability was a central node in the network of BPD-related difficulties amongst their heterogeneous clinical sample. Furthermore, they found that anger and lack of trust were also central nodes. Importantly, von Klipstein et al. (2021) also found that affective instability was a central node in the network of individuals with a primary diagnosis of BPD. The consistency of this finding suggests that changes in the degree of affective instability might play a particularly important role in activating or maintaining in BPD symptomatology.

In terms of significant edges (depicting conditional independence relations), Richetin et al. (2017) found unique relationships between aggression and impulse control and between certain psychological states and processes, such as affective instability and emptiness in their high BPD-related difficulties sample. Southward and Cheavens (2018) found similar connections between affective instability, loneliness, unhappiness and boredom in people with high BPD traits. Peckham et al. (2020), on the other hand, split their sample into younger and older participants with BPD (below and above of the age of 46 respectively). They found relationships between NSSI/suicide and emptiness in the younger group of people, while this connection seemed weaker in the older group. The older participants instead showed a stronger relationship between anger and relationship problems.

Q2: Additional factors. As Table 1 outlines, several of the other six reviewed studies included additional factors such as other mental health difficulties or unhelpful and altered cognitive functions in their network analyses of BPD-related difficulties. Some also assessed comorbidities and transdiagnostic relationships between BPD and other mental health disorders. The studies that assessed cognitive, emotional and behavioural factors included identity diffusion (Rivnyák et al., 2021), early maladaptive schemas (Esmailian et al., 2019), interpersonal difficulties (Southward & Cheavens, 2018) and emotional dysregulation (De Paoli et al., 2020; Southward & Cheavens, 2018), showing several important connections between these constructs and BPD-related features. The study of De Paoli et al. (2020) also included theory of mind (ToM), attachment style, emotion recognition and rejection sensitivity. ToM is a complex construct which refers to the ability to assign mental states to others and anticipate their behaviour based on those based on perspective taking (Baron-Cohen, Golan, Chakrabarti, & Belmonte, 2008). De Paoli et al. (2020) found that ToM showed very few and inconsistent connections with BPD-related difficulties and the additional factors.

In the context of BPD, the role of interpersonal difficulties is an important additional factor that was incorporated into the network of Southward and Cheavens (2018), as it might be particularly significant in relation to mentalisation (Fonagy & Bateman, 2008). In people with low BPD features, interpersonal difficulties were linked to BPD via chronic emptiness (which was a central node in the network), while in people with more profound BPD features, interpersonal difficulties were linked to BPD via the non-acceptance of one's own emotions. This may suggest that in people with less severe BPD-related difficulties, increased feelings of detachment from themselves and others might lead to more interpersonal difficulties (Miller, Townsend, Day, & Grenyer, 2020), whereas in those with more severe BPD traits, interpersonal difficulties seem to be associated with irritation related to their own negative feelings. The latter could be explained by their struggle to tolerate even minor upsets or frustrations, which in turn can trigger intense and uncontrolled emotions and anger outburst towards others as a way to regulate negative emotions (Fonagy et al., 2002; Keltner & Kring, 1998). While they only measured interpersonal difficulties as part of the BPD symptomatology, Peckham et al. (2020) found similar dynamics in the older age group of their clinical sample, where the network highlighted an important link between anger and interpersonal problems.

The findings by Esmailian et al. (2019) might illustrate the cognitive processes that accompany this relationship between the irritation by one's negative feelings and

interpersonal problems. Their findings demonstrated connections between interpersonal problems and abandonment and mistrust/abuse schemas. These schemas represent one's expectations about the unreliability and unpredictability of significant others' devotion and support, as well as the expectation that one would be abused or taken advantage of (Arntz & van Genderen, 2009). It has been hypothesised that the activation of these schemas may lead to intense emotional pain and a resurfacing of internalised punitive parents, which in turn results in the emergence self-punitive thoughts and behaviours (Arntz, Klokman, & Sieswerda, 2005). Feelings of anger and frustration therefore remain suppressed and build up until they eventually become expressed in an uncontrollable and impulsive need-gratifying manner, thereby undermining the relationships of a person (Arntz et al., 2005).

Table 1 outlines the key findings of the four studies assessing the role of comorbidities within their network analyses, including PTSD and Complex PTSD (Knefel et al., 2016), NSSI (Buelens et al., 2020), major depression (Köhne & Isvoranu, 2021) and eating disorder (De Paoli et al., 2020). Across these studies, BPD established itself as distinct from all other generally accepted mental health diagnoses, including PTSD and complex PTSD, NSSI, eating disorder and major depression (Buelens et al., 2020; De Paoli et al., 2020; Knefel et al., 2016; Köhne & Isvoranu, 2021). Nevertheless, several transdiagnostic bridge symptoms were identified. For instance, loneliness and impulsivity, which were central features in the network of people with higher BPD traits in the study by Southward and Cheavens (2018), were important bridge symptoms between NSSI and BPD, suggesting that feeling lonely and acting impulsively might be associated with NSSI for people with more severe BPD difficulties in particular. This might be connected to the emotion deprivation and failure schemas that NSSI was associated with (Esmailian et al., 2019). The expectation that one's emotional needs will not be met and one would be seen as fundamentally inadequate in the eyes of others might activate self-punitive behaviours as a way to regulate these difficult feelings (Young, Klosko, & Weishaar, 2003). This process might be also facilitated by the impulsivity and loneliness that people with elevated BPD traits often report, as well as by the experience of emptiness, especially in adolescence (Peckham et al., 2020).

Q3: Studies incorporating mentalisation. No network analysis studies incorporated a measure of mentalisation. The study of De Paoli et al. (2020) included the closest construct to mentalisation, namely ToM. While theory of mind and mentalisation are often used interchangeably, there are clear distinctions between the two concepts (Górska & Marszał, 2014). Mentalisation is a broader concept, encompassing several socio-cognitive functions

(such as emotion recognition and reflective functioning) alongside ToM (Ha et al., 2013). Furthermore, while mentalisation requires “hot knowledge” in terms of relational representations and the processing of emotional experiences, ToM refers to a “cold” knowledge of internal states closely related to perspective-taking abilities and detection of false beliefs (Górska & Marszał, 2014). De Paoli et al. (2020) also did not differentiate between different types of ToM difficulties (cognitive and affective). By including the Reading the Mind in the Eyes task, they measured affective ToM only (Gallant & Good, 2020).

Summary of the Findings of the Systematic Literature Review. The above systematic review revealed that several studies have been published using network analysis as part of a cross-sectional research design to understand the complex difficulties that are currently conceptualized as BPD. Affective instability was generally found to be of central importance, implying that emotional dysregulation and volatility might activate several other BPD-related difficulties or even the overall system. While some studies found similarities between network structures, several differences on the most important difficulties and the symptom-symptom connectivity were demonstrated as well.

The review also showed that additional cognitive, emotional and behavioural factors, alongside symptoms of various clinical diagnoses may influence the activation of BPD-related networks. As mentioned, only one study (De Paoli et al., 2020) included an affective ToM measurement, but no study has thus far incorporated different types of ineffective modes of mentalisation.

The Current Study

The network approach offers an appealing theoretical framework to explore the network involving BPD-related symptoms while incorporating ineffective modes of mentalising. This would move away from understanding BPD as a single- or multi-dimensional latent disorder associated with a range of corresponding underlying symptoms. Instead, it focuses on specific symptoms and their connectivity that may cause and maintain the deficits in the form of vicious, intricate and complex feedback loops across several levels of system activation. It might furthermore facilitate a conceptualisation of BPD that combines aspects of the traditional categorical and dimensional diagnostic methods. Importantly, this approach would still provide an effective technique to identify individuals in need of support, since network structures of those suffering from more versus less severe difficulties would likely differ. Finally, the network approach allows for the inclusion of various mentalisation

impairments. Research has consistently provided support that these impairments exist in individuals with BPD but they are currently not incorporated in the symptom-based diagnostic criteria. None of the systematically reviewed network analysis studies have incorporated mentalisation in their models. Including ineffective modes of mentalisation into the network models of BPD-related difficulties would enable the investigation and comparison of the unique connections between these features and across different system activation levels. This might offer further insight into the effects of poor mentalisation across different severity of BPD-related difficulties, which could ultimately inform about the rationale for therapies targeting the improvement of mentalisation.

Study Aim

Overall, the study aims to explore the ways in which ineffective modes of mentalisation activate BPD-related difficulties in those who have a diagnosis of BPD versus those who do not. To achieve this, the connections and complex interactions between mentalisation impairments (namely hypermentalisation, hypomentalisation and total lack of mentalisation) and the network of BPD symptoms will be examined in the two samples. Based on the mentalisation-based model, it is hypothesised that connections between various mentalisation impairments and BPD-related features will be found, and that the patterns of these networks will differ between the two samples of participants.

Chapter Two: Methods

Epistemological Positioning

Epistemology has been defined as the “theory of knowledge”, concerned with the body of concepts and theories that focus on questions related to truth, reality, justification and the searching for the means and conditions of knowledge (Audi, 2011). Ontology is related to epistemology, as it seeks the classification of entities, aiming to explain the form and nature of objects, processes and relations in the social world (Smith, 2012). Psychological research has long been dominated by the theory of empiricism, which recognises reality as universal, objective and quantifiable. Positivism is a paradigm founded on empiricist philosophy, which posits that human behaviour in the social world can be examined the same way as in the natural world, through the means of experimentation and the measurement of what can be observed (Mertens, 2009). In recent decades, the positivistic paradigm in psychological research has been succeeded by postpositivism, which still values objectivity and generalizability, but bases research conclusions and claims about the truth on probability, rather than certainty (Mertens, 2009). This is related to the ontological standpoint called critical realism, which assumes that reality exists but due to the human limitations of the experimenter, it can only be calculated imperfectly, within the realm of probabilities (Maxwell, 2004). Recognising that researchers’ personality, beliefs and background knowledge can influence observations to a great extent, this paradigm attempts to ensure objectivity by eliminating the personal biases of the investigators via increased awareness and standardised research protocols that are followed rigorously (Mertens, 2009; Reichardt & Rallis, 1994).

The network analysis approach is a psychometric tool that simulates complex systems based on empirical data via statistical estimation and model fitting techniques (Dalege et al., 2016). Both the collection and estimation of data occur within a postpositivist framework, with the researchers collecting observations using standardised research protocols, calculating probability values for network models and drawing generalisable conclusions about the global structures of their components (Cramer et al., 2012). In line with the postpositivist paradigm, the network theory acknowledges that no model is likely to capture the full complexity of mental health problems that might be sustained (Kendler, Zachar, & Craver, 2011). However, the approach improves on the ontological problems of the traditional psychological views that tend to lie within the realms of both realism and essentialism, which view psychological attributes as latent realities with a number of underlying objects that can,

to a varying degree, be characterised and classified by the researchers (Guyon, 2018; Zachar, 2010). The conceptualisation of mental health disorders as unique latent biopsychological entities, mostly independent from social context, has been criticised by network researchers for overlooking the high levels of heterogeneity and comorbidity consistently reported in clinical practice (Cramer et al., 2010; Fried, 2015). Instead, psychological phenomena and the related terms are understood “as the result of the biological, psychological, and environmental forces that knit some behaviors closely together” (Cramer et al., 2012, p. 453.), and are perceived to emerge from the interactions and connections between intra- and interpersonal factors. In that sense, the shared similarities in people’s mental health problems do not arise from the same underlying entity, but from complex interactions and evolutions between one’s physiology, behaviour and the surrounding environment (Kendler et al., 2011). “Illnesses” are conceptualised across time and cultures by a relatively stable sets of central traits that are mutually reinforcing in nature, leading to a general identification of mental health disorders and to more prototypical cases, while allowing room for those also who might be less typical in their presentation (Kendler et al., 2011).

While the latent variable model is questioned, the network approach accepts the reality of psychological attributes and mental health problems, conceptualising them as emerging self-sustaining equilibrium states consisting from a variety of symptoms, behaviours, emotions and contexts, inseparable from the social environment (Cramer et al., 2012; van Geert & Steenbeek, 2010). Contrary to the constructivist epistemological view that suggests that mental health disorders are socially constructed and “symptoms” hang together conveniently without describing a homogenous group of people, the network theory assumes that the pattern of these components forms in a non-arbitrary interconnected manner (Borsboom, 2008; Cramer et al., 2012). Therefore, the current research remains situated within the postpositivist epistemological paradigm, being phenomenological, empirical and objective in its scientific nature. Ontologically, although the network approach aligns itself with critical realist paradigm in emphasising probability values and focusing on underlying discourses about mental health disorders (Alvesson & Skoldberg, 2009), it is viewed as a separate paradigm, offering an alternative theory to the realism about underlying variables and mental health disorders, without collapsing into relativism as offered by the constructivist epistemological paradigm (Borsboom, 2008). As Guyon, Falissard, and Kop (2017) suggest, the materiality of psychological attributes in the network approach are seen as both objective (as a complex system generating emerging properties that cause mental distress) and intersubjective (the way society or culture conceptualises the emergent properties).

Nevertheless the speculative nature of the current study is emphasised, alongside the necessity to validate its results across various cultures and sub-populations.

Design

The current study used data collected as part of a large research project titled “*Probing Social Exchanges – A Computational Neuroscience Approach to the Understanding of Borderline and Anti-Social Personality Disorder*”. As an international research collaboration dating back to 2012, it explored social cognition and mentalisation using behavioural and neuroimaging techniques in adults with and without a diagnosis of BPD (i.e. Michael et al., 2021; Rifkin-Zybutz et al., 2021; Stagaki et al., 2022).

The design of the current study was a cross-sectional correlational analysis, whereby a network including BPD-related symptoms (defined by the items of the PAI-BOR), alongside three types of ineffective modes of mentalisation (defined by three subscales of the MASCS) was created as a mathematical representation of conditional associations between the difficulties. To investigate the structure of this network and to understand the complex associations and interplays between the symptoms, network analysis was applied as the most appropriate statistical technique to identify network architectures (Borsboom, 2017b). Networks were compared to each other across the levels of the independent variable, which was defined as group membership. For the sake of brevity and clarity, the group of people without a BPD diagnosis were referred to as the community group and the group of people with a BPD diagnosis were referred to as the clinical group.

Recruitment

Although the participant sampling strategy was clinically principled, the recruitment of the patients in the larger study followed a non-probabilistic consecutive sampling method to ensure a representativeness of the clinical sample. The clinical cohort of the study was recruited from inpatient, outpatient and community personality disorder services in the following Greater London NHS trusts: North East London NHS Foundation Trust; West London NHS Foundation Trust; South West London and St. George’s NHS Foundation Trust; Barnet, Enfield, and Haringey NHS Foundation Trust; Camden and Islington NHS Foundation Trust and Central and North West London NHS Foundation Trust. Patients were recruited during their clinical assessment periods and while they were on waiting lists or in early stages of their treatment. First, clinicians identified eligible service users (SUs) and provided basic information about the study if they were interested. If they wished to proceed,

the clinicians acquired the SU's consent and shared their contact details with research team, who pursued communication and screened them against eligibility criteria (see below).

The recruitment of community controls followed a non-probabilistic purposive sampling method. Controls were recruited from across Greater London via posters and online advertisements which provided study information and contact details, enabling interested individuals to contact the research team. Upon communication with the research team, interested individuals were recruited if they met eligibility criteria and were characterised by similar demographic characteristics as the clinical sample. This purposive sampling approach enabled closer demographic matching of the samples according to age, gender, education and socioeconomic status (SES).

Eligibility criteria.

Inclusion Criteria. To be eligible for inclusion in the current study, participants were aged between 18-60, had to understand and write in English proficiently, had to have normal vision and the ability and willingness to attend at least two assessment sessions lasting for several hours. Participants of all genders were included. For the SUs, a suspected or confirmed primary diagnosis of BPD or antisocial personality disorder (ASPD) was an essential part of the inclusion criteria.

Exclusion Criteria. Participants were excluded if they reported current or past history of neurological disorders or trauma including epilepsy, head injury and loss of consciousness. People with a learning disability requiring specialist educational support or medical treatment were also excluded. For the clinical sample, SUs with a primary psychotic or mood disorder or a substance use disorder causing a significantly altered state of mind were not eligible. In the community controls, individuals with any current or past diagnosis of a psychiatric or personality disorder were excluded.

In the clinical sample, the presence of a BPD or ASPD diagnosis was verified by the research team using the SCID-II. In the community sample, the absence of any PD was confirmed by evaluating the Standardised Assessment of Personality-Abbreviated Scale questionnaire. Anyone who scored above the threshold of four, was conducted a SCID-II with. Those who met the criteria for BPD were re-assigned to the group of people who had an official clinical diagnosis, as to eliminate bias in the severity of problems across the two groups. Further information about the psychometric characteristics of the SCID-II is provided below (see Materials).

Study Procedures

Interested and eligible participants were offered two or three personal appointments lasting 3-4 hours each. On the day of their first appointment, all participants received a detailed information sheet of the study. After discussing any remaining questions, participants provided informed signed consent and the researcher initiated the study tasks. Participants were usually asked to attend the research appointments within 2 weeks to minimize the risk of external fluctuations. Communication was upheld via a range of media (e.g. text messaging, email, phone call etc.).

The assessments took place in Wellcome Trust Centre for Neuroimaging (WTCN) and Experimental Psychology Research Department at University College London (UCL). The participants completed clinical/psychiatric, behavioural, cognitive and social cognitive assessments, as well as a range of self-report questionnaires. To reduce the workload while attending the appointments and only if preferred by the participant, they were given detailed explanations of the questionnaires and could complete these at home between appointments. Participants received a remuneration of £10 per hour for their time and effort, and travel as well as refreshment costs were reimbursed. All participants were assigned a unique study ID to pseudo-anonymise the data. Data was and remains stored in secure, anonymised electronic databases and locked storage in the building of WTCN.

Participants

Recruitment took place until March 2020. In total, 971 participants were enrolled in the study, all whom had a primary diagnosis of BPD and ASPD or were considered community controls. For the current study, individuals referred with a primary diagnosis of ASPD were excluded from the analyses, leaving 658 participants. Nine people withdrew from the study, while three cases were excluded due to errors associated with their data entry, leaving 646 participants for the current analysis. The average age of the participants was 30.24 years ($SD = 10.18$). The majority identified as female (73.8%) and White (68.3%).

Materials

All participants reported their age, gender, ethnicity, level of education and SES. The below only describes the materials selected from the larger project which were used in the current study.

BPD-related difficulties. To quantify BPD-related features, the Personality Assessment Inventory - Borderline Personality Feature Scale (PAI-BOR) was employed

(Morey, 1991). The PAI-BOR is a self-report instrument, intended to measure BPD-related psychopathological features on a 4-point Likert scale. Participants were asked to rate how accurately a set of 24 items described their actions and feelings on a scale from 0 (“Not at all”) to 3 (“Very True”, see Appendix A). The items of the measurement were developed to assess specific characteristics of BPD (e.g. “*My mood can shift quite suddenly*”) and they tap directly into the DSM-4 and DSM-5 criteria of the construct (American Psychiatric Association, 2013; Morey, 1991). Theoretically, it is possible to compute a total score and a score for four subscales based on the items of the questionnaire, where the subscales represent the historically conceptualized central “factors” or BPD: Affective Instability, Identity Disturbance, Negative Relationships and Self-Harm (Morey, 1991). However, the statistical analysis detailed below uses these specific items of the questionnaire and the raw values that people indicated for them (0-3) in order to preserve as much information of the specific BPD-related difficulties as possible. Before conducting the relevant analyses, reverse score transformation was carried out on the respective items. Reverse scored items are indicated in Table 4.

The PAI-BOR has been effectively used in various settings and both clinical and non-clinical populations, and displays adequate psychometric properties (Jacobo, Blais, Baity, & Harley, 2007). Convergent and discriminant validity (Bell-Pringle, Pate, & Brown, 1997; Morey, 1991; Trull, 1995) as well as predictive validity of the measurement have been demonstrated in numerous studies, stressing its utility not only in assessing BPD-related features, but also in examining the aetiology and development of the disorder (Trull, Ueda, Conforti, & Doan, 1997). External reports of internal consistency suggest this is high, with an average Cronbach’s alpha of .88 across clinical and non-clinical samples, and its test-retest reliability was also found to be high ($r = .86$; Morey, 1991). In this sample, the total score had excellent internal consistency (Cronbach’s alpha = .95), and the subscales had good internal consistency (Cronbach’s alpha = .80 - .89).

Ineffective mentalisation. To measure different types of ineffective mentalisation strategies, the Movie for the Assessment of Social Cognition (MASC) was used (Dziobek et al., 2006). The MASC is a video-based behavioural assessment measuring subtle mindreading abilities of participants while watching a 15-minute movie about four people at a social gathering (Dziobek et al., 2006). This movie contains 45 pauses. During each pause, participants answered a multiple choice question about the mental states (such as intentions, emotions and thoughts) of the individuals seen in the movie. The multiple choice questions include four answer options: 1) correct answer (appropriate mentalizing), 2)

hypermentalizing/over-mentalizing (excessive attribution of mental states without observable data to justify it), 3) hypomentalizing/under-mentalizing (misattribution of mental states due to reduced mentalizing), and 4) lack of mentalizing (total absence of inferring mental states in social situations, inferences are drawn based on physical causations). Four sum scores reflected the number of times each answer was chosen, with one score for 1) correct answers (MASCcorr), and three scores for the different types of errors: 2) hypermentalizing (MASCexc), 3) hypomentalizing (MASCless) and 3) lack of mentalising (MASCno). At the end of the task, six control questions were asked to test the participant's attention and their ability to draw non-social inferences.

The MASC captures mentalization in a multidimensional manner as it requires the integration of visual, auditory and verbal input channels (e.g. facial expression, tonality and linguistic messages) to infer mental states of the characters (Lahera et al., 2014). Convergent and discriminant validity of the measurement has been established on several occasions (Dziobek et al., 2006; Fossati et al., 2018; Preißler, Dziobek, Ritter, Heekeren, & Roepke, 2010) and test-retest reliability was also found to be high ($r = .97$; Dziobek et al., 2006). Adequate and high internal consistency of the MASC was demonstrated in both clinical ($\alpha = .78$) and non-clinical samples respectively ($\alpha = .80$; Fossati et al., 2018).

In this study the three subscales of ineffective modes of mentalisation were used: hypermentalising, hypomentalising and total lack of mentalising. The number of total correct answers was not included in the analysis. Since the total correct mentalisation scale is in a direct relationship with the scores of three ineffective modes of mentalisation scales (MASCcorr score = total score - sum of MASC error scale scores), this scale may eliminate some of the variance that is of interest when interpreting the 3 mentalisation impairments by introducing bias in the model, which in turn may focus on the exploration of relationship between the MASC scales rather than the relationship between the MASC scales and PAI-BOR items.

Severity of BPD-related difficulties. In the clinical sample existing BPD diagnosis was confirmed by the SCID-II (First, Spitzer, Gibbon, & Williams, 1995), while in the community sample it was used to identify participants with high severity of mental health problems. The SCID-II is a semi-structured assessment interview for clinical disorders and PDs (First & Gibbon, 2004). Studies show excellent inter-rater reliability for the SCID-II BPD diagnosis ($\kappa = .909$; Maffei et al., 1997) as well as adequate convergent and divergent item validity, and adequate associations of these with general PD features and functional impairments (Ryder, Costa, & Bagby, 2007). In the current study, the SCID-II interviews

were conducted by highly trained and experienced researchers who received regular supervision to ensure consensus across scoring. One person from the community group met the criteria for BPD diagnosis as rated by the SCID-II. This person was moved to clinical group before any further statistical analysis was conducted.

Data Analysis

All statistical analyses were computed in R, version 4.0.5 (R Core Team, 2021) and IBM SPSS Statistics, version 18.0.

Missing data analyses.

Exclusion of fully missing data. In the first instance, participants who did not complete either the PAI-BOR questionnaire or the MASC task were excluded from the analyses, leaving a total of 575 people in the study. A chi-square test of association and Mann-Whitney U tests were conducted in SPSS to examine whether this excluded group significantly differed from the remaining participants in terms of gender, age, ethnicity, education, employment, SES and household income (Field, 2013).

Data imputation. As a second step of missing data analysis, missing data was imputed where possible and required. The R “*mice*” package was applied to run a multiple imputation on the basis of the first available set of data for missing items of the PAI-BOR (van Buuren & Groothuis-Oudshoorn, 2011). For the MASC, missing scores could only occur due to non-completion of the task, meaning a systematic pattern of missingness would appear across all three error types. Imputing data that is not missing at random would likely introduce a bias in the estimate of effects (Mack, Su, & Westreich, 2018). As mentioned above, this meant that all participants who did not have data for the MASC were excluded.

Preliminary analyses. A series of preliminary statistical tests were conducted to determine whether there were differences in the scores of the PAI-BOR and MASC subscales between the group of people with and without a BPD diagnosis. The PAI-BOR and MASC data was not normally distributed (as assessed by visual inspection of their histograms), so non-parametric Mann-Whitney U-tests were done using SPSS (Field, 2013).

Nonparanormal transformation. Since the relevant variables were not normally distributed, the R “*huge*” package was used to apply nonparanormal transformation on the dataset in order to improve normality and ensure that the assumption of normality of residuals distribution has been met for all regression models (Epskamp & Fried, 2018; Haslbeck, 2022; Haslbeck, Borsboom, & Waldorp, 2018; Zhao et al., 2012). Nonparanormal transformation employs cumulative distributions to change the distribution of the observed variable by

mapping every specific outcome of the observed variable to a specific value of a standard normal variable (Epskamp & Fried, 2018).

Item redundancy. Network analysis operate on the assumption that the nodes of the network represent singular entities measuring distinct constructs (Peckham et al., 2020). If this assumption is not met and the examined items (PAI-BOR items and MASC error subscales), or nodes, load onto the same underlying construct (or a smaller latent variable), these items are redundant and the network will have poor model fit with heightened risk of false positive correlations (Christensen, Garrido, & Golino, 2020; Santiago et al., 2021). Unique Variable Analysis (UVA) was used to statistically identify potential redundant variables in the data and therefore minimize inaccurate estimates of dimensional structures (Christensen et al., 2020). The analysis was conducted in R using the “*EGAnet*” package. Following the recommendations of Christensen et al. (2020), weighted topological overlap statistics were estimated (wTO) with an adaptive alpha (Pérez & Pericchi, 2014; Zhang & Horvath, 2005). Wherever redundancies were identified between a set of items, instead of removing those (which would have led to the loss of significant information), the items were combined into new minor latent factor. Decisions about whether the identified potential redundancies should be combined into a new variable were led by clinical judgement of the researcher and will be reported in the Results section.

Mixed Graphical Model analysis. Since the data was a combination of ordinal (PAI-BOR), continuous (MASC) and binary data (BPD diagnosis), Mixed Graphical Models were used ([MGM]; Haslbeck & Waldorp, 2020) to determine whether there are differences in the patterns of connectivity in the networks of the group of people with and without a BPD diagnosis when including mentalisation in the network of BPD-related difficulties.

This method is based on multivariate Gaussian distribution and applies a l_1 -regularised [LASSO] nodewise regression, estimating regression coefficients that represent edge weights (Haslbeck et al., 2018). The regression coefficients calculated by the model depict conditional dependence relations between the nodes after the influences of all other nodes were controlled for (Haslbeck et al., 2018). The l_1 -regularization is employed to avoid overfitting of the model by having the underlying assumption (called the sparsity assumption) that most parameters in the true model equal to zero (Haslbeck et al., 2018). In line with this assumption, the thickness of edges represent the strength of associations and weak edges are being shrunk to zero (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012; Haslbeck & Waldorp, 2020).

The conditional pairwise effects are calculated by using regularized nodewise regression (each variable is regressed on all other, and the results are combined to generate the network). They can be interpreted as partial correlations, with a value ranging from -1 to 1 (Burger et al., 2020; Haslbeck et al., 2018). The value of the partial correlations was evaluated according to the guidelines by Doucouliagos (2011), who conducted a large-scale meta-analysis based on empirical effect sizes of economics papers and updated the guidelines suggested by Cohen (1988). Cohen originally developed these for zero order correlations with no covariates. Since these latter correlations are rarely the case in the field of psychology, the qualitative categories suggested by Doucouliagos (2011) were felt to be more appropriate. According to these, a partial correlation effect size less than ± 0.07 is small, between 0.07 and 0.33 is moderate, and greater than ± 0.33 is large.

For each node-wise regressions calculated, a tuning parameter (λ_s) is used to control the strength of penalty. To select an optimal value of λ_s , 10-fold cross-validation scheme was used (Haslbeck et al., 2018). This works by dividing the dataset into 10 non-overlapping folds, which all are used as held-back datasets, while all the other folds are employed as training datasets (Kuhn & Johnson, 2013). 10 models are fit and calculated on the 10 hold-back test sets, with the mean performance getting reported (Kuhn & Johnson, 2013). The OR-rule was used to combine the mean of the edge weight for the three-way interactions as default, since the AND-rule might be too conservative for the estimation of three-way interactions (Haslbeck et al., 2018). All variables were mean-centred by default before estimation (Haslbeck et al., 2018). The networks were computed in R with the “*mgm*” package and visualised with the help of the “*qgraph*” package (Epskamp et al., 2012; Haslbeck & Waldorp, 2020).

Group comparisons via moderation analysis. The calculated pairwise interactions through the MGM extend standard GGMs by allowing for certain variables to become moderators between two variables in the form of three-ways interactions (Haslbeck et al., 2018). As the result of the analysis, networks using the data of the whole sample and of the two groups were computed and conditioned on BPD diagnosis, to illuminate the differences in the network structures of BPD- and mentalisation-related difficulties between those who have a BPD-diagnosis (and therefore presumably suffer from more severe mental health difficulties) and those who do not. MGMs have been reported to outperform various other split-sample based methods (e.g. Network Comparison Test or Fused Graphical Lasso models) in finding differences between groups and moderation effects (Haslbeck et al.,

2018). Differences in pairwise associations across the two groups are reported in terms of the strength and direction of conditional dependencies between variables.

Subgroup analyses. To investigate how the moderation effect of BPD diagnosis impacts the pairwise associations found through the moderation network analysis, subgroup analyses of both groups were conducted using the same MGM method described above (without the inclusion of the independent variable). Estimating network parameters within each group separately complement the understanding of moderation effects found in the analysis of the whole network and highlights where the difference lies within the relationships detected in the two groups of people.

Stability and reliability. To calculate the stability and reliability of the estimated parameters, all networks were resampled 1000 times to acquire confidence intervals around the edge weights and moderation effects (Parsons, Songco, Booth, & Fox, 2021). The process involves bootstrapping, allowing for the extraction of the amount of moderating effects and non-zero edges (Parsons et al., 2021). Estimated means of the edge weights (which represent the strengths of dependency between variables) were calculated with low (5%) and high (95%) quantiles of the bootstrapped sample distribution. This is translatable to 95% confidence intervals. Narrower confidence intervals represent smaller variance for pairwise interaction effects in the sampling distribution, which implies stability of the estimated parameters. The mean effect of the moderator variable on the dependency between pairs of variables was also calculated with corresponding confidence intervals.

Global strength and structure. Since the MGM model does not allow for direct significance test in global structure, the Network Comparison Test was conducted as a complimentary analysis for detecting significant differences in global strength (overall connectivity=absolute sum of all edges) and global structure (maximum difference in any of the pairwise edge weights) of the two groups, applying GLASSO regularization (van Borkulo et al., 2022). To achieve this, differences in edge weights are pulled and compared between the networks of the two groups, which are then repeatedly re-assigned randomly into two groups to estimate several new pairs of networks. This procedure leads to a reference distribution of differences between the networks, serving as the null hypothesis that the networks represent the same population. After this, maximum differences in global strengths values and edge weights are identified and compared to this reference distribution. If the observed difference is greater than 95% of the reference distribution, the null hypothesis is rejected, meaning that the networks are different in global strengths and structure as a whole.

The “*NetworkComparisonTest*” package was employed in R, using 10,000 permutations for the comparison procedure.

Expected influence. In order to measure centrality, expected influence (EI) of the nodes was calculated using the R “*networktools*” package. EI is understood to be the most accurate measure to estimate node centrality in networks that contain both positive and negative edges, as it represents the summed weight of all the edges extending from a given node, while also maintaining the sign of those (Robinaugh et al., 2016). One-step EI evaluates the nodes’ influence on its immediate neighbouring nodes, connected by an edge (Robinaugh et al., 2016). While two-step EI also accounts for the immediate effect of nodes on its neighbours, it incorporates also the secondary influence that a node has on the whole network via its neighbours, evaluating the EI of the neighbouring nodes as well (Robinaugh et al., 2016). Both one-step and two-step EI were reported.

Predictability. As part of the MGM analyses, nodewise predictability was explored, which illuminates the proportion to which a node can be predicted by all the other nodes it connects with (Haslbeck & Waldorp, 2018). For continuous variables (all variables in this case), the amount of explained variance is reported (for binary variables the accuracy, or proportion of correct classification is measured) (Haslbeck & Waldorp, 2018). Node predictability estimates the proportion of influence we can have on a node (X) via the nodes it shares edges with, assuming that all these edges are directed towards the node in question (X) (Briganti, Fried, & Linkowski, 2019). While EI is considered to be a relative measure of node centrality, predictability is understood as a more “objective” centrality index, since it allows for comparison across different networks (Spiller et al., 2020).

Power analysis. Statistical power has traditionally been understood as the probability of observing a true effect when rejecting a null hypothesis based on a p-value and reporting statistically significant results (Brydges, 2019). While the presence or absence of an effect is an important question, modern statistics has moved towards the exploration of the level of accuracy of estimated values as estimates of population effects (Halsey, 2019). Since network models are novel approaches for estimating effects, to the researcher’s knowledge there are currently no gold standard tools for power analysis yet (Faelens, Hoorelbeke, Fried, De Raedt, & Koster, 2019). However, it is generally accepted that parameters of a network will be more accurate with increasing sample size (Epskamp et al., 2018). Therefore, to determine the accuracy of estimations, the rule of thumb (three participants per parameter) suggested by Fried and Cramer (2017) was followed to determine the power of the study.

Calculating and interpreting confidence intervals (CI) has also been a useful method to indicate the accuracy of effect size estimates, by providing likely error estimation (Cumming & Calin-Jageman, 2016). As mentioned above, the accuracy of network estimations were enhanced by bootstrapping methods, which calculate confidence intervals around edge-weights and moderation effects, providing information about the sampling variability and accuracy (Epskamp et al., 2018).

Reporting

The current project followed the recommendations of Borsboom et al. (2021) and von Klipstein et al. (2021) in making predominantly non-causative interpretations of the networks. Thus, even though single nodes of the networks were examined, the complex dynamics of the BPD-related psychopathology as a whole are emphasised, where highly connected difficulties, feedback loops and reciprocal associations guided the reporting and discussion of the results (Bringmann et al., 2019; Cramer et al., 2016; Schmittmann et al., 2013).

Ethical Considerations

The larger project which provided the dataset for the current study was reviewed and approved by the Research Ethics Committee (REC) of Wales (reference number: 12/WA/0283, IRAS project ID: 103075). The use of this data for the current secondary data analysis was additionally approved by the Ethics Sub Committee of University of Essex ([UoE]; application numbers ETH1920-1420 and ETH2021-0857; see Appendix B).

The current research was considered low-risk in terms of participant safety. Nonetheless, the ethics approval was obtained on the basis of identifying and minimizing potential risks around ethically sensitive domains, such as patient confidentiality, anonymity and data protection. Risk management plans were incorporated in the study protocol.

The current study analysed data which was previously collected. Participants' anonymity was protected throughout the current study. All data available to the author was non-identifiable as a result of pseudo-anonymisation. The author of the current study did not have access to the secure database linking the participant ID and identifiable personal information to participant consent forms, or any other electronic or paper-based documents that would reveal the identities of the participants. The received dataset was password-protected, only available to the author of the current study. The consent form of the larger project included participants' agreement for pseudo-anonymised data to be used for research

conducted by members of the research team. Data was stored securely and with password protection on the author's personal computer. Statistical analysis was carried out on non-identifiable datasets and any published data maintain complete anonymity. The dataset is only stored by the author until possible future manuscripts are accepted for publication after which it will be destroyed (all study-related data and documents will be archived at UCL and other international sites participating in the data collection, as outlined in their research protocol). All data received by the author is subject to good clinical practice as laid down in the General Data Protection Regulation (GDPR) and local research data management policies of University of Essex, University College London and the Wellcome Trust Centre for Neuroimaging.

Dissemination

Publication. One or more journal articles are planned to be published based on the findings of the current project. The most preferred option within topic-related journals would include the journal called *Personality Disorders: Theory, Research, and Treatment*, as the current research topic greatly aligns with its published materials and the journal obtains a very high impact factor (4.687). Further options include *PloS ONE* and *Journal of Personality Disorders*, which are preferred for being an open-access peer-reviewed journal with high impact factor (impact factor: 2.776 and 3.158 respectively). R-codes used in the study will be published online for increased transparency and reproducibility.

Conferences. Findings might be presented at the yearly conference of *International Congress on Borderline Personality and Allied Disorders*, organised by the European Society for the Study of Personality Disorders, where fellow academics could be informed of the results. Clinicians might also be reached via conferences offering Continuing Professional Development (CPD) accreditation, such as the *Improving Access, Treatment and Support for People with a Diagnosis of Personality Disorder* conference which took place in 2020. Since attachment and mentalization are psychodynamic terms, the Department of Psychosocial and Psychoanalytic Studies of UoE might also be interested in offering a platform for the dissemination of the results, whereby students from various disciplines could have access to the research findings. Moreover, since the yearly conferences on mentalization and attachment organised by the Psychoanalysis Unit of UCL typically attract clinicians, therapists and academics, delivering a poster or oral presentation there could also be an effective way of reaching people from different segments of psychology.

Services. To further ensure that the clinical implications of the study reach the clinicians of interest, services throughout Essex providing complex needs and personality disorders pathways would be contacted and provided with the abstract/journal article (e.g. C &E Centre in Chelmsford, Basildon Mental Health Unit, Psychotherapy Department Colchester etc.). Additionally, with the purpose of influencing general policies, the Policy Team of the British Psychological Society may be contacted via the external consultants who facilitate collaboration with members of the society on various topics. By sending them the results of this study, the Policy team could synthesize results with other research outcomes of studies on BPD, condensing its most important clinical implications and feed them back to the government as scientific evidence in the form policy reports and position papers.

Chapter Three: Results

Missing Data Analyses.

Exclusion of fully missing data. People who had fully missing data on the PAI-BOR items (3.6%) and on the MASC scales (8.5%) were excluded prior to analyses, leaving 575 people in the final sample. To detect whether significant differences were found in the demographic characteristics of the people who were excluded due to missing data and those who were not, chi-square tests of association and Mann-Whitney U-tests were run (Field, 2013). The general assumption of chi-square tests regarding expected counts was tested according to recommendations from Yates, Moore, and McCabe (1999), who suggested that all expected counts ought to be greater than one and no more than 20% of expected counts should be less than five.

Gender. Less than five counts of participants from the retained sample identified as transgender ($n=3$) and other ($n=2$). Therefore, they were excluded from this sensitivity analysis (but they were included in the final analyses). The retained sample was composed of a significantly greater proportion of men (27.4%) than the excluded sample (11.3%), $\chi^2(1, N=641) = 8.597, p = .003$.

Ethnicity. The retained sample had similar ethnicities as the excluded sample, $\chi^2(4, N=646) = 4.941, p = .293$.

Age. Age was not normally distributed in the retained or excluded sample, as assessed by visual inspection of the histograms. Therefore, Mann-Whitney U-test was used to determine that the retained sample was significantly older ($Mdn=28$) than the excluded sample ($Mdn=25.5$), $U=15770.5, p = .003$.

Education. The retained sample did not differ from the excluded sample in their level of education, $\chi^2(6, N=643) = 7.328, p = .292$.

Employment. Categories of employment were collapsed in order to meet the assumptions of the chi-square test of association (for collapsed categories see Table 3). The retained sample did not differ from the excluded sample, $\chi^2(3, N=641) = 2.867, p = .413$.

Socioeconomic status. Socioeconomic status was indicated by social deprivation rank according to post code. Social deprivation rank was not normally distributed for either sample, as assessed by visual inspection of the histograms. Therefore, Mann-Whitney U-test was used to show that the two samples were similar, $U=18167, p = .754$.

Household income. Categories of household income were collapsed in order to meet the assumptions of the chi-square test of association (for collapsed categories see Table 3).

The retained sample did not differ from the excluded sample in their employment status, $\chi^2(2, N=620) = 0.994, p = .608$.

Descriptive Statistics

Table 3 summarizes the demographic characteristics of the community group and the clinical group. The observed variables were not normally distributed, as assessed by visual inspection of Normal Q-Q Plots and histograms. Therefore, Mann-Whitney U-tests were run to detect differences across the two groups for continuous variables and Chi-square test of independence tests were employed for categorical ones.

The clinical group included significantly more women and significantly older individuals than the community group. The two groups also differed in ethnicity, with significantly more White participants being included in the clinical group than in the other one. Significantly more people were unemployed in the clinical group as well. Regarding education, while more people in the clinical group did not have any formal education, significantly more of them also completed higher education when compared with the participants in the community group. While no difference was detected between the two groups in terms of socioeconomic status (based on social deprivation rank), people in the community group were found to earn significantly more than those living with a diagnosis.

Table 3.
Sociodemographic Characteristics of Participants Within the Two Groups

Demographic variable	Clinical group (n=350) <i>n (%) or Median</i>	Community group (n=225) <i>n (%) or Median</i>	Value of relevant comparative statistic ^d	p-value
Gender			$X^2(1)= 24.427^a$	< .001
Male	69 (20%)	87 (39%)		
Female	277 (79%)	137 (61%)		
Transgender	2 (0.5%)	1 (0%)		
Other	2 (0.5%)	-		
Age	29 (40)	26 (44)	U=350.50	.030
Ethnicity ^b			$X^2(3)= 12.192$.007
White	261 (75%)	138 (61.5%)		
Black/Black British	27 (8%)	26 (11.5%)		
Asian/British Asian	21 (6%)	25 (11%)		
Mixed/Other	39 (11%)	35 (16%)		
Employment			$X^2(3)=82.76$	< .001
Employed	100 (29%)	123 (55%)		
Unemployed	194 (56%)	40 (18%)		
Student/Apprentice	47 (14%)	57 (25%)		
Retired/Carer	5 (1%)	5 (2%)		
Education			$X^2(6)=14.651$.023
No formal education	24 (7%)	7 (3%)		
Other qualification (e.g. certificate)	10 (3%)	6 (3%)		
Vocational level 1 (e.g. NVQ), GCSE (<5 A*-C), or equivalent	29 (8%)	16 (7%)		
GCSE (5 or more A*-C), level 2 (e.g. NVQ), or equivalent	65 (19%)	46 (20%)		
A level, vocational level 3 (e.g. NVQ), or equivalent	99 (28%)	90 (40%)		
Higher education or professional/vocational equivalent	97 (28%)	42 (19%)		
Post graduate education or professional/vocational equivalent (e.g. Masters, PhD)	24 (7%)	18 (8%)		
Household Income			$X^2(2)=35.288$	< .001
<£10k	161 (48%)	52 (24%)		
£10k-35k	128 (38%)	106 (48.5%)		
>£35k	48 (14%)	60 (27.5%)		
SES ^c	10562 (31252)	10802 (31166)	U=35795	.635

Note. N = 575. BPD=Borderline personality disorder.

^aPeople who identify as transgender or other had to be excluded from comparative analysis to meet assumptions of the relevant test; ^bWhite=White British, White Irish, Any other white; Black/Black British=Caribbean, African, Any other black; Asian/British Asian=Indian, Pakistani, Bangladeshi, Chinese, Any other Asian; Mixed/Other=White and Black Caribbean, White and Black African, White and Asian, Any other mixed, Any other background not stated; ^cSES=socioeconomic status indicated by the social deprivation rank according to post code; ^d X² for chi-square test of independence, U for Mann-Whitney U test (data was not normally distributed).

Preliminary Analyses

Scores of the PAI-BOR individual items and the MASC subscales were compared between the two groups. Since the items of the PAI-BOR and the MASC subscales were not normally distributed in either group (as evaluated by visual inspection of Normal Q-Q Plots and histograms), a series of Mann-Whitney U-tests were run to detect group differences (see Table 3). Upon visual inspection, the distributions of PAI-BOR scores were not similar between groups, so mean ranks were interpreted, whereas the distributions of the MASC subscale scores were similar, so medians were interpreted. Significant differences between the two groups were found on all PAI-BOR items and on two of the MASC subscales, namely 'adequate mentalisation' and 'hypermentalisation'. The 'lack of mentalisation' subscale approached the threshold of significant difference as well.

Table 4.
Item Abbreviations, Item Content, Medians and Mean Ranks for PAI-BOR items and MASC subscales

PAI-BOR subscale	Item	Item content	Full sample	Clinical sample	Community sample	<i>U</i> value ^b	<i>p</i> value ^c
			(<i>N</i> =575)	(<i>n</i> =350)	(<i>n</i> =225)		
			<i>Mdn</i>	<i>Mdn or Mean rank</i> ^a	<i>Mdn or Mean rank</i> ^a		
AI	BPD1	Sudden mood shifts	3	372.52	156.53	9793.5	< .001.
ID	BPD2	Attitude about self changes	2	358.29	178.67	14775	< .001.
NR	BPD3	Stormy relationships	2	366.7	165.58	11830	< .001.
AI	BPD4	Mood gets intense	3	376.3	150.65	8470.5	< .001.
ID	BPD5	Chronic emptiness	3	379.76	145.27	7260.5	< .001.
NR	BPD6	Let people know they've hurt me	2	351.67	188.95	17089.5	< .001.
AI	BPD7	Steady mood (RS)	3	362.60	171.01	13051.5	< .001.
ID	BPD8	Worry about people leaving	2	363.09	169.39	12743	< .001.
NR	BPD9	People let me down	3	349.59	188.49	17021	< .001.
AI	BPD10	Little control over anger	1	362.19	172.59	13407	< .001.
ID	BPD11	Wonder about life	3	347.09	196.08	18692	< .001.
NR	BPD12	Rarely lonely (RS)	3	342.8	200.06	19614	< .001.
SH	BPD13	Do things impulsively	1	355.5	183	15749.5	< .001.
AI	BPD14	Happy person (RS)	3	361.53	173.61	13638	< .001.
ID	BPD15	Can't handle separation	2	350.46	190.83	17512.5	< .001.
NR	BPD16	Mistakes in picking friends	1	347.99	194.68	18378.5	< .001.
SH	BPD17	Hurt self when upset	1	383.87	138.86	5819	< .001.
AI	BPD18	Can't express all of anger	2	364.47	168.11	12399.5	< .001.
ID	BPD19	Don't get bored easily (RS)	3	332.80	217.23	23451.5	< .001.
NR	BPD20	Stay friends with people	2	321.15	233.06	27013.5	< .001.
SH	BPD21	Too impulsive	1	358.52	176.38	14260.5	< .001.
SH	BPD22	Spend money easily	2	341.65	203.5	20362.5	< .001.
SH	BPD23	Reckless person	1	346.31	195.27	18511	< .001.
SH	BPD24	Careful about money (RS)	2	327.04	226.17	25465	< .001.
	MASCcorr	Correct mentalisation	34	34	35	44978.5	.004
	MASCexc	Hypermentalisation	5	5	4	33876	.004
	MASCless	Hypomentalisation	4	4	4	37795	.413
	MASCno	No mentalisation	2	2	1	35730	.055

Note. BPD=Borderline personality disorder. PAI-BOR = Personality Assessment Inventory Borderline subscale. MASC = Movie for the Assessment of Social Cognition. RS= reverse-scored. AI=Affective instability. ID=Identity diffusion. NR= Negative relationships. SH= Self-harm.
^aMean ranks were reported for the PAI-BOR items, median was reported for MASC subscales.
^bU value represents the result of the Mann-Whitney U test.
^cBolded values indicate significance ($p < .05$)

Unique Variable Analysis

UVA was used to identify redundant items of the questionnaires by measuring the level of topological overlap between them and producing minor factors to improve the accuracy of further estimated models (Christensen et al., 2020; Zhang & Horvath, 2005). The significance test used as part of the UVA identified so-called target variables and a “redundancy chain” plot associated with them, based on the threshold defined by the chosen wTO option (p value of .25). Potentially redundant variables were listed and depicted as fully connected sets of nodes offered for combination (Christensen et al., 2020). Table 5 summarizes the target variables and potential redundancies as offered by the analysis, alongside the outcome of the decisions about the combination of identified items into a new latent variable.

Table 5.
Result of UVA

Target variable ^a	Items identified as potentially redundant	Decision	New label
Item 23: <i>I'm a reckless person.</i>	Item 13: <i>I sometimes do things so impulsively that I get into trouble.</i> Item 21: <i>I'm too impulsive for my own good.</i> Item 22: <i>I spend money too easily.</i> Item 24: <i>I'm careful about how I spend my money.</i>	Combine	<i>Impulsivity and recklessness</i>
Item 1: <i>My mood can shift quite suddenly.</i>	Item 4: <i>My moods get quite intense.</i>	Combine	<i>Intense mood shifts</i>
Item 5: <i>Sometimes I feel terribly empty inside.</i>	Item 11: <i>I often wonder what I should do with my life.</i>	Not combine	
Item 6: <i>I want to let certain people know how much they've hurt me.</i>	Item 9: <i>People once close to me have let me down.</i>	Not combine	
Item 7: <i>My mood is very steady.</i>	Item 14: <i>I've always been a pretty happy person.</i>	Not combine	
Item 8: <i>I worry a lot about other people leaving me.</i>	Item 15: <i>I can't handle separation from those close to me very well.</i>	Not combine	
Item 10: <i>I have little control over my anger.</i>	Item 18: <i>I've had times when I was so mad I couldn't do enough to express all my anger.</i>	Not combine	
MASCless	MASCno	Not combine	

Note.

^aTarget variables are items from the PAI-BOR = Personality Assessment Inventory Borderline subscale and the hypermentalisation scale of the Movie for the Assessment of Social Cognition

The UVA offered a total of eight target variables that showed high topological overlap with other items; seven belonged to the PAI-BOR and one to the MASC. Clinical judgement by the researcher determined which items were made redundant, balancing the need for improving model fit with the attempt to preserve the specificity of all BPD-related traits as much as possible. As Table 5 indicates, items were made redundant in the case of only two target variables (Item 23 and Item 1). Five of the PAI-BOR target variables were judged to capture significantly different difficulties than the offered items, and thus were kept separate. The MASCless subscale was also deemed to be kept separate from the offered item of MASCno, in order to avoid compromising the research question.

Two new minor latent variables were created after combining target and redundant items: “*Impulsivity and recklessness*” and “*Intense mood shifts*”. This left a total of 19 BPD-

related features to include in the network analyses. Table 5 summarises the final variables across PAI-BOR and MASC that were included as nodes in the network analyses.

Group Differences in Global Strength and Structure

The Network Comparison Test was conducted to detect differences in the overall network connectivity. The network of the community group (global strength= 2.11) demonstrated significantly lower global strength than the network of the clinical group (global strength=5.21), $S = 3.1$, $p < .001$. This suggested greater connectivity (higher level of activation) in the clinical group than in the community one. The two groups also significantly differed on the maximum difference in any of the associations, $M = 0.36$, $p < .0001$, suggesting that the structure of the variables also differed between the groups.

Moderated Network Analysis

The moderated networks included 23 nodes in total (see Table 6): 19 BPD-related features, three types of ineffective modes of mentalisation (hypermentalisation, hypomentalisation and no mentalisation) and the presence or absence of BPD diagnosis. The estimated network of the whole sample included 575 individuals. To create a condition relating to BPD diagnosis and compare the two groups (N = 225 individuals in the community group, N = 350 individuals in the clinical group), the *condition* () function of the *mgm* package was used (Haslbeck et al., 2018).

Table 6.
Nodes included in final MGM analysis

Node number	Item description
1	Intense mood shifts
2	Attitude about self changes a lot
3	Stormy relationships
4	Chronic emptiness
5	Want to let people know they hurt me
6	Unsteady mood
7	Worry about people leaving
8	Feeling that people let one down
9	Little control over anger
10	Wonder about what to do with life
11	Feeling lonely
12	Feeling unhappy
13	Cannot handle separation
14	Making mistakes in picking friends
15	Hurt self when upset
16	Cannot express all of anger
17	Getting bored easily
18	Difficulty to stay friends for long time
19	Impulsivity and recklessness
20	Hypermentalised
21	Hypomentalised
22	Lack of mentalisation
23	BPD diagnosis

Note.

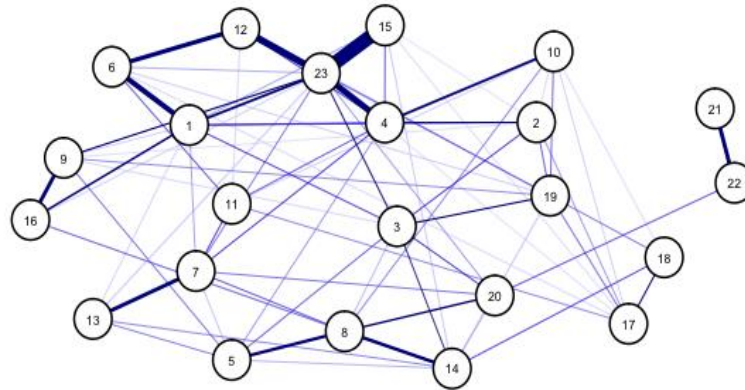
The estimated moderated network models are visualised in Figure 3, with 3/A depicting the network of the whole sample and 3/B and C depicting the network when conditioned on BPD diagnosis. Edges were calculated via multiple regression analysis; however, instead of predicting single dependent variables only, the networks reveal how variables can predict all other dependent and independent variables in the network in the presence of a moderator (Epskamp & Fried, 2018). Therefore, each edge represents the unique undirected pairwise association, or in other words, the unique conditional dependence relationship between the BPD- and mentalisation-related difficulties, while controlling for all other variable in the networks. The visualisation of the edges are adjusted for the strongest weight of the graph, so while the difference in the thickness between edges represent a

difference in their weight (the thicker the edge the larger the edge weight), it is not recommended to interpret the mean edge weights through visualisation alone (Burger et al., 2020; Epskamp et al., 2012). The edge weights are not partial correlation coefficients but have the same interpretations. Values that directly relate to the research question are reported below, while average edge weights of the networks are included in Appendix C, D and E.

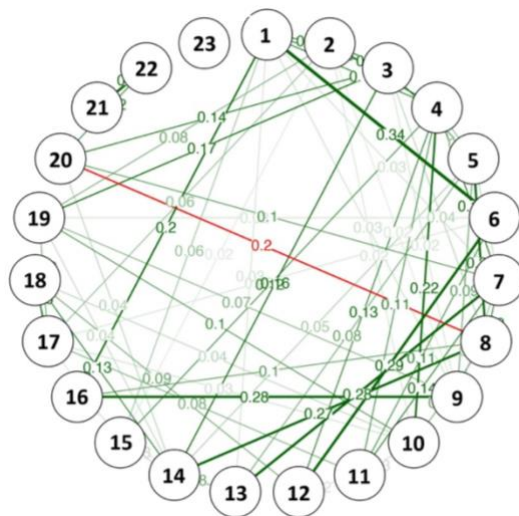
Figure 3.

Moderated network models for the whole sample and conditioned on the absence and presence of BPD diagnosis

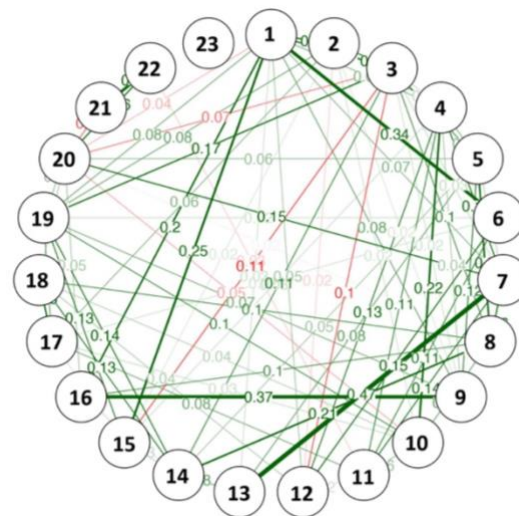
A. Whole Sample ($N=575$)



B. Community Sample ($n=225$)



C. Clinical Sample ($n=350$)



Note. BPD=Borderline Personality Disorder.

Legend. node 1= intense mood shifts, node 2= attitude about self changes, node 3=stormy relationships, node 4=chronic emptiness, node 5=let people know they hurt me, node 6= unsteady mood, node 7=worry about people leaving, node 8= 'people let me down', node 9= little control over anger, node 10= wonder about life, node 11= feeling lonely, node 12= feeling unhappy, node 13= cannot handle separation, node 14= mistakes in picking friends, node 15= hurt self when upset, node 16= cannot express all of anger, node 17= gets bored easily, node 18= difficulty with staying friends with people , node 19= impulsivity and recklessness, node 20= MASCExc (hypermentalisation), node 21= MASCLess (hypomentalisation), node 22= MASCno (no mentalisation), node 23= BPD diagnosis.

Values along the edges represent average absolute value of edge weights (pairwise partial correlation values).

In the depicted networks, green and red edges respectively indicate a positive or negative linear relationship between the variables in the presence of the moderator. Values along the edges represent the average absolute values of edge weights. Stronger relationships are visualised as thicker edges. Absent edges do not indicate lack of marginal connections between variables, but that the relationship disappears when controlled for all other variables in the network (in other words, they were spurious).

Group Differences Moderated by BPD diagnosis

To explore differences in the pairwise associations, moderation network analysis was used to examine the whole sample, with the BPD diagnosis being included as a 23rd moderator variable. This allowed for the detection of specific edges between BPD- and mentalisation-related nodes that are moderated by the presence or absence of the diagnosis (or in other words, the severity of the problem).

For all participants, the moderation network analysis revealed several pairwise associations, which have been moderated by the presence of BPD to a varying degree. To highlight the most robust results and follow a concise reporting style, it was decided by the researcher that associations are reported if they reached the edge weight of ± 0.05 (which is less than the threshold defined by Doucouliagos (2011) for small associations) or if the association was revealed in at least 50% of the bootstrapped samples. Tables including all bootstrapped edge weights, moderation effects and the corresponding confidence intervals can be found in Appendix C, D, E. Figure 3/A visualises a network that is representative of the whole sample.

To examine the stability and accuracy of the moderated network, it was resampled 1000 times. This bootstrapping approach also provided more detailed information about the estimated parameters and their reliability. Estimated means of the edge weights are reported with low (5%) and high (95%) quantiles of the bootstrapped sample distribution, which is translatable to 95% confidence intervals. Narrower confidence intervals indicate less variance in the pairwise interaction effects and thus, higher stability of the estimated parameters. Bootstrapping revealed that mean edge weights in the moderated network ranged from 0.86 (between ‘when upset hurt self’ and BPD diagnosis) to -0.21 (between ‘people let me down’ and hypermentalisation).

Hypermentalisation. A moderate positive pairwise association was found between hypermentalisation and ‘having stormy relationships’ (weight= 0.14; bootstrapped 95% CI [0-0.25]), which was negatively moderated by the presence of a BPD diagnosis (-0.15;

bootstrapped 95% CI [-0.15 to -0.26]). This indicates that the probability of hypermentalisation increasing stormy relationships and vice versa is less likely in the clinical sample (see node 3-20 in Appendix C). The association was confirmed in 90% of the 1000 bootstrap samples, and the moderation effect in 89%.

Furthermore, a moderate positive pairwise, cross-sectional association was found between hypermentalisation and ‘worrying about people leaving’ (weight=0.08; bootstrapped 95% CI [0- 0.19]), which was positively moderated by the presence of a BPD diagnosis (0.01; bootstrapped 95% CI [0 - 0.12]). This suggests that the probability of hypermentalisation increasing the worry about people leaving and vice versa is slightly more likely in the clinical sample (see node 7-20 in Appendix C). This association was confirmed in 67% of the 1000 bootstrap samples, however the moderation effect was only found in 8% of the samples.

A small positive pairwise, cross-sectional association was found between hypermentalisation and ‘mistakes in making friends’ (weight=0.05; bootstrapped 95% CI [0 – 0.17]), which was negatively moderated by the presence of a BPD diagnosis (-0.03; bootstrapped 95% CI [-0.03 to -0.14]). This indicates that the probability of hypermentalisation increasing the feeling of making mistakes when picking friends and vice versa is less likely in the clinical sample (see node 14-20 in Appendix C). This association was confirmed in 48% of the 1000 bootstrap samples, while the moderation effect was only found in 30% of the samples.

Finally, a moderate negative pairwise association was found between hypermentalisation and ‘feeling that people let one down’ (weight= - 0.21; bootstrapped 95% CI [- 0.32 to – 0.07]). This was positively moderated by the presence of a BPD diagnosis (0.16; bootstrapped 95% CI [0-0.3]), indicating that the probability of hypermentalisation decreasing the feeling that people let one down and vice versa is less likely in the clinical group (see node 8-20 in Appendix C). The association was confirmed in 99% of the 1000 bootstrap samples, and the moderation was confirmed in 92% of the samples.

Hypomentalisation. No associations were found in the representative network of the whole sample with regards to hypomentalisation or no mentalisation (Figure 3/A). However, bootstrapping across 1000 samples revealed a moderate positive pairwise association between hypomentalisation and ‘struggling to handle separation’ (weight= 0.07; bootstrapped 95% CI [0 – 0.18]), which was moderated negatively by the presence of a BPD diagnosis (-0.01; bootstrapped 95% CI [-0.01 to -0.09]). This indicates that the probability that hypomentalisation increases the struggles with handling separation and vice versa is slightly

less likely for people with BPD (see node 13-21 in Appendix C). The association was confirmed in 69% of the 1000 bootstrap samples, and the moderation effect in 10% of the samples. Bootstrapping also revealed a small positive pairwise association between hypomentalisation and ‘wanting to let people know how much they hurt one’ (weight= 0.06; bootstrapped 95% CI [0 – 0.17]), which was moderated negatively by the presence of a BPD diagnosis (-0.03; bootstrapped 95% CI [-0.03 to -0.13]). This indicates that the probability of hypomentalisation increasing the desire to let people know how much they hurt one and vice versa is less likely for people with BPD (see node 5-21 in Appendix C). This association was confirmed in 58% of the 1000 bootstrap samples, and the moderation effect in 30%.

MASC subscales. A large positive pairwise association was found between hypomentalisation and no mentalisation (weight= 0.3; bootstrapped 95% CI [0.2-0.39]), which was positively moderated by the presence of a BPD diagnosis (0.02; bootstrapped 95% CI [0 - 0.12]). This indicates that the probability of hypomentalisation increasing no mentalisation and vice versa is more likely in the clinical sample (see node 21-22 in Appendix C). The association was confirmed in 100% of the 1000 bootstrap samples, and the moderation effect in 25%. A moderate positive pairwise association was found between hypermentalisation and no mentalisation (weight= 0.11; bootstrapped 95% CI [0-0.21]), which was positively moderated by the presence of a BPD diagnosis (0.02; bootstrapped 95% CI [0 - 0.12]). This indicates that the probability of hypermentalisation increasing no mentalisation and vice versa is more likely in the clinical sample (see node 20-22 in Appendix C). The association was confirmed in 90% of the 1000 bootstrap samples, and the moderation effect in 24%.

Subgroup analysis

The moderation network analysis of the whole sample confirmed pairwise associations between the relevant variables that are moderated by the presence or absence of BPD diagnosis (see bolded nodes in Table 7). To examine how the moderator variable impacts these pairwise associations (or in other words, to highlight the ways in which the relationships are different in the two groups), networks within each group were examined separately. These networks were divided by the grouping variable (BPD diagnosis) and thus contained 22 variables in total (19 BPD-related nodes and three nodes for ineffective modes of mentalisation). To examine the stability and accuracy of the networks, they were resampled 1000 times. Estimated means of the edge weights are reported with low (5%) and

high (95%) quantiles of the bootstrapped sample distribution, which is translatable to 95% confidence intervals.

Community group. Figure 3/B visualises a network that is representative of the community group. Bootstrapping revealed that mean edge weights in the moderated network ranged from 0.37 ('impulsivity and recklessness' and 'feeling that people let one down') to -0.22 ('feeling that people let one down' and hypermentalisation; see Appendix D).

Hypermentalisation. A moderate positive pairwise association was found between hypermentalisation and 'stormy relationships' (weight=0.17; bootstrapped 95% CI [0.05 – 0.27], confirmed in 99% of 1000 bootstrapped samples), 'worrying about people leaving' (weight=0.09; bootstrapped 95% CI [0 – 0.21], confirmed in 73% of 1000 bootstrapped samples) and 'mistakes of making friends' (weight=0.08; bootstrapped 95% CI [0 – 0.19], confirmed in 71% of the bootstrapped samples). Furthermore, a moderate negative pairwise association was found between hypermentalisation and 'feeling that people let one down' (weight= -0.22; bootstrapped 95% CI [-0.22 to - 0.32]), which was confirmed in 100% of the bootstrapped samples.

Hypomentalisation. Several associations were found across 1000 bootstrapped samples (see Appendix D), which were not depicted in Figure 3/B, as the visualisation depicts only a representation of these. Across 1000 bootstrapped samples, several relationships with hypomentalisation were found (see Appendix D). A moderate positive association was revealed between hypomentalisation and 'struggling with handling separation' well (weight=0.09; bootstrapped 95% CI [0 – 0.2], confirmed in 82% of the bootstrapped samples) and 'wanting to let people know how much they hurt one' (weight=0.08; bootstrapped 95% CI [0 – 0.19], confirmed in 75% of the bootstrapped samples).

No mentalisation. Similarly to hypomentalisation, several associations were found across 1000 bootstrapped samples (see Appendix D), which were not depicted in Figure 3/B. A small positive association was found between no mentalisation and 'mistakes in picking friends' (weight=0.05; bootstrapped 95% CI [0 – 0.16], confirmed in 52% of the bootstrapped samples). Finally, in 61% of samples, a small negative association was found between no mentalisation and 'struggles with handling separation' (weight= -0.05; bootstrapped 95% CI [-0.16 – 0]).

MASC subscales. A moderate positive partial correlation was shown between the hypomentalisation and no mentalisation scales of the MASC (weight= 0.27; bootstrapped 95% CI [0.16 – 0.38], confirmed in 100% of the bootstrapped samples). A moderate positive

partial correlation was also shown between the hypermentalisation and no mentalisation scales of the MASC (weight= 0.08; bootstrapped 95% CI [0 – 0.19], confirmed in 77% of the bootstrapped samples).

Clinical group. Figure 3/C visualises a network that is representative of the clinical group. Bootstrapping revealed that mean edge weights in the moderated network ranged from 0.47 (worrying about people leaving and inability to handle separation) to -0.14 (hypomentalisation and hypermentalisation; see Appendix E).

Hypermentalisation. A moderate positive pairwise association was found between hypermentalisation and ‘worrying about people leaving’ (weight=0.13; bootstrapped 95% CI [0.03 – 0.22], confirmed in 97% of 1000 bootstrapped samples), ‘wanting to let people know that they hurt one’ (weight=0.09; bootstrapped 95% CI [0 – 0.18], confirmed in 87% of the bootstrapped samples). Hypermentalisation was negatively and moderately correlated with ‘stormy relationships’ (weight= -0.06; bootstrapped 95% CI [-0.15 – 0], confirmed in 65% of the bootstrapped samples).

Hypomentalisation. A small negative pairwise was shown between hypomentalisation and ‘stormy relationships’ (weight=-0.06; bootstrapped 95% CI [-0.15 – 0], confirmed in 69% of the bootstrapped samples) and ‘worrying about people leaving’ (weight=-0.05; bootstrapped 95% CI [-0.14 – 0], confirmed in 59% of the bootstrapped samples).

MASC subscales. A large positive pairwise association was found between the hypomentalisation and no mentalisation subscales (weight=0.35; bootstrapped 95% CI [0.26 – 0.43], confirmed in 100% of the bootstrapped samples). A moderate positive association was also shown between hypermentalisation and no mentalisation (weight=0.16; bootstrapped 95% CI [0.7 – 0.25], confirmed in 99% of the bootstrapped samples).

Further connections. The subgroup analysis also identified several pairwise associations within the two groups that were shown to be absent or very weak in the whole sample analysis. While the subgroup analyses had significantly less power than the whole sample moderation network analysis, the additional connections discovered through subgroup analysis are reported below, as they suit the exploratory nature of the study. It is however important to note that they do not indicate statistical differences between the two groups and merely indicate tendencies at best (see non-bolded nodes in Table 7)

In the community group, a small positive pairwise association was found between hypermentalisation and ‘hurting the self when feeling upset’ (weight=0.05; bootstrapped 95% CI [0 – 0.15]), which was confirmed in 65% of the bootstrapped samples. In 68% of the

samples, a small negative association was also found between hypomentalisation and ‘attitude about self-changes’ (weight= - 0.06; bootstrapped 95% CI [-0.16 – 0]). Furthermore, a small positive association was found between no mentalisation and ‘feeling empty’ (weight=0.06; bootstrapped 95% CI [0 – 0.17], confirmed in 63% of the bootstrapped samples).

In the clinical group, a moderate positive pairwise association was shown between hypermentalisation and ‘struggles with expressing anger’ (weight=0.8; bootstrapped 95% CI [0 – 0.17], confirmed in 81% of the bootstrapped samples) and ‘impulsivity and recklessness’ (weight=0.8; bootstrapped 95% CI [0 – 0.16], confirmed in 83% of the bootstrapped samples). Hypermentalisation was negatively and moderately correlated with ‘wondering about life’ as well (weight= - 0.08; bootstrapped 95% CI [- 0.16 – 0], which was confirmed in 86% of bootstrapped samples). Furthermore, a small positive pairwise association was found between hypomentalisation and ‘intense mood shifts’ (weight=0.6; bootstrapped 95% CI [0 – 0.16], confirmed in 68% of the bootstrapped samples), ‘little control over anger’ (weight=0.6; bootstrapped 95% CI [0 – 0.15], confirmed in 68% of the bootstrapped samples) and ‘feeling empty’ (weight=0.5; bootstrapped 95% CI [0 – 0.14], confirmed in 60% of the bootstrapped samples). A moderate negative pairwise was found however between hypomentalisation and ‘unsteady mood’ (weight=-0.08; bootstrapped 95% CI [-0.16 – 0], confirmed in 86% of the bootstrapped samples), while a small negative pairwise was shown between hypomentalisation and ‘unhappiness’ (weight=-0.06; bootstrapped 95% CI [-0.15 – 0], confirmed in 72% of the bootstrapped samples). In terms of no mentalisation, a small positive pairwise association was found between no mentalisation and ‘wondering about life’ (weight=0.5; bootstrapped 95% CI [0 – 0.13], confirmed in 70% of the bootstrapped samples), while a small negative partial correlation was shown between no mentalisation and ‘feeling lonely’ (weight= -0.06; bootstrapped 95% CI [-0.15 – 0], confirmed in 71% of the bootstrapped samples). Finally, a moderate negative association was found in this group between hypermentalisation and hypomentalisation (weight= -0.14; bootstrapped 95% CI [-0.23 to -0.03], confirmed in 97% of the bootstrapped sample).

Table 7.
Pairwise effects found in both groups

Node in the network ^a	Community group			Clinical group		
	MASCexc	MASCless	MASCno	MASCexc	MASCless	MASCno
N1: Intense mood shifts					+	
N2: Attitude about self changes		-				
N3: Stormy relationships	+			-	-	
N4: Feeling empty			+		+	
N5: Let people know they've hurt me		+		+		
N6: Unsteady mood			+		-	
N7: Worry about people leaving	+			+	-	
N8: People let me down	-					
N9: Little control over anger					+	
N10: Wonder about life				-		+
N11: Feeling lonely						-
N12: Feeling unhappy					-	
N13: Can't handle separation		+	-			
N14: Mistakes in picking friends	+		+			
N15: When upset hurt self	+					
N: 16 Can't express all of anger				+		
N18: Difficulty staying friends with people		+		+		
N19: Impulsivity and recklessness						
MASCexc			+			+
MASCless			+			+
MASCno	+	+		+	+	

Note. BPD= Borderline personality disorder. † = large positive partial correlation between nodes, ‡ = moderate positive partial correlation between nodes, + = small positive partial correlation between nodes, - = moderate negative partial correlation between nodes. - = small negative partial correlation between nodes. RS= Reverse scale.

^a Bolded nodes correspond to relationships with ineffective modes of mentalisation that were already demonstrated in the moderated network analysis. Non-bolded nodes represent the relationships with ineffective modes of mentalisation found solely as the result of exploratory subgroup analysis.

Group Differences in the Strength of Associations

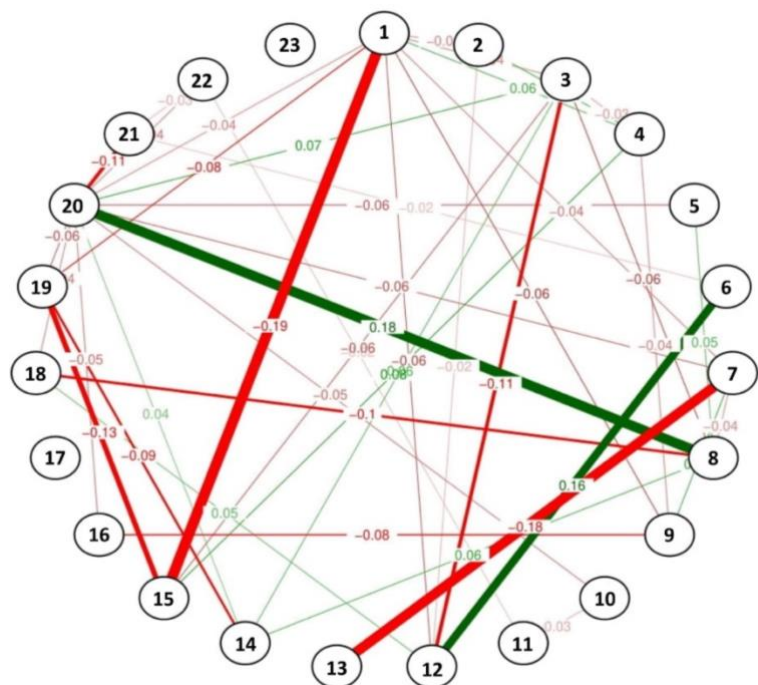
The estimated edges of the network belonging to the clinical group were subtracted from the edges of the network belonging to the community group. These difference scores indicated where and by how much the groups' pairwise associations between BPD- and mentalisation-related difficulties differed. Figure 4 visualises these difference scores on a graph, where the green edges represent a correlation that is stronger in the community group, while the red edges represent a correlation that is stronger in the clinical group

Overall, the pairwise associations between ineffective modes of mentalisation and BPD-related difficulties were stronger in the clinical group (difference scores displayed in Figure 4 and Appendix F). This included edges between hypermentalisation and the following BPD-related difficulties: 'intense mood shifts', 'wanting to let people know that they hurt one', 'worrying about people leaving', 'wondering about life', 'inability to express anger', 'difficulty in staying friends with people', and 'impulsivity/recklessness'. Hypermentalisation was also more correlated with other mentalisation difficulties such as hypermentalisation and no mentalisation in the clinical group. However, in the community group, the pairwise associations between hypermentalisation and 'stormy relationships', 'feeling that people let one down', and 'feeling that one makes mistakes in picking their friends' were stronger.

Pairwise associations related to hypomentalisation and no mentalisation were all found to be stronger in the clinical group. Thus, reduced mentalisation correlated stronger with 'unsteady mood', hypermentalisation and no mentalisation in the clinical group than in the community one. Similarly, no mentalisation correlated stronger with 'feeling lonely', hypermentalisation, and hypomentalisation in the clinical group than in the community one.

Figure 4.

Graph representing the difference scores of the pairwise associations between the groups of people with and without a BPD diagnosis



Note. BPD=Borderline Personality Disorder.

Legend. node 1= intense mood shifts, node 2= attitude about self changes, node 3=stormy relationships, node 4=chronic emptiness, node 5=let people know they hurt me, node 6= unsteady mood, node 7=worry about people leaving, node 8= ‘people let me down’, node 9= little control over anger, node 10= wonder about life, node 11= feeling lonely, node 12= feeling unhappy, node 13= cannot handle separation, node 14= mistakes in picking friends, node 15= hurt self when upset, node 16= cannot express all of anger, node 17= gets bored easily, node 18= difficulty with staying friends with people , node 19= impulsivity and recklessness, node 20= MASCexc (hypermentalisation), node 21= MASClless (hypomentalisation), node 22= MASCno (no mentalisation).

Green edges represent stronger pairwise connections between variables in the community group than in the clinical group while red edges depict a relationship of the opposite nature.

Expected Influence

The following network models included 22 variables: 19 BPD-related features and the three types of ineffective modes of mentalisation. Since the two groups were split according to the presence of BPD diagnosis, the grouping variable was not included in the centrality analyses. Nonetheless, results are reported for the whole sample, as well as for the two groups.

Table 8 summarizes the estimated one-step and two-step EI values of the nodes for the whole sample and across both groups, while Figure 5 shows the standardised (z-scored)

EI values in a decreasing order with regards to their centrality (influence) in the network. Between all modes of ineffective mentalisation, hypermentalisation yielded the highest one-step and two-step EI values across the whole sample and both groups. Still, relative to all other variables, hypermentalisation was amongst the least influential nodes in the whole sample (one-step EI=0.71, two-step EI= 1.3). Within the groups, one-step and two-step EI values for hypermentalisation ranked in the middle of the range of EI values associated with BPD-related traits (community: one-step EI= 0.59, two-step EI= 1.09; clinical: one-step EI= 0.58, two-step EI= 0.99; see Figure 5/B and C). The relative influence of hypermentalisation decreased in the community relative to the clinical sample.

EI values of hypomentalisation and no mentalisation were ranked lowest in the range of EI values across all nodes in the whole sample (hypomentalisation: one-step EI= 0.59, two-step EI= 0.99, no mentalisation: one-step EI=0.51, two step EI=0.85). However, the standardised one-step and two-step EI values of hypomentalisation increased when examined in each group separately. The clinical group (see Figure 5/C; raw scores: one-step EI=0.57, two-step EI=0.91) scored higher than the community group (see Figure 5/B; raw scores: one-step EI=0.51, two-step EI=0.81). A similar trend was observed for the EI values of the node representing no mentalisation as well (higher EI values for the clinical sample). However, the centrality of lack of mentalising remained low across both groups (see Figure 5/B and C; community group: one-step EI=0.45, two-step EI=0.75; clinical group: one-step EI=0.5, two-step EI=0.79) as compared to other nodes of the network.

One-step EI values of BPD-related difficulties ranged from 0.59 ('getting bored easily') to 1.05 ('cannot express all the anger') in the whole sample. Similarly, the node corresponding to 'getting bored easily' was confirmed to have the lowest two-step EI value amongst the BPD traits (two-step EI= 1.07), while the highest was 'intense mood shifts' (two-step EI=2.91). In the community group, the node representing 'hurting oneself when feeling upset' had the lowest EI values (one step EI=0.34, two-step EI=0.69), whilst the node representing 'chronic emptiness' yielded the highest one (one-step EI=1.21; two-step EI=1.99). Finally, in the clinical group, the lowest EI values corresponded to the node representing 'getting bored easily' (one-step EI=0.32, two-step EI=0.5). On the other hand, the highest one-step EI value was found to be the node related to 'worrying about people leaving' (one step EI=1.08) in this group, while the highest two-step EI value showed to be the node related to 'intense mood shifts'. EI values for all BPD-related features are summarised in Table 8.

Predictability

Table 8 also outlines the predictability values of the nodes of the MGM networks, revealing the proportion of variance in a node that is explained by its neighbours. Explained variance of a node is specified by R^2 (coefficient of determination; Haslbeck & Waldorp, 2018; Laerd Statistics, 2015).

The proportion of explained variance (represented as R^2 values in Table 8) reported in across all nodes ranged from 12% to 76% in the whole sample. Predictability values of ineffective modes of mentalisation were the lowest, with hypermentalisation, hypomentalisation and no mentalisation explaining 12%, 16% and 16% of variance respectively. Nodes with the highest predictability indices were 'impulsivity and recklessness' (76%), 'chronic emptiness' (67%) and 'inability to express anger' (61%).

In the model for the community group, the proportion of explained variance ranged from 16% to 47%. Predictability values of hypermentalisation, hypomentalisation and no mentalisation were the lowest (16%, 17%, and 16%, respectively). 'Chronic emptiness' and 'feeling that people are letting one down' (47%), 'impulsivity and recklessness' (46%) and 'unsteady mood' (46%) had the highest predictability indices.

In the model for the clinical group, the proportion of explained variance in ranged from 1% to 50%. Predictability values of hypermentalisation, hypomentalisation and no mentalisation were the lowest (14%, 17%, 17%, respectively). 'Impulsivity and recklessness' (50%), 'worrying about people leaving' (44%) and 'inability to express anger' (41%) had the highest predictability indices.

Table 8.
Results centrality analyses

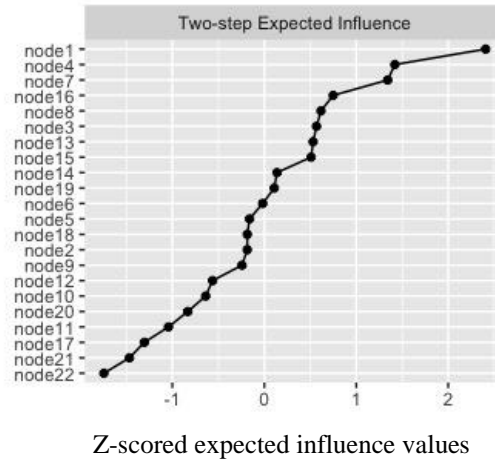
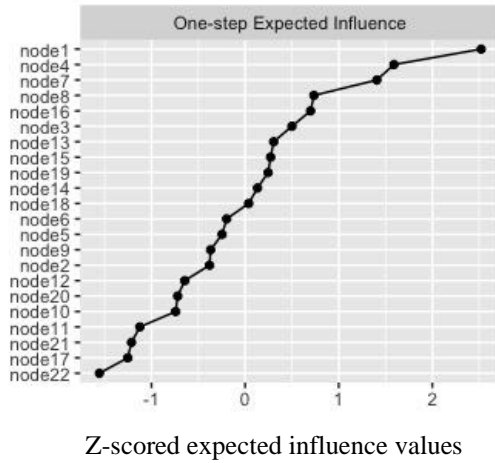
	N1 ^a : Intense mood shifts	N2: Attitude about self charges	N3: Stormy relationships	N4: Chronic emptiness	N5: Let people know they have hurt me	N6: Unsteady mood	N7: Worry about people leaving	N8: People let me down	N9: Little control over anger	N10: Wonder about life	N11: Feeling lonely	N12: Feeling unhappy	N13: Cannot handle separation	N14: Mistakes in picking friends	N15: Hurt self when upset	N16: Cannot express all of anger	N17: Gets easily bored	N18: Stay friends with people	N19: Impulsivity and recklessness	N20: MASCExc	N21: MASClless	N22: MASCN0
Whole Sample																						
Expected Influence																						
One-step	1.48	0.79	1	1.26	0.82	0.84	1.22	1.06	0.8	0.71	0.62	0.73	0.96	0.91	0.95	1.05	0.59	0.89	0.94	0.71	0.59	0.51
Two-step	2.91	1.62	2	2.42	1.64	1.71	2.38	2.02	1.6	1.4	1.2	1.44	1.98	1.79	1.97	2.09	1.07	1.63	1.77	1.3	0.99	0.85
Predictability (R ²)	0.76	0.48	0.6	0.67	0.46	0.57	0.59	0.53	0.55	0.4	0.33	0.47	0.48	0.46	0.6	0.61	0.24	0.19	0.52	0.12	0.16	0.16
Community group																						
Expected influence																						
One-step	0.96	0.9	0.84	1.21	0.81	1.01	0.82	1.07	0.47	0.49	0.45	0.53	0.49	1.01	0.34	0.6	0.52	0.71	0.75	0.59	0.51	0.45
Two-step	1.8	1.65	1.53	1.99	1.58	1.71	1.4	1.89	0.8	0.97	0.88	1.06	0.89	1.83	0.69	1.07	0.9	1.15	1.31	1.09	0.82	0.75
Predictability (R ²)	0.46	0.39	0.39	0.47	0.36	0.46	0.3	0.47	0.24	0.23	0.2	0.26	0.19	0.44	0.17	0.31	0.21	0.24	0.32	0.16	0.17	0.16
Clinical group																						
Expected influence																						
One-step	1.43	0.56	1.21	0.55	0.56	0.52	1.08	0.87	0.8	0.66	0.46	0.39	0.67	0.60	0.79	0.83	0.32	0.4	0.85	0.58	0.57	0.5
Two-step	2.51	1.12	2.15	0.97	1.06	1.05	1.87	1.55	1.48	1.05	0.79	0.74	1.29	1.08	1.56	1.55	0.5	0.65	1.61	0.99	0.91	0.79
Predictability (R ²)	0.5	0.23	0.39	0.19	0.23	0.2	0.44	0.32	0.39	0.22	0.13	0.01	0.34	0.24	0.29	0.41	0.08	0.09	0.32	0.14	0.17	0.17

Note. BPD=Borderline Personality Disorder. ^a N1-N23=Node1-Node23

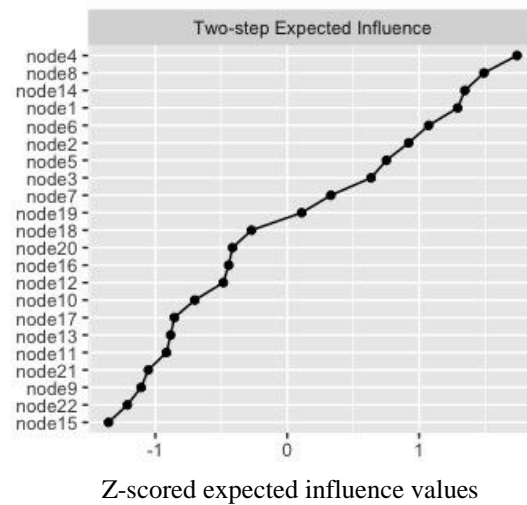
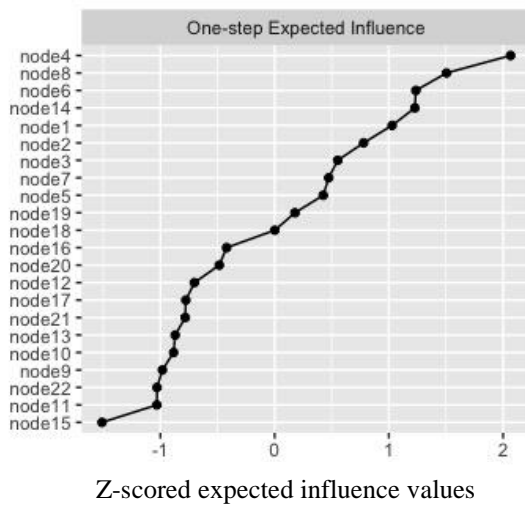
Figure 5.

One-step and two-step expected influence results

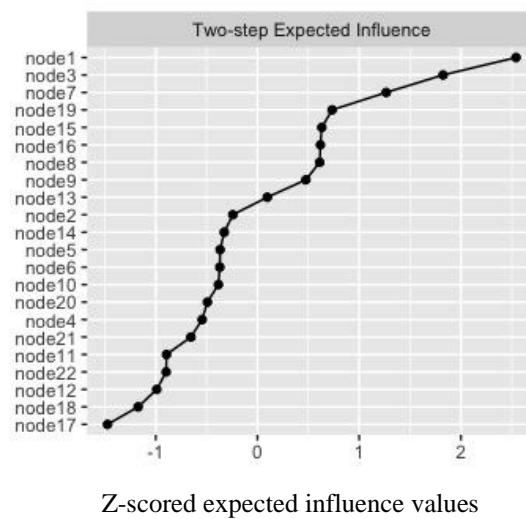
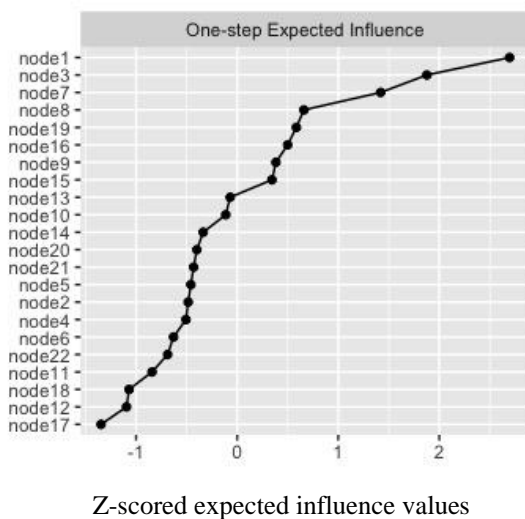
A. Whole Sample (N=575)



B. Community group (n=225)



C. Clinical group (n=350)



Note. BPD=Borderline Personality Disorder.

Legend. node 1= intense mood shifts, node 2= attitude about self changes, node 3=stormy relationships, node 4=chronic emptiness, node 5=let people know they hurt me, node 6= unsteady mood, node 7=worry about people leaving, node 8= 'people let me down', node 9= little control over anger, node 10= wonder about life, node 11= feeling lonely, node 12= feeling unhappy, node 13= cannot handle separation, node 14= mistakes in picking friends, node 15= hurt self when upset, node 16= cannot express all of anger, node 17= gets bored easily, node 18= difficulty with staying friends with people , node 19= impulsivity and recklessness, node 20= MASCExc (hypermentalisation), node 21= MASCCless (hypomentalisation), node 22= MASCCno (no mentalisation)

Power Analysis and Accuracy

As mentioned above, the estimated number of total possible edges (number of parameters) for the moderated network analysis was 253 ($(23 * 23 - 1)/2$). Based on the suggestion by Fried and Cramer (2017), 759 people (three participants per parameter) would have been the ideal sample size to achieve optimal power in the analyses. Since the study included 575 individuals in total (and significantly less for the subgroup network estimations within each of the two groups), the study is possibly underpowered, and the results have to be interpreted with caution. The relatively wide bootstrapped CIs around the estimated edge weights and moderation effects also suggest sub-optimal accuracy of estimates and therefore warrant a cautious interpretation of the findings (Epskamp et al., 2018). It is worth noting that bootstrapped CIs did not function as significance tests. This is because the presence of an edge after using LASSO regularization indicated that the edge was strong enough to be retained in the model. Therefore the presence of edges and their signs (positive or negative) were interpreted regardless of the relatively wide CIs and regardless of whether zero was spanned in these CIs (Epskamp et al., 2018).

Chapter Four: Discussion

Summary of Findings

The main objective of the current study was to evaluate the complex interactions between inadequate modes of mentalising and BPD symptomatology and to compare these between people with and without a BPD diagnosis. Building on extant literature, a network analysis approach was used to understand the role of mentalisation difficulties with BPD. Specifically, this approach could capture differences in these interactions across two levels of system activation: a less activated system in a ‘stable state’, corresponding to the group of people who did not have diagnosed mental health difficulties, and a more activated system in a ‘BPD-state’, associated with people who suffered from the severe and complex mental health problem referred to as BPD (Fried et al., 2017; van de Leemput et al., 2013). Based on the systematic literature review described in the first chapter and to the best of the researcher’s knowledge, this study is the first to explore the dynamic and interacting cross-sectional relationships between three types of ineffective modes of mentalisation and specific difficulties commonly linked to BPD using a matched sample of people with and without BPD diagnosis.

In line with the mentalisation-based model, it was hypothesised that connections between various mentalisation impairments and BPD-related features would be found, and that the patterns of these networks would differ between the two samples of participants. Overall, the findings of the study supported this hypothesis. Firstly, a significant difference was found between the two groups in terms of their overall level of connectivity. Secondly, while expected influence indices of ineffective mentalisation were amongst the lowest in both groups, predictability values of all included variables were relatively high, suggesting that the networks were determined by their interconnectedness to a significant degree. Thirdly, significant difference was shown between the two groups in their patterns of relations between difficulties BPD- and mentalisation-related difficulties. Specifically, moderation network analysis identified several pairwise associations between these variables, amongst which six were reported as the most robust and accurate ones. All of these associations were either positively or negatively moderated by the presence or absence of BPD diagnosis, suggesting that the pattern of connections differs between the two groups. In line with the mentalisation model, the BPD-related difficulties that were found to interact with ineffective modes of mentalisation were all of interpersonal nature.

The discussion of findings relies on the underlying premise that the interactions found in the network models may offer clues to causal dynamics as conditional independence relations connect network correlations to causal relations (Borsboom et al., 2021; Pearl, 2000). The results add to the growing literature on the effects of poor mentalising on BPD. They also offer a more comprehensive understanding of how mentalisation enhancing approaches can potentially deactivate the system of BPD-related features.

Discussion of Findings

The results regarding the overall connectivity of the networks will be discussed first. This will be followed by a discussion of the centrality and predictability findings. Then, differences in the structure of network (representing the pattern of relations between difficulties) with regards to specific nodes and edges linked to ineffective modes of mentalisation will be discussed in turn, starting with the most robust findings.

Network connectivity and structure. Network theory can identify topological differences in the network connectivity between people who suffer from mental health difficulties and those who do not (Borsboom, 2017b; Cramer et al., 2016; Fried et al., 2017; van Borkulo et al., 2015). The current findings revealed a significant difference in global strength (representing the total connectivity) and structure (representing maximum difference of pairwise edges) between the two groups. The network of the clinical group had higher connectivity than the network of the community group. This aligns with a central thesis of the network theory, which suggests that high network connectivity indicates an alternative and unhealthy ('disordered') state that is characterised by an increased vulnerability for symptom co-activation and a decreased ability to reduce this activation (Borsboom, 2017b; Cramer et al., 2016). It has been hypothesised that weakly connected networks (such as the one found in the community group) might respond to adverse environmental impacts by gradually and continuously increasing its connectivity rather than transitioning suddenly into a 'disordered' state (Fried et al., 2017; van de Leemput et al., 2013). In contrast, strongly connected networks, such as the one found in the clinical group, may be characterised by a prolonged period of recovery from additional external triggers due to the already high level of co-activation between the difficulties, meaning they are at higher risk for slow recovery from adverse environmental impacts (Fried et al., 2017). Therefore, the current results provide evidence for the presence of different levels of activation in people who suffer versus those who do not suffer from BPD. According to the network theory, this represents differing abilities to recover from adverse environmental events. This aligns with the mentalisation-

based theory of BPD which argues that people with BPD diagnosis are less able to regain mentalisation capacities after losing them in highly stressful situations, in turn sustaining their interpersonal difficulties (Bateman & Fonagy, 2016).

However, the study highlighted that while the clinical group have more complex interactions and stronger relationships between BPD-related difficulties and ineffective modes of mentalisation, people without any known mental health disorders are not immune to making mentalisation errors. The network analysis across the whole sample which contained group as a moderator, as well as the subsequent analyses within each group separately demonstrated several interactions between poor mentalising and BPD-related difficulties in the community sample. These findings are contrary to previous studies that suggested ineffective modes of mentalisation are uncommon in non-clinical individuals (Goueli et al., 2020; Normann-Eide et al., 2020; Quek et al., 2018). It must be noted that several other studies employing the MASC to examine mentalisation lack non-clinical control groups (Poznyak et al., 2019), and thus, cannot measure performance in such individuals. On the other hand, the current findings are consistent with a more recent hypothesis by Luyten, Fonagy, Lowyck, and Vermote (2012) which suggested that mentalisation abilities fluctuate, even in people without mental health difficulties. For instance, it may be more difficult to accurately mentalise about people outside of one's intimate social circle, particularly if an individual is experiencing attachment-related stress (Bartz, Zaki, Bolger, & Ochsner, 2011; Bowlby, 1973; Nolte et al., 2013). Fossati et al. (2018) also suggested people without mental health difficulties may at times struggle to mentalise. They found that non-clinical adolescents and adults mistakenly inferred mental states on nearly half of the MASC questions. Together, this research supports the notion that mentalising is not static, and that attachment-related stress particularly might induce automatic, non-mentalising states even in people with predominantly secure attachment styles (Bartz et al., 2011). However, since current findings showed that the overall level of network connectivity was lower in the community group, it can be speculated that co-activation between ineffective modes of mentalisation and BPD-related difficulties is more gradual and prone to recovery in the community group than in people in a more 'disordered' state (as suggested by the mentalisation framework as well).

Expected influence. Centrality analysis involving the exploration of one-step and two-step expected influence showed that modes of ineffective mentalisation had the lowest importance both in the whole sample and in the two groups. At first glance these results would suggest that the role of ineffective modes of mentalisation in the activation and

maintenance of the BPD difficulties is small. Whilst this might be a possibility, the low centrality estimates of mentalising difficulties could have stemmed from methodological issues as well. Meaningful interpretation of centrality indices in the context of psychology networks has been widely questioned in recent years (Bringmann et al., 2019). Dablander and Hinne (2019) found that centrality measures are often inappropriately used to infer causation. Several researchers have cautioned against interpreting symptoms with highest centrality indices as those with the most clinical relevance. It is particularly risky if this is used to inform primary targets of interventions, since the network merely indicates correlation (the extent of causation and directionality that can be drawn from networks are still debated) and the temporality of activations remain hidden in cross-sectional study designs (Fried et al., 2018; McNally, 2021). Some researchers have gone as far as suggesting to discard the concept of node centrality entirely in the field of psychological network research (Bringmann et al., 2019). Therefore, the low EI values do not necessarily mean that ineffective modes of mentalisation play a less important role in the activation and maintenance of BPD as a complex phenomenon. While centrality measures are still very commonly reported in network analysis studies (hence the decision to include them in the study), the suggestion of Bringmann et al. (2019) is followed, whereby the discussion of reciprocal associations found in the network is prioritised, as those may provide more accurate focus for interventions.

Predictability. Predictability is a network metric that has become a more widely accepted measure of practical relevance (McNally, 2021). Predictability provides information on the practical relevance of edges, quantifying how well nodes of the network are explained by the interconnectedness of the variables relative to the variance that is determined by other factors outside of the network (Haslbeck & Fried, 2017). Thus, it has been suggested that the absolute measure of predictability is more appropriate for identifying potential intervention targets than the relative measure of centrality (McNally, 2021). It was beyond the scope of the current study to report and discuss the predictability values of all variables included in the networks, especially the BPD-related nodes that showed no connections with the nodes representing ineffective modes of mentalisation (future studies are recommended to focus on these). It is however important to note that predictability of all the variables ranged from 10% to 47% in the community and 1% to 50% in the clinical group, with most of the variables showing predictability values between 10-40%. This means that for the majority of variables, the variance explained by neighbouring nodes reached the significance threshold recommended for social sciences ($R^2 > .04$, Ferguson (2009); interpreted as more than 4% of

variance explained by independent variables). In fact, over half of the predictability values were even higher than this threshold, indicating moderate goodness-of-fit effect sizes ($R^2 > .25$), as defined by Ferguson (2009). This strengthens the evidence for the pairwise interactions found between the variables and suggests that the networks generated in the current study were determined to a significant degree via mutual interactions between nodes (Haslbeck & Waldorp, 2018).

The moderation analysis identified six pairwise associations between BPD-related interpersonal difficulties and hypermentalisation or hypomentalisation that were moderated by the presence/absence of a BPD diagnosis. Overall, 20-45% of the variance of the respective nodes was explained by neighbouring nodes, including for the nodes representing ineffective mentalisation. This confirms the practical relevance of these edges and suggests that the respective nodes could be meaningful intervention targets. Therefore, improving ineffective modes of mentalisation could achieve effective change (Chen, 1988; Cohen, Cohen, West, & Aiken, 2003; Falk & Miller, 1992; Haslbeck & Waldorp, 2018).

Predictability values for three of the six variables ('wanting to let people know that they hurt one', 'feeling like people let one down', and 'mistakes in picking friends') were lower in the clinical group than in the community group. In other words, external factors not included in the network seem to have more effect on these variables within the clinical group. Such external factors might include childhood trauma, attachment styles, genetic dispositions, poverty and adversity (Bowlby, 1982; Cameron et al., 2019; Stepp et al., 2016). On the other hand, the predictability values for the variable 'stormy relationships' did not change between the two groups, whereas the predictability values for the variables 'worrying about people leaving' and 'struggles with handling separation' were significantly higher in the clinical group than the community group. The latter dynamic suggests that the nodes describing fear of abandonment in people with a BPD diagnosis are determined and explained by the neighbouring nodes (including hypermentalisation and hypomentalisation) to a greater extent in the clinical than in the community group (Haslbeck & Waldorp, 2018). This aligns with the mentalisation-based theory of BPD. It proposes that feelings of abandonment and separation are experienced in a non-mentalizing 'psychic equivalence' mode (defined as the inability to draw distinction between external reality and the contents of the mind) which lends these feelings unshakable ferocity and truth that reinforce the original pain via increased proximity seeking in a context where the acquisition of mentalising function is not possible (Bateman & Fonagy, 2016; Fonagy, 2008).

In addition to interpreting the predictability values of the BPD-related difficulties, it is also important to reflect on the predictability values of the ineffective modes of mentalisation. In both groups, only 14 to 17% of variance was explained in these nodes, which was lower than most other nodes. This suggests that the shift from ineffective to more effective mentalisation is largely determined by other, external factors and may not be subject to great variability when BPD-related features increase or decrease. This finding can be linked to the theory of mentalisation, which assumes the development of effective and ineffective mentalisation depends on the absence or presence of interactions with mature and sensitive minds in the contexts of attachment relationships (Fonagy & Allison, 2012). This relational aspect is a central feature of MBT, as it is hypothesised that ineffective modes of mentalisation may be corrected in time via the availability of the curious mind of a therapist, who focuses on people's subjective sense of self and helps them explore their minds (Bateman, Bales, & Hustebaut, 2012). Thereby, effective mentalisation is developed through interpersonal support and understanding rather than changes in BPD-related difficulties. Conversely however, improvement in mentalisation could activate changes in BPD-related difficulties, as evidenced by several systematic literature reviews investigating the effectiveness of MBT (Malda-Castillo et al., 2019; Vogt & Norman, 2019; Volkert et al., 2019).

Specific nodes and edges. The current study revealed several relationships between ineffective modes of mentalisation and specific BPD-related difficulties, some of which were found to be moderated by the presence of BPD diagnosis to a lesser or greater degree. Similar to the structure of the previous chapter, the discussion of these results is led by the findings of the moderation analysis conducted on the whole sample, since this provided the most robust, reliable and stable results on potential group differences (Haslbeck, 2022). However, some of the less powerful results discovered through subgroup analysis are also mentioned to provide guidance for future explorations. Results for MASC scales are discussed first, as the examination of these can facilitate the interpretation of the connections found between the ineffective modes of mentalisation and BPD-related difficulties.

MASC subscales. Hypomentalisation and no mentalisation were strongly positively related to each other in both groups, but particularly in the clinical group. The UVA analysis conducted in preparation for the network analyses found significant topological overlap between the hypomentalisation and no mentalisation subscales of the MASC, suggesting that the combination of these two factors into one overarching factor would improve the accuracy of the estimated models. While the two subscales were not merged in order to gain more

nuanced information about how they both individually relate to BPD features, it is possible that the subscales fail to distinguish differences between two modes of ineffective mentalisation (Dziobek et al., 2006) in the current sample. They may instead represent a spectrum of ‘reduced’ mentalisation, whereby each subscale refers to a different severity of hypomentalisation, depending on the extents of psychic equivalent thinking. While they may both reflect the inability to consider complex models of one’s own and others’ minds, they may not capture the total shutdown of mentalisation (representing the far end of the continuum of ‘reduced mentalisation’) and the subsequent inability to stay in contact with painful affects and thoughts that has been widely reported in people with a BPD diagnosis after being triggered by attachment-related stress (Arnsten, 1998; Halfon, Coşkun, Bekar, & Steele, 2020; Yeomans, Clarkin, Diamond, & Levy, 2008).

The concept of reflective functioning might be of potential relevance in relation to this argument. Reflective functioning has been used as a way to operationalise mentalisation for research purposes (Ha et al., 2013). The whole spectrum of reflective functioning (or in other words, mentalisation) has most effectively been captured by the Reflective Functioning Scale ([RFS]; Fonagy, Target, Steele, & Steele, 1998). The RFS was developed to measure individual differences in mentalisation in the context of attachment relationships and conceptualises mentalisation on an 11-point scale, ranging from negative, i.e., non-existent reflective functioning (-1) to exceptionally good reflective functioning (9). Evidence suggests that people with a BPD diagnosis score, on average, around 3 (‘questionable or low reflective functioning’ marked by ‘negative reflective functioning’ or ‘absent but not repudiated reflective functioning’; Chiesa & Fonagy, 2014; Fertuck, Mergenthaler, Target, Levy, & Clarkin, 2012; Fonagy et al., 1996). To the best of the researcher’s knowledge, no studies have compared performance on the MASC to scores on the RFS. Thus, it would be important that future studies examine whether the MASC no mentalisation subscale sufficiently captures the whole range of absent and negative mentalisation (represented as scores below 3 on the RFS) and thus differentiates total lack of mentalisation from hypomentalisation.

The results also showed a positive relationship between hypermentalisation and no mentalisation in both groups, which was stronger in the clinical group. Again, the RFS might come helpful in interpreting this result. On the RFS hypermentalisation is scored a 3 (thus conceptualised as ‘questionable or low reflective functioning’), but is marked by ‘overly analytical or hyperactive reflective functioning’ (Fonagy et al., 1998). Therefore, hypermentalisation is captured on the scale as a type of reduced mentalisation, even though it is described as over-involvement and sustained attention to the mental states of other people.

Hypermentalisation stems from pretend mode, which is also an essentially reduced stance of mentalisation (Bateman & Fonagy, 2016). As argued above, the non-mentalising scale of the MASC may pick up on the general reduction or disturbance of mentalisation that is intrinsically part of hypermentalisation as well, rather than the total lack of mentalisation, hence the connection between the two subscales.

Community group. As mentioned above, the current research revealed people without a mental health diagnosis also used ineffective mentalisation strategies. The following will discuss findings for each mode of ineffective mentalisation. This will be followed by a speculation of the possible roles of attachment and epistemic trust.

Hypermentalisation. Three difficulties associated with BPD (‘experiencing stormy relationships’, ‘worrying about people leaving’, and ‘feeling like one makes mistakes when picking friends’) were positively associated with hypermentalising. This suggests that from the numerous BPD-related difficulties examined in this study, these particular interpersonal problems trigger an over-analysing, over-interpretative stance, in which identifying one’s own and others’ mental states becomes excessive and overly certain without substantial evidence. In turn, hypermentalisation appears to further reinforce these difficulties, leading to self-maintaining feedback loops. These findings are congruent with previous research that found hypermentalisation is the most common mode of ineffective mentalisation in non-clinical samples (Fossati et al., 2018; Poznyak et al., 2019). Since mentalisation facilitates affect regulation (Fonagy et al., 2000; Schwarzer, Nolte, Fonagy, & Gengelmaier, 2021), it could be speculated that hypermentalising is a strategy (however unhelpful it may be on the long term) to regulate enhanced emotions that arise in interpersonal conflicts, regardless of the presence of mental health problems. Another possible interpretation is that hypermentalising functions as a self-defence to enhance one’s self-esteem and integrity in the face of interpersonal discomfort. This would occur due to an increase in attention to external indicators of mental states, generating long and overly detailed interpretations about the motives of one’s own and others’ actions, while adaptive mentalisation strategies are temporarily inaccessible due to attachment-related stress (Bateman & Fonagy, 2016; Bowlby, 1973; Fonagy et al., 2016; Liotti & Gilbert, 2011). A similar suggestion was put forward by Herrmann et al. (2018), who believed that attachment distress and the related heightened emotional arousal might initially trigger affect-centred mentalizing. This would express itself in the form of urgent attempts to understand what’s in the mind of others in order to enhance affect regulation and communication (although they assumed this process to be present in their clinical sample).

Fonagy, Luyten, Allison, and Campbell (2017) recently emphasised the role of epistemic hypervigilance in the manifestation of hypermentalisation. Epistemic trust is the openness to receive social knowledge and encode it as personally significant, relevant, and socially generalisable (Csibra & Gergely, 2009, 2011). Epistemic hypervigilance on the other hand is defined as a chronic mistrust of both attachment figures and strangers as sources of information and is thought to result from childhood maltreatment, whereby one's ability to develop epistemic trust is hindered (Fonagy & Allison, 2014). People facing the three above-mentioned interpersonal difficulties may experience epistemic hypervigilance, which is linked to their hypermentalising strategy and which appears as poor inference of intentions and the perception of malevolent motives behind people's actions (Fonagy et al., 2017).

Hypomentalisation. The BPD-related difficulty 'struggling to handle separations' and the 'desire to let one know how much they hurt one' was positively associated with hypomentalisation in the community group. A potentially similar tendency was shown in relation to hypermentalising, which was negatively correlated with 'feeling that one is let down by people'. While being disappointed in people seemed to decrease the hyperactivation of the mentalisation system, hypermentalising tendencies not only decreased but actually shifted to hypomentalisation when people feel a desire to let people know about the psychological damage done to them and when struggling with separation. In these cases, hypomentalisation may function to defend one's psyche against emotional overinvolvement and traumatisation (Brüne, Walden, Edel, & Dimaggio, 2016). This dynamic may also be related to the closing off of the epistemic channels, which leads to a decrease in the willingness for social communication (Fonagy et al., 2017).

The role of emotional arousal. As mentioned above, one explanation for the opposing tendencies of hypermentalising and hypomentalisation in the context of various interpersonal difficulties might stem from the elevated emotional arousal that often occurs in intimate relationships. The biobehavioural switch model of Fonagy and Luyten (2009) focuses exactly on the role of emotional arousal on mentalising capacities. It presumes that interpersonal stress affects mentalisation in the shape of an inverted U-curve, depending on the extent of the emotional arousal. Thereby, an increase in stress generated by interpersonal problems activates one's attachment-system and enhances mentalisation, but if it reaches a "switch point", the emotional arousal overwhelms the system and has an opposite impact, leading to the inhibition of controlled mentalisation and the rise of ineffective modes of mentalisation.

Based on this theory, the current research highlights the possibility to rely on a hypermentalising strategy when one encounters specific interpersonal difficulties as a first

step. Hypermentalising may manifest itself shortly after reaching the ‘switch point’, when efforts to understand others’ behaviours in terms of mental states may still be big, but cognitive control is already lacking (Bateman & Fonagy, 2016). Yet, as the emotional arousal continues to grow and eventually gets pushed ‘over the edge’, hypermentalising may collapse into increasing levels of hypomentalising. Therefore, as different types of interpersonal problems escalate one’s emotional arousal, the tendency to start hypermentalising or hypomentalising may depend on the level of stress that was activated. These ineffective modes of mentalisation then escalate the interpersonal problems further in the form of vicious cycles. It is important to note however that the level of attachment-related emotional arousal triggered during the MASC is not determined. The situations in the movie are meant to elicit strong emotions, such as anger, jealousy, fear, embarrassment and affection (Sharp & Venta, 2012), which likely trigger attachment-related stress (Bowlby, 1973). Yet, future researches should include further measurements that capture mentalisation in the context of emotional arousal and attachment-related stress, which would also facilitate a better understanding of how particular attachment strategies relate to ineffective modes of mentalising and varying severity of BPD-related problems.

The role of epistemic trust. Another way of understanding the variation between hypermentalising and hypomentalising is related to epistemic trust. It is possible that ‘experiencing stormy relationships’, ‘worrying about people leaving’, and ‘feeling like one makes mistakes when picking friends’ increase one’s epistemic hypervigilance and hypermentalising tendencies, characterised by decreased ability to think flexibly and mentalise, thereby maintaining rigid knowledge structures even when these are incorrect or misleading (Kruglanski & Webster, 1996). In this state individuals are seen less trustworthy and knowledgeable and social communication is less likely to be encoded as significant or worthy of one’s consideration, leading to a ‘hearing but not listening’ stance (Bateman & Fonagy, 2016; Fonagy et al., 2017). On the other hand ‘feeling that people let one down’, the ‘desire to let one know that they hurt one’, and the ‘intolerance of separation’ might have an opposite effect and close down one’s epistemic channels, leading to short periods of epistemic mistrust or outright epistemic freezing, which is described as a complete inability to trust others as source of knowledge about the world (Fonagy & Campbell, 2017).

The relationships between BPD-related difficulties and hyper- and hypomentalisation found in the community group align with the notion that ‘normal’ personality is not inherently stable or consistent in social situations. Instead, people’s epistemic trust is less likely to be impaired in this group, so they are less vulnerable to become stuck in rigid, non-

flexible thinking, and thus can return to adequate reflective functioning quicker (Fonagy et al., 2017). This interpretation is thus similar to the core assumption of network theory in that the activation of the network of difficulties in people without mental health disorders calms down faster after a triggering environmental impact such as an interpersonal conflict than the activation of the network of people with mental health problems (Fried et al., 2017). This dynamic would however have to be confirmed by network studies using longitudinal measurements.

Clinical sample. The interactions found between ineffective modes of mentalising and BPD-related difficulties were complex and not all amenable to straightforward interpretation based on existing theory and practice. Therefore, these relationships are discussed in turn in order of BPD-related difficulty (rather than ineffective modes of mentalisation as previously), and compared to those found in the community group. This is followed by a general discussion of the most important factors that could have contributed to the results.

Stormy relationships. In the clinical group, a negative association between ‘stormy relationships’ and hypermentalising existed. This is contrary to the community group, where the association was found to be positive. Subgroup analysis suggested that the relationship between these variables is likely to become negative in the clinical group contrary to the community group where hypermentalising was found to increase the likelihood for ‘stormy relationships’ and vice versa. Furthermore, subgroup analysis also found a weaker negative association between ‘stormy relationships’ and hypomentalisation in the clinical group. This indicated that as people with BPD diagnosis increasingly feel that their relationships are stormy, they hypermentalise less (and may also hypomentalise less). Hypermentalisation has been found to be positively related to BPD traits in adolescents (Sharp et al., 2013; C. Sharp et al., 2011). However, increasing amount of research has suggested that the more substantial problem in adults with BPD is hypomentalisation (Adamsons, 2015; De Meulemeester et al., 2018; Euler et al., 2021; Kvarstein et al., 2020).

The tendency for people with BPD diagnosis to reduce the excessive attempts to reflect on other’s and one’s own behaviours when encountering stormy relationships is congruent with the ‘switch model’ discussed above. Individual ‘switch points’ have been suggested to be largely underpinned by attachment history and specific attachment styles (Fonagy & Luyten, 2009). For instance, disorganised attachment style, which has been linked to BPD, is assumed to lead to hyperresponsivity to emotional distress, with alternate frantic attempts to downregulate and dismiss emotional arousal (Luyten, Campbell, Allison, &

Fonagy, 2020). It is thus possible that experiencing stormy relationships can escalate emotional arousal in people without mental health difficulties leading to hypermentalisation, but for people with a BPD diagnosis, who often have insecure attachments and traumatic childhood experiences (Gunderson, 1996), these social conflicts are so overwhelming that they lead to a declining ability to mentalise altogether. The results might also be explained by increased epistemic mistrust in people with BPD diagnosis. Epistemic mistrust can make individuals hard-to-reach on an interpersonal level and find it hard to trust what they hear (Fonagy & Campbell, 2015; Tamas, 2016). This can hinder their ability to mentalise others and themselves and thus reinforce the presence of stormy relationships in the form of a vicious cycle.

Worrying about people leaving. ‘Worrying about people leaving’ was even more strongly positively related to hypermentalisation in the clinical group than in the community group. As mentioned above, hypermentalisation has been linked with BPD (Sharp & Vanwoerden, 2015), which this study confirms, at least in the context of worrying over abandonment. Furthermore, in the clinical group, ‘worrying about people leaving’ was associated with hypomentalisation.

Excessive fear of abandonment has been a long-standing and widely observed feature of people with a BPD diagnosis (APA, 2013; Masterson, 1972), often associated with insecure attachment styles (Gunderson, 1996) and altered, ineffective mentalisation (Fonagy et al., 2002). Imagined and real abandonment has been shown to be particularly painful for people with a BPD diagnosis and is thought to be experienced in the non-mentalising stance of psychic equivalence, where worries about people leaving become ‘too real’ and automatically true in the physical world (Bateman & Fonagy, 2015; Juul, Simonsen, & Bateman, 2020). The pain of the experience of being left by people can increase depression, which itself has been linked to hypomentalising-hypermentalising cycles (Luyten & Fonagy, 2015). The increased association with hypermentalisation may represent the hypermentalising side of the cycle, just before becoming too overwhelmed and shifting into hypomentalising. This therefore also aligns with the perspective of the ‘switch theory’.

Feeling that people let one down. The BPD-related difficulty of ‘Feeling that people let one down’ was not significantly related with hypermentalisation in the clinical sample, even though it was significantly negatively associated in the community group. In other words, the feeling that others let one down decreases people’s tendency to ‘overthink’ in people without BPD diagnosis (potentially as a way to defend themselves) but this tendency cannot be confirmed in the clinical group.

At first, this is a surprising result, since being disappointed in people is often linked with non-mentalising, dismissive attitudes (Bateman & Fonagy, 2003). However, the lack of connection in the clinical group might be explained by the psychometric properties of the PAI-BOR and the MASC. The PAI-BOR is a self-report questionnaire asking people to provide information about their psychological problems that they may be unaware of or may be particularly sensitive and painful for them (Balsis, Loehle-Conger, Busch, Ungredda, & Oltmanns, 2018). Blind spots, unstable and distorted cognitions and lack of self-knowledge may all hinder people's ability to provide a clear account of their feelings regarding interpersonal situations, potentially consciously or unconsciously protecting their self-worth (Balsis et al., 2018; Vazire & Carlson, 2011). Therefore, it is possible that people with a BPD diagnosis could not provide a reliable measurement on the PAI-BOR variable 'feeling that people let one down'. Furthermore, the experience of being let down is often very familiar and chronic in people with BPD, manifesting as epistemic mistrust (Fonagy et al., 2017). As a result, the feeling of being let down may become a default state, and thus not even trigger mentalising efforts.

Furthermore, the psychometric properties of the MASC may also help to explain this surprising finding. Feeling as though one is let down by people could increase psychological arousal to the point that the mentalisation capacities are overwhelmed and the whole system shuts down in a defensive inhibition of interest in people's minds. This state may resemble the body's fight-flight-freeze response to danger, which leads to the collapse of mentalisation (Fonagy, Bateman, & Luyten, 2012). Diminished mentalisation has been shown to be a common response, for example, in the case of childhood sexual abuse (Ensink, Bégin, Normandin, Godbout, & Fonagy, 2017) or emotional over-involvement in situations where the attachment system is greatly triggered (Bowlby, 1973; Brüne et al., 2016). As a result, the representation of others' and one's own minds can literally be obliterated and empty, hostile schematic images may take over (Asen & Fonagy, 2012). In psychoanalytic literature, this response could be similar to what Klein (1946) described as the paranoid-schizoid position, dominated by powerful early anxieties of annihilation, persecution, fragmentation and disintegration, leading to the breakdown of symbiotic thinking. It is possible that the MASC non-mentalisation scale does not capture this terrifying state of non-thinking and non-mentalising. This may also explain how it was similar to the hypomentalisation scale. Moreover, it is also possible that people with a BPD diagnosis make more mistakes in relation to mentalising about their minds rather than others', which cannot be captured by the MASC. This is a major limitation of the MASC.

Struggles with handling separation. In the clinical group, the BPD-related difficulty 'difficulties with separation' was negatively related to hypomentalising, whereas in the community group, it was positively related. However, similar to the variable 'feeling that people let one down', the relationship between 'difficulties with separation' and ineffective modes of mentalisation were not strong enough to be reported. The lack of a relationship to ineffective modes of mentalisation is once again counter-intuitive and contradictory to previous accounts, because intolerance of separation has been shown to be related to insecure attachment and highly dependent and idealising interpersonal styles in people with a BPD diagnosis (Kreisman & Straus, 2010). However, just as before, the psychometric properties of the PAI-BOR and the MASC may not capture the whole range of non-mentalising experiences that people with BPD diagnosis may feel when they encounter separation in relationships where attachment anxieties are evoked. Experiences of painful separations in childhood leaves a core wound, which can alter the perception of separation from significant others later in life. In such moments later in life, significant others may become seen as persecutory, abusive, neglectful and non-trustworthy of social communication (Fonagy et al., 2002; Yeomans et al., 2008). Therefore, people with very painful experiences of past separations may be unwilling or unaware to report these struggles when assessed as they might be too difficult to acknowledge. Furthermore, the tendency to defend against psychic pain reduces mentalisation, but the MASC may not trigger such a defence, and thus, it may not capture respective impairments in mentalisation. Similarly, separation may also be more related to ineffective mentalisation about one self, not others, meaning this might also not be captured by any of the MASC scales.

Mistakes in picking friends. While the tendency in people without mental health difficulties seem to increase their efforts to understand the reasons behind choosing friends by paying inordinate attention to others and making groundless inferences about their mental state (and vice versa), in the case of people with BPD diagnosis, no ineffective modes of mentalisation was confirmed. This result was also counter-intuitive, since individuals with a BPD diagnosis are not only known to have conflicts in their intimate interpersonal relationships but also in their friendships (Ansell, Sanislow, McGlashan, & Grilo, 2007; Javaras, Zanarini, Hudson, Greenfield, & Gunderson, 2017). However, similar to previously mentioned nodes, people with BPD diagnosis might not be willing to admit this in a self-report questionnaire or they may have a blind spot about recognising the difficulty of choosing friends poorly in the first place. In this case, not only would they not report it on the

PAI-BOR, but mentalising about its reasons and effects would neither be active nor blocked (Duschinsky & Foster, 2021).

On the other hand, if awareness about feeling like one has made mistakes in picking friends is present, it could lead to intense emotions and impulsive decision-making about ending friendships to avoid abandonment and feelings of betrayal (Hayashi, 1996; Hoveidafar, Fatehizade, Ahmadi, Jazayeri, & Abedi, 2017). All these enhanced emotions could result in the complete shutdown of mentalising, whereby mentalising becomes unavailable (Bateman & Fonagy, 2016). Again, this scenario is likely not captured fully by the MASC. Alternatively, being aware of making mistakes in picking friends may primarily trigger self-mentalising inaccuracies in people with BPD diagnosis, which, as mentioned, are also not captured by the MASC. However, the difference found between the two groups on this specific BPD-related difficulty was confirmed in less than half of the bootstrap samples; thus, these results are less robust than those detailed above.

Letting people know that they hurt one. The BPD-related difficulty of ‘wanting to let people know that they hurt one’ was positively associated with a tendency to hypomentalise in the community group, which appeared to be very unlikely (possibly absent) for people with BPD diagnosis. In fact, subgroup analysis showed the presence of hypermentalisation in the clinical group in relation to this difficulty. The latter however needs to be interpreted with caution due to the low stability of this relationships. The hypomentalising found in the community group suggests a decreased clarity and confidence about one’s own and others’ mental state, leaving people anxious about how to approach others and how to react (Haukefer & Korsan, 2020). Yet, for people who have a BPD diagnosis, this tendency reduces, and possibly shifts towards hypermentalisation, which is potentially associated with higher likelihood of confronting others. This also means that people with a BPD diagnosis may become overconfident about their inferences of mental states during a conflict, meaning they may be less likely to question mental states in the midst of it (Haukefer & Korsan, 2020). In other words, the epistemic hypervigilance of people increases, which leads to a use of rigid templates relating to the self and others and an unwillingness of listening to different views (Tillman, 2018), potentially resulting in interpersonal conflict and inappropriate responses (Hatkevich, Venta, & Sharp, 2019). However, the relationship between ‘letting people know that they hurt one’ and ineffective mentalisation was only found in less than two-third of the bootstrap samples of the moderation analysis, which limits the stability and accuracy of this finding.

Further connections. Several relationships between nodes were identified in the subgroup analyses, even though they were not revealed in the whole sample network. Importantly, the power of these analyses is significantly lower since the subgroup networks included fewer participants. Nevertheless, such relationships with the strongest edges found in the clinical group is worthwhile and may guide future research.

A moderate positive pairwise association was found between hypermentalisation and ‘struggles with expressing anger’ and ‘impulsivity and recklessness’, while a moderate negative association was found between hypermentalisation and ‘wondering about life’. These results add to extant literature about the presence of hypermentalisation in people with a BPD diagnosis (Kvarstein et al., 2020; Sharp et al., 2013; C. Sharp et al., 2011; Sharp & Vanwoerden, 2015; Somma et al., 2019) in the context of affective instability and identity diffusion. Furthermore, a moderate negative association was found between hypomentalisation and ‘unsteady mood’, which confirms the presence of simultaneous hypomentalising tendencies as well (Brüne et al., 2016; Euler et al., 2021; Goueli et al., 2020; Vahidi et al., 2021).

Overall summary of results. The investigation of the unique associations revealed complex findings, which were considered in the context of several factors that might contribute to these associations within the clinical group. The BPD-related difficulties found in the whole sample through the moderation analysis were of interpersonal nature. This supports the mentalisation-based understanding of BPD that assumes that ineffective mentalisation is of fundamental importance in activating BPD-related features as it impairs one’s self-other relatedness and interpersonal functioning (Bateman & Fonagy, 2010; Daubney & Bateman, 2015).

The current study provided evidence that hypermentalisation and hypomentalisation simultaneously exert influence on BPD-related difficulties, and vice versa, when examined in a network of BPD-related difficulties. However, the activation of these network edges does depend on the type of interpersonal difficulty encountered. For instance, ‘worrying about abandonment’ and ‘wanting to let people know that they hurt one’ seem to escalate hypermentalising tendencies, while ‘stormy relationships’ seem to be linked with hypomentalising. These results differ from many previous studies that found either hypermentalisation or hypomentalisation as the main mentalisation-related difficulty in BPD, but which rarely suggest a co-existence of both. Future investigations should focus on the underlying mechanisms that lead to specific types of interpersonal and intrapsychic difficulties which in turn create reinforcing feedback loops with either excessive or reduced

mentalising. Furthermore, while comorbidity was not considered in the current study, it would also be important to control for depression in future research. Depression is known to create a hypomentalising, psychic equivalence state in people with a BPD diagnosis, which itself can trigger a period of hypermentalising state and vice versa (hypermentalising can also trigger hypomentalising), leading to ‘dysfunctional interpersonal transition cycles’, or in other words, hypermentalising-hypomentalising cycles (Duschinsky & Foster, 2021; Luyten & Fonagy, 2015; Luyten, Fonagy, Lemma, & Target, 2012). If comorbid depression was present in the sample, it might have influenced the results by impacting one side of a hypermentalising-hypomentalising cycle, fuelling it to a greater extent than what would be found in a sample where depression was absent.

The current findings highlight differences in the unique associations between the two groups. The mediating role of emotional arousal in the context of insecure attachment has been discussed. Based on the biobehavioural switch model by Fonagy and Luyten (2009), it has been proposed that emotional arousal increases until it reaches a switching point, after which mentalising diminishes. People may temporarily maintain an extent of curiosity about mental states expressed as hypermentalising, or reduce mentalising completely if thinking becomes too painful (or change rapidly between the two states in the case of comorbid depression; Duschinsky & Foster, 2021). The switch point of people is idiosyncratic, depending largely on whether developmental trauma and insecure attachment style are present, which usually are in the case of people with a BPD diagnosis (Luyten et al., 2020). The role of epistemic trust has also been suggested to contribute towards the differences between the two groups. The absence of epistemic trust is linked with the development of disorganised attachment styles and poor mentalising, and represents a state characterized by a general loss of interest in social communication and an expectation of a threatening world (Fonagy & Allison, 2014; Stubble, 2021). Epistemic mistrust is often elicited by emotional overarousal and hypermentalising tendencies (Bo, Sharp, Fonagy, & Kongerslev, 2017), resulting in a reduced level of openness to new information and different perspectives, withdrawal from interactional exchange and reduced exploration of internal states (Fonagy & Allison, 2014; Haslinger, 2014). The theory of epistemic mistrust may overlap with the network theory, which captures the self-sustaining aspect of highly connected systems that are difficult to de-activate due to their existing patterns of connectivity (Borsboom, 2017b). Both would suggest that people’s level of psychopathology depends on the level of rigidity that they exhibit in the face of new experiences and information, particularly in emotionally stressful social situations (Fonagy & Allison, 2014).

An important finding of this research is that some of the BPD-related interpersonal difficulties (namely ‘feeling that people let one down’, ‘intolerance of separation’ and ‘mistakes in picking friends’) only showed connections with ineffective modes of mentalisation in the community group. These connections were weaker or not present in the clinical group. The idea that mentalisation in the context of such difficulties is intact in the clinical group is highly unlikely both from a research and a clinical perspective, as these difficulties have been so widely observed that some are even included in diagnostic instruments ([APA]; 2013). It does however raise questions about the reliability and validity of self-report questionnaires in capturing BPD-related difficulties. People may be motivated to protect themselves by pretending to not be aware of or not admitting their most painful difficulties on a questionnaire for many conscious and unconscious reasons (Vazire & Carlson, 2011). For people with a BPD diagnosis this is particularly difficult due to the unstable self- and other-representations (Choi-Kain, Fitzmaurice, Zanarini, Laverdière, & Gunderson, 2009) and because self-report requires self-mentalisation and meta-insight in the first place (Gallrein, Carlson, Holstein, & Leising, 2013). Thus, in relation to these specific difficulties, people without mental health diagnoses might have more insight and awareness than people who have a BPD diagnosis. Indeed, few studies have shown a discrepancy between self and informant report on BPD symptomatology (Balsis et al., 2018; Busch et al., 2016), which stresses the importance of additional informant reports in future network analysis studies about BPD. However, the lack of results regarding these particular difficulties might also be related to the psychometric properties of the MASC. It has been argued that the no mentalisation scale of the MASC does not capture the whole domain of total lack of mentalisation and may instead represent the lower end of a spectrum of hypomentalisation. Other instruments such as the RFS might capture no mentalisation better. Finally, the MASC prompts people to primarily reflect on other-related mentalisation, while the self-domain of mentalisation is not measured. This could have also added to the lack of results in relation to particular interpersonal problems.

Strengths

The current study had several theoretical and methodological strengths. The chosen approach of network theory and analysis introduced a novel theoretical and statistical model that challenges the conceptualisation of mental health difficulties offered by latent variable models. The network approach provided a useful framework for the unique and ambitious research aim, which focused on the investigation of the patterns of associations between

ineffective modes of mentalisation and specific BPD-related features in individuals with and without BPD diagnosis. A systematic literature review was conducted to investigate the originality of this research question and to evaluate methods and results of previous research that used network theory to study BPD. Based on this review it was confirmed that the present study is the first application of network theory and analysis in the understanding of BPD via the mentalisation framework. Additionally, network analysis proved to be an advantageous statistical tool for analysing and representing the complexity of the connections between BPD- and mentalisation-related variables. The analysis also addressed potential fluctuation of results that could have occurred due to chance by including model selection methods, such regularisation strategies.

Further strengths of the current methodology include the robustness of the dataset, as data was collected rigorously, using validated measures and standardised research protocols. Additionally, this study relied on one of the largest clinical sample of individuals with a primary diagnosis of BPD to date. Individuals in the clinical sample received the BPD diagnosis from mental health professionals and transdiagnostic bias was screened for at recruitment, which increased homogeneity in the clinical sample and the generalisability of the findings to people with a primary diagnosis of BPD in the wider population. On the other hand, individuals from the community group who suffered mental health problems were excluded prior to analysis. Additionally, the sampling strategy reduced bias stemming from between-group difference in the community group further as community participants did not differ from the clinical group on some of the demographic characteristics. Therefore, the reduced bias allowed for the effective exploration of the interconnectedness in clinical (activated) and community (non-activated systems). Data analysis was statistically rigorous, including a constant evaluation of stability, accuracy and robustness (via UVA and bootstrapping). This increased the internal validity of the findings. Exclusion bias stemming from the exclusion of participants with fully missing data was addressed and missing data analysis was conducted for the included participants.

Overall, the study identified several interactions between different forms of mentalising difficulty and features associated with BPD presentations but of a more complex, less straightforward way than suggested in many accounts. The results have several important implications for both clinical practice and theory development and provide fertile ground for further research into the importance of mentalisation and the mechanisms that may escalate or de-escalate it. These implications are detailed below.

Limitations

Several limitations have to be considered when evaluating this research. Firstly, the bootstrapping of the network parameters showed that the variance of the sampling distribution for pairwise effects was large (i.e., the CIs around edge weights were wide), which suggests that the stability and reliability of the findings are questionable. Secondly, while the proportion of people with BPD diagnosis was remarkably great, the size of the overall sample (and especially the samples used in subgroup analyses) was too small for the statistical demands of network analysis (Fried & Cramer, 2017). The relatively small sample size might have contributed to the wider CIs and therefore sub-optimal accuracy of the estimates, or it might have introduced additional instability to the models. Thirdly, the decision to pursue an exploratory approach meant a trade-off between the sensitivity and precision was required. To err on the side of discovery rather than caution, liberal statistical penalties in the regularisation techniques of the analysis were used. This meant that the statistical approach favoured less sparse networks and sensitivity (true positives) over precision (true negatives; Burger et al., 2020). In other words, low estimation errors for group differences were preferred at the expense of high estimation errors for absent ones (Haslbeck, 2022). Due to the general instability of the estimates, results (especially weaker ones) should only be interpreted tentatively, and future research should seek to confirm these results using stricter thresholds.

Another limitation of the methodology stems from its cross-sectional and correlational nature. The cross-sectional nature of the study provided a “snapshot” of the relationships found in the networks. This has exploratory value in understanding functional within-subject dynamics in mental health problems (von Klipstein et al., 2021). However, the temporal directionality of these relationships remains unknown, alongside the changes in the associations that may occur over time (Peckham et al., 2020). Although the associations and centrality indices found in the research may be indicative of causal relationships, the extent of bidirectional causal inferences that can be drawn from these is still heavily debated (von Klipstein et al., 2021). Moreover, it is still possible that some of the relationships found in the networks are results of common causes, latent variables or indirect causal links that the current research did not account for (von Klipstein et al., 2021). In addition, the potential limitations of the measures used have already been noted. Whilst both PAI-POR and MASC have merits, they for different reasons may have been a factor in some of the less theoretically explicable results. Thus, future network studies should consider using alternative, more sensitive ways of measuring both BPD- and mentalisation-related problems

(i.e. with different instruments and inclusion of informant reports). This could help to confirm and deepen the understanding of the results generated in the current study.

Further limitations stem from the sociodemographic characteristics of the sample. The individuals excluded in the analysis due to having too much missing data had significantly different gender and age to the individuals included in the analysis. This might be indicative of bias in the results. Furthermore, there were significant sociodemographic differences within the included participants, which depended on the group they were part of. As these were not covaried for, we cannot be sure if some of these differences contributed to the results. Moreover, most participants were relatively young females from a white racial background. The low sociodemographic diversity of the sample affects the generalisability of the findings, which highlights the need for increased representation of marginalised populations in the replication of the current findings.

Finally, people with a BPD diagnosis may experience and express emotional distress in very different ways (Skodol et al., 2002). Fitting group-level models to BPD-related difficulties may conceal within-group heterogeneity and reduce the external validity of the findings. Similarly, the reporting and discussion of the results were limited to associations found with ineffective modes of mentalisation, meaning the current study could not reveal relationships with appropriate mentalisation.

Implications of the Study

Despite these limitations, the results of the current research have various important implications for clinical practice, theory development and future research, as discussed below.

Normalisation and anti-stigmatisation. The finding that ineffective modes of mentalisation increase the likelihood for several BPD-related difficulties and vice versa in both the community and the clinical samples promotes an inclusive and less stigmatising understanding of mental health difficulties and mentalisation errors. This avoids the narratives inherited in diagnostic manuals which promote a stark difference in the personality of people with and without mental health difficulties (Borsboom, 2008). Therefore, these results support the importance of psychoeducation and particularly, normalisation of mental health problems, and particularly BPD (Gunn & Potter, 2015). They also highlight the necessity for mental health being viewed less like an ‘illness’ in wider societal discussions and policy-making decisions, and more like the result of multiple forms of intersectional social and relational disadvantage (Speed & Taggart, 2019).

The results also support the notion that ineffective modes of mentalisation are present in people regardless of the presence or absence of mental health diagnosis. This was not always the dominant view in mentalisation-based theory. But more recently, the defensive, self-preserving function of insecure attachment styles, closed epistemic channels and ineffective mentalisation strategies have been reformulated as understandable attempts to adapt to difficult broader cultural and socioemotional factors, regardless of how maladaptive their consequences may be (Fonagy & Allison, 2014; Luyten, Campbell, & Fonagy, 2019). The ability to regain one's mentalisation capacities quickly has long been an important goal of MBT (Bateman & Fonagy, 2016), but the importance of this has recently been emphasised in non-clinical individuals as well in terms of resilience and coping with general adversities of life (Schwarzer et al., 2021). Mental health problems are increasingly understood as resulting from rigid socio-communicative templates and an inability to trust others enough to update one's socio-cognitive models. This is contrary to an understanding of illnesses as being intrinsic, which would differentiate people, perpetuate the 'us-them' narrative and facilitate hierarchical differences in status and power (Speed & Taggart, 2019). The current research supports this notion and future research into mentalisation and BPD (and other mental health problems) are also recommended to further facilitate this changing narrative.

Diagnosis and assessment. Despite the fact that a diagnosis facilitates the presence of stigmas to some extent (Gunn & Potter, 2015), the complete eradication of diagnostic procedures seems unlikely in the near future. However, the results of this study have an important implication for the revision of diagnostic procedures that capture the complexity of mental health problems and specifically BPD. The difference found in the level and patterns of system activation between the two groups supports a diagnostic procedure that combines categorical (i.e. looking at whether the system of BPD-related difficulties is activated) and dimensional views of mental health problems (i.e. how severe is the activation in the system for the specific individual) (Borsboom (2008). Thereby, the present finding can support the identification of people in need of further support or at risk of a psychic breakdown at the assessment stage, while promoting a formulation-led diagnostic process that considers the interconnectedness of BPD-related difficulties. To achieve all this, the investigation of the idiosyncratic psychological networks of people who report BPD-related difficulties may be of clinical utility, especially when these are compared to the networks generated in the current study and in previous network researches on BPD (David, Marshall, Evanovich, & Mumma, 2018). Individual psychological networks would also allow for the inclusion of mentalisation and further important idiosyncratic intrapsychic or environmental factors that could maintain

the problems of SUs, instead of reducing human struggles to a pre-determined list of individual symptoms (Gunn & Potter, 2015). Furthermore, general differences in mentalising patterns between participants with and without BPD diagnosis may be of relevance clinically. In particular, specific differences in the relationships between ineffective modes of mentalisation and interpersonal difficulties may alert professionals who are in the process of assessing the severity of people's mental health problems.

However, this type of research is only one possible route towards a well-informed, evidence-based paradigm shift in the diagnosis of BPD. Further network studies using longitudinal and time-series research designs are recommended to investigate the dynamic mechanisms (i.e. critical slowing down etc.) that characterise the shifts during which 'healthier' states move toward more altered and 'disturbed' ones (Borsboom et al., 2021). These research designs also allow for more reliable causal inference (Forbes, Wright, Markon, & Krueger, 2017) and would therefore provide more detailed insight into the aetiology of BPD.

Expanding the theory of mentalisation. The finding that hypermentalising and hypomentalising were simultaneously associated with BPD-related difficulties was novel insight into how ineffective modes of mentalization relate and may induce specific BPD-related difficulties. So far, literature on whether hypomentalisation or hypermentalisation embodies the core difficulty in BPD has been divisive. This research stresses that they may both be present at the same time, depending on the level of emotional arousal, attachment style and epistemic vigilance that a specific interpersonal difficulty might activate. Further research using network theory should attempt to incorporate measurements of these underlying psychological mechanisms and/or investigate their mediating effects on the pairwise associations found in this research. This would assist therapeutic interventions by further mapping out when and under what circumstances mentalising activity is 'over' or 'under' activated.

At the same time, the lack of associations that were expected to be found in the clinical group also highlighted the difficulties in measuring BPD and ineffective mentalisation. The findings of this study support the notion that self-report measurements might not capture the full range of difficulties that people with BPD diagnosis struggle with and promote the employment of informant-ratings for future research. Similarly, the MASC lacks sensitivity to assess all domains of mentalisation (particularly self-mentalisation) and the hypomentalisation and no mentalisation subscales may have too much topological overlap, meaning they do not measure independent constructs. Therefore, the study highlights

the need for improved tools that cover and assess all domains of mentalisation and define the exact level of attachment-related stress induced by the administration of them. It also draws attention to the need for clarification and operationalization of specific modes of non-mentalizing (Duschinsky & Foster, 2021) required to create ‘a complete map of the non-mentalizing world’ that has long been missing (Duschinsky, Collver, & Carel, 2019).

Further rationale for MBT. Ineffective modes of mentalisation were found to escalate the presence of several BPD-related difficulties, and vice versa, in both groups, suggesting that enhancing and stabilising mentalisation (which is the main focus of MBT; Bateman & Fonagy, 2016) could activate significant positive changes in interpersonal problems in everyone. Yet, for people with a BPD diagnosis, whose system of difficulties is highly activated and potentially self-sustaining, improved mentalisation may have an even more important de-activating effect than for people whose level of system activation reduces quickly and automatically. Therefore, the findings add to the growing literature on the possible efficacy of MBT for reducing the struggles of people with a BPD diagnosis (Malda-Castillo et al., 2019; Vogt & Norman, 2019; Volkert et al., 2019). Therefore, they provide further rationale for the employment of MBT in services for people with complex mental health needs. Moreover, the present findings provide more specialised knowledge about how intervening on hypermentalising or hypomenthalising tendencies may affect specific BPD-related difficulties, which may otherwise reinforce ineffective mentalisation. Being aware of these feedback loops could be particularly important when providing MBT for people with a BPD diagnosis, as the quick-fire triggering of the attachment system and epistemic mistrust may lead to sudden and easily-provoked losses of mentalisation during the therapy sessions. These may catch clinicians off-guard, evoking a non-thinking, non-mentalising stance in them too (Bateman & Fonagy, 2016). Anticipating various ineffective modes of mentalisation can protect the clinician from the emotional toll of working with people with complex mental health issues (Treloar, 2009) and equip them to help the patient to maintain an optimal level of arousal in a safe relationship (Bateman & Fonagy, 2010).

Self-Reflections

It is important to commence my self-reflections with the choice of using pre-collected data from an existing research study, as research designs, data collection protocols and analytic methods often shape the epistemological and ontological frameworks of the research questions (Jayawickreme, Rasmussen, Karasz, Verkuilen, & Jayawickreme, 2019). On the one hand, conducting secondary data analysis granted me the opportunity of analysing a

dataset of a size that I would have never been able to collect on my own, due to time and financial restrictions. The availability of data has also allowed me the privilege of focusing on a novel and very sophisticated data analytic procedure, instead of spending a considerable amount of time and energy seeking out and recruiting people for my research. On the other hand, the decision to conduct a secondary data analysis determined the epistemological and ontological paradigm of the project. This meant that certain types of knowledge and conceptualization were examined at the expense of alternative views of the world and mental health difficulties. Our doctorate program required the completion of a research proposal and ethics application in the middle of our first year, at a time when I was still heavily influenced by the mainstream positivist psychological research paradigms (Arocha, 2020) and a desire to undertake “real science” that is true and objective, as opposed to seeing “truth” that is open to interpretations. However, throughout my years as a trainee I have become acquainted with several alternative epistemological frameworks that opened my eyes to different, less privileged assumptions about mental health. While my research offers several important clinical and theoretical implications, it is important to examine my own relationship with the concept of BPD and what might have remained hidden or constrained as a result of this study.

My relationship to the construction of mental health problems in medical discourse and as applied particularly to BPD has changed significantly over the years of conducting this research. It has become increasingly important for me to accentuate the phenomena, the various forms of distresses and the lived experiences of people, instead of focusing on a “psychiatric disease” or “disorder”. As a result, the current problems with the diagnosis of BPD became an important theme in my study. I also attempted to use language that decreased the view of BPD as an intrinsic, unchangeable and ultimate truth about an individual (e.g. referring to people as people with BPD diagnosis rather than people with BPD; Dyson & Gorvin, 2017). However, despite my best attempts, it was not always possible to avoid potentially unhelpful language (i.e.g ‘borderline personality disorder’ in itself still carries a huge adverse emotional connotation; Gunn & Potter, 2015). Similarly, even though I used the presence of a BPD diagnosis as an indicator for more severe difficulties, the differentiation and comparison of people based on the diagnosis might have also added to the stigmatizing of people with complex mental health problems. Additionally, the pre-determined measures that I used constrained certain knowledge from being examined (i.e. the influence of political drivers, social inequalities and power differences etc.). Thus, relying on a post-positivist framework might have led to the socially constructed elements of BPD remaining unaddressed and decontextualised by the cultural-historical environment.

Thereby, it is possible that my research perpetuates the long-standing moralistic, pathologizing, medicalising and stigmatising view of BPD, stripping the construct from context and meaning (Gunn & Potter, 2015). Had I used a different set of assumptions for understanding the emotional distress that is labelled as BPD, I might have emphasised dysfunctional family dynamics, unclear boundaries and interactional processes (Thompson, 2022), or broader societal issues such as sexism in the construction of the networks (Masland et al.). Furthermore, the presumption that the items of the PAI-BOR are experienced as difficulties (or in the medical world, symptoms) by the individual and not as strengths or assets in certain situations might be pathologizing. My awareness of these issues grew gradually while conducting this study. The goal to explore, which is present in the ambition of the research, has been echoed in myself as a constant subconscious reminder to remain curious regarding the context-dependent meaning of certain problems. While the network approach allowed for contextual variables (such as ineffective modes of mentalisation) to be investigated, it is still important to remember that various difficulties do not necessarily make up what it actually means to have certain emotional struggles labelled as disorder (Hens, Evers, & Wagemans, 2019).

Remaining true to the post-positivist paradigm also meant that I took a neutral stance in this research. However, it is still important to evaluate my own motivations to present interesting results, which eventually form publications. Even though I did everything I consciously could to remain as rigorous and meticulous throughout the research as possible, it is still possible that my own blind spots created unconscious biases that might have led to flaws and inaccuracies.

It is also important to reflect on my own experiences with the phenomenon we call BPD. During my training (and even pre-training) I have worked with several people who either had BPD diagnosis or had traits and interpersonal dynamics that resembled the intense emotional states associated with BPD. Listening to some of the horrific childhood experiences and relational traumas that people with this kind of emotional struggle were brave enough to share really moved me and transformed my professional stance. Having struggled with mental health difficulties myself, I have learned to appreciate more compassionate ways of understanding emotional distress. I view all SUs as human beings trying their best to live their lives despite the long-lasting effects of some terrible things that happened to them. Thus, while I am proud of the current research and the last three years that I have spent on completing it, in the future I am committed to conduct studies that are more humanistic and compassionate in their epistemological and ontological stance.

Conclusion

In summary this study has identified some similarities as well as distinct differences between participants with and without BPD diagnosis, suggesting that there is more complex relationship between mentalising difficulties and features associated with BPD than previously assumed. The structural differences found in the networks of the two groups confirm that more severe BPD- and mentalisation-related problems translate into more highly connected networks that are assumingly slower to be calmed down and restored. However, the importance of ineffective modes of mentalisation in the activation and maintenance of these networks was found to be contradictory. While centrality measures suggested low levels of impact, predictability measures confirmed relatively high practical relevance for ineffective modes of mentalisation in the networks. Furthermore, all the strongest and most accurate pairwise associations between mentalisation problems and BPD-related difficulties were of interpersonal nature, as predicted by the mentalisation model.

Ineffective modes of mentalisation were found to interact with interpersonal difficulties regardless of the presence or absence of BPD diagnosis, which may have important implication for future campaigns targeting destigmatisation of mental health problems in the general population. As expected, interactions between poor mentalisation and BPD-related problems were present in the clinical group too but these were more complex and, at times, counter-intuitive to previous accounts. The complexity may stem from potential effects of emotional arousal and epistemic trust on the pairwise interactions or from the limitations that the psychometric properties of the employed measurements imposed on the research. Yet, it is important to emphasise that while BPD features have generally been assumed to be linked to either hypermentalising or hypomenthalising, this study suggests that both of these modes of ineffective mentalisation might simultaneously be present. Clinical implications of the findings were highlighted particularly in relation to MBT.

Overall, the study proposed a more nuanced approach that tracked the particular ways in which mentalising capacity might be disrupted in the face of interpersonal problems. In order to deepen the understanding of some of the findings, different methodologies that are more sensitive to measure mentalising and BPD-associated problems are recommended to be employed in future studies.

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Appendix A

Personality Assessment Inventory - Borderline Personality Feature Scale (PAI-BOR)

PAI-BOR

This exercise contains a number of statements. Read each statement and decide if it is an accurate statement about you. Select whether the statement is FALSE (NOT AT ALL TRUE) or SLIGHTLY TRUE or MAINLY TRUE or VERY TRUE.

Work quickly and give your immediate answer, and try to answer every question. There are no right or wrong answers since this is simply a measure of the way you react.

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
1. My mood can shift quite suddenly.				
2. My attitude about myself changes a lot.				
3. My relationships have been stormy.				
4. My moods get quite intense.				
5. Sometimes I feel terribly empty inside.				
6. I want to let certain people know how much they've hurt me.				
7. My mood is very steady.				
8. I worry a lot about other people leaving me.				
9. People once close to me have let me down.				
10. I have little control over my anger.				
11. I often wonder what I should do with my life.				
12. I rarely feel very lonely.				

	False (0)	Slightly True (1)	Mainly True (2)	Very True (3)
13. I sometimes do things so impulsively that I get into trouble.				
14. I've always been a pretty happy person.				
15. I can't handle separation from those close to me very well.				
16. I've made some real mistakes in the people I've picked as friends.				
17. When I'm upset, I typically do something to hurt myself.				
18. I've had times when I was so mad I couldn't do enough to express all my anger.				
19. I don't get bored very easily.				
20. Once someone is my friend, we stay friends.				
21. I'm too impulsive for my own good.				
22. I spend money too easily.				
23. I'm a reckless person.				
24. I'm careful about how I spend my money.				

Appendix B

University of Essex Research Ethics Committee Approval



29/01/2021

Ms Lilla Asztalos

Health and Social Care

University of Essex

Dear Lilla,

Ethics Committee Decision

I am writing to advise you that your research proposal entitled "An Examination of the Concept of Borderline Psychopathology within the Mentalization Framework: A Network Approach" has been reviewed by the Ethics Sub Committee 1.

The Committee is content to give a favourable ethical opinion of the research. I am pleased, therefore, to tell you that your application has been granted ethical approval by the Committee.

Please do not hesitate to contact me if you require any further information or have any queries.

Yours sincerely,

Gill Green

Appendix C

Edge weights and moderation effects found in the whole sample

	Variable A ^a	Variable B ^b	Mean ^c	qtl_low ^d	qtl_high ^e	propLtZ ^f	VA ^s	VB ^b	Mod_Mean ⁱ	Mod_qtl_ low ^j	Mod_qtl_ _high ^k	Mod_propLtZ ^l
1	15	23	0.85	0.65	1.09	1	15	23	0	0	0	0
2	4	23	0.42	0.13	0.67	0.99	4	23	0	0	0	0
3	12	23	0.42	0.16	0.67	1	12	23	0	0	0	0
4	1	6	0.38	0.29	0.48	1	1	6	-0.02	-0.13	0	0.2
5	6	12	0.33	0.22	0.43	1	6	12	-0.13	-0.24	0	0.82
6	8	14	0.3	0.2	0.4	1	8	14	-0.02	-0.14	0	0.17
7	21	22	0.3	0.2	0.39	1	21	22	0.02	0	0.12	0.25
8	7	13	0.28	0.17	0.39	1	7	13	0.11	0	0.22	0.77
9	5	8	0.27	0.17	0.37	1	5	8	-0.04	-0.16	0	0.32
10	9	16	0.27	0.17	0.36	1	9	16	0.03	0	0.15	0.24
11	1	23	0.24	0	0.44	0.93	1	23	0	0	0	0
12	1	16	0.23	0.15	0.31	1	1	16	-0.01	-0.13	0	0.11
13	17	18	0.23	0.14	0.33	1	17	18	-0.02	-0.12	0	0.21
14	4	10	0.2	0.06	0.29	0.98	4	10	0.02	0	0.13	0.16
15	9	23	0.2	0	0.42	0.91	9	23	0	0	0	0
16	3	14	0.18	0.07	0.28	0.99	3	14	-0.08	-0.25	0	0.51
17	2	4	0.17	0.04	0.29	0.97	2	4	-0.05	-0.18	0	0.37
18	3	19	0.17	0.07	0.27	0.98	3	19	0	0	0	0.06
19	14	18	0.15	0.04	0.23	0.97	14	18	0	-0.05	0	0.08
20	1	4	0.14	0	0.29	0.83	1	4	-0.06	-0.19	0	0.44
21	2	19	0.14	0	0.26	0.93	2	19	-0.05	-0.17	0	0.44
22	3	20	0.14	0	0.25	0.9	3	20	-0.15	-0.26	0	0.89
23	4	7	0.14	0	0.25	0.94	4	7	-0.01	-0.1	0	0.14
24	2	3	0.13	0	0.22	0.89	2	3	0.02	0	0.11	0.16
25	3	23	0.13	0	0.35	0.81	3	23	0	0	0	0
26	4	15	0.13	0	0.3	0.79	4	15	-0.04	-0.23	0	0.23
27	1	3	0.12	0	0.23	0.87	1	3	0.01	0	0.12	0.12
28	4	12	0.12	0	0.25	0.8	4	12	-0.01	-0.1	0	0.11
29	6	11	0.12	0	0.22	0.92	6	11	-0.01	-0.08	0	0.09
30	7	9	0.12	0	0.26	0.82	7	9	-0.06	-0.2	0	0.44
31	7	23	0.12	0	0.33	0.74	7	23	0	0	0	0
32	1	2	0.11	0	0.22	0.87	1	2	0.01	0	0	0.06
33	7	11	0.11	0	0.21	0.92	7	11	0	0	0.07	0.1
34	17	19	0.11	0	0.22	0.92	17	19	-0.01	-0.08	0	0.08
35	20	22	0.11	0	0.21	0.9	20	22	0.02	0	0.12	0.24
36	4	11	0.1	0	0.22	0.88	4	11	-0.02	-0.12	0	0.16
37	7	8	0.1	0	0.2	0.78	7	8	0.02	0	0.11	0.21
38	12	18	0.1	0	0.21	0.81	12	18	-0.04	-0.13	0	0.34

Table C (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ	VA	VB	Mod_Mean	Mod_qtl_ low	Mod_qtl_ _high	Mod_propLtZ
39	5	14	0.09	0	0.22	0.76	5	14	-0.04	-0.17	0	0.29
40	8	16	0.09	0	0.19	0.8	8	16	0	0	0	0.07
41	10	19	0.09	0	0.19	0.87	10	19	0	0	0.06	0.08
42	13	23	0.09	0	0.31	0.68	13	23	0	0	0	0
43	2	10	0.08	0	0.2	0.71	2	10	-0.04	-0.15	0	0.35
44	5	13	0.08	0	0.17	0.74	5	13	0.01	0	0.08	0.15
45	7	20	0.08	0	0.19	0.67	7	20	0.01	0	0.12	0.08
46	3	5	0.07	0	0.17	0.73	3	5	0.02	0	0.1	0.21
47	6	19	0.07	0	0.19	0.6	6	19	-0.01	-0.11	0	0.11
48	8	10	0.07	0	0.18	0.73	8	10	-0.01	-0.09	0	0.12
49	9	19	0.07	0	0.17	0.68	9	19	0	0	0	0.06
50	11	18	0.07	0	0.19	0.64	11	18	-0.04	-0.14	0	0.36
51	13	21	0.07	0	0.18	0.68	13	21	-0.01	-0.1	0	0.1
52	19	23	0.07	0	0.22	0.7	19	23	0	0	0	0
53	2	17	0.06	0	0.16	0.74	2	17	0	0	0.05	0.08
54	4	5	0.06	0	0.17	0.6	4	5	-0.03	-0.14	0	0.23
55	5	9	0.06	0	0.15	0.73	5	9	0.02	0	0.11	0.24
56	5	21	0.06	0	0.17	0.56	5	21	-0.03	-0.13	0	0.28
57	6	17	0.06	0	0.17	0.66	6	17	-0.04	-0.15	0	0.41
58	13	14	0.06	0	0.16	0.6	13	14	0.03	0	0.12	0.29
59	14	15	0.06	0	0.17	0.69	14	15	0	0	0	0.02
60	16	23	0.06	0	0.23	0.56	16	23	0	0	0	0
61	1	13	0.05	0	0.17	0.54	1	13	0	-0.07	0	0.07
62	2	7	0.05	0	0.18	0.5	2	7	-0.02	-0.11	0	0.16
63	5	7	0.05	0	0.16	0.55	5	7	-0.01	-0.08	0	0.1
64	6	23	0.05	0	0.23	0.48	6	23	0	0	0	0
65	11	17	0.05	0	0.15	0.65	11	17	0.01	0	0.09	0.16
66	14	20	0.05	0	0.17	0.48	14	20	-0.03	-0.14	0	0.3
67	1	9	0.04	0	0.15	0.52	1	9	0	0	0	0.04
68	1	15	0.04	0	0.09	0.65	1	15	0.06	0	0.22	0.32
69	3	13	0.04	0	0.16	0.39	3	13	-0.01	-0.1	0	0.12
70	3	18	0.04	0	0.14	0.46	3	18	0	0	0	0.04
71	5	23	0.04	0	0.14	0.5	5	23	0	0	0	0
72	8	13	0.04	0	0.16	0.44	8	13	-0.02	-0.11	0	0.17
73	10	11	0.04	0	0.13	0.5	10	11	0.03	0	0.11	0.38
74	10	17	0.04	0	0.14	0.51	10	17	0	-0.04	0.04	0.11
75	16	18	0.04	0	0.14	0.47	16	18	0	-0.06	0	0.07
76	17	23	0.04	0	0.2	0.42	17	23	0	0	0	0
77	18	21	0.04	0	0.15	0.46	18	21	0	-0.07	0	0.08
78	1	7	0.03	0	0.13	0.36	1	7	0.02	0	0.12	0.16
79	1	8	0.03	0	0.16	0.32	1	8	-0.03	-0.14	0	0.24
80	2	6	0.03	0	0.13	0.34	2	6	0	0	0	0.05

Table C (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ	VA	VB	Mod_Mean	Mod_qtl_ low	Mod_qtl_ _high	Mod_propLtZ
81	3	8	0.03	0	0.14	0.38	3	8	0.05	0	0.14	0.47
82	3	9	0.03	0	0.13	0.4	3	9	0	0	0	0.06
83	4	22	0.03	0	0.14	0.38	4	22	-0.03	-0.16	0	0.26
84	6	22	0.03	0	0.13	0.36	6	22	-0.01	-0.11	0	0.14
85	9	21	0.03	0	0.11	0.4	9	21	0.01	0	0.09	0.17
86	10	12	0.03	0	0.13	0.34	10	12	0	0	0	0.06
87	10	18	0.03	0	0.12	0.43	10	18	0.02	0	0.11	0.22
88	11	21	0.03	0	0.13	0.34	11	21	-0.01	-0.09	0	0.12
89	13	15	0.03	0	0.12	0.32	13	15	-0.01	0	0	0.03
90	14	19	0.03	0	0.13	0.33	14	19	0.1	0	0.19	0.83
91	14	21	0.03	0	0.14	0.28	14	21	-0.03	-0.12	0	0.3
92	14	22	0.03	0	0.13	0.44	14	22	0	0	0	0.05
93	18	23	0.03	0	0.13	0.33	18	23	0	0	0	0
94	2	11	0.02	0	0.09	0.28	2	11	0.01	0	0.07	0.11
95	4	17	0.02	0	0.09	0.27	4	17	0	0	0	0.05
96	5	16	0.02	0	0.1	0.34	5	16	0	0	0.06	0.09
97	5	20	0.02	0	0.11	0.23	5	20	0.02	0	0.12	0.22
98	6	9	0.02	0	0.11	0.26	6	9	0	0	0	0.06
99	6	14	0.02	0	0.14	0.26	6	14	-0.01	-0.11	0	0.11
100	7	19	0.02	0	0.11	0.21	7	19	-0.03	-0.13	0	0.31
101	8	12	0.02	0	0.12	0.19	8	12	0.01	0	0.1	0.13
102	9	14	0.02	0	0.12	0.3	9	14	0	0	0	0.05
103	10	15	0.02	0	0.07	0.31	10	15	0	0	0	0.05
104	11	12	0.02	0	0.11	0.43	11	12	0	0	0.05	0.07
105	11	13	0.02	0	0.1	0.3	11	13	0.01	0	0.09	0.19
106	11	23	0.02	0	0.16	0.29	11	23	0	0	0	0
107	14	16	0.02	0	0.11	0.35	14	16	0	0	0	0.05
108	15	20	0.02	0	0.1	0.24	15	20	0	0	0	0
109	16	20	0.02	0	0.12	0.29	16	20	0.02	0	0.11	0.14
110	1	20	0.01	0	0.07	0.1	1	20	-0.01	-0.13	0	0.09
111	2	5	0.01	0	0.08	0.14	2	5	0.01	0	0.07	0.12
112	2	9	0.01	0	0.08	0.18	2	9	0.01	0	0.07	0.12
113	2	15	0.01	0	0.06	0.26	2	15	0.01	0	0.08	0.07
114	2	16	0.01	0	0.09	0.23	2	16	0.01	0	0.07	0.13
115	3	15	0.01	0	0.1	0.17	3	15	-0.07	-0.2	0	0.58
116	3	16	0.01	0	0.1	0.22	3	16	0	0	0	0.06
117	4	6	0.01	0	0.06	0.12	4	6	0	0	0.07	0.08
118	4	16	0.01	0	0.05	0.13	4	16	0	0	0	0.04
119	5	12	0.01	0	0.09	0.11	5	12	-0.01	-0.1	0	0.1
120	5	18	0.01	0	0.08	0.16	5	18	-0.02	-0.11	0	0.27
121	6	13	0.01	0	0.1	0.12	6	13	-0.04	-0.16	0	0.32
122	7	15	0.01	0	0.07	0.21	7	15	0	0	0	0.05

Table C (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ	VA	VB	Mod_Mean	Mod_qtl_ low	Mod_qtl_ _high	Mod_propLtZ
123	8	11	0.01	0	0.1	0.21	8	11	-0.02	-0.11	0	0.18
124	8	18	0.01	0	0.09	0.21	8	18	0.05	0	0.16	0.44
125	8	21	0.01	0	0.05	0.16	8	21	0.01	0	0.08	0.07
126	9	10	0.01	0	0.06	0.13	9	10	0	0	0	0.05
127	9	12	0.01	0	0.08	0.1	9	12	-0.03	-0.13	0	0.24
128	10	22	0.01	0	0.09	0.25	10	22	0.01	0	0.09	0.19
129	11	15	0.01	0	0.06	0.27	11	15	0	0	0	0.04
130	11	19	0.01	0	0.07	0.13	11	19	-0.03	-0.12	0	0.3
131	11	22	0.01	0	0.1	0.21	11	22	-0.08	-0.25	0	0.6
132	12	17	0.01	0	0.04	0.15	12	17	0	0	0	0.02
133	12	21	0.01	0	0.07	0.13	12	21	-0.03	-0.14	0	0.26
134	13	16	0.01	0	0.07	0.13	13	16	0.01	0	0.08	0.13
135	13	19	0.01	0	0.08	0.12	13	19	0.01	0	0.07	0.08
136	15	16	0.01	0	0.05	0.2	15	16	0.01	0	0.07	0.07
137	15	17	0.01	0	0.06	0.32	15	17	0	0	0	0.04
138	15	22	0.01	0	0.07	0.27	15	22	0	0	0	0.01
139	16	19	0.01	0	0.09	0.22	16	19	0.01	0	0.08	0.15
140	17	21	0.01	0	0.1	0.18	17	21	-0.02	-0.11	0	0.24
141	18	20	0.01	0	0.09	0.17	18	20	0.03	0	0.12	0.37
142	19	20	0.01	0	0.09	0.17	19	20	0.02	0	0.11	0.25
143	22	23	0.01	0	0.08	0.17	22	23	0	0	0	0
144	1	5	0	0	0.03	0.09	1	5	0	0	0	0.05
145	1	10	0	-0.03	0.03	0.11	1	10	0	0	0	0.05
146	1	14	0	0	0	0.06	1	14	-0.01	-0.11	0	0.12
147	1	19	0	-0.05	0.05	0.21	1	19	0.05	0	0.18	0.4
148	1	21	0	-0.06	0.04	0.13	1	21	0.03	0	0.17	0.19
149	2	22	0	0	0.02	0.08	2	22	0	0	0	0.06
150	3	4	0	-0.05	0.05	0.15	3	4	0.01	0	0.11	0.12
151	3	6	0	0	0	0.05	3	6	-0.02	-0.11	0	0.22
152	3	10	0	0	0.05	0.13	3	10	0	0	0	0.06
153	3	11	0	-0.05	0	0.12	3	11	0.01	0	0.07	0.08
154	3	12	0	0	0.04	0.06	3	12	-0.08	-0.21	0	0.65
155	3	21	0	0	0.06	0.1	3	21	-0.04	-0.15	0	0.38
156	3	22	0	0	0.04	0.09	3	22	0	0	0	0.05
157	4	8	0	0	0.05	0.08	4	8	0	0	0	0.07
158	4	9	0	0	0	0.06	4	9	0.01	0	0.1	0.09
159	4	14	0	0	0.03	0.08	4	14	0.01	0	0.09	0.08
160	4	19	0	0	0	0.06	4	19	0	0	0	0.02
161	4	20	0	0	0.05	0.1	4	20	0	0	0	0.03
162	4	21	0	-0.05	0	0.1	4	21	0.02	0	0.14	0.11
163	5	10	0	0	0.03	0.12	5	10	0.01	0	0.06	0.08
164	5	11	0	-0.04	0	0.09	5	11	0.01	0	0.07	0.13

Table C (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ	VA	VB	Mod_Mean	Mod_qtl_ low	Mod_qtl_ _high	Mod_propLtZ
165	5	15	0	0	0	0.03	5	15	0	0	0	0.02
166	5	19	0	0	0	0.06	5	19	-0.02	-0.09	0	0.29
167	6	15	0	0	0	0.06	6	15	0	0	0	0
168	6	18	0	-0.03	0	0.08	6	18	0.01	0	0.08	0.07
169	6	20	0	-0.04	0	0.06	6	20	0	0	0	0.03
170	7	10	0	0	0.03	0.1	7	10	0.01	0	0.06	0.08
171	7	12	0	-0.04	0	0.07	7	12	0	0	0	0.04
172	7	22	0	0	0	0.06	7	22	-0.01	-0.07	0	0.07
173	8	9	0	0	0	0.07	8	9	0	0	0	0.05
174	8	17	0	0	0.03	0.09	8	17	0	0	0	0.05
175	8	19	0	0	0.05	0.08	8	19	-0.03	-0.12	0	0.29
176	8	22	0	0	0	0.07	8	22	0.02	0	0.11	0.14
177	8	23	0	-0.08	0.1	0.24	8	23	0	0	0	0
178	9	18	0	0	0	0.07	9	18	-0.01	-0.08	0	0.12
179	9	20	0	0	0	0.06	9	20	0.02	0	0.11	0.16
180	10	16	0	0	0.05	0.15	10	16	0.01	0	0.06	0.09
181	11	14	0	0	0	0.07	11	14	0	-0.06	0	0.08
182	11	16	0	-0.04	0.03	0.13	11	16	-0.05	-0.12	0	0.54
183	11	20	0	0	0.03	0.09	11	20	0.01	0	0.08	0.11
184	12	13	0	0	0	0.06	12	13	-0.01	-0.07	0	0.07
185	12	14	0	0	0.03	0.06	12	14	-0.01	-0.11	0	0.13
186	12	15	0	0	0.05	0.11	12	15	0	0	0	0.03
187	12	16	0	0	0.05	0.11	12	16	0.02	0	0.11	0.18
188	12	20	0	0	0	0.04	12	20	-0.01	-0.07	0	0.05
189	12	22	0	-0.03	0	0.07	12	22	0	0	0	0.02
190	13	17	0	-0.04	0	0.1	13	17	0.01	0	0.08	0.12
191	14	17	0	-0.04	0	0.1	14	17	-0.02	-0.09	0	0.21
192	15	18	0	0	0.04	0.06	15	18	0	0	0	0.02
193	16	17	0	0	0.04	0.09	16	17	-0.01	-0.08	0	0.17
194	16	22	0	0	0	0.05	16	22	0	0	0	0.02
195	17	22	0	-0.03	0	0.08	17	22	0	-0.04	0	0.07
196	19	22	0	0	0	0.06	19	22	0	0	0.04	0.06
197	21	23	0	-0.08	0.06	0.21	21	23	0	0	0	0
198	1	17	-0.01	-0.06	0	0.15	1	17	0	0	0	0.03
199	1	22	-0.01	-0.05	0	0.11	1	22	0.01	0	0.05	0.05
200	2	8	-0.01	-0.08	0	0.14	2	8	0	-0.07	0	0.08
201	2	14	-0.01	-0.07	0	0.15	2	14	0.01	0	0.07	0.1
202	2	18	-0.01	-0.07	0	0.15	2	18	0	-0.05	0	0.09
203	2	23	-0.01	-0.09	0.04	0.24	2	23	0	0	0	0
204	3	17	-0.01	-0.06	0	0.13	3	17	0	0	0	0.05
205	4	18	-0.01	-0.06	0	0.14	4	18	0	0	0	0.03
206	5	17	-0.01	-0.08	0	0.17	5	17	-0.01	-0.06	0	0.13

Table C (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ	VA	VB	Mod_Mean	Mod_qtl_ low	Mod_qtl_ _high	Mod_propLtZ
207	5	22	-0.01	-0.08	0	0.19	5	22	-0.01	-0.07	0	0.13
208	6	7	-0.01	-0.06	0	0.11	6	7	-0.01	-0.09	0	0.18
209	6	10	-0.01	-0.06	0	0.13	6	10	0.01	0	0.1	0.12
210	6	16	-0.01	-0.08	0	0.15	6	16	0	0	0	0.06
211	7	17	-0.01	-0.05	0	0.1	7	17	0.01	0	0.08	0.08
212	7	21	-0.01	-0.09	0	0.19	7	21	-0.01	-0.09	0	0.12
213	9	11	-0.01	-0.07	0	0.1	9	11	0.01	0	0.07	0.09
214	9	15	-0.01	-0.06	0	0.12	9	15	0	0	0	0.01
215	9	17	-0.01	-0.06	0	0.12	9	17	0.02	0	0.11	0.26
216	9	22	-0.01	-0.07	0	0.17	9	22	0	0	0	0.05
217	10	13	-0.01	-0.06	0	0.12	10	13	0.01	0	0.11	0.16
218	10	14	-0.01	-0.09	0	0.17	10	14	-0.01	-0.07	0	0.13
219	10	21	-0.01	-0.09	0	0.18	10	21	-0.01	-0.07	0	0.09
220	12	19	-0.01	-0.1	0	0.11	12	19	0.01	0	0.11	0.1
221	13	20	-0.01	-0.08	0	0.12	13	20	0	0	0	0.06
222	15	19	-0.01	-0.1	0	0.17	15	19	0.08	0	0.18	0.61
223	15	21	-0.01	-0.06	0	0.14	15	21	0	0	0	0.02
224	16	21	-0.01	-0.08	0	0.16	16	21	0	0	0	0.03
225	19	21	-0.01	-0.07	0	0.13	19	21	0.01	0	0.09	0.1
226	1	11	-0.02	-0.14	0	0.26	1	11	0.02	0	0.14	0.2
227	1	18	-0.02	-0.1	0	0.23	1	18	0	0	0	0.04
228	2	12	-0.02	-0.12	0	0.22	2	12	-0.01	-0.09	0	0.13
229	2	13	-0.02	-0.11	0	0.28	2	13	-0.01	-0.06	0	0.09
230	2	20	-0.02	-0.11	0	0.19	2	20	0.02	0	0.11	0.18
231	3	7	-0.02	-0.13	0	0.22	3	7	0.04	0	0.15	0.39
232	6	8	-0.02	-0.13	0	0.16	6	8	0.01	0	0.11	0.13
233	6	21	-0.02	-0.11	0	0.3	6	21	-0.03	-0.14	0	0.25
234	8	15	-0.02	-0.14	0	0.18	8	15	0.02	0	0.14	0.18
235	10	23	-0.02	-0.14	0	0.29	10	23	0	0	0	0
236	13	22	-0.02	-0.11	0	0.27	13	22	0.01	0	0.08	0.08
237	17	20	-0.02	-0.11	0	0.28	17	20	0	-0.06	0	0.09
238	18	22	-0.02	-0.11	0	0.28	18	22	-0.01	-0.06	0	0.12
239	20	21	-0.02	-0.12	0	0.28	20	21	-0.07	-0.17	0	0.59
240	4	13	-0.03	-0.15	0	0.38	4	13	0	0	0	0.06
241	7	14	-0.03	-0.14	0	0.3	7	14	0	0	0.07	0.08
242	7	16	-0.03	-0.13	0	0.32	7	16	0	-0.06	0	0.09
243	7	18	-0.03	-0.12	0	0.44	7	18	0	0	0	0.03
244	9	13	-0.03	-0.13	0	0.33	9	13	0.01	0	0.09	0.08
245	10	20	-0.03	-0.13	0	0.43	10	20	-0.02	-0.11	0	0.15
246	14	23	-0.03	-0.17	0	0.33	14	23	0	0	0	0
247	18	19	-0.03	-0.12	0	0.42	18	19	0.01	0	0.07	0.07
248	1	12	-0.04	-0.17	0	0.33	1	12	0.08	0	0.21	0.56

Table C (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ	VA	VB	Mod_Mean	Mod_qtl _low	Mod_qtl _high	Mod_propLtZ
249	2	21	-0.04	-0.16	0	0.42	2	21	0.05	0	0.16	0.44
250	13	18	-0.05	-0.17	0	0.49	13	18	0.02	0	0.14	0.19
251	5	6	-0.07	-0.18	0	0.63	5	6	0.06	0	0.18	0.47
252	20	23	-0.07	-0.21	0	0.56	20	23	0	0	0	0
253	8	20	-0.21	-0.32	-0.07	0.99	8	20	0.16	0	0.3	0.92

Note. The pairwise effects provide information about the stability of the findings across bootstrap estimations, whilst the moderation effects illustrate the effect of borderline personality disorder diagnosis on the pairwise correlations.

Legend. node 1= intense mood shifts, node 2= attitude about self changes, node 3=stormy relationships, node 4=chronic emptiness, node 5=let people know they hurt me, node 6= unsteady mood, node 7=worry about people leaving, node 8= 'people let me down', node 9= little control over anger, node 10= wonder about life, node 11= feeling lonely, node 12= feeling unhappy, node 13= cannot handle separation, node 14= mistakes in picking friends, node 15= hurt self when upset, node 16= cannot express all of anger, node 17= gets bored easily, node 18= difficulty with staying friends with people , node 19= impulsivity and recklessness, node 20= MASCexc (hypermentalisation), node 21= MASCless (hypomentalisation), node 22= MASCno (no mentalisation), node 23=borderline personality disorder diagnosis.

^aVariable A= node A; ^b Variable B= node B; ^c Mean= mean edge weight across 1000 bootstrap estimations; ^{d+e} qtl_low and qtl_high= 95% quantiles of edge weights, containing 95% of the observations across 1000 bootstrap sampling (translatable to 95% confidence intervals); ^f propLtZ= the probability for the edge to be 0 or proportion of edge weight estimates that were non-zero presented in decimals, translatable to percentage (for regularised network models, it should not be interpreted as a hypothesis test); ^g VA= variable (node) A; ^h VB= variable (node) B; ⁱ Mod_Mean=mean moderation effect of the moderator variable across 1000 bootstrap estimations; ^{j+k} Mod_qtl_low and Mod_qtl_high= 95% quantiles of moderation effects containing 95% of the observations across 1000 bootstrap sampling (translatable to 95% confidence intervals); ^lMod_propLtZ= the probability for the moderation effect to be 0 or proportion of moderation effect estimates that were non-zero (for regularised network models, it should not be interpreted as a hypothesis test).

Appendix D

Edge weight estimations in the community group

	Variable A ^a	Variable B ^b	Mean ^c	qtl_low ^d	qtl_high ^e	propLtZ ^f
1	1	6	0.37	0.27	0.46	1
2	6	12	0.34	0.22	0.45	1
3	8	14	0.3	0.2	0.41	1
4	5	8	0.27	0.17	0.38	1
5	21	22	0.27	0.16	0.38	1
6	7	13	0.25	0.12	0.37	1
7	17	18	0.25	0.14	0.36	1
8	9	16	0.24	0.11	0.35	1
9	1	16	0.23	0.14	0.34	1
10	3	14	0.21	0.1	0.33	1
11	2	4	0.2	0.07	0.32	0.98
12	2	19	0.2	0.08	0.32	0.99
13	4	15	0.18	0.04	0.3	0.98
14	3	19	0.17	0.02	0.29	0.95
15	3	20	0.17	0.05	0.27	0.99
16	7	9	0.16	0.03	0.3	0.96
17	1	4	0.14	0	0.28	0.87
18	4	7	0.14	0	0.26	0.91
19	4	10	0.14	0	0.27	0.94
20	14	18	0.14	0	0.25	0.94
21	5	14	0.13	0	0.25	0.86
22	2	10	0.12	0	0.23	0.89
23	4	11	0.12	0	0.23	0.9
24	4	12	0.12	0	0.25	0.85
25	12	18	0.12	0	0.22	0.91
26	17	19	0.12	0	0.25	0.88
27	1	2	0.11	0	0.23	0.83
28	2	3	0.11	0	0.24	0.82
29	6	11	0.11	0	0.23	0.89
30	7	11	0.1	0	0.21	0.84
31	9	19	0.1	0	0.22	0.78
32	11	18	0.1	0	0.22	0.82
33	14	15	0.1	0	0.24	0.8
34	1	3	0.09	0	0.22	0.76
35	6	19	0.09	0	0.21	0.74
36	7	20	0.09	0	0.21	0.73
37	8	10	0.09	0	0.19	0.8
38	8	16	0.09	0	0.2	0.75

Table D (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
39	13	21	0.09	0	0.2	0.82
40	4	5	0.08	0	0.19	0.75
41	5	21	0.08	0	0.19	0.75
42	6	17	0.08	0	0.19	0.8
43	7	8	0.08	0	0.21	0.7
44	8	13	0.08	0	0.2	0.69
45	10	19	0.08	0	0.2	0.77
46	14	20	0.08	0	0.19	0.71
47	20	22	0.08	0	0.19	0.77
48	2	7	0.07	0	0.19	0.68
49	5	7	0.07	0	0.19	0.66
50	5	13	0.07	0	0.19	0.61
51	1	13	0.06	0	0.18	0.59
52	2	17	0.06	0	0.17	0.61
53	3	13	0.06	0	0.2	0.57
54	4	22	0.06	0	0.17	0.63
55	16	18	0.06	0	0.16	0.59
56	1	9	0.05	0	0.17	0.56
57	3	5	0.05	0	0.16	0.54
58	3	15	0.05	0	0.17	0.56
59	6	22	0.05	0	0.15	0.54
60	9	14	0.05	0	0.16	0.53
61	13	15	0.05	0	0.19	0.53
62	14	22	0.05	0	0.15	0.52
63	15	20	0.05	0	0.15	0.65
64	18	21	0.05	0	0.16	0.55
65	1	8	0.04	0	0.16	0.42
66	3	9	0.04	0	0.15	0.47
67	3	18	0.04	0	0.16	0.5
68	5	9	0.04	0	0.14	0.45
69	6	14	0.04	0	0.14	0.41
70	7	19	0.04	0	0.15	0.44
71	10	12	0.04	0	0.15	0.51
72	10	15	0.04	0	0.15	0.53
73	10	17	0.04	0	0.15	0.49
74	11	21	0.04	0	0.14	0.52
75	13	14	0.04	0	0.15	0.48
76	14	16	0.04	0	0.14	0.45
77	14	21	0.04	0	0.16	0.49
78	2	6	0.03	0	0.13	0.41
79	5	16	0.03	0	0.13	0.39

Table D (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
80	6	9	0.03	0	0.13	0.38
81	6	13	0.03	0	0.12	0.34
82	8	11	0.03	0	0.13	0.41
83	9	21	0.03	0	0.12	0.4
84	11	12	0.03	0	0.13	0.41
85	11	17	0.03	0	0.15	0.45
86	11	22	0.03	0	0.12	0.4
87	16	20	0.03	0	0.12	0.35
88	17	21	0.03	0	0.13	0.39
89	1	7	0.02	0	0.13	0.3
90	1	15	0.02	0	0.12	0.31
91	2	11	0.02	0	0.11	0.28
92	2	15	0.02	0	0.12	0.34
93	2	16	0.02	0	0.11	0.29
94	3	8	0.02	0	0.12	0.3
95	3	16	0.02	0	0.12	0.33
96	4	16	0.02	0	0.11	0.27
97	4	17	0.02	0	0.12	0.33
98	5	12	0.02	0	0.11	0.26
99	5	18	0.02	-0.03	0.13	0.32
100	7	15	0.02	-0.03	0.14	0.35
101	8	12	0.02	0	0.11	0.24
102	9	10	0.02	0	0.11	0.29
103	9	12	0.02	0	0.12	0.31
104	10	11	0.02	0	0.11	0.33
105	10	18	0.02	0	0.12	0.36
106	11	13	0.02	0	0.11	0.29
107	11	19	0.02	0	0.13	0.35
108	12	21	0.02	0	0.1	0.28
109	14	19	0.02	0	0.11	0.25
110	15	18	0.02	0	0.1	0.29
111	16	17	0.02	0	0.11	0.31
112	1	20	0.01	0	0.11	0.26
113	2	5	0.01	0	0.09	0.2
114	2	9	0.01	-0.04	0.1	0.24
115	3	10	0.01	-0.05	0.09	0.24
116	3	12	0.01	0	0.08	0.17
117	3	21	0.01	0	0.09	0.24
118	3	22	0.01	0	0.1	0.24
119	4	6	0.01	-0.04	0.1	0.23
120	4	8	0.01	0	0.08	0.19

Table D (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
121	5	19	0.01	0	0.08	0.18
122	5	20	0.01	0	0.1	0.23
123	6	15	0.01	0	0.08	0.21
124	8	19	0.01	0	0.09	0.25
125	9	18	0.01	0	0.1	0.24
126	10	22	0.01	0	0.08	0.23
127	11	15	0.01	0	0.09	0.22
128	11	16	0.01	-0.03	0.09	0.22
129	12	14	0.01	0	0.09	0.18
130	12	17	0.01	0	0.09	0.21
131	13	16	0.01	-0.03	0.09	0.24
132	13	19	0.01	-0.03	0.1	0.25
133	15	17	0.01	0	0.09	0.22
134	15	22	0.01	-0.04	0.1	0.28
135	16	19	0.01	-0.03	0.09	0.25
136	18	20	0.01	-0.05	0.09	0.22
137	19	20	0.01	0	0.1	0.24
138	1	5	0	-0.04	0.05	0.16
139	1	10	0	-0.03	0.06	0.17
140	1	14	0	-0.04	0.05	0.15
141	2	22	0	-0.05	0.05	0.2
142	3	4	0	-0.08	0.05	0.23
143	3	6	0	0	0.04	0.14
144	4	14	0	-0.07	0.04	0.18
145	4	19	0	-0.05	0.07	0.19
146	4	20	0	-0.04	0.08	0.19
147	4	21	0	-0.06	0.04	0.2
148	5	10	0	-0.08	0.04	0.22
149	5	15	0	-0.03	0.06	0.16
150	7	10	0	-0.06	0.06	0.19
151	7	22	0	-0.03	0.08	0.19
152	8	9	0	-0.05	0.03	0.2
153	8	17	0	-0.03	0.03	0.14
154	8	18	0	-0.05	0.08	0.23
155	8	21	0	-0.04	0.05	0.2
156	8	22	0	-0.06	0.03	0.16
157	10	16	0	-0.04	0.07	0.2
158	11	14	0	-0.04	0.07	0.22
159	11	20	0	-0.07	0.07	0.23
160	12	15	0	-0.09	0.09	0.28
161	12	16	0	-0.04	0.07	0.18

Table D (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
162	12	20	0	-0.02	0.04	0.14
163	14	17	0	-0.06	0.03	0.16
164	15	16	0	-0.05	0.07	0.22
165	16	22	0	0	0.05	0.14
166	17	22	0	-0.07	0.04	0.21
167	19	22	0	-0.04	0.03	0.16
168	1	19	-0.01	-0.09	0	0.21
169	1	21	-0.01	-0.09	0	0.28
170	2	8	-0.01	-0.1	0.03	0.23
171	2	18	-0.01	-0.09	0.03	0.26
172	3	11	-0.01	-0.1	0.02	0.25
173	4	9	-0.01	-0.09	0	0.19
174	5	11	-0.01	-0.1	0	0.24
175	5	17	-0.01	-0.09	0	0.2
176	5	22	-0.01	-0.1	0	0.25
177	6	7	-0.01	-0.09	0.03	0.21
178	6	10	-0.01	-0.1	0	0.25
179	6	18	-0.01	-0.1	0	0.23
180	6	20	-0.01	-0.07	0	0.17
181	7	12	-0.01	-0.1	0	0.25
182	7	17	-0.01	-0.1	0	0.24
183	7	21	-0.01	-0.09	0	0.23
184	9	20	-0.01	-0.09	0	0.18
185	10	21	-0.01	-0.08	0.02	0.22
186	12	13	-0.01	-0.09	0.03	0.2
187	12	22	-0.01	-0.08	0	0.19
188	13	17	-0.01	-0.1	0	0.23
189	15	21	-0.01	-0.08	0	0.21
190	1	17	-0.02	-0.11	0	0.24
191	1	22	-0.02	-0.11	0	0.26
192	2	12	-0.02	-0.12	0	0.28
193	2	14	-0.02	-0.12	0	0.31
194	3	17	-0.02	-0.11	0	0.26
195	4	18	-0.02	-0.13	0	0.27
196	6	16	-0.02	-0.12	0	0.3
197	6	21	-0.02	-0.11	0	0.25
198	9	11	-0.02	-0.12	0	0.34
199	10	13	-0.02	-0.12	0	0.32
200	10	14	-0.02	-0.11	0	0.28
201	12	19	-0.02	-0.13	0	0.32
202	13	20	-0.02	-0.12	0	0.27

Table D (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
203	16	21	-0.02	-0.1	0	0.27
204	19	21	-0.02	-0.12	0	0.33
205	20	21	-0.02	-0.1	0	0.24
206	1	18	-0.03	-0.13	0	0.35
207	2	13	-0.03	-0.14	0	0.37
208	6	8	-0.03	-0.14	0	0.31
209	8	15	-0.03	-0.15	0	0.37
210	9	17	-0.03	-0.13	0	0.38
211	9	22	-0.03	-0.14	0	0.4
212	10	20	-0.03	-0.13	0	0.43
213	17	20	-0.03	-0.12	0	0.37
214	18	22	-0.03	-0.14	0	0.39
215	1	11	-0.04	-0.16	0	0.42
216	2	20	-0.04	-0.15	0	0.42
217	3	7	-0.04	-0.16	0	0.42
218	7	14	-0.04	-0.17	0	0.45
219	7	16	-0.04	-0.16	0	0.44
220	7	18	-0.04	-0.12	0	0.43
221	1	12	-0.05	-0.18	0	0.51
222	13	22	-0.05	-0.16	0	0.61
223	15	19	-0.05	-0.17	0	0.5
224	2	21	-0.06	-0.16	0	0.68
225	4	13	-0.06	-0.19	0	0.58
226	9	13	-0.06	-0.17	0	0.62
227	9	15	-0.06	-0.16	0	0.62
228	18	19	-0.06	-0.16	0	0.64
229	13	18	-0.09	-0.22	0	0.76
230	5	6	-0.1	-0.21	0	0.83
231	8	20	-0.22	-0.32	-0.11	1

Note.

Legend. node 1= intense mood shifts, node 2= attitude about self changes, node 3= stormy relationships, node 4= chronic emptiness, node 5= let people know they hurt me, node 6= unsteady mood, node 7= worry about people leaving, node 8= 'people let me down', node 9= little control over anger, node 10= wonder about life, node 11= feeling lonely, node 12= feeling unhappy, node 13= cannot handle separation, node 14= mistakes in picking friends, node 15= hurt self when upset, node 16= cannot express all of anger, node 17= gets bored easily, node 18= difficulty with staying friends with people, node 19= impulsivity and recklessness, node 20= MASCexc (hypermentalisation), node 21= MASCless (hypomentalisation), node 22= MASCno (no mentalisation).

^aVariable A= node A; ^bVariable B= node B; ^cMean= mean edge weight across 1000 bootstrap estimations; ^{d+e}qtl_low and qtl_high= 95% quantiles of edge weights, containing 95% of the observations across 1000 bootstrap sampling (translatable to 95% confidence intervals); ^fpropLtZ= the probability for the edge to be 0 or proportion of edge weight estimates that were non-zero presented in decimals, translatable to percentage (for regularised network models, it should not be interpreted as a hypothesis test)

Appendix E

Edge weight estimations in the clinical group

	Variable A ^a	Variable B ^b	Mean ^c	qtl_low ^d	qtl_high ^e	propLtZ ^f
1	7	13	0.47	0.38	0.55	1
2	9	16	0.38	0.29	0.46	1
3	21	22	0.35	0.26	0.43	1
4	1	6	0.27	0.15	0.39	1
5	4	10	0.24	0.12	0.34	1
6	1	15	0.23	0.14	0.33	1
7	14	19	0.19	0.1	0.28	1
8	1	3	0.17	0.06	0.27	0.97
9	2	3	0.17	0.08	0.26	1
10	3	19	0.17	0.08	0.26	0.99
11	8	14	0.17	0.08	0.26	1
12	7	8	0.16	0.07	0.25	0.99
13	20	22	0.16	0.07	0.25	0.99
14	3	8	0.15	0.05	0.25	0.98
15	5	8	0.15	0.04	0.24	0.97
16	1	2	0.14	0.04	0.23	0.98
17	1	16	0.14	0.03	0.24	0.96
18	1	19	0.14	0.04	0.22	0.98
19	15	19	0.14	0.03	0.22	0.96
20	7	20	0.13	0.03	0.22	0.97
21	8	18	0.13	0.04	0.22	0.98
22	17	18	0.13	0.03	0.22	0.98
23	3	5	0.12	0	0.21	0.94
24	5	9	0.12	0.02	0.21	0.97
25	7	11	0.12	0	0.22	0.94
26	10	11	0.11	0	0.2	0.94
27	13	14	0.11	0	0.2	0.92
28	14	18	0.11	0	0.2	0.93
29	1	7	0.1	0	0.2	0.87
30	5	13	0.1	0	0.19	0.92
31	10	19	0.1	0	0.19	0.88
32	1	9	0.09	0	0.18	0.87
33	1	12	0.09	0	0.18	0.9
34	5	20	0.09	0	0.18	0.87
35	8	16	0.08	0	0.17	0.82
36	10	18	0.08	0	0.18	0.85
37	16	20	0.08	0	0.17	0.81
38	19	20	0.08	0	0.16	0.83

Table E (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
39	4	12	0.07	0	0.18	0.78
40	6	11	0.07	0	0.17	0.73
41	11	17	0.07	0	0.16	0.77
42	14	15	0.07	0	0.17	0.73
43	17	19	0.07	0	0.17	0.77
44	18	20	0.07	0	0.17	0.8
45	1	21	0.06	0	0.16	0.68
46	2	15	0.06	0	0.15	0.7
47	2	17	0.06	0	0.14	0.78
48	4	7	0.06	0	0.17	0.72
49	8	12	0.06	0	0.16	0.71
50	9	21	0.06	0	0.15	0.68
51	3	7	0.05	0	0.14	0.64
52	3	9	0.05	0	0.13	0.63
53	3	14	0.05	0	0.14	0.63
54	4	21	0.05	0	0.14	0.6
55	6	12	0.05	0	0.14	0.68
56	8	15	0.05	0	0.14	0.62
57	10	15	0.05	0	0.15	0.63
58	10	22	0.05	0	0.13	0.7
59	11	13	0.05	0	0.15	0.65
60	15	17	0.05	0	0.14	0.64
61	16	19	0.05	0	0.14	0.64
62	2	4	0.04	0	0.12	0.56
63	2	11	0.04	0	0.13	0.52
64	2	16	0.04	0	0.13	0.58
65	3	4	0.04	0	0.13	0.58
66	3	18	0.04	0	0.13	0.58
67	4	9	0.04	0	0.12	0.67
68	4	14	0.04	0	0.13	0.54
69	4	15	0.04	0	0.13	0.6
70	6	9	0.04	0	0.14	0.5
71	7	15	0.04	0	0.14	0.56
72	8	10	0.04	0	0.14	0.56
73	8	22	0.04	0	0.13	0.57
74	9	17	0.04	0	0.12	0.54
75	9	19	0.04	0	0.13	0.58
76	10	17	0.04	0	0.14	0.57
77	11	12	0.04	0	0.14	0.56
78	11	15	0.04	0	0.13	0.56
79	12	16	0.04	0	0.15	0.57

Table E (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
80	15	16	0.04	0	0.14	0.52
81	15	22	0.04	0	0.12	0.52
82	1	11	0.03	0	0.13	0.51
83	2	6	0.03	0	0.12	0.43
84	2	9	0.03	0	0.12	0.53
85	3	16	0.03	0	0.12	0.39
86	4	11	0.03	0	0.11	0.46
87	5	7	0.03	0	0.12	0.5
88	5	16	0.03	0	0.13	0.48
89	8	21	0.03	0	0.12	0.41
90	9	14	0.03	0	0.11	0.46
91	9	20	0.03	0	0.12	0.52
92	10	12	0.03	0	0.12	0.47
93	10	16	0.03	0	0.12	0.45
94	13	16	0.03	0	0.12	0.45
95	14	16	0.03	0	0.11	0.49
96	16	18	0.03	0	0.12	0.4
97	1	4	0.02	0	0.11	0.44
98	1	5	0.02	0	0.11	0.38
99	1	13	0.02	0	0.11	0.35
100	2	5	0.02	0	0.11	0.41
101	2	19	0.02	-0.03	0.1	0.42
102	2	21	0.02	0	0.1	0.42
103	4	6	0.02	-0.02	0.11	0.37
104	5	6	0.02	-0.02	0.11	0.4
105	5	14	0.02	0	0.1	0.36
106	6	10	0.02	0	0.09	0.33
107	6	18	0.02	0	0.1	0.36
108	6	19	0.02	0	0.09	0.34
109	7	10	0.02	0	0.1	0.37
110	8	20	0.02	0	0.1	0.42
111	11	20	0.02	0	0.1	0.42
112	13	17	0.02	-0.01	0.1	0.36
113	13	19	0.02	0	0.12	0.35
114	13	21	0.02	0	0.1	0.32
115	14	22	0.02	0	0.1	0.4
116	15	20	0.02	0	0.1	0.35
117	18	21	0.02	0	0.12	0.42
118	1	22	0.01	-0.03	0.07	0.22
119	2	7	0.01	-0.02	0.07	0.21
120	2	14	0.01	-0.02	0.07	0.25

Table E (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
121	2	20	0.01	0	0.09	0.33
122	2	22	0.01	-0.03	0.07	0.26
123	3	11	0.01	-0.04	0.08	0.29
124	3	13	0.01	0	0.09	0.31
125	4	16	0.01	-0.03	0.08	0.34
126	4	19	0.01	-0.05	0.08	0.32
127	4	20	0.01	0	0.09	0.32
128	5	10	0.01	0	0.09	0.34
129	5	11	0.01	-0.02	0.09	0.35
130	6	8	0.01	-0.02	0.07	0.22
131	6	15	0.01	0	0.07	0.24
132	7	17	0.01	-0.02	0.08	0.35
133	8	13	0.01	-0.03	0.09	0.29
134	8	17	0.01	0	0.08	0.31
135	9	10	0.01	0	0.09	0.3
136	9	11	0.01	0	0.09	0.27
137	10	13	0.01	-0.02	0.09	0.34
138	11	21	0.01	-0.02	0.07	0.24
139	12	15	0.01	-0.04	0.09	0.36
140	12	19	0.01	0	0.08	0.29
141	13	15	0.01	0	0.08	0.27
142	19	21	0.01	-0.03	0.08	0.28
143	19	22	0.01	-0.02	0.08	0.25
144	1	17	0	-0.06	0.04	0.28
145	2	10	0	-0.06	0.05	0.22
146	3	10	0	-0.06	0.06	0.26
147	3	17	0	-0.06	0.06	0.28
148	4	5	0	-0.05	0.05	0.23
149	4	8	0	-0.06	0.07	0.31
150	4	17	0	-0.05	0.06	0.29
151	5	21	0	-0.07	0.03	0.24
152	6	14	0	-0.04	0.05	0.21
153	6	16	0	-0.07	0.08	0.29
154	8	9	0	-0.06	0.06	0.26
155	9	13	0	-0.04	0.03	0.18
156	9	22	0	-0.06	0.02	0.2
157	12	18	0	-0.04	0.06	0.28
158	13	18	0	-0.04	0.03	0.23
159	13	22	0	-0.03	0.04	0.17
160	14	20	0	-0.06	0.04	0.25
161	1	10	-0.01	-0.08	0.03	0.31

Table E (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
162	1	18	-0.01	-0.08	0.02	0.28
163	3	22	-0.01	-0.07	0.03	0.23
164	5	12	-0.01	-0.07	0.02	0.28
165	5	15	-0.01	-0.08	0	0.3
166	6	17	-0.01	-0.07	0	0.29
167	6	22	-0.01	-0.07	0.03	0.23
168	7	9	-0.01	-0.08	0	0.23
169	7	12	-0.01	-0.08	0.03	0.27
170	7	14	-0.01	-0.09	0.02	0.27
171	9	15	-0.01	-0.08	0	0.26
172	11	14	-0.01	-0.07	0.02	0.28
173	11	18	-0.01	-0.07	0.03	0.27
174	12	17	-0.01	-0.06	0.02	0.25
175	12	22	-0.01	-0.08	0	0.36
176	16	21	-0.01	-0.08	0.03	0.26
177	16	22	-0.01	-0.07	0	0.2
178	17	22	-0.01	-0.08	0	0.31
179	18	19	-0.01	-0.08	0	0.3
180	1	8	-0.02	-0.09	0	0.31
181	2	8	-0.02	-0.1	0	0.32
182	2	18	-0.02	-0.09	0	0.33
183	4	18	-0.02	-0.09	0	0.4
184	6	20	-0.02	-0.1	0	0.36
185	7	22	-0.02	-0.1	0	0.33
186	8	11	-0.02	-0.09	0	0.35
187	9	18	-0.02	-0.1	0	0.39
188	10	14	-0.02	-0.12	0.03	0.4
189	14	21	-0.02	-0.1	0	0.35
190	16	17	-0.02	-0.1	0	0.39
191	17	21	-0.02	-0.1	0	0.39
192	1	20	-0.03	-0.11	0	0.41
193	2	13	-0.03	-0.11	0	0.43
194	3	6	-0.03	-0.14	0	0.49
195	4	13	-0.03	-0.12	0	0.4
196	4	22	-0.03	-0.1	0	0.43
197	5	17	-0.03	-0.11	0	0.5
198	5	18	-0.03	-0.11	0	0.48
199	5	22	-0.03	-0.12	0	0.48
200	7	16	-0.03	-0.12	0	0.42
201	10	21	-0.03	-0.11	0	0.42
202	12	13	-0.03	-0.11	0	0.52

Table E (Continued)

	Variable A	Variable B	Mean	qtl_low	qtl_high	propLtZ
203	12	14	-0.03	-0.11	0	0.44
204	12	20	-0.03	-0.11	0	0.45
205	13	20	-0.03	-0.12	0	0.43
206	14	17	-0.03	-0.12	0	0.52
207	15	18	-0.03	-0.12	0	0.44
208	17	20	-0.03	-0.11	0	0.49
209	1	14	-0.04	-0.14	0	0.56
210	5	19	-0.04	-0.12	0	0.54
211	6	7	-0.04	-0.12	0	0.57
212	6	13	-0.04	-0.14	0	0.6
213	7	18	-0.04	-0.12	0	0.59
214	7	19	-0.04	-0.14	0	0.46
215	8	19	-0.04	-0.13	0	0.58
216	9	12	-0.04	-0.14	0	0.54
217	11	19	-0.04	-0.14	0	0.55
218	15	21	-0.04	-0.12	0	0.55
219	18	22	-0.04	-0.13	0	0.55
220	7	21	-0.05	-0.14	0	0.59
221	3	20	-0.06	-0.15	0	0.65
222	3	21	-0.06	-0.15	0	0.69
223	11	22	-0.06	-0.15	0	0.71
224	12	21	-0.06	-0.15	0	0.72
225	2	12	-0.07	-0.14	0	0.84
226	10	20	-0.08	-0.16	0	0.86
227	11	16	-0.08	-0.17	0	0.88
228	6	21	-0.1	-0.2	0	0.87
229	3	12	-0.11	-0.19	-0.02	0.95
230	3	15	-0.11	-0.2	0	0.95
231	20	21	-0.14	-0.23	-0.03	0.97

Note.

Legend. node 1= intense mood shifts, node 2= attitude about self changes, node 3=stormy relationships, node 4=chronic emptiness, node 5=let people know they hurt me, node 6= unsteady mood, node 7=worry about people leaving, node 8= 'people let me down', node 9= little control over anger, node 10= wonder about life, node 11= feeling lonely, node 12= feeling unhappy, node 13= cannot handle separation, node 14= mistakes in picking friends, node 15= hurt self when upset, node 16= cannot express all of anger, node 17= gets bored easily, node 18= difficulty with staying friends with people , node 19= impulsivity and recklessness, node 20= MASExc (hypermentalisation), node 21= MASClless (hypomentalisation), node 22= MASClno (no mentalisation).

^aVariable A= node A; ^b Variable B= node B; ^c Mean= mean edge weight across 1000 bootstrap estimations; ^{d+e} qtl_low and qtl_high= 95% quantiles of edge weights, containing 95% of the observations across 1000 bootstrap sampling (translatable to 95% confidence intervals);^f propLtZ= the probability for the edge to be 0 or proportion of edge weight estimates that were non-zero presented in decimals, translatable to percentage (for regularised network models, it should not be interpreted as a hypothesis test)

Appendix F

Differences in the strength of average edge weights between variables across the two groups

	N:1	N:2	N:3	N:4	N:5	N:6	N:7	N:8	N:9	N:10	N:11	N:12	N:13	N:14	N:15	N:16	N:17	N:18	N:19	N:20	N:21	N:22	N:23
N: 1	0,00	-0,04	-0,04	0,06	0,00	0,00	-0,04	0,00	-0,06	0,00	0,00	-0,06	0,00	0,00	-0,19	0,00	0,00	0,00	-0,08	-0,04	0,00	0,00	0,00
N: 2	-0,04	0,00	0,00	0,07	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,02	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00
N: 3	-0,04	0,00	0,00	-0,03	0,00	0,00	0,00	-0,06	0,00	0,00	0,00	-0,11	0,00	0,06	-0,06	0,00	0,00	0,00	0,00	0,07	0,00	0,00	0,00
N: 4	0,06	0,07	-0,03	0,00	0,00	0,00	0,00	0,00	-0,04	0,00	0,00	0,00	0,00	0,00	0,08	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00
N: 5	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,05	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,06	0,00	0,00	0,00
N: 6	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,16	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,02	0,00	0,00
N: 7	-0,04	0,00	0,00	0,00	0,00	0,00	0,00	-0,04	0,06	0,00	0,00	0,00	-0,18	0,00	0,00	0,00	0,00	0,00	0,00	-0,06	0,00	0,00	0,00
N: 8	0,00	0,00	-0,06	0,00	0,05	0,00	-0,04	0,00	0,00	0,00	0,00	0,00	0,00	0,06	0,00	0,00	0,00	-0,10	0,00	0,18	0,00	0,00	0,00
N: 9	-0,06	0,00	0,00	-0,04	0,00	0,00	0,06	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,08	0,00	0,00	0,00	0,00	0,00	0,00	0,00
N: 10	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,03	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,05	0,00	0,00	0,00
N: 11	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,03	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,02	0,00
N: 12	-0,06	-0,02	-0,11	0,00	0,00	0,16	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,05	0,00	0,00	0,00	0,00	0,00
N: 13	0,00	0,00	0,00	0,00	0,00	0,00	-0,18	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00
N: 14	0,00	0,00	0,06	0,00	0,00	0,00	0,00	0,06	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,09	0,04	0,00	0,00	0,00
N: 15	-0,19	0,00	-0,06	0,08	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,13	0,00	0,00	0,00	0,00
N: 16	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,08	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,05	0,00	0,00	0,00
N: 17	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00
N: 18	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,10	0,00	0,00	0,00	0,05	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,04	0,00	0,00	0,00
N: 19	-0,08	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,09	-0,13	0,00	0,00	0,00	0,00	-0,06	0,00	0,00	0,00
N: 20	-0,04	0,00	0,07	0,00	-0,06	0,00	-0,06	0,18	0,00	-0,05	0,00	0,00	0,00	0,04	0,00	-0,05	0,00	-0,04	-0,06	0,00	-0,11	-0,04	0,00
N: 21	0,00	0,00	0,00	0,00	0,00	-0,02	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,11	0,00	-0,03	0,00
N: 22	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,02	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	-0,04	-0,03	0,00	0,00
N: 23	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00	0,00

Note. BPD= Borderline Personality Disorder. N=Node.

Estimates representing the difference in the strength of average edge weights were calculated by subtracting the estimated edges in the network of the group of people with diagnosis from the edges in the network of the group of people without a diagnosis. Negative estimates refer to a pairwise association that is stronger in the group of people with a BPD diagnosis, while positive estimates refer to stronger pairwise interactions in the group of people without a diagnosis.