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**Nosographic and Comorbidity Structure, and
Etiopathogenic Trajectories of Posttraumatic Stress
Disorder**

An Approach Based on Complex Psychological Networks

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of Posttraumatic Stress Disorder: An Approach Based on Complex Psychological
Networks**

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Dissertação de Doutoramento em Psicologia, Especialidade de Psicologia Clínica

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Aos meus tesouros,

Ao meu pai,

À minha mãe,

Ao meu companheiro.

“QNPVSC”

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Resumo

A Perturbação de Stress Pós-Traumático é uma perturbação mental complexa e com uma elevada comorbilidade. Esta condição está associada a uma deterioração severa no funcionamento dos indivíduos, a inúmeros problemas psicossociais e tentativas de suicídio. Estas implicações estendem-se a idades precoces. A eficácia dos tratamentos é insatisfatória com um elevado número de indivíduos a continuar a demonstrar sintomas e com uma elevada probabilidade de desenvolver outras perturbações mentais, como psicose. A estrutura nosográfica permanece amplamente desconhecida, bem como as suas trajetórias etiopatogénicas. Além disto, os sistemas tradicionais da psicopatologia (i.e., categoriais e dimensionais) não permitiram superar estes problemas. O tipo de trauma tem sido sugerido como uma possível explicação para a diversidade estruturas nosográficas encontradas na literatura, no entanto, os resultados encontrados têm sido contraditórios. Neste contexto, uma abordagem baseada em redes complexas emergiu no campo da psicologia e permitiu ultrapassar várias das dificuldades mencionadas anteriormente. Contudo, os avanços realizados por esta abordagem permitiram somente uma visão parcial da estrutura nosográfica e de comorbilidade da PTSD e das suas trajetórias etiopatogénicas. Assim, este trabalho teve como objetivo explorar e introduzir novas ferramentas metodológicas que permitissem superar estes obstáculos e obter uma visão global e detalhada destas estruturas. E do impacto diferencial do tipo de trauma na organização sintomática da PTSD e no desenvolvimento de outras perturbações mentais. Assim, a exploração e introdução destes novos métodos poderá contribuir para elucidar e obter uma perspetiva sustentada sobre o funcionamento complexo das perturbações mentais.

Abstract

Posttraumatic Stress Disorder (PTSD) is a complex and highly comorbid mental disorder. This condition is associated with severe deterioration in individuals' functioning, numerous psychosocial problems, and suicide attempts. These implications extend to younger ages. The effectiveness of treatments is unsatisfactory, with a high proportion of individuals continuing to show symptoms and a high probability of developing other mental disorders, such as psychosis. The nosographic structure of PTSD remains largely unknown, as well as its etiopathogenic trajectories. In turn, the traditional psychopathological systems (i.e., categorical and dimensional) did not allow to surpass these problems. The type of trauma has been suggested as a possible explanation for the diversity of nosographic structures found in previous literature. However, results showed contradictory results. In this context, a novel theory based on the complex networks emerged in the psychological field that enabled overcome some of the difficulties mentioned above. However, the advances made by this theory only allowed for a partial view of the nosographic and comorbidity structure of PTSD and its etiopathogenic trajectories. Thus, this thesis aimed to explore and introduce new methodological tools that could address these gaps and obtain a global and detailed perspective of these structures; and assess the differential impact of the type of trauma in the symptomatic organization of PTSD and in the development of other mental disorders. The exploration and introduction of these frameworks may contribute to elucidate and obtain a sustained perspective on the complex functioning of mental disorders.

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Introduction

The nosographic structure of Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) is a complex and heterogeneous mental disorder with high comorbidity, severe implications to an individual's well-being (Bryant, 2019), and increased economic costs to society (McGowan, 2019). The knowledge regarding its nosographic structure and comorbidity remains uncertain (Lee et al., 2019; Knefel et al., 2016), as well as its etiopathogenic trajectories and therapeutic mechanisms (Bryant et al., 2017), which results in poor treatment outcomes (Resick et al., 2017).

Over the last decades, the nosographic structure of PTSD in adult and younger ages has been exhaustively questioned (Bryant, 2019; Miller et al., 2014; Ayer et al., 2011; Elhai et al., 2009). Since its initial formalization in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980) and revisions in the DSM-IV (American Psychiatric Association, 1994) and the DSM-5 (American Psychiatric Association, 2013), numerous definitions of trauma (Bryant, 2019), exposure criteria (North et al., 2016), and several symptoms that reliably depict PTSD (Cao et al., 2017) have been proposed. In the DSM-III (American Psychiatric Association, 1980), the diagnosis of PTSD encompassed 12 symptoms; five symptoms (i.e., physiological reactivity, emotional reactivity, foreshortened future, irritability, and hypervigilance) were added in the DSM-IV (American Psychiatric Association, 1994) and afterward, three more symptoms (i.e., blame, negative beliefs, recklessness, or destructive behavior) were added in the DSM-5 (American Psychiatric Association, 2013).

This gradual inclusion of new symptoms had the primary goal of improving the diagnosis of PTSD (Weathers, 2017) by decreasing the overlap between symptoms of

other mental disorders (Pai et al., 2017). Given the high comorbidity rates of PTSD (Bryant et al., 2010), with, for example, depression (Shah et al., 2012), anxiety (Spinhoven et al., 2014), alcohol use disorder (Norman et al., 2018), autism (Haruvi-Lamdan et al., 2018), psychotic disorders (Swan et al., 2017), and other mental disorders (Greene et al., 2016), this addition was expected to reduce the comorbidity rates, adding more clarity and precision to the diagnosis (Armour, et al., 2016). Consequently, this enabled a more fine-grained view of the PTSD nosographic structure. Nevertheless, the inclusion of new symptoms leads to different prevalence rates of PTSD (North et al., 2016), which constitutes an obstacle, because it maintains the uncertainties about the best symptomatic organization of PTSD.

To a significant extent, efforts to clarify the nosographic structure of PTSD have relied predominantly on factor analytic methods (Marshall et al., 2013). However, these studies' results have been mixed, originating numerous distinct latent structures (Elhai & Palmeri, 2011). These structures varied between one to seven factor-models in adults (Armour et al., 2016), extending to the literature in adolescents (Ayer et al., 2011). Among these models, two four-factor models have received increased attention (Wang et al., 2011), namely, the Emotional Numbing model (King et al., 1998) and the Dysphoria model (Simms et al., 2002). These proposals differed from the three-factor structure proposed in DSM-IV by stating that a four-factor structure was the best symptomatic representation of PTSD (Armour et al., 2016). Essentially, these models vary in the allocation of three symptoms (i.e., sleep difficulties, irritability, and concentration difficulties). In the Emotional Numbing model, these symptoms fit in the cluster of hyperarousal symptoms, whereas in the Dysphoric model, they were placed along with dysphoric arousal symptoms (Armour et al., 2016). These models revealed superior regarding the three-structure proposed by the DSM-IV (i.e., re-experiencing,

avoidance/numbing, hyper-arousal), and several studies were performed to uncover which proposal best captures the structure of PTSD (Elhai & Palmeri, 2011). However, results were mixed, with the Dysphoria model revealing a slighter better performance regardless of the type of trauma or questionnaire used (Yufik & Simms, 2010). Nevertheless, other factor models were developed, namely the Dysphoric Arousal model (Elhai et al., 2011). This model is constituted by five factors and revealed superior to the two-four factor proposals previously mentioned (Armour et al., 2016). This controversy extended after the publication of the DSM-5, which reorganized the symptomatic structure of PTSD in four-factor clusters (i.e., intrusions, avoidance, negative alterations in cognition and mood, altered arousal and reactivity). In this context, factor models ranging between six (i.e., Anhedonia model; Liu et al., 2014; Externalizing Behavior model, Tsai et al., 2015) to seven factors (Hybrid model; Armour et al., 2016; see Table S1 in Chapter III of the Appendix for a detailed description of the factor models proposed in previous literature) were presented.

These results might be related to the use of factor analytical studies that focus on the items (i.e., which represent PTSD symptoms) of the assessment instruments, leading to the shared variance of the PTSD symptoms (Armour et al., 2016). This shared variance suggests that the symptoms of PTSD interact with each other. However, factor analytical proposals cannot explain these interactions because they assume that the items are independent of each other (see Limitations of categorical and dimensional models, bellow). Finding appropriate ways to accommodate the symptoms interactions might elucidate the diversity of distinct conceptualizations of the nosographic structure of PTSD (Borsboom & Cramer, 2013; see Limitations of categorical and dimensional models, bellow). In more specific terms, the multiplicity of symptomatic structures compatible with the shared variance patterns in factor analysis leads to numerous potential

symptomatic combinations (Galatzer-Levy & Bryant, 2013). It also leads to difficulties in the assessment and providing an effective diagnosis (Elhai & Palmieri, 2011; Elhai et al., 2012). Also, it decreases the probability of delivering effective and personalized treatments (Armour et al., 2016). In this context, a clear view of the nosographic structure of PTSD is needed since it would also help clarify the particularities of this mental condition in younger age groups.

Beyond the factor models, the type of trauma has been proposed as a possible explanation for the variety of PTSD nosographic structures (Fink et al., 2017; Shea et al., 2017; Kelley et al., 2009). This suggestion has been made since some studies showed that particular symptoms of PTSD were associated with specific types of trauma (Kessler et al., 2017). For example, symptom avoidance has been found in individuals experiencing sexual trauma (Kelley et al., 2009; Smith et al., 2016). The symptom detachment has been found in military veterans (Holowka et al., 2012). This way, it seems that according to the type of trauma, a specific symptomatic configuration might emerge (Kelley et al., 2009). In turn, these associations have been revealing valuable since they might inform and guide clinicians in the selection of more adequate treatment protocols and intervention techniques to a specific population that experienced a particular type of trauma (Smith et al., 2016).

However, some studies have found some particular symptoms associated with a specific type of trauma in other kinds of trauma. For example, Ge, Yuan, Li, Zhang, and Zhang (2019) and Bryant and colleagues (2017) concluded that physiological reactivity was associated with earthquake trauma and traumatic injury. This enhances the difficulties in the comprehension of the nosographic structure of PTSD and exposes its complexity and heterogeneity. Additionally, other variables, such as sex and gender (Lancaster et al., 2014), the type of sample (i.e., non-clinical or clinical), and the study

isolated of a traumatic event (Smith et al., 2016) have been suggested as potentials indicators that influence the symptomatic configurations of PTSD. Furthermore, the type of trauma has been associated with developing other mental disorders, such as major depression (Negele et al., 2015) and bipolar disorder (Assion et al., 2009).

Moreover, previous studies have shown that exposure to a traumatic event has been associated with the development of psychotic symptoms (Varese et al., 2012). It has been proposed that individuals who suffer from psychotic symptoms might have been exposed to a traumatic event during psychosis since they are individuals more vulnerable to social isolation, financial problems, and homelessness (Maniglio, 2009; Bosson et al., 2011). On the other hand, it has been suggested that exposure to a traumatic event could result in the development of psychosis (Swan et al., 2017). Indeed, two types of PTSD with psychosis have been proposed – post psychotic PTSD (PP-PTSD) and PTSD with secondary psychotic features (PTSD-SP) (Compean & Hamner, 2019), which displays the high comorbidity of this mental disorder.

However, it remains unclear how the type of trauma influences mental disorders' clinical configurations (Guina et al., 2018). Specifically, it remains uncertain if exposure to a traumatic event impacts the symptomatology directly or if other symptoms mediate this association. In this context and considering the growth of studies showing the associations between PTSD and psychosis, uncover how exposure to a traumatic event impacts the development of psychotic symptoms might open new insights to inform therapists, specifically, in their clinical practice to prevent the burden of these two comorbid disorders (Bosson et al., 2011).

Altogether, uncertainties remain about the symptomatic structure of PTSD and the impact of the type of trauma in its nosographic structure and the development of other mental disorders. Previous studies only allowed a partial view about these topics, and a

global and detailed perspective of the nosographic structure of PTSD and its etiopathogenic and maintenance mechanisms is needed.

Limitations of categorical and dimensional models

All the problems mentioned in the previous section seem to be related. In common, they have a conceptual approach based on the latent variable model. According to this framework, symptoms are viewed as observable variables explained by a mental disorder. In other words, symptoms are measurements of a mental disorder (Borsboom, 2008). For example, this model conceptualizes posttraumatic stress disorder as a latent variable that might cause the 20 symptoms described in the DSM. This perspective underlines the traditional psychopathology models, namely, the categorical systems, such as DSM (American Psychiatric Association, 2013) and the International Statistical Classification of Diseases and Related Health Problems (CDI-11; World Health Organization, 2018). And the dimensional systems, such as the Research Domain and Criteria (RdoC; National Institute of Mental Health, 2021). These models assume that symptoms (i.e., observable variables) are equal and interchangeable indicators of a mental disorder (i.e., latent construct) (Armour et al., 2016).

This view has hampered advances regarding the nosology and etiopathogenic and maintenance mechanisms of mental disorders. Categorical systems have been criticized for offering only partial validity to the diagnosis of several mental disorders (Goldberg, 2015), being unable to explain the diversity of clinical configurations (Monroe & Anderson, 2015) and the high rate of comorbidity between mental conditions (Kessler et al., 2005). The dimensional systems attempted to overcome the difficulties of categorical systems. However, they remain predominantly descriptive, unable to explain the variety of clinical configurations and comorbidity patterns (Nuijten et al., 2016; Giudice, 2016),

as well as the etiopathogenic and maintenance mechanisms (Tio et al., 2016; Fried & Nesse, 2015) of mental disorders.

In the particular context of PTSD, the DSM-5 requires a minimum of six symptoms to diagnose this mental condition. According to DSM-5, 20 symptoms divided across four clusters (i.e., intrusions, avoidance, negative alterations in cognition and mood (NACM), and alterations in arousal and reactivity (AAR)) depicts PTSD. Moreover, at least one symptom of the cluster intrusion and avoidance and two symptoms from NACM and AAR clusters must endorse the diagnose. Remarkably, this results in 636,120 possible combinations of PTSD symptoms (Galatzer-Levy & Bryant, 2013), which illustrates the diversity of clinical configurations. Moreover, the fact that DSM requires a predetermined number of symptoms in a particular cluster results in a significant number of individuals being excluded (Armour et al., 2016). The dimensional perspective attempted to surpass some of the categorical perspective's limitations by identifying a set of dimensions (i.e., cognitive, affective, behaviors) to fulfill the loss of information of categorical systems (Clark et al., 2017). This system conceptualizes PTSD in a continuum, where individuals that display a higher severity also showed higher levels of distress (Armour et al., 2016). However, this system remained primarily descriptive without explaining and specifying the associations between the different dimensions and PTSD symptoms (Schmidt & Vermetten, 2017).

Moreover, a growing body of research has recognized the impact of the type of trauma in the development of symptoms of PTSD and other mental disorders (Armour & Shevlin, 2010; Shafer & Fisher, 2011), as well as the different patterns of comorbidity with other mental disorders (Contractor et al., 2013). This way, it appears that symptoms of PTSD are not a reflection of a latent construct. On the contrary, it seems that external variables deepen influence the configuration of PTSD symptoms.

Overall, the perspective of categorical and dimensional models precludes the identification of nosographic and comorbidity structures and clarifies the etiopathogenic and maintenance mechanisms of mental conditions, such as PTSD. This fragmented perspective has significant implications since it hampers the precision of the diagnoses (Armour et al., 2016), resulting in poor case formulations and, consequently, leading to ineffective treatments (Resick et al., 2017) that might lead to a significant deterioration in the individuals' functioning (Koenen et al., 2008).

Complex networks in psychology

The partial view obtained about the nosographic structure and the impact of the type of trauma in the symptomatic organization of PTSD and other mental disorders might be related to the use of methods such as profile analysis (Kelley et al., 2009), confirmatory factor mixed modeling (Shevlin & Elkit, 2012) and logistic regressions (Graham et al., 2016). These methods are based on the latent variable paradigm, which despite the advances that they have achieved, are unable to explain how the symptoms covary with each other. In this context, alternative methodological frameworks that contribute to stabilize further and detail the nosographic structure of PTSD and the impact of the type of trauma are needed.

In recent years, a novel framework has been used in psychology to examine mental disorders – the network model (Borsboom & Cramer, 2013). This theory contrasts with the latent variable model by conceptualizing a mental disorder as a dynamical system in which the symptoms (i.e., nodes) interact (i.e., edges) with each other. This framework argues that it is in the dynamic complexity of the interactions between the symptoms that nosographic and comorbidity structures of a mental condition emerge, and other phenomena might be explained (e.g., clinical configurations, therapeutic outcomes).

In the past decade, a significant body of research has applied the network theory to a diversity of mental disorders (Borsboom, 2017). Significant advances based on the focus of the interactions between the symptoms have been achieved (Borsboom et al., 2019). Some examples are noteworthy in major depression disorder (Fried et al., 2016), generalized anxiety disorder (Beard et al., 2016), substance abuse disorder (Rhemtulla et al., 2016), autism (Anderson et al., 2016), eating disorders (Levinson et al., 2017) and PTSD (Armour et al., 2016).

Based on the theory that mental disorders result from the interaction between symptoms (Borsboom et al., 2019), efforts were made to formalize this theory. Thus, network methods were developed to identify, quantify, and visualize the interactions between the symptoms in a network (Borsboom, 2017). These methods enabled us to surpass some of the traditional latent-variable models' limitations and provide significant contributions to studying the nosographic and comorbidity structure of mental disorders. Broadly, these advances were achieved based on the identification and characterization of two types of symptoms: central and bridge symptoms (Levinson et al., 2017; Marcus, et al., 2018; Smith et al., 2018).

Central symptoms are defined as those symptoms that show the strongest relationships with the other symptoms in the network (Opshal et al., 2010). In other words, these symptoms are responsible for the aggregation of the remaining symptoms in the network (Epskamp et al., 2018). In turn, several descriptions and types of bridge symptoms have been debated. The first authors who applied the network theory to psychopathology defined that bridge symptom connected two different disorders (i.e., bridge symptoms) (Cramer et al., 2010). However, it was also possible a symptom be part of two mental conditions simultaneously (i.e., overlap symptoms) (Cramer et al., 2010). It was recently proposed that bridge symptoms were symptoms responsible for

connecting different clusters of symptoms within a disorder. Additionally, it has been suggested that these symptoms might play different roles or functions in the network (Blanken et al., 2018).

The identification of central symptoms has been achieved based on four metrics: degree, strength, closeness, and betweenness (a detailed description of each of these measures is found in Chapter II). On the other hand, bridge symptoms have been ascertained based on identifying network modules or communities (i.e., clusters of symptoms with strong relationships between themselves). A variety of methods have been proposed to identify bridge symptoms (i.e., bridge centrality metrics; Jones et al., 2018; Clique Percolation; Adamcsek, 2006; Palla et al., 2005). However, these frameworks have shown significant limitations. Specifically, the method proposed by Jones and colleagues (2018) assumes that bridge symptoms can only bridge different disorders, discarding the possibility of symptoms and modules overlap. This relevant because it was shown that bridge centrality is equal to the sum of the weights of the links between symptoms on the network, which decreases its discriminant validity precisely in the case of comorbidity structures (Christensen et al., 2019).

On the other hand, the Clique Percolation method focuses exclusively on the assessment of overlap symptoms. This way, there is an absence of a framework that might identify, quantify, and characterize the two types of bridge symptoms simultaneously and provide information about how the modules are arranged in the network. This knowledge is of significant relevance because it would allow a detailed understanding of an intermediate level of the psychopathological network structures, specifically a level that embraces how the symptom's functions interact with each other.

Despite this, central and bridge symptoms have been hypothesized to be core treatment targets (Armour et al., 2016; Afzali et al., 2017). Based on the assumption that

these types of symptoms have specific roles or functions, it has been discussed that targeting these symptoms could impact the disposal of the interactions in the network (Jones et al., 2018; Blanken, 2018). For example, it has been argued that if a central symptom is targeted (i.e., clinical intervention in this symptom), their centrality levels would probably decrease, and a transformation in the network might occur. It is expected that the connections become weaker and sparser, and the path that connects the symptoms increase, which might result in a diminution of the severity of the symptoms in the network (Isvoranu et al., 2018). On the other hand, targeting a bridge symptom might hamper other disorders' development and the emergence of comorbidity structures (Afzali et al., 2017). However, this continues to be a disputed hypothesis since previous studies found contradictory results (Bringmann et al., 2018; Snippe et al., 2017; Bos et al., 2018; see Chapter II for a detailed discussion). With this in mind, and although previous evidence remains scarce on these topics, this constituted valuable insights that might help guide treatment plans.

Complex networks in Posttraumatic Stress Disorder

As mentioned, the complex network approach conceptualizes PTSD as a dynamic system in which symptoms interact with each other. It is based on these interactions that this mental disorder arises (Birkeland et al., 2020). This perspective contributed to important advances regarding its nosographic and comorbidity structures and the identification of possible therapeutic targets, essentially through identifying central and bridge symptoms. For example, in the study conducted by McNally and colleagues (2015), in a sample of earthquake survivors, the symptom hypervigilance was the most central symptom, suggesting that this symptom might be an important therapeutic target for individuals that experienced a natural disaster. Armour and colleagues (2017) also

performed a study in a military veteran sample and found that negative trauma-related emotions, flashbacks, detachment, and physiological reactivity were the most central symptoms. Similarly, the authors suggested that these symptoms might be important therapeutic targets since they might help attenuate the severity of the other symptoms in the network.

On the other hand, identifying bridge symptoms reveals a critical step to prevent the emergence of comorbidity structures and poor therapeutic outcomes (Armour et al., 2017). Given the high comorbidity levels of PTSD with other mental conditions, such as major depressive disorder, Afzali and colleagues (2017) conducted a study to identify the bridge symptoms among these mental disorders. The authors found that irritability, concentration problems, sleep problems, and loss of interest were symptoms that bridge PTSD and major depression disorder. Furthermore, PTSD reveals high levels of comorbidity with alcohol use disorder (Sareen, 2014). In this context, in another study, Afzali and colleagues (2017) attempted to unveil the bridge symptoms that connected these disorders and found that alcohol use in a dangerous situation, loss of interest, and reckless behavior were the symptoms that bridged these disorders. This way, identifying central and bridge symptoms of PTSD constituted a significant step forward to inform clinicians and help develop effective and personalized treatments.

Despite the valuable contribution of the complex network approach, previous literature found inconsistent results regarding central and bridge symptoms across various mental disorders (Contreras et al., 2019). For example, in major depression disorder, the symptom loss of interest was identified as a central symptom in Park and Kim (2020) study. Nevertheless, in the study performed by Boschloo and colleagues (2016), the concentration difficulties symptom was identified as the most central symptom central. This controversy extended to the literature on bridge symptoms. For example, Kaiser and

colleagues (2021) found that bridge anxiety and depression disorders were the symptoms of psychomotor agitation or retardation. In turn, Garabiles and colleagues (2019) found that the symptoms that bridge these disorders were fatigue, depressed mood, and anhedonia. In this context, identify central and bridge symptoms appears to be a central issue.

Within PTSD, the diversity of central and bridge symptoms is remarkable (Birkeland et al., 2020). A plethora of central symptoms has been found in previous network studies. For example, the symptom flashbacks were the symptom with the highest strength levels in McBride and colleagues' study (2020). However, in the study performed by Birkeland and colleagues (2017), the emotional numbing symptom was identified as the most central symptom. At the same time, this scenario extended to the network literature on bridge symptoms. The detachment symptom and loss of interest were found as bridge symptoms within PTSD (Ross et al., 2018). Nevertheless, in another study performed by Ross and colleagues (2020), the symptom concentration problem was identified as the symptom with the highest bridge centrality levels.

This diversity of central and bridge symptoms might be related to different types of trauma (Ross et al., 2018). Previous network studies have suggested that the type of trauma might influence the nosographic structures of PTSD (Benfer et al., 2018). Also, earlier network studies have proposed that the type of trauma might influence the development of symptoms of PTSD and other mental disorders, such as psychosis (Sawn et al., 2017). However, PTSD network studies on these topics have been scarce and only allowed a partial view of how the type of trauma might influence the nosographic structure of PTSD and the development of other mental disorders.

This way, the accurate and precise identification of central and bridge symptoms is essential. Following the psychopathological network model, the intervention in these

symptoms might be of extreme clinical relevance (van Borkulo et al., 2015). Essentially, because it could help prevent the spread of information to the other symptoms in the network in an initial stage after the traumatic event (Borsboom & Cramer, 2013). Also, independently of the type of trauma and psychopathological stage, it could promote the symptoms networks' disaggregation faster (Borsboom, 2017). Therefore, a detailed characterization of how the type of trauma influences PTSD and other symptoms is needed.

To surpass these obstacles, the Fused Graphical Lasso (FGL; Dahaner et al., 2014) and the Network Comparison Test (NCT; van Borkulo et al., 2019) have been explored. The FGL is a framework that allows the joint estimation of a set of networks. Essentially, this method enables the quantification of the similarity between networks and, consequently, the networks' replicability (Dahaner et al., 2014). The NCT is a framework that allows the comparison of a set of networks. Specifically, the assessment of particular aspects of the networks, such as structure invariance, global strength invariance, and edge strength invariance (for a detailed description of these methods, see Chapter IV).

These methods provided significant advances in clarifying the nosographic structure of PTSD (see Fried et al., 2018, for a detailed review). However, at the same time, they revealed a significant set of limitations that restricted a deeper understanding of the symptomatic organization of PTSD. Specifically, the FGL ensures the networks' validity and replicability inhibit the differences that might be clinically significant (Liu et al., 2018). Also, the NCT cannot compare more than two networks each time, which hampers the networks' comparison. Moreover, the NCT method to evaluate the differences between the edge strength invariance might lead to type I error and needs significant sample sizes to ensure statistical power (Fried et al., 2018).

Hence, to overcome these obstacles, alternative methodological frameworks are needed. Essentially, those that could provide a clear view of (1) the nosographic structure of PTSD; (2) the differential impact of the type of trauma in the nosographic structure of PTSD, and (3) the impact of the type of trauma in the clinical configuration of mental disorders.

Goals, methodology, and the dissertation structure

The beginning of this work concurred with the growth of the network theory in psychology. At the time, this theoretical framework allowed surpassing several obstacles, as previously mentioned. This new conceptualization of mental disorders was formalized based on methods that enabled the quantitative analysis of the importance of symptoms and their interactions.

Despite the relevant contributions made by these methods, a whole set of questions remained partially unanswered. With the growing number of studies in network analysis, a diversity of central and bridge symptoms started to emerge, highlighting these methods' difficulties in identifying these symptoms and their precise and accurate role. Also, it exposes the scarcity of methods able to accommodate the theoretical proposals made by the network theory of psychopathology regarding the two types of bridge symptoms, as well as the difficulties in identifying the most promising therapeutic targets, that according to this framework, might have an essential role in the deactivation of the interactions between the symptoms.

In this context, the focus of this thesis was two-fold. This work's first aim covered the exploration of new methods that could identify accurate and precise nosographic structures of mental disorders and clarify the symptoms' functions in the network to identify the most relevant therapeutic targets. The second aim consisted of applying these methods to PTSD to promote a deeper understanding of this mental condition. Specifically: (1) identify the nosographic structure of PTSD; (2) clarify the comorbidity structure of PTSD; (3) characterize the role of central and bridge symptoms within PTSD; (4) explore the intermediate level that might be understood by the analysis and characterization of modules; (5) ascertain the differential impact of the type of trauma in the nosographic structure of PTSD and (6) clarify the influence of the types of trauma in

the development of psychotic symptoms. Altogether, the integration of these frameworks and the advances achieved by the traditional methods in the analysis of psychological networks would contribute to obtaining a fine-grained view of the symptomatic organization of PTSD and how this arrangement might be transformed considering the type of trauma.

Two novel methods were explored: ModuLand (Kóvacs et al., 2010) and CoDiNA (Gysi et al., 2020). Initially, ModuLand's was tested through the analysis of the network of personality traits to assess its precision in evaluating the structure of psychological constructs and assess its efficacy in the detection and characterization of central and bridge nodes and modules (Chapter I). Also, it was used to estimate the two types of bridge symptoms in a sample of psychopathological networks to address the theoretical proposal of the network model regarding the potential of bridge symptoms as valuable treatment targets (Chapter II). After ModuLand's assessment, it was applied to PTSD (Chapter III) to uncover the modular structure of PTSD and the role of the different symptoms in the network. Subsequently, CoDiNA was used to characterize the structure of several PTSD networks covering different types of trauma and identify differences between these networks (Chapter IV). A distance-based measure of the network structure – shortest paths – was used to evaluate the intermediate role of general and PTSD symptoms on the pathway between different traumatic events and the development of psychotic symptoms (Chapter V).

CHAPTER I ^{1,2,3}

Modularity of the Personality Network

Note:

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² Supplementary materials of this paper are available in the Chapter I of the Appendix.

³ The first and the second author contributed equally to the development of the paper.

Abstract

The Five Factor Model (FFM) is the most widely used personality model; it proposes a hierarchical structure of personality with personality characteristics, facets, and factors. An increasing number of studies has challenged the FFM and a plethora of factor models with varying numbers of facets and factors have been proposed, leading to uncertainties about the structure of personality. The networked system of interactions between personality characteristics has stimulated promising progresses, however, the methodological developments needed to map the topological structure and functional organisation remain scarce. This study aims to explore the hierarchical modular structure of the personality network and the functional role of personality characteristics. A sample of 345,780 individuals ($M_{age} = 24.99$, $SD_{age} = 10.00$, 59.18% female) that completed the International Personality Item Pool – NEO – 120 in a previous study was reanalysed. A non-regularised method was used to estimate the personality network and ModuLand was used to characterise its modular structure. Results revealed a modular structure comprising three levels: one level with the 120 personality characteristics, a second level with 35 modules, and a third with 9 modules. Such results suggest that specific personality characteristics and modules have specialised roles in the topological structure of the personality network.

Modularity of the Personality Network

The Five Factor Model (FFM; Digman 1989) is the most influential multidimensional personality model. This model recognises that personality is hierarchically structured (McCrae & John, 1992) on three levels: factors, facets, and specific personality characteristics. The higher level comprises 5 factors/dimensions defined as Extraversion, Agreeableness, Conscientiousness, Neuroticism, and Openness to Experience (McCrae & Costa, 1997). Subsequently, each of these factors contains subgroups of characteristics identified as facets, which, cover more specific personality characteristics (Mottus et al., 2018). Although a substantial number of studies support this model (see John, Naumann, & Soto, 2008 for a review), there remains no consensus about the structure of personality due to a lack of consistent results pertaining to the different descriptive levels proposed by the FFM. The number of factors has been contested due to the lack of reproducibility across personality inventories, with different studies supporting a variable number of factors ranging from one to twelve (e.g. Backstrom, 2007, Digman, 1997; Wakabayashi, 2014; Shedler & Westen, 2004). Furthermore, there is no generally recognised method of deriving facets (McCrae, 2009; Ziegler & Backstrom, 2016), which causes the number of facets to change according to the inventory that is used (Soto & John, 2016). In addition, the number of levels has also been contested and alternative proposals have emerged that attempt to provide more fine-grained views of personality (e.g., DeYoung, Quilty, & Peterson, 2007; Mottus, Kandler, Bleidorn, Riemann, & McCrae, 2017).

From a psychometric perspective, these discrepancies were attributed to the selection of personality characteristics included in the personality inventories and the choice of factor rotations (Franic, Borsboom, Dolan, & Boomsma, 2014), the presence of cross-loadings (McCrae & Costa, 2008), and the misinterpretation of the statistical

techniques (usually exploratory factor analysis or principal components analysis), from which the FFM is derived (Borsboom, 2006). Additionally, the personality structures originating from these techniques tend not to adequately fit in a confirmatory context (McCrae, Zonderman, Costa, Bond, & Paunonen, 1996). As such, a theoretical interpretation of these results is difficult because the FFM lacks any formal theory (Franic, Borsboom, Dolan, & Boomsma, 2014).

In this context, it is significant that recent studies have found personality characteristics to be better predictors in a variety of outcomes than the FFM factors themselves (Seeboth & Möttus, 2018; Mottus, Kandler, Bleidorn, Riemann, & McCrae, 2017), suggesting that personality characteristics are more accurate descriptors and might provide better explanations for the personality structure than the factors. This is especially noteworthy in light of the recognition that there exists a lack of clarification in the FFM, of the interactions that emerge between the personality characteristics (Goekoop, Goekoop, & Scholte, 2012), and a lack of integration of these interactions in the personality models (Baumert et al., 2017). In this way, the FFM largely remains descriptive in its nature and so is unable to provide an explanatory theory regarding these interactions (John, Naumann, & Soto, 2008). In fact, some authors have recently proposed that these interactions between the personality characteristics are responsible for the emergence of the personality structure (Cramer et al., 2012). Altogether, this suggests that the interactions between the personality characteristics are more relevant to the comprehension of the personality structure than acknowledged and implied by previous models. The recently developed network approach for psychology focuses precisely on these interactions (Cramer et al., 2012).

A network is composed of nodes or vertices that are connected by edges or links and can be used to explain various kinds of phenomena such as social, biological, and

semantic (Kovács, Palotai, Szalay, & Csermeley, 2010). For psychology, this implies a change in the nature of psychological phenomena from the traditional latent variable perspective to a perspective centred on the interactions between psychological variables. While common factor analytic techniques use these variables to identify an unobserved or latent construct (Wright, 2017), network analysis focuses on the interactions between variables to explain the psychological phenomena without referring to any latent construct beyond the psychological variables in question.

Recently, this approach has been applied to the study of personality (Goekoop et al., 2012), conceptualising personality as a networked system of interacting personality characteristics (Cramer & Borsboom, 2015; Cramer, Waldorp, Maas, Borsboom, 2010). In this context, personality characteristics (i.e., items) are viewed as nodes in a network and the interactions (i.e., partial correlations) between them as links. In this perspective, it is through these interactions that the personality structure develops and stabilises. However, previous studies on the personality network have focused predominantly on establishing the theoretical (Cramer et al., 2012) and methodological (Costantini et al., 2015; Costantini et al., 2019; Goekoop et al., 2012) potential of network analysis or on the exploration of specific dimensions of the FFM (Costantini & Perugini, 2016; Christensen, Cotter, & Silvia, 2018). This leaves a large amount of ground uncovered in comparison with the advances made in psychopathological networks. Within the context of psychopathology networks, symptoms are conceptualised as nodes and the interaction between them links (Cramer et al., 2010). Focusing on the interaction between symptoms has facilitated advancements in the nosology and comorbidity of mental disorders (Curtiss & Klemanski, 2016). This has largely been accomplished through the detection of network communities or modules and the identification of bridge symptoms within the

network (McNally, Mair, Mugno, & Riemann, 2017; Bekhuis, Schoevers, van Borkulo, Rosmalen, & Boschloo, 2016).

Modules are sets of highly interconnected symptoms and have been used to identify the structure of various mental disorders (e.g., Price, Legrand, Brier, & Hébert-Dufresne, 2019, Blaken et al., 2018). This has been achieved either by estimating a network of only one disorder and determining its intrinsic organisation (Birkeland & Heir, 2017) or by combining, in the same network, two different but highly comorbid disorders, and establishing which symptoms belong to each disorder (Castro, Ferreira, Mendes, & Ferreira, 2018; Jones, Mair, Riemann, Mugno, & McNally, 2018). This assumes that the symptoms that are more connected with each other correspond to a specific mental disorder or to a specific group of disorder characteristics (Borsboom, 2017). Moreover, by identifying these modules, we can identify the symptoms that connect them. These symptoms are known as bridge symptoms (Jones, Ma, & McNally, 2019) and have been proposed to be the mechanisms behind the development of comorbid disorders (Cramer et al., 2010).

In previous psychopathological network studies, bridge symptoms have been divided into two subcategories: symptoms that connect both disorders due to their interactions with other modules in the network (Levinson et al., 2017) and symptoms that connect disorders by belonging to more than one module (Cramer et al., 2010). The identification of these symptoms and modules has been done with two major methods in psychopathological networks. One of these methods was proposed by Jones and colleagues (2019), which uses theory guided defined modules (i.e., the researcher defines the modules) followed by the estimation of a bridge centrality measure for each symptom; this determines the strength of the connections that the symptom has with the other modules. Thus, the higher the bridge centrality of a specific symptom, the more important

its role in connecting the modules. However, this method fails to specify the modules based on the network properties and is not able to determine symptoms that belong to both disorders. The other proposed method is the Clique Percolation Method (CPM; Adamcsek, Palla, Farkas, Derényi, & Vicsek, 2006; Palla, Derényi, Farkas, & Vicsek, 2005), introduced into the psychopathological network's literature by Blanken and colleagues (2019), which determines the symptoms that belong to both modules; however, the method cannot determine the number of connections of a symptom with the other modules. Translating this into personality networks, modules represent sets of highly interconnected personality characteristics that comprise the personality structure (Goekoop et al., 2012). For example, while in the traditional factor analytic models the extraversion factor occurs due to patterns of covariance in certain items (Wright, 2017), in the network perspective extraversion emerges from the interactions between these items (Cramer et al., 2012). In turn, the characteristics that connect the modules would be bridge characteristics. As with bridge symptoms, these might be characteristics that are responsible for the development of the personality structure.

However, none of the aforementioned algorithms for module and bridge detection are able to determine a hierarchical structure, which is central to the FFM proposal (McCrae & John, 1992). The only approximation to an estimation of a hierarchical structure proposed in the psychological networks studies is the Minimum Spanning Tree method (MST; Mantegna, 1999). The MST gives us indirect information on hierarchical levels, but it does not produce measures for any of the two types of bridges proposed or for the modules in each hierarchical level. Consequently, none of the methods introduced in psychological networks allow for a complete and detailed exploration of the personality structure.

Therefore, the modular structure of the personality network remains largely unexplored, maintaining current uncertainties about the constitution of personality modules, as well as the mechanisms underlying the interactions between the personality characteristics both within and across the modules. In this context, there exists a need for new methods that can identify the hierarchical structure of personality and both types of bridge characteristics between the various modules in the different hierarchical levels. This paper aims to address these issues by exploring the ModuLand framework (Kovács et al., 2010). ModuLand (Kovács et al., 2010) is an integrated framework for the identification of the hierarchical modular structure of complex networks; it can quantify both types of bridge characteristics and provides information regarding the connections within and across modules for all hierarchical levels. This fine-grained view of personality could provide valuable information about the inner workings and organisation of personality, which could, consequently, improve research and clinical practice (Goekoop et al., 2012; Knefel & Lueger-Schuster, 2016).

Therefore, with this framework we aim to characterise (1) the modular structure of personality; (2) the modular connectivity; and (3) the functional roles of the personality characteristics.

Method

Participants

The open access data from a previous study on the International Personality Item Pool – NEO – 120 (IPIP-NEO-120; see Johnson, 2014 for a detailed description) was analysed. Originally, 690,863 anonymous individuals completed a web-version of the 120 items of the IPIP-NEO-120. The percentage of missing values across the 120 items ranged from 0.1% to 0.7%, and the percentage of mismatch across all pairs of items from 0.13%

to 1.37%. The number of missing values did not significantly varied between genders. After removing participants with missing responses, a sample of 345,780 participants ($M_{age} = 24.99$, $SD_{age} = 10.00$, 59.18% female) was analysed.

Measures

International Personality Item Pool – NEO –120 (IPIP-NEO-120). The IPIP-NEO-120 is a 120-item self-report inventory that measures the five domains of personality and the 30 facets of the FFM. The IPIP-NEO-120 revealed good reliability, with mean alphas for facets ranging from .63 to .88, and between .81 to .90 for the dimensions (Johnson, 2014).

Network Estimation and Analysis

The personality network was estimated using a non-regularised method based on multiple regression, implemented in the GGMnonreg package (version 0.1.0; for a detailed description see Williams, Rhemtulla, Wysocki, & Rast, 2019) for R (version 3.5.1; R Development Core Team, 2018). This method was used since it recently achieved high specificity, as well as a low false positive rate, in comparison with traditional estimation methods (Epskamp, Borsboom, Fried, 2018). The R package qgraph (version 1.5; Epskamp et al., 2012) was used for the graphical representation of the personality network. The quality of partitions was evaluated through the modularity measure available in igraph package (version 1.2.4.1; Csárdi, 2019) for R (version 3.5.1; R Development Core Team, 2018). Modularity is the number of optimal edges that can be removed to identify communities (Newman, 2006).

Hierarchical Modular Structure

The ModuLand algorithm (Kovács et al., 2010), implemented in Cytoscape 3.5.1. (Shannon et al., 2003), was used to estimate and characterise the hierarchical modular structure of the personality network. This algorithm defines a module as a set of nodes that mutually influence each other. So, in order to estimate the modular structure, ModuLand initially applies the Linkland function to assess the influence zone of each edge in the network. The influence zone is a subgraph of the network that contains the starting edge and all other edges in the network, which has an indirect impact on the starting edge. Subsequently, the community centrality of each edge is estimated by computing the influence zones covered by a given edge. From this, the community centrality of the node is derived (see Kovács et al., 2010 for detailed formulas). Nodes are then expressed in a community centrality landscape, where nodes with higher community centrality (module centres) form hills; overlapping hills are considered overlapping modules. In this way, the number of modules corresponds to the number of hills, i.e., the number of nodes with the highest levels of mutual influence (community centrality). According to their positioning in the community centrality landscape, module assignment values for the remaining nodes are estimated.

In order to create the hierarchical levels, modules from the previous level become meta-nodes and the overlapping values between them the links. Thus, if the first level has five modules, the subsequent level will be a network composed by five nodes and the links between them will be the overlapping values of the modules in the previous level. It is from this network that the next hierarchical level is estimated, repeating the initial procedure. These steps are repeated until the network becomes just one single meta-node.

Moreover, ModuLand allows the characterisation of the hierarchical modular structure of the personality network and the functions of the personality characteristics.

This framework thereby provides a set of measures that allow a more comprehensive view of the hierarchical modular structure, namely community centrality and effective degree. Community centrality is a measure that displays the extent of the influence each personality characteristic or module possesses. Effective degree corresponds to the effective number of weighted connections of a given characteristic or module. In addition, ModuLand enables the characterisation of two types of bridge symptoms through measures of modular bridgeness and modular overlap. Modular bridgeness is a measure of the inter-modularity that corresponds to the effective number of the modules that a personality characteristic is connected to. Similarly, modular overlap is a measure of the trans-modularity of each personality characteristic and corresponds to the effective number of modules that a personality characteristic belongs to.

Network Robustness

The robustness of the personality network was examined according to the procedures of previous studies (e.g., Letina, Blanken, Deserno, & Borsboom, 2019). The initial sample was divided into two random sub-samples by attributing each participant a 50% probability of being assigned to either sub-sample. This procedure was repeated 100 times resulting in 100 pairs of sub-samples. For each sub-sample, the network was estimated according to the procedure described above. The networks corresponding to each pair were compared on several indicators. As presented in Table 1, high reliability was evidenced by all indicators. Procedures and results for robustness under different sample sizes and partitioning are available in the Supplementary Materials in Figures S21-S28.

Table 1*Descriptives of the robustness indicators*

Robustness Indicator	M (SD)	Minimum - Maximum
Difference in the total number of estimated edges	0.000	.000 – .000
Similarity Index	.871 (.006)	.856 – .885
Proportion of edges that failed to replicate	.129 (.006)	.115 – .144
Correlation between edges weights	.992 (.000)	.992 – .993
Correlation between nodes degree	.982 (.002)	.977 – .986
Correlation between nodes closeness	.993 (.001)	.989 – .996
Correlation between nodes betweenness	.984 (.004)	.973 – .990
Correlation between nodes expected influence	.995 (.001)	.992 – .997
Number of estimated second-level modules on split-halves samples	36 (.761)	33 - 37
Number of estimated third-level modules on split-halves samples	10 (3.30)	1 - 12

Results

Personality Network

Figure 1 depicts the personality network estimated from the 120 personality characteristics included in the IPIP-NEO-120 (Johnson, 2014). Nodes (the circles) represent the 120 personality characteristics of the IPIP-NEO-120 and the edges (the lines connecting the circles) represent the connections apparent between the nodes. The width of the edges between the nodes represents the connection strength. Edges in red represent negative connections, whilst positive connections are represented in blue. The network is constituted of 4410 connections (density = .618), of which 2484 (56.32%) are positive connections and 1926 (43.67%) are negative. The weights of positive connections range from .005 to .628 ($M = .031$, $SD = .050$); negative connections weigh from .005 to .344 ($M = .017$, $SD = .016$). More network descriptives can be found in Table S2 in supplementary materials.

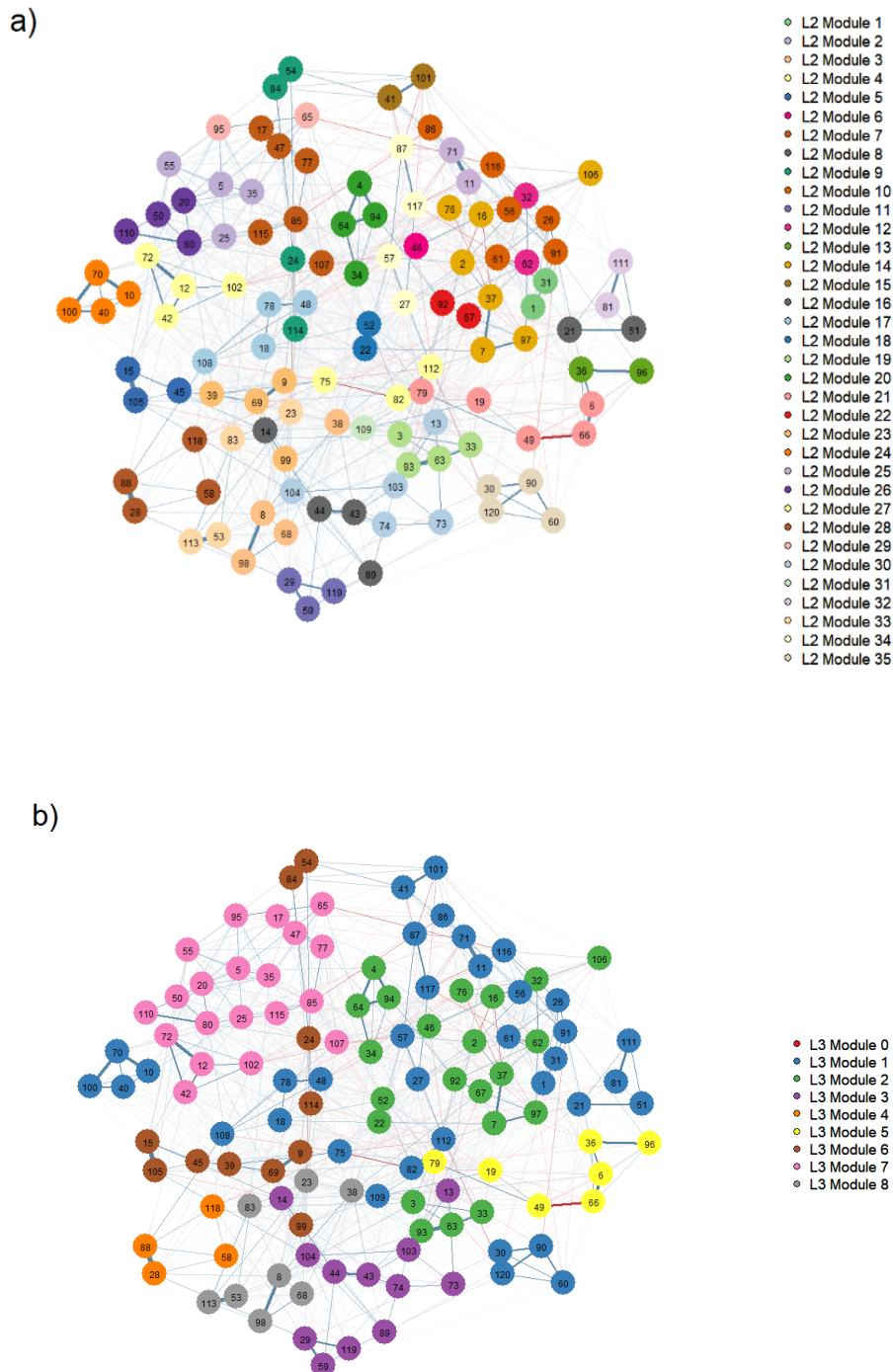


Figure 1. Graphical representation of the first level network. Nodes represent the 120 items in the IPIP-NEO-120. Panel (a) shows a projection of the second level modules in the first level network and panel (b) a projection of the third level modules in the first level network. In panel (a), the colours of the nodes represent the second level modules to which the node is most connected to. In panel (b), the colours of the nodes represent the third level modules to which the nodes are most assigned to. In both panels, the edges between the nodes represent the interaction between the nodes and the width of the edge represents the interaction strength. Only edges with weights superior to .3 are represented in the figure. Non-thresholder figures are available in the Supplementary Materials.

Modular Structure of the Personality Network

The modular structure of the personality network was comprised of three levels. The first level corresponds to the 120 personality characteristics (i.e., items), the second level comprised 35 modules, and the third comprised 9 modules. The interactions between the second-level and third-level modules was estimated through the overlap values in the previous level. Figure 2 represents the hierarchical, modular organisation of the personality network. The modularity score for the second level was -0.0087, whereas the third level achieved -0.0292.

Level 1: The 120 personality characteristics.

For the first level, two centrality measures were computed: effective degree and community centrality (Figure S2 and S3). The personality characteristics, “try not to think about the needy” (119), “have difficulty starting tasks” (115), and “remain calm under pressure” (116) showed a higher effective degree. This exposes the strong connections between these personality characteristics, thereby they not only have a high number of connections but also strong connections with the neighbouring personality characteristics in the network. Consequently, they may play an important role in the stability of the network.

In terms of community centrality, “am not bothered by difficult social situations” (106), “only feel comfortable with friends” (76), and “have a high opinion of myself” (84) were the characteristics with the highest values. Thus, these characteristics may have a high capacity to reach various other characteristics and could have an important impact on personality development.

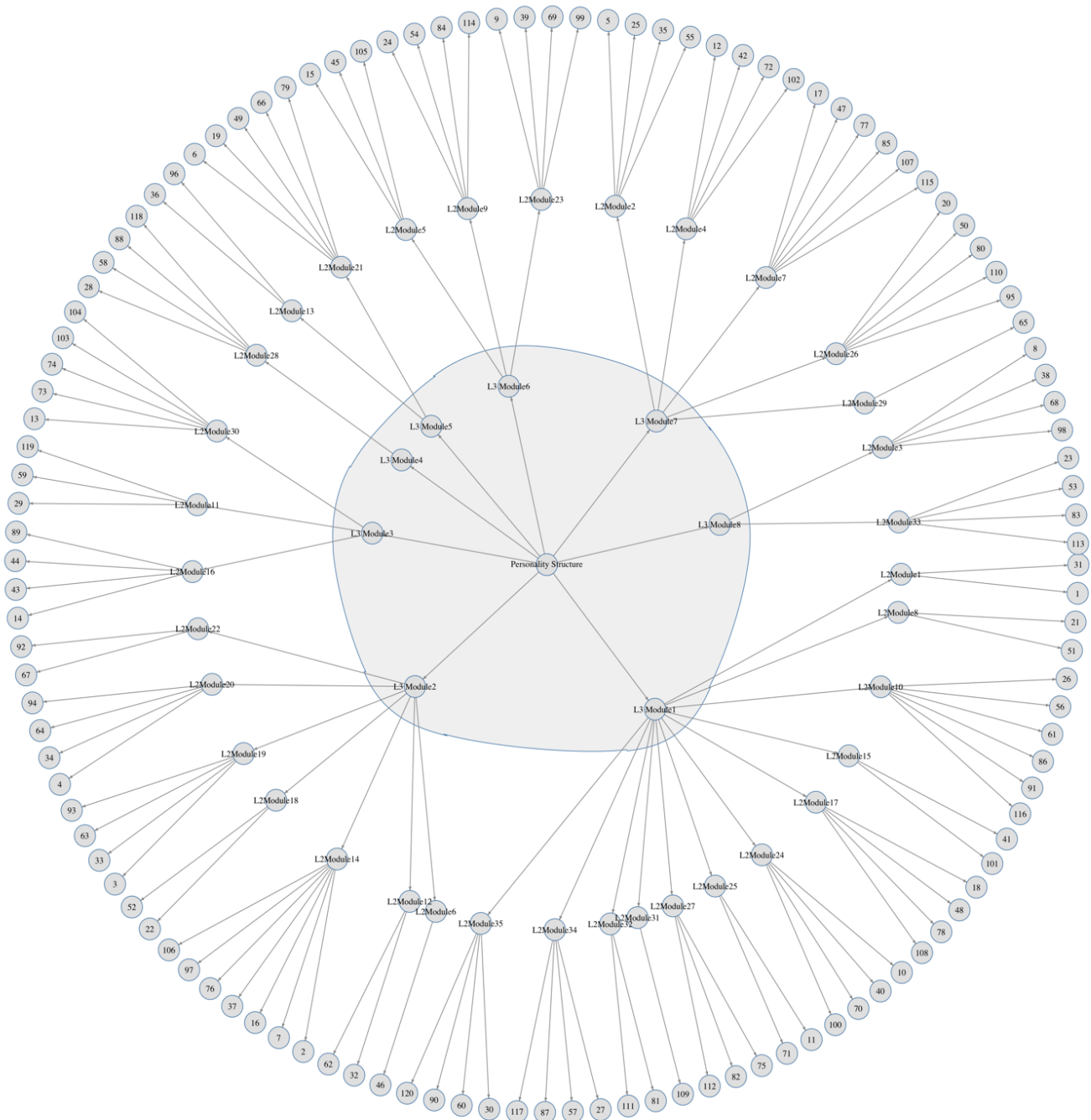


Figure 2. Hierarchical structure of personality. The outer ring represents the first hierarchical level with its 120 personality characteristics. The subsequent ring represents the 35 modules of the second hierarchical level. The third hierarchical level is represented by the central ring. The area surrounding the second level modules corresponds to the L3 Module 0. This representation of L3 Module 0 was implemented as none of the second level modules were primarily assigned to these modules.

Level 2: 35 Modules

In the second level, a network of 35 modules was identified (Figure S19). This is similar to the 30 facets proposed for the IPIP-NEO-120, with the main differences concerning splits in specific facets; for example, the depression facets “feel comfortable with myself” (101) and “dislike myself” (41) solely form module 15 and the items “often feel blue” (11) and “am often down in the dumps” (71) exclusively form module 25. A detailed description and comparison between the FFM facets and second level modules can be found in Table S1.

Regarding the centrality measures, the personality characteristics that revealed the highest values of modular bridgeness (Figure S9) were “believe that I’m better than others” (24), “radiate joy” (27), “love to read challenging material” (23), “like to take it easy” (107), and “am afraid to draw attention to myself” (46). These characteristics seem to be positioned between modules, playing a vital role in connecting the different modules of this level. At a facet level, therefore, these seem to be the most important characteristics in the connection of the facets due to their positioning. Moreover, the personality characteristic “am afraid to draw attention to myself” (46) also displayed a high value of modular overlap, along with the “am not bothered by difficult social situations” (106) and “like to take it easy” (107) personality characteristics. So, these characteristics seem to belong to various facets, and this might have important consequences for the development and stabilisation of the network. Interestingly, the items “am afraid to draw attention to myself” (46) and “like to take it easy” (107) showed high levels of both modular bridgeness and overlap. In this case, these two characteristics seem to be able to connect the modules due to their positioning in the networks and by belonging to various facets. The dual role of these characteristics might increase their importance in the development and stability of the personality network.

Concerning the modules, Figure S13 shows the modular overlap and bridgeness for each module in this hierarchical level. Modules 31, 22, and 2 tend to group the personality characteristics with higher overlap. It can also be seen that modules 6, 27, 22, and 18 tend to cluster the characteristics with higher bridgeness. Regarding community centrality (Figure S6), modules 14, 10, and 34 exhibited the higher values. In addition, module 14 also showed the higher values of effective degree, followed by modules 18 and 28 (Figure S10). In contrast, module 31 had the lowest values of effective degree and community centrality measures. These results point to a certain level of specialisation of the modules, with some modules being more closely related with trans-modular processes (i.e., grouping high overlapping characteristics) or to an intermodular role (i.e., grouping high bridgeness characteristics).

Level 3: 9 Modules

In the third hierarchical level, a 9-module network (Figure S20) was derived from the second level (Figure S19). In comparison with the FFM factors, only modules 8 and 4 were composed by personality characteristics of the same FFM factor, Openness to Experience. The remaining modules were all composed of personality characteristics from different factors. For example, module 1 had personality characteristics corresponding to all the FFM factors. Despite this, each module consistently demonstrated more personality characteristics of a given factor. For example, module 1, while possessing personality characteristics from each factor, has a higher number of personality characteristics from Neuroticism (see Table S1 for a detailed description of all the modules).

Regarding the centrality measures, the personality characteristics (Figure S4) “go on binges” (21), “easily resist temptations” (81), and “am able to control my cravings”

(111) showed the highest values of modular bridgeness. This means that, when looking at the personality characteristics that connect these 9 modules, these characteristics are those that are positioned between them. Consequently, these characteristics provide access to the different modules and might help develop high order ones, which, in turn, might provide access to the development of the specific second level modules (facets). The personality characteristics “easily resist temptations” (81), “rarely overindulge” (51), and “am able to control my cravings” (111) exhibited the highest values of modular overlap. These characteristics belong to more than one of the 9 modules in this level, meaning that these characteristics also connect the modules and due to their overlap might also contribute to their stability. Regarding the modules in this level, Figure S14 shows the modular bridgeness and overlap; clearly, modules 4, 6, and 8 tend to group the personality characteristics with the highest overlap. It can also be observed that the bridgeness values of the personality characteristics were broadly similar across the 9 modules, with modules 4 and 8 tending to group personality characteristics with slightly higher values of this measure. Across these 9 modules, the higher values of community centrality were on modules 1 and 2 (Figure S7), with modules 5, 3, and 8 revealing the highest values of effective degree (Figure S12). Despite this, none of these modules simultaneously had high values in both effective degree and community centrality measures. This could indicate that modules 1 and 2 are more associated with the development of the other modules and that modules 5, 3, and 8 are more associated with the stability of the high order level of the personality structure. The second level modules with the highest assignment values in the third level are illustrated in Table S1. Interestingly, none of the second level modules had the highest assignment value on module 0.

Discussion

We aimed to contribute to the clarification of the personality structure by proposing a hierarchical model and outlining the distinct functions for the different personality characteristics through network analysis and ModuLand framework. ModuLand allowed for the estimation and characterisation of the hierarchical modular structure of the personality network rooted on the theoretical grounds that have been proposed for psychological networks (Borsboom, 2017, Borsboom, Cramer, & Kalis, 2019). Although other cluster and hierarchy detection methods have been proposed in psychological networks, such as the Exploratory Graph Analysis (EGA; Golino & Epskamp, 2017) and the Minimal Spanning Tree (MST; Letina et al., 2019). There are several theoretical proposals that have been made in psychopathological and personality networks that aren't embodied in these methods, i.e., bridge and overlap personality characteristics (Jones, Ma, & McNally, 2019; Cramer et al., 2012). Moreover, the structure of personality is assumed to encompass 3 hierarchical levels (McCrae & John, 1992) and none of the cluster detection methods introduced in psychopathological and personality networks can estimate more than one hierarchical level. For example, in EGA the clustering method only allows for the estimation of one hierarchical level. Moreover, in EGA clusters are determined via the walktrap algorithm, which assumes that there are no overlapping features in the network (Golino & Epskamp, 2017). Even in other clustering methods introduced in psychological networks where the overlapping features can be estimated, e.g. Clique Percolation Method (Blaken et al., 2018), the hierarchical structure is limited to one level, which in personality research would be the equivalent to the facets level. In turn, in ModuLand the modules from one level become nodes in the next hierarchical level and their overlapping values in the previous level become the edges between the modules. This allows for the estimation of a hierarchical structure with

various levels, resulting in a hierarchical structure grounded in the overlapping features of the network.

Thus, to study the personality structure from a network perspective, from the proposed network theory of mental disorders and in the previous theoretical approaches in personality, methods like ModuLand allow for an integrative framework. With this in mind, we have opted for ModuLand in order to determine a personality structure that could accommodate these theoretical perspectives and provide an increased detail in its characterisation. ModuLand allows for a fine-grained characterisation of each characteristic and module that might shed some light in the mechanisms at play in the development and maintenance of the personality structure.

In the present paper, we propose that centrality measures might represent different roles for modules and for personality characteristics (see Results section), however, none of these roles have been evaluated and are solely derivations from the other fields of network science. Future research should focus on examining the centrality measures and their possible roles in the development and stability of the personality structure. We envision two ways of examining the proposed roles for the personality characteristics. First, as typically done in network science, simulated contagion might give us a first glimpse into the personality characteristics that are more prone to disseminate information across the network and, consequently, play an important role in the development of personality. We suggest characteristics with high bridgeness or overlap as an initial hypothesis. Second, we suggest that some characteristics might act as stabilisers and might be harder to change. In order to test this hypothesis, we suggest the deactivation of these characteristics and the analysis of the impact that this deactivation has on the network structure (for a detailed discussion on the role of centrality measures see the supplementary discussion in Supplementary Materials).

However, the results of this study should be interpreted carefully and considering some limitations. Cross-sectional data might not be suited to directly examine the hypothesis pertaining to the functions of the personality characteristics and modules. Additionally, the use of a set of items from a specific inventory (IPIP-NEO-120) might have constrained the correlational structure of the personality characteristics and the modular structure of the personality network. Moreover, although we have used the most up-to-date methods for psychological networks estimation, our network still has a high number of small edges that might have driven the modular structure found. As can be seen in the Table 1 and Figure S28, the modular structure of the split halves samples in the second level has a mean of 36 modules and the second level has a mean of 10 modules. And, although the network shows high robustness at this sample size (see Figures S25, S26 and S27 in the Supplementary Materials), the mean number of modules found was different for each hierarchical level from those obtained with the full sample. The large number of small edges might have driven these small differences in module detection between the full sample and the split halves. Moreover, to estimate the hierarchical structure with ModuLand algorithm, the negative edges can't be considered. While this might be an important limitation of the method used, a clear theoretical interpretation of these edges hasn't been put forward. These limitations call for caution on the interpretation of the results and more studies regarding the robustness of ModuLand algorithm in psychological networks are required. Consequently, future studies should, firstly, try to replicate these results, then to specifically test the hypothesis suggested for the different centrality measures. Furthermore, this is the first time that the ModuLand algorithm has been applied to the study of the personality structure and its replicability across other samples and direct comparison with other module detection algorithms remains to be seen.

Despite these limitations, the analysis of the hierarchical modular structure of the personality network is promising as it overcomes a number of the limitations observed in previous studies and promotes a modular perspective of the personality network (see also Goekoop, Goekoop, & Scholte, 2012), encompassing the inter-modularity and trans-modularity of the personality characteristics. Besides, our proposed framework can accommodate all the previous theoretical contributions made in the personality field and network theory for psychological phenomena, adding detail to the characterisation of the personality structure.

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CHAPTER II ^{1,2,3}

**The Differential Role of Central and Bridge Symptoms in Deactivating
Psychopathological Networks**

Note:

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² Supplementary materials of this paper are available in Chapter II of the Appendix.

³ The first and the second author contributed equally to the development of the paper.

Abstract

The network model of psychopathology suggests that central and bridge symptoms represent promising treatment targets because they may accelerate the deactivation of the network of interactions between the symptoms of mental disorders. However, the evidence confirming this hypothesis is scarce. This study reanalysed a convenience sample of 51 cross-sectional psychopathological networks, published in previous studies addressing diverse mental disorders or clinically relevant problems. In order to address the hypothesis that central and bridge symptoms are valuable treatment targets, this study simulated five distinct attack conditions on the psychopathological networks by deactivating symptoms based on two characteristics of central symptoms (degree and strength), two characteristics of bridge symptoms (overlap and bridgeness), and at random. The differential impact of these symptoms' characteristics was assessed in terms of the magnitude and the extent of the attack required to achieve maximum impact on the number of components, average path lengths and connectivity. Only moderate evidence to sustain the hypothesis that central and bridge symptoms constitute preferential treatment targets was obtained. The results suggest that degree, strength and bridgeness attack conditions are more effective than the random attack condition only in increasing the number of components of the psychopathological networks. The degree attack condition seemed to perform better than the strength, bridgeness and overlap attack conditions. Overlapping symptoms evidenced limited impact on the psychopathological networks. The need to address the basic mechanisms underlying the structure and dynamics of psychopathological networks through the expansion of the current methodological framework and its consolidation in more robust theories is stressed.

The Differential Role of Central and Bridge Symptoms in Deactivating Psychopathological Networks

Traditional models of psychopathology (e.g., categorical, and dimensional) continue to display a vast array of limitations, including a lack of explanation for high rates of comorbidity (Goldberg, 2015; Kessler, Chiu, Demler, & Walters, 2005) and the diversity of clinical presentations (Boschloo et al., 2015; Nuijten, Deserno, Cramer, & Borsboom, 2016). They have also been unable to explain the direct interactions between symptoms (Cramer & Borsboom, 2015), and offer poor support for the identification of etiopathogenic mechanisms and biomarkers of mental disorders (Fried & Nesse, 2015). In recent years, the network theory of mental disorders (Borsboom, 2017; Borsboom & Cramer, 2013; Borsboom, Cramer, & Kalis, 2019; Cramer, Waldorp, van der Maas, & Borsboom, 2010) has received increased attention as an alternative model that may overcome such persistent limitations. Instead of assuming that symptoms are the effects and measures of a latent dimension (psychological, genetic, neurophysiological or otherwise) at the origin of mental disorders, this perspective theorizes that mental disorders emerge from a complex network of causal interactions between symptoms (Borsboom, Cramer, & Kalis, 2019; Cramer, Waldorp, van der Maas, & Borsboom, 2010; Hofmann, Curtiss, & McNally, 2016). Figure 1.A. presents a typical illustration of these psychopathological networks. In it, the nodes (circles) represent symptoms and the edges (lines) connecting the nodes, the multiple interactions that constitute the causal structure at the origin of mental disorders (see Epskamp & Fried, 2018 for a brief discussion on the issue of causality in psychopathological networks). On this basis, it is hypothesised (Borsboom, 2017), and current evidence supports this hypothesis (Cramer, et al., 2016), that in an asymptomatic state this structure remains inactive, but as some symptoms are activated, for example by external events, this generates a cascading effect that spreads

through the network activating the other symptoms to which they are connected. As symptoms' activation spreads following the paths defined by the structure of interactions between symptoms, they become increasingly connected until the system transitions into a disease state in which the strongly connected network of symptoms sustains itself, even in the absence of the original activating event (Cramer, et al., 2016).

This perspective changes the focus from the syndromatic boundaries of mental disorders or their shared causes to the intricate interplay between symptoms. The inspection of the network of interactions between symptoms has resulted in new understandings of the nosography and comorbidity structures of different mental disorders like, for example, depression and anxiety (Beard, Millner, Forgeard, & Fried, 2016), complex posttraumatic stress disorder (Knefel, Karatzias, Ben-Ezra, Cloitre, Lueger-Schuster, & Maercker, 2019), or borderline personality disorder and bipolar disorder (Castro, Ferreira, Mendes, & Bento, 2018). To a significant extent, these advances have been made via the identification of central and bridge symptoms (e.g., Bekhuis, Schoevers, van Borkulo, Rosmalen, & Boschloo, 2016; Borsboom, 2017; Knefel, Tran, & Lueger-Schuster, 2016; Levinson et al., 2017; Marcus, Preszler, & Zeigler-Hill, 2018; Robinaugh, LeBlanc, Vuletich, & McNally, 2014; Smith et al., 2018).

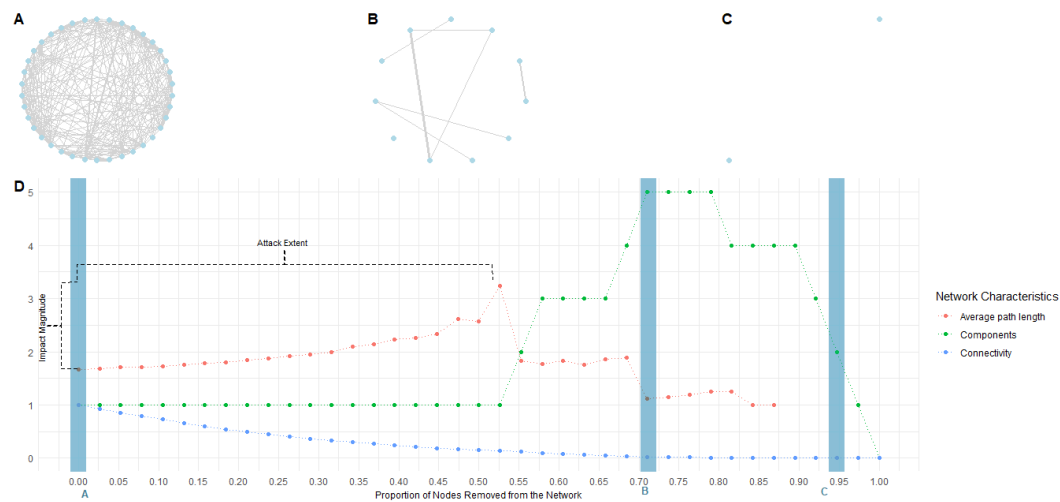


Figure 1. Illustrative example of network attack. Symptoms' networks are usually represented in a graph constituted by nodes representing symptoms (the blue circles in Figures 1.A., 1.B. and 1.C) and edges representing the interactions between symptoms (the grey lines in Figures 1.A., 1.B., and 1.C.; lines' widths represent the strength of each interaction). The complete graph represents the causal structure of the interactions between the symptoms. Its exploration provides information regarding the centrality of diverse symptoms (e.g., the number of interactions with other symptoms), as well as the network characteristics (e.g., the number of symptoms, or groups of symptoms, disconnected from the remaining). Figure 1.A. represents the original psychopathological networks. Under attack, the symptoms (nodes) were sequentially removed, one at a time, in the decreasing order of two centrality measures – (1) degree and (2) strength – and two bridging measures – (3) bridgeness and (4) overlap. Figures 1.B. and 1.C. represent the evolution of the initial network as symptoms are removed. At each point, network characteristics were measured (average path length, number of components, connectivity and diameter). The evolution of these measures throughout the attack is represented in Figure 1.D. The blue rectangles signal the moments to which the networks represented in Figures 1.A., 1.B. and 1.C. correspond. Finally, the attack extent and the magnitude of its impact were measured considering the number of symptoms that had to be removed to achieve peak average path length, number of components, connectivity and diameter, as well as the difference between peak and initial values.

Central and Bridge Symptoms

Central symptoms are defined and identified through a set of centrality measures, namely degree, strength, closeness and betweenness (Opsahl, Agneessens, Skvoretz, 2010). Degree centrality corresponds to the number of connections a symptom has with the other symptoms in a network and strength to the sum of the absolute weights of these connections (Epskamp, Borsboom, & Fried, 2018; Fried, van Borkulo, Cramer, Boschloo, Schoevers, & Borsboom, 2017). Closeness refers the average proximity of a given

symptom to all other symptoms and betweenness measures the number of times a symptom is on the shortest path between two other symptoms (Epskamp, Borsboom, & Fried, 2018). However, recent research has concluded that closeness and betweenness may be affected by sampling variability and spurious covariance between symptoms (Hallquist, Wright, & Molenaar, 2019), and are not always adequately stable (Epskamp, Borsboom, & Fried, 2018). It was also concluded that strength centrality might signal a symptom's association with a latent variable instead of its role in the network (Hallquist, Wright, & Molenaar, 2019). Moreover, the theoretical foundation for centrality measures in psychopathological networks is still limited (Bringmann, et al., 2018). This constrains the meaningful interpretation of high-scoring symptoms in these measures, and the determination of central symptoms remains mostly restricted to those identified via degree or strength centralities.

In turn, bridge symptoms are broadly defined as symptoms that connect different clusters of symptoms corresponding to different mental disorders or subgroups of symptoms within the same mental disorder. In the early stages of the field, Cramer and colleagues (2010) defined bridge symptoms as those symptoms that overlap perfectly between mental disorders, meaning that bridge symptoms are those that belong to the diagnostic criteria of distinct disorders. For example, fatigue belongs to the diagnostic criteria of major depression disorder and generalized anxiety disorder (American Psychiatric Association, 2013) and should therefore constitute a bridge symptom between these two disorders. Although this initial formulation, as well as subsequent studies that have utilised it (Afzali et al., 2017; Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011), continue to be generative, other conceptualisations of bridge symptoms have emerged. Some authors defined bridge symptoms as those that connect different disorders, regardless of any overlap between them (e.g., Levinson et al., 2017; Marcus et

al., 2018; McNally, Mair, Mugno, & Riemann, 2017; Robinaugh et al., 2014; Smith et al., 2018). For example, Levinson and colleagues (2017) suggested that symptoms related to physical sensations might constitute the bridge symptoms between bulimia and anxiety because they are the symptoms of anxiety that are connected to the symptoms of bulimia. These different conceptualisations imply two different kinds of bridge symptoms: (1) those that belong to two or more mental disorders (overlapping symptoms), and (2) those that belong to just one disorder, or alternatively are not specific symptoms of a disorder, but nevertheless still play an important role in connecting different disorders (bridging symptoms). Significantly, the conceptual distinction between overlapping and bridging symptoms has a correspondence in the alternative methodological approaches that have addressed the problem of identifying bridge symptoms. Advances in the identification of bridge symptoms have been achieved by exploring the concept of network communities or modules (e.g., McNally, Mair, Mugno, & Riemann, 2017). Network modules (Fortunato, 2010; Fortunato & Hric, 2016) are sets of symptoms that tend to interact more strongly and therefore exert a greater influence on each other than on the rest of the symptoms in the network. In psychopathological networks, these modules correspond to a mental disorder or to subgroups of symptoms within the same disorder. Jones, Ma, and McNally (2018) proposed a set of measures of the symptoms' bridge centrality, specifically designed to detect and quantify interacting symptoms between modules and found that deactivating symptoms based on their bridge strength was more effective in preventing symptoms activation from spreading than deactivating symptoms based on their strength or betweenness. This provides evidence in support of the theoretical proposal that bridge symptoms are implicated in the emergence of comorbidity structures between mental disorders (Cramer, Waldorp, van der Mass, & Borsboom, 2010). By assuming modules do not overlap, this method implies a concept of bridge symptoms that

is closer to the one of bridging symptoms. To date, studies characterizing the modular structure of psychopathological networks mostly followed this perspective as they typically involve module detection algorithms (for example, walktrapp, Price, Legrand, Brier, & Hébert-Dufresne, 2019; spin-glass, Birkeland & Heir, 2017) that result in non-overlapping modules. Blanken and colleagues (2018) have assumed an alternative perspective in exploring the usefulness of the Clique Percolation Method (CPM; mcseckmcsek, Palla, Farkas, Derényi, & Vicsek, 2006; Palla, Derényi, Farkas, & Vicsek, 2005) to detect overlapping modules in psychopathological networks. In the CPM, modules may share overlapping symptoms, which means that one symptom can belong simultaneously to more than one module (Blanken et al., 2018), and therefore it endorses a concept of bridge symptoms that coincides with the one of overlapping symptoms. On this basis, Blanken and colleagues (2018) illustrated how the symptoms that communicate between different modules may explain the diversity of possible pathways resulting the clinical heterogeneity of mental disorders.

Although differences between these two conceptualizations are not always apparent, preliminary evidence suggests that these types of bridge symptoms may perform different functions on psychopathological networks. In a previous study (Castro, Ferreira, Mendes, & Ferreira, 2018) on the modularity of the network of bipolar and borderline personality disorders, an alternative algorithm – ModuLand (Kovács, Palotai, Szalay, & Csermely, 2010) – was used to explore the comorbidity structure of these mental disorders and differentiate the symptoms functions on that structure. ModuLand also allows the modules to overlap and computes two different measures – modular overlap and modular bridgeness – each of which enables the identification of different types of bridge symptoms. Akin to Jones, Ma, and McNally (2018) proposal, modular bridgeness focus on the symptoms' inter-modular role and provides the effective number

of modules to which a symptom is connected (Szalay et al., 2012). In parallel with the proposal of Blanken and colleagues (2018), modular overlap focus on symptoms' trans-modular role and specifies the overlap of a given symptom between two or more modules relative to all other symptoms (Szalay et al., 2012). In that study, Castro, Ferreira, Mendes, & Ferreira (2018) found a moderate correlation between symptoms' strength centrality and modular bridgeness but not between strength centrality and modular overlap. This suggests that the distinction between bridging and overlapping symptoms may increase the conceptual clarity of the construct of bridge symptom and stimulate further theoretical refinement of the network theory of mental disorders.

Despite these initial observations, and the potential pathways that they open to develop further the network theory on mental disorders, more studies are needed that compare both types of bridge symptoms. In this case, an integrated framework, which provides a coherent rationale for the identification of both types of bridge symptoms is still required. Without it, the theoretical potential of this construct will remain limited. In order to contribute to consolidate an integrated methodological framework for the measurement of bridge symptoms, this study further explores the ModuLand algorithm (Kovács, Palotai, Szalay, & Csermely, 2010). Kovács and colleagues (2010) found ModuLand to be robust in characterizing the networks' modular structure over several benchmark networks representing different phenomena (social, biological, semantic), and to be more sensitive and also provide more detailed functional specificity than CPM.

Central and Bridge Symptoms as Psychotherapeutic Targets

As mentioned, central and bridge symptoms play an important part in the analysis of psychopathological networks and are on the basis of some of the most promising hypothesis pertaining the treatment of mental disorders that are currently emerging within

the field. From the network theory perspective, treatments of mental disorders exert some influence on the network of interactions between symptoms by deactivating symptoms, inhibiting their interactions, or removing external events that trigger activation cascades (see Borsboom, 2017; Isvoranu, Boyette, Guloksuz, & Borsboom, 2018). Irrespectively of the pathway through which treatments exert its influence, as they unfold, psychopathological networks are expected to follow a trajectory similar to the one depicted in Figures 1.A., 1.B., and 1.C. As represented in those figures, the inhibition of the interactions between symptoms should be associated with a general decrease in the networks' connectivity (number of interactions present in the network) due to the decrease in the number of active interactions. Similarly, symptoms' deactivation should also contribute to this decrease, since it also deactivates the interactions in which symptoms are involved. As networks' connections become sparser, the number of interactions needed to connect the symptoms that remain active is expected to increase (i.e., the pathways between them become longer). Finally, the decrease in networks' connectivity and the increase in the length of the pathways between symptoms should cause some symptoms to become isolated, and therefore be accompanied by an increase in the number of disconnected components. In addition to being theoretically coherent, this proposal is also consistent with routine clinical observations since recovered patients, by definition, report a minimal number of mild residual symptoms to no symptoms at all. It is therefore surprising that studies comparing the connectivity of psychopathological networks at treatment admission and discharge or follow-up have provided mixed evidence for a decrease in the networks' connectivity. Some studies revealed a pattern of networks' transformation consistent with the one described above, with decreased connectivity at discharge (and, in some samples, isolated symptoms; Snippe et al., 2017), but no statistically significant differences compared to admission (Snippe et al., 2017)

and placebo or wait-list control groups (Snippe et al., 2017) were found. A previous study also found higher connectivity in a group of poor-responders to treatment, but no statistically significant difference compared to a group of good-responders (Schweren, van Borkulo, Fried, & Goodyer, 2018). In addition, other studies observed an increase in connectivity from admission to discharge (Beard, et al., 2016; Bos et al., 2018). These studies have proposed a number of possible explanations (see also Fried, van Borkulo, Epskamp, Shoevers, Tuerlinckx, & Borsboom, 2016) related, for example, to the persistence of an underlying vulnerability to the development of mental disorders associated with networks' connectivity even after symptoms decrease (Snippe et al., 2017), naturalistic versus controlled (Schweren, van Borkulo, Fried, & Goodyer, 2018) or within versus between-subjects designs (Bos et al., 2018), and response bias arising from the repeated administration of symptoms measures and changes in the interpretation of measures' items as treatment unfolds (Bos et al., 2018; Fried, van Borkulo, Epskamp, Shoevers, Tuerlinckx, & Borsboom, 2016). Additionally, some of the previous studies (Beard, et al., 2016; Bos et al., 2018; Snippe et al., 2017) are based on complete samples at discharge. These samples are likely to include participants that did not respond to treatment, other participants that responded to treatment but did not recovered, participants that deteriorated, and also participants which recovered across treatment. Other studies (Schweren, van Borkulo, Fried, & Goodyer, 2018) are based on participants which responded to treatment but for whom it's unclear that they have recovered. Consequently, in some of these studies post-treatment samples displayed average symptoms severity within the clinically relevant realm (Beard, et al., 2016; Bos et al., 2018; Kraft, et al., 2019; Snippe et al., 2017), or minimal change in relation to admission (Kraft, et al., 2019), and studies analysing clearly recovered participants are still missing. The lack of distinction between different therapeutic trajectories may contribute to

explain the observation that despite no statistically significant differences are found in networks' connectivity from pre- to post-treatment, participants which networks display higher connectivity at admission also display a poorer response to treatment (Smith, et al., 2019). It is also increasing the recognition that these studies pose significant challenges (see Terluin, de Boer, & de Vet, 2016). Reports from recovered participants will necessarily be highly skewed and display a restriction of range which affects both network estimation due to deviations from normality (Epskamp, Borsboom, & Fried, 2018) and comparison due to the impact of differential variance on the networks' connectivity (Terluin, de Boer, & de Vet, 2016). Although the precise impact of these (and other) possible explanations remains to be further clarified, it was recognized that exploration of the assumption that higher connectivity characterizes psychopathological states is warranted (Schweren, van Borkulo, Fried, & Goodyer, 2018) and consistent with existing evidence (Snippe et al., 2017) suggesting that it is in fact associated with transition to a psychopathological state (Cramer, et al., 2016). Also, in natural settings, it was observed that higher connectivity of cross-sectional psychopathological networks is associated with symptoms persistence (compared to remittance; van Borkulo et al., 2015; van Rooijen, et al., 2018), and is higher in clinical populations compared to healthy populations (Santos, Fried, Asafu-Adjei, & Ruiz, 2017; Stone, DuBois, DeJesus, Rodgers, & Rizzo, 2017), as well as between individuals diagnosed with a mental disorder but displaying distinct clinical presentations (DuBois, Rodgers, Franko, Eddy, & Thomas, 2017) and course types (Koenders, et al., 2015). The analysis of the global connectivity of longitudinal dynamic networks has produced mixed results. Two studies concluded that the networks of individuals diagnosed with mental disorders displayed higher connectivity (Pe et al., 2015; Wichers, et al., 2016) but these results may be dependent on the methodological options during data pre-processing and network estimation (de Vos,

et al., 2017). Another study (Groen, et al., 2019) did not observe differences in the connectivity of the dynamic networks of individuals with persisting symptoms compared to individuals displaying symptoms remission. However, in this study (Groen, et al., 2019), differences between those groups were observed in the structure of interactions between symptoms (networks' topological structure) suggesting that the role of specific symptoms may be relevant beyond the global connectivity of the networks. This is consistent with the results of other studies which observed changes in the network structure but not in its global connectivity after a brief intervention in remitted symptoms (Kraft, et al., 2019) and underlined that treatments impact some specific symptoms (Blanken, et al., 2019; Mullarkey, Stein, Pearson, & Beevers, 2019). Similar results were obtained when comparing healthy individuals or community-based samples with individuals diagnosed with mental disorders or other medical conditions, both on dynamic (Curtiss, Fulford, Hoffmann, & Gershon, 2019) and cross-sectional (Hartung, Fried, Mehnert, Hinz, & Vehling, 2019; Montazeri, de Bildt, & Anderson, 2019; Silk, et al., 2019) networks; and also the cross-sectional networks of individuals displaying low versus high behavioral risk for medical conditions (Choi, Batchelder, Ehlinger, Safren, & O'Cleirigh, 2017). Together, these results suggest that the specific role each symptom plays within the network (as defined by the symptoms' centrality and bridgeness) may carry particular significance independently of the networks' global connectivity. This is consistent with an early hypothesis within this research field that central and bridge symptoms constitute priority therapeutic targets (Cramer, Waldorp, van der Maas, & Borsboom, 2010). Central symptoms were hypothesised to be responsible for maintaining mental disorders as they are involved in stronger interactions, or in the majority of interactions that constitute psychopathological networks (Borsboom & Cramer, 2013). For this reason, it has often been proposed that these symptoms can provide valuable

psychotherapeutic targets because they may accelerate the deactivation of the network and consequently catalyse treatments (e.g. Bekhuis et al., 2016; Borsboom & Cramer, 2013; Bryant, et al., 2017; Goldchmidt, et al., 2018; Knefel et al., 2016; McNally, Robinaugh, Wu, Wang, Deserno, & Borsboom, 2015; Martel, Levinson, Lee, & Smith, 2016; Montazeri, de Bildt, Dekker, & Anderson, 2019; Olatunji, Levinson, & Calebs, 2018; Richetin, Preti, Constantini, & De Panfilis, 2017; Robinaugh, Millner, & McNally, 2016; see also Fried et al., 2017 for a general overview). Simultaneously, bridge symptoms were hypothesised to be associated with the emergence of comorbidity structures (Borsboom, 2017; Cramer et al., 2010) that are known to hamper a treatment's progress (Kessler et al., 2005). Accordingly, as with central symptoms, bridge symptoms are considered important treatment targets because the deactivation of these symptoms might prevent the development of comorbidity between mental disorders (e.g. Afzali et al., 2017; Borsboom, 2017; Choi et al., 2017; Cramer et al., 2010; DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Garabiles, Lao, Xiong, & Hall, 2019; Jones et al., 2018; Levinson et al., 2017; Rouquette et al., 2018; Solmi, Collantoni, Meneguzzo, Tenconi, & Favaro, 2019). Studies that specifically addressed symptoms' centrality, noted that it predicts changes in the remaining symptoms (Robinaugh, Millner, & McNally, 2016), and facilitates evolution to a psychopathological condition (Boschloo, van Borkulo, Borsboom, & Schoevers, 2016), whereas others did not find evidence to support the hypothesis that symptoms' centrality, estimated from cross-sectional networks, is associated with changes in symptoms over time (Bos et al., 2017). To date, the study to most closely test this hypothesis was conducted by Rodebaugh and colleagues (2018), who explored whether central symptoms in a cross-sectional network of social anxiety disorder predicted changes in symptoms across treatment in another sample of individuals who undertook treatment for the same disorder. They found only moderate support for

this hypothesis, as the symptoms' centrality was not generalised across measures and the frequency of symptoms' endorsement also predicted change and was generalised across measures of social anxiety disorder.

Therefore, the initial hypothesis remains open and its evaluation will contribute to increase psychopathological networks' utility and validity for routine practice (see Contreras, Nieto, Valiente, Espinosa, & Vazquez, 2019). In this paper we aim to contribute to the clarification of the role of central and bridge symptoms in the deactivation of psychopathological networks and deepen current knowledge on the most effective ways to decrease their connectivity. We offer an alternative perspective by focusing on the actual impact of symptoms' deactivation. In order to deepen our comprehension of the mechanisms that promote the deactivation of psychopathological networks, this study simulates symptoms' deactivation by removing them from the network according to different regimes dependent on various centrality measures and their roles as bridge symptoms (for studies following similar modelling methods see Afzali et al., 2017; Jones, Ma, & McNally, 2019; Robinaugh, Millner, & McNally, 2016). As is typical of network science, we evaluated the impact of the deactivation of these symptoms on the global properties of the networks compared with the deactivation of random symptoms (e.g., Albert, Jeong, & Barabasi, 2000; Costa, Rodrigues, Travieso, & Villas Boas, 2007; Latora & Marchiori, 2001) in order to explore the differential efficacy of central and bridge symptoms in deactivating psychopathological networks. Since previous studies provide limited information to differentiate the impact of the different centrality measures, it is expected that the deactivation of the distinct types of central symptoms will display similar effects on psychopathological networks. Based on the previous observation that symptoms' bridgeness is more strongly correlated with their strength than overlapping (Castro, Ferreira, Mendes, & Ferreira, 2018), it is expected that

the deactivation of bridging symptoms will display a stronger impact on the network than overlapping symptoms.

Method

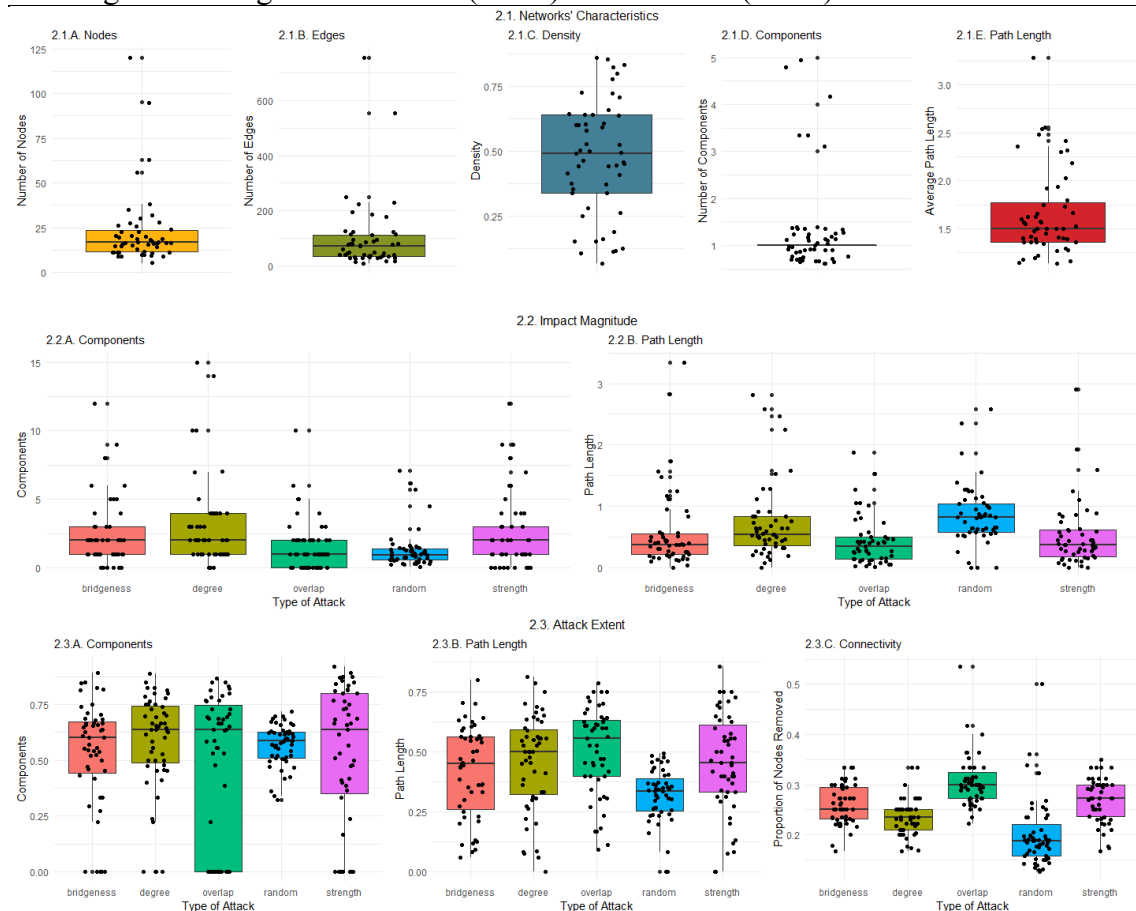
Networks

In order to constitute a sample of networks from the published literature, the databases PsychInfo, Web of Knowledge, Academic Search Complete, and Google Scholar were searched for studies addressing the cross-sectional networks of symptoms of mental disorders or other clinically relevant problems (e.g., hopelessness, alexithymia) using a combination of keywords including the names of mental disorders and “psychopathological networks” or “network analysis”. Studies addressing the personality structure or other psychological phenomena that are not directly related to mental disorders or clinically relevant problems were not included. Studies meeting the above criteria were checked for supporting data availability. An initial sample of 34 networks was collected. Seventeen additional networks were included, having been made available in a previous review by Halsbeck and Fried (2017). A convenience sample of 51 cross-sectional networks from 36 previous studies addressing mental disorders or clinically relevant problems was reanalysed. These networks and the original studies are presented in Table S1 of the Supplementary Materials. They are also identified with an asterisk in the reference list.

From the 51 networks, 19 (37.3%) pertained to clinical samples and the remainder to community-based samples; 18 (35.3%) included symptoms from different mental disorders or psychological problems. The networks’ descriptives are presented in Table 1 and the distributions of the networks’ characteristics are summarised in Figure 2.1.

Table 1*Networks' descriptives*

Network Characteristics	$M (SD)$	Networks ($N = 51$) $M_{trimmed} (SE)$	$Min - Max$
Nodes	22.725 (20.576)	17.419 (1.335)	5.000 – 120.000
Edges	99.392 (129.186)	65.645 (8.492)	8.000 – 756.000
Density	0.475 (0.221)	0.485 (0.035)	0.067 – .861
Components	1.333 (0.973)	1.000 (0.000)	1.000 – 5.000
Average Path Length	1.658 (0.454)	1.544 (0.053)	1.139 – 3.283

**Figure 2.** Boxplots summarising the distributions of the networks' characteristics, impact magnitude and attack extent.

Data Analyses

Originally, networks were estimated using correlation matrices (e.g., Goekoop & Goekoop, 2014; Koenders et al., 2015; Robinaugh et al., 2014), partial correlation matrices (e.g., Anderson, Montazeri, & de Bildt, 2015), PC algorithm (Ruzzano, Borsboom, & Geurts, 2015), Gaussian Graphical Models (GGM; e.g., Fried, Epskamp,

Nesse, Tuerlinckx, & Borsboom, 2016; Fried, et al., 2018), or Ising models (e.g., Boschloo et al., 2015; Kendler, Aggen, Flint, Borsboom, & Fried, 2017). Following current standards in psychopathological network estimation (Epskamp, Borsboom, & Fried, 2018), where raw data or correlation matrixes were available, networks were re-estimated using GGM (Epskamp & Fried, 2018) or Ising models (van Borkulo et al., 2014) in the case of continuous and binary data, respectively. GGM implemented in the qgraph package (version 1.5; Epskamp et al., 2012) for R (version 3.5.1; R Core Team, 2019) represents the most widely used method for estimating psychopathological networks from continuous data. This model estimates the partial correlation coefficients' networks (Epskamp, Waldorp, Mottus, & Borsboom, 2018). The Ising model is based on logistic regressions and is a commonly used procedure to estimate networks from binary data (van Borkulo et al., 2014). This model is implemented through the IsingFit package (version 0.3.1; van Borkulo et al., 2014) for R (version 3.5.1; R Core Team, 2019). Both methods use the Least Absolute Shrinkage and Selection Operator (LASSO) regularization technique (Tibshirani, 1996) in order to control for spurious interactions and the Extended Bayesian Information Criterion (EBIC; Chen & Chen, 2008) for model selection (see Epskamp, Borsboom, & Fried, 2018 for a detailed discussion of these methods for psychopathological networks estimation).

Where previous studies have analysed different networks estimated from the same sample (e.g., DuBois, Rodgers, Franko, Eddy, & Thomas, 2017; Santos, Fried, Asafu-Adjei, & Ruiz, 2017; Smith et al., 2018), only one was included in the current study. Analyses were based on the adjacency matrixes provided by the original authors or by Haslbeck and Fried (2017) when raw data or correlation matrixes were unavailable.

The graphical representation of the networks was performed using the qgraph package (version 1.5; Epskamp et al., 2012) for R (version 3.5.1; R Core Team, 2019).

The measures of symptoms' centrality, namely degree and strength, were also computed using the *qgraph* package. Degree corresponds to the number of connections a symptom has with the other symptoms. Strength corresponds to the sum of the weight of all connections from one symptom to the remainder. These measures were selected because they are consistently stable across studies, unlike other measures like betweenness and closeness, which are frequently unstable (Epskamp, Borsboom, & Fried, 2018).

The ModuLand algorithm (Szalay et al., 2012), implemented in Cytoscape 3.5.1 (Shannon et al., 2003), was used to measure the two types of bridge symptoms discussed above. ModuLand allocates each symptom to a module based on assignment values that represent how much a symptom belongs to each module. Thus, this framework permits the identification of modular overlap, which is a trans-modularity measure of the number of modules to which a symptom is assigned, and modular bridgeness, an inter-modularity measure of the effective number of modules to which a symptom is connected (Szalay et al., 2012).

In order to explore the characteristics of the deactivation of psychopathological networks, symptoms deactivation was simulated by removing them successively, one at a time, and the networks' characteristics were measured after the removal of each symptom (see Barabási, 2016; also see Albert, Jeong & Barabasi, 2000; Latora & Marchiori, 2001; Mishkovski, Biey & Kocarev, 2011 for examples of the same procedure in different domains of network science). The differential impact of the central and bridge symptoms was examined by simulating five attack conditions. Symptoms were removed by the decreasing order of (1) degree, (2) strength, (3) bridgeness, and (4) overlap. These were also compared to a fifth condition, in which symptoms were randomly removed from the network. This was performed using ProNet package (version 1.0.0; Wu & Xia, 2015) for R (version 3.5.1; R Core Team, 2019). The number of simulations for the

random attack was set at 2,000. At each time, three characteristics of the networks were measured: connectivity, components and average path length. The *igraph* package (version 1.2.2; Csárdi & Nepusz, 2006) for R (version 3.5.1; R Core Team, 2019) was used to compute the characteristics of the networks. Connectivity measures the sum of the number of interactions between all symptoms in the network. Components refer to portions (symptoms or groups of symptoms) of the network that are disconnected from the rest of the network. Finally, the average path length is the mean of the shortest paths in the network (Costa et al., 2007).

The impact generated by each attack condition was measured by computing the magnitude of the impact and the extent of the attack required to achieve maximum impact. First, we computed the difference between the average path length and number of components' maximum value and the initial value displayed by the networks. Second, the proportion of symptoms that had to be removed to achieve the maximum average path length and number of components was computed. Finally, the impact on the networks' connectivity was measured by computing the proportion of symptoms that needed to be removed to achieve a 50% drop in the networks' connectivity. This procedure is illustrated in Figure 1.

The analysis of the distributions of the characteristics of the networks, impact magnitude and attack extent (summarised in Figures 2.2 and 2.3) revealed high dispersion, extreme values and skewed distributions. For this reason, the robust bootstrap-t method proposed by Wilcox (2017; see also Field & Wilcox, 2017 for an introduction to this method) for comparing multiple dependent trimmed means was used to compare the effect of the attack conditions on the magnitude of the impact as well as the extent of the attack on the networks' number of components, average path length and connectivity. The robust test includes the test statistic and the critical value for the test statistic (at $\alpha =$

0.05). Robust post hoc tests display the difference between trimmed means (ψ_{ihat}), its bootstrap 95% confidence interval, the test statistics for this difference and the critical value for the test. ψ_{ihat} is negative if the trimmed mean for the first element in the comparison (for example, the trimmed mean number of components in the random versus degree attack conditions) is lower than the trimmed mean for the second element and positive if it is higher. The test is considered statistically significant ($p < 0.05$) if the test statistics exceeds the critical value. The amount of trim in computing trimmed means was set at 0.2, and 2,000 bootstrap samples were considered. R package WRS2 (version 0.10-0; Mair & Wilcox, 2018) was used to compute robust statistics. All data analysis on R was performed under RStudio 1.1.379 (RStudio Team, 2017). R-code is available from the contact author upon request.

Results

The extent of the attack and the magnitude of its impact under the different attack conditions are presented in Table 2. (The evolution of each network's characteristics under the different attack conditions is included in the Supplementary Materials.) The distributions of the attack extent and impact magnitude for the different attack conditions are summarised in Figures 2.2 and 2.3.

The robust bootstrap-t method for comparing multiple dependent means was conducted to compare the effect of the attack conditions on the magnitude of the impact on the networks' number of components, and average path length. A statistically significant difference was observed between the attack conditions on the trimmed mean impact on the number of components, $F_t = 14.514$, $F_{\text{crit}} = 2.499$, $p < .05$. The post-hoc comparisons between attack conditions, reported in Table 3, suggest that degree, strength and bridgeness attack conditions (but not the overlap attack) yielded a significantly higher

impact than the random attack on the number of components. No significant differences between these three conditions on the magnitude of the impact on the number of components were observed. Degree and bridgeness attack conditions also yielded a significantly higher impact than the overlap attack on the number of components. A statistically significant difference between attack conditions on the trimmed mean impact on the networks' average path length was also observed, $F_t = 28.790$, $F_{crit} = 2.387$, $p < .05$. The post-hoc comparisons between attack conditions, reported in Table 4, suggest that the random attack condition had a higher impact on the networks' average path length than the remaining attack conditions; moreover, degree attack had a stronger impact on the networks' average path length than the strength, bridgeness and overlap conditions.

The robust bootstrap-t method for comparing multiple dependent means was also conducted to compare the effect of the attack conditions on the extent of the attack required to achieve maximum impact on the networks' number of components, average path length and connectivity. A non-statistically significant difference was observed between the trimmed mean extent of the attack on the number of components between attack conditions, $F_t = 0.446$, $F_{crit} = 3.912$, $p > .05$. A statistically significant difference was observed between the trimmed mean extent of the attack on the average path length between attack conditions, $F_t = 17.752$, $F_{crit} = 2.511$, $p < .05$. The post-hoc comparisons between attack conditions, reported in Table 5, suggest that the random attack performs better than all of the remaining attack conditions in extending the average path length. A statistically significant difference between attack conditions on the trimmed mean extent of the attack on the networks' connectivity was observed, $F_t = 57.293$, $F_{crit} = 2.808$, $p < .05$. The post-hoc comparisons between attacks, reported in Table 6, suggest that the random attack condition performed better than any of the remaining attack conditions in reducing the networks' connectivity. They also suggest that degree attack performed

better than strength, bridgeness and overlap conditions, and that bridgeness attack better than overlap attack.

Table 2

Descriptives of impact magnitude and attack extent

Network Characteristics / Attack Condition	Impact Magnitude			Attack Extent		
	$M (SD)$	$M_{trimmed}$ (SE)	$Min -$ Max	$M (SD)$	$M_{trimmed}$ (SE)	$Min -$ Max
Components						
Random	1.300 (1.469)	0.926 (0.089)	0.060 – 7.096	0.568 (0.093)	0.577 (0.013)	0.323 – 0.718
Degree	3.098 (3.183)	2.290 (0.295)	0.000 – 15.000	0.587 (0.213)	0.623 (0.027)	0.000 – 0.889
Strength	2.471 (2.701)	1.806 (0.291)	0.000 – 12.000	0.523 (0.316)	0.582 (0.058)	0.000 – 0.921
Bridgeness	2.412 (2.401)	1.774 (0.198)	0.000 – 12.000	0.527 (0.245)	0.574 (0.032)	0.000 – 0.893
Overlap	1.608 (1.866)	1.194 (0.190)	0.000 – 10.000	0.493 (0.327)	0.545 (0.074)	0.000 – 0.868
Path Length						
Random	0.855 (0.502)	0.801 (0.054)	0.000 – 2.588	0.315 (0.119)	0.326 (0.016)	0.000 – 0.493
Degree	0.735 (0.629)	0.576 (0.052)	0.000 – 2.820	0.459 (0.200)	0.482 (0.031)	0.000 – 0.812
Strength	0.511 (0.521)	0.391 (0.052)	0.000 – 2.904	0.454 (0.208)	0.467 (0.031)	0.000 – 0.857
Bridgeness	0.575 (0.650)	0.382 (0.057)	0.001 – 3.345	0.420 (0.191)	0.436 (0.034)	0.059 – 0.800
Overlap	0.431 (0.398)	0.333 (0.041)	0.010 – 1.879	0.503 (0.182)	0.526 (0.028)	0.091 – 0.786
Connectivity						
Random	--	--	--	0.201 (0.067)	0.187 (0.007)	0.127 – 0.501
Degree	--	--	--	0.236 (0.036)	0.235 (0.005)	0.167 – 0.333
Strength	--	--	--	0.270 – 0.044	0.275 (0.007)	0.167 – 0.350
Bridgeness	--	--	--	0.260 (0.039)	0.259 (0.007)	0.167 – 0.333
Overlap	--	--	--	0.306 (0.051)	0.300 (0.006)	0.222 – 0.536

Table 3

Post-hoc comparisons of the magnitude of the impact on the number of components between attack conditions

Pairwise comparisons	<i>psihat</i>	95% CI	Test statistics	Critical value of the test
Random vs. Degree	-1.364	[-2.124, -0.604]	-5.624*	3.132
Random vs. Strength	-0.880	[-1.627, -0.134]	-3.694*	3.132
Random vs. Bridgeness	-0.848	[-1.330, -0.366]	-5.513*	3.132
Random vs. Overlap	-0.2671	[-0.787, 0.253]	-1.610	3.132
Degree vs. Strength	0.484	[-0.084, 1.052]	2.668	3.132
Degree vs. Bridgeness	0.516	[-0.015, 1.047]	3.044	3.132
Degree vs. Overlap	1.097	[0.373, 1.821]	4.745*	3.132
Strength vs. Bridgeness	0.032	[-0.572, 0.637]	0.167	3.132
Strength vs. Overlap	0.613	[-0.098, 1.324]	2.700	3.132
Bridgeness vs. Overlap	0.581	[0.022, 1.140]	3.250*	3.132

Note. *Statistically significant, $p < .05$.

Table 4

Post-hoc comparisons of the magnitude of the impact on the average path length between attack conditions

Pairwise comparisons	<i>psihat</i>	95% CI	Test statistics	Critical value of the test
Random vs. Degree	0.225	[0.079, 0.372]	4.346*	2.826
Random vs. Strength	0.410	[0.250, 0.570]	7.242*	2.826
Random vs. Bridgeness	0.419	[0.260, 0.579]	7.437*	2.826
Random vs. Overlap	0.468	[0.312, 0.625]	8.447*	2.826
Degree vs. Strength	0.185	[0.038, 0.332]	3.554*	2.826
Degree vs. Bridgeness	0.194	[0.065, 0.323]	4.237*	2.826
Degree vs. Overlap	0.243	[0.090, 0.396]	4.479*	2.826
Strength vs. Bridgeness	0.009	[-0.122, 0.140]	0.195	2.826

Strength vs. Overlap	0.058	[-0.078, 0.194]	1.202	2.826
Bridgeness vs. Overlap	0.049	[-0.068, 0.166]	1.186	2.826

Note. *Statistically significant, $p < .05$.

Table 5

Post-hoc comparisons of the extent of the attack on the networks' average path length between attack conditions

Pairwise comparisons	<i>psihat</i>	95% CI	Test statistics	Critical value of the test
Random vs. Degree	-0.155	[-0.215, -0.096]	-7.702*	2.970
Random vs. Strength	-0.141	[-0.204, -0.078]	-6.618*	2.970
Random vs. Bridgeness	-0.110	[-0.189, -0.030]	-4.111*	2.970
Random vs. Overlap	-0.200	[-0.270, -0.130]	-8.498*	2.970
Degree vs. Strength	0.015	[-0.052, 0.081]	0.655	2.970
Degree vs. Bridgeness	0.046	[-0.024, 0.115]	1.953	2.970
Degree vs. Overlap	-0.045	[-0.123, 0.034]	-1.686	2.970
Strength vs. Bridgeness	0.031	[-0.044, 0.106]	1.220	2.970
Strength vs. Overlap	-0.059	[-0.148, 0.030]	-1.972	2.970
Bridgeness vs. Overlap	-0.090	[-0.181, 0.001]	-2.943	2.970

Note. *Statistically significant, $p < .05$.

Table 6

Post-hoc comparisons of the extent of the attack on the networks' connectivity between attack conditions

Pairwise comparisons	<i>psihat</i>	95% CI	Test statistics	Critical value of the test
Random vs. Degree	-0.049	[-0.075, -0.022]	-5.506*	3.017
Random vs. Strength	-0.088	[-0.122, -0.054]	-7.894*	3.017

Random vs. Bridgeness	- 0.073	[-0.101, - 0.044]	-7.711*	3.017
Random vs. Overlap	- 0.114	[-0.135, - 0.092]	-16.200*	3.017
Degree vs. Strength	- 0.039	[-0.059, - 0.020]	-6.015*	3.017
Degree vs. Bridgeness	- 0.024	[-0.041, - 0.008]	-4.422*	3.017
Degree vs. Overlap	- 0.065	[-0.084, - 0.046]	-10.289*	3.017
Strength vs. Bridgeness	0.015	[-0.005, 0.035]	2.298	3.017
Strength vs. Overlap	- 0.026	[-0.054, 0.003]	-2.740	3.017
Bridgeness vs. Overlap	- 0.041	[-0.065, - 0.016]	-5.026*	3.017

Note. *Statistically significant, $p < .05$.

Discussion

The network theory of psychopathology has suggested that central and bridge symptoms might constitute priority therapeutic targets due to their ability to accelerate the deactivation of the network of interactions between symptoms (Borsboom, 2017; Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010). This hypothesis had a strong impact on the field and it is common for studies using cross-sectional networks in order to detail the nosographic structure of mental disorders, to conclude that the most central or bridge symptoms in those networks could constitute important therapeutic targets. However, there are few studies that directly tested this hypothesis and the actual impact to the psychopathological networks of manipulating central and bridge symptoms remains unclear. Mapping this impact will have important consequences for the field as it may support current innovations in diverse domains, like treatment personalization for example (see e.g. Fisher et al., 2019), and therefore contribute to increase the relevance of psychopathological networks to routine practice which has been recognized to remain limited (Contreras, Nieto, Valiente, Espinosa, & Vazquez, 2019; see also Bringmann, & Eronen, 2019). In order to contribute to this effort,

this study compared the impact of deactivating symptoms (as it is assumed to occur during treatment; Boorsboom, 2017) according to different conditions corresponding to the different definitions of bridge symptoms and the most common measures of symptoms centrality. Given that, in the case of bridge symptoms, different definitions coexist in the literature, we explored an alternative method for their identification based on the modular properties of the psychopathological networks. Through ModuLand (Kovács, Palotai, Szalay, & Csermely, 2010) it was possible to specify both the inter-modular and trans-modular roles that bridge symptoms have been proposed to play in psychopathological networks, which may offer a framework for future studies that directly compare these roles of bridge symptoms.

Globally, the results suggest that attacks based on bridge and central symptoms are less proficient than random attacks in transforming networks' path length and connectivity but tend (except for overlapping symptoms) to generate a higher number of isolated symptoms. They also suggest that symptoms with the highest degree are more efficient (than symptoms with the highest strength, bridgeness or overlap) in increasing the networks' paths length and in decreasing its connectivity. In contrast, overlapping symptoms revealed the smallest impact on the network. As in the previous studies (Rodebaugh, et al., 2018), these results provide only moderate evidence in support of the hypothesis that central and bridge symptoms constitute priority treatment targets and are consistent with the observation that common centrality measures (degree, strength, closeness, and betweenness) are only weakly correlated with symptoms' causal influence on the network (Dablander, & Hinne, 2018).

However, they also add to a small number of studies (Dablander, & Hinne, 2018; Robinaugh, Millner, & McNally, 2016) which converge in highlighting that distinct centrality measures perform differently and that there are measures of the networks'

operation and symptoms centrality, other than the ones most commonly used in the field, that constitute relevant alternatives. For example, psychopathological networks have been explored with emphasis placed on the global connectivity of the network, such as through comparisons between pre-treatment and post-treatment connectivity (Beard et al., 2016; Bos et al., 2018; Snippe, et al., 2017). Although results from these studies are not always consistent (e.g. Bos et al., 2018; van Borkulo et al., 2015), the suggestion remains that the connectivity of the network is the prime property for detecting changes in networks. This is grounded in strong theoretical assumptions (Borsboom, 2017) and empirical evidence (Cramer et al., 2016), but the results from this study suggest that other characteristics of psychopathological networks should be further explored since changes in the number of components appear to be easier to accomplish with targeted attacks (guided by symptoms degree, strength and bridgeness) and therefore this might represent a better indicator for detecting changes in psychopathological networks. Nonetheless, the number of components is an unexplored measure of psychopathological networks and the meaning of these changes does not currently have a theoretical foundation to enable us to derive a decisive conclusion regarding its importance for psychopathological networks. In fact, in most cases after a successful therapeutic process the remission of symptoms isn't total (see Rottenberg, Devendorf, Kashdan, & Disabato, 2018 for an example in depression) so it's fair to assume that these symptoms might be still forming separate components. Connectivity within these components might still be high so the change in connectivity is not particularly noticeable and an increase in number of components might be a better measure for therapeutic success.

Another example stems from the finding that attacks based on symptoms with higher overlap do not to produce significant changes in the networks' characteristics, in contrast to the other attack conditions. This is consistent with another study (Castro,

Ferreira, Mendes, & Ferreira, 2018) which reported a moderate correlation between symptoms' strength and bridgeness but not between strength and overlap. Although this suggests that overlapping symptoms may not be of special relevance to inform developments in the psychotherapeutic treatments based on psychopathological networks, since they do not seem to pinpoint a vulnerability of these networks, it does not exclude the hypothesis that they may play an important role in their constitution. In fact, recent studies have proposed methods for the identification of these symptoms on the basis that they might be more akin to the nature of psychopathological processes (Blanken et al., 2018). The two types of bridge symptoms may therefore play distinctive roles on the psychopathological networks and future research should aim to provide further knowledge on the roles of bridging and overlapping symptoms. It is reasonable to hypothesize, since symptoms' displaying higher modular bridgeness also seem to display stronger connections to other symptoms in the network, that bridging symptoms may be more involved in aggregating symptoms and maintaining the integrity of the network of interactions between them than the overlapping symptoms. If this is the case, then bridging symptoms may be implicated in networks' vulnerability to transition to a mental disorder state since vulnerability is associated with stronger connectivity (Cramer et al., 2016) and bridge symptoms may regulate the flux of information in psychopathological networks (Jones, Ma, & McNally, 2018). On the other hand, it was suggested that overlapping elements in complex networks, with few and weak links to other elements, but that connect nodes in distinct regions of the network, contribute to the networks' flexibility and adaptability (Csermely, 2008) which may help to explain the variability of mental disorders' clinical presentations and subtypes (e.g., Galatzer-Levy & Bryant, 2013; Fried & Nesse, 2015). If the different functions of bridging and overlapping symptoms are confirmed in future studies, it would add to that general hypothesis the

possibility that they contribute to the constitution of comorbidity structures through distinct mechanisms: bridging symptoms by connecting otherwise distinct modules from distant regions of the network and overlapping symptoms by creating a shared vulnerability for the synchronized activation of several modules.

The type of attack is another important aspect of this study. We have only computed the symptoms' centrality and bridge measures for the initial network and the order for the symptoms' removal was established based on this first estimation. This approach was used because most of the research identifies the central and bridge symptoms and proposes them as therapeutic targets based only on a single time-point estimation of the network. However, considering the particularities of psychopathological networks, such as the high density and small number of symptoms, this might not be the most adequate approach to model symptoms deactivation. In some cases, these characteristics of psychopathological networks result in the removal of a symptom without the ability to meaningfully affect the networks' characteristics. If all of the highest scoring symptoms have high degree values mostly due to the connections between them, the removal of these symptoms based on the first estimation will arrive at a point where the symptom being removed is no longer relevant for the network. Given that removing one symptom also removes the existing connections, with the symptoms being largely interconnected, their removal after the initial attack leaves the remaining network unaffected. In these cases, the random attack has a higher probability of removing a more connected symptom after the initial attack. In order to overcome this limitation future research should test other types of targeted attacks, such as cascading attacks (Motter & Lai, 2002; Wang & Rong, 2009). In a cascading attack, symptoms centrality is re-estimated after the deactivation of each symptom. Consequently, at each step, the symptom with highest centrality reflects the networks' state at that point and not the initial

state of the network. These attacks are more harmful to certain networks (Holme, Kim, Yoon, & Han, 2002) and, in light of this study's results, this may be a more appropriate strategy for exploring the vulnerability of the network.

Another limitation of this study is that it tested a restricted range of centrality measures. We have excluded two commonly used measures in psychopathological networks (betweenness and closeness) based on the lack of stability they have exhibited in psychopathological networks (Epskamp, Borsboom, & Fried, 2018) and opted for the inclusion of only the most frequently used centrality measures in the field. This was also done in favour of the results consistency and interpretability. However, other measures of symptom's impact have emerged within the field (Jones, 2018; Robinaugh, Millner, & McNally, 2016) and network science offers a vast array of centrality measures (e.g. Wang, Huang, Xu, & Xiao, 2014) that might be generative in the study of the vulnerability of psychopathological networks but remain unexplored. This suggestion to use alternative centrality measures has recently been proposed by Bringmann and colleagues (2018). It is consistent with the findings that alternative centrality measures (e.g., eigenvector centrality) reveal higher correlations with the symptoms' causal role than the most common ones (Dablander, & Hinne, 2018), and that these common measures may not adequately account for some of the characteristics of psychopathological networks (e.g., the negative weights of some of the connections between symptoms; Robinaugh, Millner, & McNally, 2016). The examination of alternative measures is necessary for the continued development of the network theory of psychopathology, but it should not be done in a random way. Evidence from other fields suggests that the nature of the network dictates its specific vulnerability to different types of attack (Holme, Kim, Yoon, & Han, 2002) and the most relevant centrality measures (Borgatti, 2005). An initial study (Borsboom, Cramer, Schmittmann, Epskamp, &

Waldorp, 2011) explored the topological structure of the network of the DSM-IV (APA, 1994) symptoms concluding that it is consistent with the characteristics of a general type of complex networks, named small-world networks (Watts & Strogatz, 1998), meaning that most symptoms, even those from traditionally considered distant mental disorders, are connected by short paths and this was offered as an explanation to the pervasiveness of comorbidity between mental disorders and the unsuccessful search for causal mechanisms and biomarkers. No further attempts were made to replicate this study and most of the recent networks in the field refer to smaller sets of symptoms pertaining to specific mental disorders. Therefore, the characteristics of the topological structure of psychopathological network remain largely unknown. As these characteristics, as well as the specific processes unfolding within the network, determine the most relevant measures of its operation (Borgatti, 2005), increased efforts to clarify the characteristics of the topological structure of psychopathological networks could guide the principled identification of the most relevant centrality measures for psychopathological networks. It could also contribute to the study of the vulnerability of psychopathological networks and advance the identification of the characteristics that may more productively be explored by psychotherapeutic treatments.

It should also be noted that all of the networks used in this study were based on cross-sectional data, which has been pointed out as a limitation in the identification of central symptoms (van Borkulo et al., 2015; Robinaugh, LeBlan, Vuletich, & McNally, 2014; Fried et al., 2015). In fact, previous studies found differences in the central symptoms between cross-sectional and longitudinal networks (e.g., Bos et al., 2017; see also Contreras, Nieto, Valiente, Espinosa, & Vazquez, 2019 for a review). In this study, cross-sectional networks were used because, to date, research suggesting central and

bridge symptoms are important therapeutic targets are mostly based on cross-sectional networks. However, replication studies based on longitudinal networks are needed.

Finally, this study examined a convenience sample of previously published data sets that encompass different mental disorders. Its findings aggregate all of the networks, potentially resulting in the omission of specific mechanisms associated with the differential vulnerability of distinct mental disorders. Whether the psychopathological networks characterizing different mental disorders have different characteristics and therefore display different vulnerabilities remains unknown.

In conclusion, central and bridging symptoms were more effective than randomly selected symptoms in increasing the number of components of psychopathological networks but not in decreasing their connectivity and increasing their average path length. Overlapping symptoms displayed the smallest impact on the networks. These results provide only partial support for the initial hypothesis that central and bridge symptoms are therapeutically relevant. They also suggest that caution is advised when concluding that central and bridge symptoms are preferential treatment targets based solely on a single time-point identification of these symptoms and that this might not be the most prolific road for network-guided interventions. Consequently, the identification of treatment targets remains an open problem in the field and an important pathway for future research. These findings add to the large body of research highlighting the need to explore new methodological frameworks that may enable a more fine-grained perspective of the mechanisms responsible for the development of psychopathological networks. Moreover, these alternative methodological frameworks will contribute to further develop previous theoretical assumptions and hypothesis and open new pathways for future research since a significant amount of network properties and centrality measures from other fields of network science are already available. Their examination might

contribute to deepening the knowledge about the behaviour of psychopathological networks.

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Chapter III ^{1,2}

The Modular Structure of Posttraumatic Stress Disorder in Adolescents

Note:

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² Supplementary materials of this paper are available in Chapter III of the Appendix.

Abstract

The nosographic structure of the symptoms of posttraumatic stress disorder (PTSD) remains unclear. Previous studies which performed factor analysis of these symptoms in adults found several distinct models. These inconsistencies have been transported to the analysis of the symptoms of PTSD in adolescents and are amplified by the different expressions that these symptoms have in adolescents in comparison to adults. The network perspective of mental disorders is a promising approach to clarifying the structure of PTSD. This study aims to identify the hierarchical modular structure of the network of PTSD symptoms in adolescents and the role of the different symptoms in that network. A sample collected in a previous study (Guo, Xue, Shao, Long, & Cao, 2015) of 662 adolescents ($M_{\text{age}} = 13.05$, $SD_{\text{age}} = 0.83$) exposed to a pipeline explosion was reanalyzed. The PTSD Checklist-Civilian Version (PCL-C) was used to assess symptoms of PTSD. The network of symptoms was estimated with a non-regularized method and the identification and characterization of the modular structure and the role of symptoms was performed using the ModuLand algorithm. A structure of two levels was found with a first level with 17 symptoms and five high-order modules. The module encompassing the reexperiencing symptoms showed the highest centrality values. Symptoms transversal to various disorders have high values of overlap. Our findings suggest that a modular structure encompassing five modules captures the best representation of the PTSD structure in adolescents.

The Modular Structure of Posttraumatic Stress Disorder in Adolescents

Over the years, the nosographic structure of the symptoms of posttraumatic stress disorder (PTSD) has remained elusive (Lee et al., 2019), and this uncertainty about its organization extends to younger age groups (Russell et al., 2017). Since its introduction in the third version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980) and its subsequent revisions (Bartels et al., 2019), considerable disagreement has emerged about the conceptualization (McNally, 2012), number (Cao, Wang, Cao, Zhang, & Elhai, 2017) and organization (Russell et al., 2017) of PTSD symptoms. In addition, the structure of PTSD has been mostly examined through factor analysis methods (Marshall, Schell, & Miles, 2013) which have revealed different factor structures (Armour, Carragher, & Elhai, 2013), ranging between one to seven factors (Armour, Mullerová, & Elhai, 2016; Table S1 in Supplementary Materials).

In children and adolescents exposed to traumatic and stressful events, research has only recently begun to address the nosography of PTSD symptoms (Saul, Grant, & Carter, 2008) and their impact on development and functioning in later stages of life (Kolaitis, 2017). To a significant extent, this has been achieved through studies based on factor analysis; however contradictory results have been found (Cao et al., 2017). For example, Ayer and colleagues (2011a) performed a confirmatory factor analysis in which they compared eight PTSD models in two samples of adolescents and found that all the proposals provided an adequate fit. In turn, Elhai and colleagues (2009) compared the two four-factor structures of PTSD (i.e., King, King, Leskin, & Weathers, 1998; Simms, Watson, & Doebbeling, 2002; Table S1 in Supplementary Materials) in an epidemiological sample of adolescents and concluded that both proposals were suitable, with only slight differences favoring the model of Simms and colleagues (2002). However, in another study, the Dysphoric Arousal Model (Elhai & Palmeiri, 2011) was

found to display a slightly better fit (Wang et al., 2011) and considerable evidence has shown the suitability of this model in the study of the nosology in adolescents (e.g., Armour et al., 2013; Bennett, Kerig, Chaplo, McGee, & Baucom, 2014; Sumner, Pietrzack, Danielson, Adams, & Ruggiero, 2014; Wang et al., 2013; Wang, Long, Li, & Armour, 2011).

This particular model is constituted by five factors (i.e., reexperience, avoidance, numbing, dysphoric, and arousal) and is similar with the four-factor proposal of King and colleagues (1998). It distinguishes itself from the previous proposal by splitting the numbing factor into a dysphoric and an anxious arousal factor. Thus, the symptoms, sleep problems, irritability, and concentration problems, characterize the dysphoric factor, and the symptoms, hypervigilance and startle response characterize the anxious arousal factor (Contractor et al., 2003). Essentially, this division is related to the fact that the dysphoric factor, as described in King's model, did not allow the distinction of symptoms of fear-based disorders (i.e., anxious arousal in the five-factor proposal), from symptoms related to mood responses (i.e., dysphoria-arousal in the five-factor proposal; Cao et al., 2017).

Even though the Dysphoric Arousal Model has been extensively supported (e.g., Wang et al., 2013; Wang et al., 2013), the nosographic structure of PTSD in children and adolescents has been continuously assessed with factor models developed to uncover the nosography of PTSD in adults (Wang et al., 2013). In parallel with the controversy concerning the nosography of PTSD among adults (Wang et al., 2011), the application of these models to young populations might not be adequate (Ayer et al., 2011b). One of the main limitations of the use of models of PTSD developed for adults to characterize children and adolescents' PTSD structure is the lack of a developmentally informed perspective (Gregorowski & Seedat, 2013), which might be crucial in these age groups (Scheeringa, Zeanah, & Cohen, 2011). This might constitute a possible explanation for

the contradictory results found in previous literature (Denton, Frogley, Jackson, John, & Querstret, 2017).

It has been recognized that maturational, cognitive, and affective processes, as well as earlier experiences of life, influence the acquisition and development of coping mechanisms and emotion regulation strategies in youth (Nkyi, 2015; Pynoos et al., 2009). These are important aspects that influence the course of PTSD (Olf, Langeland, Draijer, & Gersons, 2007). Particularly in adolescents, the development of PTSD has been associated with earlier developmental experiences, in which they felt that their needs and perceptions of safety given by significant others were not corresponding (Pynoos et al., 2009). Consequently, this could predispose them to negative cognitive appraisals (Mitchell, Brennan, Curran, Hanna, & Dayer, 2017) and to the engagement in high-risk behaviours (Pynoos et al., 2009) such as delinquent and aggressive behaviours and social withdrawal (Becker & Kerig, 2011; Saigh, Yasik, Oberfield, Halamandaris, & McHugh, 2002). Nevertheless, often these behaviours are interpreted in the context of other disorders (e.g., conduct problems, personality disorders) (Pynoos et al., 2009). Therefore, the clarification of the nosographic structure of PTSD in young ages would be of extreme importance and help to design models that can better capture the particular features of children and adolescents with PTSD.

In fact, although research in these age groups is still scarce (Bennett et al., 2014), previous studies have shown that symptoms of PTSD are expressed differently in children and adolescents when compared with adult samples (Sachser et al., 2018; Saul et al., 2008). For example, children frequently display symptoms of PTSD through re-enactment in play or in drawings (Broman-Fulks et al., 2009), while adolescents are more prone to difficulties in emotional processing and cognitive functioning and have a higher vulnerability of engaging in aggressive behaviours and substance use (Doric et al., 2019;

Sumner et al., 2014). Moreover, previous studies found that specific traumatic experiences might be associated with distinct symptomatic expressions (Kessler et al., 2017), and that this extends to children and adolescents (Bennett et al., 2014). Therefore, this supports the notion that changes in the structure of PTSD symptoms might occur over the course of life (Broman-Fulks et al., 2009).

Altogether, this suggests that it is necessary to explore the structure of PTSD at earlier ages, taking into consideration a developmental perspective, and capturing its specific affective, cognitive, and behavioral expressions. It also suggests the need to study the structure of PTSD in children and adolescents with different empirical methods. Essentially, this knowledge would provide a new pathway to uncover the core symptoms of PTSD, further enabling the comprehension of the etiopathogenic and maintenance mechanisms underlying this condition, as well providing information about potential relationships between clusters of symptoms (Elhai & Palmeri, 2011). Subsequently, this would help in the development of more accurate diagnostic criteria and assessment instruments, as well as supporting the caregivers with the ultimate goal of providing an adequate treatment consistent with the needs of this population (Kolaitis, 2017; Pynoos et al., 2009).

In this context, network theory emerges as a promising approach to surpass these limitations (Borsboom, 2017). In psychopathology, this perspective conceptualizes mental disorders as complex systems in which the symptoms interact with each other (Borsboom & Cramer, 2013). According to this framework, PTSD is the result of a network of connections between a set of symptoms (conceptualized as nodes in that network) and the associations among them (which are conceptualized as the network's edges; McNally et al., 2014). This approach focuses on this network of connections between symptoms, resulting in important insights into the nosographic structure of

mental disorders (Curtiss & Klemanski, 2016), such as central and bridge symptoms (Borsboom, 2017). Thus, identification of central and bridge symptoms in the network have been the main route to ascertaining the nosographic structure of mental disorders (e.g., Levinson et al., 2017; Smith et al., 2019).

Central symptoms are conceptualized as the nodes that exhibit the strongest connections with other symptoms, thereby exerting a significant influence in the structure of the network (Epskamp, Borsboom, & Fried, 2018). These symptoms have been identified through diverse centrality indices that usually focus on the number and strength of the connections of a symptom with the remaining symptoms in the network (Opsahl, Agneessens, & Skvoretz, 2010). In turn, bridge symptoms are probably implicated in the development of the structure of a mental disorder since they may act as information spreaders between different clusters of symptoms (Blanken et al., 2018) and/or on the emergence of comorbidity structures since they might play a role in the connections between different disorders (Cramer, Waldorp, Van der Maas, & Borsboom, 2010).

These symptoms have been classified into two subtypes: overlapping and bridging symptoms. Overlapping symptoms refer to symptoms that belong to multiple clusters of symptoms or disorders (Blanken et al., 2018; Borsboom & Cramer, 2013). On the other hand, bridging symptoms are positioned between clusters of symptoms or different disorders, playing a role in the interconnection of the network (Jones, Ma, & McNally, 2019). The identification of these symptoms has been obtained through the examination of the network of communities or modules (Kovács, Palotai, Szalay, & Csermely, 2010). A community or module consists of a set of nodes that have the highest connections between themselves (Fortunato & Hric, 2016). In this context, in psychopathological networks, modules are sets of symptoms that show the stronger relationships between them; these sets of symptoms might represent a disorder or specific subgroups within a

mental condition (Blanken et al., 2018). Hence, the identification of these subsets of symptoms and, consequently, the identification of central and bridge symptoms could provide valuable insights about the structural organization of mental disorders.

Previous studies of the network of PTSD symptoms were predominantly performed in adult samples and revealed different central symptoms (Ross et al., 2018). For example, in a study by Birkeland and Heir (2017), emotional numbing was identified as the central symptom; in contradiction to these results, McNally and colleagues (2014) found hypervigilance and concentration problems to be the ones with highest centrality. This problem extends to the PTSD network literature in adolescents (i.e., Bartels et al., 2019; Cao et al., 2018; De Haan et al., 2020; Pfeiffer et al., 2019; Ross et al., 2020; Russell et al., 2017; Yung, Li, Zhang & Zhang, 2019). Although some similarities in the central symptoms were observable between some studies (for example, intrusion was one of the central symptoms identified in both Russell and colleagues [2017] and Cao and colleagues [2018]), several inconsistencies also emerged (emotional numbing, happiness/love valence, and detachment or flashbacks were specific to the studies of Russell and colleagues [2017] and Cao and colleagues [2018], respectively). Additionally, Bartels and colleagues (2019) and De Haan and colleagues (2020) found as central symptoms negative trauma related emotions and persistent negative emotional state and physiological and emotional reactivity, respectively. It is important to note that the differences between the studies of Russell and colleagues (2017) and Cao and colleagues (2018) could be related to the use of different measures and versions of DSM. Moreover, these studies did not focus on the identification of the different types of bridge symptoms, which could carry valuable information about the symptoms structure of PTSD. Here, the identification of modules seems to be a potential pathway to uncover how the network of PTSD is organized and to identify different roles for each symptom

(i.e., the symptoms responsible for connecting the different modules and the symptoms that belong to more than one module; Blanken et al., 2018).

Moreover, previous studies on the structure of PTSD in adolescents did not explore the structure of this disorder through communities or modules detection methods. Some of these methods (i.e., spinglass, walktrap) have been used in the examination of the structure of PTSD in adult samples (i.e., Birkeland & Heir, 2017) and enabled a more fine-grained view of the organization of the symptoms of PTSD. Despite this, the community detection algorithms previously used do not incorporate, in the same framework, a method that is able identify both bridge and overlapping symptoms. The application of hierarchical algorithms could address this problem.

In the context of psychological networks, the use of hierarchical algorithms could provide a detailed view of the symptoms' connections; specifically, a clear identification of the bridge and overlapping symptoms derived from a single unified framework. This is important since these symptoms have been recognized as the symptoms that are responsible for the development and maintenance of the disorder (Blanken et al., 2018). To our knowledge, the framework in the psychology field that enabled an approximation of a hierarchical perspective is the Minimum Spanning Tree method (MST; Mantegna, 1999). However, this framework does not contain measures to identify and characterize bridge and overlapping symptoms. On the other hand, ModuLand (Kovács et al., 2010) is a framework that has been used to evaluate the hierarchical modular structure across several areas (i.e., protein, biological, psychological; Castro et al., 2018; Castro et al., 2019; Sandhu et al., 2012; Szalay-Beko et al., 2012), essentially, because it allows the estimation of these two types of bridge symptoms (i.e., bridge and overlapping), as well as information regarding the characteristics of the modules. Therefore, the use of this method in this age group would allow a deeper comprehension of the nosographic

structure of PTSD, and improve the diagnosis of adolescent PTSD, assessment instruments, and prevention and treatment strategies (Armour et al., 2016; Kolaitis, 2017).

In order to accomplish this, our study aimed to (1) identify the hierarchical modular structure in a sample of traumatized adolescents; (2) characterize the modules within the network; and (3) pinpoint central and bridge symptoms within the modular structure.

Method

Participants

Data from a previous study (for a detailed description, see Guo et al., 2015) that explored the relationship between severity of trauma and neuroticism in the development of symptoms of PTSD was analyzed. The sample was comprised of 662 Chinese adolescents ($M_{age} = 13.05$, $SD_{age} = 0.83$, 55.1% male) exposed to a pipeline explosion in China in November of 2013. The original study was approved by the Research Ethics Committee of Shandong University School of Nursing. All the participants and their parents provided written informed consent.

In the present study, only the data pertaining to PTSD symptoms was analyzed. For a detailed description of the participants' characteristics see Table 1.

Table 1*Sociodemographic Characteristics of Participants*

		<i>n (%)</i>	<i>M (SD)</i>
Age			13.05(0.83)
Gender	Male	365(55.1)	
	Female	297(44.9)	
Grade	7th	371(56.0)	
	8th	291(44.0)	
Types of trauma	Injury/death of a family member	17(2.6)	
	Injury/death of friends	115(17.4)	
	Witnessed injury/death	520(78.5)	
	House damage	68(10.7)	
	Property loss	47(7.1)	

Measures

The PTSD Checklist-Civilian Version (PCL-C; Weathers, Litz, Herman, Huska, & Keane, 1993) is a 17-item self-report measure developed to assess the presence of PTSD symptoms according to the DSM. This measure was developed for adults, although it has been consistently used to assess symptoms of PTSD in adolescents (e.g., Jin, Liu, & Guan, 2014; Radcliffe et al., 2007; Tian, Wong, Li, & Jiang, 2014). The PCL-C Chinese version was used in the original study to evaluate the symptoms of PTSD in adolescents. This measure has acceptable internal consistency ($\alpha = .82$; Xiaoyun, Hongai, & Qigui, 2007). This study also showed a good internal consistency ($\alpha = .93$, 95% *CI* [.92, .95]).

Estimation and Analysis of the Adolescent PTSD Symptoms Network

The adolescent PTSD symptoms network was estimated using non-regularized methods. These methods are based on multiple regression and non-parametric procedures and have been shown to be suitable to estimate the structure of psychopathological networks accurately since they control for false positives and ensure high specificity (Williams et al., 2019). In the present study, non-regularized methods were used since they achieve a higher specificity and a low false positive rate in comparison with traditional methods (Epskamp et al., 2018). This method was implemented in the GGMnonreg package (version 1.0.0; for a detailed description see Williams et al., 2019) for R (R Core Team, 2019).

The R (R Core Team, 2019) package qgraph (version 1.5; Epskamp et al., 2012) was used for the graphical representation of the network and to compute the strength centrality index. This measure estimates the sum of the weighted connections of a symptom to all other symptoms in the network (Opsahl et al., 2010).

Moreover, the density coefficient of the network was estimated with the R package igraph (version 1.2.4.1; Csárdi & Nepusz, 2006; R Core Team, 2019). The density coefficient is a propriety of the network that corresponds to the proportion of the number of edges regarding the possible number of edges in the network (Epskamp et al., 2018).

Hierarchical Modular Structure of the Adolescent PTSD Symptoms Network

The identification and characterization of the hierarchical modular structure of the adolescent PTSD symptoms network was performed using the ModuLand algorithm (Kovács et al., 2010) implemented in Cytoscape 3.5.1 (Shannon et al., 2003). This framework estimates the hierarchical modular structure through the application of an

established set of parameters. This way, through Linkland function, ModuLand recognizes the influence zone (i.e., the subgraph of the network that contains all the edges indirectly affected by a given edge) of the edges in the network. Then, for each edge, community centrality values are estimated. This measure sums up all the influence zones of a given edge and also provides the community centrality values for each node (i.e., symptom). All nodes are then represented in a landscape where the nodes with the highest values in community centrality form hills. The number of hilltops represents the number of modules. The intersection between the hills determines the overlapping values for each node. Sequentially, the hierarchical structure is constructed based on the modules identified in the previous level, which become meta-nodes in the next hierarchical level. The edges strength between these meta-nodes are determined by the values of the overlap between the modules (i.e., modules with higher overlap will have a stronger edge connecting them). This procedure is repeated until only one meta-node prevails (for a more detailed description on the modules estimation, see Kóvacs et al., 2010).

Additionally, this framework provides a group of measures that enables the characterization of the different functions of symptoms and modules of the adolescent PTSD symptoms network. Within these measures, modular bridgeness and modular overlap were used to identify bridge and overlap symptoms. The first measure assesses the intermodularity of a given symptom; in other words, the effective number of modules to which a given node (i.e., symptom) is connected. The second measure evaluates the transmodularity of a given symptom, representing the effective number of modules to which a symptom belongs (Kovács et al., 2010). In turn, the characterization of the modules was achieved through measures of effective degree and community centrality. Effective degree represents the number of the weighted connections of a module, while

community centrality measures the influence area of each module in the network (Kovács et al., 2010).

Results

Adolescent PTSD Symptoms Network

The adolescent PTSD symptoms network is depicted in Figure 1. The network is constituted by 36 connections (density = .265); 35 are positive connections (97.2%) and one is negative (2.8%). Positive connections' weights vary between .099 to .522 (M .199, SD = .103) and the negative connection has a weight of -.124. Stability results are available in Figure S6 in the Supplementary Materials.

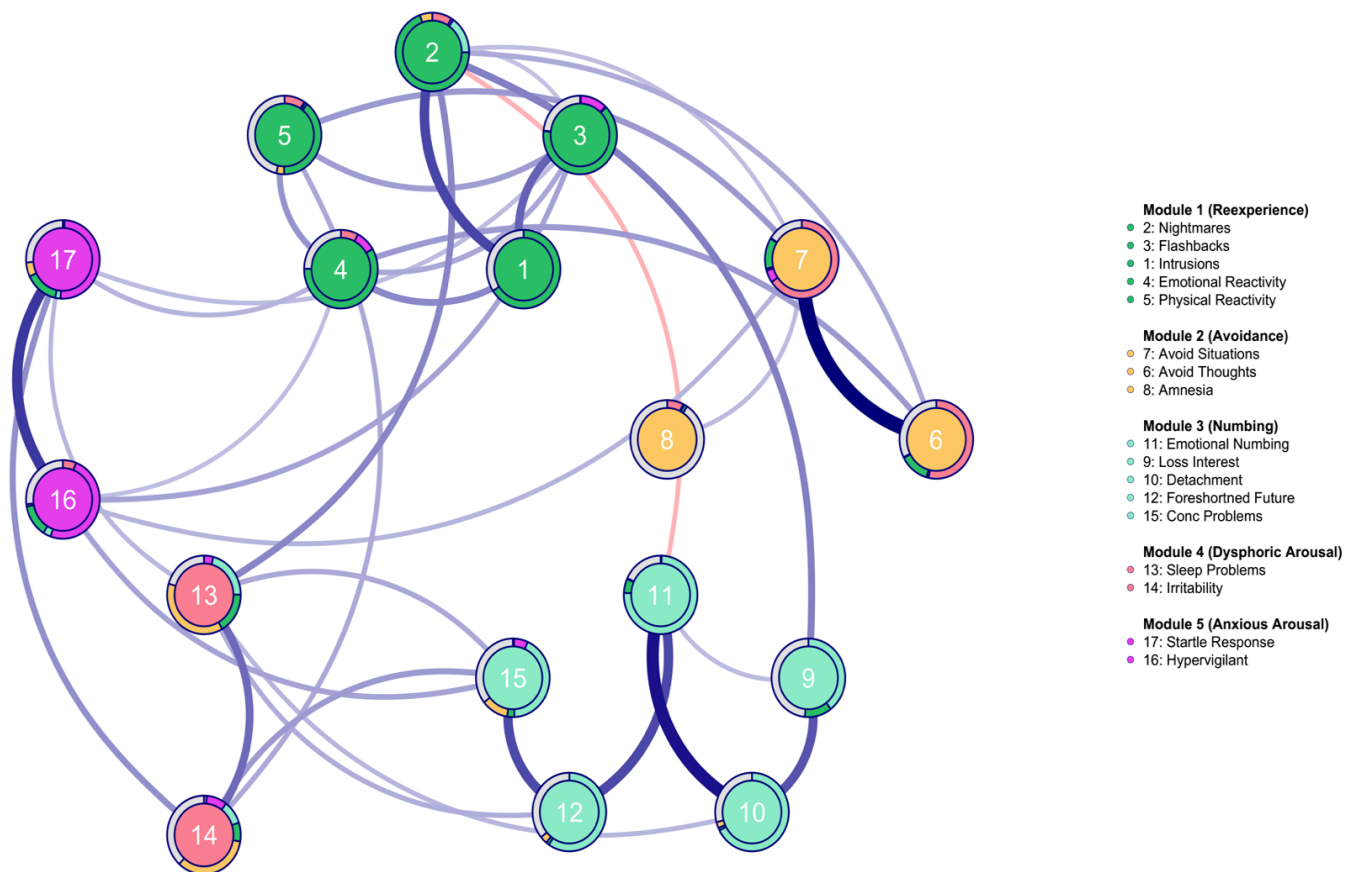


Figure 1. Hierarchical modular structure of the PTSD network. The symptoms in the network are represented by nodes (circles) of different colors and the edges (lines) connecting them depict their connections. The width of the edge represents the strength of the connection. The color of the nodes represents the module to which they most belong and the colors in the outer ring of the node display the amount of connection to other modules. The grey in the outer ring represents the absence of overlap. Light rose represents module 1; green nodes represent module 2; light green represents module 3; rose nodes represent module 4, and yellow nodes represent module 5. After the module number, in parenthesis, is the name of the factor of the Dysphoric Arousal Model most similar to the estimated modules.

Hierarchical Modular Structure of the Adolescent PTSD Symptoms Network

The hierarchical modular structure of the adolescent PTSD symptoms network was constituted by two levels. The first level corresponded to the 17 PTSD symptoms network (Figure 1) and the second level consisted of a five-module network (for a detailed description of each module see Table S1; and for the graphical representation of modules network see Figure S4).

Figure 2 displays the symptoms centrality in the first level. Within this level, nightmares (2), emotional numbing (11), avoidance of situations (7), and sleep problems (13) were the central symptoms, that is, they were at higher levels of strength. The bridge symptoms with a higher number of connections with other modules, that is with high values of bridgeness, were nightmares (2), sleep problems (13), and hypervigilance (16). The symptom of irritability (14) and concentration problems (15) showed high values of overlap, meaning that these were the symptoms that simultaneously belonged to a higher number of modules.

In the second level, five modules were identified. Figure 3 represents the measures of community centrality and effective degree for these five modules. Module 1 stands as the module with both higher levels of effective degree and community centrality. Nevertheless, modules 2 and 4 presented superior levels in effective degree; and module 5 revealed lower levels in both measures.

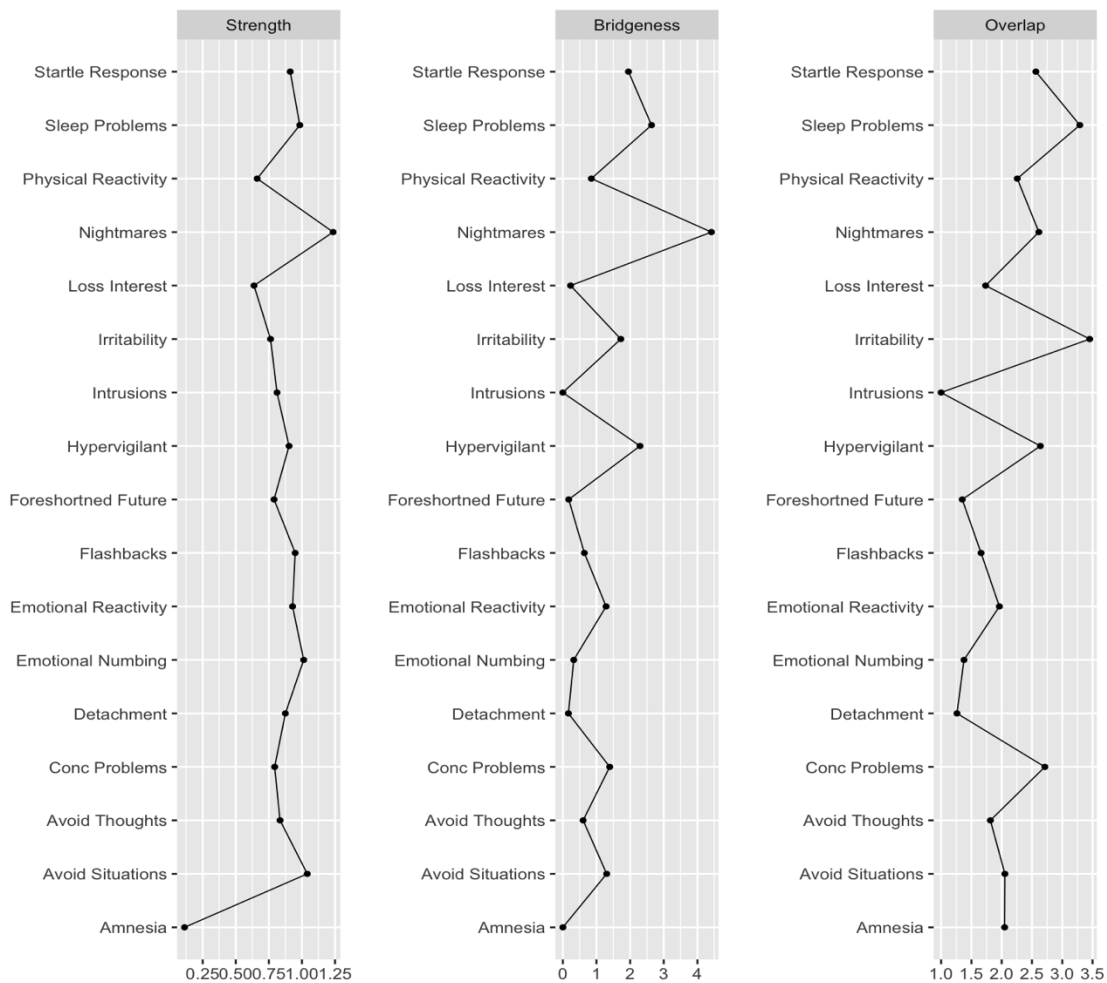


Fig. 2 Measures of strength, modular bridgeness, and modular overlap of the PTSD symptoms in the first level

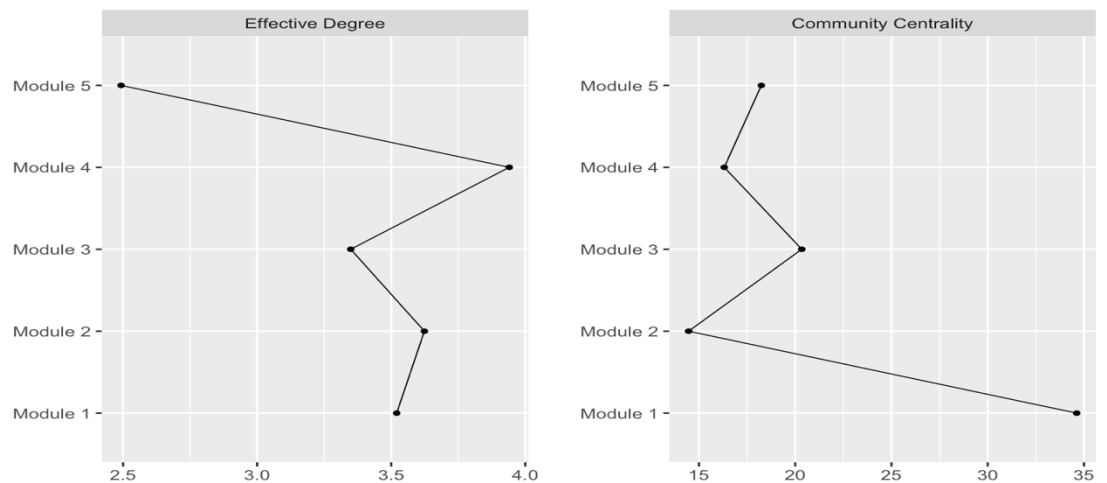


Fig. 3 Measures of effective degree and community centrality of the modules in the second level

Discussion

The present study aimed to uncover the modular structure of PTSD in adolescents. Through the use of the ModuLand algorithm, we identified a two-level structure. The first level corresponded to the 17 PTSD symptoms assessed by the DSM-IV. Then, based on these symptoms, a second level of 5 modules was found. This higher hierarchical level is in line with the five-factor model proposed by Elhai and Palmieri (2011), termed Dysphoric Arousal Model, and with previous studies based on factor analysis that revealed the superior fit of this model (Armour et al., 2013; Bennett et al., 2014; Sumner et al., 2014; Wang et al., 2011; Wang et al., 2013).

However, the use of this methods might prevent a deeper understanding of the hierarchical modular structure of PTSD in children and adolescents. This might be due to the exclusive focus on the items that best capture the symptoms, and its presentation (Marshall, Schell, & Milles, 2003), which could contribute to the shared variance of the PTSD symptoms (Armour et al., 2016). Moreover, these methods only provide the number of groups in which the items – which represent symptoms – belong to (Armour et al., 2016). Therefore, the use of alternative methods, such as the ModuLand algorithm, might add valuable information.

Globally, most of our modules were congruent with the Dysphoric Arousal Model, with only two exceptions. Concentration problems appear in module 3 encompassing numbing symptoms, instead of appearing in the group of dysphoric arousal symptoms as proposed by the Dysphoric Arousal Model (module 5 in the PTSD network). Amnesia in our structure belongs to module 2, characterized by avoidance related symptoms, instead of the group constituted by numbing symptoms, as proposed by the Dysphoric Arousal Model. A possible explanation for the different allocation of concentration problem symptoms, is related to its high level of bridgeness, which puts it in between different

clusters of symptoms. Regarding amnesia, it was assigned to module 2 alongside avoidance of situations and thoughts. In previous studies with adults (e.g., Armour, Fried, Deserno, Tsai, & Pietrzack, 2017; Fried et al., 2018) and with adolescents (e.g., Bartels et al., 2019; Cao et al., 2018), amnesia exhibited low centrality values and it has been suggested that it might not be a prototypical symptom of PTSD. Nevertheless, our results suggest that, even with low centrality, amnesia might be an additional element in the avoidance mechanisms module in adolescents. In fact, in DSM-IV amnesia was considered to belong to the avoidance cluster (American Psychiatric Association, 1994). However, recent proposals placed amnesia alongside negative alterations in cognition and mood (American Psychiatric Association, 2013), numbing (Elhai et al., 2011; Tsai et al., 2015), or negative affect (Armour et al., 2016; Liu et al., 2014) symptoms. In this context, future research should continue to explore its role in adolescents.

Despite these differences, our results are consistent with the five-factor proposal and provide support for the distinction between dysphoric arousal and anxious arousal symptoms (Watson, 2005). However, uncertainties remain about which cluster of symptoms accounts for each mechanism (Armour et al., 2013). In order to address this, we have calculated centrality measures for each of the modules, these measures might give us new insights to their mechanisms (Castro et al., 2018). Our results revealed that the module that encompassed the dysphoric symptoms (module 4) displayed the highest levels of effective degree. This shows that this module has strong connections with all other modules and might have an important role in the maintenance of the disorder, since these connections could contribute to keeping the modules more linked with each other. Curiously, in the study performed by Bennett and colleagues (2014) with youth from juvenile detention centers, the group of dysphoric symptoms was associated with several mental problems, supporting the fact that the symptoms within this module have an

important role in the connectivity with the other symptoms. In contrast, the module that covered anxious arousal symptoms (module 5) displayed low values in the measures used to characterize the modules. It has been proposed that the anxious arousal symptoms reflect an underlying dimension of fear (Hetzel-Riggin & Harbke, 2014), which is a common feeling in adolescents that experienced a traumatic event, but it is very often expressed through a pattern of emotional numbing (Bennett et al., 2014). The expression of these symptoms through emotional numbing (module 3) might explain the low scores in the centrality measures revealed by the anxious arousal module. Moreover, the anxious arousal symptoms are among those with the highest values of bridgeness and overlap, which indicates that these symptoms are positioned between modules and belong to various modules. The assumption that, in these age groups, the anxious arousal symptoms might be expressed through the emotional numbing symptoms could be explained by these high values in bridgeness and overlap.

Our results seem to suggest that dysphoric symptoms might be a relevant indicator for the maintenance of PTSD due to the elevated number of connections that can help spread information to other modules. In turn, the anxious arousal module might be more associated with development of the disorder since the symptoms in these modules seem to be positioned between the modules, and this positioning might promote an easy access route to other symptoms during development. In this context, the assessment and monitoring of these symptoms might help in the elaboration of treatment models for recovery after a traumatic event in these age groups (Sumner et al., 2014), thereby assisting in prevention strategies.

Notably, our results revealed that the module that grouped the re-experiencing symptoms (module 1) was the one displaying highest community centrality, suggesting that it might also have a significant role in the flow of information in the network by

accessing various symptoms (high community centrality). This means that this module has various paths to it and, consequently, can be reached from various other places (i.e., symptoms) in the network. In this way, this module might also have an important role in the development of PTSD (Bryant et al., 2017). However, it is important to notice that the distinctions between the roles of these two centrality measures, effective degree and community centrality, are yet to be proven in psychological networks, and future research should focus on identifying whether these roles are in fact represented by these measures.

Regarding the first level, the centrality measures used revealed that the symptoms of nightmares, emotional numbing, and avoidance of situations, were those with the higher levels of strength. These findings are similar to those reported by Russell and colleagues (2017) and Pfeiffer and colleagues (2019) that identified emotional numbing and nightmares, respectively, as the symptoms with highest strength. The results of the present study are also in line with the investigation performed by Cao and colleagues (2018) in which avoidance of situations was ascertained as one of the central symptoms, specifically among girls. It is important to note that both studies, as well as the present one, were conducted using the DSM-IV (American Psychiatric Association, 1994) diagnostic criteria. In contrast, the study performed by Bartels and colleagues (2019) used DSM-5 (American Psychiatric Association, 2013) diagnostic criteria and identified the two recently added symptoms, negative trauma related cognitions and persistent negative emotional state, as the most central symptoms. However, as in this study, restricted affect (i.e., emotional numbing) was also one of the symptoms with elevated centrality values, and amnesia was the symptom with the lowest strength (Bartels et al., 2019).

In this context, previous literature has recognized that adolescents might reexperience distressing dreams after a traumatic event (Pynoos et al., 2009). In adolescents, considering their specific developmental features, i.e., cognitive function,

emotional regulation techniques, and coping mechanisms, emotional numbing could be the observable behavior adopted in order to downregulate the distress provoked by the traumatic event (Bennett et al., 2014). Also, avoidance has been conceived as a coping mechanism used by adolescents to distance themselves from the distress provoked by the traumatic or stressful event (Woodward et al., 2019). Despite the temporary relief that this coping mechanism might be able to evoke, the suppression of these feelings can lead youth to engage in high risk and reckless behaviors (Pynoos et al., 2009). Therefore, these results highlight the need to assess these symptoms and their expressions in order to help prevent the symptoms' severity.

Moreover, the most central symptoms in our study are in line with the recent changes introduced in the ICD-11 (World Health Organization, 2018), which reduces the diagnostic criteria to six symptoms (i.e., nightmares, avoidance of situations, avoidance of thoughts, flashbacks, startle response, and hypervigilance). Although these symptoms are amongst those with the highest strength centrality, emotional numbing, sleeping problems, emotional reactivity, and detachment also displayed high centrality values. This seems to suggest that reduction in the ICD-11 (World Health Organization, 2018) might have been excessive. Nevertheless, the basis for this reduction was to promote a more specific diagnostic with less comorbid symptoms (Haravuori, Kiviruusu, Suomalainen, & Marttunen, 2016). In fact, sleeping problems, irritability, and concentration problems, which are symptoms that belong to other mental disorders (e.g., depression), and were removed from the ICD-11 criteria, were the symptoms with the higher overlap values in our study, pointing to a non-specificity of these symptoms in the PTSD diagnostic. Price and colleagues (2019) also found that these symptoms were those with the higher density of connections with generalized anxiety disorder (GAD) and depression and suggested that these symptoms might be responsible for the comorbidity

between PTSD, GAD, and depression. Thus, the reduction of symptoms implemented in the ICD-11 might indeed result in less comorbid symptoms. Although, with the removal of some of the most central symptoms it might, as well, result in less PTSD diagnoses in adolescents.

The results of the present study should be interpreted carefully, considering some limitations. First, data reanalyzed in this study comes from a non-clinical setting, whereby the generalization of these findings needs to be performed with caution. Second, the use of cross-sectional data constitutes an obstacle, since it prevents a longitudinal perspective of the development of PTSD symptoms between adolescence and adulthood. Future research should replicate this study in dynamic networks, thus allowing for the unveiling of causal influences. Third, the type of trauma and severity of the traumatic event might contribute to the differences found in comparison with other studies. Additionally, the PTSD symptoms were assessed with the DSM-IV, which does not cover the three new symptoms introduced in the DSM-5. Thus, we suggest the replication of this study with the DSM-5 to include the new modifications and the assessment of PTSD. Also, the use of the PCL-C to evaluate the presence of PTSD symptoms in adolescents may constitute a limitation; although this questionnaire was developed with the aim of assessing PTSD symptoms in adults, it has been consistently used in adolescents and has revealed itself to be suitable for evaluating the symptoms of PTSD in this age group (e.g., Jin & Guan, 2014; Radcliffe et al., 2007; Tian et al., 2014). However, its application to young populations might obscure specific symptomatic expressions typical of these younger groups. The items in the PCL-C aim to assess the presence of each of the symptoms of PTSD as established in DSM-IV; however, eight of the 17 criteria require the recognition of verbal experiences and emotions, which young people might not be able to understand, because of their specific developmental stage. Therefore, there is a possibility that this

measure might not capture the PTSD symptoms in adolescents (Hawkins & Radcliffe, 2006). Thus, it is essential to include a developmental perspective, as well as considering cultural differences, and the words that better capture the presence of the symptoms of PTSD in the elaboration of instruments of assessment of this disorder in youth.

Despite these limitations, the present study provides an important contribution to the debate on the structure of PTSD in adolescents. The exploration of the hierarchical modular structure of PTSD in this younger group and the characterization of the different functions of modules and symptoms yield a more detailed view about the nosography and maintenance mechanisms of PTSD.

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Chapter IV ^{1,2}

**The Nosographic Structure of Posttraumatic Stress Symptoms across
Different Types of Trauma: An Exploratory Network Analysis Approach**

Note:

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² Supplementary materials of this paper are available in Chapter IV of the Appendix.

Abstract

The nosographic structure of posttraumatic stress disorder (PTSD) remains unclear and attempts to determine its symptomatic organization have been unsatisfactory. Several explanations have been suggested, and the impact of the type of trauma is receiving increasing attention. Little is known about the differential impact of the type of trauma in the nosographic structure of PTSD, however, and thus we explore the nosology of PTSD and the effect of the type of trauma on its symptomatic organization. We reanalyzed five cross-sectional psychopathological networks involving four types of trauma (war-related trauma, sexual abuse, terrorist attack, and refugees). The weighted topological overlap was used to estimate the networks and attribute weights to their links. Co-expression differential network analysis was used to identify the common and specific network structures of the connections across different types of trauma and to determine the relevance of the symptoms across the networks. We found a set of symptoms with more common connections with the other symptoms, suggesting that these connections might constitute the prototypic nosographic structure of PTSD. We also found a set of symptoms that had a high number of specific connections with other symptoms. These connections varied according to the type of trauma. Finally, we ascertained the relevance of the symptoms across the common and specific networks. Our findings offer new insights into the symptomatic organization of PTSD and support previous research into the impact of the type of trauma on the nosology of this condition.

The Nosographic Structure of Posttraumatic Stress Symptoms across Different Types of Trauma: An Exploratory Network Analysis Approach

Posttraumatic stress disorder (PTSD) is a complex, heterogeneous, and debilitating disorder (Birkeland et al., 2020). The nosographic structure (i.e., symptomatic organization) of PTSD has been exhaustively examined (Bryant, 2019), revealing numerous symptomatic structures (Armour et al., 2016). The diversity of distinct conceptualizations of the nosographic structure of PTSD has severe implications for research and clinical practice: it leads to a variety of clinical presentations (Galatzer-Levy & Bryant, 2013), which hinder assessment (Elhai & Palmieri, 2011) and, subsequently, increases the potential an incorrect diagnosis (Elhai et al., 2012), as well as hampering the development and selection of personalized treatments (Armour et al., 2016). In this context, a greater comprehension of the nosographic structure of PTSD would be valuable, since it would help ascertain the core symptoms, and identify its etiopathogenic and maintenance mechanisms (Elhai et al., 2011). In turn, this would help develop and refine assessment instruments and treatment plans (Armour et al., 2016).

The clarification of PTSD nosographic structure might be improved if the type of trauma experienced is considered. Previous studies have suggested that specific symptoms might be associated with certain types of trauma (Kessler et al., 2017); for example, Kelley and colleagues (2009) and Smith et al., (2016) found avoidance symptoms in a sample of individuals who had experienced sexual trauma. Graham and colleagues (2016) found that the symptoms of detachment and loss of interest were associated with war-related trauma. Physiological reactivity has also been associated with motor vehicle accidents (Kelley et al., 2009). However, the same symptoms have also been associated with different types of traumatic events. For example, Ge et al., (2019)

and Bryant et al. (2017) found that physiological reactivity was associated with both earthquake trauma and traumatic injury.

Several factors have been proposed to explain these discrepancies: the use of non-representative samples; the study of a single traumatic event (Smith et al., 2016); demographic features, such as age and sex (Lancaster et al., 2014); and time, and recurrent exposure to a traumatic event (Fink et al., 2017; Shea et al., 2017). The methodologies used to determine these associations (i.e., profile analysis [Kelley et al., 2009]), confirmatory factor mixed modeling (Shevlin & Elkit, 2012), and logistic regressions (Graham et al., 2016), have been insufficient to obtain an accurate view of the symptomatic organization across different types of trauma. This is because, essentially, they only acknowledge the presence of differences, and not the identification of specificities and commonalities of one symptom at a time. The variability of the results and the limitations of the current methods might therefore inhibit a deeper understanding of the nosographic structure of specific types of trauma, since the primary goal is identifying of the unique symptoms of PTSD. Recently, several authors (e.g., Borsboom & Cramer, 2013) have proposed expanding this focus by looking at the level of the connections between the symptoms.

Network theory (Borsboom et al., 2019) might be a promising path to overcoming these difficulties. Theoretically, this framework conceptualizes mental disorders as complex systems of interacting symptoms (Borsboom & Cramer, 2013). From this perspective, PTSD is perceived as a set of symptoms (nodes) that interact (link) with each other (Birkeland et al., 2020) forming a complex psychopathological network. In recent years, this focus on the interplay between symptoms has led to important insights into the nosographic structures of mental disorders (Boschloo et al., 2015; Fried et al., 2018). In contrast to factor analytical studies, the network analysis approach allows the overall

disposal of the symptomatic organization of PTSD to be identified, in and across clusters of symptoms.

Central symptoms (i.e., symptoms with the strongest connections with other symptoms) have been the main route used to ascertain the nosographic structure of mental disorders (Epskamp et al., 2018), however, previous studies on the network structure of PTSD symptoms have had inconsistent results regarding the centrality of symptoms (Ross et al., 2018). For example, Birkeland and Heir (2017) found that the symptom with the highest strength was emotional numbing (i.e., the sum of the weighted connections of a node to the other nodes in the network; Opsahl et al., 2010) in a sample of individuals who had experienced a terrorist attack. Conversely, hypervigilance and concentration problems had the highest strength in a sample of people who had survived an earthquake (McNally et al., 2015). Armour and colleagues (2017) found that negative trauma-related emotions and flashbacks had the highest levels of strength in a sample of military veterans. In this context, and due to the lack of consistency regarding the most central symptoms across studies, we used the fused graphical lasso (FGL; Danaher et al., 2014) and the network comparison test (NCT; van Borkulo et al., 2017) to identify the nosographic network structure of PTSD.

The FGL is an extension of the graphical lasso method which is commonly used to estimate the network of connections between the symptoms of PTSD (see Fried et al., 2018), and allows the joint estimation of a set of networks. This is accomplished using two tuning parameters. The first parameter (λ_1) is responsible for the process of regularizing the network. The second parameter (λ_2) performs the same procedure on the differences between the edge weights of the networks. Specifically, high levels of the second parameter lead to the estimation of similar networks, whereas small values make the differences between the networks clear (Danaher et al., 2014). In this way, the FGL

enables the amount of similarity and the differences across the networks to be estimated, and the validity and replicability of the networks to be verified.

The NCT is a framework used to compare the structure of networks (van Borkulo et al., 2017). The NCT compares key aspects of the structure of the network by applying a permutation test. First, the NCT allows the structure of the networks to be estimated and obtains a global structure considering the particularities of all the networks (i.e., structure invariance). Second, it estimates the overall connectivity; in other words, the weighted absolute sum of all edges in the network (i.e., global strength invariance). Third, it estimates the differences in strength of the edges (i.e., edge strength invariance). Generally, it is a framework that allows the identification of significant aspects in which the networks vary. In the context of a severe replicability crisis in the psychology field (Lewandowsky & Oberauer, 2020) these frameworks thus allowed considerable achievements regarding the replicability of psychopathological networks (Fried et al., 2018).

These methods are limited, however, in their ability to capture the differences in networks in detail. For example, in order to secure the validity and replicability of the networks, the FGL (Danaher et al., 2014) inhibits differences that might be of significant clinical relevance (Lyu et al., 2018). The NCT (van Borkulo et al., 2017) is unable to compare more than two networks simultaneously, making a comparison between various networks a daunting task. Moreover, the method used to assess differences between the strengths of the edge could generate type I errors, and the size of the sample affects the statistical power (Fried et al., 2018). In the particular context of PTSD, Fried and colleagues (2018) applied both methods to explore the differences and similarities across four datasets of individuals with symptoms of PTSD according to the DSM-IV. Their primary focus was to demonstrate the replicability of the network structure of PTSD,

concluding that the networks were replicated, with moderate to high coefficients of similarity (0.62–0.74) and centrality indices (0.63–0.75) across the four networks. Despite the advances attained in this study, the problem underlying the nosographic structure of PTSD is not completely solved, since there are variations in the central symptoms and network structures across several PTSD studies (Birkeland et al., 2020) that remain unexplained (Borsboom, 2017).

In this context, it has been proposed that the differential impact of the type of trauma is a possible explanation for the distinct network structures of PTSD found (Benfer et al., 2018). The exploration of the network perspective in this topic is still in its infancy, however, since, to our knowledge, only one network study (Benfer et al., 2018) has addressed this specific topic, where the authors examined and compared the network structure of PTSD symptoms, according to the DSM-5, in three types of trauma: motor vehicle accident, sudden accidental/violent death of a loved one, and sexual assault. Using the NCT, Benfer and colleagues (2018) found differences in the network structure across the three types of trauma (compared in pairs). Differences were found in the global strength of the network that included sexual assault and motor vehicle accident, and in the association of negative beliefs and anhedonia which was not present in sexual assault trauma. The network that included the trauma of sudden accidental/violent death of a loved one revealed smaller global strength values than the other networks. There were also differences in the centrality of symptoms, where flashbacks and irritability were identified as central in the networks involving sexual assault and motor vehicle accidents, respectively.

This preliminary evidence suggests that different types of trauma might give rise to different network structures. This deserves further exploration, with a more detailed view of which connections occur in PTSD despite the traumatic event, and which

connections are dependent on the traumatic event. This detail might allow us to detect specific patterns in the development and the maintenance of PTSD after specific traumatic events, and, consequently, improve treatment and prevention responses. Identifying which development paths are present despite the traumatic event would also help in clarifying the prototypical network structure of PTSD.

In this context, it seems reasonable to use alternative approaches from other fields of network science that might help elucidate these issues. The co-expression differential network analysis (CoDiNA; Gysi et al., 2020) method was recently developed with the aim of providing a more fine-grained view of complex systems (e.g., biological, social, financial). Specifically, this framework allows the comparison of multiple weighted networks, and the identification and classification of nodes and their connections among the networks (Gysi et al., 2020). There are three main categories of nodes and their connections: common, different, and specific (a detailed description of these categories can be found in the methods section). This framework also allows the importance of each symptom in each of the networks to be identified. These details are key steps to a greater comprehension of the network structures. CoDiNA has been used in previous studies to ascertain the expression of HIV in children and adults with or without tuberculosis, and to identify specific transcription factors in distinct types of cancer (Gysi et al., 2020). Remarkably, this method identified the presence of specific genes and connections in the network of HIV in children and in the network of HIV of adults and sets of genes associated with tuberculosis (Gysi et al., 2020).

In PTSD, the application of this framework might promote important advances regarding its nosology, such as the identification of connections that are specific to certain types of trauma, or connections that are transversal to all types of trauma, and could identify the importance of each symptom in the network. Understanding the differential

impact of each trauma type might promote the development of more personalized and effective treatments. This study thus aimed to (1) explore the nosographic structure of PTSD, and (2) the impact of four types of trauma in the symptomatic organization of PTSD. The CoDiNA framework was used to achieve these goals, since its ability to identify the overall structure of PTSD, and the specificities related to each trauma might promote a better understanding of the disorder.

Method

Networks Collection

Three previous PTSD published networks were collected from databases (PsychInfo, PsyArticles, and Academic Search Complete). Two of the PTSD networks were provided upon request of the authors (Birkeland et al., 2017; Spiller et al., 2017), this was done to increase the variety of traumatic events. The stability of these networks was evaluated in the original studies. A convenience sample of five cross-sectional PTSD networks were collected and reanalyzed (see Table 1, for details of the original network studies). Two of these networks were estimated according to the symptoms in the DSM-IV (17 symptoms) and the others according to the symptoms in the DSM-5 (20 symptoms). The networks covered different types of trauma: sexual abuse (N=179), war-related trauma (N=221; N = 378), terrorist attack (N=190), and refugee trauma (N=151). Comorbidity networks were not included in the analysis.

Table 1*Descriptive variables of the original studies*

Network	Study	Population	Type of Trauma	Sample	DSM	N	Data Type	Original Analysis
1	(Armour et al., 2017)	Adults	War-related trauma	Clinical	DSM-5	221 (86.7% male)	Continuous	GGM
2	(Birkeland & Heir, 2017)	Adults	Terrorist attack	Community	DSM-IV	190 (38% male)	Continuous	GGM
3	(McNally et al., 2017)	Adults	Sexual abuse	Clinical	DSM-IV	179 (16.2% male)	Continuous	GGM
4	(Spiller et al., 2017)	Adults	Refugees	Community	DSM-5	151 (70% male)	Continuous	GGM
5	(Moshier et al., 2018)	Adults	War-related trauma	Community	DSM-5	378 (93.6% male)	Continuous	GGM

Data Analysis

CoDiNA (version 1.1.2; Gysi et al., 2020) R package (version 4.0.2; R Core Team, 2020) was used to explore the general network structure of PTSD and the structure of specific connections across different types of trauma. We first used the weighted topological overlap (wTO) (version 1.6.3; Gysi et al., 2018) R package to estimate the networks. The wTO is derived from a Topological Overlap Matrix, and a pair of nodes have its weight given by a normalized correlation. It means, its correlation is normalized by the correlation of all its shared neighbors. The wTO was calculated using the Pearson correlation coefficients between each pair of nodes, according to the following equation:

$$\omega_{i,j} = \frac{\sum_u a_{i,u} a_{u,j} + a_{i,j}}{\min(k_i, k_j) + 1 - |a_{i,j}|}$$

Where:

$$k_i = \sum_j a_{i,j}, \text{ and}$$

$$A = [a_{i,j}] = \begin{cases} \rho_{i,j} & \text{if } i \neq j \\ 0 & \text{if } i = j \end{cases}$$

CoDiNA was then used to explore and characterize the common and specific structure of connections between the different types of trauma. The CoDiNA method considers the weight of the link in which the values ranged between -1 and +1, and if the networks were derived from different experiments that are not under the same procedure, then the networks must be re-parametrized; a stretch parameter is used to permit this comparison, performing a minmax normalization, allowing all networks to be in the same range of values, and per consequence, comparable. The CoDiNA method is based on the weight of the connections between the nodes included in the networks and uses a predefined threshold to remove the spurious connections. In our case, we set the threshold to zero; this threshold identifies the minimum link weight to be considered for the comparison. When a link is under the absolute value of the defined threshold, it is set to zero; if the weight value is above the threshold, it is assigned +1, and if under the threshold, then -1 is assigned to the link, if a link receives zero in all networks under evaluation it is then removed from the analysis. Next, CoDiNA classifies each of the connections (i.e., links) according to three types: common (α), different (β), and specific (γ). Common connections are those present in all the networks; different connections are those present in all the networks but with different weight signs. Specific connections are those that are not present in all networks. As an example, assume that a link exists between symptoms A and B in all trauma types; this link is common to all networks under evaluation – with similar weight, and the same sign – and therefore, we say it is a common link, however, there could be another link that only exists in one particular trauma, which CoDiNA will call a specific link. However, when saying a link is only specific in a

multiple comparison set up, the need to understand where this link exists becomes evident, and a sub-classification is required. A sub-categorization is therefore performed for different and specific links in order to clarify the network in which the connection is present.

Degree centrality (i.e., the absolute number of connections between a node to all other nodes in the network; Opsahl et al., 2010) was used to characterize the common and specific structure of connections between the networks. The qgraph package (Epskamp et al., 2012) for R (version 1.6.3) was used to estimate this centrality measure. The importance of each of the symptoms in each network was calculated based on their score ratios, from CoDiNA. This score measures how well classified a link is, and how strong that link is for a particular network. On this basis, we can easily derive their strength in each network, and compare it to the overall strength of all other symptoms, per category, by calculating its Z-Score, and later correcting for multiple corrections; if a symptom was important overall in more than one network, we selected the one with the lower *p*-adj-value (BH FDR corrected). The igraph package (version 1.2.6; Csardi & Nepusz, 2006) for R was used to create the graphical representation of the common and specific network structure of connections according to the type of trauma.

Results

Network Structure of Common Connections

The common connections between the symptoms of PTSD across all the networks endorsing different types of trauma is depicted in Figure 1. The symptoms with a higher number of common connections to the other symptoms in the network, and higher levels of degree, were physiological reactivity, emotional reactivity, startle response, concentration problems, and loss of interest.

Network Structure of Specific Connections across Types of Trauma

The structure of the specific connections varied according to the type of trauma (Figures 2–3). Based on the scores provided by CoDiNA (see Table 1 in the supplementary materials), the war-related trauma and refugees networks revealed a distinct specific connection structure (Figure 2). There was no connection between flashbacks and avoidance of reminders, destructive behavior, irritability, and loss of interest in the network of refugees. The specific connection between the concentration problems symptom, and the avoidance of reminders and avoidance of situations previously found was not detected in war-related trauma. Similarly, no association was found between amnesia, avoidance of thoughts, irritability, or nightmares among refugees. No specific connection was found between irritability as a symptom and the avoidance of reminders, concentration problems, detachment, flashbacks, loss of interest, or sleep difficulties in the sample of refugees. No connection was found between loss of interest as a symptom, in the sample of refugees, and amnesia, avoidance of thoughts, flashbacks, irritability, nightmares, sleep difficulties, or destructive behavior. All the specific connections of the aforementioned symptoms were present in the remaining types of trauma (i.e., sexual abuse and terrorist attack), with the exception of the three new symptoms of DSM-5. No different connections were found. All symptoms were positively correlated with others, which makes it impossible, by definition, to have different connections.

Differences were found regarding degree centrality across the networks (Figure 4). The network concerning war-related trauma revealed that the symptoms of negative emotions, destructive behavior, and flashbacks had the highest levels in this measure, whereas there were higher degree of negative emotions, blame, and flashbacks in the network pertaining to refugees. The symptoms with higher levels of degree in the

networks of a terrorist attack and sexual abuse were flashbacks, concentration problems, irritability, loss of interest, and avoidance of thoughts.

The most important symptoms in the network of common connections were found to be intrusive thoughts, nightmares, emotional reactivity, physiological reactivity, avoidance of reminders, negative beliefs, loss of interest, detachment, restricted affect, hypervigilance, and startle response (see Table 1 in the supplementary materials). In turn, a set of symptoms were more important across the specific networks: flashbacks, concentration problems and sleep difficulties were more relevant in networks involving war-related trauma, terrorist attack, sexual abuse, and refugees. Negative emotions and blame were more relevant in war-related trauma and in refugees' networks. Destructive behavior was more relevant in only one of the two networks of war-related trauma and in the network of refugees. Finally, the symptom irritability was more relevant in the networks involving war-related trauma, sexual abuse, and terrorist attack. Amnesia and avoidance of thoughts were not found to be relevant either in the common or specific networks analyzed.

Network Structure of Common Interactions

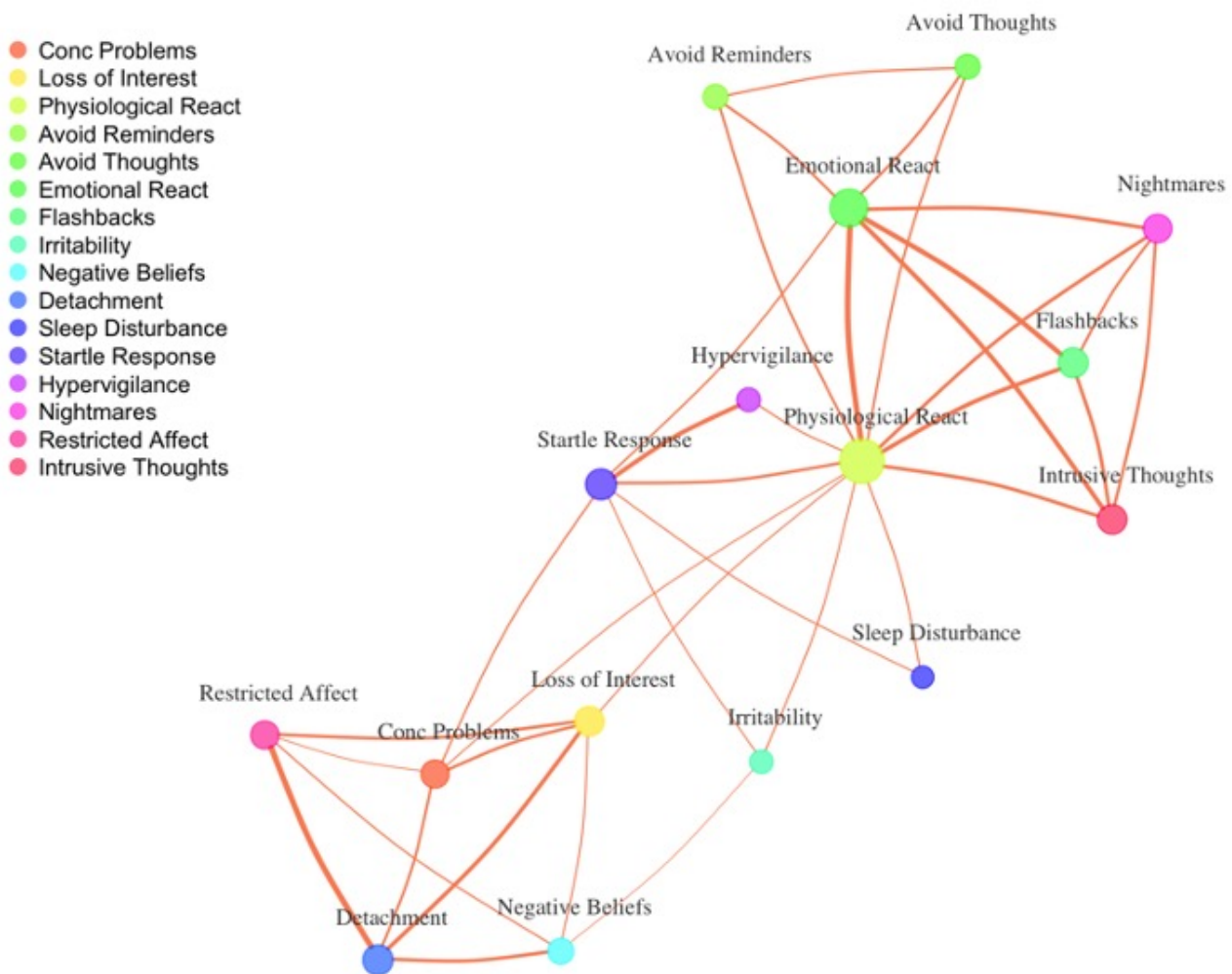


Figure 1. Network structure of common connections across the different types of trauma. Nodes represent the symptoms of PTSD and the edges between the nodes represent the weighted topological overlap values between the symptoms across the different types of trauma. The size of the nodes represents the degree centrality values. The thicker edges represent the higher weighted topological overlap values between the symptoms across the networks.

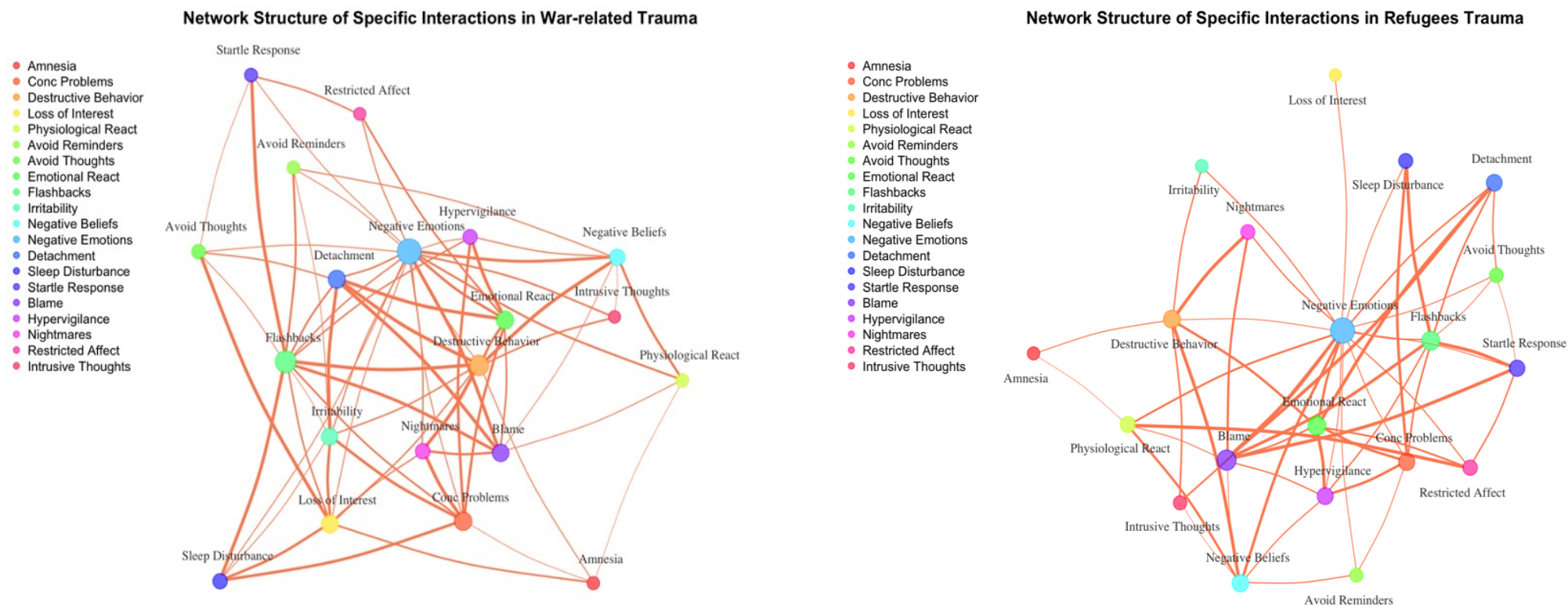


Figure 2. War-related and refugees trauma network structure of specific connections. Nodes represent the symptoms of PTSD and the edges between the nodes represent weighted topological overlap values between the nodes in the network pertaining to war-related and refugee trauma. The thicker edges represent the higher weighted topological overlap values between the symptoms in the networks.

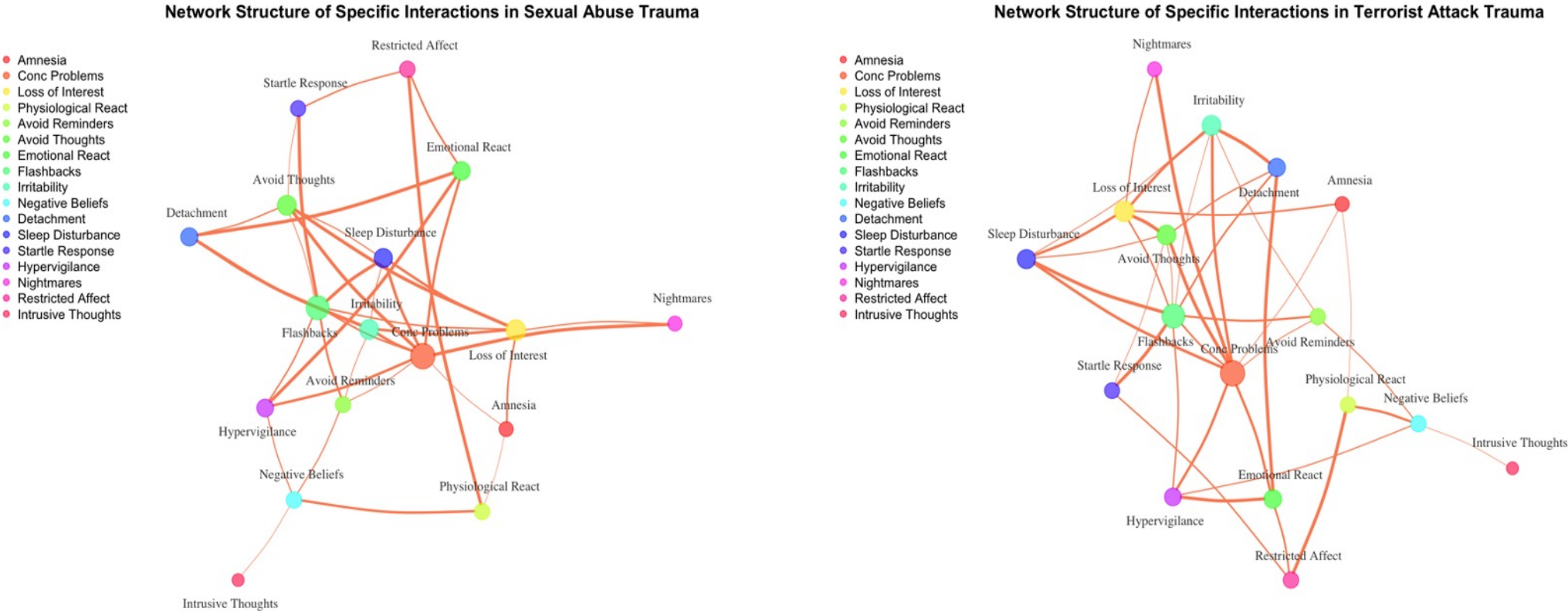


Figure 3. Sexual abuse and terrorist attack trauma network structure of specific connections. Nodes represent the symptoms of PTSD and the edges between the nodes represent the weighted topological overlap values between the nodes in the network pertaining to sexual abuse and terrorist attack trauma. The thicker edges represent the higher weighted topological overlap values between the symptoms in the networks.

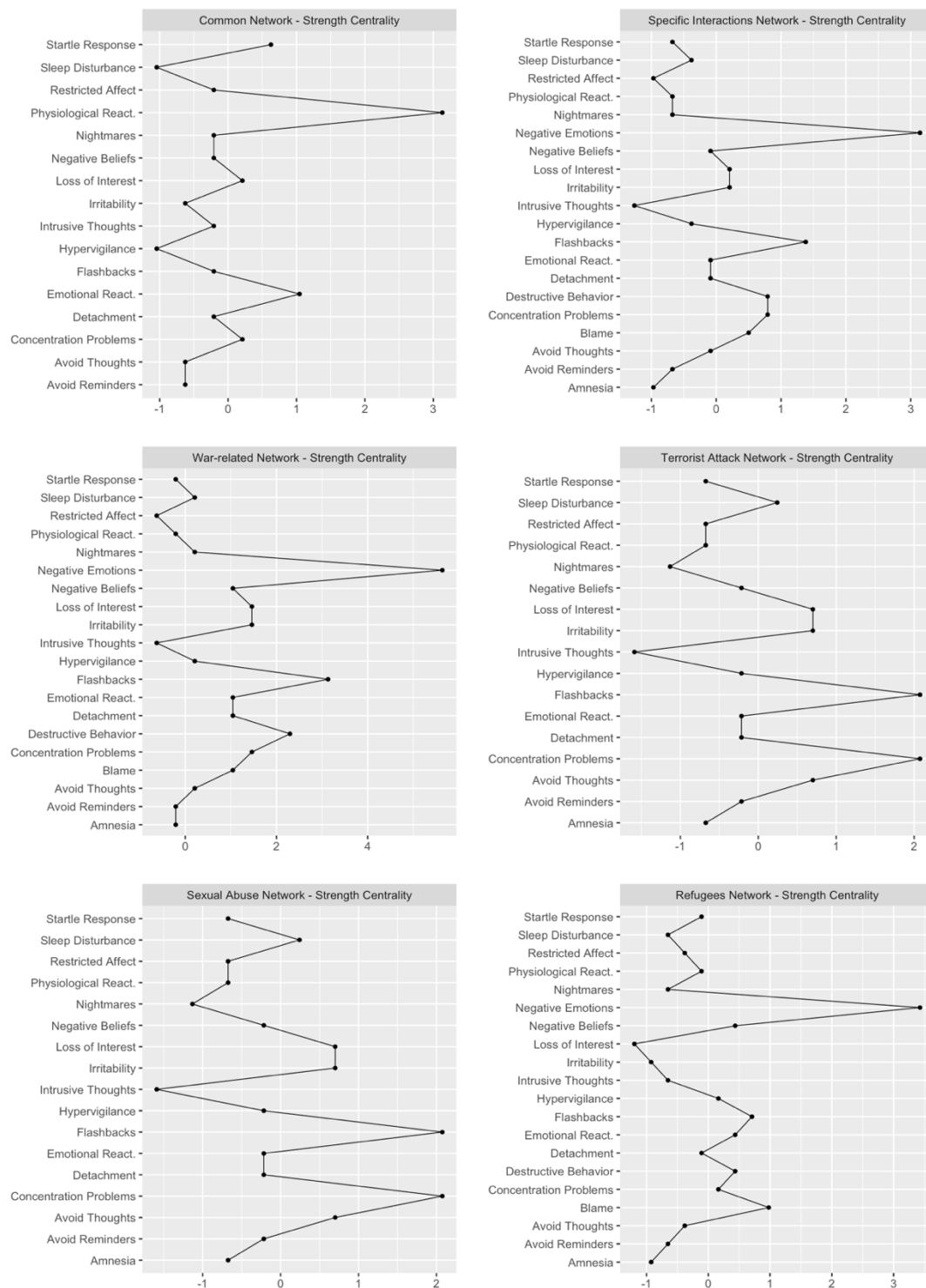


Figure 4. Degree centrality values across the networks. Top left figure shows the degree centrality values of the network of common connections; top right figure shows the degree centrality values the network of specific connections; center left figure shows the degree centrality values of the war-related network; center right figure shows the degree centrality values of the terrorist attack network; bottom left figure shows the degree centrality values of the sexual abuse network and bottom right figure shows the degree centrality values of the refugees network.

Discussion

The nosographic structure of PTSD remains uncertain (Bryant, 2019) and attempts to uncover the symptomatic structure of this condition have turned out to be unsatisfactory (Birkeland et al., 2020). This is amplified by the reported association between the type of trauma and distinctive clinical configurations (Kessler et al., 2017). This study thus took a novel approach to clarify these topics, and found that the symptoms physiological reactivity, emotional reactivity, startle response, concentration problems and loss of interest had the highest number of common interactions with the other symptoms. This suggests that these central symptoms play an important role in the common structure of PTSD. Interestingly, these symptoms, except for startle response, have previously been suggested as non-specific symptoms of PTSD (Gros et al., 2010). These symptoms overlap with symptoms from other mental conditions, such as major depression or generalized anxiety disorder (i.e., concentration problems and loss of interest; Gros et al., 2012; Price & Stolke-Cooke, 2015), and panic disorder or specific phobia (i.e., physiological, and emotional reactivity; Walton, 2017). In fact, it has been suggested that these non-specific symptoms are responsible for the high levels of comorbidity between these disorders and PTSD (Spinhoven et al., 2014) or constitute a general component of distress that is transversal to several mental disorders (Watson, 2009). Our results point in this direction, suggesting that these symptoms are central to the common structure of PTSD, although apparently not specifically related to any of the traumatic events analyzed in the present study.

In addition to being central symptoms, except for concentration problems, our results also suggest that these symptoms are more relevant to the common network structure than to any of the trauma specific networks, further supporting the idea that, although relevant in the common network, these symptoms might not be distinctively

related to any of the traumas analyzed in the present study. This is important, since the cognitive model of PTSD (Ehlers & Clark, 2000) suggests that the experience of general symptoms (e.g., physiological reactivity, emotional reactivity, and loss of interest) are common reactions after a traumatic event (Ehlers & Clark, 2000). For example, individuals with PTSD tend to re-experience symptoms (i.e., physiological, and emotional reactivity), independent of the traumatic event when their sense of danger is activated (Ehlers & Clark, 2000). The importance of these symptoms in the common network might also emphasize their role in the maintenance of PTSD, no matter which traumatic event occurred. Ehlers and Clark (2000) note that mood and arousal (i.e., loss of interest, startle response) symptoms play an important role in the negative appraisal of a traumatic event, which is considered an important factor in the maintenance of PTSD. The high level of degree of these symptoms and their importance in the common network also support this view.

Negative emotions, flashbacks, concentration problems, destructive behavior, blame, irritability and loss of interest showed a high number of specific connections with the other symptoms in the networks. Remarkably, these connections varied according to the type of trauma (Figure 2–3), which seems to support previous research which has acknowledged the differential impact of the type of trauma on the network structure of PTSD (Kelley et al., 2009). In our study, war-related trauma had a cluster of specific connections between the symptoms of flashbacks and the avoidance of reminders, destructive behavior, irritability, and loss of interest, but which were not present in the network of refugees. We also did not find a connection between concentration problems and the avoidance of reminders in the network associated with war-related trauma, although this was present in the network of refugees. Previous studies have shown that refugees often have concentration difficulties involving the memory of painful and

distressful experiences (Minihan et al., 2018) and veterans usually experience depressive symptoms (i.e., irritability and loss of interest) along with PTSD symptoms (Rytwinski et al., 2013). The different specific connections identified suggest different network structures in each trauma and, consequently, different central symptoms. Degree centrality results suggest slight differences between the war-related trauma and refugees' networks. Specifically, destructive behavior and depressive symptoms seem to be more central in the war-related trauma than in the refugees' network, whereas blame and symptoms of arousal and re-experience appear more central. Despite this, negative emotions were the most central symptom in both networks.

These results might be explained by the different and common characteristics of the traumatic events, such as the varying duration of the exposure and the unpredictability, or not, of the event (Goral et al., 2017). For example, veterans and refugees are exposed to a type of trauma that features continuous exposure to stressful and traumatic events (Jakob et al., 2017; Li et al., 2016), which might explain the commonalities found by our study in these types of trauma. There are important differences between these types of trauma, however, which might explain the distinct network structures found. In veterans, the impact of the traumatic event seems to promote a negative view of themselves, of others, and of the future (Benner et al., 2018), which makes them susceptible to an elevated number of depressive symptoms (Armenta et al., 2019). This might be because the acts perpetuated and testified by them in war changes the way they evaluate themselves and the others (Benner et al., 2018). In turn, refugees live in continuous fear of leaving their homes, being persecuted, torture, sexually assaulted, murdered or witnessing the murder of their loved ones (Liddell et al., 2019; Li et al., 2016) which leaves in a constant state of uncertainty about their future (Liddell et

al., 2019), possibly leading to the increased number of connections in arousal and re-experiencing symptoms observed in our study.

Interestingly, sexual abuse and terrorism attack trauma revealed a similar structure of connections and central symptoms. This might be related to the common contextual characteristics of these types of traumas, such as their unpredictability (Pereda, 2013) and the perpetrators' deliberate intention of causing harm (Santiago et al., 2013). For example, previous studies have suggested that victims of sexual abuse gain awareness of their experience through one of the most central symptoms in both networks, flashbacks (Schröder et al., 2018), and this was found to be a central symptom by McBride et al. (2020). In turn, in a study conducted with victims of a terrorist attack, 49.5% experienced DSM-IV group B, re-experiencing symptoms, which includes flashbacks (DiGrande et al., 2010), and might explain the elevated centrality of this symptom in our network.

Our method was able to identify symptoms that were more relevant to the specific networks of each trauma. Flashbacks, blame, negative emotions, destructive behavior, irritability, concentration problems and sleep difficulties were found to be more relevant symptoms across the specific networks of each trauma. Remarkably, flashbacks, destructive behavior, concentration problems and sleep disturbances were not present in one of the networks that covered war-related trauma. This might be related to the features of this network, such as the higher number of participants (i.e., $N=378$), the inclusion criteria (i.e., age of recruitment was 18), or the fact that the evaluation of the traumatic event was based on the worst event. In contrast, the other network (i.e., $N=221$) assessed all the traumatic events described by the participants, which might explain the higher incidence of, and per consequence, the relevance of the symptoms.

Irritability was not a relevant symptom in the network of refugees, although previous research has found strong relationships between irritability and destructive

behavior (Pfeiffer et al., 2019). The three new symptoms of DSM-5 were not present in the networks pertaining to sexual abuse and terrorism attack, as these were networks evaluated with the DSM-IV. Amnesia and avoidance of thoughts were not relevant in the common, or across specific networks. Our results thus support previous research which suggests the removal of amnesia from the DSM (e.g., Fried et al., 2018; Birkeland et al., 2020), and that future research should explore the role of avoidance of thoughts as a symptom across the different types of trauma analyzed in this study, to ascertain its relevance to the diagnosis. Overall, this suggests that these symptoms might be markers of the type of trauma to which victims were exposed, in the same way that CoDiNA was used in genetics to identify specific genes associated with particular types of cancer. This remains to be assessed for PTSD, however, since the types of trauma assessed in this study were all exclusively interpersonal and a relatively small number of networks was used in our study.

The inclusion of more networks and types of traumatic events might promote a more refined view of which symptoms might be more important and possible markers for each trauma. In this context, future research should also include intrapersonal traumas (i.e., natural disasters) to clarify the specific network structure of connections in this type of trauma, as well as including and comparing more types of traumas. Another limitation is that we did not explore the differential impact of other confounding variables that might also affect the network structure and explain the differences in the networks. For example, sex and gender have been shown to have an effect in symptomatic profiles (Olff et al., 2007), and this might have an important impact on the network structure of each trauma.

The networks included in the present study were also all cross-sectional. It has been acknowledged that the use of cross-sectional data hampers comprehension of the evolution of symptomatology because the evaluation of symptoms is made at a single

point in time. To overcome this limitation, we suggest the replication of these analyses in longitudinal networks, which are intended to identify possible fluctuations in these findings across the course of the mental disorder. One of the networks showed low reliability, and although the remaining networks displayed moderate to high reliability values, this might have affected the results. The networks analyzed in this study were assessed with different versions of DSM (i.e., DSM-IV and DSM-5). These versions include different numbers of symptoms, which might constrain the analysis of possible connections between the symptoms. We believe future research should therefore replicate this study in larger networks with the same number of symptoms. Two of the five networks analyzed included war-related trauma, which resulted in a network with a high number of connections in comparison to the other networks and their types of trauma.

Most of the central symptoms found were symptoms present in DSM-IV and DSM-5, and their presence in both versions of DSM increases the possibility of a higher number of connections with the other symptoms. The centrality of these symptoms should therefore be interpreted carefully, since it might be a result of method variance. Similarly, this is the first time that the wTO and the CoDiNA frameworks have been used in the psychopathological network field, so it makes sense to interpret these results carefully, since the weight of the connections is not the traditional partial correlations estimated in psychopathological networks, but the weighted topological overlap.

In conclusion, despite these limitations, we believe the use of these frameworks needs to be improved and examined to accommodate the particularities of psychological symptomatic networks, because they contribute to revealing new insights about the nosographic structure of PTSD and its specificities in different types of trauma.

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Chapter V ^{1,2}

**Exposure to Traumatic Events and Development of Psychotic Symptoms in
a Prison Population: A Network Analysis Approach**

Note:

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² Supplementary materials of this paper are available in Chapter V of the Appendix.

Abstract

Previous studies consistently observed an association between exposure to traumatic events and psychotic symptoms. However, little is known about the differential impact of distinct traumatic events and the role of general symptoms in mediating this relationship. Thus, our study aimed to explore the differential association of several traumatic events to the psychotic symptoms in a sample of prisoners and whether this association is mediated by general symptoms. The total sample from the *Survey of Psychiatric Morbidity Among Prisoners in England and Wales* ($N = 3039$; 75.4% male) was used. Participants completed a list of traumatic events experienced before reclusion, the Psychosis Screening Questionnaire, Clinical Review Schedule-Revised. Network analysis was used to estimate the network of interactions between traumatic events and general and psychotic symptoms. Shortest paths analysis was performed to identify the different development trajectories. Results suggested that memory problems, compulsions, and irritability might be key mediating symptoms for most traumatic events. However, sexual abuse showed alternative mediators that might be specific of this traumatic event. Finally, the traumatic events, suffered from violence at work, separation/divorce and been homeless showed direct associations with specific psychotic symptoms.

Exposure to Traumatic Events and Development of Psychotic Symptoms in a Prison Population: A Network Analysis Approach

The development of the psychotic symptoms has been the topic of extensive debate (Sideli et al., 2012). Several perspectives have arisen that explain the emergence of psychotic symptoms with genetic variants (Zwicker et al., 2018), pre and perinatal neurodevelopment (e.g., maternal stress, maternal anemia, viral infection; Brown and Patterson, 2011; Fineberg et al., 2016; Nielsen et al., 2016) and substance use (e.g., cannabis, tobacco; Gurillo et al., 2015; Schlosser et al., 2012). Another perspective associates the psychotic symptoms with childhood adversities (Trotta et al., 2015) and repeated exposure to traumatic events (Schlosser et al., 2012). In fact, poly-victimization, that is, the experience of repeated traumatic experiences throughout the course of life, seems more common in individuals diagnosed with psychotic symptoms (Arseneault et al., 2011; Crush et al., 2018; Kelleher et al., 2013).

Incarcerated individuals seem to have been through more traumatic events than the general population (Baillargeon et al., 2009) and, in this population, the prevalence rates of psychotic disorders are 5 to 10 times higher than in the general population (Brugha et al., 2005). Indeed, psychotic symptoms have been continually associated with violent crime, particularly homicide (Fazel et al., 2009, 2014). This leads to a large number of prisoners with psychotic symptoms (Saavedra et al., 2017) in institutions that are historically unable to provide specialized treatment (Kinsler & Saxman, 2007).

Recent reviews suggest that traumatic events are indeed associated with the development of psychosis both in the general population (Varese et al., 2012) and in incarcerated individuals (Fazel et al., 2014). Unfortunately, the boundaries of what constitutes a traumatic event are unclear (Greenberg et al., 2015). So, for this study, we have broadened the definition of traumatic events, including some events that might be

otherwise considered psychosocial stressors. Regardless, it has been suggested that the relationship between these events and psychotic symptoms might be mediated by the individuals' neurodevelopmental characteristics (Read et al., 2014) and cognitive affective processes (Hardy et al., 2016).

Nevertheless, as pointed out by Bentall and colleagues (2014), these results implicitly assume a single pathway to psychosis, but its complex nature, the lack of support for its current categorical taxonomy, and the complex covariation of symptoms are unlikely to be the product of one single causal process. However, it's still not clear what these specific complex interactions are between traumatic events and psychotic symptoms (Gibson et al., 2016). In light of this, the authors initiated a search for relationships between exposure to specific traumatic events and psychotic symptoms and found that specific traumas have associations with specific psychotic symptoms (e.g., childhood sexual abuse was related with a high risk of developing hallucinations [Bentall, Wickham, Shevlin et al., 2012] and childhood neglect was associated with paranoid symptoms [Sitko, Bentall, Shevlin et al., 2014]). Also, Shevlin and colleagues (2015) found similar conclusions in a sample of incarcerated individuals, observing that prisoners with a past history of violence at home, institutional care, and bullying were more likely to develop paranoid symptoms.

Until now, studies exploring the association between exposure to traumatic events and psychotic symptoms assumed that these paths are direct; however, as aforementioned, there are potential mediators in this relationship. In fact, the role of general psychopathological symptoms has been recognized as a potential mediational mechanism between traumatic events and the development of psychotic symptoms (Isvoranu et al., 2017). Isvoranu and colleagues (2017) explored this by estimating a model of the complex network of interactions between traumatic events, general psychopathological symptoms,

and psychotic symptoms. In network models, these interactions are modeled as part of a causal system that contributes to explaining the underlying mechanisms that lead to the development of mental disorders (Borsboom & Cramer, 2013b). Recently, this approach has been providing a deeper understanding about the etiology of mental disorders (Borsboom & Cramer, 2013b), their nosography (Galderisi et al., 2018; van Rooijen et al., 2017; Wigman et al., 2017) and comorbidity structures (Cramer et al., 2010). Also, it was suggested that network models can help disentangle the mechanisms behind the relationship amongst traumatic events and the psychosis spectrum (Isvoranu et al., 2016). Unfortunately, only a strict number of studies have explored this relationship through this framework (Guloksuz et al., 2016; Isvoranu et al., 2016, 2017; Moffa et al., 2017). Guloksuz and colleagues (2016) showed that the exposure to environmental risk factors (childhood trauma, urbanicity, cannabis use, and discrimination) increases the connectivity of the network. This might mean that an increase in the exposure to environmental risk factors increases the expression of psychosis. However, this study only supports previous findings that partially explain this phenomenon, failing to clarify how traumatic events interact and possibly lead to the expression of psychotic symptoms. To solve this, Isvoranu and colleagues (2016) studied the specificity of interactions between environmental risk factors, general psychopathology, and psychotic symptoms. Their results point to the impact of traumatic events (i.e., physical neglect, emotional neglect, sexual abuse, emotional abuse, physical abuse) in psychotic symptoms being mostly mediated by general psychopathology. Moffa and colleagues (2017) studied the impact of bullying in the development of psychosis through network analysis and found a direct relation with persecutory ideation and a mediated relationship by general symptoms with hallucinations, which opens the possibility that some environmental risk and traumatic events are directly associated with psychotic symptoms whereas others are

indirectly associated with psychotic symptoms. This, however, awaits further clarification and replication. Furthermore, Isvoranu and colleagues (2016) and Moffa and colleagues (2017) focused on a limited selection of traumatic events and general psychopathological symptoms that might be oversimplifying the complex interaction between traumatic events and symptoms. In this context, we aim to contribute to the clarification of the interactions between traumatic events and psychotic symptoms by exploring the impact of diverse traumatic events associated with the development of psychosis and the mediational role of a large number of general psychopathological symptoms.

Method

Participants

We analyzed data from a previous epidemiological study, the *Survey of Psychiatric Morbidity Among Prisoners in England and Wales, 1997* (see Brugha et al., 2005, for a detailed description). This survey was carried out by the Social Survey Division of the Office for National Statistics and commissioned by the Department of Health of the UK (Singleton et al., 1997). This study assessed the prevalence of psychiatric morbidity among prisoners in order to inform general policy decisions (Singleton et al., 1997). A total of 131 penal establishments were included in the survey (Coid et al., 2002) and all the participants included in the study provided written informed consent. In this way, a group of psychiatric disorders were assessed, namely, personality disorders, psychosis, neurotic symptoms, suicide and alcohol misuse and dependence. Regarding psychosis, the disorders covered were schizophrenia and other non-organic psychotic disorders, and a set of affective psychosis, namely, manic episode, bipolar affective disorder, severe or recurrent depression with psychosis. The prevalence of any

functional psychosis was 7% for male sentenced, 10% for male remand and 14% for female (see Singleton et al., 1997, for a detailed description).

In the present study, we excluded the prisoners that had not been exposed to any traumatic event before reclusion, and a distinction by type of sentence wasn't performed. In this way, data from 3,039 prisoners were analyzed (75.4% male). There was no missing data in any of the variables in our study. Sociodemographic characteristics are presented in Table 1.

Table 1

Sociodemographic Characteristics of Participants

	<i>n (%)</i>
Sex	
Male	2291 (75.4)
Female	748 (24.6)
Age	
16 to 17	152 (5.0)
18 to 19	293 (9.6)
20	129 (4.2)
21 to 24	576 (19.0)
25 to 29	678 (22.3)
30 to 34	516 (17.0)
35 to 39	276 (9.1)
40 to 44	189 (6.2)
45 to 49	112 (3.7)
50 to 54	64 (2.1)
55 to 59	37 (1.2)
60+	17 (.6)
Marital Status	
Married	345 (11.4)
Cohabiting	838 (27.6)
Single	1450 (47.7)
Widowed/ divorced/separated	388 (12.8)
Ethnic Origin	
White	2442 (80.4)
Black - Caribbean	250 (8.2)
Black - African	115 (3.8)
Black – Other black group	45 (1.5)
Indian	38 (1.3)
Pakistani	32 (1.1)

Bangladeshi	8 (0.3)
Chinese	3 (0.1)
None of these	105 (3.5)
Education Levels	
Higher	391 (12.9)
GCES	725 (23.9)
Others qualifications	435 (14.3)
No qualifications	1482 (48.8)
Employment Status before Prison	
Working	1122 (36.9)
Seeking work	622 (20.5)
Living of crime	453 (14.9)
Economically inactive	798 (26.3)
Sentence Length	
Less than 3 months	28 (0.9)
3 - 5 months	86 (2.8)
6 – 11 months	151 (5.0)
1 -3 years	693 (22.8)
4 – 9 years	472 (15.5)
10 years or more	94 (3.1)
Life sentence	105 (3.5)
Crimes	
Violence	585 (19.2)
Sex offence	159 (5.2)
Robbery	1174 (38.6)
Frauds/ forgery	88 (2.9)
Drugs	558 (18.4)
Other	345 (11.4)
Unknown	125 (4.1)
Previous Criminal Convictions	
Yes	2289 (75.3)
No	750 (24.7)

Measures

Key Life Events and Posttraumatic Stress. The Key Life Events and Posttraumatic Stress questionnaire was used to assess the traumatic events. This questionnaire covers a list of traumas extracted from the “List of Threatening Experiences” (Brugha et al., 1985) and was presented to the participants in the form of cards. This way, prisoners were asked to look at the cards and acknowledge which events they endured.

The responses were coded as “Yes” or “No” and to ensure reliability of the answers the cards were presented by trained clinicians.

The traumatic events assessed were: suffered bullying; suffered violence at work; suffered violence at home; suffered sexual abuse; suffered serious illness/injury; suffered separation/divorced; suffered death of spouse/partner; suffered death of parent/sibling; suffered death of a close friend/relative; suffered stillbirth; expelled from school; been sacked /made redundant; run away from home; been homeless; and experienced serious money problems.

Psychosis Screening Questionnaire. In the *Survey of Psychiatric Morbidity Among Prisoners in England and Wales, 1997*, the Psychosis Screening Questionnaire (PSQ; Bebbington and Nayani, 1995) was used to assess positive psychotic symptoms. The PSQ is a 12-item questionnaire with five main items relative to hypomania, thought insertion, paranoia, strange experiences, and hallucinations. Participants answers to these items were used in the present study ($\alpha = .59$). In the original study, the complete PSQ displayed a sensitivity of 97% and a specificity of 95% (Bebbington & Nayani, 1995).

Clinical Interview Schedule—Revised. The Clinical Interview Schedule—Revised (CIS-R; Lewis and Pelosi, 1990) was used to evaluate the presence of general psychopathology symptoms as well assess their nature and severity. The CIS-R covers 14 sections: somatic symptoms, fatigue, concentration and forgetfulness, sleep problems, irritability, worry about physical health, depression, depression ideas, worry, anxiety, phobias, panic, compulsions, and obsessions. Moreover, in a series of studies, the CIS-R

showed a good interrater reliability ranging from .53 to .56 (Glyn Lewis et al., 1992). The symptoms analyzed in the present study are detailed in Table 2.

Table 2

Items of Psychosis Screening Questionnaire and from the Revised Clinical Interview Schedule and List of the Traumatic Events

Item Label	Domain Color	Item Description	n (%)
te1	Blue	Suffered bullying	882 (29.0)
te2	Blue	Suffered violence at work	169 (5.6)
te3	Blue	Suffered domestic violence	1006 (33.1)
te4	Blue	Suffered sexual abuse	446 (4.7)
te5	Blue	Suffered serious illness/injury	486 (16.0)
te6	Blue	Suffered separation/divorced	1378 (45.3)
te7	Blue	Suffered death of spouse/partner	268 (8.8)
te8	Blue	Suffered death of parent/sibling	855 (28.1)
te9	Blue	Suffered death of close friend	1440 (47.4)
te10	Blue	Suffered stillbirth	256 (8.4)
te11	Blue	Expelled from school	1494 (49.2)
te12	Blue	Been sacked/ made redundant	1323 (43.5)
te13	Blue	Run away from home	1561 (51.4)
te14	Blue	Been homeless	1306 (43.0)
te15	Blue	Had serious money problems	1608 (52.9)
ps16	Red	Hipomania	1863 (61.3)
ps17	Red	Thought Insertion	829 (27.3)
ps18	Red	Paranoia	1821 (59.9)
ps19	Red	Strange Experiences	1228 (40.4)
ps20	Red	Hallucinations	638 (21.0)
gs21	Green	Loss of appetite	1127 (37.1)
gs22	Green	Weight loss	1065 (35.0)
gs23	Green	Increase in appetite	800 (26.3)
gs24	Green	Weight increase	967 (31.8)
gs25	Green	Somatic complains	1836 (60.4)
gs26	Green	Fatigue	1472 (48.4)
gs27	Green	Concentration problems	1395 (45.9)
gs28	Green	Memory problems	1355 (44.6)
gs29	Green	Insomnia	2031 (66.8)
gs30	Green	Irritability	1945 (64.0)
gs31	Green	Health concerns	1305 (42.9)
gs32	Green	Depressive mood	2359 (77.6)
gs33	Green	Anhedonia	1378 (45.3)
gs34	Green	Lack of sexual interest	710 (23.4)
gs35	Green	Restlessness	1165 (38.3)
gs36	Green	Do things more slowly	1025 (33.7)
gs37	Green	Quiet	1409 (46.4)
gs38	Green	Guilt	927 (30.5)

gs39	Green	Inferiority	807 (26.6)
gs40	Green	Hopelessness	1243 (40.9)
gs41	Green	Worry	2026 (66.7)
gs42	Green	Anxiety	1818 (59.8)
gs43	Green	Phobias	222 (7.3)
gs44	Green	Panic	646 (21.3)
gs45	Green	Compulsions	842 (27.7)
gs46	Green	Obsessions	1417 (46.6)
gs47	Green	Not worth living	1424 (46.9)
gs48	Green	Wished was death	1352 (44.5)
gs49	Green	Suicidal ideation	1376 (45.3)
gs50	Green	Suicidal attempt	855 (28.1)
gs51	Green	Flashbacks	682 (22.4)
gs52	Green	Nightmares	500 (16.5)
gs53	Green	Intrusive memories	601 (19.8)
gs54	Green	Avoidance	644 (21.2)

Network Estimation and Analysis

The Ising model, implemented in the IsingFit package (version 0.3.1.; Van Borkulo et al., 2014) for R (version 3.6.1; R Development Core Team, 2018) was used to estimate the network connections between traumatic events, general psychopathology symptoms, and positive psychotic symptoms. The graphical representation of the network was computed using the Fruchterman-Reingold layout algorithm (Fruchterman & Reingold, 1991) in the package qgraph (version 1.6.3; Epskamp et al., 2019) for R (version 3.6.1.; R Development Core Team, 2018). This package was also used to estimate the shortest paths between traumatic events and psychotic symptoms and the shortest paths analysis by gender. Dijkstra's algorithm was used to compute the shortest paths (Dijkstra, 1959).

The accuracy and stability of the network was analyzed with R package bootnet (version 1.3; Epskamp & Fried, 2020), which estimates 95% bootstrapped confidence intervals (CIs) for each one of the connections. The correlation stability coefficient (CS-Coefficient; Epskamp et al., 2018) was also computed using R package bootnet. The CS-Coefficient estimates the maximum number of cases that can be dropped from the original

data while maintaining a correlation of at least 0.7 (95%) between the original network and the one with the missing data (Epskamp et al., 2018b).

Results

Network Descriptives

Figure 1 represents the network of interactions between traumatic events, general psychopathological symptoms, and positive psychotic symptoms. The network is constituted by 280 connections (density = .195), 272 positive connections, and eight negative connections. Positive connection weights varied between .012 and 2.814 ($M = .347$; $SD = .416$) and negative connections between .088 and 3.153 ($M = .937$; $SD = 1.149$). One psychotic symptom, “hypomania” (ps16), and one traumatic event, “suffered death of a close friend/relative” (te9), were isolated.

Regarding the stability of the network, the bootstrapped CIs and the CS-coefficient for the centrality strength was .75 and the CS-coefficient for betweenness centrality was .28. Closeness centrality did not show any variance. Analyses of network accuracy and stability are available in Figure 3, 4, 5 and 6 of supplementary materials.

Shortest Paths Between Traumatic Events and Psychotic Symptoms

Figure 2 summarizes the shortest paths between traumatic events and positive psychotic symptoms (shortest path for each traumatic event are available in Figure 2 of the supplementary materials). The shortest paths between each one of the traumatic events and the psychotic symptoms are detailed in the supplementary materials. Some traumatic events, namely, “suffered violence at work” (te2), “suffered separation/divorced” (te6), and “been homeless” (te14), showed direct associations with “strange experiences” (ps219), “thought insertion” (ps17), and “paranoia” (ps18), respectively. The remaining

traumatic events are indirectly associated with psychotic symptoms either through one of these traumatic events or through general symptoms. For example, the shortest path between the exposure to “had serious money problems” (te15) and “paranoia” (ps18) includes “been homeless” (te14). In fact, frequent connections between traumatic events were observed. Three general symptoms mediated the association between traumatic events and psychotic symptoms: “memory problems” (gs28), “irritability” (gs30), and “compulsions” (gs45). “Memory problems” (gs28) were implicated in the shortest paths involving exposure to “suffered violence at work” (te2) and “hallucinations” (ps20); “irritability” (gs30) was implicated in the shortest paths involving “suffered violence at home” (te3) and “paranoia” (ps18); and “compulsions” (gs45) were implicated in the shortest paths involving “expelled from school” (te11) and “hallucinations” (ps20).

The impact of “suffered sexual abuse” (te4) followed a different pattern. The shortest path between “suffered sexual abuse” (te4) and “hallucinations” (ps20) was mediated only by “suicide attempt” (gs50). Moreover, the shortest path from “suffered sexual abuse” (te4) to “paranoia” (ps18) was mediated by “suicide attempt” (gs50), “suicidal ideation” (gs49). In turn, the shortest path from “suffered sexual abuse” to “strange experiences (ps19) and “thought insertion” (ps17) was mediated by “avoidance” (gs54), “flashbacks” (gs51), “nightmares” (gs52), and symptoms associated with “panic” (gs44).

Traumatic Events, General Symptoms and Psychosis Network

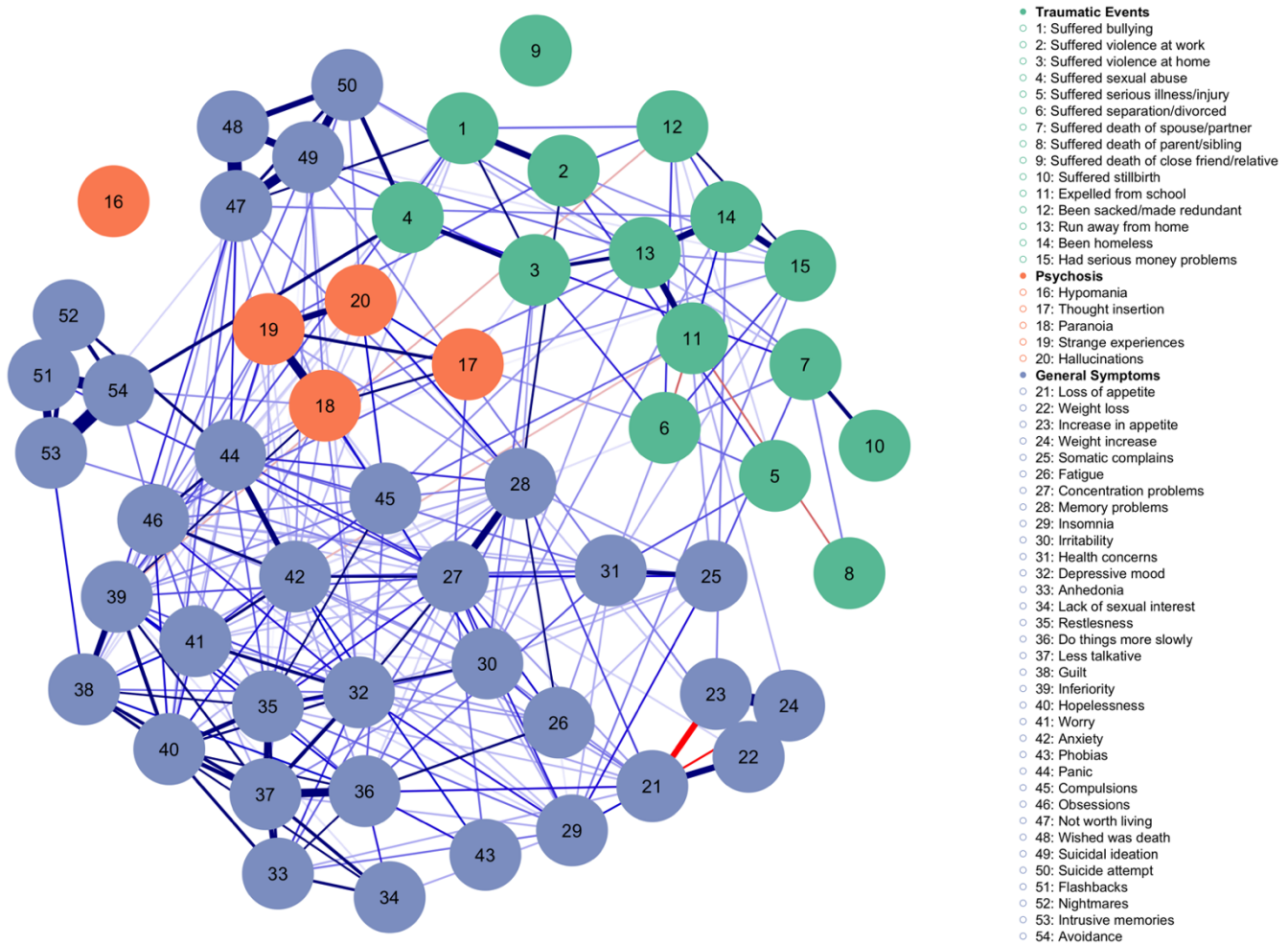


Figure 1. Network depicts the interactions between traumatic events, general symptoms and positive psychotic symptoms. Symptom groups are differentiated by colors. Green nodes represent traumatic events; orange nodes represent positive psychotic symptoms and blue nodes represent general symptoms. The thicker the lines are the stronger the connections between symptoms are. Positive correlations are represented in blue and negative correlations in red.

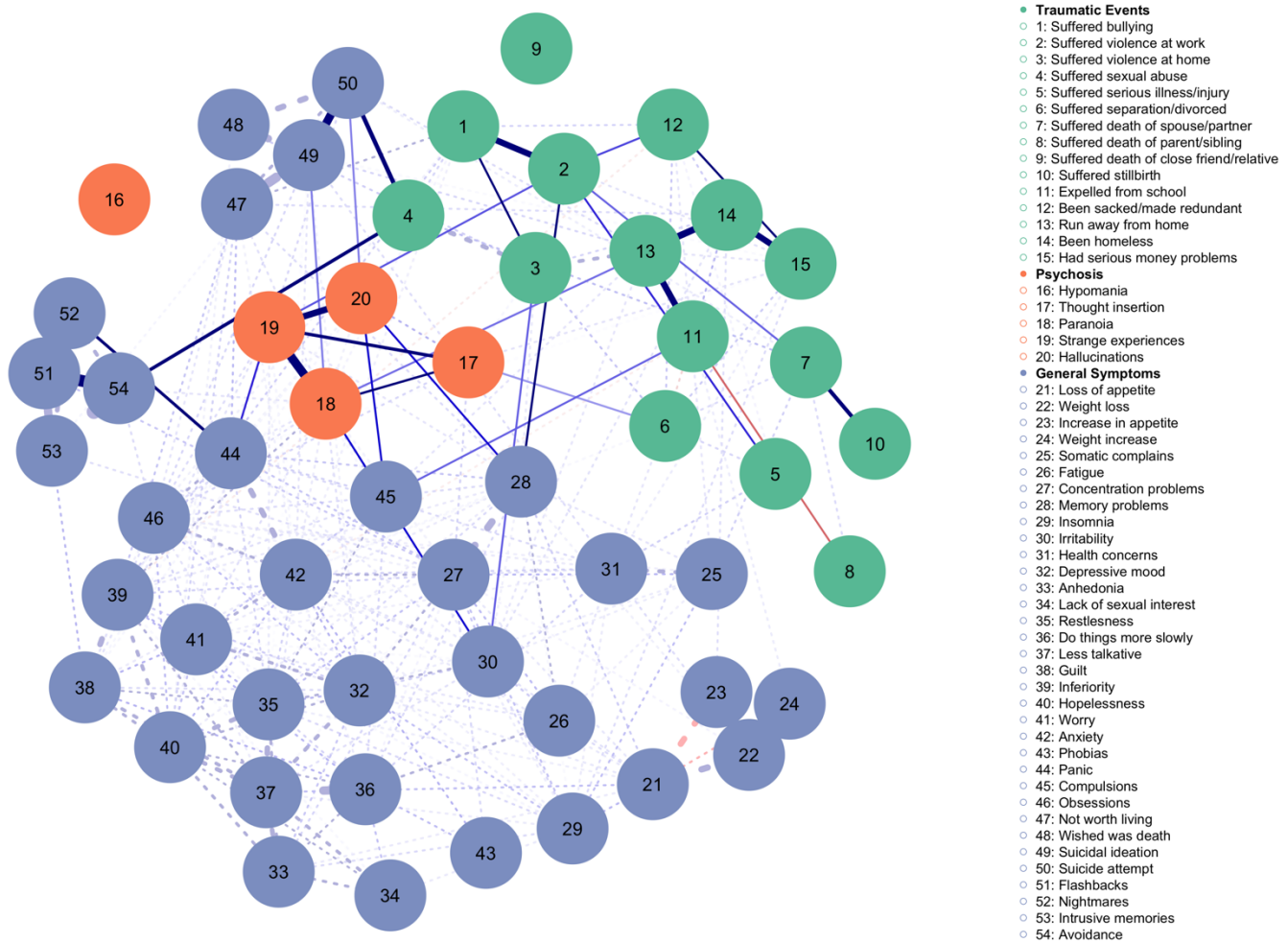
Shortest Paths from **Traumatic Events** to Psychosis

Figure 2. Network depicts the shortest paths between traumatic events, general symptoms and positive psychotic symptoms. Symptom groups are differentiated by colors. Green nodes represent traumatic events; orange nodes represent positive psychotic symptoms and blue nodes represent general symptoms. The thicker the lines are the stronger the connections between symptoms are. Positive correlations are represented in blue and negative correlations in red.

Discussion

In this study, we aimed to detail the specificity of the relationship between different traumatic events and symptoms of psychosis. Our results showed that the majority of the shortest paths between traumatic events and psychotic symptoms were mediated by symptoms of common mental health disorders. These results are in line with previous research in network analysis (Isvoranu et al., 2016, 2017; Moffa et al., 2017) and expand the earlier results by focusing on more general levels of trauma, not limiting to a particular traumatic event (i.e. childhood trauma).

Our results pointed to a restricted number of general symptoms involved in the paths between the traumatic events and psychotic symptoms, specifically, memory problems, compulsions, and irritability. Memory problems are common after exposure to a traumatic event (Brewin et al., 2007) and have been consistently associated with the development of psychosis (Bora & Murray, 2014; Geddes et al., 2016; Reichenberg et al., 2010). In particular, in this study memory problems were consistently implicated on the shortest path involving distinct traumatic events and hallucinations, which is consistent with the observation that the way trauma is cognitively processed can in part determine the occurrence of hallucinations (Geddes et al., 2016). Overall, these results suggest that difficulties in processing diverse traumatic experiences derived from cognitive problems may contribute to the emergence of psychotic symptoms, specifically hallucinations.

Compulsions were also implicated in most paths between traumatic events and psychotic symptoms, particularly hallucinations. Childhood adversities are one of the factors that contribute to the etiology of obsessive-compulsive disorders (Bey et al., 2017; Dhuri and Parkar, 2014.) and compulsions might be present in the prodromal period of psychosis (Bottas et al., 2005; Poyurovsky et al., 2012). Therefore, compulsions may

have an important role in the development of psychosis after a traumatic event. This is important since it might help explain the high comorbidity rates between obsessive-compulsive disorder and psychotic symptoms (Cunill et al., 2013) and the severity of the latest symptoms (Schreuder et al., 2017).

Irritability was also involved in the interaction between traumatic events and psychotic symptoms, which is congruent with previous studies that revealed that a history of childhood trauma leads to fear and anger (Russo et al., 2015) that can turn into irritability due to the inability of psychotic patients to recognize their emotions (Bilgi et al., 2017). This inability to recognize emotions in psychotic patients and the anger originated by childhood trauma might explain why irritability appears as a mediator. Also, it helps explain the high rates of violence, hostility, impulsivity, and poor impulse control in patients with psychotic symptoms (Fazel et al., 2009; Witt et al., 2013).

A different set of general symptoms were implicated in the shortest paths between sexual abuse and psychotic symptoms related with suicide behaviors, traumatic symptoms, and panic. Suicidal behaviors are frequently reported by victims of sexual abuse (Bedi et al., 2012; Miller et al., 2017) and both are highly prevalent in individuals presenting psychotic disorders (Conus et al., 2010; Phillips et al., 2009). In the study of Kilcommons and Morrison, (2005), posttraumatic symptoms showed several associations with psychotic symptoms, and sexual assault was related specifically to hallucinations. In this study, this was also the case with the shortest path involving hallucinations and also paranoia. Another network study that explored the impact of sexual abuse in psychosis found that anxiety is involved the path between sexual abuse and psychosis (Isvoranu et al., 2017). This study found similar results, with panic intervening in the pathway between sexual abuse and psychosis along with posttraumatic stress symptoms.

In addition to these indirect paths between traumatic events and psychotic symptoms, direct paths were also observed. These traumatic events covered exposure to violence at work, separation/divorce and homelessness. Previous research has been acknowledging the relationship between work and mental health problems, such as, depression or anxiety (Bowling & Beehr, 2006; Nolfé et al., 2007). Yet, in a smaller extent, some research found the presence of psychotic symptoms among individuals that experienced violence, often recognized as bullying at work (Verkuil et al., 2015). In this way, work contexts depicted as authoritarian or in which a strong hierarchy is present (Ulaş et al., 2018), as well as, in environments where the realization of tasks is performed alone and with distress (Villotti et al., 2019) might favor the disposition to the development of psychotic symptoms.

Regarding separation/divorce, it has been argued that this condition is frequently experienced as a stressful event and is related with the development of mental health problems (Knöpfli et al., 2016). However, previous research has predominantly associated this situation with depression, anxiety and substance abuse disorders (e.g. Chatav and Whisman, 2007; Lin et al., 2019). Interestingly, in our study, we found a direct association with a psychotic symptom (i.e. thought insertion). We believe, that this finding might constitute a new insight about other possible mental health outcomes related with this stressful event. This way, we encourage future research to focus on this particular event and their possible mental health outcomes.

Likewise, homelessness is perceived as a distressing and painful situation whereby, are considered high-risk group for the development of psychotic symptoms (Ayano et al., 2019; Fazel et al., 2008). This might occur through the repeated exposure to stressful and traumatic situations over the course of life (Nilsson et al., 2019), which might predispose them to a more vulnerable state for the development of psychotic

symptoms. Nevertheless, research is limited about the interaction between these specific traumatic events and the development of psychotic symptoms. Importantly, these direct paths might also point to a lack of information in our network and the absence of other possible mediator symptoms for these specific events. Thus, our results open new venues for future research concerning the development of psychotic symptoms.

Lastly, we found various connections between different traumatic events. This result has also been found in similar studies using network analysis (Isvoranu et al., 2017), and it is consistent with the experience of repeated traumatic events (i.e., poly-victimization, which is more common in incarcerated and psychotic individuals; Arseneault et al., 2011; Crush et al., 2018; Kelleher et al., 2013).

All these results point to the benefits of network analysis on the examination of patterns of interaction, allowing for a more complex view of the interactions between symptoms and events, contributing to a better understanding of the specific relationships between traumatic events and psychotic symptoms.

Nevertheless, our results should be cautiously interpreted in light of some limitations of the present study. Firstly, our results pertain to a specific population, namely, a sample of prisoners, whereby the generalization of our findings to diagnosed patients' needs to be considered prudently. Secondly, we use cross-sectional data, whereby the positive psychotic symptoms would probably be at a subclinical level. Moreover, the use of cross-sectional data on a specific sample whose traumatic events were experienced at different ages may obscure developmental processes that modulate the psychological response to trauma (Sedgwick, 2014). In order to surpass these limitations, we suggest that future studies should perform clinical interviews to obtain a more comprehensive view of the different stages of the psychotic symptomatology and adopt an Ecological Momentary Assessment (EMA; Shiffman et al., 2008) design, in

which it would be possible to assess general and psychotic symptoms at different times after the traumatic event. The use of this experimental procedure would enable a more detailed view of the symptoms developmental processes. Also, it should be noted that while we have estimated the shortest paths between traumatic events and psychosis, the development of psychosis might occur through different and possibly longer paths. Likewise, the exclusive assessment of positive psychotic symptoms constitutes a limitation; future research should also include negative symptoms to unveil and clarify other possible relationships between traumatic events and psychotic symptoms. Furthermore, the shortest paths do not allow the inference of direction and causality effects in the network. One way to advance in this direction is to use Bayesian techniques, such as Directed Acyclic Graphs (Jones et al., 2018) to explore the causality of the interactions between traumatic events, general symptoms, and psychotic symptoms. Alternatively, experimental studies could constitute a possibility to overcome the difficulties in establishing causal effects in the network.

In conclusion, our findings open new insights by detailing a vast number of associations between traumatic events and general and psychotic symptoms, which for treatment purposes might help design specific interventions and prevent the development of the worst prognosis among prisoners.

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Final Considerations

As is debated throughout this dissertation, the nosographic structure of PTSD remains a topic of intensive debate (Birkeland et al., 2020). A diversity of symptomatic structures of PTSD have been proposed (Armour et al., 2016), and the methodological attempts to identify the nosographic structure of PTSD have been unsatisfactory (Benfer et al., 2018). Several explanations have been suggested and among them, the type of trauma has been gaining attention (Kelley et al., 2009). However, it remains unclear how the type of trauma influences the nosographic structure of PTSD and its impact on developing other mental disorders (Greene et al., 2016), such as psychosis (Swan et al., 2017). Specifically, it remains unclear how the type of trauma influences the development of psychotic symptoms, namely, if this impact is direct or mediated by other symptoms, such as PTSD symptoms.

The theory of psychopathological networks constitutes an alternative, promising approach to the traditional categorical and dimensional perspectives. However, despite the advances made, some limitations remain (Ross et al., 2018). As this is a new approach to psychopathology, it is only natural that the methodological repertoire within this field is also limited at this time. Consequently, the first steps in this dissertation intended to explore new methodological tools that could address the gaps in our knowledge on the nosographic structure of PTSD. To a significant extent, initial advances on the nosographic and comorbidity structures of mental disorders generated by this theory have been achieved based on the detection of communities or modules of symptoms within the psychopathological networks (McNally et al., 2017). This allowed for a better understanding of the symptomatic organization of mental disorders (Blanken et al., 2018). However, it lacked a framework that enabled a more fine-grained view of the networks' communities or modules.

In this context, the ModuLand (Kóvacs et al., 2010) was explored as an alternative method to address this topic. This method has several benefits compared to the usual module detection algorithms that are used in this domain. First, it identifies the hierarchical modular structure of psychopathological networks, which is essential because it allows a more detailed characterization of the modules and their interactions within and across the modules. Second, it provides a more detailed description of the modules and the symptoms' roles and functions in the network, which is needed to clarify potential mechanisms responsible for developing and maintaining mental conditions. And the mechanisms responsible for the emergence of the comorbidity structures. Third, it is more theoretically consistent with the core proposals of the theory of psychopathological networks, which may contribute to test the different assumptions proposed by the network theory. For example, it allows the estimation of two types of bridge symptoms proposed in the network theory (i.e., bridge and overlap symptoms), which have been suggested as potential treatment targets, since they might be responsible for the emergence of comorbidity structures. Therefore, evaluating this hypothesis contributes to the development and improvement of the theory of psychopathological networks and it can produce new insights for a sustainable theory about mental disorders.

ModuLand was initially tested in a network of personality traits (Chapter I). The structure of personality traits is a topic of intense debate in psychology (Cramer & Borsboom, 2015). Previous studies proposed different personality structures (e.g., Shedler & Westen, 2004; Wakabayashi, 2014), however, it is recognized that personality structure is hierarchical structured (McCrae & John, 1992; McCrae & Costa, 1997). Therefore, this psychological construct reveals an excellent model to assess the stability and efficacy of ModuLand. The results supported the conceptual validity of ModuLand in the context of psychological networks. It was found that the diverse traits are associated

with distinct roles and functions in the network (see Chapter I for a detailed view). For example, personality characteristics with high bridgeness levels seem to be positioned between the modules. Therefore, they might play an essential role in connecting the modules. The personality characteristics with high levels of overlap appeared to stabilize the network and might impact the network's development. Bearing this in mind, modules with high levels of bridgeness (i.e., elements with high levels of bridgeness) seems to have an inter-modular role. In contrast, modules with high levels of overlap (i.e., elements with high levels of overlap) might have a trans-modular role. Also, in this study, it was found that psychological networks are distinct from random networks. This suggests that the theory of psychopathological networks could be a valid and alternative framework to the traditional models of conceptualization of mental disorders.

Towards these results and considering the proposal of the network theory of psychopathology that central and bridge symptoms might be important treatment targets, the deactivation of these symptoms could significantly impact the network of interactions of mental disorders. Specifically, it has been suggested that the deactivation of these symptoms might help disaggregate the network of interactions faster (Bekhuis et al., 2016; Robinaugh et al., 2016; Olatunji et al., 2018). However, evidence confirming this hypothesis is scarce, with several studies showing contradictory results (e.g., Snippe et al., 2017; Bos et al., 2018). This led to it being directly tested in Chapter II through an alternative methodological approach (compared to the usual frameworks in the field). Even though only moderated evidence was found regarding this hypothesis, it was found that degree centrality performed better in the identification of potential treatment targets, whereby future research should continue to explore the role of this measure. Also, this study's results provided further support to ModuLand's conceptual validity since it was found that bridgeness and overlap performed differently. Bridgeness appears to be a

measure important in increasing the number of components (i.e., isolated symptoms). On the other hand, symptoms with high overlap did not seem to impact the networks' disaggregation. These results open new pathways to the continuous exploration of the impact of different centrality measures and distinctive network properties in identifying potential therapeutic targets. In fact, in a follow-up study (Castro, Ferreira, & Ferreira, 2021) in which we explored the impact of the symptom deactivation in idiographic networks according to different centrality measures in several network properties. Results suggested that eigenvector centrality might identify symptoms with an important role in the network, which might be due to its close relationship with the causality (Dablander & Hinne, 2019). Regarding the network properties, we found that the deactivation of symptoms in idiographic networks promotes changes in the network structure that are distinct from those that occur in cross-sectional networks. In addition, these changes in the network structure were more significant if the centrality of each symptom was re-estimated each time a symptom was deactivated. These results might have important implications in future identification of potential treatments targets and the subsequential development of networks-based psychological treatments.

Considering the new insights obtained in these papers, ModuLand was then applied to identify the modular structure of PTSD in a community-based sample of adolescents exposed to a pipeline explosion (Chapter III). The problems in the nosographic structure of PTSD in children and adolescents are even more accentuated than in adults since this structure has been explored predominantly with factor models developed for adults (Wang et al., 2013). Notably, ModuLand allowed the assertion of the modules responsible for sustaining the disorder and those that might impact the development of PTSD. The module that covered the reexperiencing and dysphoric symptoms seemed responsible for the maintenance of this mental disorder, due to its high

number of connections with the other symptoms that might help spread the information through the network. The module that encompassed arousal symptoms might be responsible for the development of PTSD. Since its symptoms are positioned between the modules, which might facilitate the route to other symptoms during the development. With this detailed characterization of the modules, it is possible to identify the symptom's role and functions, and it was found that the symptom nightmares showed high levels of centrality and bridgeness, suggesting that might be a valuable treatment target. Overall, through the use of ModuLand, this dissertation offered a new global and local perspective of the functioning of PTSD and contributed to identifying potential symptoms that might be valuable treatment targets due to their role in the network.

Another issue that awaits further clarification pertains to the role of the type of trauma in determining the nosographic structure of PTSD. To address this issue (Chapter IV), a new method was used (CoDiNA; Gysi et al., 2020) that provided a promising pathway to disentangle the prototypical network structure of PTSD and elucidate the symptomatic patterns that characterize exposure to specific types of trauma. In this context, we found a set of interactions between symptoms that were present regardless of the type of trauma which may constitute the prototypical network structure of PTSD. In turn, our results also point to a set of connections that might be specific to each type of trauma and symptoms that might be potential markers for each of the traumas. This detailed view contributes to inform and guide clinicians in their clinical practice by proposing possible treatment targets and delivering personalized treatments and more appropriate therapeutic techniques (Armour et al., 2017).

In line with the clarification of the nosographic structure and the impact of the type of trauma in the symptomatic organization of PTSD, evaluating the impact of traumatic events in general and PTSD symptoms might explain the emergence of

comorbidity structures. PTSD is a highly comorbid disorder (Greene et al., 2016). However, its association with psychotic disorders remains unclear (Swan et al., 2017). In this context, uncovering the different symptomatic trajectories between traumatic events and psychotic symptoms might provide clinicians with relevant information to prevent evolution into more severe and treatment-resistant mental disorders. Using a distance-based measure (i.e., shortest paths) in Chapter V, it was possible to unveil the general symptoms that mediate these trajectories between particular types of trauma and several mental disorders. These results provided further specificity to the results obtained in the previous Chapter. In Chapter IV, the symptom flashbacks was found to be a relevant symptom across the four types of trauma analyzed, which included sexual abuse, making it a potential marker for this trauma. In parallel, in Chapter V, this symptom mediated the relationship between sexual abuse and the development of psychotic symptoms. Bearing this in mind, flashbacks might be an important symptom for the identification the structure of interactions of PTSD due to sexual trauma and alert the clinicians to a potential treatment target that prevents the evolution of PTSD into a psychotic disorder.

Overall, this work clarified and detailed the innumerable associations between traumatic events and psychotic symptoms. Moreover, it opened new insights about the impact of the type of trauma in symptomatology, namely, that it might occur through distinctive patterns of symptoms. In turn, these different trajectories might help explain the diversity of nosographic and comorbidity structures found in the previous literature.

Altogether, this work contributed with significant advances to the psychological network field and PTSD by introducing and exploring new methodological frameworks that can deal with the complexity of this mental condition and surpass some of the obstacles found in previous studies. Notably, in clinical terms, it could help inform clinicians about the etiopathogenic and maintenance mechanisms of PTSD and,

consequently, provide effective treatment plans. Furthermore, this knowledge might help develop more precise and effective techniques and treatment protocols (Armour et al., 2017) to reduce this mental condition's burden and the high economic costs for society in research terms.

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Chapter I ^{1,2}

Modularity of the Personality Network

Note:

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Comparison between the Five Factor Model facets and factors, and the modular structure of the personality network

Table S1

IPIP-NEO-120 itens (Johnson, 2014), FFM facets and factors, Personality Network Modules

Personality characteristic	IPIP-NEO-120	Facets FFM	L2 Module	FFM Factors	L3 Module
31	Fear for the worst	Anxiety	L2Module1	Neuroticism	L3Module1
*1	Worry about things	Anxiety	L2Module1	Neuroticism	L3Module1
61	Am afraid of many things	Anxiety	L2Module10	Neuroticism	L3Module1
91	Get stressed out easily	Anxiety	L2Module10	Neuroticism	L3Module1
86	Feel that I'm unable to deal with things	Vulnerability	L2Module10	Neuroticism	L3Module1
56	Become overwhelmed by events	Vulnerability	L2Module10	Neuroticism	L3Module1
*. **26	Panic easily	Vulnerability	L2Module10	Neuroticism	L3Module1
116	Remain calm under preasure	Vulnerability	L2Module10	Neuroticism	L3Module1
101	Feel comfortable with myself	Depression	L2Module15	Neuroticism	L3Module1
*41	Dislike myself	Depression	L2Module15	Neuroticism	L3Module1
78	Dislike changes	Adventurousness	L2Module17	Openness to experience	L3Module1
18	Prefer variety to routine	Adventurousness	L2Module17	Openness to experience	L3Module1
108	Am attached to conventional ways	Adventurousness	L2Module17	Openness to experience	L3Module1
*. **48	Prefer to stick with things that I know	Adventurousness	L2Module17	Openness to experience	L3Module1
100	Leave my belongings around	Orderliness	L2Module24	Conscientiousness	L3Module1
40	Often forget to put things back in their proper place	Orderliness	L2Module24	Conscientiousness	L3Module1
*70	Leave a mess in my room	Orderliness	L2Module24	Conscientiousness	L3Module1
10	Like to tidy up	Orderliness	L2Module24	Conscientiousness	L3Module1
11	Often feel blue	Depression	L2Module25	Neuroticism	L3Module1
*71	Am often down in the dumps	Depression	L2Module25	Neuroticism	L3Module1
75	Break rules	Dutifulness	L2Module27	Conscientiousness	L3Module1
*82	Enjoy being reckless	Excitement Seeking	L2Module27	Extraversion	L3Module1
112	Act wild and crazy	Excitement Seeking	L2Module27	Extraversion	L3Module1
*109	Get back at others	Cooperation	L2Module31	Agreeableness	L3Module1
*111	Am able to control my cravings	Immoderation	L2Module32	Neuroticism	L3Module1
81	Easily resist temptations	Immoderation	L2Module32	Neuroticism	L3Module1
87	Love life	Cheerfulness	L2Module34	Extraversion	L3Module1
*117	Look at the side bright of life	Cheerfulness	L2Module34	Extraversion	L3Module1
27	Radiate joy	Cheerfulness	L2Module34	Extraversion	L3Module1
57	Have a lot of fun	Cheerfulness	L2Module34	Extraversion	L3Module1
60	Make rash decisions	Cautiousness	L2Module35	Conscientiousness	L3Module1
90	Rush into things	Cautiousness	L2Module35	Conscientiousness	L3Module1
30	Jump into things without thinking	Cautiousness	L2Module35	Conscientiousness	L3Module1
*120	Act without thinking	Cautiousness	L2Module35	Conscientiousness	L3Module1
51	Rarely overindulge	Immoderation	L2Module8	Neuroticism	L3Module1
*21	Go on binges	immoderation	L2Module8	Neuroticism	L3Module1
62	Avoid contact with others	Friendliness	L2Module12	Extraversion	L3Module2
*32	Feel comfortable around people	Friendliness	L2Module12	Extraversion	L3Module2
2	Make friends easily	Friendliness	L2Module14	Extraversion	L3Module2
7	Love large parties	Friendliness	L2Module14	Extraversion	L3Module2
97	Avoid crowds	Gregariousness	L2Module14	Extraversion	L3Module2
*. **37	Talk to a lot of different people at parties	Gregariousness	L2Module14	Extraversion	L3Module2
106	Am not bothered by difficult social situations	Self-Consciousness	L2Module14	Neuroticism	L3Module2
76	Only feel comfortable with friends	Self-Consciousness	L2Module14	Neuroticism	L3Module2
*16	Find it difficult to approach others	Self-Consciousness	L2Module14	Neuroticism	L3Module2
*52	Seek adventure	Excitement Seeking	L2Module18	Extraversion	L3Module2
22	Love excitement	Excitement Seeking	L2Module18	Extraversion	L3Module2
3	Have a vivid imagination	Imagination	L2Module19	Openness to experience	L3Module2
*63	Love to daydream	Imagination	L2Module19	Openness to experience	L3Module2

(continue)

Table S1

IPIP-NEO-120 itens (Johnson, 2014), FFM facets and factors, Personality Network Modules (continued)

Personality characteristic	IPIP-NEO-120	Facets FFM	L2 Module	FFM Factors	L3 Module
33	Enjoy wild flights of fantasy	Imagination	L2Module19	Openness to experience	L3Module2
93	Like to get lost in thought	Imagination	L2Module19	Openness to experience	L3Module2
34	Believe that others have good intentions	Trust	L2Module20	Agreeableness	L3Module2
4	Trust others	Trust	L2Module20	Agreeableness	L3Module2
94	Distrust people	Trust	L2Module20	Agreeableness	L3Module2
*64	Trust what people say	Trust	L2Module20	Agreeableness	L3Module2
92	Keep others at a distance	Friendliness	L2Module22	Extraversion	L3Module2
*67	Prefer to be alone	Gregariousness	L2Module22	Extraversion	L3Module2
46	Am afraid to draw attention to myself	Self-Consciousness	L2Module6	Neuroticism	L3Module2
59	Feel empathy for those who are worse off than myself	Sympathy	L2Module11	Agreeableness	L3Module3
119	Try not to think about the needy	Sympathy	L2Module11	Agreeableness	L3Module3
*29	Sympathize with the homeless	Sympathy	L2Module11	Agreeableness	L3Module3
*. **44	Am concerned about others	Altruism	L2Module16	Agreeableness	L3Module3
14	Love to help others	Altruism	L2Module16	Agreeableness	L3Module3
89	Am not interested in other people's problems	Sympathy	L2Module16	Agreeableness	L3Module3
43	Feel other's emotions	Emotionality	L2Module16	Openness to experience	L3Module3
104	Take no time for others	Altruism	L2Module30	Agreeableness	L3Module3
74	Am indifferent to the feelings of others	Altruism	L2Module30	Agreeableness	L3Module3
73	Rarely notice my emotional reactions	Emotionality	L2Module30	Openness to experience	L3Module3
*103	Don't understand people who get emotional	Emotionality	L2Module30	Openness to experience	L3Module3
13	Experience my emotions intensely	Emotionality	L2Module30	Openness to experience	L3Module3
58	Believe that is no absolute right or wrong	Liberalism	L2Module28	Openness to experience	L3Module4
118	Believe that we should be tough on crime	Liberalism	L2Module28	Openness to experience	L3Module4
28	Tend to vote for liberal political candidates	Liberalism	L2Module28	Openness to experience	L3Module4
*88	Tend to vote for conservative political	Liberalism	L2Module28	Openness to experience	L3Module4
96	Am not easily annoyed	Anger	L2Module13	Neuroticism	L3Module5
*36	Get irritated easily	Anger	L2Module13	Neuroticism	L3Module5
19	Love a good fight	Cooperation	L2Module21	Agreeableness	L3Module5
49	Yell at people	Cooperation	L2Module21	Agreeableness	L3Module5
79	Insult people	Cooperation	L2Module21	Agreeableness	L3Module5
6	Get angry easily	Anger	L2Module21	Neuroticism	L3Module5
*. **66	Lose my temper	Anger	L2Module21	Neuroticism	L3Module5
99	Obstruct other's plans	Morality	L2Module23	Agreeableness	L3Module6
39	Cheat to get ahead	Morality	L2Module23	Agreeableness	L3Module6
9	Use others for my own ends	Morality	L2Module23	Agreeableness	L3Module6
*. **69	Take advantage of others	Morality	L2Module23	Agreeableness	L3Module6
45	Tell the truth	Dutifulness	L2Module5	Conscientiousness	L3Module6
*15	Keep my promises	Dutifulness	L2Module5	Conscientiousness	L3Module6
105	Break my promises	Dutifulness	L2Module5	Conscientiousness	L3Module6
84	Have a high opinion of myself	Modesty	L2Module9	Agreeableness	L3Module6
114	Boast about my virtues	Modesty	L2Module9	Agreeableness	L3Module6
54	Think highly of myself	Modesty	L2Module9	Agreeableness	L3Module6
*24	Believe that I am better than others	Modesty	L2Module9	Agreeableness	L3Module6
55	Carry out my plans	Self-Discipline	L2Module2	Conscientiousness	L3Module7
25	Am always prepared	Self-Discipline	L2Module2	Conscientiousness	L3Module7
*5	Complete tasks sucessfully	Self-Efficacy	L2Module2	Conscientiousness	L3Module7
35	Excel in what I do	Sympathy	L2Module2	Conscientiousness	L3Module7
110	Put little time and effort into my work	Achievement-striving	L2Module26	Conscientiousness	L3Module7

(continue)

Table S1

IPIP-NEO-120 items (Johnson, 2014), FFM facets and factors, Personality Network Modules (continued)

Personality characteristic	IPIP-NEO-120	Facets FFM	L2 Module	FFM Factors	L3 Module
50	Do more than what's expected of me	Achievement-striving	L2Module26	Conscientiousness	L3Module7
*. **80	Do just enough work to get by	Achievement-striving	L2Module26	Conscientiousness	L3Module7
20	Work hard	Achievement-striving	L2Module26	Conscientiousness	L3Module7
65	Handle tasks smoothly	Self-Efficacy	L2Module29	Conscientiousness	L3Module7
*95	Know how to get things done	Self-Efficacy	L2Module29	Conscientiousness	L3Module7
72	Take control of things	Assertiveness	L2Module4	Extraversion	L3Module7
*12	Take charge	Assertiveness	L2Module4	Extraversion	L3Module7
42	Try to lead others	Assertiveness	L2Module4	Extraversion	L3Module7
102	Wait for others to lead the way	Assertiveness	L2Module4	Extraversion	L3Module7
115	Have difficult starting tasks	Self-Discipline	L2Module7	Conscientiousness	L3Module7
85	Waste my time	Self-Discipline	L2Module7	Conscientiousness	L3Module7
77	Do a lot in my spare time	Activity level	L2Module7	Extraversion	L3Module7
*17	Am always busy	Activity level	L2Module7	Extraversion	L3Module7
47	Am always on the go	Activity level	L2Module7	Extraversion	L3Module7
107	Like to take it easy	Activity level	L2Module7	Extraversion	L3Module7
98	Do not enjoy going to art museums	Artistic interests	L2Module3	Openness to experience	L3Module8
68	Do not like poetry	Artistic interests	L2Module3	Openness to experience	L3Module8
38	See beauty in things that others might not	Artistic interests	L2Module3	Openness to experience	L3Module8
*8	Believe in the importance of art	Artistic interests	L2Module3	Openness to experience	L3Module8
*. **113	Am not interested in theoretical discussions	Intellect	L2Module33	Openness to experience	L3Module8
53	Avoid philosophical discussions	Intellect	L2Module33	Openness to experience	L3Module8
83	Have difficulty understanding abstract ideas	Intellect	L2Module33	Openness to experience	L3Module8
23	Love to read challenging material	Intellect	L2Module33	Openness to experience	L3Module8

Note. * Modular cores in the second level, ** Modular cores in the third level

Global properties of the network

Table S2.

Global properties of the three hierarchical levels. Assortativity measures the level of homophily of the graph. A high coefficient of assortativity indicates that connected nodes have the similar values (Newman, 2002; 2003). Cohesion measures the node connectivity of the graph (White & Harary, 2001). Average path length calculates the average path length, calculating the shortest paths between all pairs of nodes (West, 1996). Density is the ratio of the number of edges and the number of possible edges (Wasserman & Faust, 1994). Transitivity is the ratio of triangles and the connected triples in the graph (Wasserman & Faust, 1994; Barrat, Barthélemy, Pastor-Satorras, & Vespignani, 2004). Radius it's a measure derived from eccentricity, which is the shortest path distance from the farthest other node in the network. The lowest eccentricity in the graph is called radius (Harary, 1994). Centralization calculates a graph-level centrality score based on the node-level centrality measure (Wasserman & Faust, 1994; Freeman, 1979). Diameter is the largest undirected shortest path connecting to nodes (Csardi & Nepusz, 2006). Number of components determines the maximal number of components of a graph (Csardi & Nepusz, 2006).

Network Property	Score		
	First level	Second level	Third level
Assortativity	-0.0207	-0.0085	--
Cohesion	38	32	8
Average Path Length	1.3823	1.0100	1
Density	0.6176	1	1
Transitivity	0.6298	0.9900	1
Radius	2	1	1
Centralization	0.172	0.0588	0
Diameter	0.0219	0.2662	1.9826
Number of components	1	1	1

Effective degree, community centrality, bridgeness and overlap

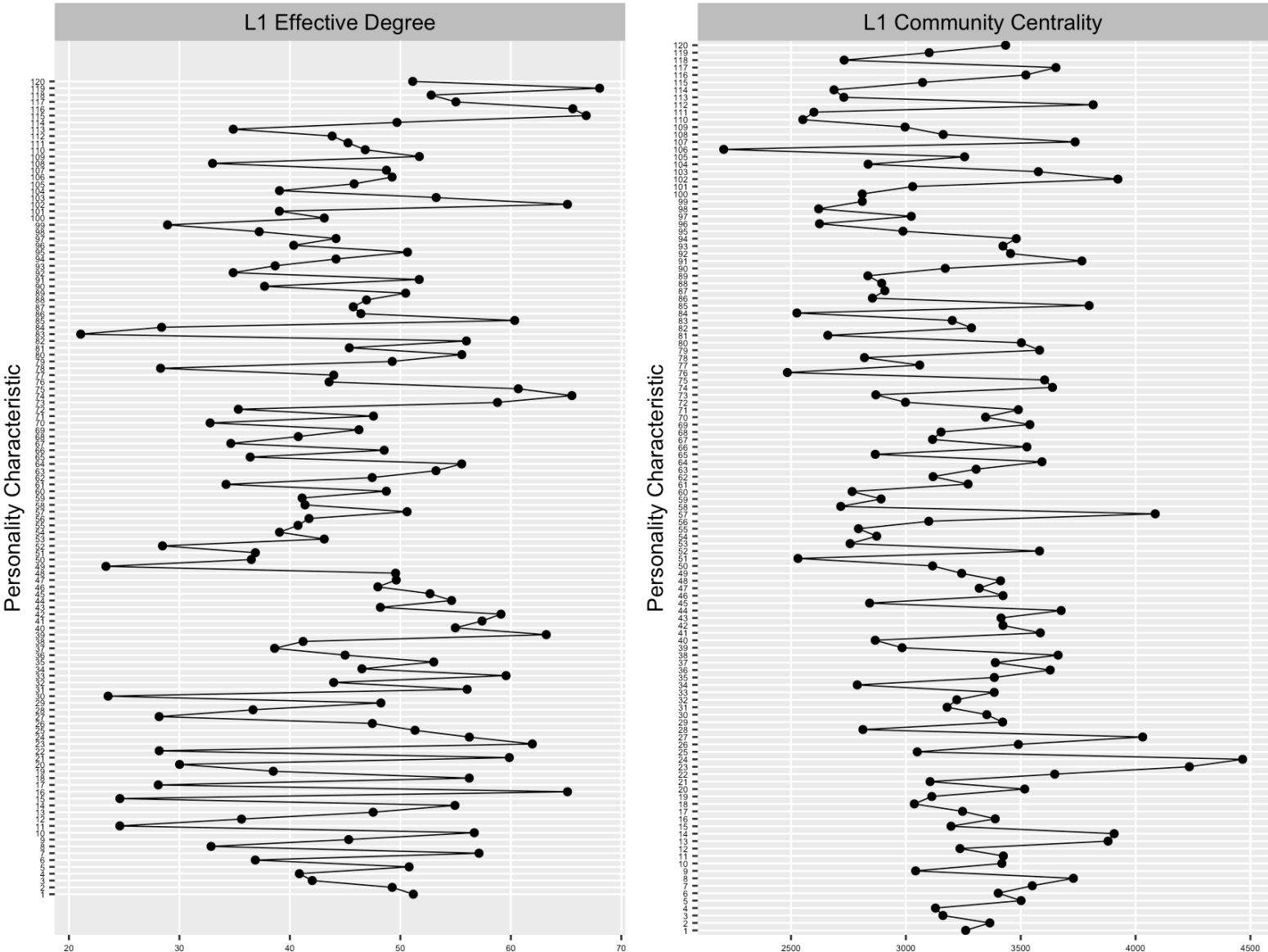


Figure S2. Community centrality and effective degree of the personality characteristics in the first level

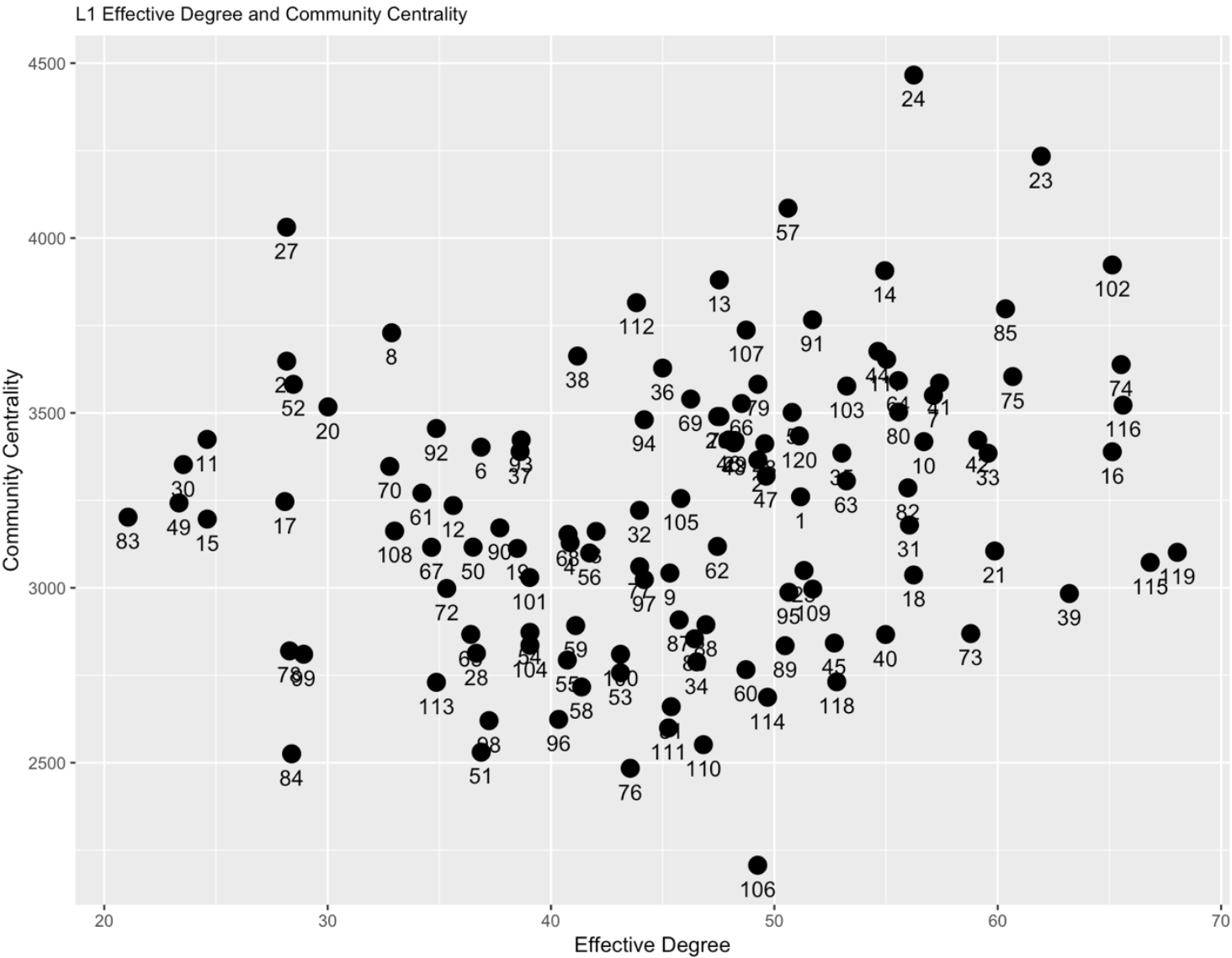


Figure S3. Joint representation of community centrality and effective degree in the first hierarchical level. Black dots represent the 120 items (i.e., personality characteristics) of the IPIP-NEO-120.

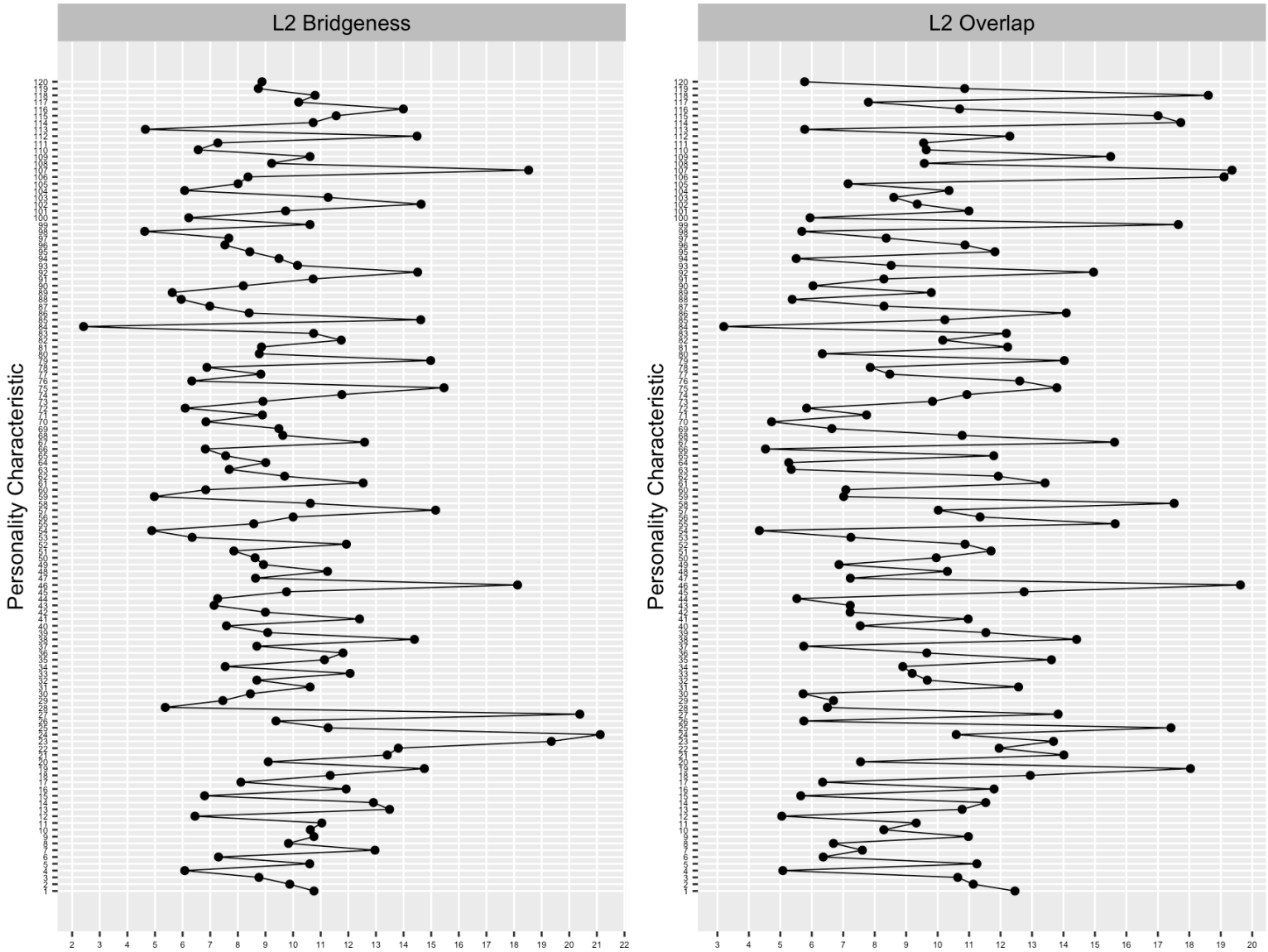


Figure S4. Bridgeness and overlap of the personality characteristics in the second level.

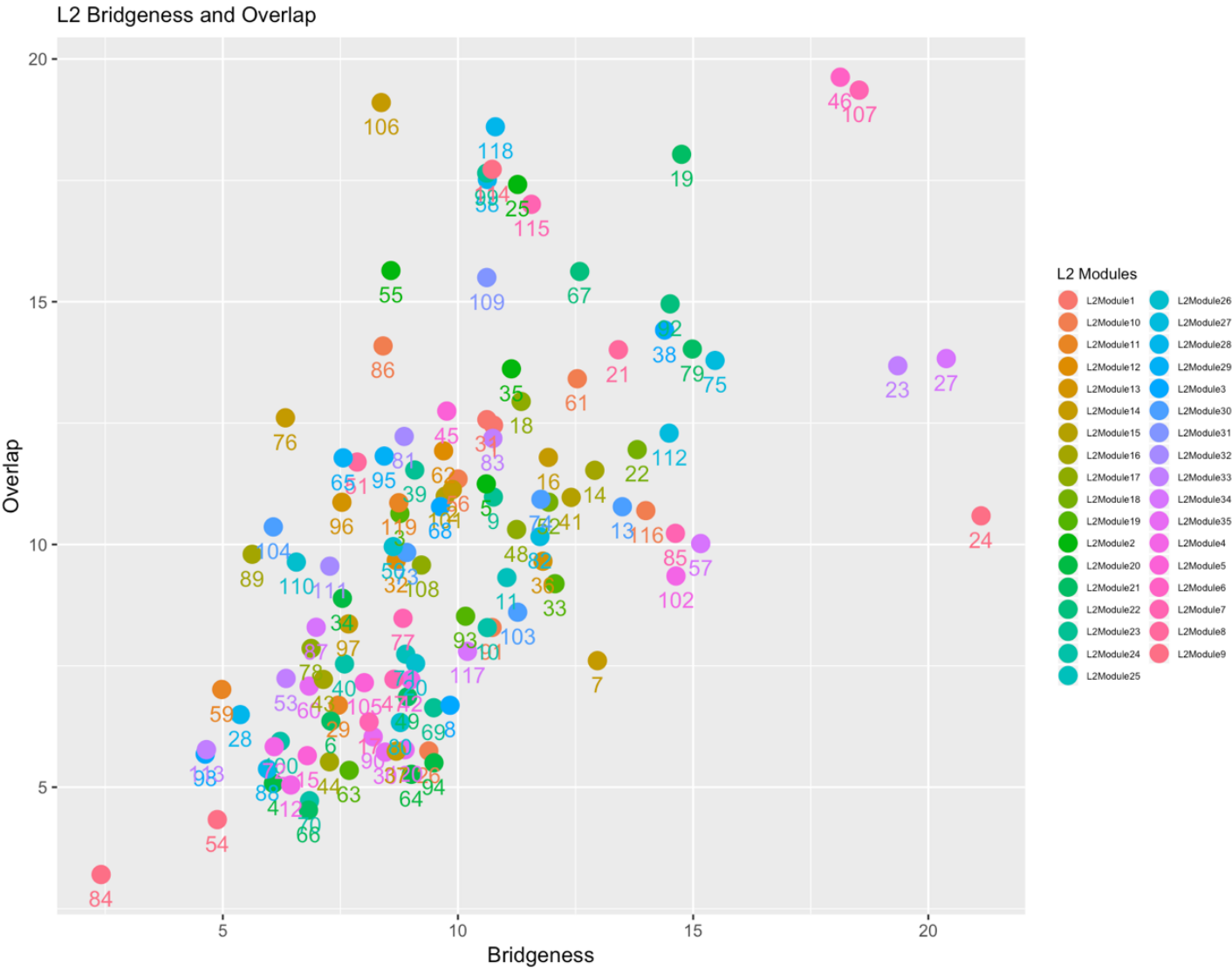


Figure S5. Joint representation of bridgeness and overlap in the second hierarchical level. Dots represent the 120 items (i.e., personality characteristics) of the IPIP-NEO-120. Each of the 35 modules is represented by a different color. Dots with the same color belong to the same module.

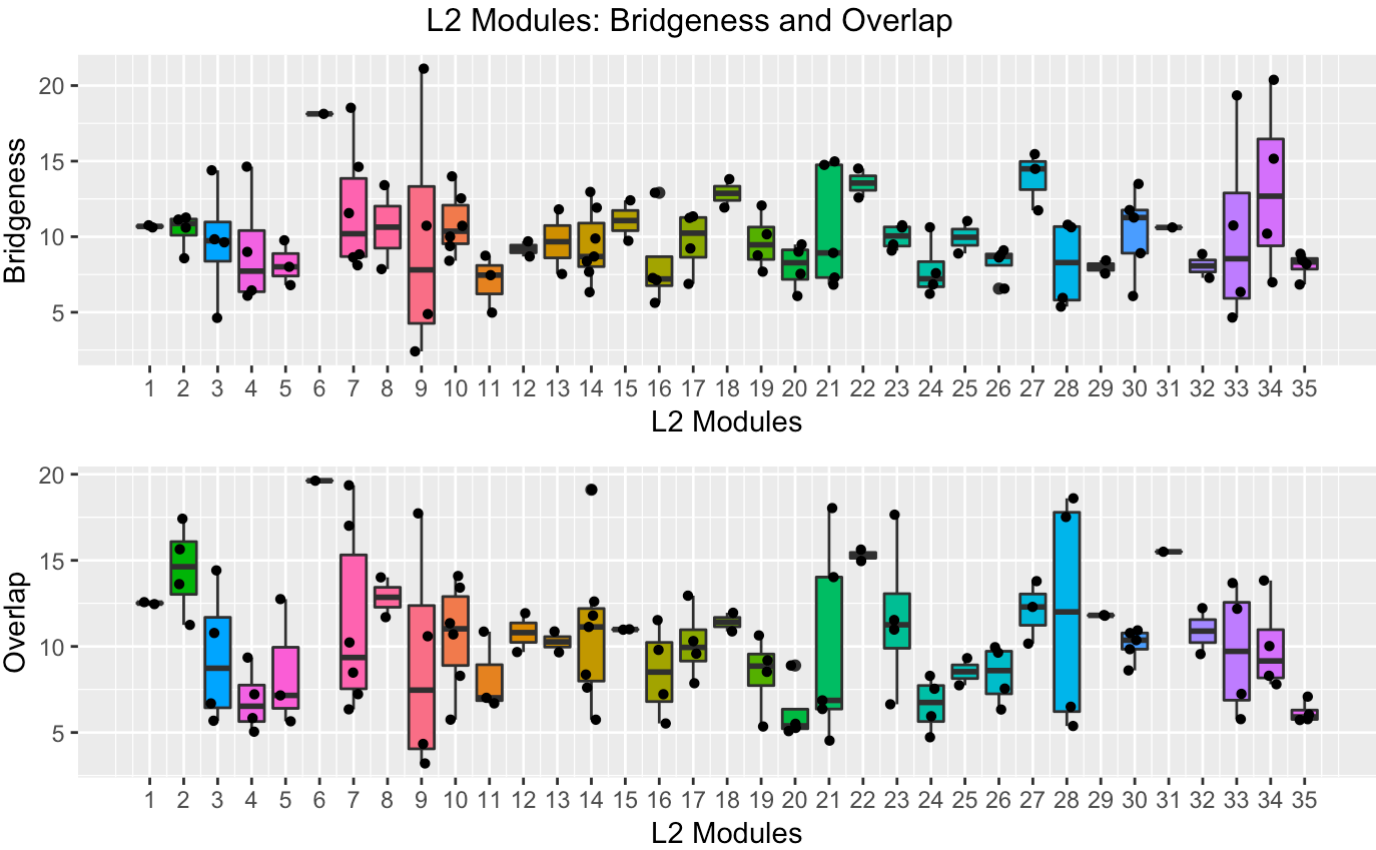


Figure S6. Overlap and bridgeness for each of the second level modules. Each boxplot corresponds to a second level module. Dots represent the personality characteristics in the module.

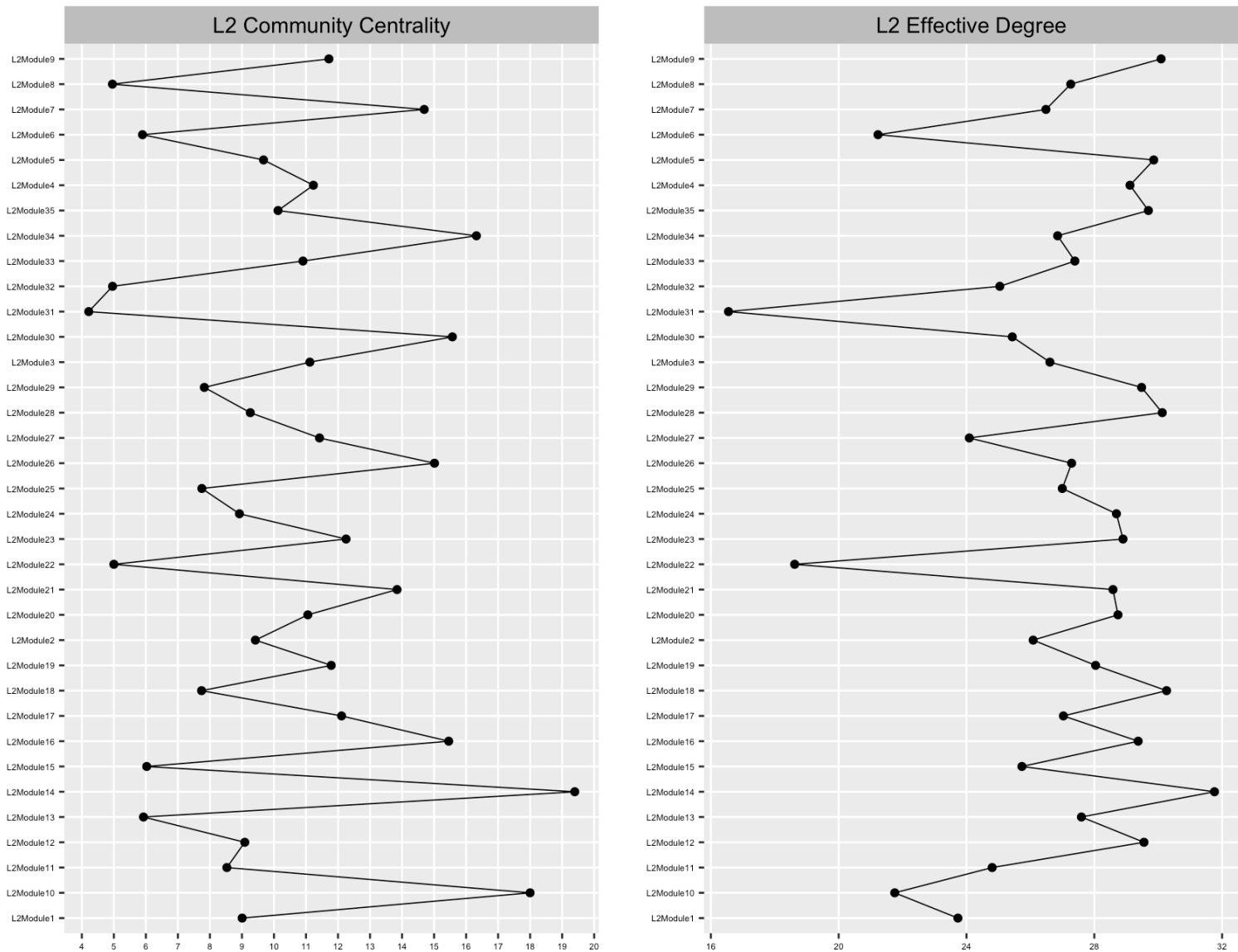


Figure S7. Community centrality and effective degree of the modules in the second level.

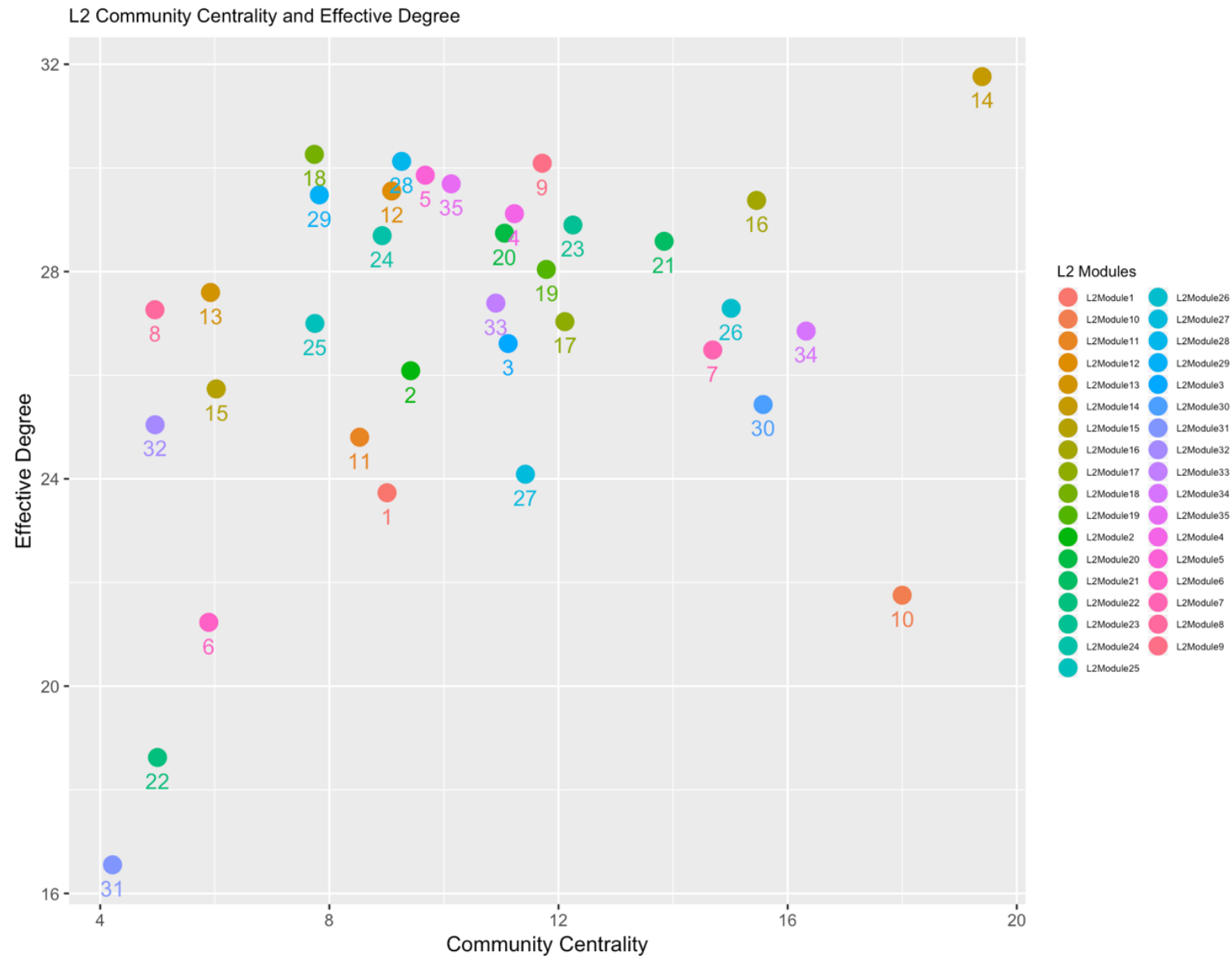


Figure S8. Joint representation of effective degree and community centrality in the second hierarchical level. Dots represent the 35 modules. Each module is represented by a different color.

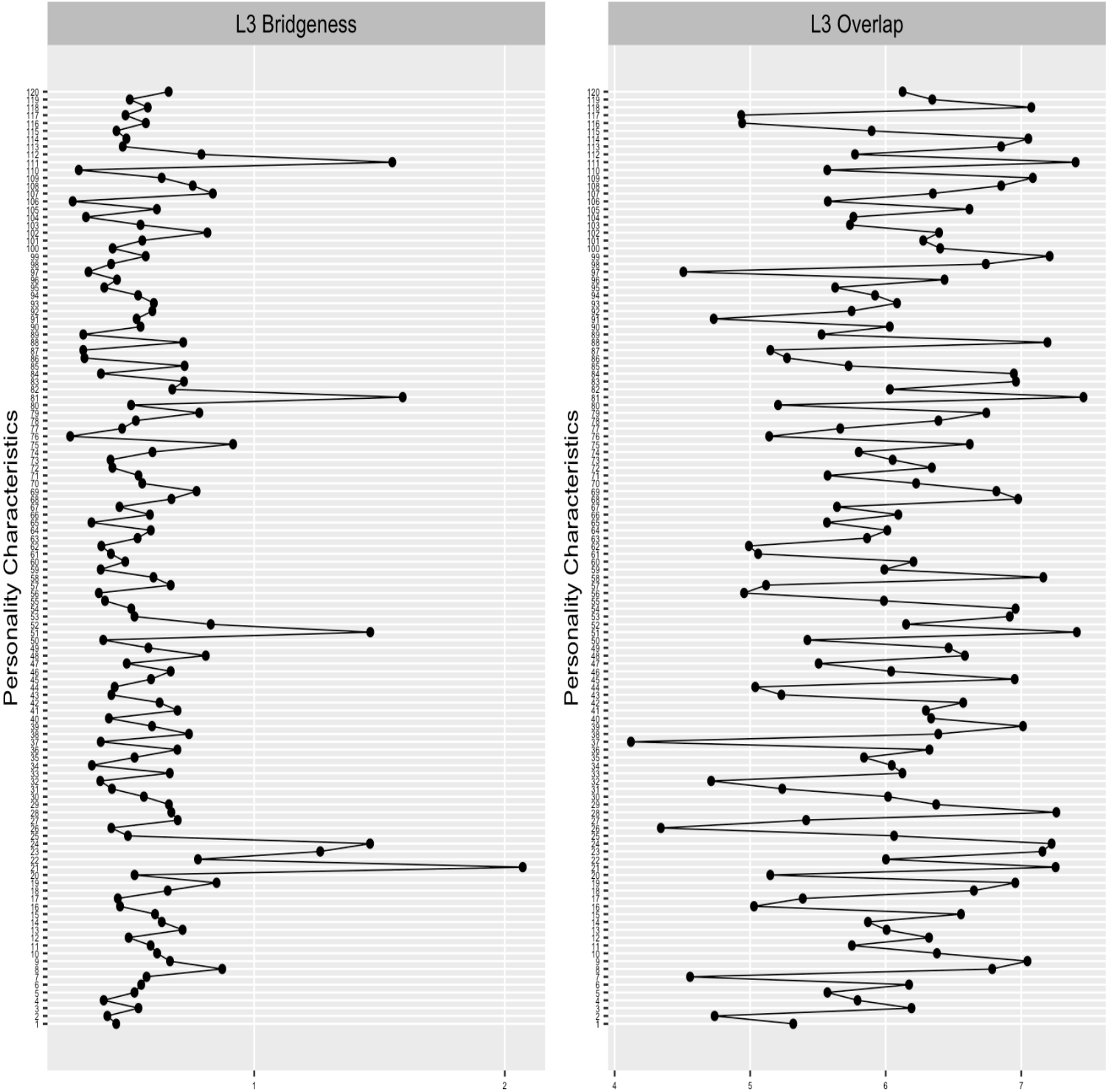


Figure S9. Bridgeness and overlap of the personality characteristics in the third level.

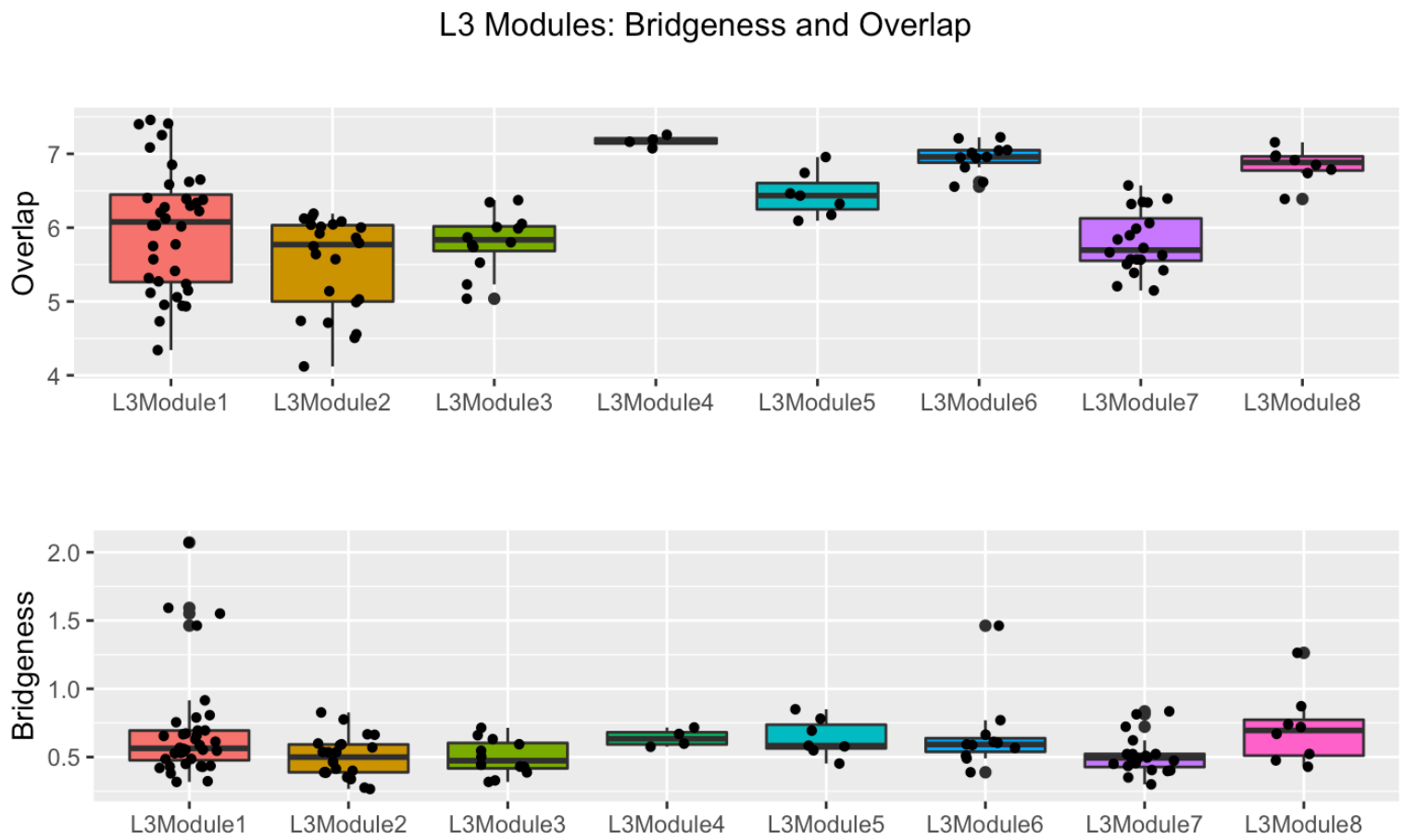


Figure S10. Overlap and bridgeness for each of the third level modules. Each boxplot corresponds to a third level module. Dots represent the personality characteristics in the module.

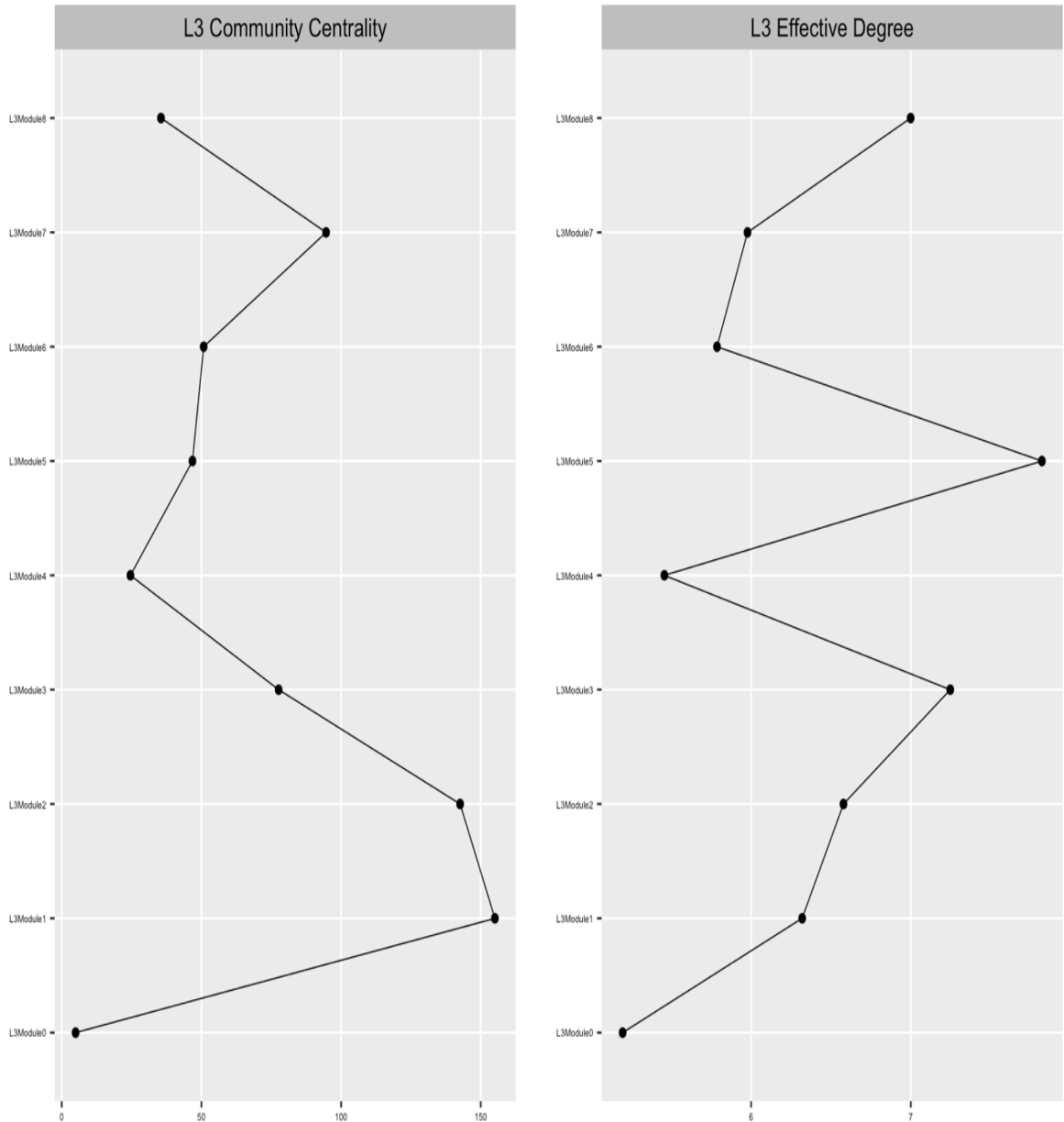


Figure S11. Community centrality and effective degree of the modules in the third hierarchical level.

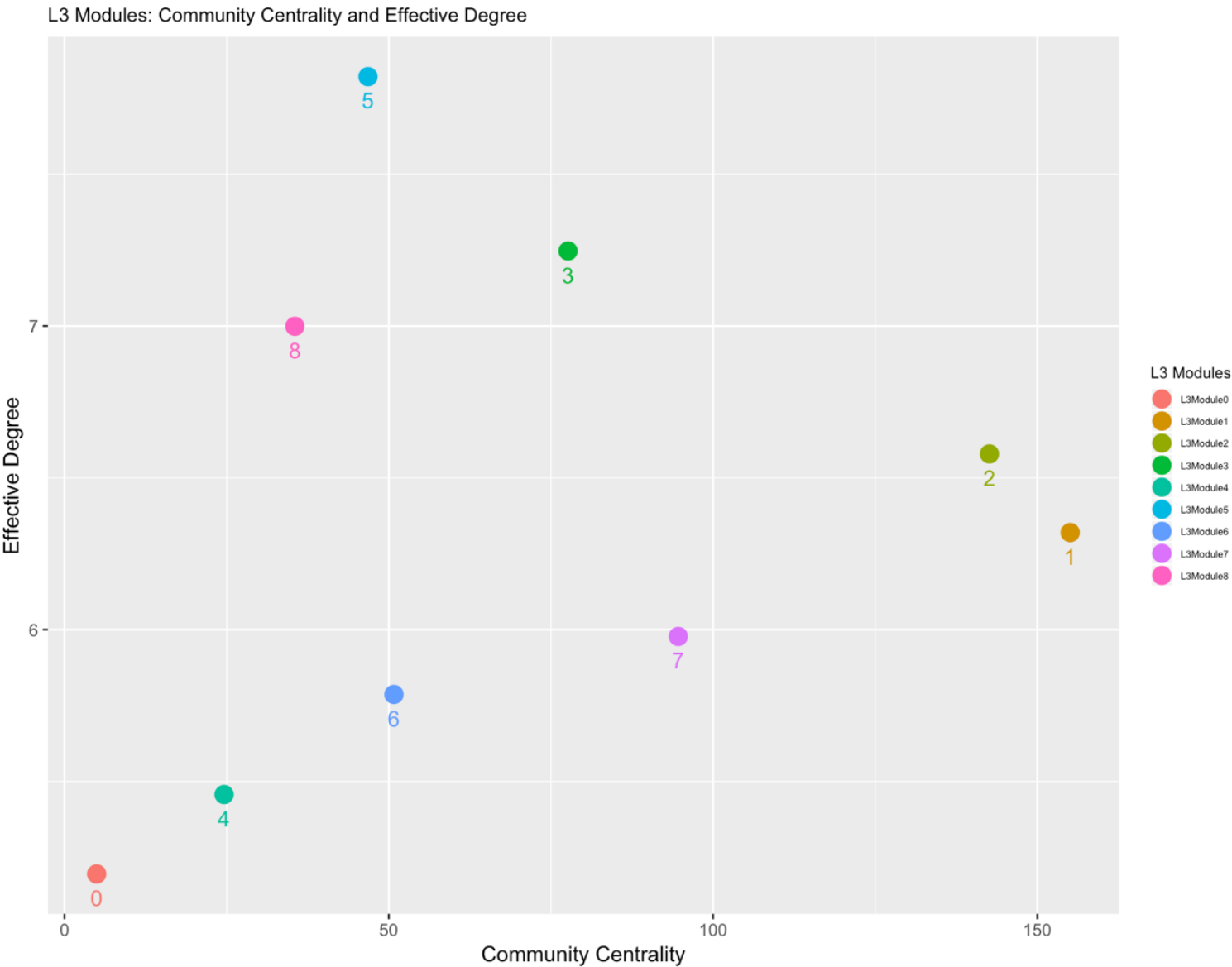


Figure S12. Joint representation of effective degree and community centrality in the third hierarchical level. Dots represent the 9 modules of this level. Each module is represented by a different color.

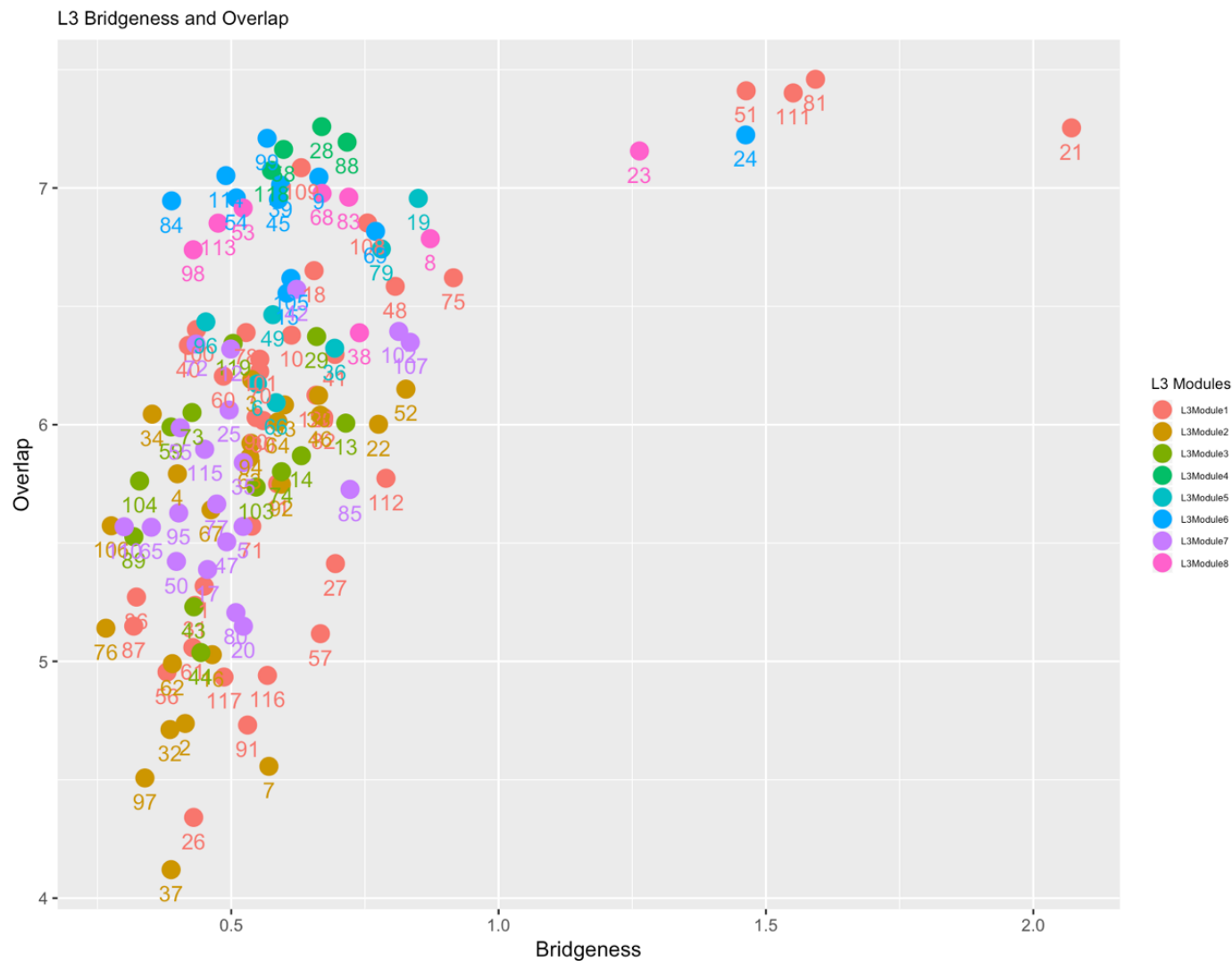


Figure S13. Joint representation of bridgeness and overlap in the third hierarchical level. Dots represent the 120 items (i.e., personality characteristics) of the IPIP-NEO-120. Each of the 9 modules is represented by a different color. Dots with the same color belong to the same module.

Correlation between modules' profiles

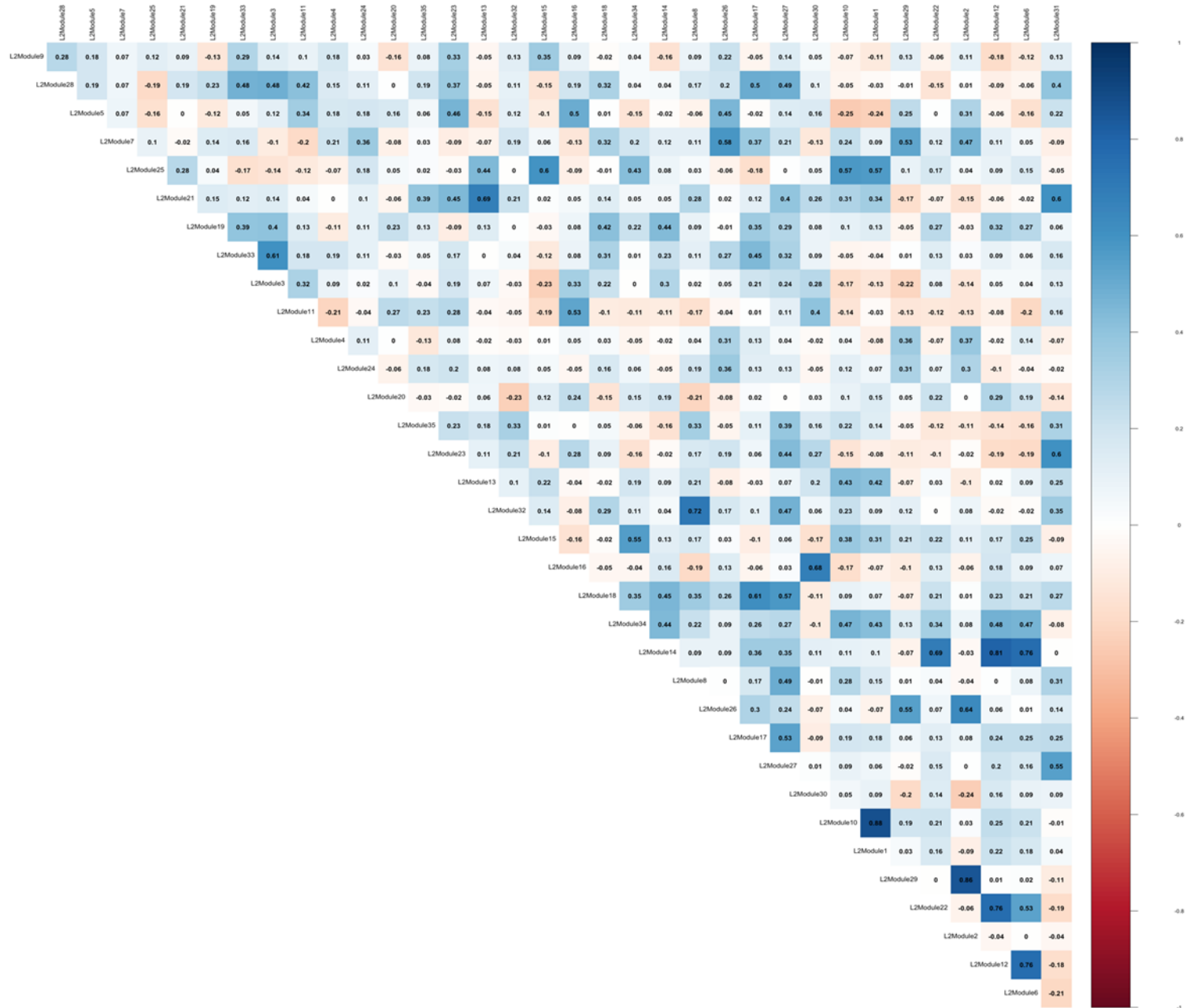


Figure S14. Correlations between the modules' profiles in the second level. Positive correlations are represented in blue and the negative correlations in red. Strength of the correlation is represented by the darkness of the respective color.

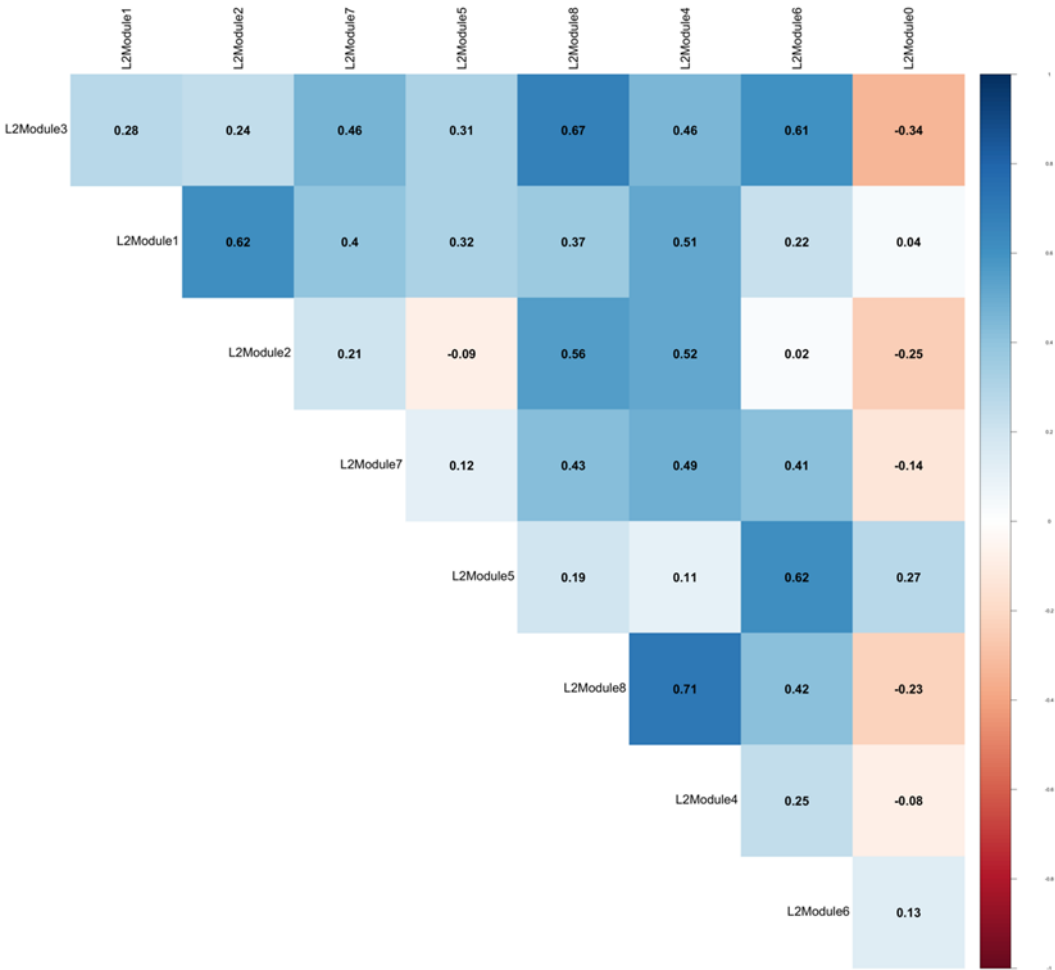


Figure S15. Correlations between modules' profiles in the third level. Positive correlations are represented in blue and the negative correlations in red. Strength of the correlation is represented by the darkness of the respective color.

Graphical representations of the network

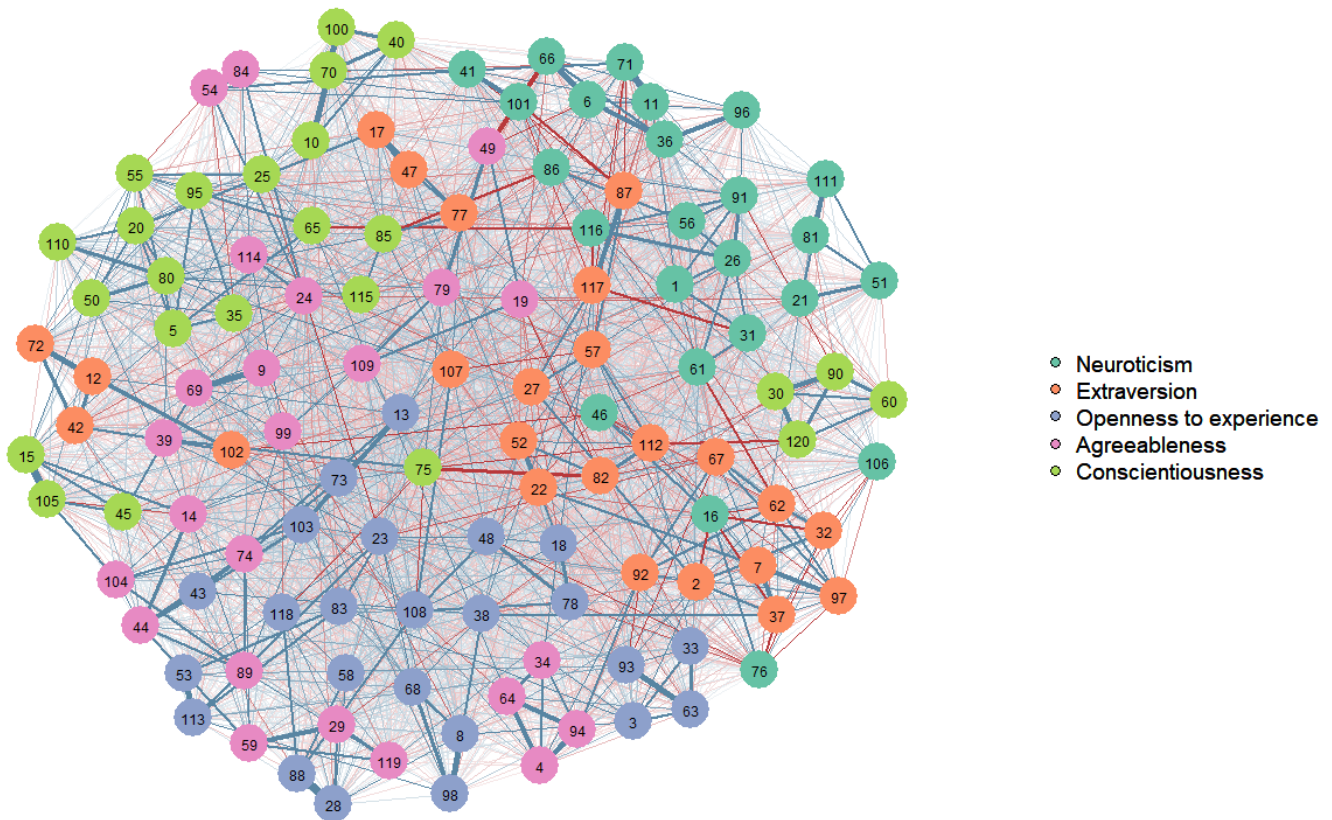


Figure S16. Graphical representation of the FFM on the non-thresholded personality network. Nodes represent the 120 personality characteristics (i.e., items) in the IPIP-NEO-120. Edges between the nodes represent the interaction between the nodes and the edges width represents the strength of the interaction. Nodes colors represent the FFM factors proposal. Green nodes correspond to the conscientiousness factor; orange nodes correspond to the extraversion factor; pink nodes represent the agreeableness factor; openness to experience is represented by the blue nodes; and the neuroticism factor is represented in ocean green.

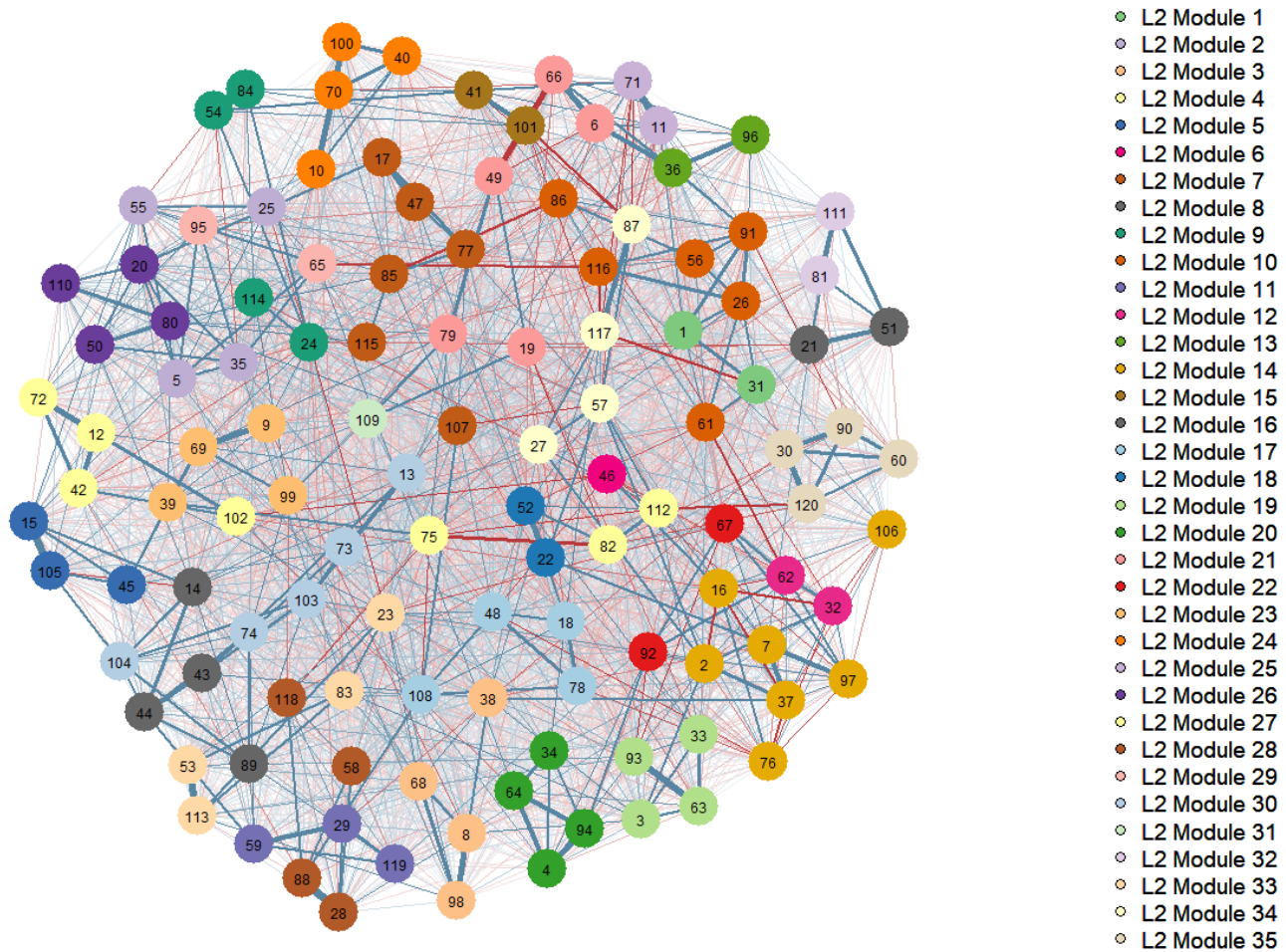


Figure S17. Graphical representation of the non-thresholded first level network with the second level modules projected. Partial correlation network. Nodes represent the 120 personality characteristics (i.e., items) in the IPIP-NEO-120. Edges between the nodes represent the interaction between the nodes and the edges width represents the strength of the interaction. Nodes colors represent 35 modules of the second level to which the node is most assigned to.

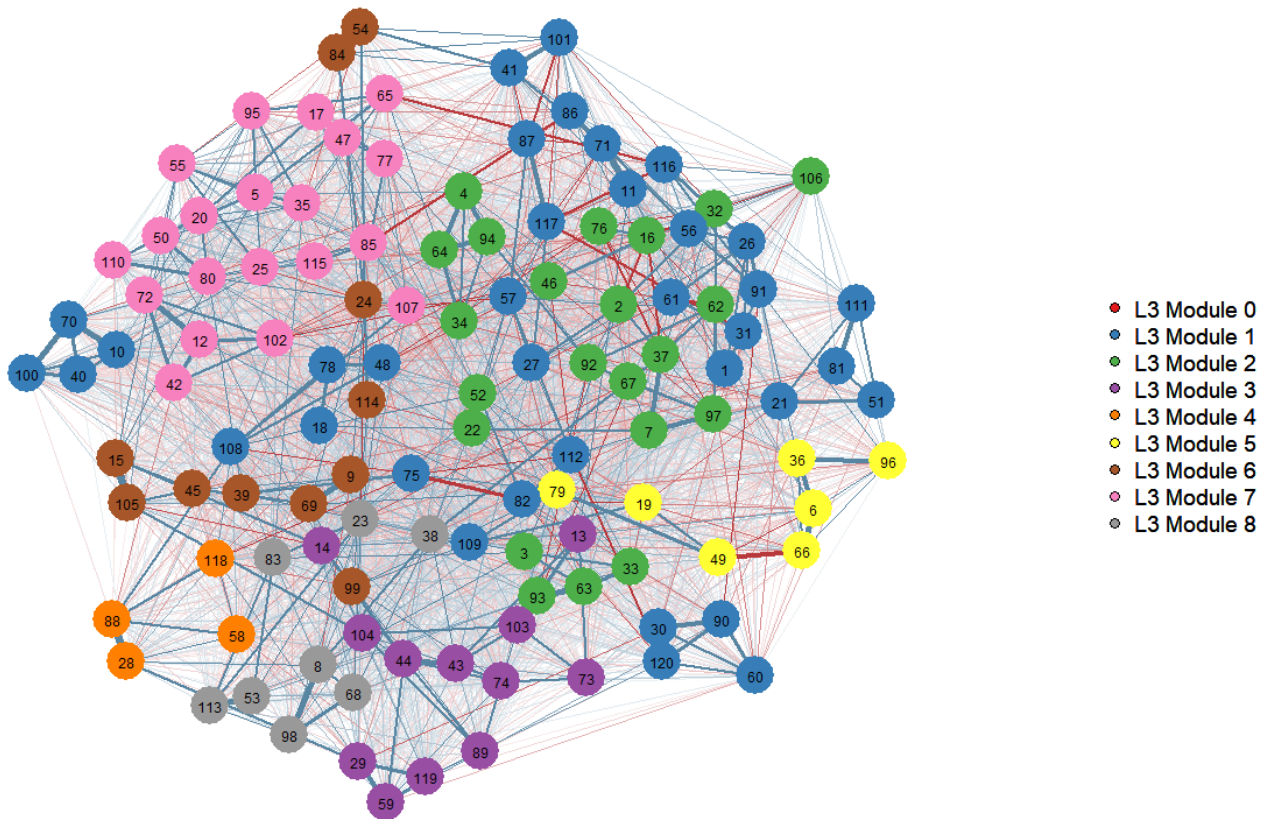


Figure S18. Graphical representation of the non-thresholded first level network with the third level modules projected. Partial correlation network. Nodes represent the 120 personality characteristics (i.e., items) in the IPIP-NEO-120. Edges between the nodes represent the interaction between the nodes and the edges width represents the strength of the interaction. Nodes colors represent the 9 modules of the third level to which the node is most assigned to. None of the nodes are colored with L3 Module 0, since none of them belong mostly to this module.

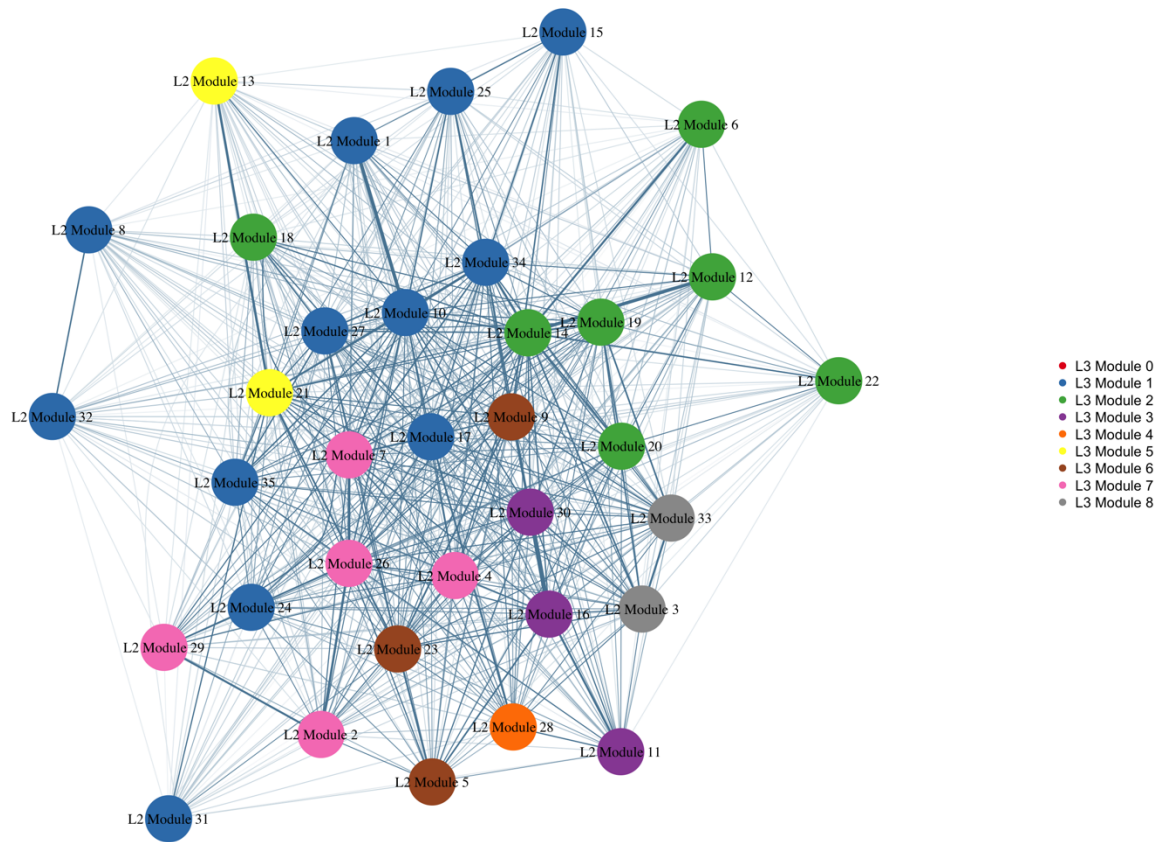


Figure S19. Graphical representation of the second level network with the third level modules projected. Overlap network. Nodes represent the 35 modules in the second hierarchical level. The colours of the nodes represent the third hierarchical level module to which the second level module is most assigned. None of the nodes have the colour of L3 Module 0, since none of the second level modules are mostly assigned to it. The edges between the nodes represent the overlap value between the modules, the higher the overlap value the larger the width of the edge.

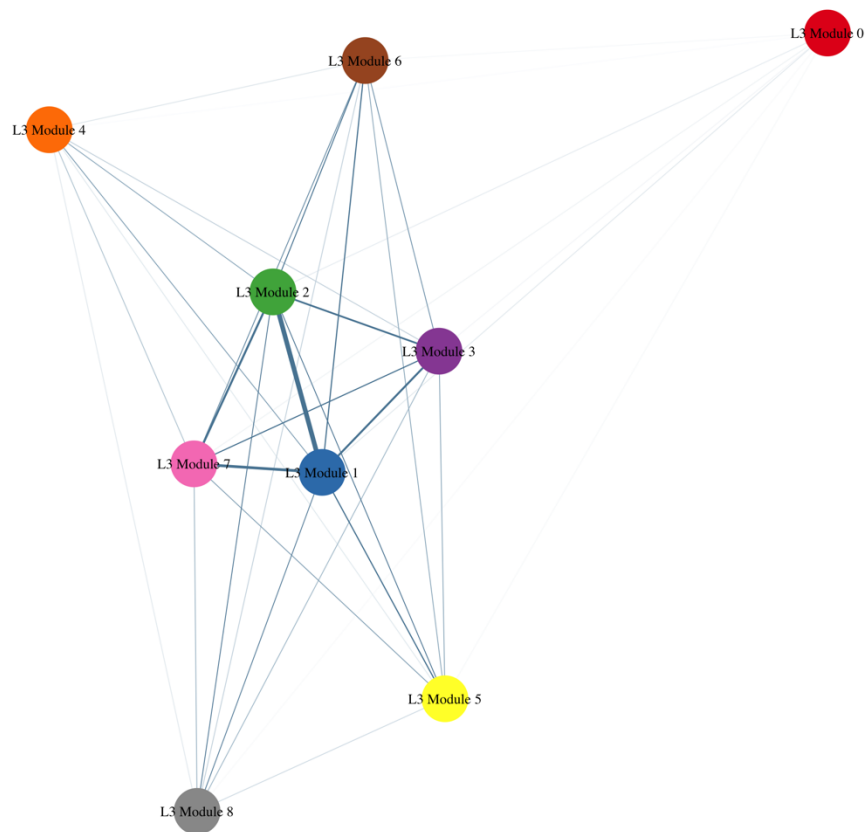


Figure S20. Graphical representation of the third level network. Overlap network. The nodes represent the 9 modules in the third hierarchical level. The edges between the nodes represent the overlap values between the modules, the higher the overlap value the larger the width of the edge.

Robustness of the personality network

As described in the main text, the analysis of the robustness of the personality network involved the comparison of the networks estimated from 100 pairs of sub-samples that were randomly generated from the initial sample. This comparison was performed on the following indicators: difference in the total number of estimated edges, similarity index, proportion of edges that failed to replicate, as well as correlations between edges weights and centrality measures (degree, closeness, betweenness, and expected influence) (see Borsboom, et al., 2017; Letina, Blanken, Deserno, & Borsboom, 2019). Results from this comparison are presented in Figure S19 bellow. As observed, results suggest that the personality network was highly robust and stable.

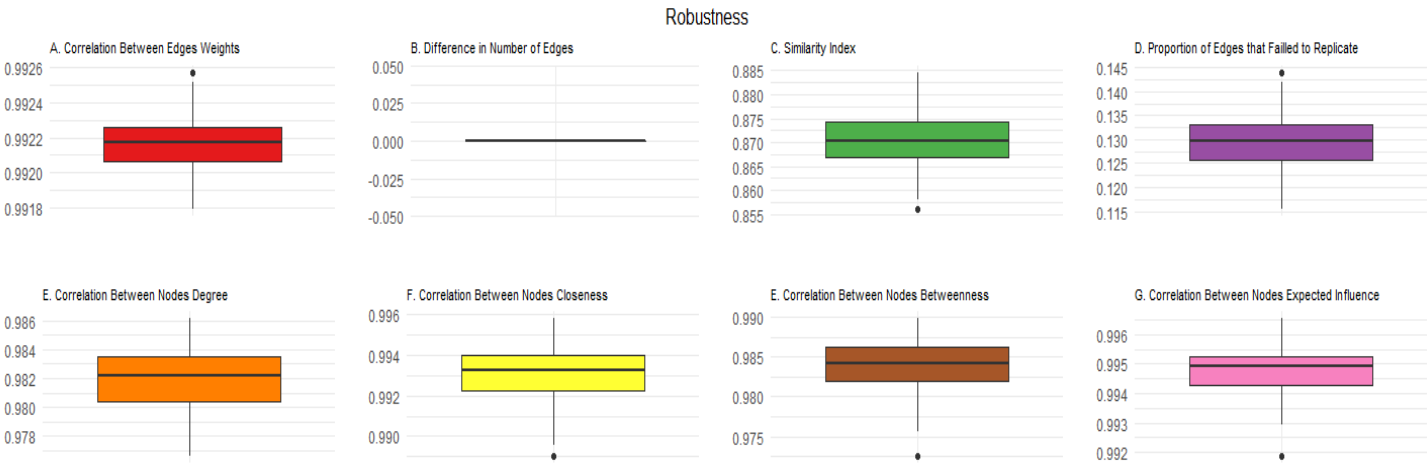


Figure S21. Robustness of the personality network

Comparison with unweighted null models

The properties of the personality network at the first level were compared to the same properties in three different unweighted null models. The networks' assortativity, transitivity, cohesion and centralization were used in this comparison. (See page 6 of the Supplementary Materials for a brief description of these properties.) Figures S20, S21, and S22 below depict the results. As observed in those Figures, the properties of the personality network are unlikely to be the result of random processes.

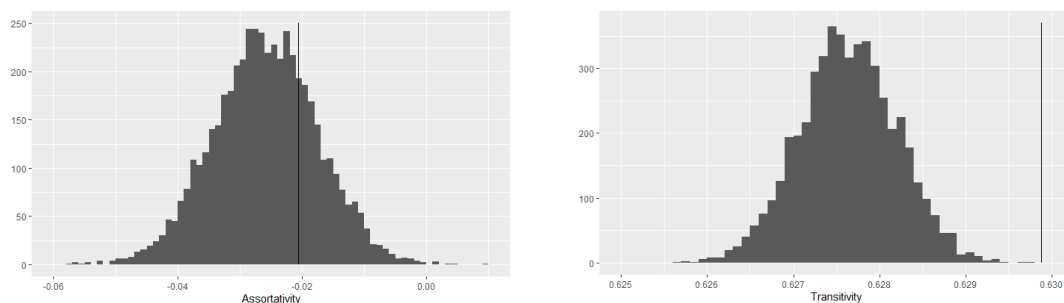


Figure S22. Histogram of the network properties in 5000 simulated networks obtained by randomizing the edges of the personality network while maintaining the same degree distribution. Vertical line identifies the observed value for each property in the personality network. Cohesion and centralization were not included because, as expected, simulated networks in this model have the same values as the personality network.

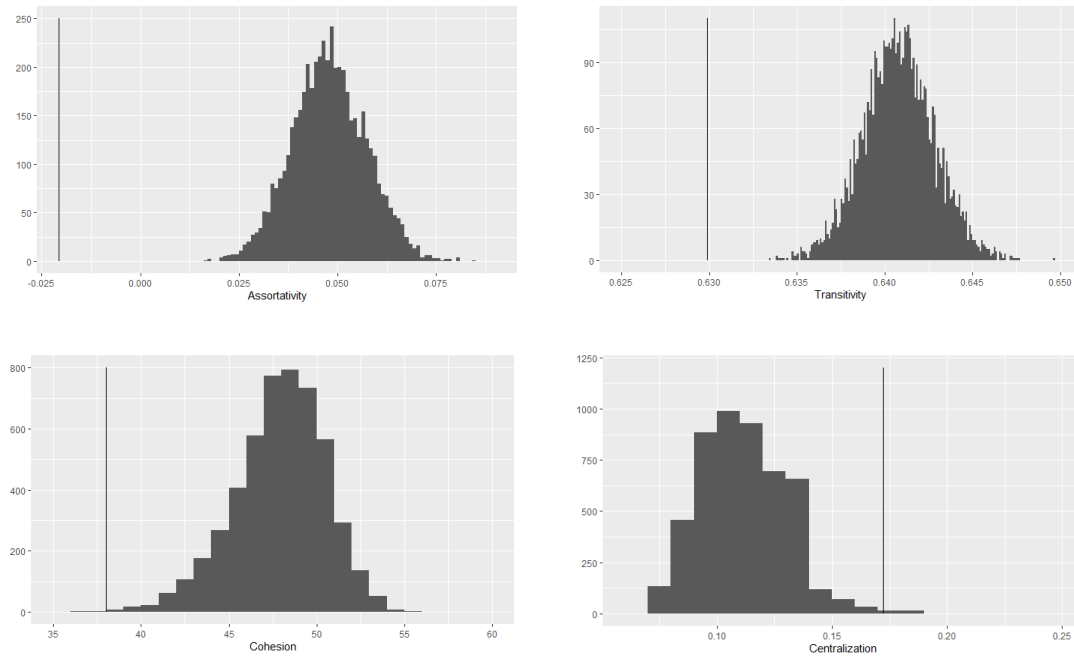


Figure S23. Histogram of the network properties in 5000 simulated networks obtained by randomizing the edges of the personality network. Vertical line identifies the observed value for each property in the personality network.

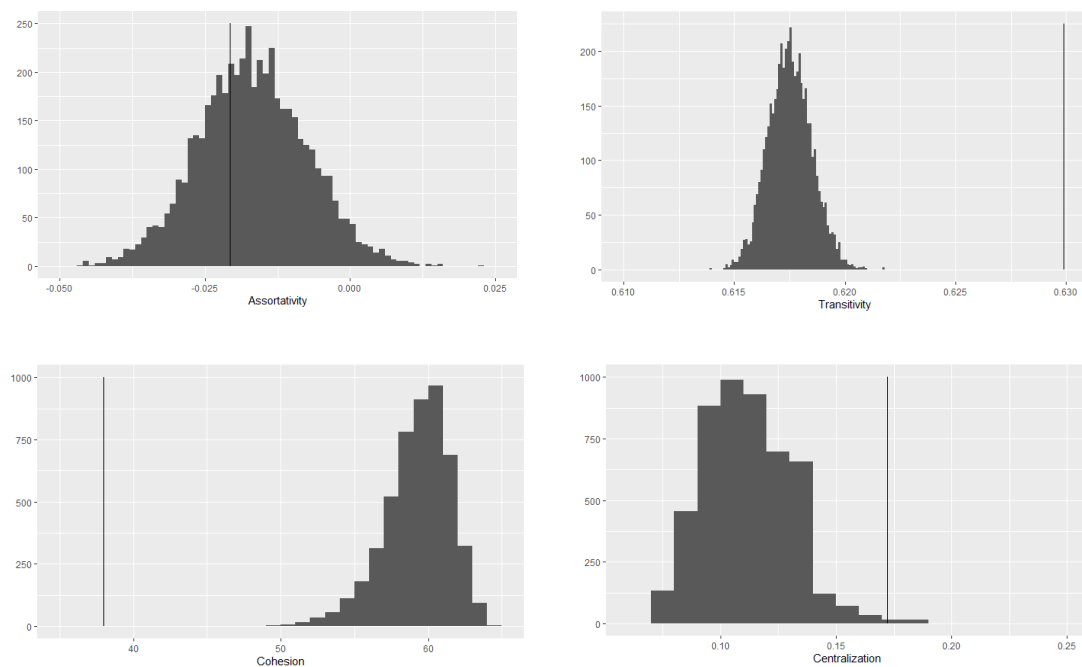


Figure S24. Histogram of the network properties in 5000 simulated networks obtained by generating a random graph with the same number of vertices and edges. Vertical line identifies the observed value for each property in the personality network.

Robustness of the personality network at different sample sizes

The procedure used to explore the personality network robustness (see p. 3 of the Supplementary Materials and main text) was repeated using randomly generated sub-samples of different sizes. Results are depicted in Figure S23 bellow. Overall, results suggest that with 1200 participants the proportion of edges that fail to replicate is consistently below 50%, and the similarity index consistently over 50%. However, with the exception of expected influence, the correlation between centrality measures is only consistently over .50 with samples with 2400 participants.

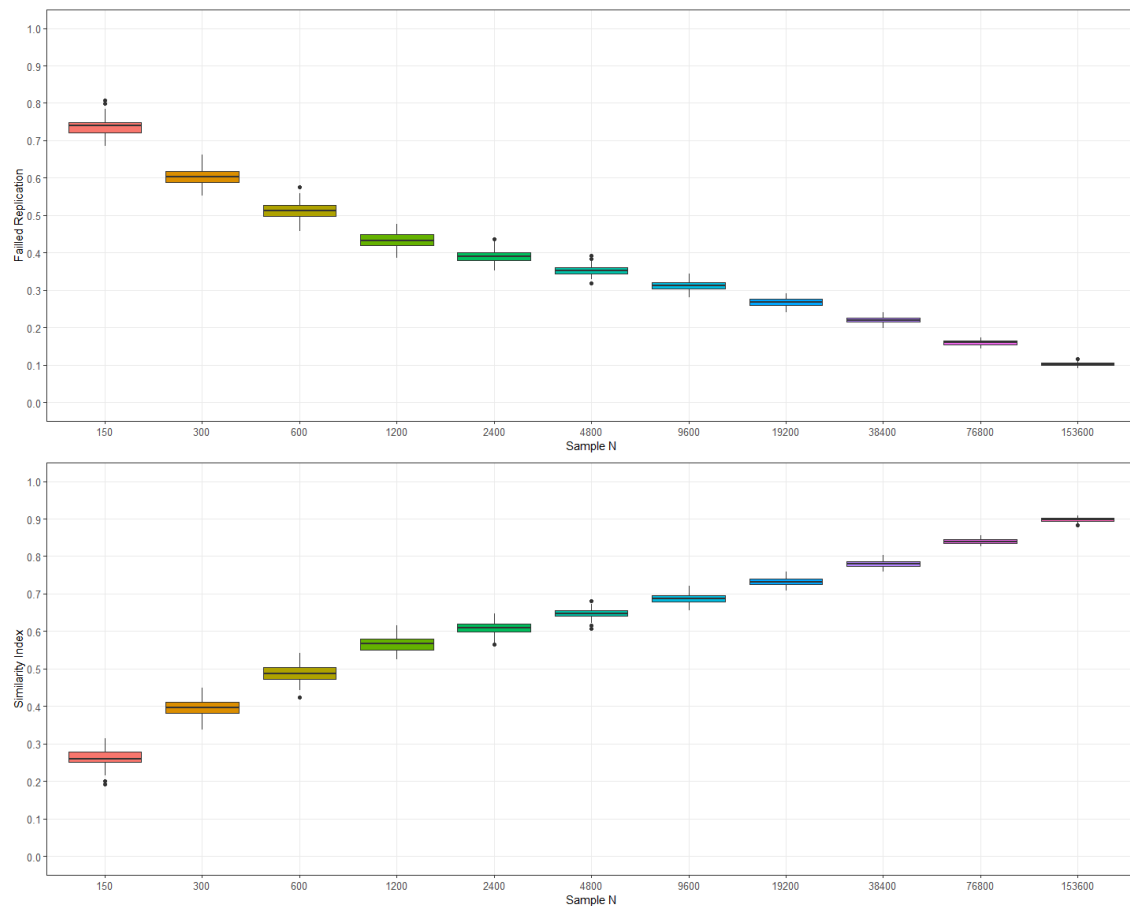


Figure S25. Robustness of the personality network at different sample sizes (continue)

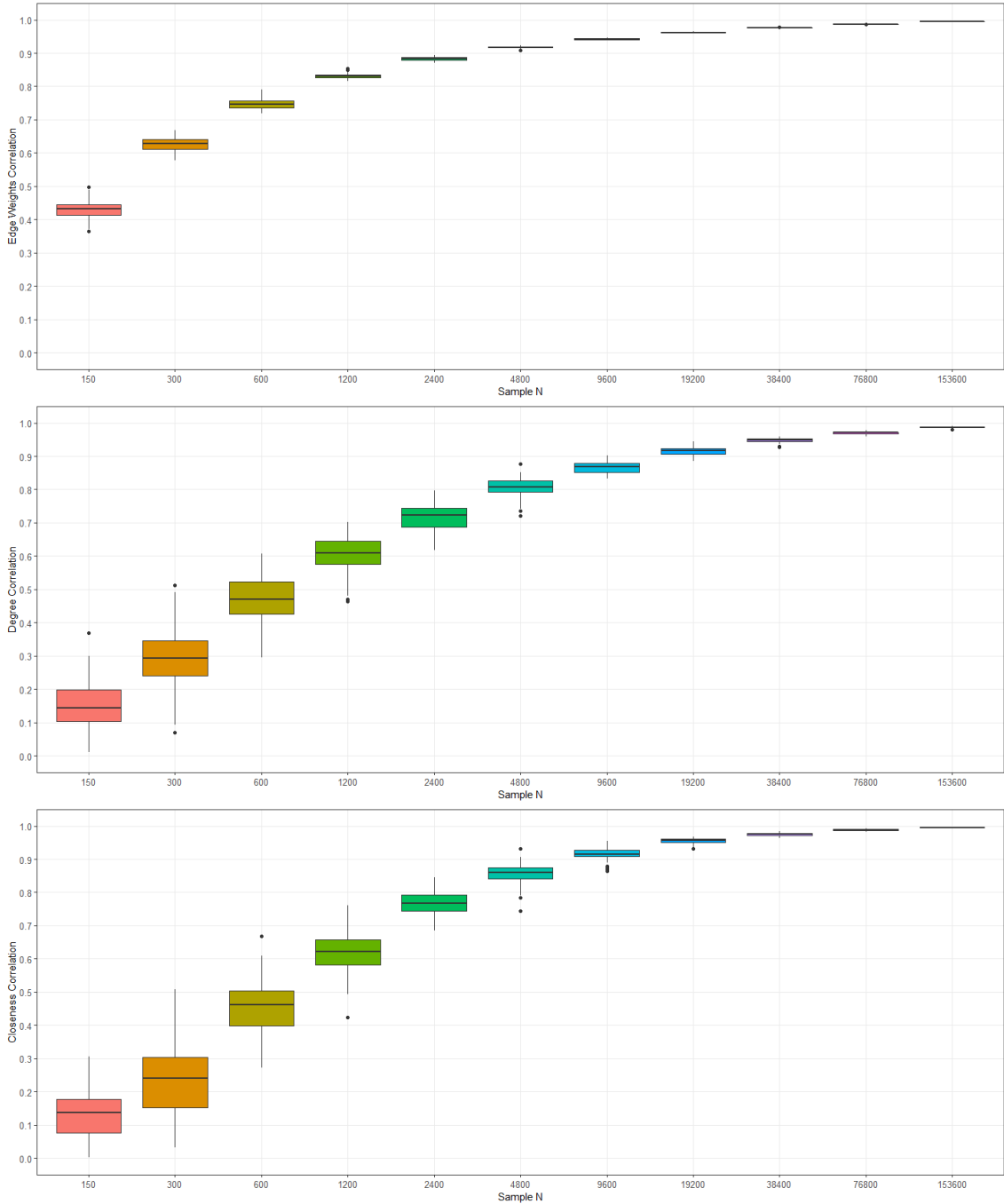


Figure S26. Robustness of the personality network at different sample sizes (continue)

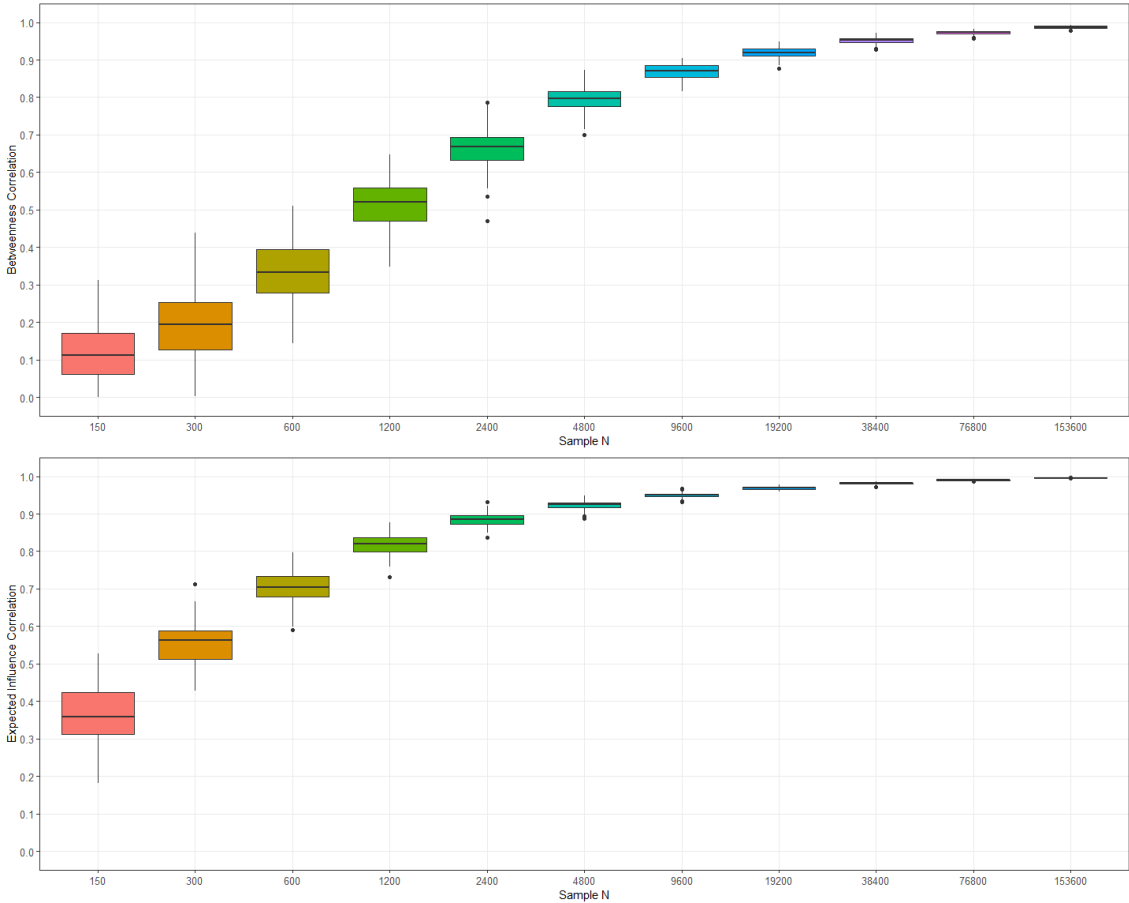


Figure S27. Robustness of the personality network at different sample sizes

Stability of the ModuLand algorithm in split-halves samples

Stability of the ModuLand algorithm was assessed by estimating a hierarchical structure for 200 randomly generated samples with half of the sample size. Results show that for the second hierarchical level a mean of 36 (SD = .761) modules was found and in the third hierarchical level a mean of 10 (SD = 3.30) modules.

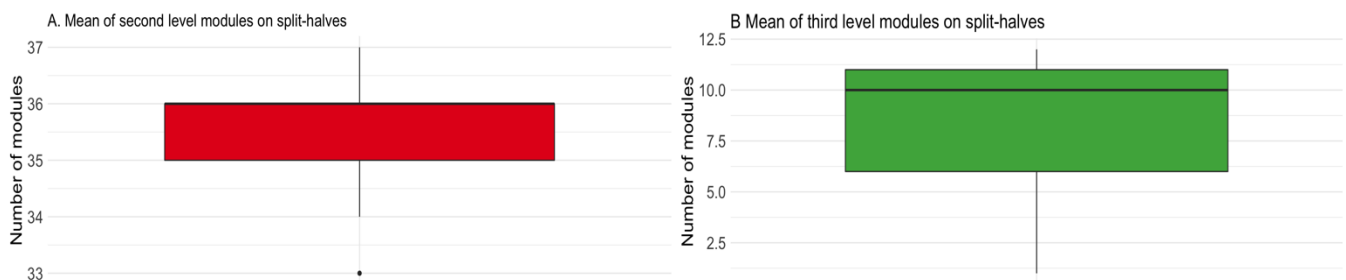


Figure S28. Stability of ModuLand algorithm in 200 split-halves samples with 50% of participants randomly selected. The panel a) shows the second-level modules estimated across samples (M=36, SD=.761) and panel b) represents the third-level modules (M=10, SD=3.30).

Supplementary discussion

In this study we explored the modular structure of personality network through ModuLand. In the main text we have explained the main reasoning behind the choice of this particular algorithm, as well as, its limitations and future needs of research. In this discussion we will deepen the results found with the ModuLand algorithm and detail how further research can be done with this promising algorithm.

Results revealed a three-level structure in line with the proposal of the FFM (McCrae & John, 1992). However, the FFM of the IPIP-NEO-120 proposes a second hierarchical level of 30 facets (Johnson, 2014) and our results suggest that this hierarchical level is comprised of 35 modules (see Table S1). Albeit this increase in partitions on the second-level, most of our modules are similar to the facets proposed for the IPIP-NEO-120, with the increase in partitions being driven mostly by splits within the proposed facets (e.g. depression facet splits into two different modules). In turn, our third hierarchical level was more distinct from the structure proposed by the FFM (Costa & McCrae, 1992) and inconsistent with the previous proposals (Digman, 1997; Shedler & Westen, 2004; Gerlach, Farb, Revelle, & Amaral, 2018). Although each module had more personality characteristics than a specific FFM factor, most of our 9 modules were constituted by personality characteristics from all the proposed factors in the FFM (see Table S1). In addition, in the third hierarchical level, the existence of a module without a clear constitution (i.e., aggregating all of the second-level modules without any being mostly assigned to it) might be similar to the results of previous studies on the bifactor models of personality (Chen, Hayes, Carver, Laurenceau, & Zhang, 2012). This implies the presence of a general factor that assesses the general effect shared by the personality characteristics, which, in spite of it, are grouped in specific facets due to their specific effects (Chen et al., 2012). Therefore, this third level module might encompass the general

effects shared by the second level modules, while the remaining third level modules might account for the specific effects of the second level modules.

On more specific terms, the analysis of centrality measures (Figure S2 – S13) suggests that each hierarchical level might have personality characteristics and modules with specialised roles in the structure of personality; these could reflect the developmental mechanisms which explain the different developmental trends found for each factor (Soto & Tackett, 2015). The personality characteristics with high overlap belong to different modules and thus they may facilitate the development of other specific personality modules or, conversely, could be harder to change since they belong to various modules. For example, as seen in Figure S4, item 46 “am afraid to draw attention to myself” might be a personality characteristic which facilitates communication between the second-level modules and/or that is harder to change. In turn, modular bridgeness has been regarded as another possible explanation for the connection between disorders in psychopathological networks due to their positioning between disorders, i.e., modules (Castro et al., 2018). Thus, in the personality networks, these modules might be responsible for keeping the personality network interconnected and providing paths for the development of personality characteristics and modules that would have been otherwise unreachable. One of these characteristics is item 24, “believe that I’m better than others” (Figure S4), which might be a personality characteristic that connects several different second-level modules in the network.

Looking how these types of personality characteristics (i.e., overlapping and bridging) tend to group we see that in the third-level modules, for example, modules 4, 6 and 8 tend to group characteristics with higher bridgeness and overlap (Figure S10; for the second-level modules see figure S6). In fact, research has shown that Agreeableness and Openness to Experience are factors prone to change during development (Soto &

Tackett, 2015), which, in our results, are the most represented factors in modules 4, 6, and 8 (Table S1); these are the modules where the personality characteristics with more bridgeness and overlap tend to group. So, high bridgeness or overlap might be a mechanism for the development of certain characteristics due to their position in the network.

There were also personality characteristics and modules with high values in both modular overlap and bridgeness (see Figure S5 for second-level modules and Figure S6 for third-level modules), which indicates that these personality characteristics and modules simultaneously have an inter-modular and a trans-modular role (i.e., are positioned between modules and at the same time belong to various modules). Consequently, these characteristics and modules might have a more vital role in the development of personality.

Regarding community centrality and effective degree, these two measures also seem to characterise different roles of the modules in the personality structure (see Figure S2, S7 and S11 for each level respectively). Modules with elevated community centrality tend to branch out more, connecting with other modules. This increased access might assign these modules a critical role in the development of personality. In turn, modules with a high effective degree have an elevated number of connections with their immediate neighbouring modules. These connections could have a stabilising role in the structure of personality by keeping these modules strongly connected. For example, in our third level the differentiation between the modules with a high effective degree and high community centrality is clear (Figure S12). Modules 1 and 2 have high community centrality and modules 3 and 5 high effective degree. Module 1 is comprised mostly of Neuroticism items and module 2 of Extraversion items; these two factors have been shown to have differential developmental trends (Soto & Tackett, 2015) and to change during

intervention (Roberts et al., 2017). This could be explained, not by their position in the network, but through the capacity of these modules to reach a vast number of personality characteristics in the network. This capacity provides new paths for developing new personality characteristics and potentially changing ones' personality structure.

Concerning modules 3 and 5, modules with higher effective degree, these modules are mostly composed by neuroticism and agreeableness items, since items from these two factors have also been identified in other modules characterised by other measures, potentially meaning that not all of the items from these two factors have the same role in the personality network. For example, specific personality characteristics from agreeableness might have a role in guiding the development of the personality structure, while other personality characteristics might have a role in maintaining the stability of the personality structure over time. Results for the remaining levels can be seen in Figure S8 and Figure S3.

In conclusion, our results present a more refined view of personality, where the personality characteristics take the stage. Moreover, our study proposes that there might be characteristics with different roles in the network and these different roles could offer new and important insights on how personality develops and changes.

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<https://doi.org/10.1111/0081-1750.00098>

Chapter II ^{1,2}

**The Differential Role of Central and Bridge Symptoms in the Disaggregation of
Psychopathological Networks**

Note:

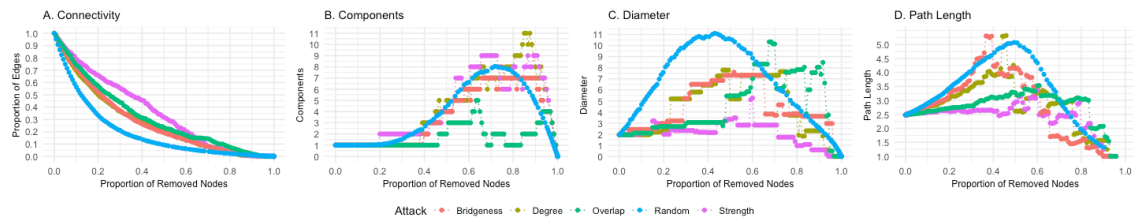
¹ This chapter was published as an independent paper:

Castro, D., Ferreira, F., de Castro, I., Rodrigues, A. R., Correia, M., Ribeiro, J., & Ferreira, T. B. (2019). The Differential Role of Central and Bridge Symptoms in Deactivating Psychopathological Networks. *Frontiers in Psychology*, 10. <https://doi.org/10.3389/fpsyg.2019.02448>.

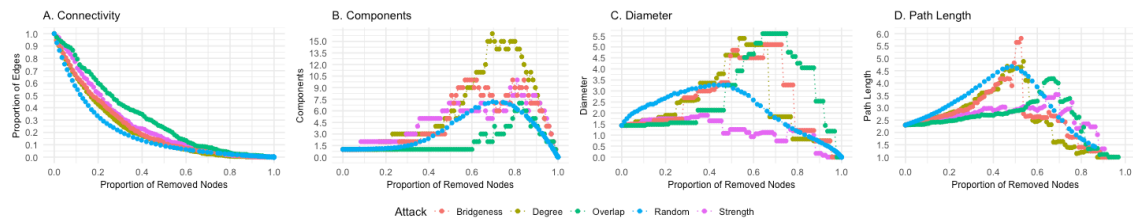
² The first and the second author contributed equally to the development of the paper.

Plots displaying attack results for each individual network

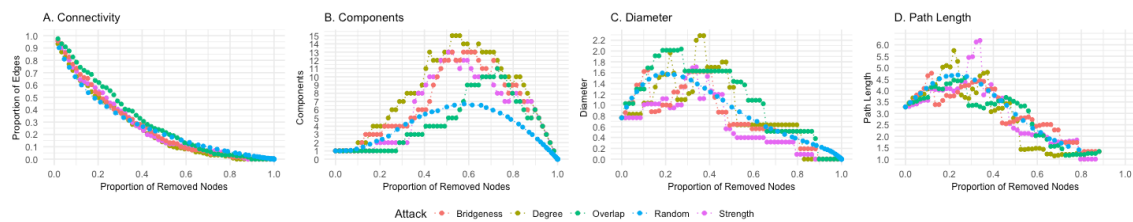
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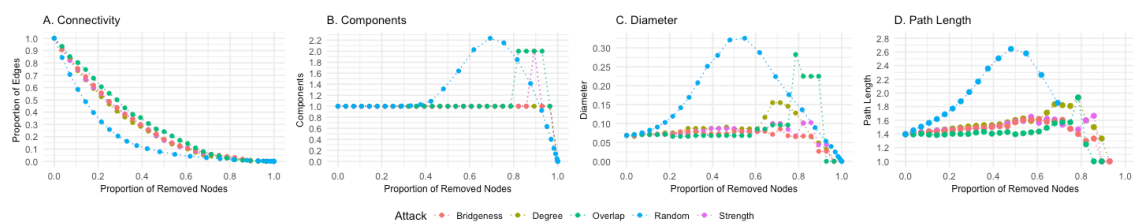
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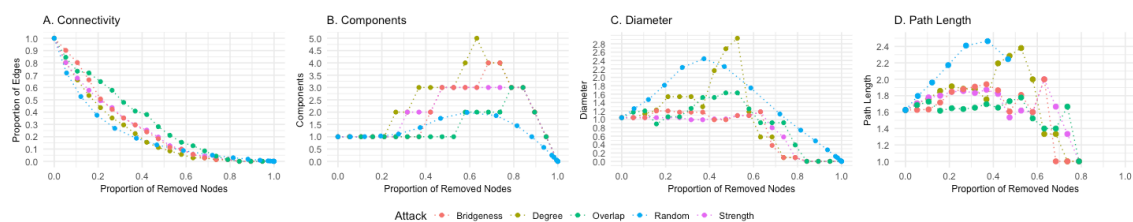
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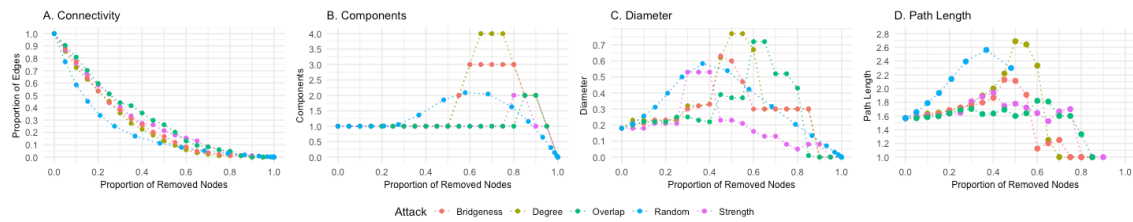
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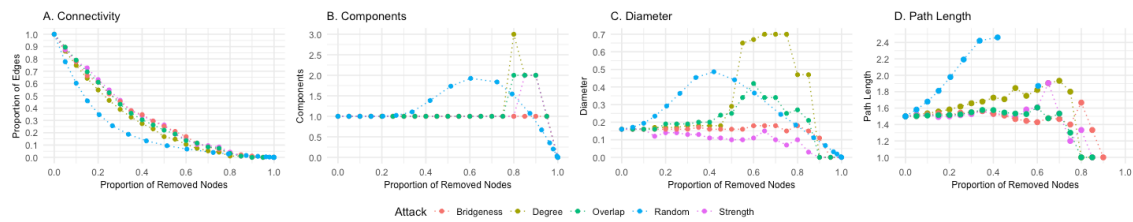
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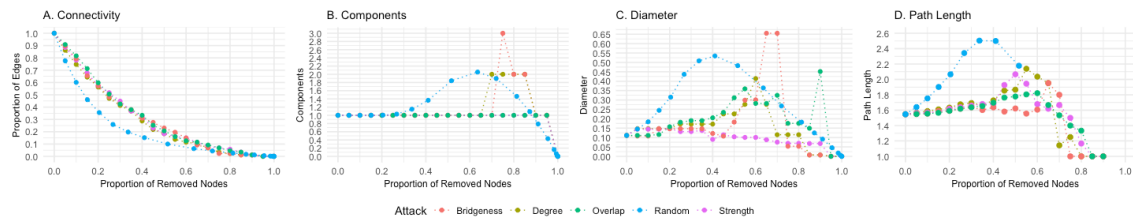
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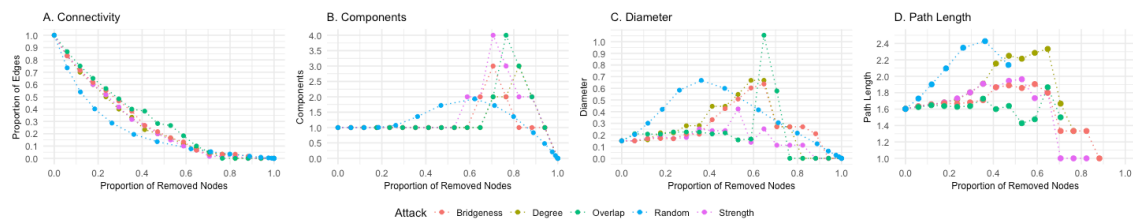
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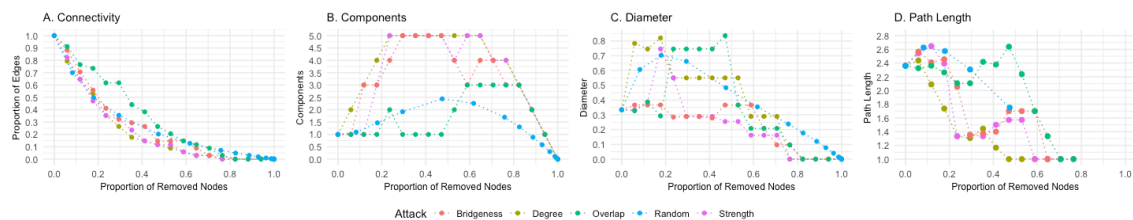
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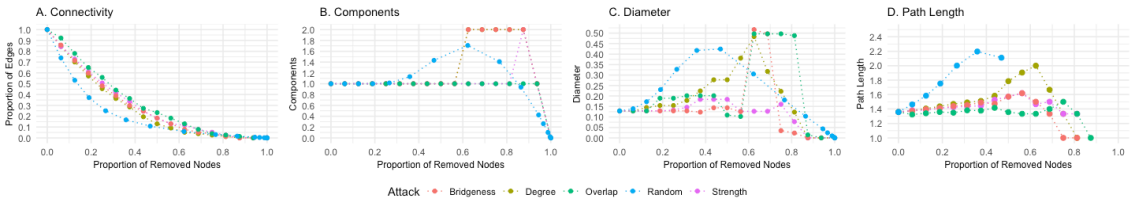
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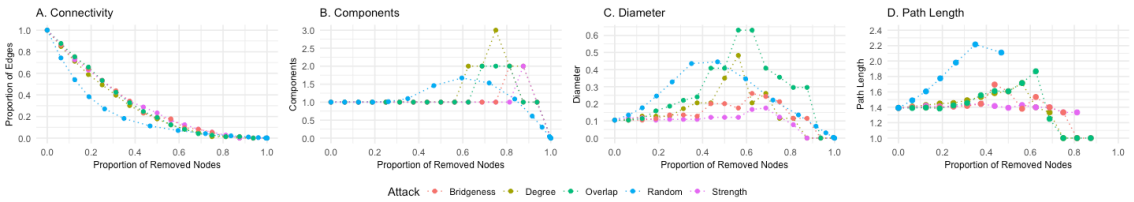
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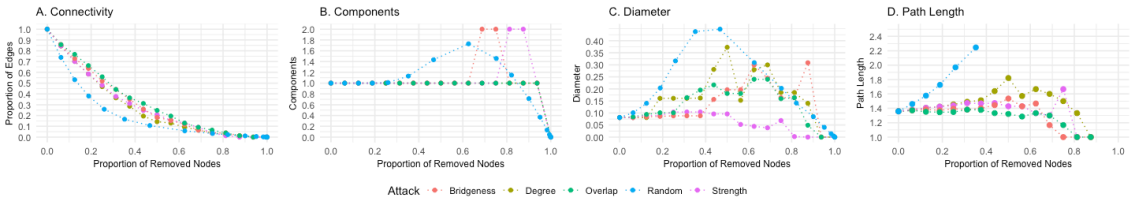
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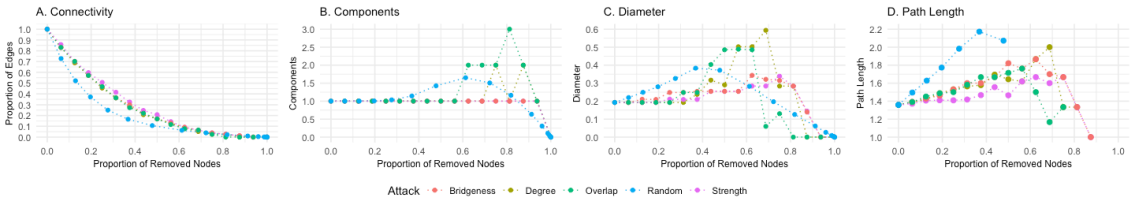
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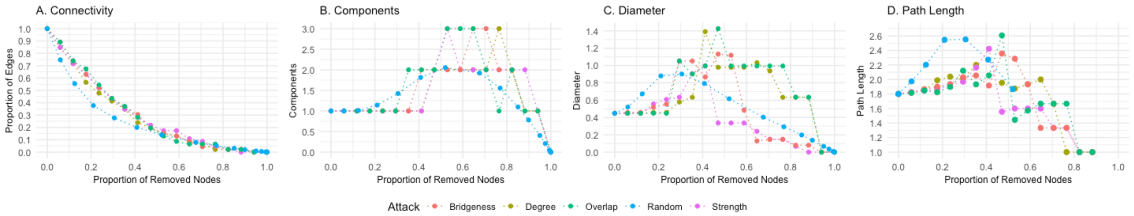
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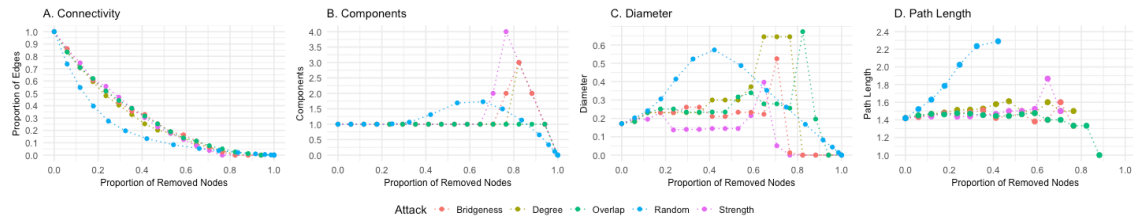
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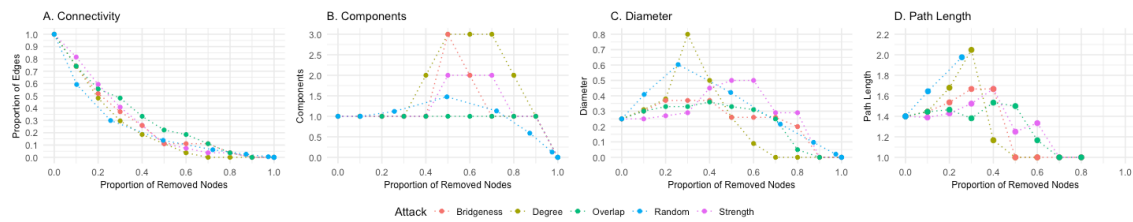
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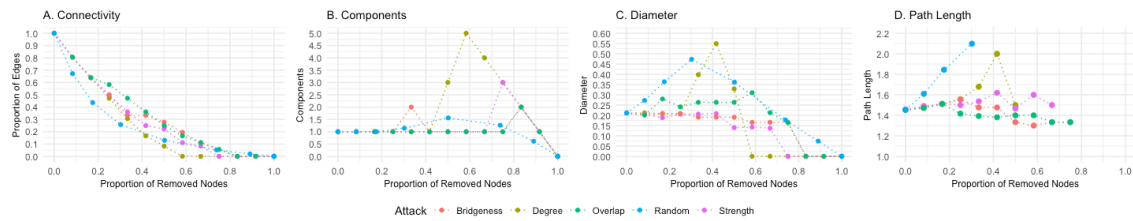
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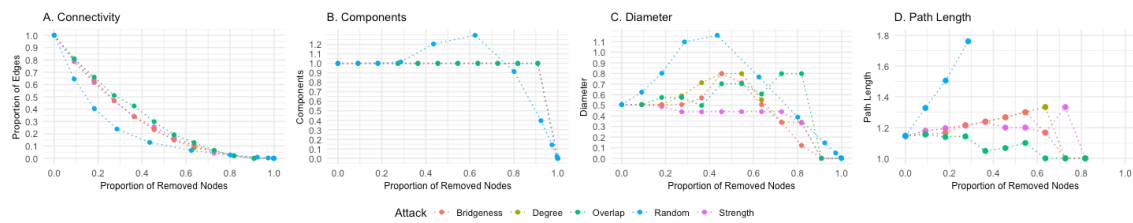
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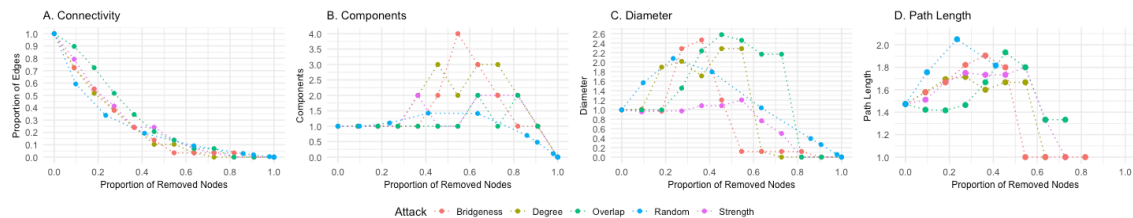
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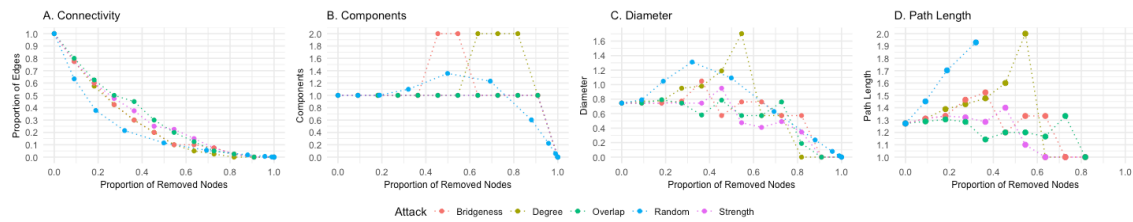
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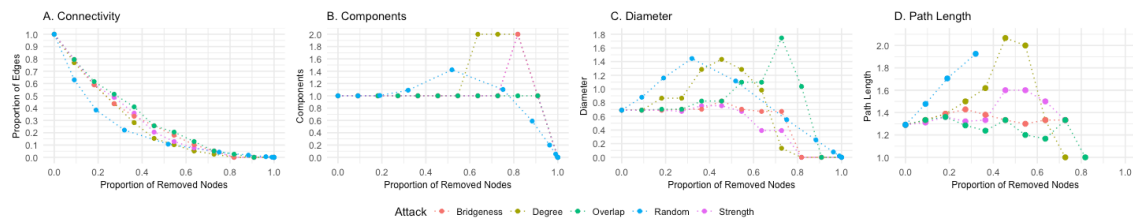
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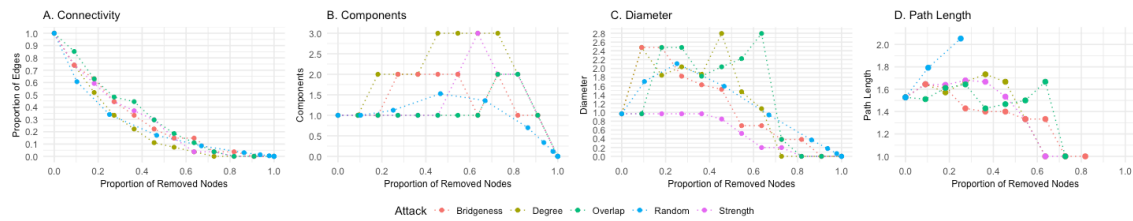
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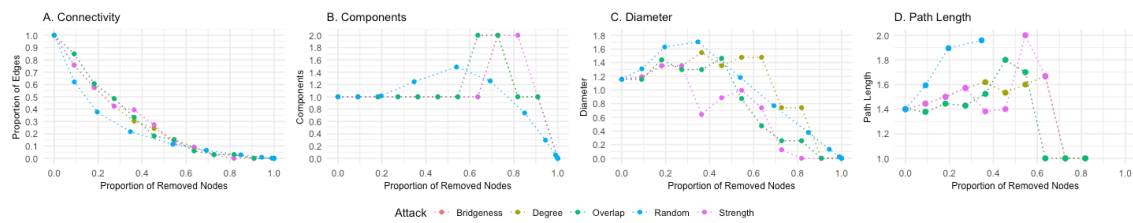
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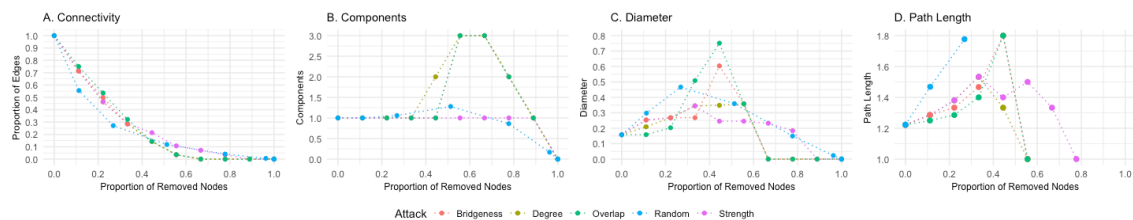
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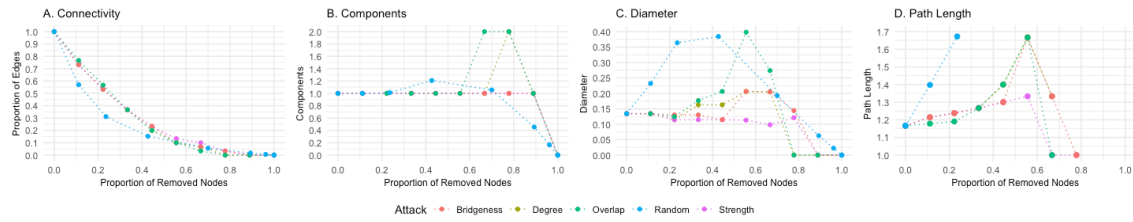
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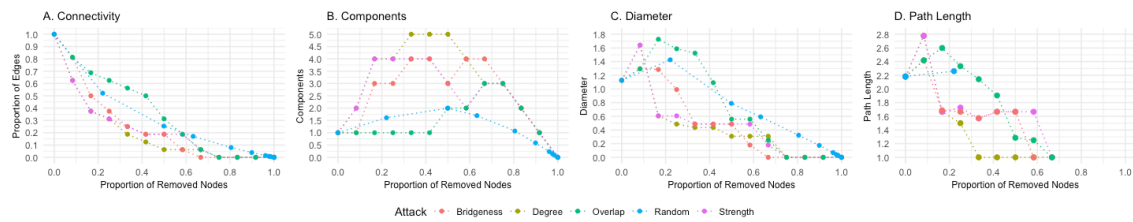
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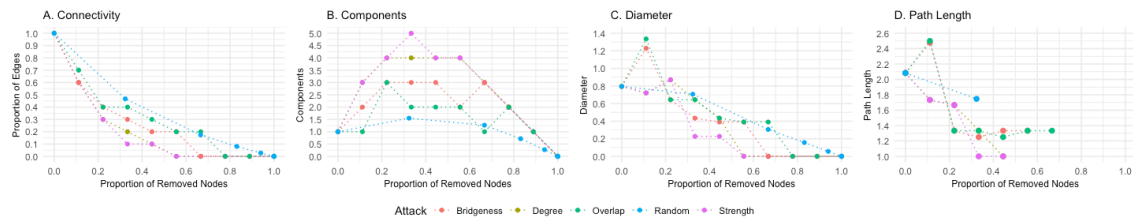
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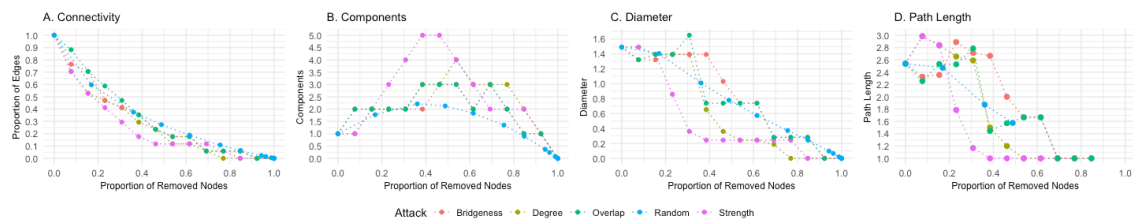
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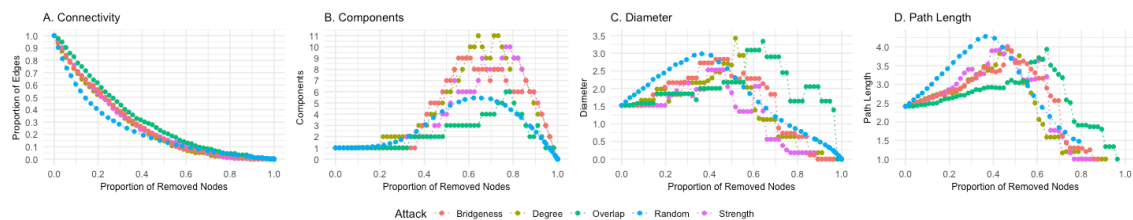
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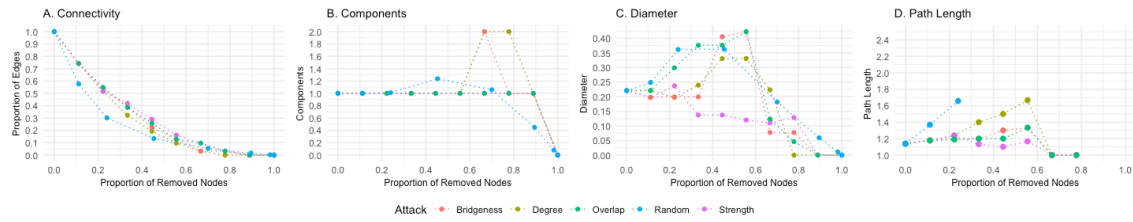
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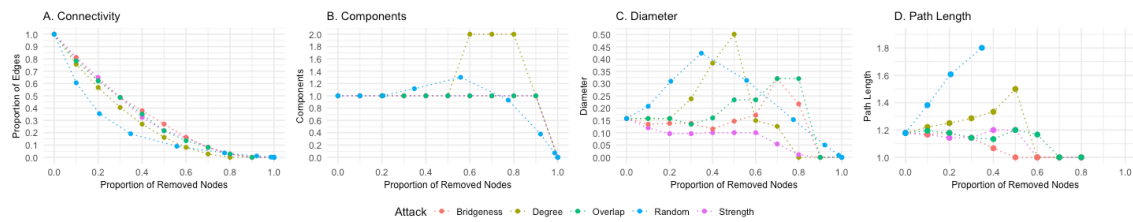
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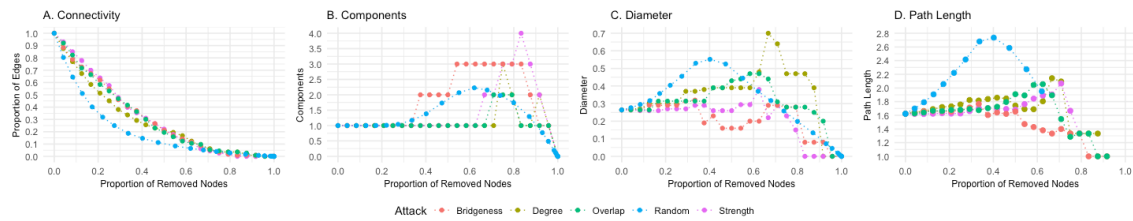
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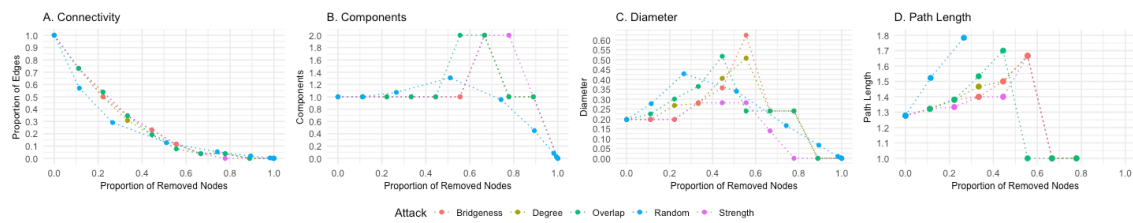
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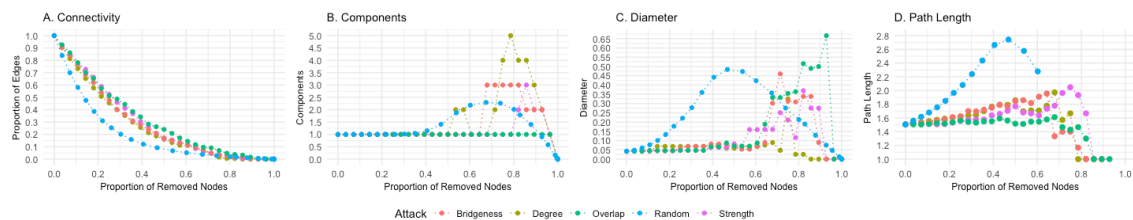
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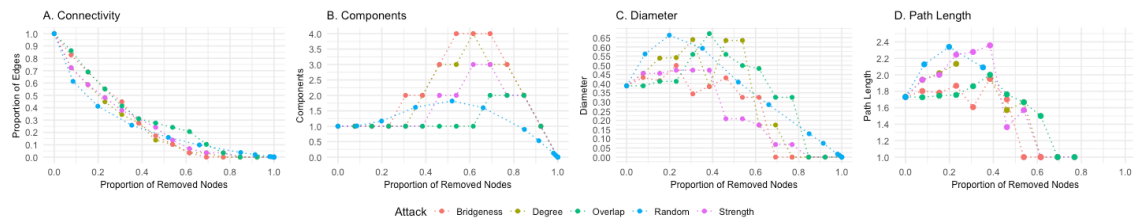
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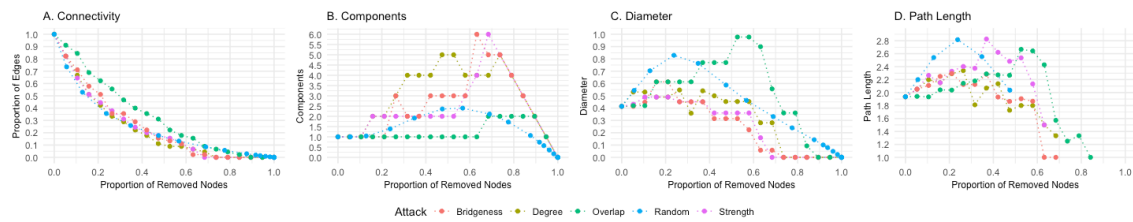
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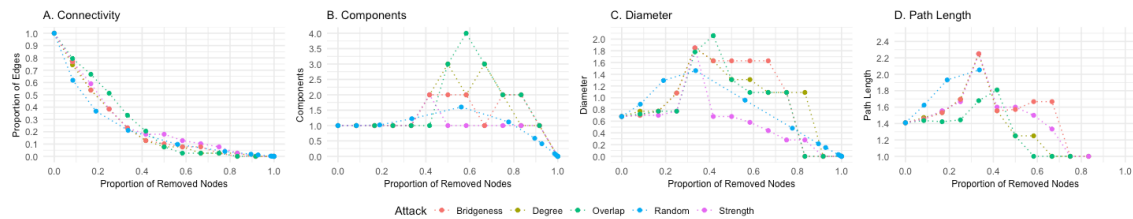
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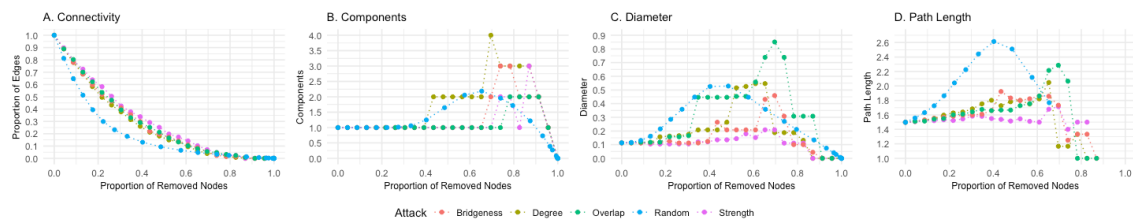
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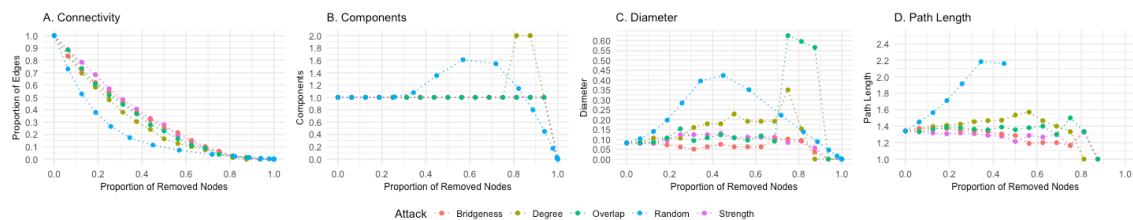
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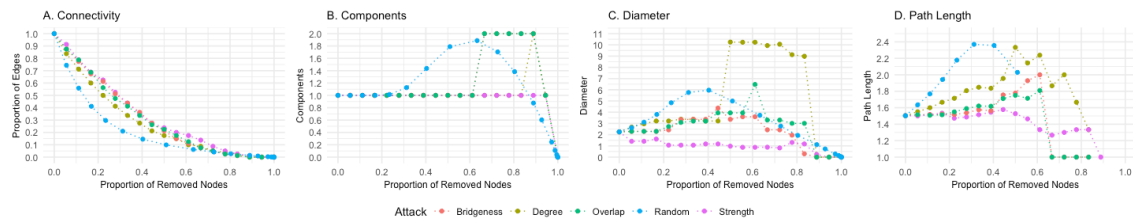
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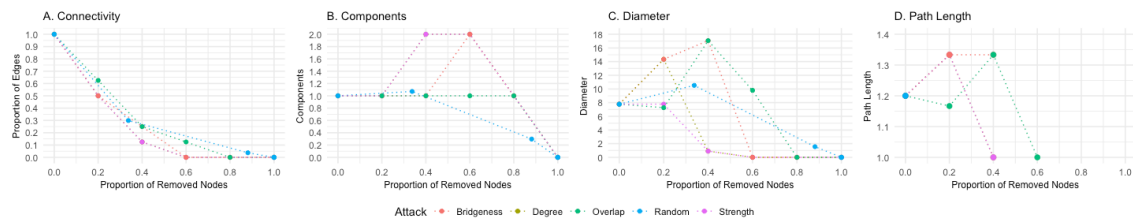
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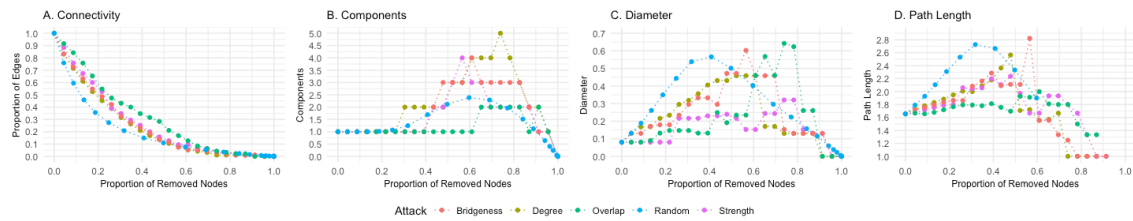
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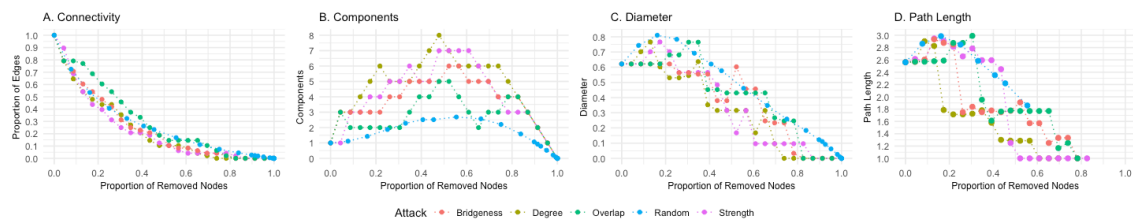
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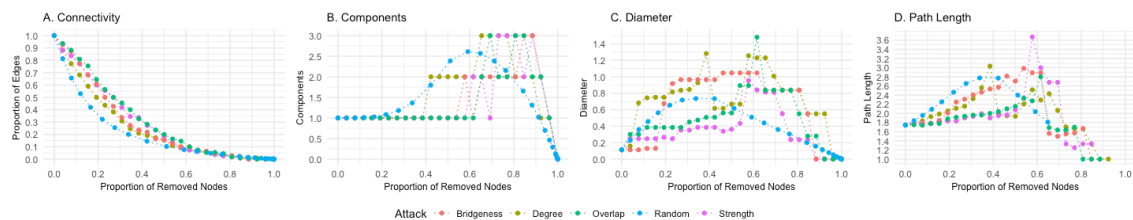
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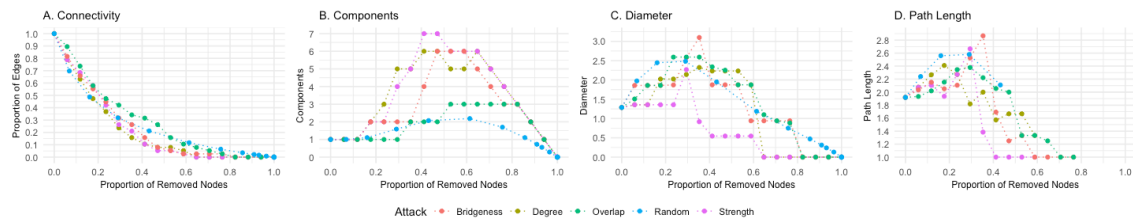
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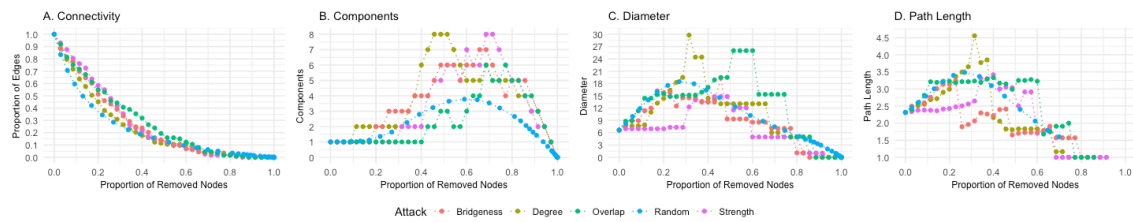
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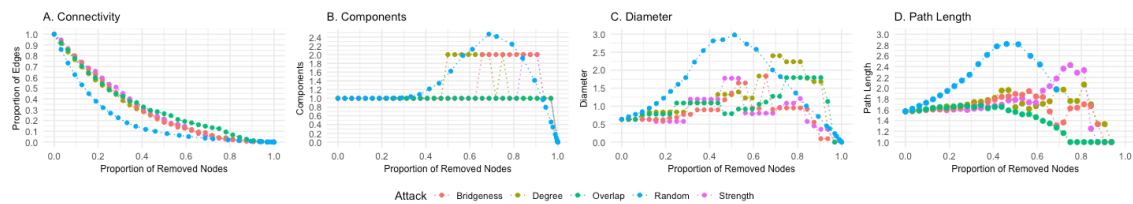
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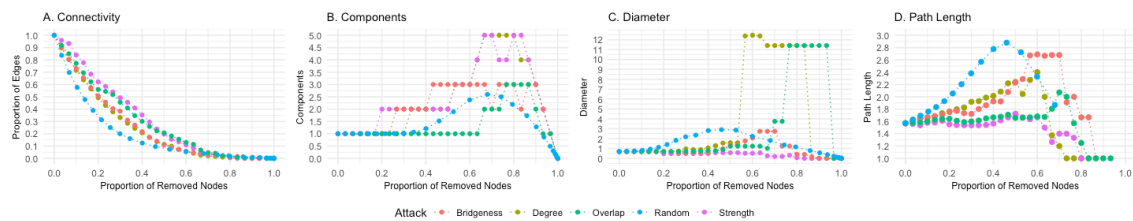
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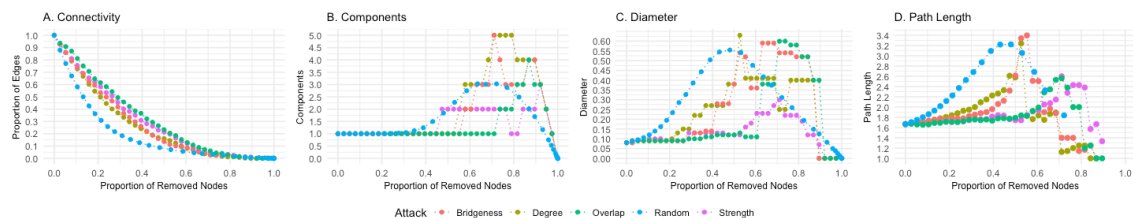
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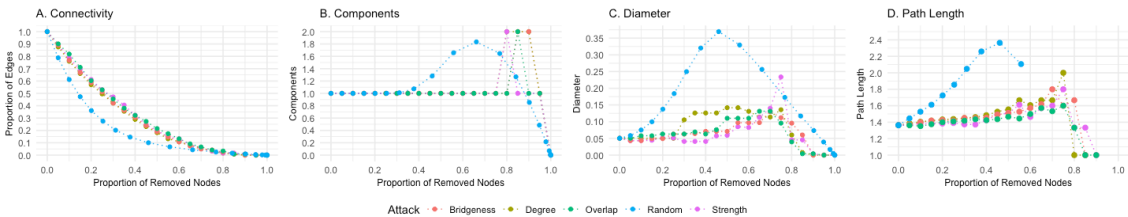
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**Comparison of the number of modules identified through the Clique Percolation
and ModuLand algorithms**

Network Number	Study	Number of Modules				
		ModuLand	Clique Percolation			
			K = 3	K = 4	K = 5	K = 6
1	Boschloo et al. (2015)	29	14	14	15	12
2	Boschloo et al. (2016)	24	16	19	16	2
3	Goekoop et al. (2014)	13	1	-	-	-
4	Fried et al. (2016)	8	5	6	2	3
5	Kendler et al. (2017)	5	3	2	2	-
6	Santos et al. (2017).a	7	6	6	1	-
7	Santos et al. (2017).b	5	6	5	2	1
8	Armour et al. (2017)	8	6	3	3	1
9	Birkeland et al. (2017).a	7	7	5	1	-
10	Birkeland et al. (2017).b	6	4	3	-	-
11	Fried et al. (2018).a	6	4	6	6	2
12	Fried et al. (2018).b	4	3	5	3	1
13	Fried et al. (2018).c	7	3	6	4	3
14	Fried et al. (2018).d	6	5	6	4	4

15	McNally et al. (2015)	6	8	2	-	-
16	McNally et al. (2017)	7	7	5	5	-
17	Sullivan et al. (2018)	4	3	2	-	-
18	Anderson et al. (2015)	3	2	2	-	-
19	Rhemtulla et al. (2016).a	3	3	2	2	2
20	Rhemtulla et al. (2016).b	4	3	2	-	-
21	Rhemtulla et al. (2016).c	3	3	3	3	-
22	Rhemtulla et al. (2016).d	3	2	1	2	-
23	Rhemtulla et al. (2016).e	2	3	1	-	-
24	Rhemtulla et al. (2016).f	1	5	4	-	-
25	Richetin et al. (2017).a	4	1	2	1	-
26	Richetin et al. (2017).b	3	1	2	1	-
27	Koenders et al. (2015).a	2	2	-	-	-
28	Koenders et al. (2015).b	3	1	-	-	-
29	Koenders et al. (2015).c	3	2	-	-	-
30	Wigman at al. (2016)	15	18	2	-	-
31	Marcus et al. (2018).a	3	3	2	2	1
32	Marcus et al. (2018).b	3	3	2	1	2
33	Watters et al. (2016)	5	6	4	3	-

34	DuBois et al. (2017)	3	2	2	1	-
35	Goldschmidt et al. (2018)	7	7	4	3	4
36	Robinaugh et al. (2016)	4	5	-	-	-
37	Robinaugh et al. (2014)	4	5	2	-	-
38	Fried et al. (2015)	5	4	2	2	1
39	Bellet et al. (2018)	9	7	4	3	3
40	Beard et al. (2016)	4	3	3	3	2
41	Borsboom et al. (2013).a	2	3	3	3	2
42	Borsboom et al. (2013).b	2	2	1	-	-
43	Van Rooijen et al. (2018)	8	6	7	4	-
44	Jones et al. (2018)	6	8	1	2	-
45	McNally et al. (2017)	7	8	7	2	1
46	Ruzzano et al. (2015)	4	6	2	-	-
47	Afzali et al. (2017)	10	9	5	-	-
48	Castro et al. (2018)	9	7	6	5	6
49	Bekhuis et al. (2016)	8	7	6	5	5
50	Smith et al. (2018)	12	10	10	14	4
51	Marchetti (2018)	5	4	3	3	3

Description of the original networks and studies

Table S1*Description of the original networks and studies*

Network Number	Study	Psychopathology	Different Disorders or Problems	Population	Sample	N	Datatype	Supplementary Material	Original Analysis	Analysis	Haslbeck Analysis	Haslbeck N
1	Boschloo et al. (2015)	Mood, Anxiety, and Substance Abuse or Dependence Disorders	Yes	Adults	Community	34653	Binary	Adjacency Matrix	Ising	--	--	--
2	Boschloo et al. (2016)	Internalizing and Externalizing Problems	Yes	Preadolescents	Community	2175	Binary	Adjacency Matrix	Ising	--	--	--
3	Goekoop et al. (2014)	Mood, Anxiety, Schizophrenia, Adjustment, and Personality Disorders	Yes	Adults	Clinical	192	Continuous	Raw Data	Correlations	--	GGM	--
4	Fried et al. (2016)	Depression	No	Adults	Clinical	3463	Continuous	--	GGM		GGM	3463
5	Kendler et al. (2017)	Depression	No	Adults	Clinical	5952	Binary	Adjacency Matrix	Ising	--	--	--
6	Santos et al. (2017).a	Depression	No	Adults	Clinical	264	Continuous	Adjacency Matrix	GGM	--	--	--
7	Santos et al. (2017).b	Depression	No	Adults	Community	240	Continuous	Adjacency Matrix	Ising	--	--	--
8	Armour et al. (2017)	Posttraumatic Stress Disorder	No	Adults	Clinical	221	Continuous	Raw Data	GGM	GGM	--	--
9	Birkeland et al. (2017).a	Posttraumatic Stress Disorder	No	Adults	Community	117	Continuous	Network Data	GGM	--	--	--
10	Birkeland et al. (2017).b	Posttraumatic Stress Disorder	No	Adults	Community	73	Continuous	Network Data	GGM	--	--	--
11	Fried et al. (2018).a	Posttraumatic Stress Disorder	No	Adults	Clinical	526	Continuous	Network Data	GGM	--	--	--

Table S1
Description of the original networks and studies (continued)

Network Number	Study	Psychopathology	Different Disorders or Problems	Population	Sample	N	Datatype	Supplementary Material	Original Analysis	Analysis	Haslbeck Analysis	Haslbeck N
12	Fried et al. (2018).b	Posttraumatic Stress Disorder	No	Adults	Clinical	365	Continuous	Network Data	GGM	--	--	--
13	Fried et al. (2018).c	Posttraumatic Stress Disorder	No	Adults	Clinical	926	Continuous	Network Data	GGM	--	--	--
14	Fried et al. (2018).d	Posttraumatic Stress Disorder	No	Adults	Clinical	965	Continuous	Network Data	GGM	--	--	--
15	McNally et al. (2015)	Posttraumatic Stress Disorder	No	Adults	Clinical	362	Continuous	--	Partial Correlations	--	GGM	362
16	McNally et al. (2017)	Posttraumatic Stress Disorder	No	Adults	Clinical	179	Continuous	Raw Data	GGM	GGM	--	--
17	Sullivan et al. (2018)	Posttraumatic Stress Disorder	No	Adults	Community	4639	Continuous	Adjacency Matrix	GGM	--	--	--
18	Anderson et al. (2015)	Autism	No	Children	Clinical	477	Continuous	--	Partial Correlations	--	GGM	477
19	Rhemtulla et al. (2016).a	Substance Abuse	No	Adults	Clinical	2230	Binary	--	Ising	--	Ising	2405
20	Rhemtulla et al. (2016).b	Substance Abuse	No	Adults	Clinical	358	Binary	--	Ising	--	Ising	
21	Rhemtulla et al. (2016).c	Substance Abuse	No	Adults	Clinical	677	Binary	--	Ising	--	Ising	
22	Rhemtulla et al. (2016).d	Substance Abuse	No	Adults	Clinical	639	Binary	--	Ising	--	Ising	

Table S1
Description of the original networks and studies (continued)

Network Number	Study	Psychopathology	Different Disorders or Problems	Population	Sample	N	Datatype	Supplementary Material	Original Analysis	Analysis	Haslbeck Analysis	Haslbeck N
23	Rhemtulla et al. (2016).e	Substance Abuse	No	Adults	Clinical	199	Binary	--	Ising	--	Ising	
24	Rhemtulla et al. (2016).f	Substance Abuse	No	Adults	Clinical	350	Binary	--	Ising	--	Ising	
25	Richetin et al. (2017).a	Borderline Personality Disorder	No	Adults	Clinical	96	Continuous	Raw Data	FGM	GGM	--	--
26	Richetin et al. (2017).b	Borderline Personality Disorder	No	Adults	Community	1317	Continuous	Raw Data	FGM	GGM	--	--
27	Koenders et al. (2015).a	Bipolar Disorder	No	Adults	Clinical	47	Continuous	Raw Data	Correlations	--	GGM	126
28	Koenders et al. (2015).b	Bipolar Disorder	No	Adults	Clinical	42	Continuous	Raw Data	Correlations	--	GGM	
29	Koenders et al. (2015).c	Bipolar Disorder	No	Adults	Clinical	36	Continuous	Raw Data	Correlations	--	GGM	
30	Wigman et al. (2016)	Psychosis	Yes	Children	Clinical	283	Binary	--	Ising	--	Ising	283
31	Marcus et al. (2018).a	Dark Personality Traits	Yes	Adults	Community	2831	Continuous	Correlation Matrix	Adaptive Lasso	GGM	--	--
32	Marcus et al. (2018).b	Dark Personality Traits	Yes	Adults	Community	844	Continuous	Correlation Matrix	Adaptive Lasso	GGM	--	--
33	Watters et al. (2016)	Alexithymia	No	Adults	Community	839	Continuous	Adjacency Matrix	Adaptive Lasso	--	--	--

Table S1
Description of the original networks and studies (continued)

Network Number	Study	Psychopathology	Different Disorders or Problems	Population	Sample	N	Datatype	Supplementary Material	Original Analysis	Analysis	Haslbeck Analysis	Haslbeck N
34	DuBois et al. (2017)	Eating Disorders	Yes	Adolescents, Adults	Clinical	194	Continuous	Adjacency Matrix	GGM	--	--	--
35	Goldschmidt et al. (2018)	Eating Disorders	Yes	Children, Adolescents	Clinical	636	Continuous	Correlation Matrix	GGM	GGM	--	--
36	Robinaugh et al. (2016)	Grief	No	Adults	Community	195	Continuous	--	GGM	--	GGM	195
37	Robinaugh et al. (2014)	Grief, Depression	Yes	Adults	Community	265	Continuous	--	Correlation	--	GGM	1532
38	Fried et al. (2015)	Grief, Depression	Yes	Adults	Community	515	Binary	--	Ising	--	Ising	515
39	Bellet et al. (2018)	Grief, Posttraumatic Growth	Yes	Adults	Community	485	Continuous	Correlation Matrix	GGM	GGM	--	--
40	Beard et al. (2016)	Depression, Anxiety	Yes	Adults	Clinical	742	Continuous	Covariance Matrix	GGM	GGM	--	--
41	Borsboom et al. (2013).a	Depression, Anxiety	Yes	Adults	Community	9282	Binary	Raw Data	Correlations	Ising	--	--
42	Borsboom et al. (2013).b	Anxiety	No	Adults	Community		Binary	Raw Data	Partial Correlations	Ising	--	--
43	Van Rooijen et al. (2018)	Depression, Psychosis	Yes	Adults	Clinical	470	Continuous	Correlation Matrix	GGM	GGM	--	--
44	Jones et al. (2018)	Depression, Obsessive Compulsive Disorder	Yes	Adolescents	Clinical	87	Continuous	Raw Data	GGM	GGM	--	--

Table S1

Description of the original networks and studies (continued)

Network Number	Study	Psychopathology	Different Disorders or Problems	Population	Sample	N	Datatype	Supplementary Material	Original Analysis	Analysis	Haslbeck Analysis	Haslbeck N
45	McNally et al. (2017)	Depression, Obsessive Compulsive Disorder	Yes	Adults	Clinical	408	Continuous	Raw Data	GGM	GGM	--	--
46	Ruzzano et al. (2015)	Autism, Obsessive Compulsive Disorder	Yes	Children	Clinical	213	Binary	--	PC	--	Ising	213
47	Afzali et al. (2017)	PTSD, Alcohol Use	Yes	Adults	Community	449	Binary	Adjacency Matrix	Ising	--	--	--
48	Castro et al. (2018)	Bipolar, Borderline Personality Disorder	Yes	Adults	Community	7556	Binary	Raw Data	Ising	GGM	--	--
49	Bekhuis et al. (2016)	Depression, Anxiety, Somatic Symptoms	Yes	Adults	Clinical	2704	Binary	Adjacency Matrix	Ising	--	--	--
50	Smith et al. (2018)	Eating Disorder, Depression, Anxiety	Yes	Adults	Clinical	446	Continuous	Adjacency Matrix	GGM	--	--	--
51	Marchetti (2018)	Hopelessness	No	Adults	Community	1985	Continuous	Correlation Matrix	GGM	GGM	--	--

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Chapter III ¹

The Modular Structure of Posttraumatic Stress Disorder in Adolescents

Note:

¹ This paper was submitted for review:

Ferreira, F., Castro, D. & Ferreira, T. (2020). The modular structure of Posttraumatic Stress Disorder in adolescents [Manuscript submitted for publication].

Comparison between the DSM-IV and DSM 5 models of Posttraumatic Stress Disorder and the estimated ModuLand modules

Table S1

DSM-IV and DSM 5 models of Posttraumatic Stress Disorder and the estimated ModuLand modules

Symptoms	Two Factor Model (Taylor et al., 1998)	Two Factor Model (Taylor et al., 1998)	Three Factor Model (DSM-IV, APA, 1994)	Four Factor Model (DSM-5, APA, 2013)	Four Factor Model (King et al., 1998)	Four Factor Model (Simms et al., 2002)	Five Factor Model (Elhai et al., 2011)	Six Factor Model Anhedonia (Liu et al., 2014)	Six Factor Model Externalizing (Tsai et al., 2015)	Seven Factor Hybrid Model (Armour et al., 2016)	ModuLand Structure
Intrusions	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Module 1
Nightmares	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Module 1
Flashbacks	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Module 1
Emotional Reactivity	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Module 1
Physiological Reactivity	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Reexperiencing	Module 1
Avoidance of Thoughts	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Module 2
Avoidance of Situations	Reexperiencing/ Avoidance	Reexperiencing/ Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Avoidance	Module 2
Amnesia	Numbing/ Hyper Arousal	Reexperiencing/ Avoidance	Avoidance	Negative Alt. in Cognitions and Mood	Numbing	Dysphoria Arousal	Numbing	Negative Affect	Numbing	Negative Affect	Module 2
Loss of Interest	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Avoidance	Negative Alt. in Cognitions and Mood	Numbing	Dysphoria Arousal	Numbing	Anhedonia	Numbing	Anhedonia	Module 3
Detachment	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Avoidance	Negative Alt. in Cognitions and Mood	Numbing	Dysphoria Arousal	Numbing	Anhedonia	Numbing	Anhedonia	Module 3
Emotional Numbing	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Avoidance	Negative Alt. in Cognitions and Mood	Numbing	Dysphoria Arousal	Numbing	Anhedonia	Numbing	Anhedonia	Module 3
Foreshortened Future	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Avoidance	Negative Alt. in Cognitions and Mood	Numbing	Dysphoria Arousal	Numbing	Negative Affect	Numbing	Negative Affect	Module 3
Sleep Problems	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Hyper Arousal	Alt. in Arousal and Reactivity	Hyper Arousal	Dysphoria Arousal	Dysphoric Arousal	Dysphoric Arousal	Dysphoric Arousal	Dysphoric Arousal	Module 4
Irritability	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Hyper Arousal	Alt. in Arousal and Reactivity	Hyper Arousal	Dysphoria Arousal	Dysphoric Arousal	Dysphoric Arousal	Externalizing Behavior	Externalizing Behavior	Module 4
Concentration Problems	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Hyper Arousal	Alt. in Arousal and Reactivity	Hyper Arousal	Dysphoria Arousal	Dysphoric Arousal	Dysphoric Arousal	Dysphoric Arousal	Dysphoric Arousal	Module 3
Hypervigilance	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Hyper Arousal	Alt. in Arousal and Reactivity	Hyper Arousal	Hyper Arousal	Anxious Arousal	Anxious Arousal	Anxious Arousal	Anxious Arousal	Module 5
Startle Response	Numbing/ Hyper Arousal	Numbing/ Hyper Arousal	Hyper Arousal	Alt. in Arousal and Reactivity	Hyper Arousal	Hyper Arousal	Anxious Arousal	Anxious Arousal	Anxious Arousal	Anxious Arousal	Module 5
Blame*	-	-	-	Negative Alt. in Cognitions and Mood	-	-	-	Negative Affect	Numbing	Negative Affect	-
Negative Emotions*	-	-	-	Negative Alt. in Cognitions and Mood	-	-	-	Negative Affect	Numbing	Negative Affect	-
Destructive Behavior*	-	-	-	Alt. in Arousal and Reactivity	-	-	-	Dysphoric Arousal	Externalizing Behavior	Externalizing Behavior	-

Note. *DSM-5 new sy

Spearman correlation coefficients between centrality measures

Table S2

Descriptives of the Spearman correlation coefficients between centrality measures

		Bridgeness	Overlap	Strength
Bridgeness	Spearman's rho	—		
	p-value	—		
	Upper 95% CI	—		
	Lower 95% CI	—		
Overlap	Spearman's rho	0.833	—	
	p-value	< .001	—	
	Upper 95% CI	0.938	—	
	Lower 95% CI	0.587	—	
Strength	Spearman's rho	0.496	0.038	—
	p-value	0.043	0.885	—
	Upper 95% CI	0.788	0.509	—
	Lower 95% CI	0.020	-0.451	—

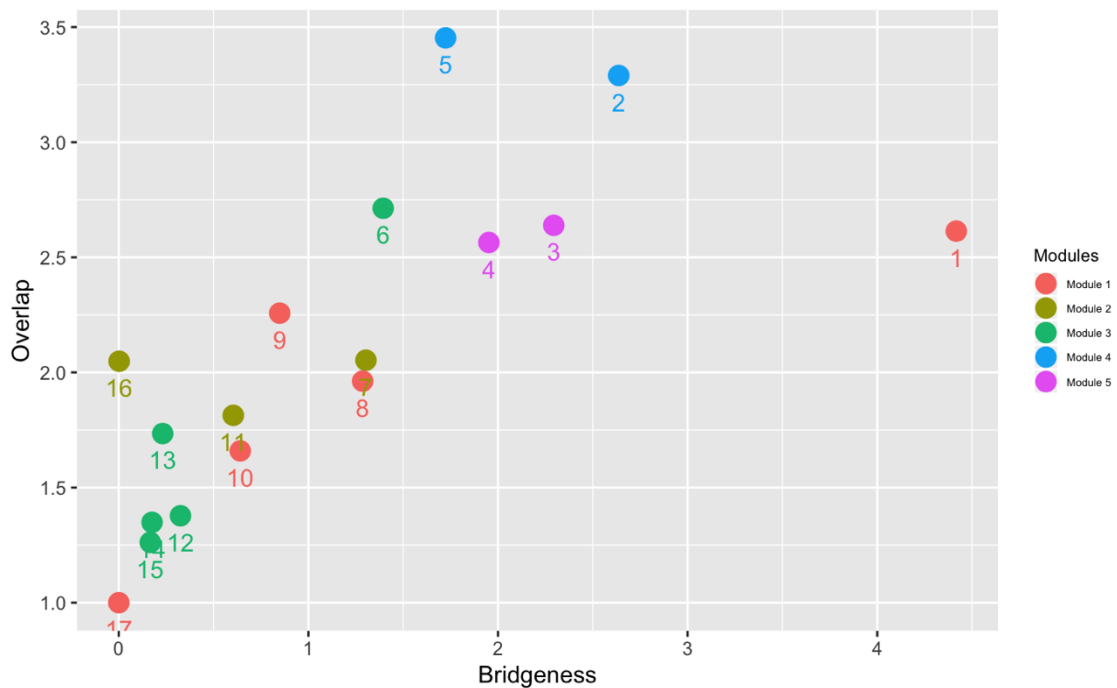
Joint representation of the centrality measures in the first hierarchical level

Figure S1. Joint representation of overlap and bridgeness in the first hierarchical level. Each dot represents a PTSD symptom, (1) intrusions, (2) nightmares, (3) flashbacks, (4) emotional reactivity, (5) physical reactivity, (6) avoidance of thoughts, (7) avoidance of situations, (8) amnesia, (9) loss of interest, (10) detachment, (11) emotional numbing, (12) foreshortened future, (13) sleep problems, (14) irritability, (15) Concentration problems, (16) hypervigilance, (17) startle response. The color of the dots represents the module to which the symptom is most assigned to.

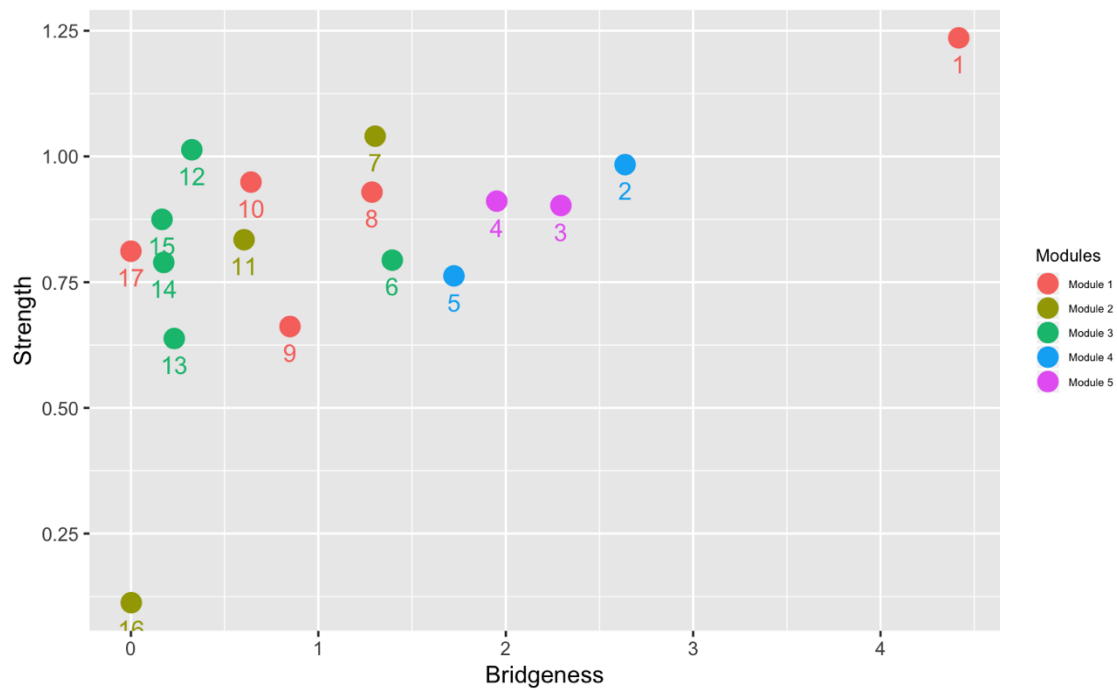


Figure S2. Joint representation of strength and bridgeness in the first hierarchical level. Each dot represents a PTSD symptom, (1) intrusions, (2) nightmares, (3) flashbacks, (4) emotional reactivity, (5) physical reactivity, (6) avoidance of thoughts, (7) avoidance of situations, (8) amnesia, (9) loss of interest, (10) detachment, (11) emotional numbing, (12) foreshortened future, (13) sleep problems, (14) irritability, (15) Concentration problems, (16) hypervigilance, (17) startle response. The color of the dots represents the module to which the symptom is most assigned to.

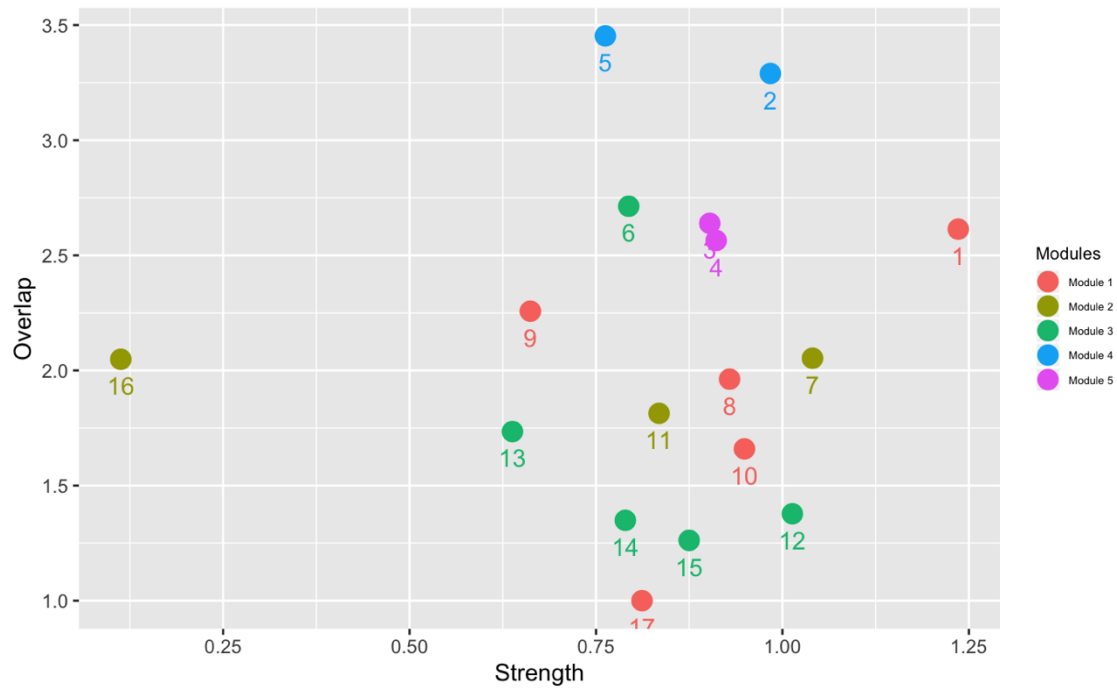


Figure S3. Joint representation of overlap and strength in the first hierarchical level. Each dot represents a PTSD symptom, (1) intrusions, (2) nightmares, (3) flashbacks, (4) emotional reactivity, (5) physical reactivity, (6) avoidance of thoughts, (7) avoidance of situations, (8) amnesia, (9) loss of interest, (10) detachment, (11) emotional numbing, (12) foreshortened future, (13) sleep problems, (14) irritability, (15) Concentration problems, (16) hypervigilance, (17) startle response. The color of the dots represents the module to which the symptom is most assigned to.

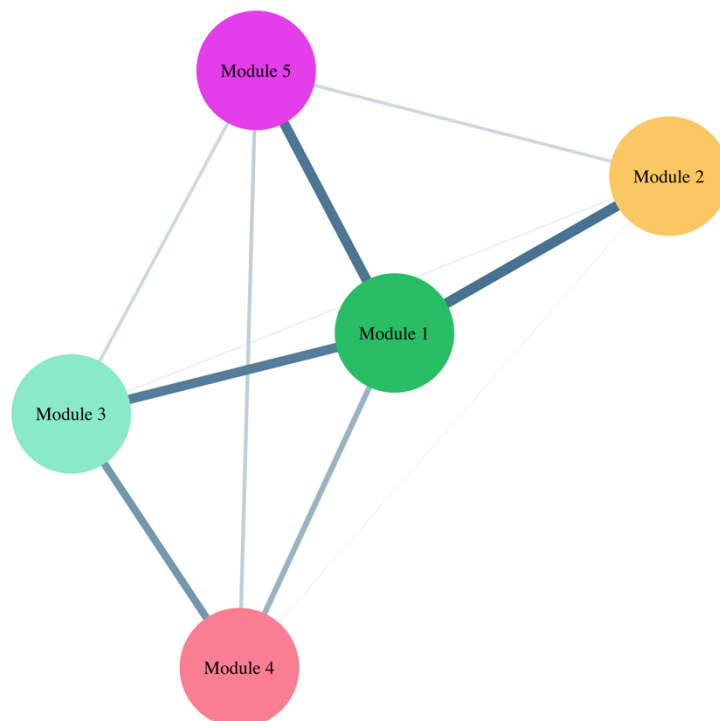
Graphical representation of the module's network

Figure S4. Graphical representation of the module's network. Nodes represent the 5 modules in the second hierarchical level. Edges between the nodes represent the interaction between the modules and the width of the edge represents the strength of the interaction.

Joint representation of the centrality measures in the second hierarchical level

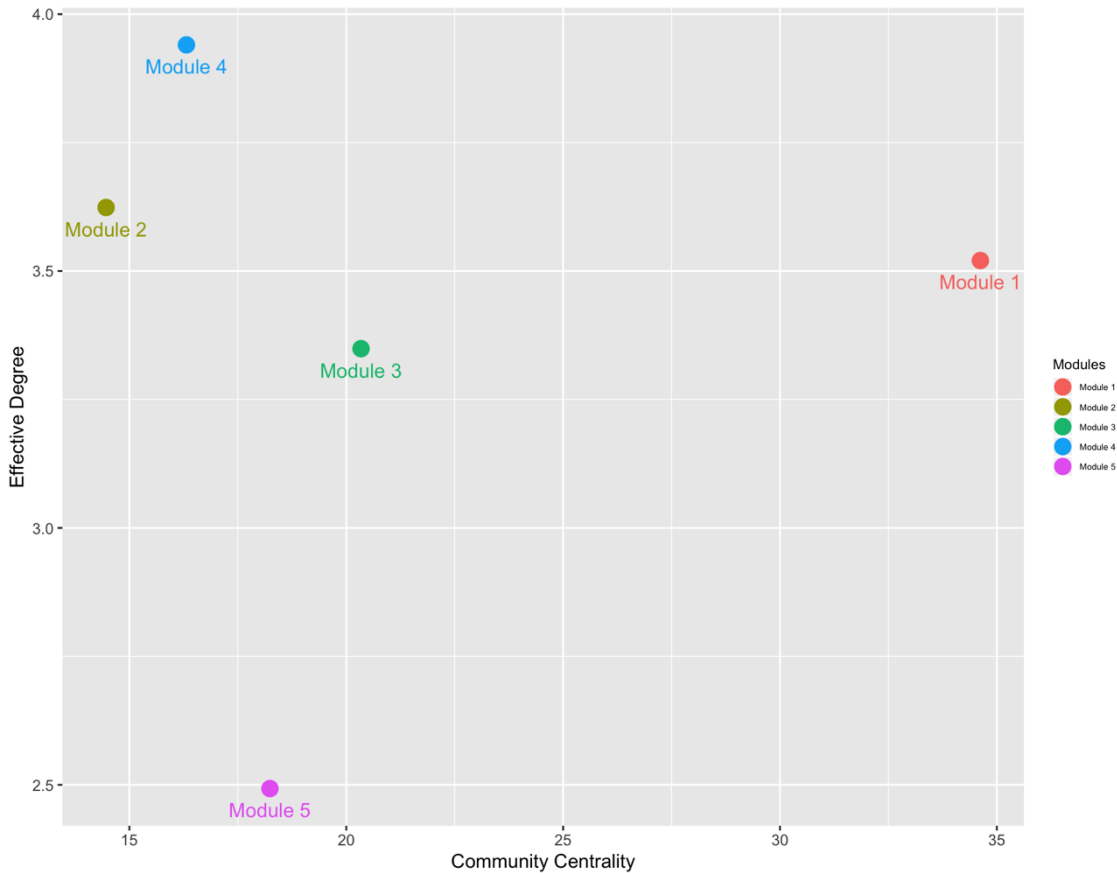


Figure S5. Joint representation of effective degree and community centrality in the second hierarchical level. Each dot represents a module of the second level network.

Robustness of the adolescents’ network

The robustness of the adolescent’s network was estimated based on the comparison of the networks estimated from 100 pairs of sub-samples that were randomly generated from the initial sample. A total of five indicators were evaluated: correlations between edges weights, difference in the total number of estimated edges, similarity index, proportion of edges that failed to replicate, and degree centrality measure (see Borsboom, et al., 2017; Letina, Blanken, Deserno, & Borsboom, 2019). Figure S6 displays the result of this comparisons.

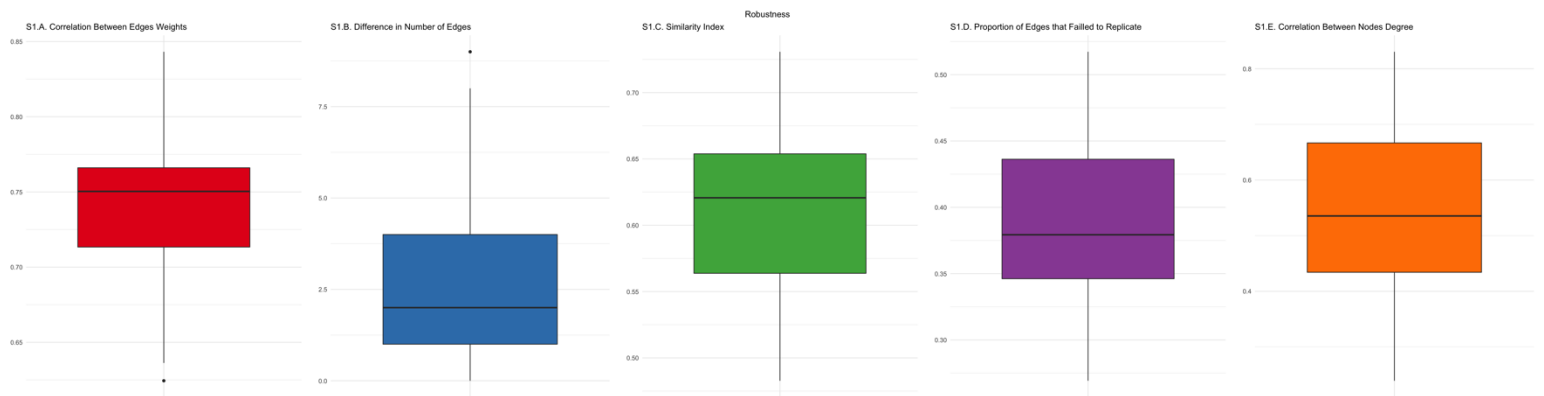


Figure S6. Robustness of the adolescent’s network

Global proprieties of the adolescent's network

Table S3

Global proprieties of the adolescent's network. Assortativity is a propriety that measures the level of homophily of a graph (Newman, 2002; 2003). Average path length it's the result of the estimation of the shortest paths between all pairs of nodes (West, 1996). Transitivity is the ratio of triangles and the connected triples in the graph (Wasserman & Faust, 1994; Barrat, Barthelemy, Pastor-Satorras, & Vespignani, 2004).

Network Propriety	Scores
Assortativity	-0.169
Average Path Length	2.037
Transitivity	0.289

Comparison with unweighted null methods

The properties of the adolescent's network were compared to the same properties in two different unweighted null models. Three proprieties were evaluated (i.e., assortativity, transitivity and average path; see page 11 of the Supplementary Materials for a brief description of these measures). Results from these comparisons are presented in Figures S7 and S8.

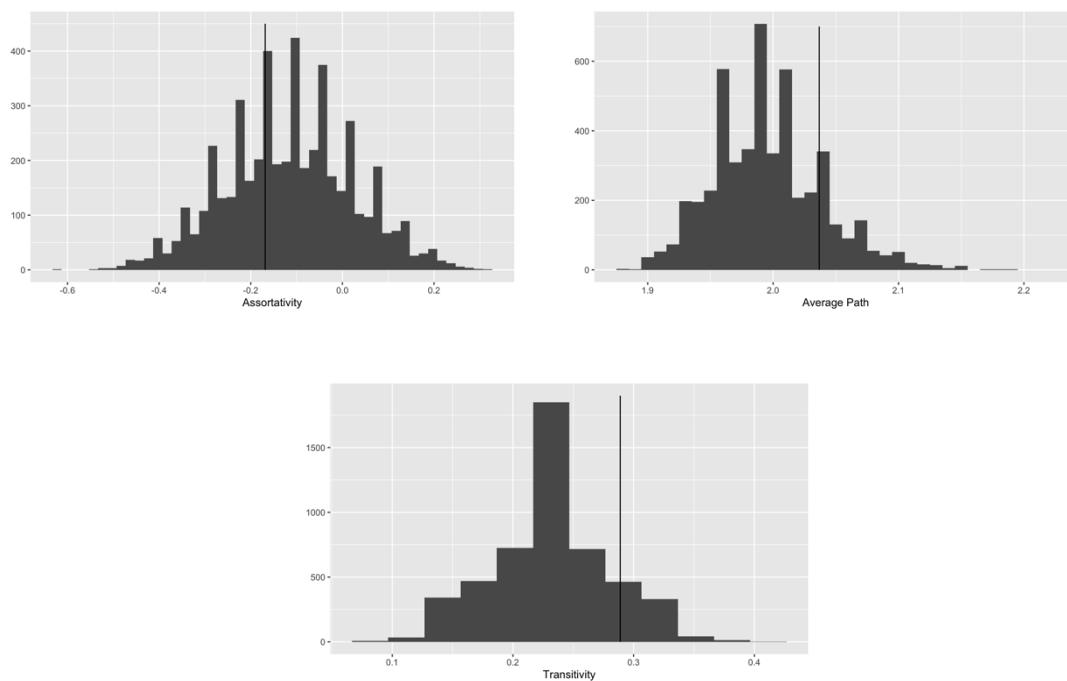


Figure S7. Histogram of the network properties in 5000 simulated networks obtained by randomizing the edges of the adolescent's network while maintaining the same degree distribution. Vertical line identifies the observed value for each property in the adolescent's network.

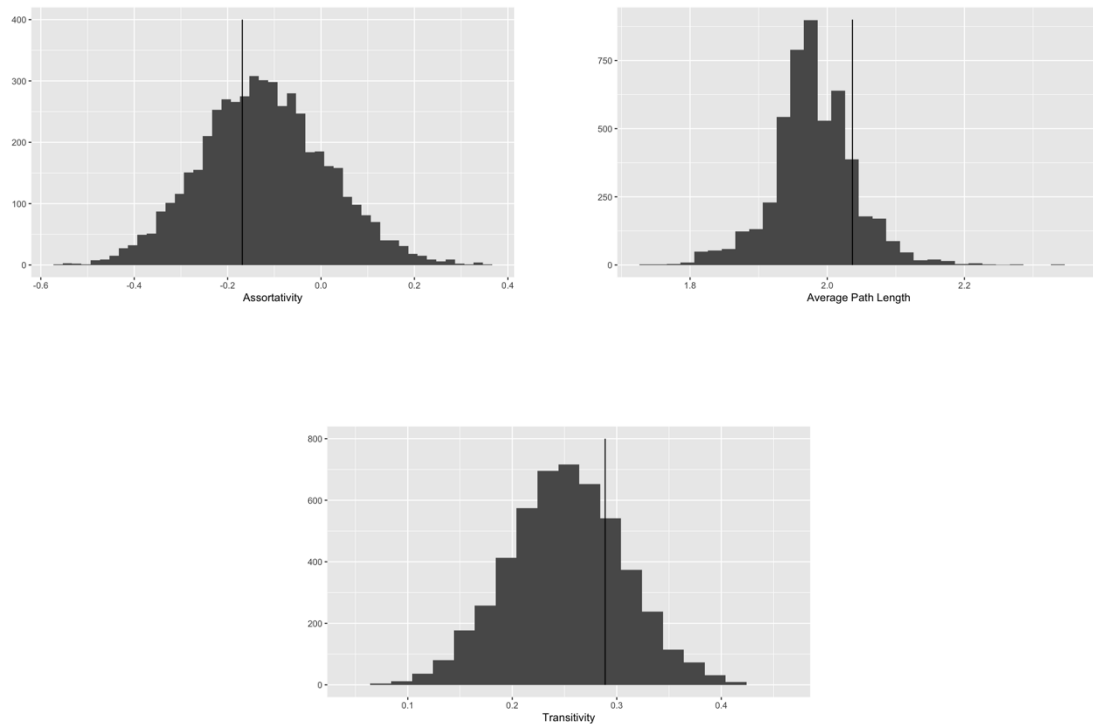


Figure S8. Histogram of the network properties in 5000 simulated networks obtained by generating a random graph with the same number of vertices and edges. Vertical line identifies the observed value for each property in the adolescent's network.

Robustness of the adolescent's network at different sample sizes

Randomized sub-samples of different sizes were generated, in order to explore the robustness of the adolescent's network. The procedure and the indicators used to analyze the samples of different sizes are described in page 10 of the Supplementary Materials. Results are showed in Figure *S9* and Figure *S10*.

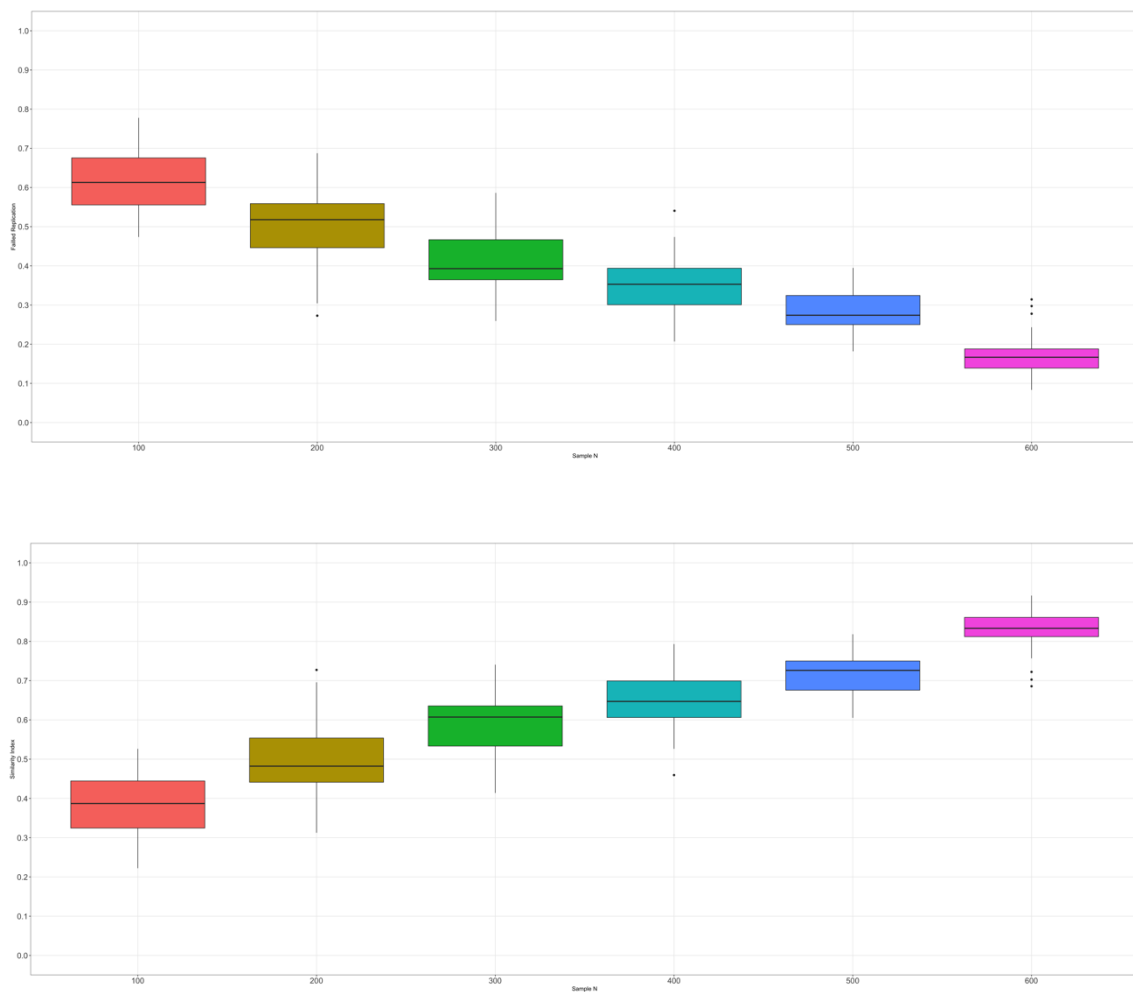


Figure *S9*. Robustness of the adolescent's network at different sample sizes

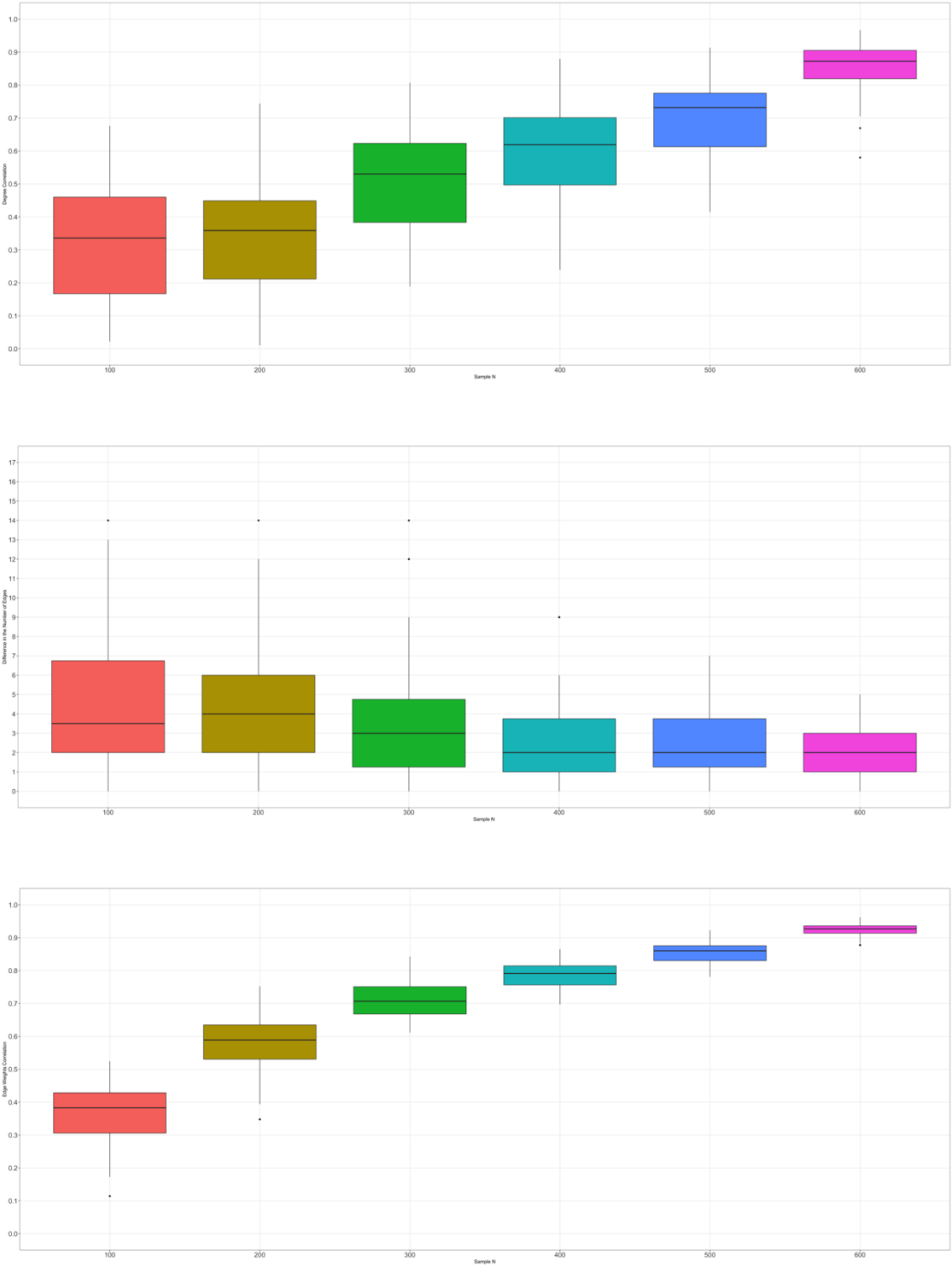


Figure S10. Robustness of the adolescent’s network at different sample sizes

Distribution scores for each item of the PCL

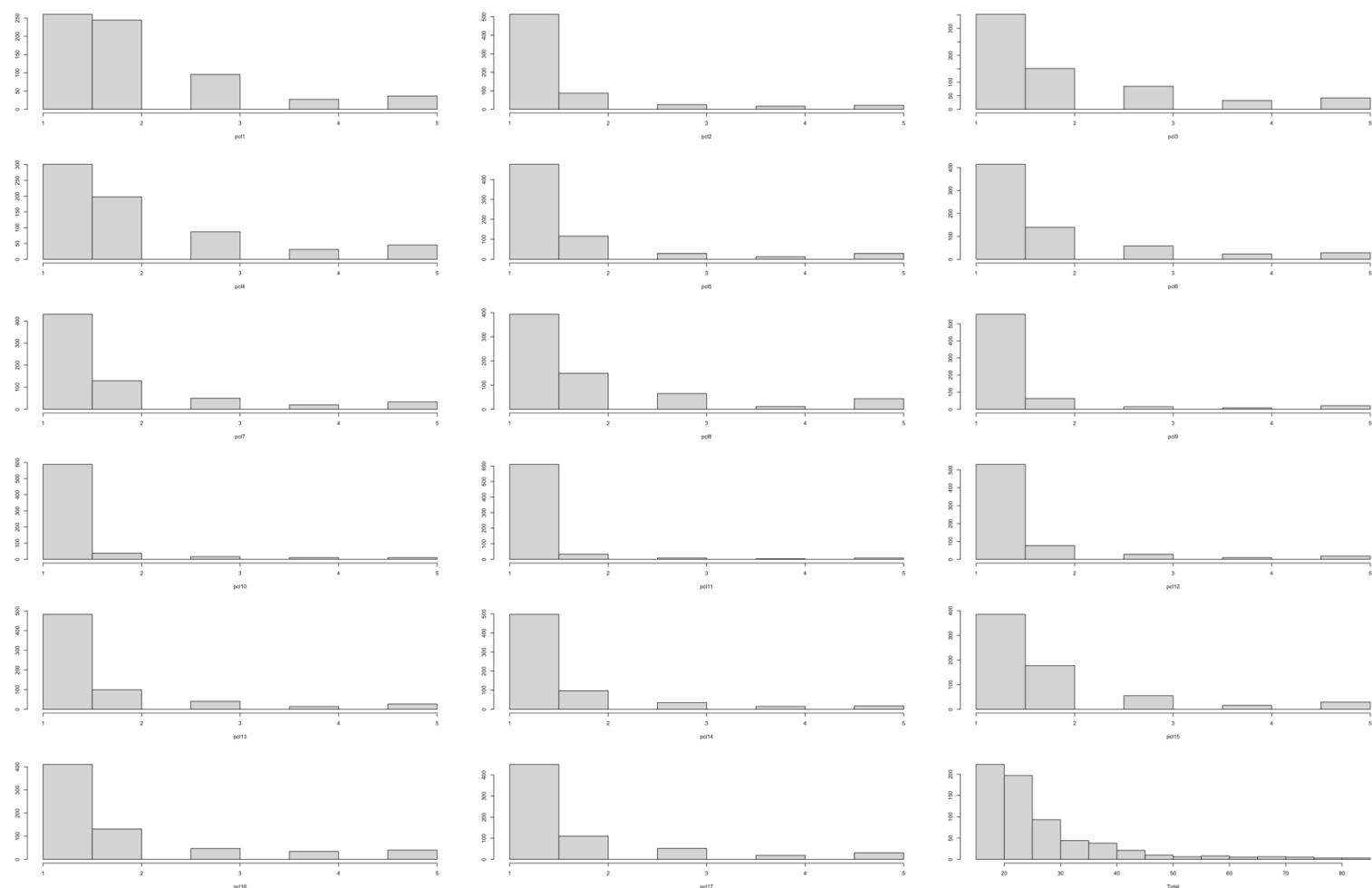


Figure S11. Distribution scores for each item of the PCL

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Chapter IV ¹

**The Nosographic Structure of Posttraumatic Stress Symptoms across Different
Types of Trauma: An Exploratory Network Analysis Approach**

Note:

¹ This chapter was published as an independent paper:

Ferreira, F., Gysi, D., Castro, D. & Ferreira, T. (2021). The nosographic structure of posttraumatic stress symptoms across different types of trauma: An exploratory network analysis approach [Manuscript submitted for publication].

Table 1 – Results from CoDiNa for specific and common interactions

Node 1	Node 2	Armour <i>et al.</i> , 2017	Birkeland and Heir, 2017	McNally <i>et al.</i> , 2017	Spiller <i>et al.</i> , 2017	Moshier <i>et al.</i> , 2018	Phi	Phi tilde	Score center	Score Phi	Score Phi tilde	Score internal	Score ratio
Intrusive Thoughts	Nightmares	0,785	0,820	0,860	0,680	0,819	a		a 0,795	0,614	0,614	0,216	2,846
Flashbacks	Intrusive Thoughts	0,874	0,806	0,924	0,722	0,751	a		a 0,819	0,684	0,684	0,199	3,427
Flashbacks	Nightmares	1,000	0,775	0,847	0,544	0,630	a		a 0,776	0,557	0,557	0,289	1,925
Emotional React	Intrusive Thoughts	0,864	0,910	0,889	0,899	0,758	a		a 0,866	0,822	0,822	0,147	5,604
Emotional React	Nightmares	0,775	0,862	0,894	0,870	0,726	a		a 0,828	0,710	0,710	0,186	3,817
Emotional React	Flashbacks	0,963	0,881	0,902	0,930	0,747	a		a 0,888	0,888	0,888	0,137	6,483
Intrusive Thoughts	Physiological React	0,804	0,870	0,857	0,896	0,644	a		a 0,819	0,685	0,685	0,207	3,316
Nightmares	Physiological React	0,785	0,857	0,905	0,889	0,612	a		a 0,817	0,678	0,678	0,218	3,106
Flashbacks	Physiological React	0,911	0,898	0,901	0,848	0,694	a		a 0,854	0,789	0,789	0,170	4,634
Emotional React	Physiological React	0,961	0,909	0,955	0,953	0,779	a		a 0,914	0,964	0,964	0,112	8,599
Avoid Thoughts	Intrusive Thoughts	0,476	0,754	0,830	0,396	0,527	a		a 0,619	0,095	0,095	0,437	0,217
Avoid Thoughts	Nightmares	0,539	0,707	0,799	0,611	0,356	a		a 0,621	0,100	0,100	0,425	0,235
Avoid Thoughts	Flashbacks	0,654	0,726	0,808	0,437	0,295	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,687	0,546	0,495	0,399	1,242
Avoid Thoughts	Emotional React	0,618	0,857	0,847	0,864	0,687	a		a 0,781	0,573	0,573	0,248	2,314
Avoid Thoughts	Physiological React	0,785	0,838	0,870	0,785	0,452	a		a 0,761	0,513	0,513	0,295	1,739
Avoid Reminders	Intrusive Thoughts	0,579	0,756	0,875	0,415	0,473	a		a 0,643	0,164	0,164	0,418	0,393
Avoid Reminders	Nightmares	0,683	0,747	0,850	0,443	0,377	a		a 0,646	0,173	0,173	0,421	0,411
Avoid Reminders	Flashbacks	0,785	0,753	0,897	0,323	0,356	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,745	0,652	0,745	0,399	1,868
Avoid Reminders	Emotional React	0,644	0,867	0,892	0,778	0,665	a		a 0,776	0,558	0,558	0,252	2,216
Avoid Reminders	Physiological React	0,772	0,848	0,911	0,801	0,427	a		a 0,771	0,542	0,542	0,300	1,803
Avoid Reminders	Avoid Thoughts	0,639	0,837	0,913	0,430	0,840	a		a 0,753	0,488	0,488	0,321	1,520
Amnesia	Intrusive Thoughts	0,390	0,707	0,761	0,532	0,164	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,621	0,425	0,171	0,436	0,392
Amnesia	Nightmares	0,424	0,701	0,729	0,408	0,053	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,586	0,361	0,000	0,460	0,000
Amnesia	Flashbacks	0,487	0,730	0,734	0,038	0,000	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,661	0,499	0,000	0,369	0,000
Amnesia	Emotional React	0,270	0,791	0,754	0,554	0,345	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,650	0,480	0,385	0,449	0,859
Amnesia	Physiological React	0,377	0,823	0,757	0,617	0,199	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,673	0,521	0,429	0,408	1,053
Amnesia	Avoid Thoughts	0,147	0,677	0,626	0,193	0,310	g	Birkeland and Heir, 2017; McNally et al., 2017	0,709	0,587	0,000	0,446	0,000
Amnesia	Avoid Reminders	0,351	0,730	0,726	0,130	0,445	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018;	0,591	0,372	0,000	0,473	0,000
Intrusive Thoughts	Negative Beliefs	0,476	0,697	0,850	0,547	0,295	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,675	0,524	0,437	0,413	1,060
Negative Beliefs	Nightmares	0,335	0,701	0,690	0,687	0,153	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,627	0,437	0,204	0,433	0,472
Flashbacks	Negative Beliefs	0,505	0,717	0,814	0,497	0,135	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,651	0,481	0,322	0,397	0,810
Emotional React	Negative Beliefs	0,356	0,794	0,780	0,918	0,448	a		a 0,694	0,315	0,315	0,404	0,781
Negative Beliefs	Physiological React	0,398	0,810	0,787	0,880	0,249	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,753	0,668	0,823	0,361	2,281
Avoid Thoughts	Negative Beliefs	0,000	0,730	0,700	0,690	0,342	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,636	0,453	0,322	0,416	0,774
Avoid Reminders	Negative Beliefs	0,293	0,742	0,872	0,601	0,395	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,692	0,555	0,563	0,416	1,351
Amnesia	Negative Beliefs	0,285	0,711	0,614	0,475	0,427	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,586	0,361	0,108	0,479	0,226
Blame	Intrusive Thoughts	0,620	0,000	0,000	0,497	0,448	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,527	0,254	0,254	0,484	0,524
Blame	Nightmares	0,573	0,000	0,000	0,570	0,199	g	Armour et al., 2017; Spiller et al., 2017	0,589	0,366	0,800	0,451	1,773
Blame	Flashbacks	0,686	0,000	0,000	0,509	0,214	g	Armour et al., 2017; Spiller et al., 2017	0,623	0,429	0,944	0,439	2,152
Blame	Emotional React	0,649	0,000	0,000	0,987	0,569	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,757	0,675	0,675	0,321	2,104
Blame	Physiological React	0,660	0,000	0,000	0,880	0,374	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,671	0,516	0,516	0,417	1,237
Avoid Thoughts	Blame	0,309	0,000	0,000	0,528	0,381	g	Spiller et al., 2017; Moshier et al., 2018	0,510	0,222	0,093	0,592	0,158
Avoid Reminders	Blame	0,393	0,000	0,000	0,484	0,420	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,434	0,084	0,084	0,569	0,147
Amnesia	Blame	0,348	0,000	0,000	0,244	0,267	g	Armour et al., 2017	0,502	0,208	0,066	0,745	0,088
Blame	Negative Beliefs	0,429	0,000	0,000	0,826	0,630	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,649	0,477	0,477	0,405	1,176
Intrusive Thoughts	Negative Emotions	0,814	0,000	0,000	0,747	0,726	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,763	0,686	0,686	0,241	2,850
Negative Emotions	Nightmares	0,688	0,000	0,000	0,832	0,626	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,721	0,608	0,608	0,297	2,047
Flashbacks	Negative Emotions	0,746	0,000	0,000	0,820	0,594	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,726	0,618	0,618	0,295	2,092
Emotional React	Negative Emotions	0,812	0,000	0,000	1,000	0,722	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,853	0,849	0,849	0,194	4,383
Negative Emotions	Physiological React	0,720	0,000	0,000	0,943	0,687	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,791	0,737	0,737	0,245	3,012

Node 1	Node 2	Armour <i>et al.</i> , 2017	Birkeland and Heir, 2017	McNally <i>et al.</i> , 2017	Spiller <i>et al.</i> , 2017	Moshier <i>et al.</i> , 2018	Phi	Phi tilde	Score center	Score Phi	Score Phi tilde	Score internal	Score ratio
Avoid Thoughts	Negative Emotions	0,469	0,000	0,000	0,826	0,690	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,678	0,530	0,530	0,369	1,435
Avoid Reminders	Negative Emotions	0,618	0,000	0,000	0,718	0,662	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,667	0,510	0,510	0,337	1,517
Amnesia	Negative Emotions	0,571	0,000	0,000	0,658	0,580	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,604	0,395	0,395	0,399	0,990
Negative Beliefs	Negative Emotions	0,822	0,000	0,000	0,937	0,872	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,878	0,896	0,896	0,132	6,797
Blame	Negative Emotions	1,000	0,000	0,000	0,905	0,897	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,935	1,000	1,000	0,081	12,352
Intrusive Thoughts	Loss of Interest	0,571	0,829	0,846	0,383	0,463	a	a	0,646	0,175	0,175	0,426	0,410
Loss of Interest	Nightmares	0,589	0,838	0,882	0,256	0,384	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,714	0,596	0,598	0,404	1,478
Flashbacks	Loss of Interest	0,715	0,837	0,828	0,269	0,381	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,727	0,619	0,659	0,385	1,712
Emotional React	Loss of Interest	0,406	0,862	0,881	0,680	0,534	a	a	0,697	0,325	0,325	0,376	0,864
Loss of Interest	Physiological React	0,510	0,866	0,901	0,741	0,495	a	a	0,723	0,401	0,401	0,344	1,169
Avoid Thoughts	Loss of Interest	0,306	0,838	0,850	0,165	0,423	g	Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,758	0,677	1,000	0,409	2,445
Avoid Reminders	Loss of Interest	0,552	0,851	0,886	0,348	0,470	a	a	0,657	0,205	0,205	0,434	0,474
Amnesia	Loss of Interest	0,421	0,832	0,817	0,149	0,324	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017	0,745	0,652	0,688	0,418	1,647
Loss of Interest	Negative Beliefs	0,738	0,932	0,811	0,367	0,665	a	a	0,728	0,415	0,415	0,353	1,178
Blame	Loss of Interest	0,385	0,000	0,000	0,269	0,505	g	Armour et al., 2017; Moshier et al., 2018	0,488	0,182	0,000	0,590	0,000
Loss of Interest	Negative Emotions	0,636	0,000	0,000	0,585	0,797	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,679	0,532	0,532	0,339	1,567
Detachment	Intrusive Thoughts	0,424	0,835	0,901	0,443	0,438	a	a	0,644	0,169	0,169	0,446	0,379
Detachment	Nightmares	0,476	0,856	0,889	0,361	0,370	a	a	0,635	0,141	0,141	0,472	0,300
Detachment	Flashbacks	0,589	0,856	0,886	0,437	0,256	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,728	0,622	0,699	0,383	1,827
Detachment	Emotional React	0,209	0,866	0,870	0,864	0,473	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,794	0,742	1,000	0,306	3,268
Detachment	Physiological React	0,401	0,879	0,878	0,728	0,456	a	a	0,699	0,329	0,329	0,390	0,844
Avoid Thoughts	Detachment	0,230	0,856	0,891	0,494	0,395	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,703	0,576	0,612	0,421	1,453
Avoid Reminders	Detachment	0,366	0,857	0,917	0,513	0,495	a	a	0,666	0,232	0,232	0,429	0,541
Amnesia	Detachment	0,351	0,844	0,774	0,215	0,406	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,641	0,463	0,243	0,473	0,514
Detachment	Negative Beliefs	0,662	0,950	0,951	0,661	0,808	a	a	0,817	0,677	0,677	0,233	2,911
Blame	Detachment	0,312	0,000	0,000	0,617	0,612	g	Spiller et al., 2017; Moshier et al., 2018	0,653	0,484	0,999	0,444	2,251
Detachment	Negative Emotions	0,584	0,000	0,000	0,794	0,843	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,749	0,660	0,660	0,283	2,332
Detachment	Loss of Interest	0,788	0,975	0,918	0,459	0,975	a	a	0,846	0,763	0,763	0,263	2,902
Intrusive Thoughts	Restricted Affect	0,359	0,683	0,865	0,519	0,335	a	a	0,587	0,000	0,000	0,491	0,000
Nightmares	Restricted Affect	0,366	0,670	0,758	0,503	0,263	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,608	0,403	0,111	0,470	0,237
Flashbacks	Restricted Affect	0,526	0,685	0,841	0,389	0,210	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,642	0,464	0,276	0,438	0,632
Emotional React	Restricted Affect	0,071	0,778	0,837	0,772	0,448	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,726	0,617	0,708	0,331	2,142
Physiological React	Restricted Affect	0,309	0,804	0,836	0,801	0,324	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,854	0,851	1,000	0,319	3,134
Avoid Thoughts	Restricted Affect	0,021	0,686	0,734	0,373	0,331	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,561	0,316	0,002	0,502	0,004
Avoid Reminders	Restricted Affect	0,301	0,710	0,843	0,367	0,413	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,634	0,450	0,317	0,486	0,651
Amnesia	Restricted Affect	0,144	0,676	0,640	0,484	0,367	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,561	0,315	0,000	0,480	0,000
Negative Beliefs	Restricted Affect	0,565	0,865	0,809	0,734	0,662	a	a	0,735	0,435	0,435	0,293	1,487
Blame	Restricted Affect	0,099	0,000	0,000	0,522	0,456	g	Spiller et al., 2017; Moshier et al., 2018	0,495	0,195	0,000	0,517	0,000
Negative Emotions	Restricted Affect	0,500	0,000	0,000	0,772	0,797	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,703	0,575	0,575	0,338	1,701
Loss of Interest	Restricted Affect	0,809	0,929	0,854	0,472	0,865	a	a	0,802	0,635	0,635	0,268	2,365
Detachment	Restricted Affect	0,984	0,966	1,000	0,620	1,000	a	a	0,926	1,000	1,000	0,171	5,860
Intrusive Thoughts	Irritability	0,586	0,809	0,815	0,446	0,399	a	a	0,636	0,143	0,143	0,427	0,335
Irritability	Nightmares	0,534	0,813	0,786	0,617	0,356	a	a	0,644	0,166	0,166	0,415	0,401
Flashbacks	Irritability	0,670	0,810	0,806	0,127	0,331	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,686	0,544	0,459	0,402	1,142
Emotional React	Irritability	0,542	0,835	0,868	0,557	0,548	a	a	0,686	0,292	0,292	0,362	0,807
Irritability	Physiological React	0,628	0,850	0,885	0,797	0,370	a	a	0,731	0,425	0,425	0,350	1,215
Avoid Thoughts	Irritability	0,351	0,828	0,706	0,000	0,420	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,609	0,403	0,085	0,467	0,182
Avoid Reminders	Irritability	0,589	0,822	0,834	0,073	0,431	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,691	0,553	0,483	0,373	1,294
Amnesia	Irritability	0,490	0,795	0,632	0,253	0,317	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017	0,692	0,555	0,253	0,448	0,565
Irritability	Negative Beliefs	0,688	0,870	0,700	0,579	0,623	a	a	0,699	0,331	0,331	0,323	1,022
Blame	Irritability	0,524	0,000	0,000	0,500	0,470	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,498	0,201	0,201	0,503	0,400
Irritability	Negative Emotions	0,743	0,000	0,000	0,592	0,769	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,706	0,580	0,580	0,309	1,880

Node 1	Node 2	Armour <i>et al.</i> , 2017	Birkeland and Heir, 2017	McNally <i>et al.</i> , 2017	Spiller <i>et al.</i> , 2017	Moshier <i>et al.</i> , 2018	Phi	Phi tilde	Score center	Score Phi	Score Phi tilde	Score internal	Score ratio
Irritability	Loss of Interest	0,652	0,915	0,863	0,044	0,616	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,773	0,703	0,881	0,272	3,234
Detachment	Irritability	0,636	0,929	0,892	0,098	0,683	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,797	0,747	1,000	0,254	3,930
Irritability	Restricted Affect	0,670	0,876	0,767	0,478	0,584	a	a	0,689	0,301	0,301	0,353	0,851
Destructive Behavior	Intrusive Thoughts	0,424	0,000	0,000	0,617	0,253	g	Armour et al., 2017; Spiller et al., 2017	0,559	0,312	0,674	0,521	1,295
Destructive Behavior	Nightmares	0,647	0,000	0,000	0,595	0,192	g	Armour et al., 2017; Spiller et al., 2017	0,636	0,453	1,000	0,404	2,477
Destructive Behavior	Flashbacks	0,647	0,000	0,000	0,310	0,093	g	Armour et al., 2017	0,723	0,612	1,000	0,479	2,087
Destructive Behavior	Emotional React	0,325	0,000	0,000	0,658	0,498	g	Spiller et al., 2017; Moshier et al., 2018	0,627	0,437	0,837	0,487	1,719
Destructive Behavior	Physiological React	0,534	0,000	0,000	0,826	0,349	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,602	0,392	0,392	0,473	0,828
Avoid Reminders	Destructive Behavior	0,448	0,000	0,000	0,339	0,370	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,388	0,000	0,000	0,616	0,000
Amnesia	Destructive Behavior	0,416	0,000	0,000	0,608	0,096	g	Armour et al., 2017; Spiller et al., 2017	0,525	0,251	0,533	0,502	1,061
Destructive Behavior	Negative Beliefs	0,317	0,000	0,000	0,703	0,509	g	Spiller et al., 2017; Moshier et al., 2018	0,653	0,484	1,000	0,464	2,157
Blame	Destructive Behavior	0,393	0,000	0,000	0,513	0,270	g	Armour et al., 2017; Spiller et al., 2017	0,495	0,195	0,406	0,583	0,696
Destructive Behavior	Negative Emotions	0,623	0,000	0,000	0,734	0,626	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,663	0,503	0,503	0,343	1,467
Destructive Behavior	Loss of Interest	0,560	0,000	0,000	0,326	0,452	g	Armour et al., 2017; Moshier et al., 2018	0,559	0,312	0,618	0,548	1,128
Destructive Behavior	Detachment	0,547	0,000	0,000	0,301	0,580	g	g Armour et al., 2017 Moshier et al., 2018	0,603	0,392	1,000	0,486	2,059
Destructive Behavior	Restricted Affect	0,421	0,000	0,000	0,642	0,498	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,529	0,257	0,257	0,488	0,527
Destructive Behavior	Irritability	0,709	0,000	0,000	0,864	0,591	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,730	0,625	0,625	0,300	2,081
Hypervigilance	Intrusive Thoughts	0,380	0,817	0,840	0,598	0,409	a	a	0,639	0,153	0,153	0,437	0,351
Hypervigilance	Nightmares	0,560	0,785	0,805	0,516	0,359	a	a	0,628	0,121	0,121	0,430	0,281
Flashbacks	Hypervigilance	0,702	0,794	0,808	0,484	0,203	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,716	0,599	0,639	0,345	1,853
Emotional React	Hypervigilance	0,327	0,859	0,875	0,731	0,594	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,790	0,735	0,983	0,308	3,192
Hypervigilance	Physiological React	0,521	0,853	0,875	0,861	0,423	a	a	0,733	0,429	0,429	0,352	1,221
Avoid Thoughts	Hypervigilance	0,288	0,776	0,786	0,392	0,427	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,640	0,461	0,341	0,468	0,729
Avoid Reminders	Hypervigilance	0,374	0,792	0,878	0,478	0,488	a	a	0,633	0,135	0,135	0,444	0,305
Amnesia	Hypervigilance	0,288	0,738	0,715	0,522	0,256	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,702	0,573	0,000	0,419	0,000
Hypervigilance	Negative Beliefs	0,325	0,766	0,785	0,661	0,431	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,695	0,560	0,575	0,402	1,430
Blame	Hypervigilance	0,173	0,000	0,000	0,582	0,327	g	Spiller et al., 2017	0,690	0,552	0,604	0,558	1,082
Hypervigilance	Negative Emotions	0,526	0,000	0,000	0,766	0,708	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,675	0,524	0,524	0,349	1,502
Hypervigilance	Loss of Interest	0,560	0,848	0,846	0,421	0,459	a	a	0,654	0,196	0,196	0,417	0,471
Detachment	Hypervigilance	0,568	0,857	0,857	0,468	0,544	a	a	0,679	0,272	0,272	0,379	0,718
Hypervigilance	Restricted Affect	0,372	0,736	0,741	0,614	0,363	a	a	0,590	0,007	0,007	0,466	0,015
Hypervigilance	Irritability	0,521	0,842	0,799	0,576	0,566	a	a	0,674	0,256	0,256	0,364	0,703
Destructive Behavior	Hypervigilance	0,429	0,000	0,000	0,677	0,342	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,503	0,210	0,210	0,536	0,392
Intrusive Thoughts	Startle Response	0,458	0,916	0,836	0,658	0,377	a	a	0,682	0,278	0,278	0,408	0,682
Nightmares	Startle Response	0,696	0,910	0,833	0,623	0,356	a	a	0,710	0,363	0,363	0,370	0,980
Flashbacks	Startle Response	0,801	0,898	0,812	0,560	0,267	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,790	0,734	1,000	0,296	3,381
Emotional React	Startle Response	0,440	0,922	0,892	0,782	0,587	a	a	0,748	0,474	0,474	0,332	1,429
Physiological React	Startle Response	0,657	0,931	0,901	0,883	0,473	a	a	0,789	0,596	0,596	0,291	2,048
Avoid Thoughts	Startle Response	0,353	0,875	0,802	0,487	0,278	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,680	0,533	0,461	0,451	1,023
Avoid Reminders	Startle Response	0,476	0,897	0,865	0,665	0,391	a	a	0,689	0,300	0,300	0,396	0,758
Amnesia	Startle Response	0,296	0,834	0,741	0,481	0,299	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,742	0,647	0,267	0,425	0,628
Negative Beliefs	Startle Response	0,398	0,876	0,744	0,627	0,359	a	a	0,633	0,134	0,134	0,446	0,301
Blame	Startle Response	0,304	0,000	0,000	0,658	0,256	g	Spiller et al., 2017	0,769	0,696	1,000	0,524	1,908
Negative Emotions	Startle Response	0,579	0,000	0,000	0,766	0,644	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,667	0,510	0,510	0,346	1,475
Loss of Interest	Startle Response	0,639	0,900	0,856	0,519	0,498	a	a	0,703	0,340	0,340	0,359	0,948
Detachment	Startle Response	0,607	0,901	0,882	0,516	0,505	a	a	0,704	0,346	0,346	0,362	0,954
Restricted Affect	Startle Response	0,469	0,854	0,787	0,617	0,320	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,716	0,600	0,641	0,387	1,658
Irritability	Startle Response	0,576	0,928	0,859	0,604	0,562	a	a	0,723	0,400	0,400	0,333	1,202
Destructive Behavior	Startle Response	0,581	0,000	0,000	0,699	0,352	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,563	0,320	0,320	0,478	0,669
Hypervigilance	Startle Response	0,725	1,000	0,936	0,896	0,726	a	a	0,864	0,817	0,817	0,182	4,486
Conc Problems	Intrusive Thoughts	0,482	0,894	0,854	0,415	0,406	a	a	0,648	0,178	0,178	0,447	0,400
Conc Problems	Nightmares	0,487	0,885	0,838	0,263	0,214	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017	0,783	0,721	1,000	0,373	2,682

Node 1	Node 2	Armour <i>et al.</i> , 2017	Birkeland and Heir, 2017	McNally <i>et al.</i> , 2017	Spiller <i>et al.</i> , 2017	Moshier <i>et al.</i> , 2018	Phi	Phi tilde	Score center	Score Phi	Score Phi tilde	Score internal	Score ratio
Conc Problems	Flashbacks	0,644	0,872	0,853	0,411	0,210	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,727	0,620	0,694	0,373	1,864
Conc Problems	Emotional React	0,233	0,909	0,872	0,703	0,409	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,758	0,677	0,848	0,359	2,360
Conc Problems	Physiological React	0,445	0,909	0,882	0,778	0,441	a	a	0,721	0,396	0,396	0,372	1,066
Avoid Thoughts	Conc Problems	0,131	0,890	0,849	0,234	0,196	g	Birkeland and Heir, 2017; McNally et al., 2017	0,900	0,937	1,000	0,270	3,709
Avoid Reminders	Conc Problems	0,223	0,887	0,856	0,427	0,267	g	Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,779	0,715	0,511	0,401	1,275
Amnesia	Conc Problems	0,411	0,847	0,732	0,193	0,292	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017	0,718	0,602	0,465	0,434	1,071
Conc Problems	Negative Beliefs	0,589	0,926	0,847	0,503	0,441	a	a	0,689	0,299	0,299	0,389	0,769
Blame	Conc Problems	0,356	0,000	0,000	0,449	0,267	g	Armour et al., 2017; Spiller et al., 2017	0,447	0,108	0,204	0,628	0,324
Conc Problems	Negative Emotions	0,620	0,000	0,000	0,699	0,715	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,680	0,533	0,533	0,324	1,643
Conc Problems	Loss of Interest	0,819	0,957	0,886	0,513	0,705	a	a	0,792	0,603	0,603	0,273	2,211
Conc Problems	Detachment	0,751	0,956	0,913	0,383	0,733	a	a	0,774	0,551	0,551	0,324	1,704
Conc Problems	Restricted Affect	0,673	0,910	0,865	0,443	0,555	a	a	0,712	0,368	0,368	0,358	1,026
Conc Problems	Irritability	0,597	0,997	0,895	0,209	0,466	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,777	0,710	0,901	0,354	2,543
Conc Problems	Destructive Behavior	0,406	0,000	0,000	0,354	0,167	g	Armour et al., 2017; Spiller et al., 2017	0,399	0,020	0,000	0,632	0,000
Conc Problems	Hypervigilance	0,424	0,925	0,873	0,671	0,253	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,760	0,680	0,856	0,362	2,362
Conc Problems	Startle Response	0,589	0,954	0,918	0,728	0,359	a	a	0,743	0,460	0,460	0,364	1,264
Intrusive Thoughts	Sleep Disturbance	0,539	0,798	0,821	0,661	0,552	a	a	0,685	0,287	0,287	0,347	0,829
Nightmares	Sleep Disturbance	0,688	0,838	0,844	0,437	0,488	a	a	0,681	0,276	0,276	0,381	0,724
Flashbacks	Sleep Disturbance	0,788	0,782	0,805	0,734	0,235	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,787	0,728	0,985	0,253	3,892
Emotional React	Sleep Disturbance	0,380	0,826	0,849	0,804	0,559	a	a	0,708	0,356	0,356	0,366	0,972
Physiological React	Sleep Disturbance	0,605	0,848	0,859	0,839	0,427	a	a	0,736	0,439	0,439	0,333	1,320
Avoid Thoughts	Sleep Disturbance	0,243	0,798	0,709	0,190	0,317	g	Birkeland and Heir, 2017; McNally et al., 2017	0,817	0,784	0,565	0,401	1,409
Avoid Reminders	Sleep Disturbance	0,332	0,797	0,833	0,237	0,331	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,633	0,448	0,204	0,505	0,404
Amnesia	Sleep Disturbance	0,233	0,769	0,680	0,073	0,060	g	Birkeland and Heir, 2017; McNally et al., 2017	0,747	0,656	0,199	0,331	0,601
Negative Beliefs	Sleep Disturbance	0,351	0,842	0,710	0,342	0,292	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,620	0,424	0,170	0,512	0,331
Blame	Sleep Disturbance	0,277	0,000	0,000	0,437	0,238	g	Spiller et al., 2017	0,570	0,332	0,000	0,672	0,000
Negative Emotions	Sleep Disturbance	0,552	0,000	0,000	0,766	0,698	g	Armour et al., 2017; Spiller et al., 2017; Moshier et al., 2018	0,678	0,529	0,529	0,340	1,557
Loss of Interest	Sleep Disturbance	0,647	0,907	0,854	0,241	0,573	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,768	0,694	0,857	0,314	2,729
Detachment	Sleep Disturbance	0,634	0,922	0,908	0,339	0,580	a	a	0,711	0,365	0,365	0,391	0,935
Restricted Affect	Sleep Disturbance	0,505	0,826	0,716	0,348	0,420	a	a	0,591	0,012	0,012	0,473	0,025
Irritability	Sleep Disturbance	0,537	0,918	0,803	0,079	0,377	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Moshier et al., 2018	0,694	0,558	0,497	0,404	1,228
Destructive Behavior	Sleep Disturbance	0,366	0,000	0,000	0,294	0,125	g	Armour et al., 2017	0,486	0,179	0,000	0,710	0,000
Hypervigilance	Sleep Disturbance	0,455	0,809	0,812	0,544	0,427	a	a	0,632	0,133	0,133	0,425	0,313
Sleep Disturbance	Startle Response	0,634	0,913	0,884	0,671	0,420	a	a	0,727	0,412	0,412	0,346	1,190
Conc Problems	Sleep Disturbance	0,620	0,972	0,891	0,389	0,306	g	Armour et al., 2017; Birkeland and Heir, 2017; McNally et al., 2017; Spiller et al., 2017	0,769	0,697	0,902	0,395	2,284

a) Identifies the edges that are common to all networks

g) Identifies all the edges that are specific to a certain group of networks

Post-traumatic stress disorder symptoms across DSM-III, DSM-IV and DSM-5 versions

Table S1

Posttraumatic Stress Disorder Symptoms across DSM-III, DSM-IV and DSM-5 versions

DSM-III (American Psychological Association, 1980)	DSM-IV (American Psychological Association, 1994)	DSM-5 (American Psychological Association, 2013)
Intrusive Thoughts	Intrusive Thoughts	Intrusive Thoughts
Nightmares	Nightmares	Nightmares
Flashbacks	Flashbacks	Flashbacks
	Physiological Reactivity	Physiological Reactivity
Loss Interest	Emotional Reactivity	Emotional Reactivity
Detachment		
Emotional Numbing	Avoidance of Situations	Avoidance of Situations
	Avoidance of Thoughts	Avoidance of Thoughts
Startle Response or Hypervigilance	<u>Amnesia</u>	
Sleep Disturbance	Loss of Interest	Amnesia
Amnesia or Concentration Problems	Detachment	Foreshortened Future
Avoidance of Situations	Emotional Numbing	Blame
Avoidance of Thoughts	Foreshortened Future	Negative Emotional States
Guilt		Loss of Interest
	Sleep Disturbances	Detachment
	Irritability	Restricted Affect (i.e. Emotional Numbing)
	<u>Concentration Difficulties</u>	
	Hypervigilance	Irritability
	Startle Response	Destructive Behavior
		Hypervigilance
		Startle Response
		Concentration Problems
		Sleep Disturbance

Note: Symptoms that are in bold reflect the symptoms that were added across the versions of DSM

Symptoms that are underlined reflect the symptoms that were split and included as separate symptoms in next version of DS

Network with all the specific connections from all traumatic events

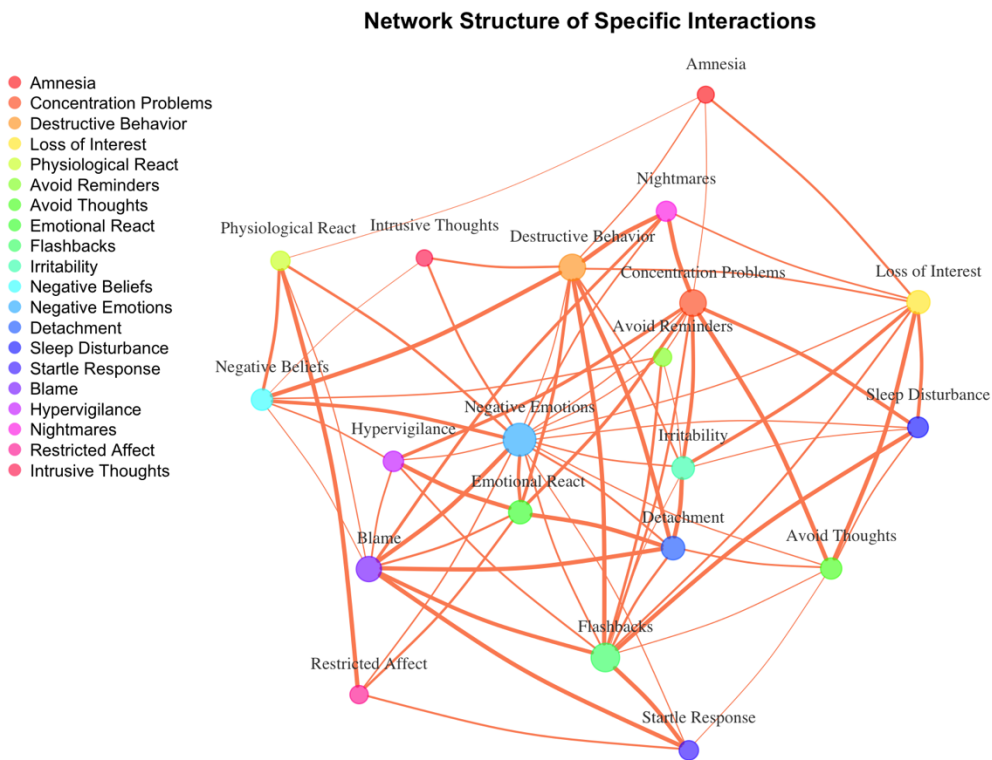


Figure 1. Network structure of specific connections across the different types of trauma. Nodes represent the symptoms of PTSD and the edges between the nodes represent the weighted topological overlap values between the symptoms across the different types of trauma. The thicker edges represent the higher weighted topological overlap values between the symptoms across the networks.

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Chapter V ¹

**Exposure to Traumatic Events and Development of Psychotic Symptoms in a
Prison Population: A Network Analysis Approach**

Note:

¹ This chapter was published as an independent paper:

Ferreira, F., Castro, D., Araújo, A. S., Fonseca, A. R., & Ferreira, T. B. (2020). Exposure to traumatic events and development of psychotic symptoms in a prison population: A network analysis approach. *Psychiatry Research*, 286, 112894. <https://doi.org/10.1016/j.psychres.2020.112894>

Shortest paths analysis

Shortest Paths from **Traumatic Events** to Psychosis

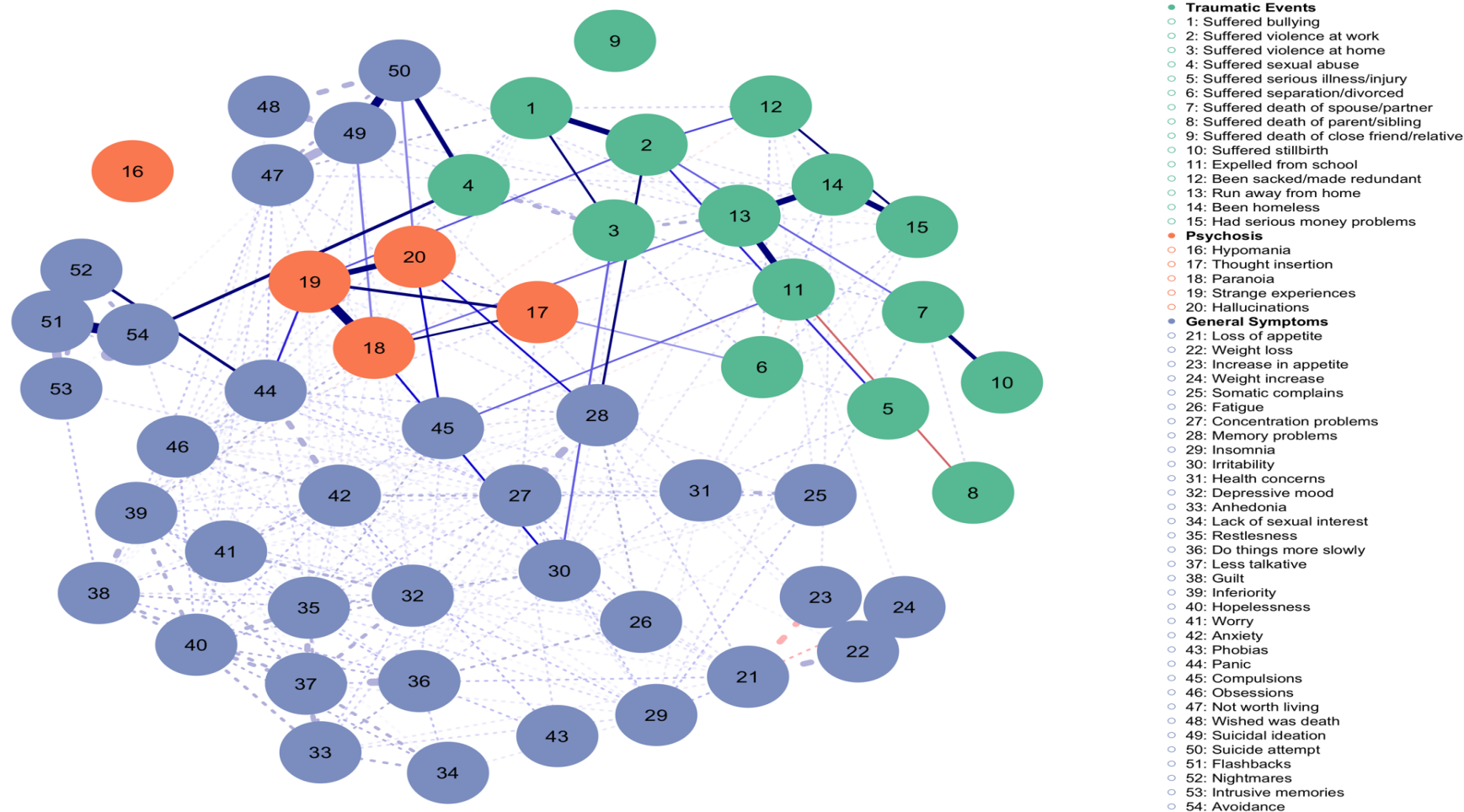


Figure 1. Shortest paths analysis from all traumatic events to the positive psychotic symptoms.

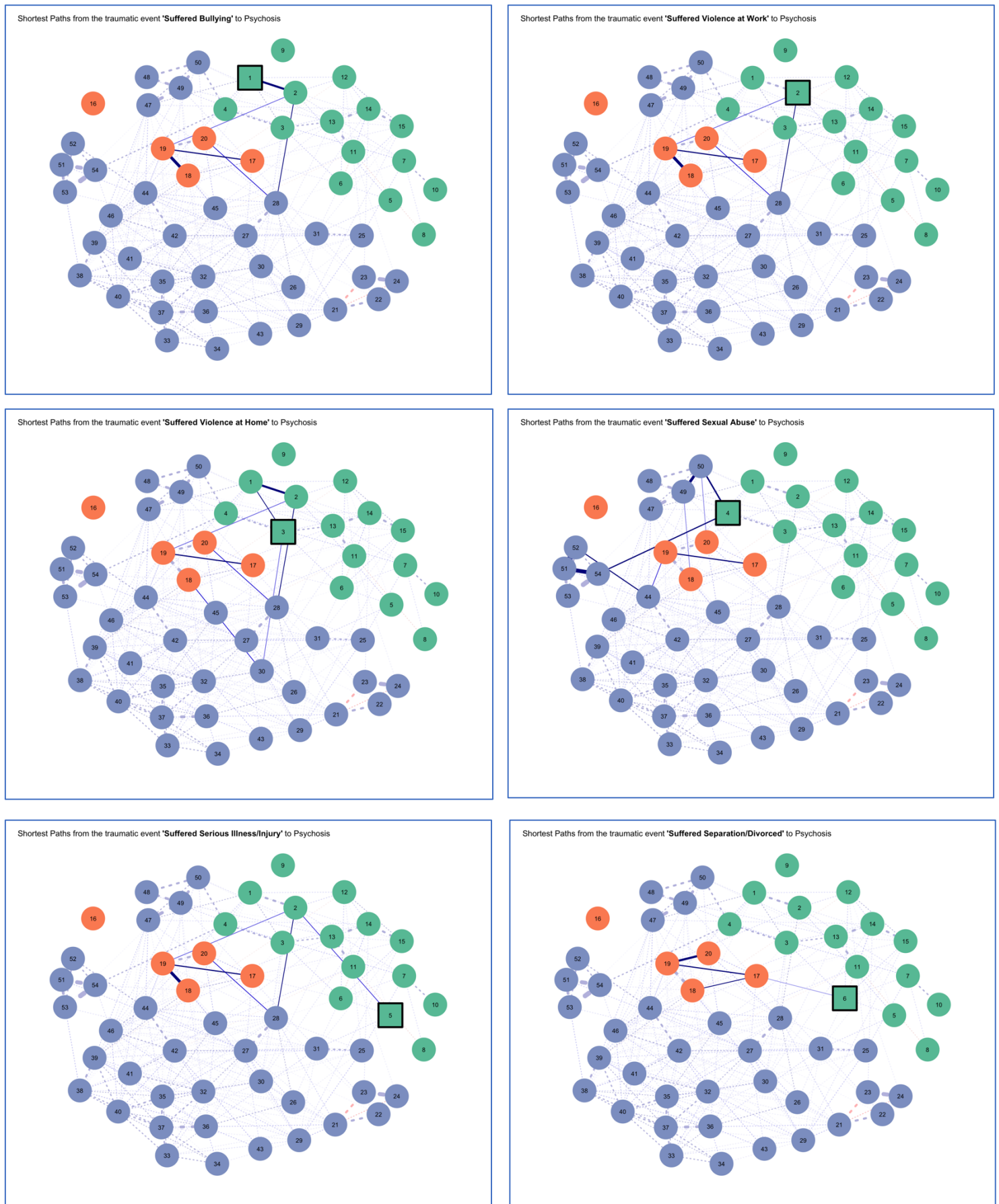


Figure 2. Shortest path from the traumatic events, suffered bullying, suffered violence at work, suffered violence at home, suffered sexual abuse, suffered serious illness/injury, suffered separation/divorce to psychotic symptoms.

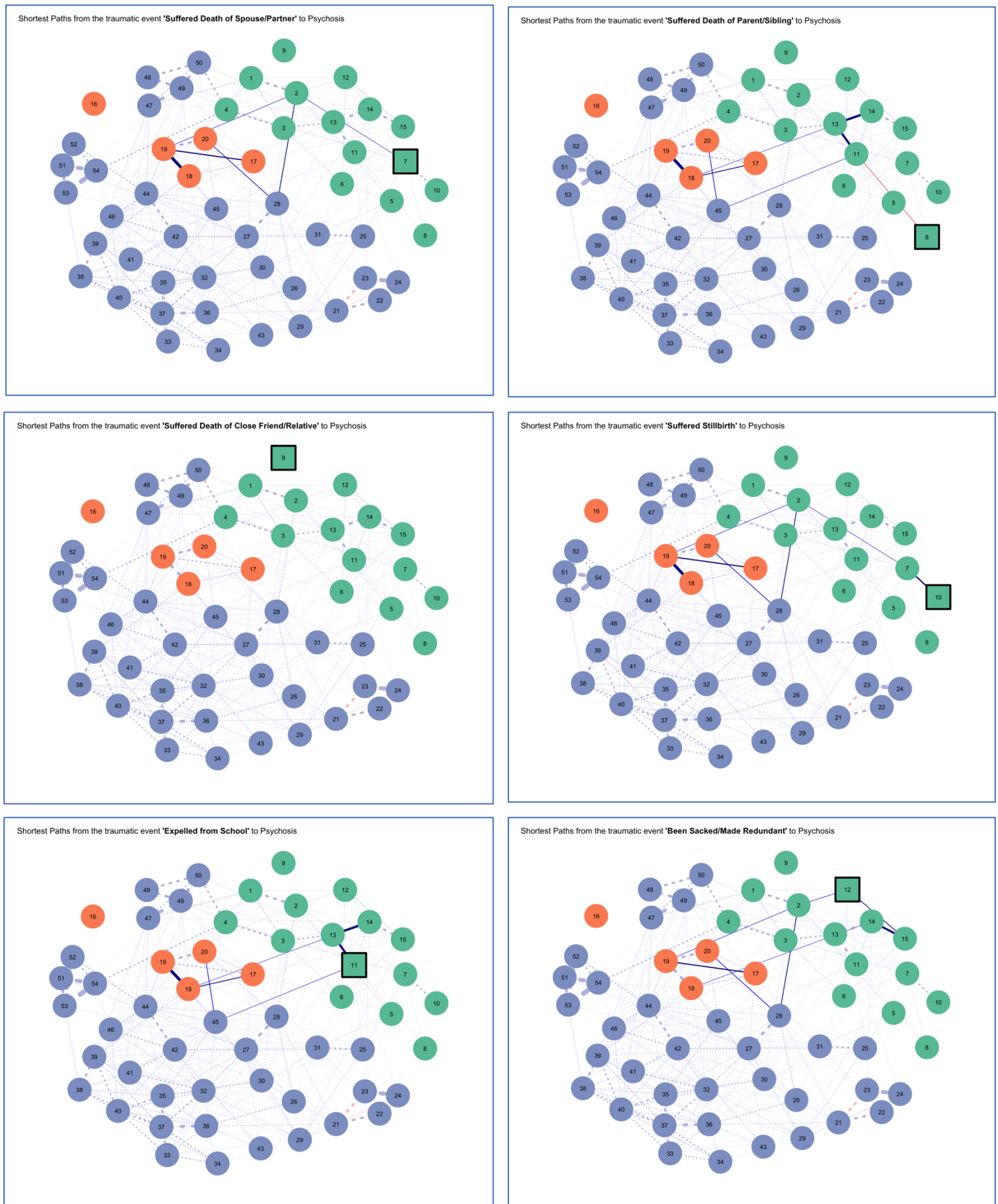


Figure 3. Shortest paths from the traumatic events, suffered death of spouse/partner, suffered death of parent/sibling, suffered death of close friend/relative, suffered stillbirth, expelled from school and been sacked/made redundant to psychotic symptoms.



Figure 4. Shortest paths from traumatic events, run away from home, been homeless and had serious money problems to psychotic symptoms.

Network stability and accuracy

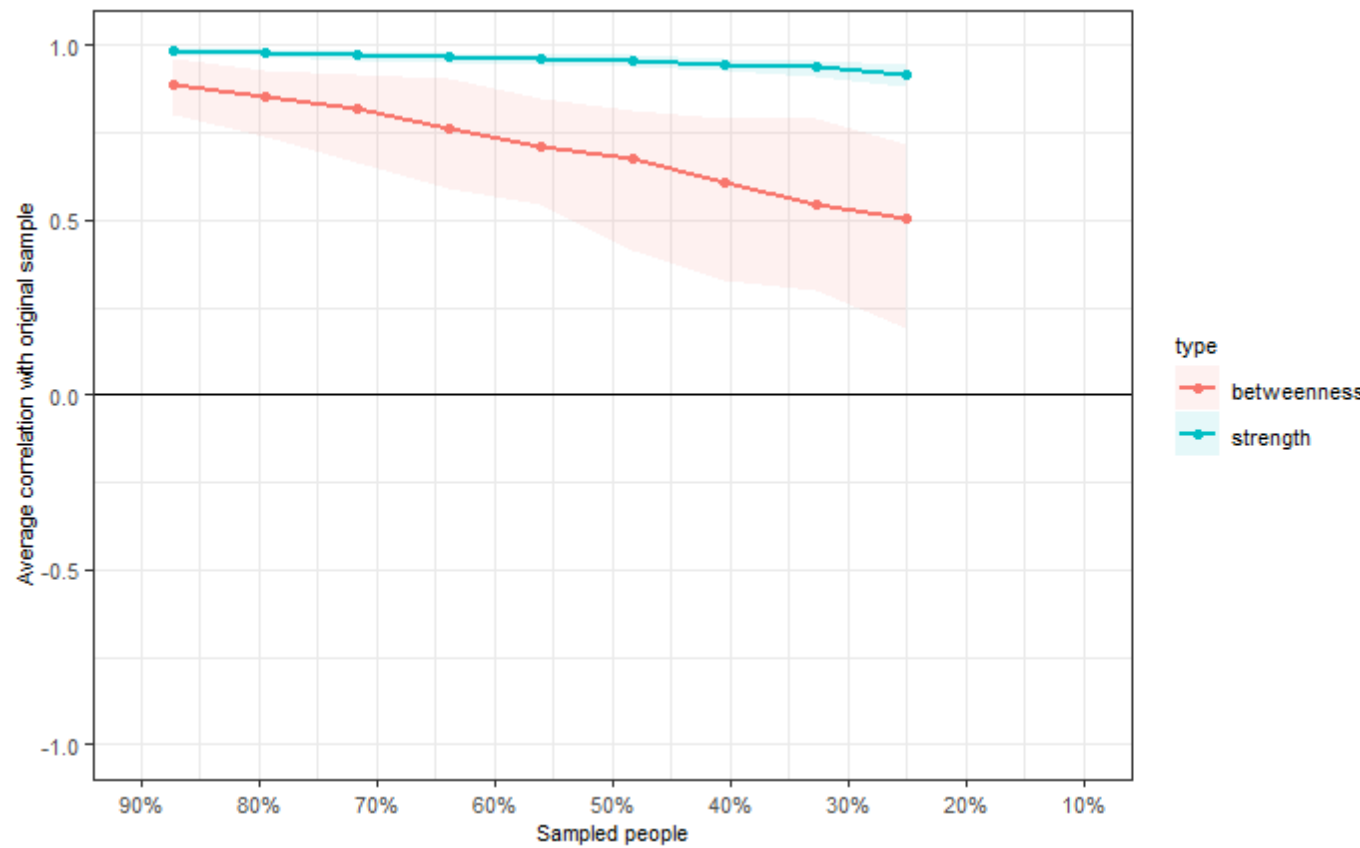


Figure 5. Average correlations between a sample of people dropped and the original sample with the centrality indices of networks. Means are indicated by the lines and the range of quantile is represented by the areas. The CS coefficient for strength was .75 and the CS coefficient for betweenness was .28. Closeness does not contain any variance and is therefore not shown.

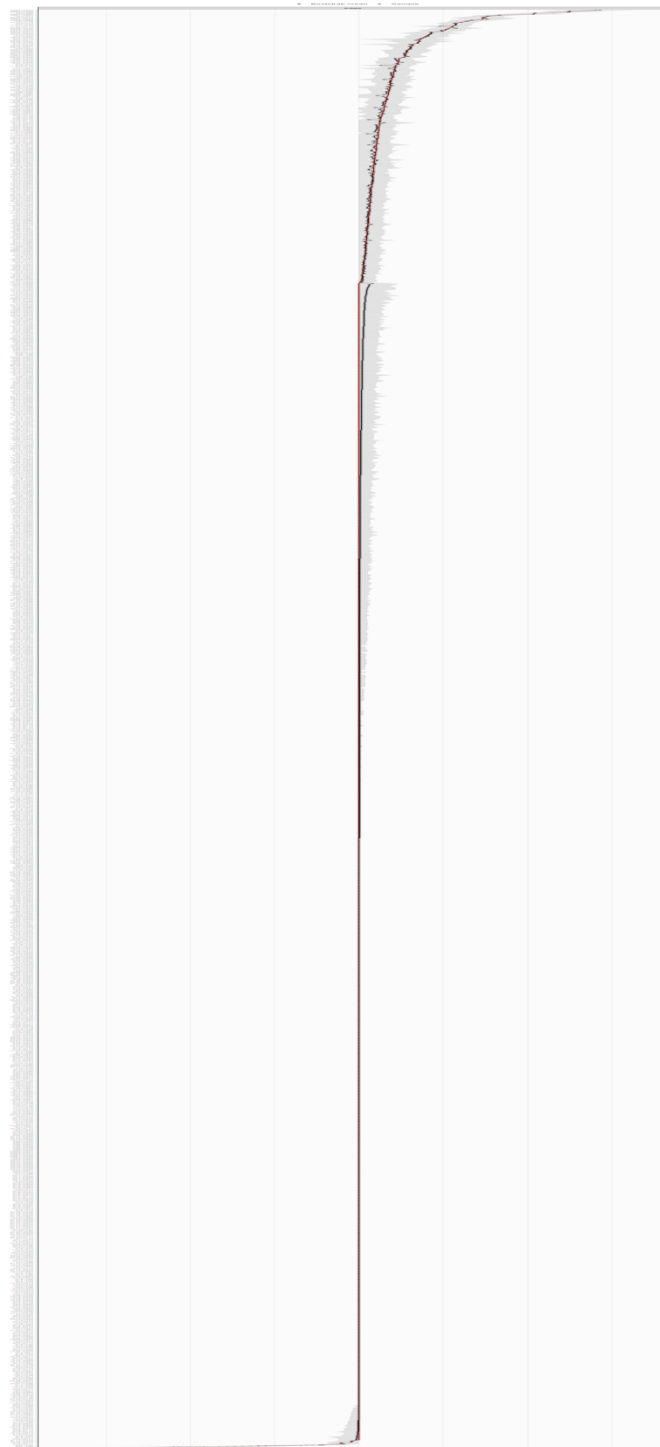


Figure 6. 95% bootstrapped CI of the estimated edge weights of the network. The red lines indicate the estimated edge weight. The red dots correspond to the estimated edge weigh for each edge. The gray areas indicate the corresponding 95% bootstrap.

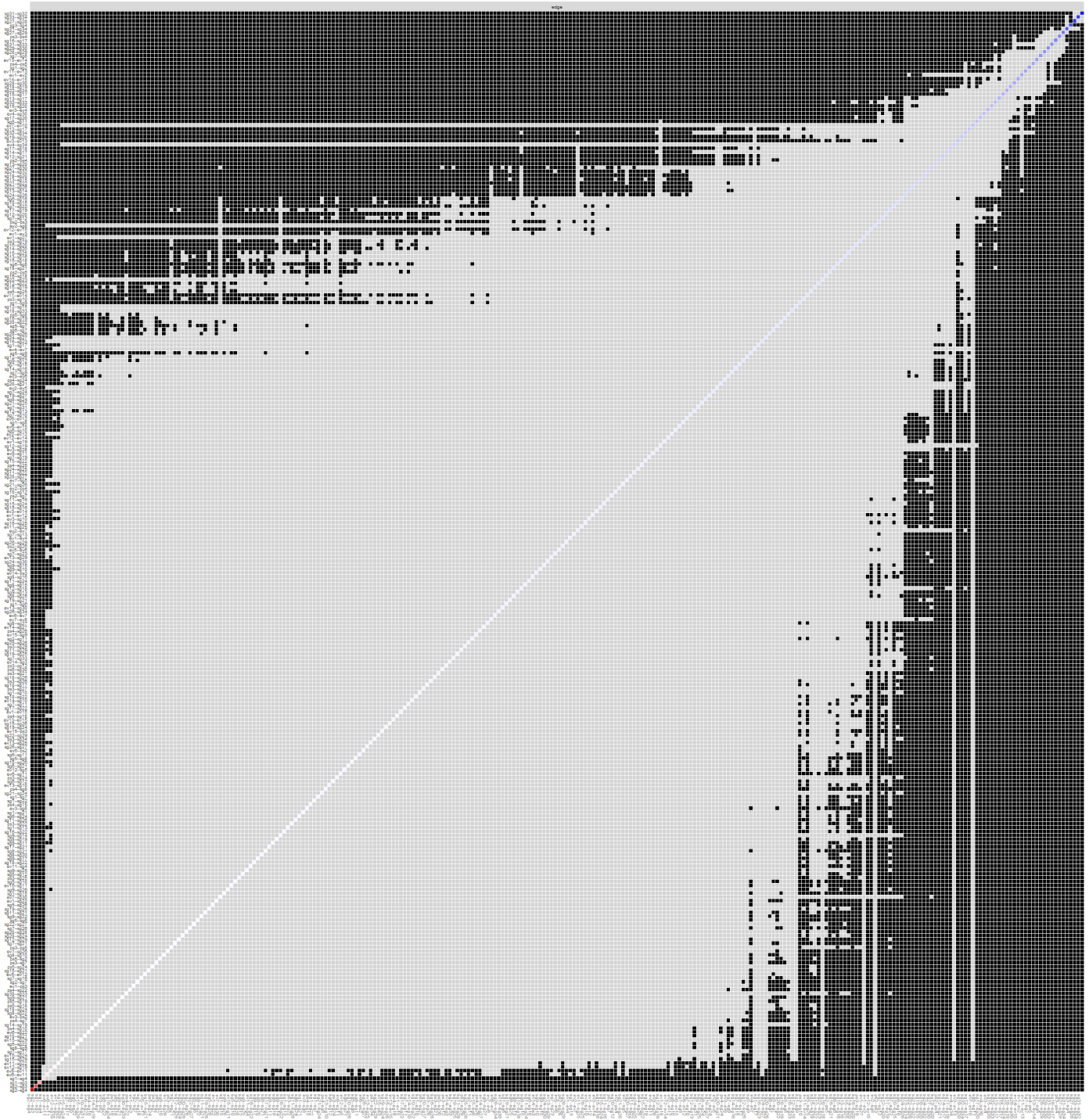


Figure 7. Bootstrap difference tests ($\alpha = 0.05$) between the weights of the connections between the traumatic events, general symptoms and positive psychotic symptoms that were non-zero in the estimated network. Connections do not differ significantly from one-another are represented in gray boxes and the black boxes represent the connections that do differ significantly.

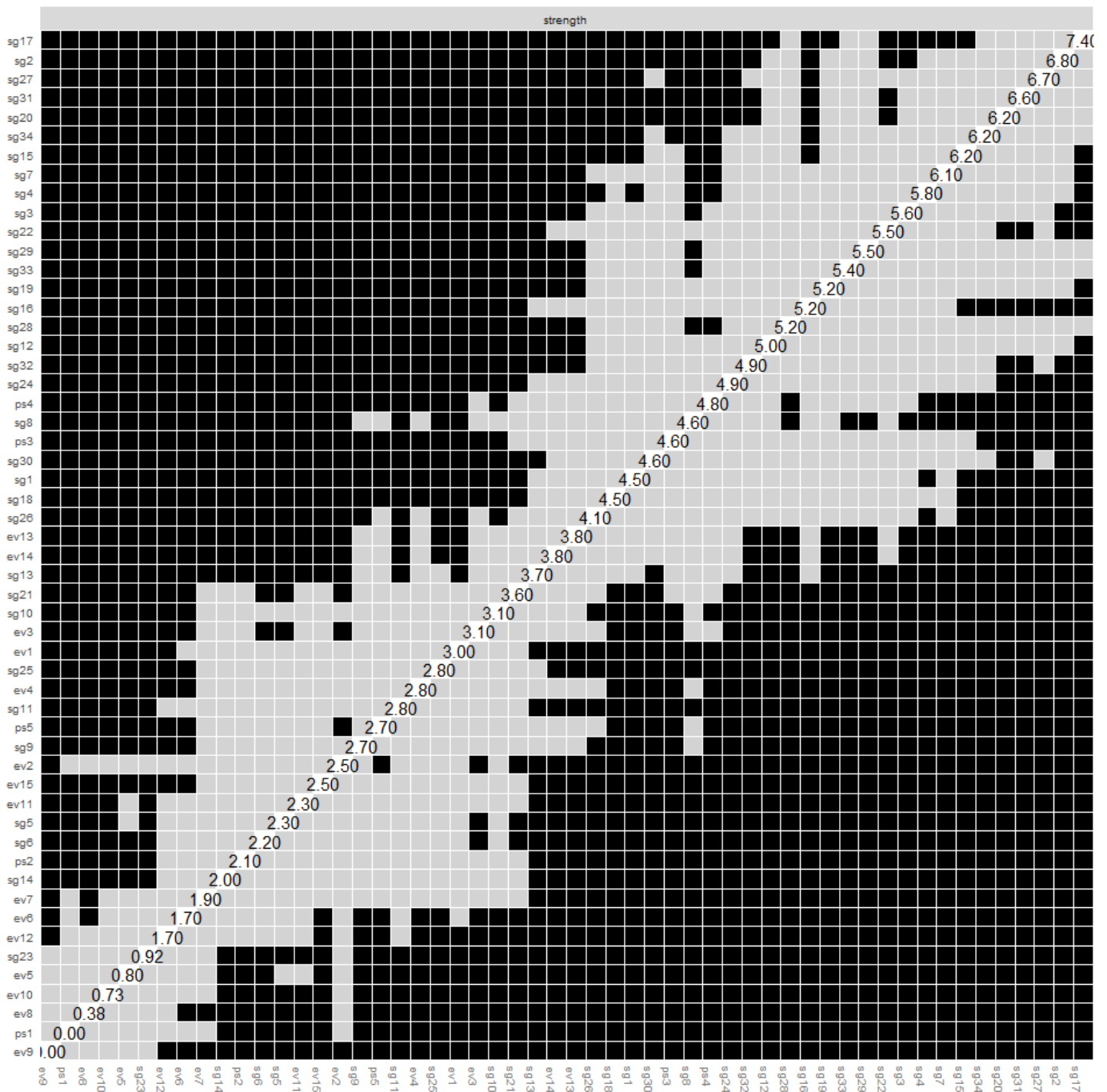


Figure 8. Bootstrap difference test ($\alpha = 0.05$) between the strength centrality of traumatic events, general symptoms and positive psychotic symptoms. Gray boxes represent the traumatic events, general and positive psychotic symptoms that do not differ significantly from one-another and the black boxes represent the traumatic events, general and positive psychotic symptoms that do not differ significantly. The value of each variable strength centrality is indicated in the white boxes.