



ALMA MATER STUDIORUM  
UNIVERSITÀ DI BOLOGNA

DOTTORATO DI RICERCA IN  
PSYCHOLOGY

Ciclo 36

**Settore Concorsuale:** 11/E4 - PSICOLOGIA CLINICA E DINAMICA

**Settore Scientifico Disciplinare:** M-PSI/08 - PSICOLOGIA CLINICA

NETWORK ANALYSIS MODELS OF EATING DISORDERS (NAMED): AN  
EXPLORATORY STUDY OF SYMPTOMS CONFIGURATION AT DIFFERENT  
STAGES OF ILLNESS

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Esame finale anno 2024

## Acknowledgements

In contrast with the stereotypical image of the scientist alone in their lab concocting weird mixtures, research is made possible only through the collaborative effort of many different people. Less mentioned is how research work is shaped not only by the contributions of our colleagues but also by our family and friends through their continued – and often discreet - support. In this spirit, I would like to express my gratitude to the “co-authors” who accompanied me along this journey. The last three and a half years have been full of personal upheavals, yet here we are, right in front of the finish line. This work would have not been possible without you, and it is as much yours as it is mine.

To prof. Elena Tomba and the EATeam (Lucia, Valentina, Elena, Edoardo), for welcoming me, and believing in me and this project. You’ve been my academic home for the last three years, and for that, I’m deeply grateful. While we might have arrived at the end of this particular journey, I’m confident more will follow.

To my Ph.D. programme colleagues, and in particular to Matteo, Luca, and Giovanbattista. Thank you for making these last three years so professionally rich and enjoyable. I hope I returned even in a small part all the energy and warmth you passed on to me.

To Gerardo, Alessia, and Annalisa for being the friends we didn’t know we needed. I could spend this whole section writing about how I hold you in high regard professionally, but the truth is that I just love spending time with you all, whether it is organizing seminars, visiting medieval-but-not-so-medieval towns, drafting research projects, or cooking (you) and eating (me) tons of parmigiana.

To Sam, Constantin, Charlotte, Lauriane, my beloved Isolde, and Ludovico. You are living proof that family is more than blood ties, and love goes past language barriers or geographical distance. While I wish I could spend more time with you all, I still feel luckier than most for having the privilege of being your friend. I will always be there for you no matter where I am or what I do.

To my family. Each in your way, you've supported me, cheered for me, believed and invested in me. Maybe it was by inspiring me academically, maybe it was by giving me advice, taking a step back and giving me space when things got rough, or maybe it was just by letting me know you were there for me. My parents in particular believed in me and supported me throughout the years, even when I might not have deserved it. This is but one way I'm repaying your efforts and sacrifices.

To Cecilia. If I were to describe all the adventures (others might call them challenges, but we're not the others) we faced together over the last three years, I'd have to write another dissertation – let alone if I were to recollect all the events we lived through over more than 10 years. You've lifted me when I needed to be lifted, you challenged me when I needed to be challenged, and you took care of me even when taking care of me meant not taking care of me. Even though it may have cost you, you let me stand on my own when I needed proof that I could stand unyielding on my own, and that is the closest anyone could ever be to me. As this chapter closes and another one opens, I look forward to keep adventuring into the world, discovering new things and places, overcoming new trials, and conquering new heights – together, always.

“[...] truth is that as a man's real power grows and his knowledge widens, ever the way he can follow grows narrower: until at last he chooses nothing, but does only and wholly what he *must* do...”

— Ursula K. Le Guin, *A Wizard of Earthsea*

“Where passion dominates, that does not signify the presence of greater desire and ambition, but rather the misdirection of these qualities toward an isolated and false goal [...]. Those who direct the maximum force of their desires toward the centre, toward true being, toward perfection, seem quieter than the passionate souls because the flame of their fervour cannot always be seen. In argument, for example, they will not shout or wave their arms. But, I assure you, they are nevertheless, burning with subdued fires.”

— Herman Hesse, *The Glass Bead Game*

## **Abstract**

Eating disorders (ED) represent a complex category of psychiatric conditions, characterized by dysfunctional thoughts and behaviours about food and excessive preoccupation with body weight or shape, heterogeneous presentations, diagnostic migration and comorbidity. Such complexity underlined the limits on conventional diagnostic classification systems that do not include trajectories of symptoms and possible comorbidities. An alternative conceptualization of disease is the clinimetric staging model, which aims to determine the current stage of a psychiatric disorder, ranging from prodromal to chronic, by focusing on the characteristics of a condition at a particular point in time. The staging model helps differentiate between the progression of clinical manifestations. Recently network analysis (NA) has been widely applied to explore ED at the symptom level, in an attempt to tackle its intricacies. NA allows to model psychiatric conditions as a constellation of interacting symptoms and appears particularly relevant to explore the complexity of ED. The following explored the characteristics of ED at different stages, modelling symptom networks and comparing their structures. The results highlight the central role of positive functioning and transdiagnostic dimensions in all but the clinical population, where ED symptoms were more prominent. These results support the idea that ED is a complex category of conditions in which clinically meaningful manifestations reach beyond the symptomatology strictly related to eating behaviours and cognitions. While it was past the scope of this work to establish a full staging model for ED, the results obtained provide relevant information regarding clinical manifestations of ED at different levels of ED symptom intensity, thus supporting not only the development of a more refined staging model for these conditions but also providing potential clinical targets for stage-specific interventions.



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## Chapter 1. Feeding and Eating Disorders

Feeding and Eating Disorders, alternatively called simply eating disorders (ED), represent a complex category of psychiatric conditions, characterized by dysfunctional thoughts and behaviours about food, accompanied by excessive preoccupation with body weight or shape (American Psychiatric Association, 2013). All ED cause significant impairment in psychosocial functioning and can impact significantly on physical health (American Psychiatric Association, 2013). The clinical identification of ED is a complex process compounded by a heterogeneous presentation (Smith et al., 2019; Wildes & Marcus, 2013), diagnostic migration and comorbidity (Castellini et al., 2011), and high rates of residual diagnoses and symptoms (Tomba et al., 2019).

### 1.1. Principal Feeding and Eating Disorder Diagnoses

Following the classification proposed in the 5<sup>th</sup> edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), four major ED diagnoses can be identified: Anorexia Nervosa (AN), Bulimia Nervosa (BN), Binge Eating Disorder (BED) and Otherwise Specified Feeding and Eating Disorder (OSFED).

#### *Anorexia Nervosa*

Anorexia nervosa is a severe psychiatric disorder that is characterized by starvation and malnutrition, a high incidence of coexisting psychiatric conditions and treatment resistance (American Psychiatric Association, 2013; Mitchell & Peterson, 2020). For AN to be diagnosed, the individual must enact behaviours directed at restricting energy intake, resulting in significantly low body weight compared to what would be expected considering sex, age, and physical health. The individual must also experience intense fear of gaining weight or of becoming fat, or persistent

behaviours that interfere with weight gain, even if already significantly underweight. Finally, the individual must experience disturbance in the way in which they perceive their body's shape or weight, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight. Anorexia Nervosa presents two subtypes based on the behavioural patterns of the individual: the *restricting* subtype requires the person to accomplish weight loss mainly through dieting, fasting or excessive exercise, with a lack of being-eating or purging behaviours for at least three consecutive months (American Psychiatric Association, 2013). The *binge-eating and purging* subtype, on the contrary, requires the individual to engage in binge-eating behaviours, followed by purging behaviours such as self-induced vomiting or excessive use of laxatives or diuretics (American Psychiatric Association, 2013). The severity of the AN presentation is based on the BMI of the individual (from mild, presenting a BMI  $> 17$  kg/m<sup>2</sup>, to extreme, presenting a BMI  $< 15$  kg/m<sup>2</sup>) (American Psychiatric Association, 2013).

### *Bulimia Nervosa*

Bulimia nervosa manifests through excessive concerns regarding body weight and shape, which is attributed to an undue influence over self-evaluation. From a behavioural standpoint, individuals suffering from BN experience recurrent episodes of binge eating, characterised by a sense of loss of control over the amount of food intake, followed by inappropriate compensatory behaviours in an attempt to mitigate the consequences of bingeing (American Psychiatric Association, 2013; Harrington et al., 2015). To establish a diagnosis of BN, binge episodes and inappropriate compensatory behaviours must occur at least once a week for at least three months. Despite the necessary presence of compensatory behaviours to diagnose BN, a person's weight is not factored in the diagnostic process; this reflects the high caloric intake that follows bingeing behaviours even when followed by purging practices (Kaye et al., 1993). In BN, the severity of the presentation is calculated based on the compensatory conducts per week, ranging from mild (an average of 1-3

episodes of purging episodes per week) to extreme (an average of 14 or more purging episodes per week) (American Psychiatric Association, 2013).

### *Binge Eating Disorder*

Binge eating disorder is characterized by regular binge eating episodes, during which individuals ingest comparably large amounts of food and experience loss of control over their eating behaviour (American Psychiatric Association, 2013; Giel et al., 2022), similar to the episodes experienced by persons with BN. To establish a diagnosis of BED, however, the binge episodes must not be associated with recurrent use of compensatory behaviours, and the binge episodes must be associated with feelings of disgust towards oneself or embarrassment regarding the amount of food eaten, eating more rapidly than normal, eating until uncomfortably full or when not physically hungry (American Psychiatric Association, 2013). To establish a diagnosis of BED, binge must occur at least once a week for at least three months. The severity of the BED presentation is established based on the episodes of binge-eating per week, ranging from mild (1-3 episodes per week) to extreme (14 or more episodes per week) (American Psychiatric Association, 2013).

### *Other Specified Feeding and Eating Disorders*

Other Specified Feeding and Eating Disorders (OSFED) encompass all those presentations in which symptoms characteristic of an ED that cause clinically significant distress or impairment in important areas of functioning predominate but do not meet the full criteria for any of the disorders in the ED diagnostic category. This class of ED includes a wide range of different presentations, including Atypical AN (a presentation similar to AN but without satisfying the BMI criteria), low frequency/limited duration BN (bingeing episodes or compensatory behaviours present less than once a week or for less than three months respectively), low frequency/limited duration BED (bingeing

episodes happening less than once a week or for less than three months respectively), and Purging Disorder, consisting of recurrent purging behaviours carried out to influence one own's weight or shape without presence of binge eating episodes.

Eating disorders can have serious consequences for physical health (Fichter & Quadflieg, 2016; Himmerich et al., 2021), and are considered among the most lethal mental health conditions (Fichter & Quadflieg, 2016), either because of medical sequelae of the disorders, such as cardiovascular or metabolic complications (Fichter & Quadflieg, 2016), or risk behaviours, substance abuse or self-harm (Baker et al., 2017; Crow et al., 2009). From a medical point of view, the severe electrolyte imbalances that follow prolonged starvation, malnutrition or purging behaviours are among the most dangerous and potentially fatal consequences of ED (Himmerich et al., 2021; Voderholzer et al., 2020), hyponatremia and hypokalemia in particular (Voderholzer et al., 2020). Diminished bone density is another frequent medical complication: a study by Zipfel and colleagues (2001) reported over 54% of AN patients, but not BN patients, satisfying the criteria for osteopenia. From a neurological point of view, diffuse atrophy of the grey and white matter is found, with consequences on cognitive functioning (Vidal et al., 2021) and, consequently, available resources to engage with psychological treatment. Eating disorders in general are associated with a high risk of suicide, especially in BN patients where suicidality represents the stronger predictor of a shorter time to death (Fichter & Quadflieg, 2016).

## 1.2. Epidemiological outline of Feeding and Eating Disorders

Feeding and Eating Disorders are the 12<sup>th</sup> leading cause globally for the reduced expectation of the length of healthy living measured disability-adjusted life year (DALY) among adolescent females in high-income countries (Erskine et al., 2016). Furthermore, the burden of ED on the

population has probably increased following the COVID-19 pandemic (Cappelletto et al., 2023; McLean et al., 2022), especially in those age groups more susceptible to ED onset (Schreyer et al., 2023).

An exact estimation of the prevalence of ED is not an easy task: epidemiological data are usually obtained from medical records or psychiatric admissions, thus probably underestimating the real prevalence due to delayed or absent help-seeking behaviours in ED populations (van Eeden et al., 2021). There is no clear consensus on the average age of onset of ED (Favaro et al., 2019), though the literature on this topic identifies the teen years as the peak of risk of onset (Favaro et al., 2019; Solmi et al., 2022). While a trend towards earlier onset of ED has been identified, it remains unclear whether this reflects a real shift in the age of onset or is a consequence of the changes implemented in the DSM-5 diagnostic criteria (Favaro et al., 2019).

The prevalence and distribution of ED seem to vary depending on gender and cultural background, and appear to affect predominantly Caucasian populations from high-income countries (Kolar et al., 2016). This is especially true for AN compared to the other ED diagnostic categories (Goode et al., 2020; Perez et al., 2016; van Hoeken et al., 2016). Among European women, the incidence of AN ranges from 1% to 4%, the incidence of BN ranges from 1% to 2%, the incidence of BED ranges from 1% to 4% and the incidence of subthreshold ED ranges from 2% to 3%, with considerable variations by area, age group and ethnic origin (Keski-Rahkonen & Mustelin, 2016). These data however might not provide a full and precise picture of ED incidence. Indeed, several studies reported critical issues in the identification and treatment of ED in minorities and among lower-income communities (Calzo et al., 2017; Chowbey et al., 2012; Hahn et al., 2023; Nagata et al., 2020)

Specifically for the Italian context, studies mostly focus on the population at the regional level, thus a comprehensive overview of epidemiological features of ED in the Italian population is challenging (Faravelli et al., 2006; Favaro et al., 2003). Data from the Study on the Epidemiology of

Mental Disorders (ESEMED) study (Alonso et al., 2004), however, provides an estimate of the lifetime prevalence of ED in the Italian adult population of 3.3%.

### 1.3. Transdiagnostic model of Feeding and Eating Disorders

The DSM-5 classification of ED operates a clear-cut distinction between the various diagnostic categories, but clinical reality belies such an approach which does not reflect the complexity and variety of clinical reality. Most ED patients ultimately receive a diagnosis of OSFED, which nonetheless remains the least studied ED diagnosis (Levinson et al., 2022), rather than another more specific diagnosis, and diagnostic migration in this population is extremely frequent over time (Cooper & Grave, 2017; Forbush et al., 2017; Galmiche et al., 2019). Diagnostic migration and lifetime prevalence of ED diagnoses different from the one currently affecting a patient are, in particular, phenomena which disproportionately affect ED patients more than other clinical populations (Agras et al., 2000). Indeed, roughly half the patients who initially receive a diagnosis of AN, at some point will fulfil the diagnostic criteria for BN; similarly, a significant number of individuals who received a diagnosis of OSFED did previously fulfil the diagnostic criteria for AN or BN. The consistency and specificity of this phenomenon typical of ED populations made researchers and clinicians question the validity of the categorical distinction adopted in the various editions of the DSM (Smink et al., 2013). A possible explanation for such dynamic typical of ED is provided by the transdiagnostic cognitive-behavioural model of ED (Fairburn et al., 2003), currently the most widely adopted theoretical model that explains the psychological and behavioural mechanisms underlying the onset and maintenance of ED (Figure 1).

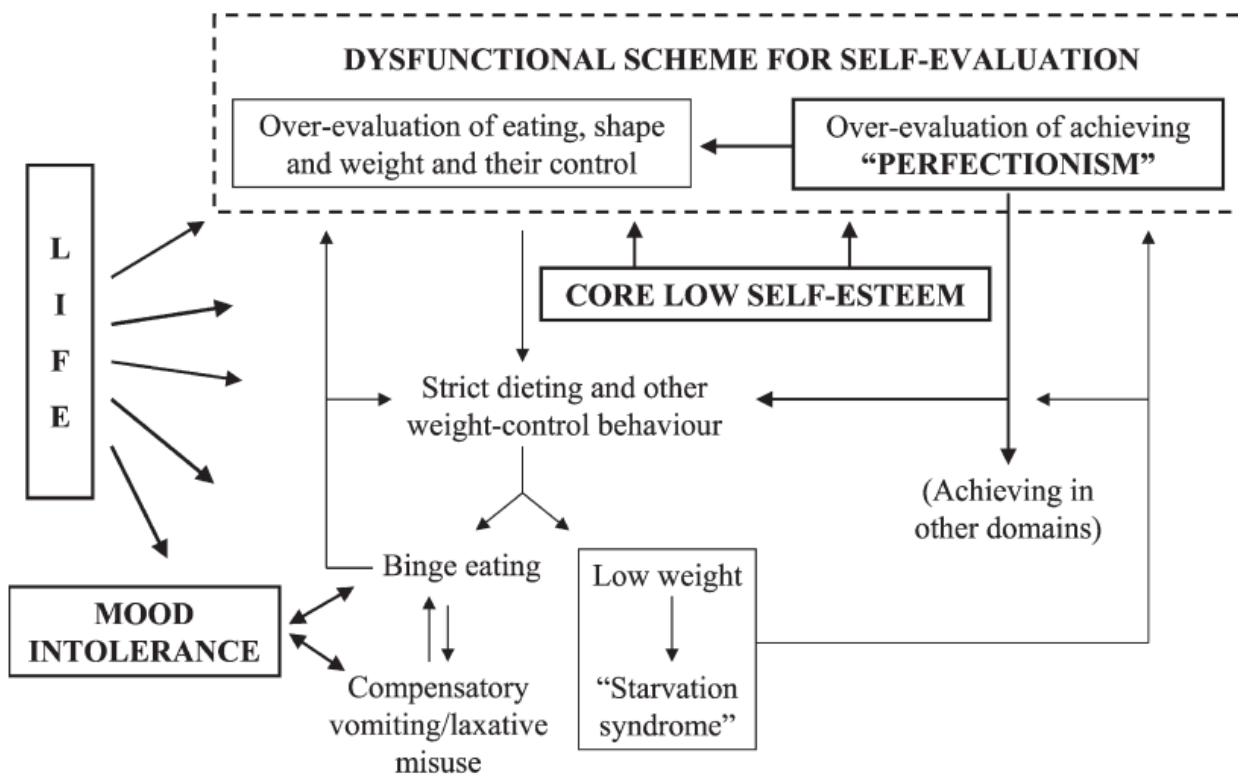


Figure 1: Transdiagnostic Cognitive-Behavioural Model of Eating Disorders (Fairburn et al., 2003).

The transdiagnostic cognitive behavioural model of ED postulates the existence of a shared psychopathological core underlying all ED, which has to do with the tendency of patients to base their sense of worth and value as an individual solely on their body weight and shape, and their ability to control them (Fairburn et al., 2003; Murphy et al., 2010). The control of their bodily weight and body shape becomes the main source of gratification for the individual, to the point where all other areas of functioning become progressively less relevant and the efforts to avoid weight gain become more and more central in the individual's life (Fairburn et al., 2003). According to the transdiagnostic model, all symptoms typical of ED, such as dietary restrictions, purging behaviours, excessive physical exercise and worries about body weight and shape are therefore the expression and consequence of the individual's overevaluation of weight and shape. Within the model, binge eating episodes are postulated to be the result of severe self-imposed dietary restrictions, so rigid as to inevitably lead to a physiological need to feed. However, the slightest disinhibition towards food, even when physiological in nature, is interpreted by the individual as proof of being a failure for not

being able to adhere to the overly rigid self-imposed dietary rules. These feelings of self-worthlessness and defeat then typically trigger the actual binge eating episode. These negative emotions and thoughts linked to failed dietary restrictions contribute to reinforce the idea that one's self-worth is determined by their ability to control eating, weight and body shape; the response to the failed adherence to the dietary rules is then usually the adoption of even stricter dietary rules, which consequently determined an even higher risk of binge eating behaviours (Fairburn et al., 2003). At this point, the vicious circle of maintaining the ED is established. Specifically, the cognitive-behavioural transdiagnostic model of ED identifies four main elements outside of specific ED symptoms which are involved in the maintenance of the eating symptomatology. The first element is represented by clinical perfectionism, a risk factor for ED which can persist even years after a person has achieved recovery (Bardone-Cone et al., 2007; Lilenfeld et al., 2000). The definition of clinically relevant perfectionism itself is similar to the description of the constructs at the core of the transdiagnostic model: it is defined as attributing undue relevance to very high self-imposed performance standards, no matter what sacrifices are necessary to achieve them (Shafran et al., 2002). Such rigid beliefs result in excessive self-criticism and the consolidation of a negative view of oneself when confronted with failures. Thus, perfectionism feeds into ED psychopathology through increasingly strict dietary rules to achieve weight loss; failure to adhere to these rules, as discussed above, negatively influences self-esteem and precipitates symptomatology (Fairburn et al., 2003).

The attitude of heavy self-criticism following failure is also an indication of low basic self-esteem, which constitutes the second of the four maintenance mechanisms postulated by Fairburn and colleagues (2003). In this regard, the authors proposed the definition of *core low self-esteem* to highlight how the negative view of self in DNA patients is much more pervasive than a simple self-reprimanding in the face of failure and also has to do with an unconditionally negative judgment of self, so deep that it merges with their own identity. The depth of this feeling is what transforms low self-esteem into a dangerous obstacle to treatment: not only because it generates an inexorable loss



of hope for one's ability to change, but above all because it involves a very strong attachment to all those behaviours that, although dysfunctional, are perceived by them as the only way to be able to give themselves a value.

Binge eating is a cross-functional problem, very often triggered by severe dietary restrictions, as already described. However, in at least 50% of cases, patients report that the *trigger* is more strictly emotional, and often represented by feelings of loneliness, anxiety, anger, boredom, and even depressive states (Waller, 2002). In light of this evidence, it has been suggested that a further mechanism of maintenance of DNA psychopathology can be traced in emotional *intolerance (mood intolerance)*, extreme difficulty in accepting negative (but also positive) emotional states that results in the use of dysfunctional emotional regulation strategies aimed at losing awareness of the emotion itself and, therefore, at neutralizing the tension that accompanies it (Fairburn et al., 2003). In people suffering from ED, binge eating, self-induced vomiting, and exhausting exercise are all behaviours that can serve this purpose, but often substance abuse and self-harm are also part of this dynamic, which are quite prevalent behaviours in eating and ED (Holderness et al., 1994; Paul et al., 2002).

Finally, it is important to recognize that the emotional states that are triggered by the binge eating episode often originate in an interpersonal context (Fairburn et al., 2003). In the specific case of bulimia, the presence of interpersonal hypersensitivity has often been recognized, which, associated with an unstable self-image and low self-esteem, makes patients extremely sensitive to criticism, rejection and disapproval of others (Steiger et al., 1999). Interpersonal difficulties have also been recognized in the course of anorexia nervosa, in which the control that the patient establishes over food seems to take on a particular significance when dysfunctional tensions and relational dynamics are present within the family (Fairburn et al., 1999). It has therefore been proposed by Fairburn et al. (2003) that the different interpersonal processes and the difficulties potentially related to them can contribute significantly to the maintenance of ED, thus representing the fourth of the maintenance mechanisms identified by the authors.

While the application of this model to explain BN and BED is clear, this model can also explain the dynamics underlying both subtypes of AN. Patients with the *purging* AN subtype engage in compensatory behaviours like BN patients and, therefore intuitively fit into the transdiagnostic model. The core components of the model, the over-evaluation of eating, weight, and shape control, however, can also explain the mechanism of *restricting the* AN subtype. If self-evaluation is strictly linked to an individual's ability to adhere to self-imposed dietary rules, then actually managing to stick to those rules will give the individual positive reinforcement, which will then motivate them to maintain the dysfunctional nutritional behaviours (Coniglio et al., 2017). Walsh (2013) hypothesized that persistent restriction is the result of a well-ingrained, maladaptive habit that has been reinforced during illness by highly salient rewards such as a perceived sense of control and accomplishment, external praise, and successful weight loss.

#### 1.4. The enhanced cognitive-behavioural treatment for Feeding and Eating Disorders (CBT-E)

In the early 2000s, Fairburn and colleagues proposed an "enhanced" version of cognitive-behavioural theory and treatment, specifically tailored for treating ED (CBT-E) (Fairburn et al., 2003a). This update of the previous CBT treatment model for ED mostly focused on BN (CBT-BN), introduced two major changes. First, it promoted a transdiagnostic perspective, positing that all ED share core cognitive components, such as shape and weight over-evaluation. This formulation led to the hypothesis that targeting these mechanisms could result in improvements over a broad spectrum of ED (Fairburn et al., 2003a). Secondly, CBT-E expanded its scope to include the maintaining mechanisms identified in the transdiagnostic theoretical model — clinical perfectionism, core low self-esteem, mood intolerance, and interpersonal difficulties. These mechanisms are purported to exacerbate the core features of ED, particularly in individuals who did not respond to CBT-BN. Specifically for this latter group of patients, Fairburn and colleagues developed a more comprehensive form of CBT-E (CBT-Eb), incorporating additional modules to intervene in the

additional maintaining processes, as relevant to the patient's specific conceptualization. For the rest, a focused version of CBT-E (CBT-Ef) was developed, which targets core ED psychopathology and mood intolerance.

The superiority of CBT-E in reducing ED symptomatology has been consistently demonstrated in multiple randomized controlled trials, in which CBT-E has shown both short and long-term superiority over waiting list control groups and active psychological treatment comparisons, such as Interpersonal Psychotherapy (IPT) (Fairburn et al., 2015), psychoanalysis (Poulsen et al., 2014), or classic CBT (de Jong et al., 2020). However, despite the demonstrated effectiveness of CBT-E in ED treatment, at most only half of the patients achieve a lasting recovery (Calugi et al., 2015; Dalle Grave et al., 2019); these outcome rates, while on par or above other psychotherapeutic interventions, call for a careful consideration of other potential areas of clinical interest in ED. Indeed, Monteleone and Cascino (2021) reported robust evidence on the role of non-specific cognitive factors that might influence the course and treatment response of ED, underlining the importance of including such aspects in future studies.

### 1.5. Non-specific cognitive factors in Feeding and Eating Disorders

As already mentioned, the transdiagnostic conceptualization of ED identifies a cognitive element at the core of this category of disorders. Maladaptive and distorted cognitions regarding eating, weight and body shape are hypothesized to influence disordered eating behaviours (Legenbauer et al., 2018). The widespread adoption of the transdiagnostic model, however, led researchers to focus on ED-specific cognitive aspects, and despite the well-established relevance of cognitive structures in the onset and maintenance of maladaptive eating behaviours (Jackman et al., 1995; Markus et al., 1987; O'Connor & Dowrick, 1987; Rosen et al., 1989), relatively few studies

investigated cognitively oriented conceptualizations beyond strictly ED-related themes in ED patients (Del Pozo et al., 2018; Legenbauer et al., 2018; Waller et al., 2003).

Two transdiagnostic cognitive constructs in particular appear especially relevant for improving our understanding of the dynamics underlying the onset and maintenance of ED: Ellis' Rational and Irrational Beliefs (Ellis, 1994) and psychological flexibility (Kashdan & Rottenberg, 2010).

#### 1.5.1. Rational and irrational beliefs

According to Ellis (1962), irrational beliefs (IBs) are rigid, absolutistic, false cognitive evaluations that cause emotional distress to the individual. DiGiuseppe and Doyle (2019) describe IBs as: inconsistent, unsubstantiated by empirical evidence, absolutistic, and hindering the pursuit of personal goals. In line with these formulations, IBs can be assimilated into the maladaptive cognitions later incorporated into cognitive behavioural therapy (Ellis & Dryden, 2007) and, consequently, in Fairburn's transdiagnostic model (2003). In the final formulation of Ellis' model, four core IBs are described and have been clinically and empirically identified: awfulizing thoughts, irrational demandingness, low frustration tolerance beliefs, and negative global self-evaluations/self-downing. Awfulizing thoughts refer to a cognitive distortion where individuals perceive a situation or outcome as absolutely terrible, unbearable, or catastrophic. It involves magnifying the negative aspects of an event and viewing it as far worse than it is (Ellis, 1994). Irrational demandingness refers to the tendency of individuals to hold rigid and unrealistic demands or expectations about themselves, others, and the world. These demands are irrational because they are extreme, absolutist, and unrealistic, often leading to emotional distress when not met (Ellis, 1994). Low frustration tolerance beliefs are irrational beliefs related to one's inability to tolerate frustration, discomfort, or difficult situations, including the belief one should always be treated fairly and any unfair treatment is

unbearable, the belief that life must be easy, without discomfort or inconvenience, and the belief that one must be thoroughly competent, intelligent, and achieving at all times to be happy (Ellis, 1994). Finally, Negative global self-evaluations/self-downing is the tendency to express excessively critical and generalized evaluations (e.g., “I am stupid and worthless because I did not pass the exam”) of oneself, others, and the world (Ellis, 1994).

In contrast, rational beliefs (RBs) are the pragmatic, logical, flexible, counterparts of IBs and lead to better overall functioning in the individual. The RB equivalent of awfulizing is realistic negative expectations, a more moderate approach to evaluating the badness of a given event, and a style more prone to assess negative episodes as “bad” rather than “awful”. The RB counterpart of irrational demandingness is a cognitive style more oriented towards non-demanding preferences, in which the individual expresses wishes or desires, rather than more dogmatic demands from themselves or others. The RB analogue of low frustration tolerance is frustration tolerance, expressed through balanced and flexible statements about one’s ability to tolerate undesirable outcomes or negative events. The fourth and last RB is self-acceptance, which represents the counterpart of global negative self-evaluations, and portrays a style oriented towards acknowledging and accepting the fallibility of oneself and recognising that the self is too complex to be globally rated.

The presence and relevance of IBs and conceptually close constructs such as perfectionism (Ellis, 2002) and low self-esteem (McLennan, 1987) in populations with ED have been documented in several studies (Halmi et al., 2000; McLaren et al., 2001; Tecuta, Gardini, Diguseppe, et al., 2021; Tecuta, Gardini, Schumann, et al., 2021; Tecuta et al., 2023). In a study by Tecuta and colleagues (Tecuta, Gardini, Schumann, et al., 2021) a clinical sample of outpatients with ED reported higher scores on all IBs measures when compared to healthy controls from the general population. More specifically, awfulizing combined with higher BMI appeared to be the most prominent predictor of ED-specific risk. This result mirrors the objective of the main therapeutic techniques of cognitive restructuring in ED, which aim to reduce the overly catastrophic cognitions related to weight gain

and body shape (Fairburn et al., 2003; Nezu & Nezu, 2016). The same study by Tecuta and colleagues also identified Negative global self-evaluations/self-downing, which encompasses aspects related to perfectionism, self-esteem, ineffectiveness and interpersonal difficulties, to be the most significant predictor of general psychological maladjustment in the study's ED population. Feelings of inefficacy in particular, for example, related to the inability to adhere to strict dietary rules, have been hypothesized to play a central role in clinical models of ED and have been empirically supported in network analysis models (Monteleone & Cascino, 2021). Low self-esteem and self-efficacy have been frequently linked to ED symptoms reported in ED populations (Krauss et al., 2023), even when confounding factors, such as depressive symptomatology, were accounted for (Tecuta & Tomba, 2018). Furthermore, the same study by Tecuta and colleagues (Tecuta, Gardini, Schumann, et al., 2021) found Negative global self-evaluations/self-downing to play a significant role in explaining the variance of cognitive reappraisal, so that greater endorsement of negative self-beliefs was associated with lower ability to reevaluate events appropriately. Negative self-beliefs would be associated with impaired cognitive reappraisal, as they represent a form of rigid and partial thinking combined with hypersensitivity to emotionally negative salient information.

Considering the information presented, IBs emerge as a promising research line that could improve our understanding of ED (Atwood & Friedman, 2020; Tecuta, Gardini, Schumann, et al., 2021; Tecuta et al., 2023) and shed light on the dynamics underpinning the relationship between general psychopathology and ED symptomatology (Monteleone & Cascino, 2021).

### 1.5.2. Psychological flexibility

Another transdiagnostic cognitive factor implicated in ED is psychological flexibility (Hill et al., 2013). Psychological flexibility is defined as being in contact with the present moment, fully aware of emotions, sensations, and thoughts, welcoming them, including the undesired ones, and

maintaining or changing one's behaviour so that it is aligned with personal values (Hayes et al., 2006). The negative counterpart of psychological flexibility and strongly associated with psychopathological outcomes (Arch et al., 2012; Zettle et al., 2011) is psychological inflexibility, defined as a rigid style of thinking that involves dwelling on one's thoughts and rigid attempts to control, alter or minimize unpleasant internal experiences, which results in an inability to change and/or persist in value-guided behaviours (Hayes et al., 2006). Psychological flexibility is composed of six separate adaptive cognitive processes, each of which has a maladaptive version that feeds into psychological inflexibility (Hayes, Pistorello, et al., 2012). The six components of psychological flexibility/inflexibility are acceptance/experiential avoidance, cognitive defusion/fusion, being present in the moment/attentional rigidity to the past and future, noticing self/conceptualized self, values/unclear motives, and committed action/avoidant persistence.

Hayes and colleagues (2012) define acceptance as an active and aware embrace of thoughts. Its counterpart, experiential avoidance, on the other hand, represents the efforts to alter the frequency or form of unwanted thoughts and emotions even when this might cause harm.

Cognitive defusion can be described as the process of distancing oneself from cognitive events, for example acknowledging that thoughts are just thoughts, or memories are just memories, with no factual meaning or value beyond their existence (Hayes, Pistorello, et al., 2012; Ruiz et al., 2021). Its counterpart, cognitive fusion, indicates the tendency to respond behaviourally and cognitively to internal stimuli without considering external variables that might contribute to regulating the response.

Being present in the moment is defined by Hayes and colleagues as the process of focusing one's attention on the present moment when events and life, in general, are unravelling; its opposite is an attentional rigidity focused on the past and/or future (Hayes, Pistorello, et al., 2012), which has been observed to exacerbate negative experiences such as pain (Schütze et al., 2010) or rumination (Davis & Nolen-Hoeksema, 2000).

The process of noticing the self is the process in which, through mindful awareness, a person can

develop a sense of self that is separate from his or her unhelpful internal experiences (Godbee & Kangas, 2020; Hayes, Strosahl, et al., 2012); its opposite, the conceptualized self, represent a rigid overidentification with a self-narrative which reduces behavioural flexibility by rejecting external events or contents that challenge such narrative.

The values component refers to the aspects of life that an individual finds most meaningful or important. These chosen, verbally constructed, consequences of patterns of activity, for which the predominant reinforcer becomes intrinsic to the behavioural pattern itself (Hayes, Pistorello, et al., 2012). Their counterpart is behaviours which are not driven by an individual's values but stem from, for example, guilt or compliance.

Finally, committed action represents the behavioural component and the underlying cognitive processes aimed at producing larger and larger patterns of effective action linked to chosen values even in the presence of obstacles, whereas its opposite represents self-defeating patterns of behaviours characterised by lack of action, or behaviours that are reactive, impulsive, or automatic (Hayes, Pistorello, et al., 2012).

Kashdan and Rottenberg (2010) argue that psychological flexibility is a critical element of mental and emotional well-being. A growing body of evidence has shown that reduced psychological flexibility is associated with a wide range of behavioural problems (Hayes et al., 2006), including disordered eating (Masuda et al., 2010; Rawal et al., 2010) and clinical perfectionism (Miles et al., 2023), which is in itself associated with ED symptomatology. A study by Merwin and colleagues (2011) reported a correlation between AN and psychological inflexibility linked to worry about controlling both their thoughts and their bodies. In addition, psychological inflexibility was found to be related to disordered eating (Masuda et al., 2010; Morton et al., 2020), either directly or through the effect of low tolerance of negative thoughts and feelings (Masuda et al., 2011). Psychological flexibility has also been suggested as one of the possible main mechanisms of action of mindfulness-based treatment for ED, possibly by facilitating following treatment recommendations, such as



adhering to a meal plan despite negative thoughts and emotions (Vanzhula & Levinson, 2020). A specific type of psychological inflexibility regarding body image has also been conceptualized specifically for ED (Sandoz et al., 2013). More in detail, body image inflexibility represents the use of maladaptive regulation strategies while experiencing body dissatisfaction and disordered eating (Sandoz et al., 2013), and has been shown to partially mediate disordered eating cognitions and pathology (Wendell et al., 2012). These findings indicate that reduced psychological flexibility may be a key mechanism in the development of unhealthy eating patterns. On the other hand, body image flexibility may also be a protective factor that weakens the association between disordered eating cognitions and disordered eating behaviour (Hill et al., 2013).

#### 1.6. Positive psychological functioning

Decades of research produced a rich body of literature focused on the dysfunctional behaviours and cognitions characteristic of ED. Less attention has been paid, on the other hand, to those aspects that might mitigate symptoms, prevent onset, or improve the quality of life of ED patients despite their symptomatology, such as positive psychological functioning. Such dimension only relatively recently started receiving attention from researchers (de Vos et al., 2017; Tomba et al., 2014), despite, in the patient's own words, it being a critical factor in achieving a satisfactory and lasting recovery from ED (Tomba et al., 2019). Traditionally, the concept of well-being in psychology has been associated with the absence of symptoms of a disorder, without actually considering an individual's positive functioning. (Ryff & Singer, 1996). Jahoda first (Jahoda, 1958), followed by Ryff (1989), extended the conceptualization of well-being to include the importance of assessing the presence of specific dimensions, which allow the development of optimal functioning of an individual (Ryan & Deci, 2001). This formulation would become known as "psychological well-being" (Ryff, 1989). Ryff's psychological well-being model encompasses six interconnected psychological dimensions: autonomy, environmental mastery, positive relationships with others, self-acceptance,

personal growth, and purpose in life. Ryff's model stresses the importance of placing psychological well-being on a separate continuum from psychopathology, that is to say, the two are not mutually exclusive but can coexist more or less independently from each other. (Ryff & Singer, 1996). This conceptualisation has also found support in biomarker studies, which have shown the independence of the two phenomena of well-being and malaise (Ryff et al., 2006). The presence of symptoms, or even full-blown disorders, is, according to Ryff and Singer, the result of the simultaneous and independent effects of well-being and ill-being (Ryff et al., 2006). This hypothesis found support in several studies; for example, Rafanelli and colleagues (2000) found that despite remission from affective disorders, the patient group showed lower levels of psychological well-being compared to healthy controls. Similarly, Tomba and colleagues (2019) observed how remitted and recovered ED patients still exhibited impaired well-being compared to healthy controls. Lower psychological well-being combined with the persistence of residual symptoms also determined an increased risk of relapse in clinical populations (Fava et al., 1998; Fava & Tomba, 2009). Tomba and colleagues (2014) observed how altered levels of psychological well-being were independent of the presence of psychopathology, supporting the idea that the presence of well-being might not be on the same continuum as psychological distress. Despite the potential relevance, especially for the conceptualization of remission and recovery from ED (Richmond et al., 2020; Tomba et al., 2019), relatively few studies so far examined both positive and negative psychological functioning in individuals with ED (de Vos et al., 2017; de Vos et al., 2021, 2023; Tecuta et al., 2020; Tomba et al., 2014; Tomba et al., 2017).

A study by Tomba and colleagues (Tomba et al., 2014) investigated psychological well-being in an outpatient population with ED, observing how autonomy, environmental mastery, positive relationships with others, and self-acceptance were consistently lower in the clinical population compared to healthy controls. Interestingly, ED patients with BN showed greater impairment in all dimensions of psychological well-being than healthy controls, in particular, lower levels of

environmental mastery, lower optimism, and lower self-esteem, whereas patients with AN were found to be more similar to their healthy counterparts (Bulik et al., 2001; Mond et al., 2005). These differences could stem from the egosyntonic nature of AN, especially in outpatients and individuals in the early stages of the disorder, who often lack insight into their condition. The combination of these two elements might induce the individual to perceive higher levels of psychological well-being despite their compromised condition (Gregertsen et al., 2017; Gunnard et al., 2012). A sense of self-worth, self-efficacy, self-mastery, and accomplishment has also been found in patients with AN concerning their ability to control weight (Nordbø et al., 2006). It has also been suggested that certain sociocultural values such as the desirability of thinness and positive social feedback to being thin may be associated with higher levels of self-reported psychological well-being in women with anorexia nervosa (Gunnard et al., 2012; Mond et al., 2005). This may be particularly relevant in outpatients with AN with a non-life-threatening BMI, who may positively view their thinness as associated with success and popularity (Nordbø et al., 2006). The relevance of well-being dimensions at various stages of ED (de Vos et al., 2017; Tomba et al., 2019) also emerges in the accounts of recovered ED patients, which identify in several well-being dimensions some of the crucial elements in achieving recovery (de Vos et al., 2017).

### 1.7. Risk factors

One of the core assumptions of the leading ED theoretical model is the disorder is maintained by the interaction of some cognitive factors (Fairburn et al., 2003); such a number has been recently postulated as possibly being larger than what previously thought (Monteleone & Cascino, 2021). However, as originally maintained by Fairburn (Fairburn et al., 1993), and amply elaborated by Stice (2002), ED maintenance factors might not necessarily be the same factors involved in the onset of the disorder. Considering the potential differences in causes of onset and maintenance of an ED, combined with the fact that roughly only half of ED patients show long-term improvement following

treatment, it is fundamental to improve our understanding of the leading causes of ED to prevent the disorders from developing in the first place.

Risk factors can be defined as measurable entities that increase the likelihood of symptom onset in a specific population (Kraemer et al., 1997). In ED, risk factors are often common to multiple diagnoses, reflecting the transdiagnostic nature of this group of disorders. As such, risk factors are not as useful to differentiate between individual ED as to differentiate between ED and other psychiatric conditions (Hilbert et al., 2014).

The presence of aspects connected to the transdiagnostic model's cognitive core of ED, overevaluation and concerns regarding shape and weight, are considered typical indicators of risk of ED. Indeed, the self-judgment and self-esteem of people with an ED are more strongly influenced by body shape rather than performance in other areas of functioning (such as work, relationships, and family) (American Psychiatric Association, 2013; Murphy et al., 2010). The centrality of weight and eating control over self-evaluation and self-esteem is supported by its subjective importance according to ED patients, though the ability to maintain such control may vary depending on the specific ED (for example maintaining restriction in AN versus binge episodes in BN). Attempts at controlling nutrition through dieting are also retrospectively correlated with ED onset regardless of individual diagnostic categories (Hilbert et al., 2014). For example, a study by Patton and colleagues (1999) found that participants adhering to strict and rigid dietary rules were 18 times more likely to develop an ED in the following 6 months, and one in five had an increased likelihood of ED onset within 12 months (Patton et al., 1999).

Over the years, research studies identified many potential psychological and cognitive aspects that might increase the odds of ED onset. Comorbidity with psychiatric symptoms, anxiety and depressive symptomatology in particular, is an extremely common phenomenon in ED populations (Treasure et al., 2020), and their presence is a well-established risk factor for ED. (Bulik et al., 1997; Fragkos & Frangos, 2013; Rojo et al., 2006; Stice et al., 2017). Similarly, impaired psychosocial

functioning has long been identified as a risk factor for the onset and exacerbation of ED symptomatology (Allen et al., 2014; The McKnight Investigators, 2003). More recently, researchers turned their attention to investigating the role of other transdiagnostic dimensions, such as positive functioning, with particular regard to social interactions, and more cognitively oriented aspects such as irrational beliefs (IBs) (Tecuta, Gardini, Schumann, et al., 2021) and psychological inflexibility (Masuda & Latzman, 2012; Miles et al., 2022).

Psychiatric conditions, mood disorders and anxiety disorders in particular represent possibly the most studied risk factor, with extremely high rates of comorbidity in populations with full-blown ED (Treasure et al., 2020), and associated with worse treatment outcomes and poorer long-term prognosis (Herpertz-Dahlmann, 2015). Both groups of disorders have been observed to be associated with increased disordered eating behaviours in non-clinical samples, prompting the researchers to recommend screening for ED in populations with affective or anxiety symptomatology (Garcia et al., 2020). Notably, both depression and anxiety symptoms have been documented to act as mediators between chronic stress and the onset of ED, with this relationship losing significance in the absence of the mediating symptomatology (Rojo et al., 2006). Depression has been hypothesized to increase vulnerability to the development of ED by exacerbating feelings of body dissatisfaction (Stice et al., 2011). Such a notion is supported by studies that have demonstrated how negative affect (Stice et al., 2017; Zerwas et al., 2013) precede and/or predict eating pathology and the development of ED. In particular, Stice and colleagues (Stice et al., 2017) hypothesized that mood disorders might influence ED onset by promoting reward-seeking behaviours involving the assumption of highly caloric food, subsequently triggering feelings of guilt and compensatory behaviours, or alternatively by directly reducing appetite. Similarly, anxiety has been observed to precede the onset of ED (Bulik et al., 1997; Schaumberg et al., 2019), even more frequently than mood disorders (Garcia et al., 2020). One possible explanation for the role played by anxiety in increasing the likelihood of ED onset posits that unhealthy weight control behaviours may be enacted to reduce anxiety about excess body fat

(Stice et al., 2017). This theory is consistent with cognitive theoretical models as well as physiological data. The fear-conditioning model sees core features of dietary restriction and excessive exercise as exerting an anxiolytic function against the fear of weight gain that is out of the patient's cognitive control (Zerwas et al., 2013). Physiologically, brain studies of patients with ED have indicated that, due to disturbances in regular serotonin and dopamine reward pathways, this population might use restricted eating as a mechanism to reduce anxiety (Kaye et al., 2013). In one study of patients with AN and BN, mutations in genes with heightened expression in brain tissue were associated with a lower lifetime BMI and earlier ED onset (Gratacos et al., 2010).

Psychosocial factors are also implied to be involved in increased risk for ED, mainly through dysfunctional relationships with family members and peers (Keel & Forney, 2013; Tozzi et al., 2005). A longitudinal study on risk factors of ED found that social pressure, including media modelling, social eating, dieting, and weight teasing, significantly predicted the onset of ED in young women (The McKnight Investigators, 2003). Social withdrawal has been observed to predict the onset of any ED (Allen et al., 2014), and impaired social functioning and lower sensitivity to positive social interaction have also been suggested as an element involved in both the onset and maintenance of ED (Treasure et al., 2012). Shame and teasing about one's weight or eating also consistently emerged as a major risk factor for ED compared to other psychiatric conditions (Fairburn et al., 1998; Fairburn et al., 1997; Troop & Redshaw, 2012), possibly via the internalization by the individual of self-shame and self-hate, as the victim of shaming loathes themselves for inducing the others in enacting such behaviours (Duarte et al., 2015).

Finally, irrational beliefs, lack of psychological flexibility and impaired psychological well-being, have been recently observed to play a central role in the onset and maintenance of ED symptoms, and are thus postulated to be important therapeutic targets (Emelianchik-Key et al., 2023; Monteleone & Cascino, 2021; Monteleone et al., 2022). The relevance of these internal experiences in the onset and maintenance of ED supports the idea that psychopathology is not only linked to

external triggers and events, but also to how individuals respond to them (Masuda et al., 2011; Masuda & Latzman, 2012; Tecuta, Gardini, Schumann, et al., 2021; Tomba et al., 2014). These dysfunctional cognitive styles are characteristic of many psychopathological frameworks: it has been demonstrated, however, that the IB Awfulizing is especially common in individuals with an ED, and that the pervasiveness of this cognitive style in this population is comparable to that of individuals with a primary diagnosis of Generalized Anxiety Disorder (Sternheim et al., 2012; Tecuta, Gardini, Schumann, et al., 2021). In their study, Tecuta and colleagues (2021) reported IB awfulizing to significantly predict ED-specific risk, that is, the greater the tendency to awfulize, the greater the ED-specific symptomatology. A maladaptive cognitive style prone to catastrophizing negative events might feed into the anxiety and preoccupation related to control over eating, weight and shape, thus triggering anxious feelings that the individual controls through ED-related behaviours. Tecuta and colleagues (2021) also reported that IBs underlie non-specific psychopathological attitudes such as lack of self-esteem or perfectionism, themselves risk factors for the onset of ED. Psychological flexibility was also observed to be associated with an increased risk of ED as well as with depression, with participants at high risk of ED reporting lower psychological flexibility compared to the low- and medium-risk groups (Miles et al., 2022). Notably, in the same study, the authors observed that the group at high risk exhibited lower psychological flexibility than even the group with a lifetime diagnosis of ED. Furthermore, low psychological flexibility has also been observed to mediate between disordered eating behaviours and self-concealment (Masuda et al., 2011), as well as self-esteem (Koushiou et al., 2021). In patients with ED, it has been shown that low psychological flexibility is associated with a restrictive diet or preoccupation with nutrition (Masuda & Latzman, 2012). In terms of maladaptive cognitions regarding weight and shape, it is possible that individuals with low psychological flexibility may rigidly interpret weight gain and failed dieting attempts as failures, while successful dieting or compensatory behaviour are regarded as positive achievements regardless of how damaging or unreasonable they may be (Rawal et al., 2010).

Clinical perfectionism, a construct related to psychological flexibility (Miles et al., 2023; Miles et al., 2022; Ong et al., 2019), has also been implicated in the onset and maintenance of ED (Miles et al., 2023; Shafran et al., 2002; Stackpole et al., 2023; Vervaet et al., 2021). This construct mainly manifests itself through a marked fear of failure, excessive focus on one's own performance, and an undue devaluation of self when not performing at excessively high standards (Fairburn et al., 2003; Shafran et al., 2002). Perfectionism has been observed to be a risk factor in both itself and in combination with other psychological dimensions, often magnifying other constructs' own impact on the risk of ED onset (Boone et al., 2014; Byrne et al., 2016). It has been hypothesized that perfectionism might influence ED either by pushing individuals towards dysfunctional goals, for example promoting higher levels of drive for thinness (Boone et al., 2014), or by hypercritical attitudes towards lack of control over eating (Howard & Porzelius, 1999).

#### 1.8. Staging models of eating disorders

One of the main challenges of treating ED, and arguably most psychological conditions at large, is that usually individuals reach clinical services only when the disease has already developed acute symptomatology. Indeed, despite the body of knowledge available on which factors influence ED risk, early recognition and interventions for early-stage ED is still compounded by a lack of unitary models for early detection that are clinically meaningful (Hart et al., 2011; Koreshe et al., 2023; Tomba et al., 2023; Treasure et al., 2015). The complexity of the ED presentation and early detection through conventional taxonomy has led clinicians and researchers to question the exclusive reliance on such diagnostic classification systems, which appear more focused on identifying the disorders at later stages, when the symptomatology has become more severe and harder to treat (Koreshe et al., 2023). In psychiatry, conventional diagnostic systems have not traditionally considered illness trajectories, symptoms severity, or comorbid conditions, focusing instead on cross-sectional descriptions (Cosci & Fava, 2013; Schnyder, 2023). Traditional nosological approaches tend



to ignore certain aspects that characterize illness experience of an individual, and that are considered clinically meaningful by clinicians, with possible consequences for the identification of efficacious treatments (Fava & Kellner, 1993; Guidi et al., 2017). Such an approach contributed to the difficulties in defining the early stages of psychiatric disorders, specifically for ED contributed to the difficulties in defining outcomes (Tomba et al., 2019) and exacerbated the difficulties in categorization caused by the frequent diagnostic cross-over (Keski-Rahkonen & Mustelin, 2016). In response to the shortcomings affecting psychiatric taxonomy, in 1993 Fava and Kellner proposed an alternative conceptualization of illness that took the staging model of disease already established in other medical specialities and adapted it for use in psychiatry (Fava & Kellner, 1993). The staging model aims to determine the current discrete stage of a psychiatric disorder, ranging from prodromal to chronic or recovered stages. By focusing on the extent and timing of disease progression at a particular point in time, the staging model helps identify patients along a continuum of illness development, differentiate between early, milder or nonspecific clinical manifestations (Cross et al., 2014), as well as features of progression and chronicity (Guidi et al., 2017; Tomba et al., 2019), improving upon classification systems based on the acute model disease (Hetrick et al., 2008). Following Fava and Kellner's proposal of staging models of schizophrenia, unipolar and bipolar depression, and panic disorder (Fava & Kellner, 1993), staging models have been proposed for a wide range of psychiatric conditions such as schizophrenia and psychosis (McGorry et al., 2010), mood disorders (McGorry et al., 2010; Vieta et al., 2011), and anxiety disorders (Fava et al., 2008). The utility and effectiveness of this approach in treatment planning have been shown also for subthreshold mental disorders (Cross et al., 2014). Despite the potential clinical benefits of applying clinical staging in mental health, unitary and clinically available staging models of ED are still missing, and most of the literature on the topic only includes theoretical proposals (Tomba et al., 2023). Existing staging models focused mainly on AN (Beaumont & Touyz, 2003; Maguire et al., 2008; Ramos et al., 2023; Steinglass et al., 2020), transdiagnostic models (Hay & Touyz, 2015; Treasure et al., 2015) (Treasure et al., 2015a; Treasure et al., 2015b; Hay & Touyz, 2015), BN (Cosci & Fava, 2013), and BED (Bodell & Racine, 2023).

While a shared definition of a number of stages and of the criteria for their classification is lacking, there is general consensus about defining stages of ED based on the severity of the illness and about including early or subsyndromal forms (Bodell & Racine, 2023; Cosci & Fava, 2013; Maguire et al., 2008; Steinglass et al., 2020; Treasure et al., 2015). These proposed early/subsyndromal forms are mostly characterized by body-image disturbances or moderate eating restrictions or, in high-risk stages, by predisposing factors such as obsessive-compulsive traits or a family history of obesity (Treasure et al., 2015). The early phases of the illness may then progress to the acute stage, or full syndrome, corresponding to the fulfilment of the diagnostic criteria. This acute phase is when patients usually are seen by a clinician and start receiving treatment, following which partial or complete remission can be observed in some cases (Beumont & Touyz, 2003; Steinglass et al., 2020), despite the debate about a proper definition of the outcome is still open (Tomba et al., 2019). A more severe and persistent form of the illness can present itself in case of remission failure, defined as severe and enduring, or chronic (Beumont & Touyz, 2003; Cosci & Fava, 2013; Ramos et al., 2023; Treasure et al., 2015). The persistence of symptoms might be a consequence of the neurobiological progression of the illness, deriving from dysfunctional eating behaviors, such as fasting, feasting and the oscillation between these behaviors (Treasure et al., 2015). BMI has been identified as the only biometrical marker in staging models of ED (Steinglass et al., 2020), though the notion of BMI as an indicator of illness severity has been criticized by multiple studies as preventing individuals with normal to high BMI from accessing the support needed for their disordered eating symptoms (Beaulieu & Best, 2023; Ralph-Nearman et al., 2020). Distress, measured through the DASS-21 (Henry & Crawford, 2005), has also been evaluated as a possible criterion for allocating patients in the severe and enduring stage of an ED (Ambwani et al., 2020). Similarly, assessment of psychological symptoms from common comorbidities, such as anxiety and depression, might be useful indicators of stages of illness in ED. The presence of comorbidities might indeed represent an important severity indicator in patients with ED, as it has been found to partially explain mortality and to be associated with greater psychopathology and illness duration (Himmerich et al., 2021; Van

Alsten & Duncan, 2020). Contrary to the acute and severe stages of an ED, it is still unclear which symptoms might constitute reliable prodromal markers. Along with body dissatisfaction, additional common prodromal symptoms have been identified, including eating difficulties, dietary restrictions, fasting, and weight/shape concerns and other psychiatric disorder-related symptoms, such as anxiety and depression (Bulik et al., 1997; McClelland et al., 2020; Stice et al., 2021). Recent evidence has also underlined the need to further explore the role of premorbid psychiatric diagnoses to understand whether they represent a risk factor for a longer duration of the ED, or if they are indicative of a distinct premorbid phase, meaning that an intervention tackling this phase might prevent the progression of illness (Van Alsten & Duncan, 2020). Consequently, it might be clinically relevant to include premorbid indicators and transdiagnostic features, such as irrational beliefs, psychological inflexibility, and impaired well-being through a complexity approach. Despite the potential clinical benefits of such complexity approach, relatively few studies proposed staging models for ED, through empirical studies and mostly focused solely on the disorder characteristics (Cosci & Fava, 2013), forgoing the complexity which is an essential characteristic of ED conditions (Levinson et al., 2022)

Recently a large number of studies turned to network analysis (NA) to explore the complexity of ED at symptom level, in an attempt to tackle their intricacies (Monteleone & Cascino, 2021). The conceptual basis of NA, that is the representation of psychiatric conditions as a constellation of interacting symptoms (Borsboom & Cramer, 2013), seems particularly relevant to the study of ED, allowing researchers to model ED as they present in everyday clinical practice (Treasure et al., 2020). In general, network analysis is an ensemble of statistical tools which can be applied in a variety of disciplines (Barabási et al., 2011) to study the dynamic interactions between phenomena through the modelling of networks. These networks are composed of units called nodes, representing a variable of choice, which are connected by edges, representing the correlations between nodes. Through its granular level analysis, NA allows one to differentiate symptoms in terms of clinical relevance, rather than considering them equivalent indicators of an underlying disorder, and allows to describe how

they influence and reinforce each other (as postulated by many psychopathological theories of specific disorders) beyond diagnostic categories (i.e., to explain psychiatric comorbidities) representing a powerful approach to isolate and differentiate specific symptoms configurations at different time-points (Ashaie et al., 2021; He et al., 2023; McElroy et al., 2019), thus providing important information to support the development and identification of different stages of disease.

## Chapter 2. Network Models: Theory and Applications in Psychopathology

### 2.1. The latent variable model of mental health conditions

Mental health conditions are complex entities that encompass biological, social and psychological aspects (Engel, 1977). This complexity, and the interconnectedness of various psychiatric symptoms and associated behaviours, makes the identification and assessment of mental health phenomena a complex and challenging task (Kirkpatrick et al., 2021), further compounded by diagnostic heterogeneity (Allsopp et al., 2019) and lack of clear biomarkers or pathognomy signs (García-Gutiérrez et al., 2020; Kirkpatrick et al., 2021). An additional hurdle in mental health assessment and measurement is represented by the nature of the phenomena themselves. It is not possible to directly measure “depression” or “schizophrenia”; thus, to establish the presence and intensity of a disorder a clinician has to rely on the assessment of their individual symptoms. Once identified, symptoms are then grouped and assigned to a latent variable, the “disorder”, based on indications from the diagnostic system of reference, which are in turn based on statistical attributes of the symptoms, such as frequency of co-occurrence and correlation between each other. For example, if a clinician notes the presence of poor quality of sleep, persistent low mood, reduced appetite, fatigue, and excessive feelings of guilt, they might diagnose a Major Depressive Disorder, following the guidelines of the DSM-5 (American Psychiatric Association, 2013). The taxonomical classification utilized in the various editions of the DSM to categorize psychiatric conditions derives from the direct translation of the medical model of illness to psychiatry by Feighner and colleagues (1972) as an alternative to the diagnosis-averse psychoanalytic approach, the dominant theoretical model during '50s and '60s (Kendler et al., 2010) and represented a dramatic change compared to the idiosyncratic and subjective methods of assessing mental disorders until then in use. This reconceptualization of the identification of psychiatric conditions was explicitly aimed to replicate the nosological approach used in clinical medicine (Fava, 2022), homogenizing individuals with the

same diagnosis in the same way insulin deficiency identifies individuals affected by diabetes (Borsboom, 2008). The medical roots of this approach are also reflected in the enthusiasm and investments in support of the discovery of genetic and neurobiological mechanisms that could constitute the primary cause of specific mental disorders (Borsboom et al., 2019); a body of research that, however, has often led to controversial and inconclusive results despite over 20 years of work by research groups across the globe (Borsboom et al., 2019; Frisch, 2021). The weak results of these etiological studies conducted on mental disorders seem to suggest the inadequacy of a model that considers the disorder itself as autonomous from its symptoms (Borsboom & Cramer, 2013). The affinities between psychopathology and medicine are indeed superficial: in both cases, the diagnosis is elaborated starting from the presence of a set of symptoms but, whereas in medicine it is common to observe the presence of a disease even in the absence of directly observable indicators (as in the case of tumours in the early stages), in psychopathology, it is impossible to even conceptualize a disorder without relying on its symptoms. Another problematic assumption of the psychometric latent variable model is local independence. This is the statistical assumption for which, when we identify a latent variable (a disorder) capable of explaining the correlations between observed variables (the symptoms), then all the direct correlations between the observed variables must be considered spurious since covariance between the variables derives from the common factor (Bollen, 2002; Cattell, 1928; Cramer et al., 2010). However, this model does not reflect the complex dynamics of clinical reality (Radden, 2018). In fact, in clinical practice, clinicians adopt a perspective based on the existence of latent variables, while also assuming the existence of direct relationships between symptoms. The tendency to consider disorders as complex dynamic systems in which symptoms influence each other, rather than being independent of each other, is in line with a way of reasoning that seems to come very naturally to specialists in the field (Kim & Ahn, 2002; Levinson et al., 2022), but is at odds with the psychometric properties of that latent variable model on which the DSM itself is based and to which clinicians rely to establish a diagnosis (Cramer et al., 2010). The hypothesis of a direct relationship between symptoms of a disorder, in addition to coming intuitively to clinicians

when representing mental disorders, is also one of the basic assumptions on which gold-standard psychotherapeutic interventions, such as cognitive therapy and exposure therapy, are based (Barbara Olasov Rothbaum & Ann C. Schwartz, 2002; Cramer et al., 2016). The problematic implications of the latent variable approach, as well as its apparent detachment from clinical reality, led researchers to look for new perspectives through which to reconceptualize the correlations between symptoms of psychological distress (Borsboom & Cramer, 2013).

## 2.2. From latent models to clinimetrics and psychological networks

It has been discussed how mental health conditions are primarily diagnosed based on clinical observations and reported symptoms, and as a result, are affected by huge variability between individual cases. These hurdles underscore the importance of promoting a comprehensive approach to the assessment and classification of mental health symptomatology and conditions. Thus, psychometrics was developed to apply mathematical modelling to the measurement of latent variables. Despite its significant contribution to developing an evidence-based approach to studying mental health constructs, psychometrics presented one major weakness in assessing phenomena in a clinical setting: it was developed independently from the clinical setting, and as such its core assumptions find limited validity and applicability in such context (Fava, 2022). As discussed by Fava and colleagues (2004), this detachment between psychometrics and clinical experience caused two main problems affecting the validity of measuring instruments: the idea that all items (or symptoms) of a psychometric questionnaire carry equal importance (Faravelli, 2004; Fava et al., 2004) and the problems surrounding lack of interrelatedness between comorbid conditions (Feinstein, 1970).

Similar difficulties in translating statistical models to the reality of clinical medicine have already been noted. Within the field, a remarkable attempt to integrate data obtained from statistical methods with information from clinical medical experience was made by Alvan R. Feinstein, who in 1982 introduced the term “clinimetrics” to indicate the domain of measurements concerning medical indexes, rating scales and other expressions used to describe or assess physical symptoms, signs or

other manifestations of illnesses (Fava, Tomba, et al., 2012). The original intent of Feinstein was to overcome the roadblock faced by clinical medicine at that time (Feinstein, 1983), to expand the medical taxonomy of his time to encompass also clinical experiences and start considering those relevant clinical phenomena that should be a core part of any clinical assessment or research in medicine. These included, among others, the patient's own experience of illness, their available social resources, and the disorder's progression rate (Feinstein, 1987). Thus, Feinstein proposed to consider additional indexes along the ones normally included when conducting a medical assessment, to differentiate between patients that would otherwise be considered similar by sharing the same medical diagnosis (Feinstein, 1987). Remarkably, this was nothing new for clinicians, who, except for using the word itself, have been communicating using clinimetric indexes all along (Feinstein, 1987). Progression of illness, overall severity of the medical disorder and functioning impairment, available social support and response to previous treatments are all aspects that are regularly factored in by clinicians despite the absence of formal methods to capture this information (Fava, Rafanelli, et al., 2012). Through clinimetrics, Feinstein attempted to bring back the process of clinical assessment within the realm of clinical practice, stepping back from delegating the assessment process to scientists from nonclinical domains (Tomba & Bech, 2012). Feinstein stressed the importance of developing "new approaches [...] of clinical investigation that can augment the scientific basis of clinical practice while rehumanizing the contents of research data and restoring analytic emphasis to the art of patient care" (Feinstein, 1983). Examples of clinimetric indexes commonly used in clinical medicine are the Jones criteria for rheumatic fever (Feinstein, 1982), the New York Heart Association Functional Classification (The Criteria Committee of the New York Heart Association, 1964) and Apgar's method of scoring the newborn's condition (Feinstein, 1999).

Several researchers, with Fava (Fava et al., 2004) at the forefront, followed by Bech (Bech, 2012) and Emmelkamp (Emmelkamp, 2004), highlighted the limitations of the psychometric model in the field of mental health and promoted a conceptual revision of clinical assessment to include clinimetrics principles. Their work integrated psychometrics with clinimetric principles to pave the



way for significant advancements in the clinical assessment processes in psychology and psychiatry, by promoting the introduction of new criteria to develop and choose clinical measures (Feinstein, 1999; Tomba & Fava, 2007), the introduction of new areas and methods of clinical assessment (Tomba & Bech, 2012; Tomba & Fava, 2007), and the conceptualization of new approaches to clinical reasoning to improve standardization of clinical data collection, such as the macro- and micro-analysis (Bech, 2012; Emmelkamp, 2004). While clinimetrics has been enthusiastically adopted by some researchers and clinicians, others raised concerns about this model. Streiner (Streiner, 2003a, 2003b) argued that clinimetrics is not an innovation as much as a redundant and ultimately unnecessary reinterpretation of the psychometric model. Emmelkamp himself, a key figure in the field of clinimetrics, while acknowledging the benefits of the clinimetric approach (such as the focus on a scale's sensitivity to change), warns against abandoning classic psychometrics before providing definitive proof of the clinimetric approach effectiveness (Emmelkamp, 2004).

### 2.3. A network theory of mental disorders

In the second decade of the 2000s, a group of researchers from statistical and research methodology backgrounds, independently from the clinimetric approach, also sought to address the limitations of classical psychometrics in measuring mental health symptoms. Borsboom first (Borsboom, 2008) and later Cramer (Cramer et al., 2010) and Fried (Fried, 2015) questioned the fitness of the Latent Variable model characterising most available psychometric screening instruments and psychiatric taxonomy, in which all symptoms are considered roughly independent and equally relevant to a specific disorder because all resulting from the same underlying condition. Such an assumption, according to Fried (Fried, 2015), is hardly tenable as it contradicts real-life clinical observations, where symptoms interact and vary in importance; this position appears aligned with what Fava and colleagues observed in previous works (Fava et al., 2004).

An alternative approach to the Latent Variable Model was then proposed, initially called *network perspective* (Borsboom, 2008; Cramer et al., 2010; Schmittmann et al., 2013), and later known as

*network theory of mental disorders* (Borsboom, 2017; Borsboom et al., 2021; van Bork et al., 2018). At the core of this theory, there is the idea that mental states act as networks, where individual entities (from psychopathology symptoms to social or medical variables) are represented as *nodes* of the network. These nodes are connected by *edges*, which represent the probabilistic dependency between two nodes after conditioning on all other variables in the data (Borsboom, 2017; Borsboom et al., 2021). An example of a psychological network can be seen in Figure 2.

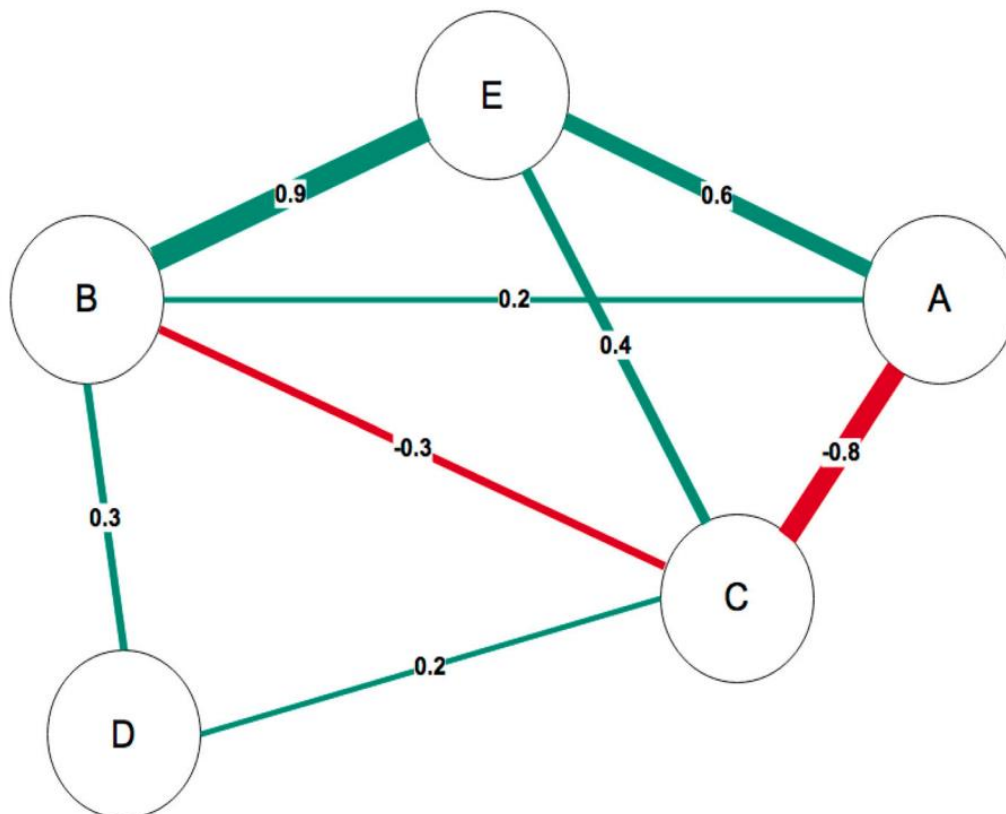


Figure 2 Example of a psychological network. The circles represent the different nodes of the network, the green and red lines represent positive and negative edges, and the number represent the correlation coefficient of the edges (from Hevey, 2018)

Each psychological network is a complex system that persists in a state of equilibrium; external factors (life events, biological triggers, prolonged exposure to stress) can activate a node, triggering a cascade effect of activation of other nodes (Figure 3).

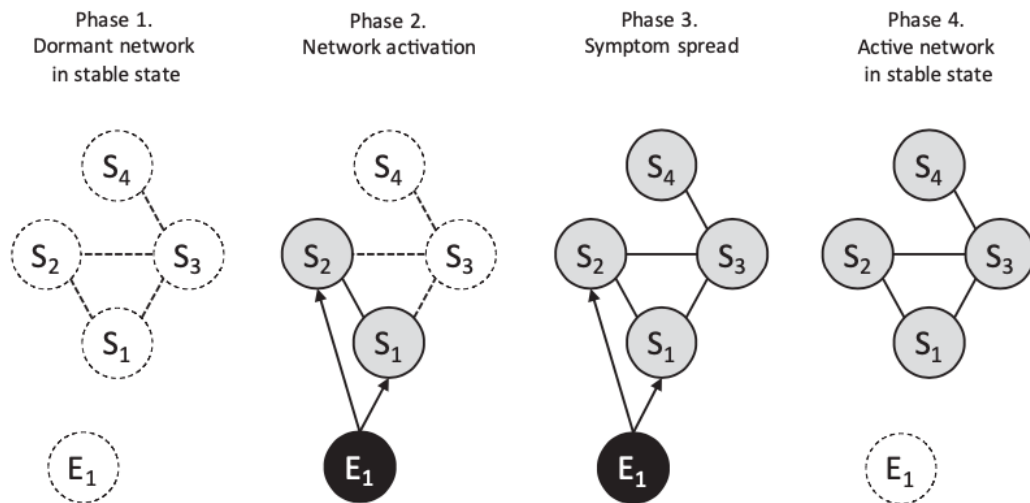


Figure 3 Network progression from health to illness (from Borsboom, 2017)

The propagation of this cascade depends on the strength of the edges involved; stronger edges will carry the perturbation further. This model represents a significant step away from the latent model approach and proposes a novel conceptual alternative: symptoms do not cluster because of a shared underlying disorder that originates them (the latent variable), rather they group together because they can cause, and maintain, each other (Fried, 2015). The formulation of the network theory of mental disorders provides an alternative to the monocausal approaches to psychopathology that assume that symptoms have a clear univocal, often neurobiological, cause (Borsboom et al., 2019) and establishes the individual symptoms and mental states as autonomous entities capable of triggering and maintaining mental health conditions (Borsboom et al., 2021). This is in line with the dynamics affecting the cognitive components of an individual and postulated to be at the core of the onset and maintenance of mental health conditions in cognitively-based therapeutic approaches (Beck, 1967; Ellis, 1962); in clinical practice, when asked to reflect on the pathogenesis of mental disorders, both

clinical experts and patients often report a dense set of causal relations between their symptoms (Borsboom & Cramer, 2013).

#### 2.4. Network models of psychological symptoms

At its core, a network is an abstract model composed of nodes, representing various variables of choice, and a set of edges, representing pairwise conditional associations, connecting said nodes (Borsboom et al., 2021). In psychological network analysis, nodes usually represent psychological variables (e.g. psychological symptoms, beliefs, behaviours), while edges represent their statistical relationships. Depending on the research question, a node can represent the global score of a scale, a sub-scale, or a single item. In psychology we currently do not have objective, definitive, and empirically established theories on how symptoms influence each other; it is therefore impossible to estimate the structure of a psychological network based on theory alone. Such a structure must therefore be inferred from the data, posing the question of how to extract it (van Borkulo et al., 2014). This question eventually led to the conception of network analysis, an ensemble of statistical methods that allow to determine the structure and interactions between the components of a network by estimating dependencies between variables while conditioning on other variables (van Bork et al., 2018). A conditional association between two variables holds when these variables are probabilistically dependent, after conditioning on all other variables in the data. Should this hold true, the graphical model obtained from the data will represent an edge connecting the two nodes. However, should the association between two variables be explained by other variables in the network, their conditional association will vanish when controlling for these other variables, and the corresponding nodes will lose their connecting edge in the graphical representation. In disciplines such as physics and computer science, probabilistic graphical models are used to model the conditional dependencies between variables. The description of the joint probability distribution of a set of variables in terms of pairwise statistical interactions is a graphical model known as the pairwise Markov random field (PMRF) (Epskamp & Fried, 2018). In a PMRF, the edges connecting two nodes can be weighted,

where the weight of an edge represents the strength of the association between two variables after conditioning on all other variables in the model (Epskamp et al., 2018). The general term PRMF does in fact encompass different types of network models; however, for the purpose of psychological network modelling, three main subclasses have been adopted: *Ising model* for binary data, *Gaussian graphical models (GGM)* for continuous data, and *Mixed Graphical Models* for mixed continuous and binary data (Borsboom et al., 2021; van Bork et al., 2018). For the purpose of the present work, only GGMs will be discussed.

#### 2.4.1. Gaussian Graphical Models

To build a GGM from continuous cross-sectional data it is possible to use the correlation matrix constructed from the scores obtained by the sample to the items of the psychological tests. Within the correlation matrix, each cell represents the correlation index between the subjects' responses to two particular items, and for each non-zero correlation, it is possible to define an edge between the two nodes.

Variable	A	B	C	D
B	.2	–	–	–
C	–.8	–.3	–	–
D	0	.3	.2	–
E	.6	.9	.4	0

Figure 4 Correlation matrix of the 5 variables used in the network from Figure 2 (from Hevey, 2018)

Correlation, however, as a simple measure of the covariance between variables, does not allow us to understand whether two variables correlate either as a result of a direct causal link or because they share a common cause; therefore, it is preferable to estimate partial correlations (van Bork et al., 2018). A partial correlation is a correlation that is observed between two variables after controlling for the effect that the other variables have on them; consequently, in a network of partial correlations,

an edge between two nodes is estimated when the relationship between those two nodes cannot be explained by any other of the nodes within the network. For this reason, concerning the network of correlations, in a network of partial correlations, when we observe a partial correlation equal to zero (i.e. absence of a link), we can actually speak of conditional independence, an aspect that is found in Markov Random Fields; in fact, networks of partial correlations are defined as the Gaussian multivariate version of the Markov Random Field (van Bork et al., 2018). However, representing an edge for each non-zero correlation implies obtaining a fully connected network: the correlation and partial correlation values will be influenced by sample variation and therefore exact zeros will be matrices will be rare. Consequently, correlation networks will nearly always be fully connected networks, possibly with small weights on many of the edges that reflect weak and potentially spurious correlations. Such spurious relationships will be problematic in terms of the network interpretation and will compromise the potential for network replication (Epskamp et al., 2018; Hevey, 2018). For this reason, some thresholds for inclusion and model selection are established, defining either a minimum correlation coefficient that must be exceeded to represent the correlation with a link, or including only correlations that exceed the threshold of statistical significance (van Bork et al., 2018).

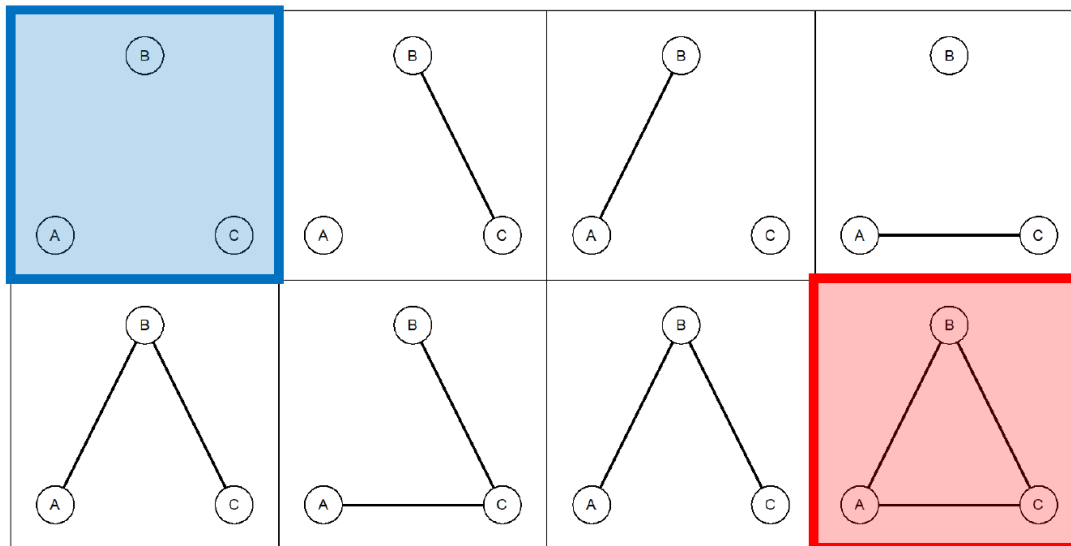
#### 2.4.2. Model selection in psychological network analysis

The PMRF is a multivariate statistical model, and as such there are multiple ways in which the model's parameters (the edges, in our case) could be estimated, ranging from an independence model, the simplest possible model in which all nodes are independent of each other, to a saturated model, the most complex model in which all nodes are connected to all other nodes. Between them there is a plethora of models placed on a continuum that encompasses all possible variations going from the independence model to the saturated model (Figure 4); somewhere along this continuum lies the "true model", the model from which the data originated and which recovery represent the researcher's goal (Blanken et al., 2022). In this sense, neither choosing the independence model nor

the saturated model is desirable: adopting the independence model will lead to type 1 error (exclusion of real edges), meaning the removal of “true” edges, while the saturated model will lead to type 2 error (inclusion of false edges), meaning the inclusion of spurious edges. Following Occam’s razor principle, the objective is therefore to retrieve the simplest possible model without substantially altering its performance. In other words, it is desirable to remove all edges for which there is no substantial evidence available supporting their existence within the model (Blanken et al., 2022; Epskamp & Fried, 2018).

When it comes to algorithms to retrieve the preferred model, there are many choices available and the choice should be based on the type of data, the size of the sample, and the objective of the study; however, the most important criteria that should be considered are the sensitivity and specificity of the algorithm (Epskamp & Fried, 2018; Isvoranu & Epskamp, 2023). Generally speaking, model retrieval algorithms can be divided into 4 categories depending on their heuristic: thresholding, pruning, regularization, and model search.

A simple (independence) model



A complicated (saturated) model

Figure 5 Range of possible models for a 3 nodes network. Within this range of models lies the "true" model which should be retrieved. (From Blanken et al., 2022)

Thresholding and pruning are conceptually similar and represent among the simplest approaches to model selection. Broadly speaking, in both approaches the rationale for inclusion of an edge is that it satisfies a predetermined criterion (e.g.  $p$ -value, Baye's factor). In thresholding, when an edge does not satisfy the criterion, it is reduced to zero and simply excluded from the network representation, though it still exists within the model. As such, non-zero edges will be estimated and included in the visual representation of the model, while other non-zero edges will still be estimated but excluded from the model. In pruning, on the other hand, edges that do not satisfy the criterion are effectively reduced to zero within the model, and then the model is re-estimated. This process can be repeated multiple times until a final model is retrieved in which non-substantial edges – as defined by the predetermined criterion – are set to zero. Both thresholding and pruning benefit from being fast model selection methods, and guarantee that the specificity of the model is known as it is based on the predetermined criterion (Williams & Rast, 2020). Additionally, in both approaches the edge selection is unbiased, as the criterion is indiscriminately applied to all edges of the network. Both methodologies, however, present considerable downsides that should be taken into account when deciding how to perform model selection. Thresholding in particular does not actually perform any model selection, but simply hides edges from the saturated model based on a specific criterion; this means that there is a high risk of type 2 error. Finally, it must be noted that both thresholding and pruning might fail to detect real edges, thus incurring in type 1 error, due to insufficient statistical power (Blanken et al., 2022).

A third approach to retrieving a network model consists of statistical regularization, in which edge weights are estimated through penalized maximum pseudo-likelihood estimation (Hastie et al., 2001). In other words, when estimating the edges of a network, the fit is penalized by how many edges are included in the network. One particular type of regularization that has been frequently adopted for PMRF is the “least absolute shrinkage and selection operator” (LASSO) (Tibshirani, 1996). The LASSO causes edges to automatically shrink to be exactly zero and results in a



parsimonious network. If the data is generated from a sparse network with pairwise interactions, this approach will in fact converge on the true model. The level of penalization of a LASSO estimation is decided manually by setting the value of the tuning parameter  $\lambda$ , where zero equals to no regularization. Specifically for GGMs, the graphical LASSO (GLASSO) (Friedman et al., 2008) is also available. The GLASSO uses the correlation matrix to estimate the GGM using regularized multivariate estimation (Blanken et al., 2022). It is also possible to estimate the optimal  $\lambda$  turning parameter, as opposed to setting it manually, using the Extended Bayesian Information Criterion (EBIC) (Chen & Chen, 2008). The EBIC relies on a hypertuningparameter  $\gamma$ , essentially a parameter that tries to constrain estimation by the size of the space of possible models: the more edges, the larger the space. The LASSO regularization has proven a powerful technique, especially in those cases where the sample size might be relatively small (Isvoranu & Epskamp, 2023). However, in the case of larger datasets, LASSO might not be the optimal choice, and may lead to type 2 error just as with thresholding and pruning, with the additional downside of less predictable false positive edges (Blanken et al., 2022).

Finally, another approach to estimate the structure of a PMRF is to perform a model search through the space of possible models, an approach which is conceptually similar to repeated pruning processes but which can be executed through much more refined algorithms. When estimating GGM, one such algorithm which has demonstrated excellent performance is the *ggmModSelect* (Epskamp et al., 2018). The *ggmModSelect* algorithm uses regularized estimation, as explained above, to first perform a quick search through model space for a model that can be then used as a starting point for a stepwise model search. Then, the algorithm proceeds with combining step-up (adding edges) and step-down (removing edges) estimations to explore all possible models in which an edge is either added or removed. This process is repeated until the algorithm reaches the optimal information criterion. Model search application has relevant advantages over the other approaches: they can retrieve accurate network structures with a good balance between sensitivity and specificity, and they

ensure that the final model retrieved is the best possible fit for the data. However, due to the extremely numerous estimations involved in this approach, the model search can be extremely slow, especially for bootstrapping operations (Blanken et al., 2022).

Once a network is estimated, it is possible to analyse its structure both globally and at the node level. The analysis of the global structure of the network consists of examining how large is it, the global strength of the edges, and whether it is sparse or densely connected. At a node level, it is possible to examine how nodes differ in various characteristics, such as which nodes are most the most strongly connected in general, or which nodes present particularly strong connections with other nodes. Assessing the global structure of a network and the strength of all its edges has relevant implications: according to network theory, networks with several strong connections between nodes and higher global edge strength – either because of stronger edges, more numerous edges, or both – will more easily trigger a vicious circle where symptoms activate each other (Borsboom, 2017). Network structure invariance, specific edges invariance, and global network strength invariance are the indices used to compare networks from different populations through the *NetworkComparisonTest* (Van Borkulo et al., 2019). The same reasoning applies for individual nodes, where it might be interesting to isolate which nodes present the strongest connections, and thus are more likely to trigger a cascade of nodes (symptoms) activation. Given their relevance within a network, these nodes might also represent preferential targets for clinical interventions, though their actual usefulness as therapeutic targets is still being debated (Dablander & Hinne, 2019). The node centrality measures traditionally used in psychological network analysis are *strength*, *closeness*, and *betweenness*; however, recent works have questioned the fitness of the last two indices in psychological sciences (Bringmann et al., 2019) and have been mostly considered obsolete since. The remaining centrality index, strength centrality, while still presenting some limits in its interpretation (Robinaugh et al., 2016; Spillet et al., 2020), is by far the most common index, representing the absolute sum of the

weights of all the edges connected to it, meaning how much a given node is capable of influencing or is influenced by the other nodes directly connected to it.

Once a network model has been retrieved and the various indexes have been computed, it is also important to test its replicability and generalizability. Since network models in psychological research are generally built from the data obtained by a small percentage of the target population, likely, the estimate of the population value is likely not perfect, and analysing a different sample from the same population would produce different results (Fried et al., 2022). To assess whether the identified connections are accurate resampling methods such as bootstrapping can be applied (Epskamp et al., 2018). The estimation of the accuracy of a network usually involves estimating the accuracy of the edge weights by drawing bootstrapped confidence intervals (CIs), investigating the stability of centrality indices after resampling, and performing bootstrapped difference tests for edge weights and centrality indices to test whether these differ significantly from each other. It is important to note, that when applying model selection strategies, such as the ones previously discussed, the bootstrapped CIs lose the fundamental property of a CI, meaning they no longer contain the true value in 95% of the intervals. This is because many edges are might be set to zero if there is not enough evidence that they are non-zero. As such, bootstrapped CIs should not be interpreted as estimated intervals around the true value, but as measures of the variability that can be expected in parameter estimates (Fried et al., 2022). To examine the stability of centrality indices, it has been proposed to adopt the case-dropping bootstrap to compute the *Correlation Stability Coefficient* (CS-coefficient) (Epskamp et al., 2018), which represents the maximum proportion of subjects that can be dropped from the original data while maintaining an in at least 95% of the cases a correlation coefficient of at least 0.70 between the original centrality coefficients and those observed in the bootstrapped sample (Fried et al., 2022).

## 2.5. Clinimetrics and network theory

It has recently been suggested that clinimetrics and network theory and analysis would seem to converge over various key areas of relevance for clinical assessment, to the point where they might complement each other to improve the conceptualization and collection of clinical information (Tomba & Tomei, 2024). While different in origin, both approaches converge on the necessity of integrating multiple information sources to create a more dynamic and representative system for clinical assessment. Arguably, this convergence of separate and independent research lines is, in and of itself, an indicator of the aforementioned issues that affect the translation of the psychometric model to clinical practice. Even more striking, though possibly not surprising, is the fact that both approaches independently identify comparable critical areas in traditional psychiatric taxonomy and questionnaires, and propose conceptually similar solutions (Borsboom, 2022; Fava, 2022). While there are various areas of convergence between clinimetrics and network theory (Tomba & Tomei, 2024), for the purpose of the present work the focus will be on two specific topics: the role of individual symptoms, and the importance of identifying stages of illness.

Both clinimetrics and network theory emphasize the importance of evaluating the specific role and impact of individual symptoms in a disorder's presentation, which provides valuable information for case formulation and treatment planning. In clinical practice, the number of symptoms a patient reports does not necessarily mirror the severity of their clinical presentation. The intensity and quality of each symptom and the patient's perceived impairment must also be considered. Identifying the most impairing symptoms, and understanding their origin, triggers, and maintenance dynamics are all critical components of the clinical assessment and case formulation process (Fava & Belaise, 2005). Clinimetrics therefore encourages clinicians to adopt an approach in which the significance of symptoms is interpreted in light of their relationship with other symptoms, suggesting that their reciprocal influence should be carefully taken into account during a clinical evaluation (Fava, Rafanelli, et al., 2012). Network theory proposes a similar approach to that of clinimetrics, focusing on the importance of evaluating the specific role and impact of individual symptoms in the clinical

presentation of a disorder through centrality metrics (Borsboom et al., 2021), emphasizing the importance of a node's centrality and the weight of its edges within a network as an index of the real impact of that specific symptom in the clinical presentation (Bringmann et al., 2019).

Clinimetrics and network theory also postulate that symptoms configure themselves and interact differently at different states (i.e. health, prodromal, acute, chronic), and agree on the importance of identifying specific configurations of symptoms that characterize such states, arguing that the progression of symptoms severity and the influence of specific symptoms in subsequent clinical presentation are critical aspects for selecting the appropriate treatment (Borsboom, 2017; Fava & Kellner, 1993; Robinaugh et al., 2020; von Klipstein et al., 2020). Through the staging model, clinimetrics offers a powerful conceptual framework and practical tool for clinicians to model their intervention on the patient's current stage of disease (Fava & Kellner, 1993; Roefs et al., 2022), rather than relying on a one-size-fits-all approach (Cosci & Fava, 2013). In a similar fashion network psychometrics introduces instead the concept of attractor states, which are equilibrium states that networks converge to and in which stabilize after a perturbation (Schmittmann et al., 2013; van de Leemput et al., 2014). This formulation reflects well the uneven progress observed during clinical interventions, which often involve a series of improvements and setbacks until a stable state of health with minimal symptoms is achieved. (Fava & Tomba, 2010; Tomba et al., 2019).

Finally, another critical element that heavily influences the heterogeneity of clinical presentations in psychiatry is comorbidity. Both network theory and clinimetrics acknowledge the limitations of traditional evaluation processes, and the advantage of introducing complementary methods that may be better equipped to capture the complexity and heterogeneity of co-occurring psychiatric conditions (Bogenschutz & Nurnberg, 2000; Borsboom, 2008, 2017; Cramer et al., 2010; Fava, Rafanelli, et al., 2012; Fava et al., 2014; Feinstein, 1970, 1987)

Comorbidity, which can be defined as the contemporaneous presence of two or more mental health conditions (van Loo & Romeijn, 2015), has been an ever-increasing presence in psychiatric

practice. Clinimetrics, however, proposes a different definition for comorbidity (Feinstein, 1987). Feinstein defined comorbidities as “any distinct additional clinical entity that has existed or that may occur during the clinical course of a disease that is under study” (Feinstein, 1970), which clinicians need to investigate and contextualize within individual clinical presentations. With true comorbidity, Feinstein (Feinstein, 1970) identified the co-presence of two clearly separate diseases, either contemporaneously present due to a common aetiological cause, or due to one disorder causing the other (Bogenschutz & Nurnberg, 2000). Conversely, with spurious comorbidity Feinstein (Feinstein, 1970) described a blurred clinical picture where the relationship between two diagnoses may not reflect a true relationship between the disorders; this misclassification could be determined by phenotypic heterogeneity, or due to the construction of artificial categories that share the same underlying dimensions or create a discrete boundary where none actually exists, such as in the psychiatric nosological approach (Bogenschutz & Nurnberg, 2000). Feinstein's conceptualization of comorbid entities emphasizes their clinical relevance. Thus, the concept of comorbidity goes beyond just the co-occurrence of disorders and focuses on a more detailed examination of how the co-existing symptoms interact and impact the clinical picture. The complex nature of psychiatric comorbidities and the challenges in their identification calls for a standardized procedure that will help the clinician in identifying and separate the various clusters of symptoms based on how they present in clinical reality, rather than following predetermined categories. Fava and colleagues (Fava, Rafanelli, et al., 2012) built upon Feinstein’s work, expanding the process of comorbidity assessment to include subsyndromal symptoms, illness behaviour, functional capacity and psychological well-being. Furthermore, Fava, Tossani, and colleagues highlighted various key aspects of clinimetric comorbidities, such as the need to identify appropriate statistical methods to reduce the discrepancy between comorbidity across studies, the need to develop and test causal models of comorbidity, and the application of the clinimetric method to move past the disorder-based comorbidity concept by including clinically significant subthreshold conditions and other psychological factors that may influence the choice of treatment (Fava et al., 2014).

This granular level of detection of comorbidities is also present in network theory by its focus on the complex connections and dynamics between individual symptoms or edges of the network (Cramer et al., 2010). In network models, as reported by Cramer et al. (2010), nodes tend to cluster together, forming highly connected communities. These communities are usually only weakly connected to nodes from other clusters. However, nodes from different clusters may present strong connections between each other, not unlike nodes within the same cluster (Borsboom, 2017). In clinical terms, symptoms from different disorders may be as strongly related as symptoms from the same disorder, leading to comorbidity by reciprocal influence. Nodes that connect clusters in this way are called bridge nodes; comorbidity arises as a node triggers the activation of nodes from neighbouring cluster(s) connected by bridge nodes. This can cause the simultaneous activation of different groups of symptoms. It is noteworthy that the notion of certain symptoms transmitting "pathological" activation from one group of nodes to another hinges on the premise that symptoms are not interchangeable, which challenges a fundamental tenet of the traditional taxonomy of mental disorders. This model suggests that symptoms are not equivalent, and a diagnosis cannot solely rely on the number of symptoms present, but rather on their intensity and their contribution to the overall clinical presentation (Borsboom, 2008).

## 2.6. Network analysis literature on Feeding and Eating Disorders

As already discussed, the current principal nosological approaches to ED are based on a conceptualization of illness which may not be adequate to capture the complexity of ED presentations as they appear in clinical reality (Levinson et al., 2022; Treasure et al., 2020). Levinson and colleagues (2022) argued that there are three main aspects that make traditional psychiatric taxonomy inadequate to capture the nature of ED: first, it is unlikely that all the symptoms of an ED derive from a single latent cause; second, it is not possible to view symptoms independent of their cause; third, symptoms of co-occurring disorders are unlikely to exist because of covariation in latent constructs. On all these

fronts, network theory and its statistical techniques could represent a more clinically valid approach. Moreover, it should be noted that it is possible for the network conceptualization of a condition and its latent variable model counterpart to co-exist. Fried and Cramer (2017) suggested that mental health conditions represent such a complex phenomenon that the simple propensity for one approach or the other is very difficult to maintain: a view that favours an interpretation based exclusively on latent variables is as unrealistic as a model based solely on the reciprocal relationship between symptoms; it seems much more plausible that the two models can complement each other in improve our representation and understanding of psychological phenomena. In the case of ED, for example, a latent cause such as the experience of being mocked and stigmatized for one's weight can lead to overestimating the importance of body shape and weight, and therefore to developing unhappiness about these aspects of one's own body, eventually leading to food restriction behaviours (Smith et al., 2018). Such behaviours can lead to the development of and ED and of additional symptoms that will support its maintenance even when the subject is no longer exposed to the trigger event (the teasing); it is indeed feasible to assume that the nodes of the symptom network are tightly connected, it will be relatively easy for "hits" on a given area of functioning (i.e. well-being, mood, anxiety, beliefs) to triggerd a cascade powerful enough to keep activating nodes representing ED symptoms.

The application of network analysis to study ED has focused mainly on the structure of the ED symptom networks, comorbid conditions, and relevant psychological constructs typically associated with ED (Monteleone & Cascino, 2021). The results from these studies provided further support to the transdiagnostic model of ED, though other transdiagnostic elements appeared just as relevant, inviting researchers to consider the possibility that ineffectiveness, interoceptive awareness and affective problems might be as central as overvaluation of weight and shape and ED network studies must include a wider range of potentially relevant psychological factors (Monteleone & Cascino, 2021). Nonetheless, the construction of transdiagnostic ED symptom networks showed that overvaluation of body shape and weight is consistently among the most central nodes (Calugi et al.,



2020; DuBois et al., 2017; Forrest et al., 2018; Forrest et al., 2019; Wang et al., 2019) and that higher overvaluation of shape and weight correlated with stronger and more densely connected networks (DuBois et al., 2017). These pieces of evidence suggest the possibility that the degree of overevaluation of body shape and weight may directly influence prognosis, since the presence of stronger connections between nodes of a network is associated with worse clinical presentation (Fried et al., 2017).

As reported by Monteleone and Cascino (Monteleone & Cascino, 2021), and Tomei and colleagues (Tomei et al., 2022), the majority of network studies in ED published so far focused on AN, followed by studies exploring network structures in transdiagnostic ED samples (Monteleone & Cascino, 2021; Tomei et al., 2022). Comparatively, few studies focused on BN, BED, or OSFED, despite the latter two conditions being widely represented in the ED population (Santomauro et al., 2021).

Multiple studies that analyzed the central symptoms of AN observed that aspects related to shape and weight concerns were the ED-related nodes most frequently resulting as central (Calugi et al., 2020; de Vos et al., 2021; Goldschmidt et al., 2018; Hagan et al., 2021; Kerr-Gaffney et al., 2020; Ralph-Nearman et al., 2021). This however might be linked to the use of the Eating Disorders Examination – Questionnaire (EDE-Q) (Fairburn & Beglin, 1994), a scale built on the cognitive-behavioural theory of ED. Indeed, studies using other assessment scales observed that other ED dimensions such as interoceptive awareness, feelings of ineffectiveness and preoccupation with emotional regulation were just as central (Monteleone et al., 2021; Schlegl et al., 2021; Solmi et al., 2018). With regard to non ED-specific nodes, depressive symptomatology resulted in highly central in the works by Monteleone and colleagues (Monteleone et al., 2019), de Vos and colleagues (de Vos et al., 2021), and in the work by Ralph-Nearman and colleagues (Ralph-Nearman et al., 2021). Nodes representing difficulties in social functioning, such as problems with communication or a tendency

to isolation, were also reported as highly central when included in the analyses (Kerr-Gaffney et al., 2020).

About central nodes of BN, studies identified a desire to lose weight and preoccupation with shape and weight as the most prominent ED-related symptoms, further supporting Fairburn's model (Forrest et al., 2018; Goldschmidt et al., 2018; Levinson et al., 2017; Schlegl et al., 2021). Similarly, as for AN, non-specific ED symptoms such as feelings of ineffectiveness (Schlegl et al., 2021; Solmi et al., 2018), and symptoms of depression (Levinson et al., 2017) featured as the most central nodes. Network studies on BED patients, again identified concerns and overvaluation of weight and shape as the most central ED-related nodes (Hilbert et al., 2020; Solmi et al., 2018; Wang et al., 2019). However, non-ED related nodes representing low self-esteem (Hilbert et al., 2020) and depression (Solmi et al., 2018) showed higher overall strength centrality.

Several studies that retrieved network models from the data of transdiagnostic ED populations observed that nodes representing shape and weight concerns or dissatisfaction, eating restraint, and drive for thinness consistently featured as the most prominent ED nodes (Brown et al., 2020; Goldschmidt et al., 2018; Smith et al., 2020; Wong et al., 2021). Similarly to specific ED diagnoses, the nodes representing subjective ineffectiveness (Olatunji et al., 2018; Vervaet et al., 2021), depression (de Vos et al., 2021; Solmi et al., 2018), and anxiety (Forrest et al., 2019; Smith et al., 2019) resulted highly central in transdiagnostic study samples as well. Interestingly, de Vos and colleagues (2021) reported how aspects of positive functioning, such as purpose in life and self-acceptance, resulted highly relevant when included in the network.

So far, results from network studies in ED consistently stressed the importance of non-ED related psychopathology and psychological dimensions as much as, or even over, ED-specific manifestations (Monteleone & Cascino, 2021). The relevance of non-ED specific nodes such as ineffectiveness, difficulties in communication, and depression seem to suggest that emotional

components and interpersonal difficulties might act both as triggers and maintaining factors for the disorder. Their potential as therapeutic targets has also been highlighted previously by Monteleone and Cascino (2021) and constitutes a relevant aspect which requires further investigation. Despite this, it would seem published studies so far focused mostly on the typical features of ED, such as behaviours and attitudes towards food and body image, at the expense of other manifestations just as relevant for clinical interventions (de Vos et al., 2021). The pictures that consistently emerge from the application of network analysis in disordered eating populations are of high relevance to the affective symptomatology in any network where such symptoms were included (Monteleone & Cascino, 2021). This is consistent with a large body of non-network studies describing the relevance and severity of affective symptoms in ED. Affective symptoms have long been observed to accompany ED as often as in 80% of the cases (Godart et al., 2015) and the non-linear effects of their influence has been object of study for more than 40 years (Williamson et al., 2004). In fact, it has been observed that the intensity of negative affective symptomatology in ED and in a primary diagnosis of an affective disorder are comparable (Voderholzer et al., 2019), somewhat in contrast with the regulating role of disordered eating on emotional experiences postulated by the classical cognitive model of ED (Fairburn et al., 2003). To further complicate the picture, studies also highlighted the different impact of affective symptomatology depending on the timing of their appearance in relation to the ED onset (Godart et al., 2015). Another category of non-specific ED characteristics that were highlighted as particularly relevant in network studies are cognitive aspects such as, self-perception, overvigilance, inhibition and high personal standards in particular (Monteleone & Cascino, 2021). On other hand, other cognitive dimensions of relevance for ED, such as psychological flexibility or rational and irrational beliefs, have been so far neglected by the network literature, with the exception of a recent work by Tecuta and colleagues (2023) which however focused on the balance between rational and irrational beliefs, rather than their individual role. In line with the broader cognitive framework for ED conceptualization (Cooper et al., 2005), cognitive profiles such as irrational beliefs and low psychological flexibility are particularly relevant in adult with ED. This conceptualization has also

found empirical support in the works of Espel-Huynh, Muratore, Virzi, Brooks, & Zandberg (2019). Furthermore, a strong correlation between irrational beliefs and ED psychopathology has been observed in non-clinical populations (Mayhew & Edelmann, 1989; Steinglass et al., 2007; Tecuta et al., 2019) suggesting a role for these cognitive aspects also at an etiological level and might inform the formulation of specific stages of illness (Barton et al., 2022). Finally, positive psychological functioning emerges as a promising research direction in ED. Consistently with previous studies (Tomba et al., 2014), aspects of positive functioning resulted strongly relevant when included (de Vos et al. 2021), and aspects of positive functioning might be another relevant indicator to identify specific stages of illness in ED (Tomba et al., 2019).

## Chapter 3. Research Studies

### 3.1. Rationale

To improve our understanding and further refine clinical interventions for ED, it is necessary to account for their complexity and variety of presentations (Levinson et al., 2018; Monteleone & Cascino, 2021), including how symptoms may surface or recede over the course of illness (Cosci & Fava, 2013). Considering the benefits of staging models to clinical research and practice and the absence of a staging model of ED based on clinical and cognitive data, the following project sought to explore the characteristics of ED at different stages by recruiting populations with various degrees of ED symptom severity, modelling network structures of their symptomatology and comparing the structures of these networks to identify nodes and edges specific for each of these populations. At the time of writing, however, no study tried to use NA to delineate different stages of ED encompassing wider cognitive aspects, such as rational and irrational beliefs, psychological inflexibility, or positive functioning from both general population (healthy, at risk for ED, acute ED) and clinical population at different levels of ED symptoms severity.

### 3.2. Aims and hypotheses

This project's goal is to evaluate the feasibility of applying NA to determine clinical stages of ED illness on the base of the severity of ED symptomatology, psychiatric comorbidities, cognitive factors, and positive functioning. To do so, an exploratory study on the psychological network structures of groups with different ED symptom severity has been conducted (Study 1); subsequently, the structures modelled in the previous study have been compared to observe differences across multiple indexes (Study 2).

Study 1 aimed to retrieve the network structure and observe the interactions between ED symptomatology, psychiatric comorbidities, cognitive factors, and positive functioning of different

groups of individuals with various degrees of ED symptom severity (healthy controls, at ED risk, acute ED, ED clinical population).

Study 2 aimed to isolate the significant differences in the network structures among these groups, to determine which ED symptoms, psychiatric comorbidities, cognitive factors, or positive functioning dimensions might help differentiate between these groups, and possibly be used as markers to differentiate between no disorders, prodromal and acute stages.

Despite the exploratory nature of Study 1 and Study 2, some hypotheses can be advanced based on existing literature. Specifically:

Study 1 hypotheses:

- (a) For the healthy control group, network nodes representing positive functioning (MHC-SF scales) will be the strongest nodes in the network (Tomba et al., 2019), followed by the node representing rational beliefs (ABS2-SF Rational Beliefs scale) (Balkis, 2024).
- (b) In the at-risk population, nodes representing weight concerns (EDE-Q Weight Concerns) (Stice et al., 2017), shape concerns (EDE-Q Shape Concerns) (Smith et al., 2018), and depression (DASS-21 Depression) (Stice et al., 2017) will be the most central nodes in the network.
- (c) In the acute population nodes representing psychological inflexibility (AAQ-II) (Sandoz et al., 2013), restricted eating (EDE-Q Restriction) (Smith et al., 2018), and depression (DASS-21 Depression) (Rojo et al., 2006) will be the most central nodes in the network.
- (d) In the clinical ED population the nodes representing restricted eating (EDE-Q Restriction) (Walsh et al., 2013), weight and shape concerns (EDE-Q Weight Concerns; EDE-Q Shape Concerns) (Tecuta et al., 2021), and anxiety (DASS-21 Anxiety) (Treasure et al., 2020) will be the most central nodes in the network.

Study 2 hypotheses:

- (a) The network models from the at-risk population will show significantly higher global strength, higher absolute strength of the EDE-Q and DASS-21 nodes clusters, and lower absolute strength of the MHC-SF nodes cluster, and significantly different network structure compared to the network model of the healthy control group.
- (b) The network model from the acute population will show significantly higher global strength, higher absolute strength of the EDE-Q and DASS-21, and lower absolute strength of the MHC-SF node clusters, and significant differences in network structure compared to the network model of the at-risk group
- (c) The network model from the clinical population will show no significant differences in global strength and no significant differences in network structure compared to the network model of the acute group.

### 3.3. Methods

#### 3.3.1. Participants

Recruitment of study participants began in March 2021. The last study participant was recruited in September 2023. Participants were recruited from the general population (n=1186) and from an outpatient clinic specialized in ED treatment (CP; n=139) in northern Italy. Participants from the general population were recruited through online advertisement on social media platforms (Facebook, Instagram, LinkedIn, Reddit), and subsequently divided into three groups (healthy controls, at risk, acute) based on the severity of their ED symptomatology (see Inclusion and exclusion criteria). Participants from this population who exhibited ED symptomatology of clinical relevance (EDE-Q global score  $\geq 3,5$ ) were emailed by a clinical psychologist involved in the study offering to provide the contact details of the closets National Health Service ED centre. Data from

the general population was collected through an online questionnaire after participants were accepted to take part in the study and passed a CAPTCHA© test to avoid collecting data from online automated response bots. Participants from the outpatient clinic were enrolled in the study at their admission to the clinic and included if they presented with a diagnosis of AN, BN, BED, or OSFED, established by a psychiatrist or qualified clinical psychologist. Data were collected through questionnaires provided as part of the routine baseline clinical evaluation carried out prior to start receiving treatment. In both cases, prior to data collection all participants were given a Research Informed Consent Form explaining in detail the study rationale and procedures, and were given the contact details of the researchers involved to get in touch with and ask questions at any point during their participation in the study.

### 3.3.2. Inclusion and exclusion criteria

Inclusion criteria for the healthy controls (HC) subsample were the following:

- (a) between 18 and 65 of age;
- (b) no prior or current diagnosis of any ED according to ICD-11 (World Health Organization, 2019) or DSM-5 (American Psychiatric Association, 2013) diagnostic criteria;
- (c) not having previously received or being currently receiving treatment for ED symptoms;
- (d) an EDE-Q global scale score below 2.3

Exclusion criteria for the HC sample were the following:

- (a) being below 18 or above 65 of age;
- (b) Having a lifetime diagnosis of any ED in line with the ICD-11 (World Health Organization, 2019) or DSM-5 (American Psychiatric Association, 2013) diagnostic criteria;
- (c) having previously received or being currently receiving treatment for ED symptoms.
- (d) not giving or withdrawing consent to take part in the study at any point.
- (e) an EDE-Q global scale score  $\geq 2.3$



Inclusion criteria for the at risk subsample (RK) were the following:

- (a) between 18 and 65 of age;
- (b) no prior or current diagnosis of any ED according to ICD-11 (World Health Organization, 2019) or DSM-5 (American Psychiatric Association, 2013) diagnostic criteria;
- (c) not having previously received or being currently receiving treatment for ED symptoms;
- (d) an EDE-Q global scale score  $\geq 2.3$  and  $\leq 3.5$

Exclusion criteria for the RK subsample were the following:

- (a) being below 18 or above 65 of age;
- (b) Having a lifetime diagnosis of any ED in line with the ICD-11 (World Health Organization, 2019) or DSM-5 (American Psychiatric Association, 2013) diagnostic criteria;
- (c) having previously received or being currently receiving treatment for ED symptoms.
- (d) not giving or withdrawing consent to take part in the study at any point.
- (e) an EDE-Q global scale score  $\leq 2.3$  or  $\geq 3.4$

Inclusion criteria for the acute subsample (AC) were the following:

- (a) between 18 and 65 of age;
- (b) no prior or current diagnosis of any ED according to ICD-11 (World Health Organization, 2019) or DSM-5 (American Psychiatric Association, 2013) diagnostic criteria;
- (c) not having previously received or being currently receiving treatment for ED symptoms;
- (d) an EDE-Q global scale score  $\geq 3.4$

Exclusion criteria for the AC subsample were the following:

- (a) being below 18 or above 65 of age;
- (b) Having a lifetime diagnosis of any ED in line with the ICD-11 (World Health Organization, 2019) or DSM-5 (American Psychiatric Association, 2013) diagnostic criteria;

- (c) having previously received or being currently receiving treatment for ED symptoms.
- (d) not giving or withdrawing consent to take part in the study at any point.
- (e) an EDE-Q global scale score  $\leq 3.4$

Inclusion criteria for the ED clinical population (CP) were the following:

- (a) between 18 and 65 of age;
- (b) presence of an ED symptomatology of sufficient severity to warrant an ED diagnosis of either AN, BN, BED, or OSFED as established by a certified professional (psychiatrist, clinical psychologist) justifying the admission to the outpatient clinic;
- (c) Not having received yet treatment as part of their admission;
- (d) Established illness duration below 7 years.

Exclusion criteria for the ED clinical population (CP) were the following:

- (a) being below 18 or above 65 of age;
- (b) not having yet being assessed by a certified professional (psychiatrist, clinical psychologist) for ED symptoms, or severity of symptomatology not being confirmed to be at clinically relevant severity yet;
- (c) Currently receiving treatment for ED symptoms as part of their admission to the outpatient clinic.
- (d) established illness duration longer than 7 years, or no information on illness duration available
- (e) not giving or withdrawing consent to take part in the study at any point

Participants who did not meet all of the inclusion criteria or met any one of the exclusion criteria for their respective group were excluded from the study.

### 3.3.3. Ethics committee approval and data management

The study protocol received the approval of the Bioethics Research University Committee of the University of Bologna with protocol number 0061318 on the 15<sup>th</sup> of March 2021.

All data collected from the HC sample was downloaded from the data collection platform (Qualtrics) directly to an encrypted university online drive, where it has been stored for the full duration of the study and for as long as necessary to comply with European GDRP regulations 101/2018. As the European Qualtrics servers are located within European territory, no data transfer agreement was necessary.

All physical copies of the clinical research forms (CRFs) collected from the CP sample of the study are stored in a locked cabinet and accessible only to authorized staff within the outpatient clinic premises. All data pertaining to study participants has been securely stored for the full duration of the study and will be kept for as long as necessary to comply with European GDRP regulations 101/2018. Electronic versions of the CRFs are securely stored on an encrypted university online drive which has been stored for the full duration of the study and for as long as necessary to comply with European GDRP regulations 101/2018.

For the whole duration of the study, any data has been accessed only by authorized researchers or clinical staff, and used in anonymized form for research purposes only, in line with the appropriate data uses approved by the Bioethics Research University Committee of the University of Bologna.

#### 3.3.4. Measures

Data from both general population and CP participants groups were collected using the following questionnaires:

**Sociodemographic questionnaire:** A sociodemographic questionnaire collecting general information such as age, biological sex, height, weight, previous history of ED, and previous history of mental health conditions.

**Eating Disorders Examination Questionnaire (EDE-Q)** (Fairburn & Beglin, 1994) (Italian validation by Calugi et al., 2017) to evaluate the spectrum and intensity of ED symptoms according to DSM 5 criteria. It is a 36-item self-report measure of general ED symptoms and features, comprising four sub-scales (restraint, eating concern, shape concern, and weight concern) plus a global score. The Italian version of EDE-Q demonstrated excellent internal consistency (Cronbach's  $\alpha = .94$ ) and test-retest reliability (Spearman's  $\rho = .80$ ). EDE-Q subscales each demonstrated good discriminant validity against collateral measures for mood, BMI and self-esteem (Grilo et al., 2015)

**Attitudes and Beliefs Scale 2-Short form (ABS2-SF)** (DiGiuseppe et al., 2021) for assessing irrational beliefs (IBs). It is a 24-item self-report scale investigating rational and irrational beliefs. Items are divided between Irrationality and Rationality scales, evaluating various cognitive aspects. The ABS2-SF has demonstrated excellent internal consistency (Rationality scale's Cronbach's  $\alpha = .86$ ; Irrationality scale Cronbach's  $\alpha = .87$ ). Correlational analyses shown good discriminant validity when compared with Outcomes Questionnaire (Lambert et al., 2004) and Psychiatric Disorders Screening Questionnaire (Zimmerman & Mattia, 2001) scales conceptually related to irrational beliefs, while not correlating with other sub-scales from the same instruments (DiGiuseppe et al., 2021).

**Acceptance and Action Questionnaire-II (AAQ-II)** (Bond et al., 2011) (Italian validation by Pennato et al., 2013) for assessing psychological flexibility. It consists of 10 items self-report measure to assess experiential avoidance/psychological flexibility. The Italian version of AAQ-II shows good internal consistency (Cronbach's  $\alpha = .53$ ) and stability over time (Spearman's  $\rho = .61$ ). Evidence for AAQ-II discriminant validity has been demonstrated by virtue of the non-significant correlation with social desirability (Bond et al., 2011), measured using the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1960).

**Mental Health Continuum-Short Form (MHC-SF)** (Keyes et al., 2008) (Italian validation by Petrillo, Capone, Caso, & Keyes, 2014) for assessing general well-being. It is a 14-item self-report assessment, comprising three scales measuring psychological, emotional and social well-being. The Italian version of MHC-SF demonstrated excellent internal consistency (Cronbach's  $\alpha = .86$ ) and stability over time yet sensitivity to change at crossover path analyses ( $\beta = .31$ ). Available studies on discriminant validity of MHC-SF in relation to measures of psychopathology shown adequate ability of the scale to discriminate between mental health well-being and mental illness (Lamers et al., 2011).

**Depression Anxiety Stress Scales (DASS-21)** (Henry & Crawford, 2005) (Italian validation by Bottesi et al., 2015) for assessing symptoms of depression, anxiety and stress. It is a 21-item self-report comprising three sub-scales: Depression Anxiety, and Stress. The Italian version of DASS-21 has shown excellent internal consistency (Cronbach's  $\alpha = .90$ ) and stability over time (Spearman's  $\rho = .76$ ). Adequate discriminant validity for the three subscales has been reported (Lee, 2019).

### 3.3.5. Data analysis procedures

Prior to any statistical analysis, various data cleaning procedures were carried out on the general population (n=1186) dataset. For the data obtained from the general population, duplicate entries were removed (n= 42) as a first step. Subsequently, data from participants who did not complete all the questionnaires were removed. This did not apply to participants with occasional missing data, but rather to participants who at some point completely stopped answering questionnaires and never resumed their participation in the data collection (n= 353). This led to a final sample size of 792 cases. No such procedure was necessary for the CP (n= 139) sample, as no duplicate data and no dropout from the study were recorded.

From the cleaned general population dataset (n= 792), three subpopulations were identified to be used as samples for network analysis procedures: a group of healthy controls, a subset of the population deemed at very high risk / at the prodromal stage of ED, and an acute group. The at-risk/prodromal subgroup (RK; n= 128) was selected based on their EDE-Q total score, adopting a similar approach to that used in Melisse et al. (2021): scores higher than 2.3 and below 3.4 were selected as threshold for presence of high risk/prodromal ED, based on the EDE-Q norms for the Italian population (Calugi et al., 2017) and consistently with the approach utilised in Melisse et al. (2021) and the reference threshold scores in Bryant et al. (2021). Participants with an EDE-Q total score of 3.5 or above were considered at the acute stage of the disorder and grouped within the acute subsample (AC). The remaining participants from the general population who did meet inclusion criteria for the RK or AC subsamples were included in the healthy control group (HC; n=527).

The full general population sample (n=792) presented 20.4% of data missing at random, while the CP sample presented 8% of data missing at random. In both cases, multivariate imputation by chained equations (MICE) were conducted with 10 cycles of imputations (Raghunathan et al., 2002) for each dataset using the *mice* R package (van Buuren & Groothuis-Oudshoorn, 2011), after which complete-

data was fit to each imputed dataset, and the results combined to obtain estimates that properly account for the missing data.

Descriptive analyses for age, gender distribution and BMI were conducted for all 4 subsamples of the study.

Prior to network estimation procedures, the *goldbricker* function from the *NetworkTools* R package (Jones, 2020) to control for topological overlaps of network nodes. In this study, we set the limit at less than 50% correlations of nodes significantly different from each other.

Individual network model estimation for each sample (HC, RK, AC, CP) was conducted using the *ggmModSelect* function in the *bootnet* R package (Epskamp et al., 2018). The *ggmModSelect* algorithm is a nonregularized algorithm for network estimation. This algorithm generates a range of regularized network structures and, subsequently, generates as many structures without applying regularization. This is done through maximum likelihood estimation, in which only the edges that were nonzero in the regularized networks are estimated to be nonzero. After this process, the *ggmModSelect* algorithm continues optimization by stepwise adding and removing edges until the EBIC criterion is optimized (Isvoranu & Epskamp, 2023). Node strength centrality and bridge strength were estimated for all nodes of all the networks. Strength centrality (STR) represents the absolute sum of all edges of a node and gives a measure of a node's overall involvement in the network (McNally, 2016). Bridge strength (BS) centrality represents the strength of a symptom with symptoms in another community and was estimated with the bridge function of the *network* R package (Jones, 2020). Node communities were determined based on the different instruments used, so that, for example, the four EDE-Q subscales were considered part of the EDE-Q nodes community, the two scales for Rational and Irrational beliefs of the ABS2-SF were considered part of the ABS2-SF nodes community, and so forth.

The accuracy and replicability of the resulting network models were examined by computing (a) the strength centrality stability coefficient (CS-coefficient); (b) accuracy of edge weights using bootstrapped confidence intervals (CI); and (c) bridge strength stability coefficient (BS-coefficient). The stability of all networks was assessed with the *bootnet* R package (Epskamp et al., 2018). Specifically, (a) the stability of the strength centrality was estimated using the CS-coefficient with 2500 bootstraps; (b) accuracy of the edge weights was estimated by drawing nonparametric bootstrapped 95% CIs with 2500 bootstraps, and (c) the stability of the bridge strength centrality was estimated using the BS-coefficient with 2500 bootstraps. Coefficients reflect the maximum proportion of cases that can be dropped such that the correlation between original centrality indices and the reduced sample is at least .70. Coefficients between .25 and .50 are considered acceptable, coefficients above .50 and below .70 are considered good, and coefficients above .70 are considered excellent (Epskamp et al., 2018).

The network models obtained from the 4 groups (HC, RK, AC, CP) were compared using the *NCT* function of the *NetworkComparisonTest* R package (Van Borkulo et al., 2019). The NCT is a 2-tailed permutation test in which the difference between 2 groups is calculated repeatedly for randomly regrouped individuals. This can be used to test the observed difference between the groups (Van Borkulo et al., 2015). First, the index of similarity between network edges and centrality indices will be obtained, and then between-group differences will be obtained using NCT, with  $p < 0.05$  indicating a significant difference between the networks. All network comparison tests were conducted on a base of 1000 permutations and investigated network invariance (possible edge weight differences) and global strength invariance (possible difference on the absolute sum of network edge weights). The overall network structure, global strength centrality, differences between individual clusters, and significant differences in individual edges between different networks were examined, with p-values  $< 0.05$  indicating a significant difference where appropriate.



### 3.4. Results

#### 3.4.1. Study 1

The final subsamples consisted of 527 participants in the HC group, 128 participants in the RK group, 118 participants in the AC group, and 139 participants in the CP group. Sociodemographic analyses revealed a majority of female participants in all groups (HC: 85.2%; RK: 85.9%; AC:89.9%; CP: 100%). Participants from the various groups also presented similar age means; full breakdown of sex distribution, age and BMI for each group is reported in Table 1.

	Sex % (f; m; nd)	Age (mean $\pm$ sd; min-max)	BMI
HC	85.2; 14; 0.2	26.6 $\pm$ 8.5; 18-67	22.5 $\pm$ 3.9
RK	85.9; 14.1; 0.0	27.0 $\pm$ 8.63; 18-59	23.4 $\pm$ 4.6
AC	89.0; 11.0; 0.0	30.4 $\pm$ 9.8; 19-62	22.8 $\pm$ 4.8
CP	100.0; 0.0; 0.0	24.2 $\pm$ 10.4; 18-63	22.2 $\pm$ 7.3

Table 1 Demographic data for healthy controls (HC), at-risk (RK), acute (AC), and clinical (CP) groups. F: females; m: males; nd: not disclosed. BMI calculated as weight in kilograms divided by height in meters squared.

The *goldbricker* test identified the following nodes as overlapping for EDE-Q\_WEIGHT.CONC and EDE-Q\_SHAPE.CONC (0.09), AAQ-II and ABS2SF\_IRR (0.27), MHC-SF\_PWB and ABS2SF\_RAT (0.36), and DASS-21\_STRS and AAQ-II (0.36). However, as the nodes represented conceptually similar but distinct constructs they were not removed from the network or collapsed in a single node. This section will first focus on the characteristics of the individual network models retrieved for each of the four populations examined in the study. In the second part, the results from the various network comparison tests between network structures will be reported. Larger figures for each of the four network plots and the network comparison plot are provided in the appendix.

### 3.4.1.1. Healthy controls (HC) network model

The network model retrieved from the HC sample data can be observed in Figure 6. The network nodes with the higher STR coefficient were MHC-SF Psychological Well-Being (STR=1.9), EDE-Q

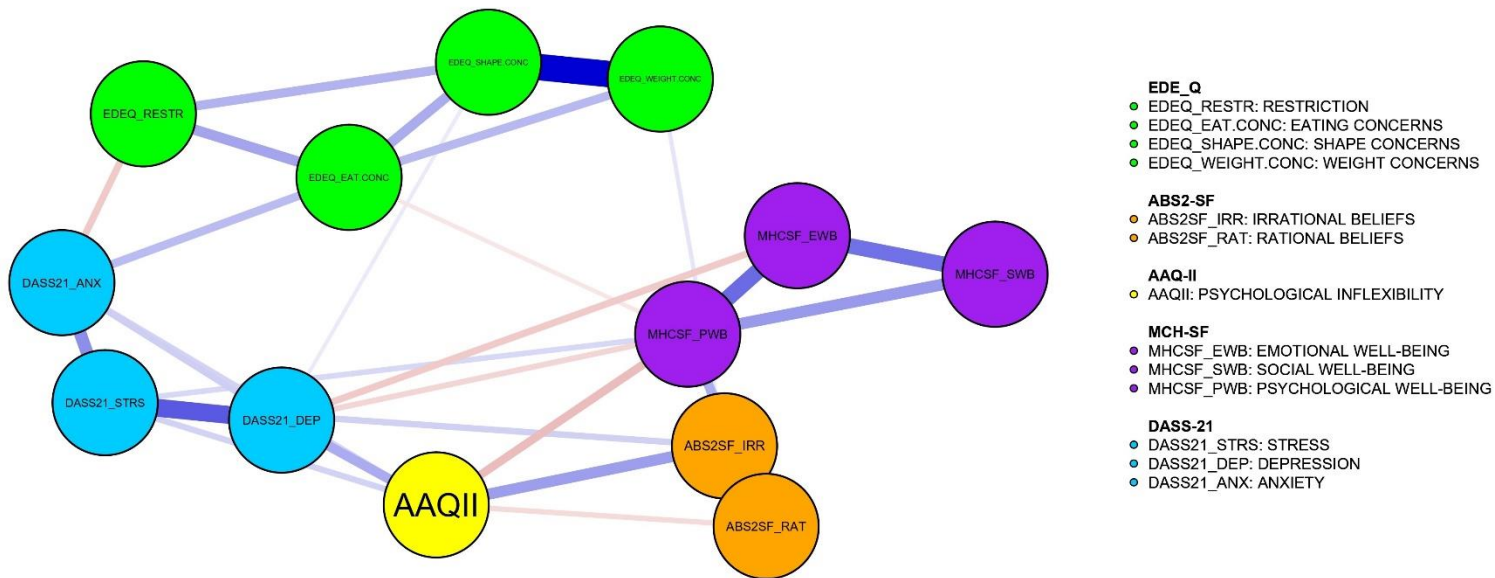


Figure 6 Network model from the HC study sample

Shape Concerns (STR=1.0) and DASS-21 Depression (STR= 0.79). The plot for the strength centrality coefficient of all nodes is reported in Figure 7. The absolute sum of edges strength for the EDE-Q nodes cluster (EDE-Q\_STR) in this population was = 1.55; the absolute sum of edges strength for the MHC-SF nodes cluster (MHC-SF\_STR) in this population was = 1.07; the absolute sum of edges strength for the DASS-21 nodes cluster (DASS-21\_STR) in this population was = 0.89.

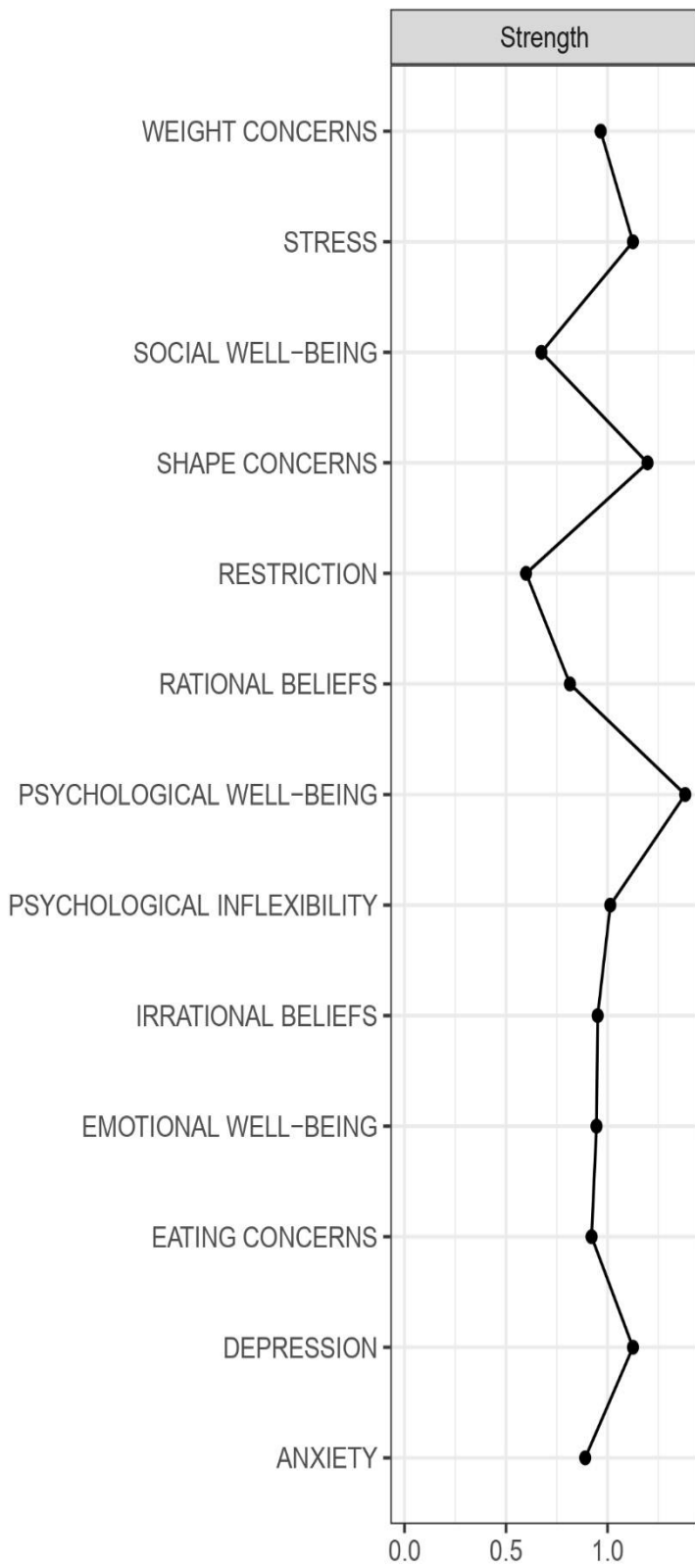


Figure 7 Node centrality plot for the HC sample network

Figure 8 represents the table of significant differences in STR between the nodes of the network, Figure 9 reports significant differences between individual edges, and Figure 10 reports the bootstrapped confidence interval of the HC network edges.

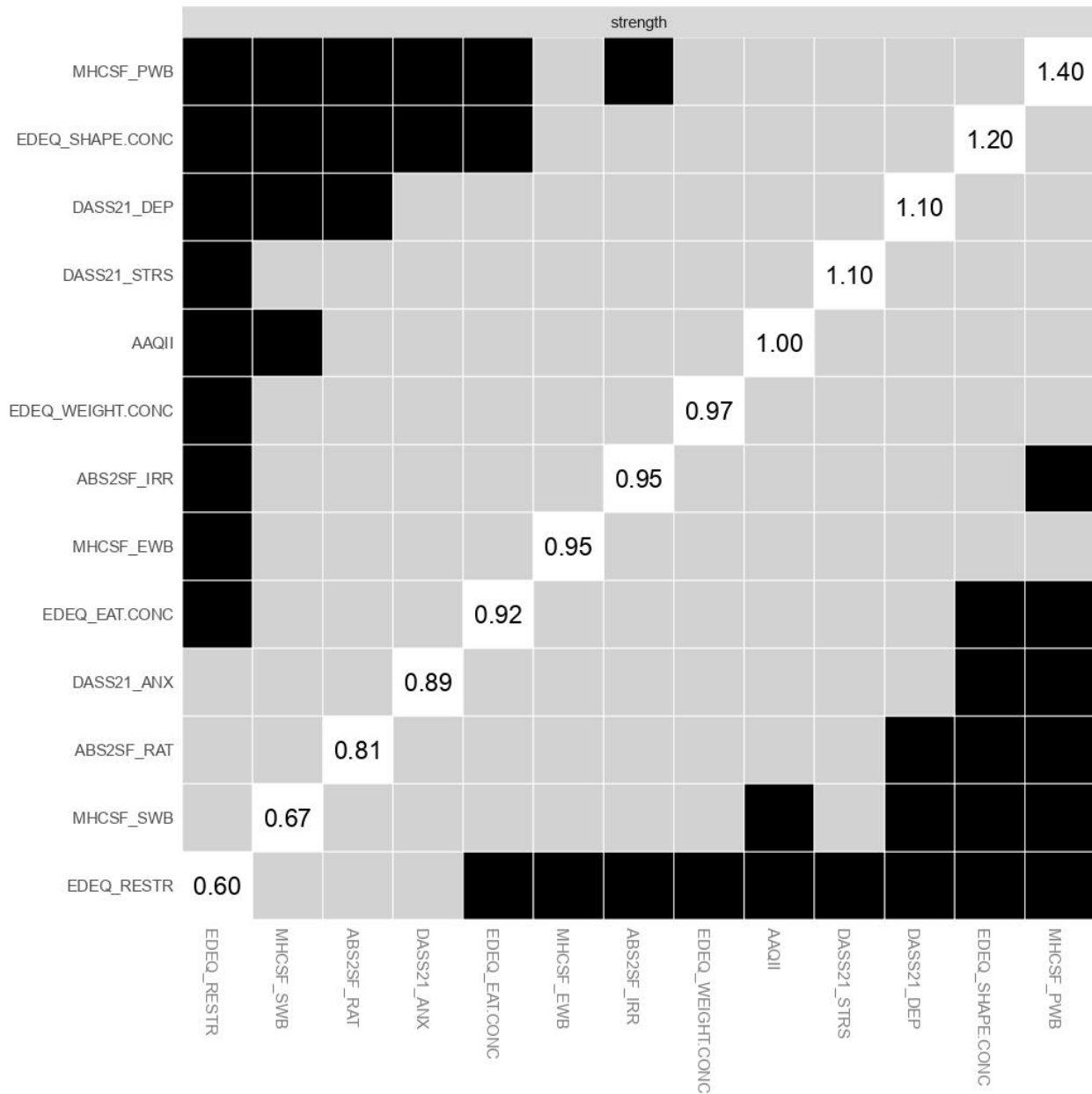


Figure 8 Strength centrality differences between HC network nodes. Black squares indicate a significant difference between the nodes on the X and Y axis.

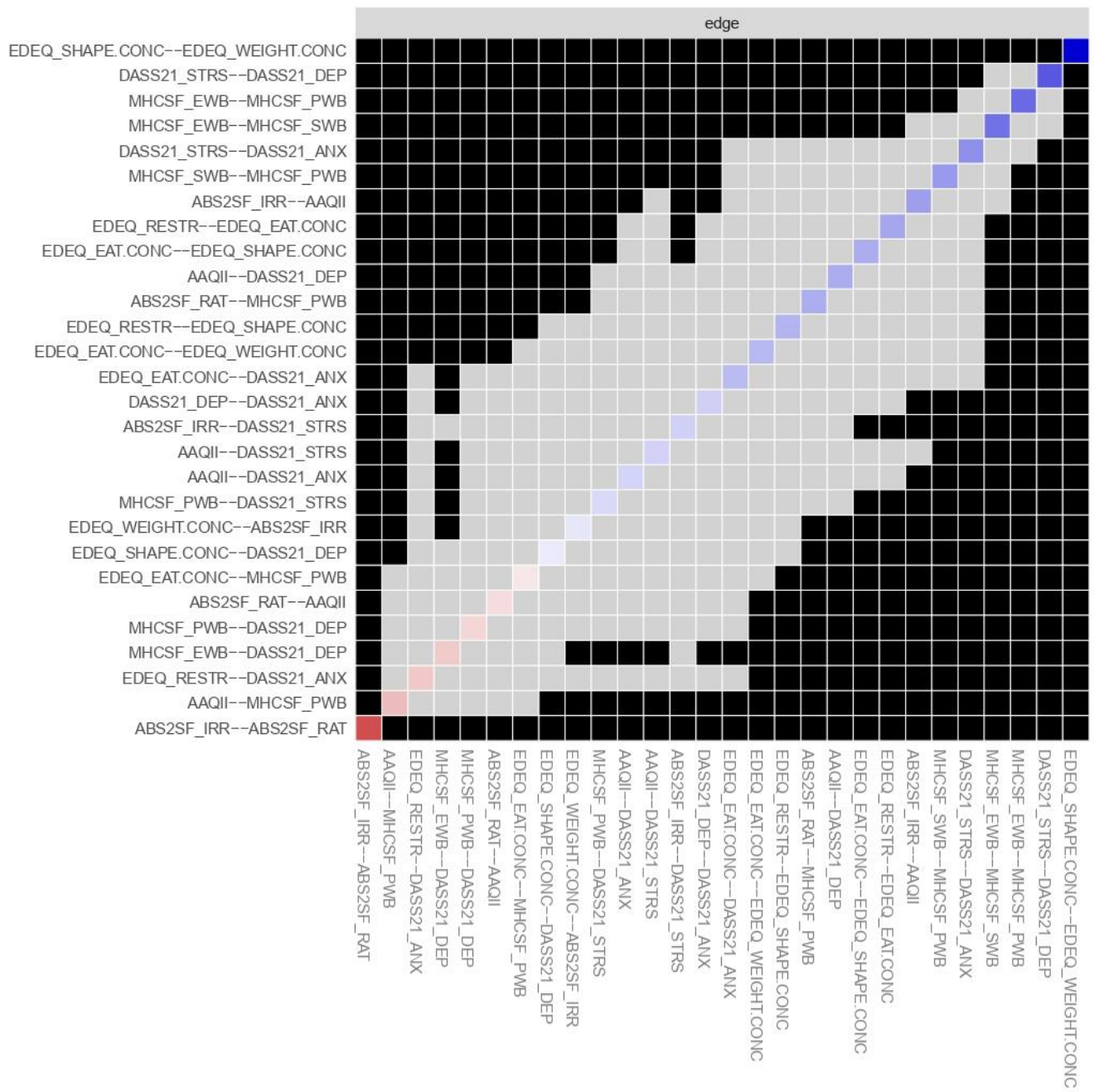


Figure 9 Edge difference for all HC network edges. Black squares indicate a significant difference between the edges on the X and Y axis

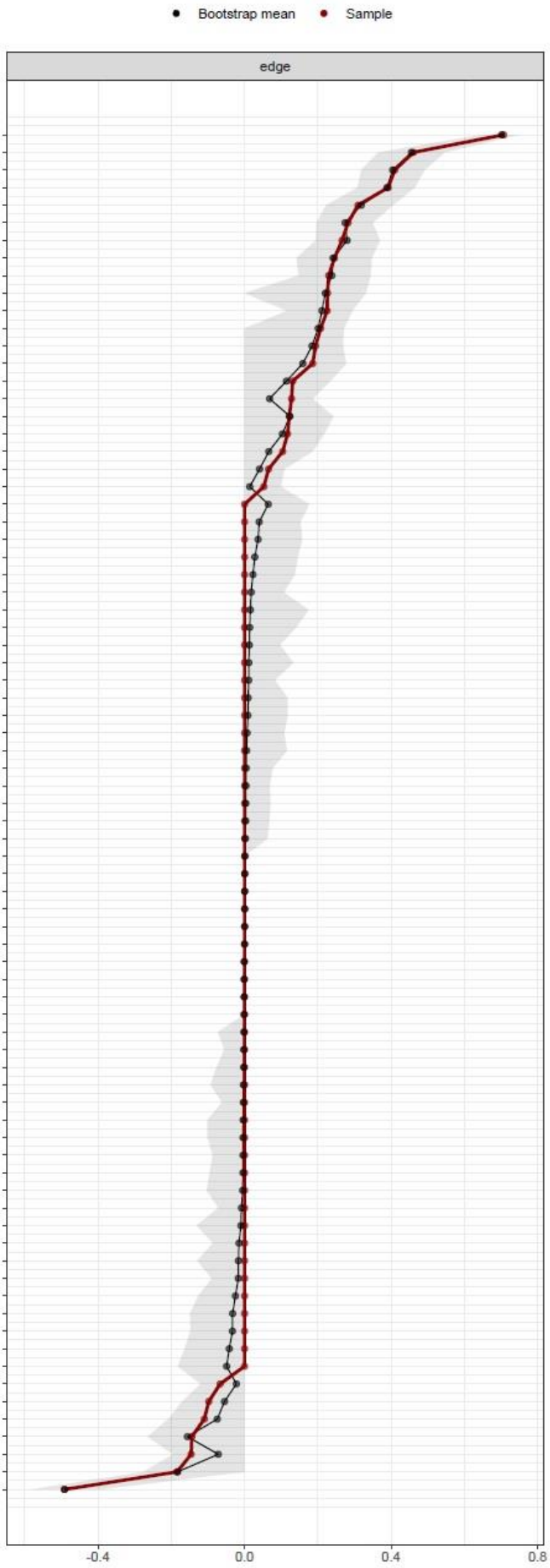


Figure 10 Bootstrapped confidence interval (BCI) of HC network edges.

The network demonstrated good stability for strength centrality at case dropping bootstrap (CS-coefficient = 0.51). Psychological inflexibility appeared as the node with the highest BS (BS=1.0). The nodes with the second and third highest BS were MHC-SF Psychological Well-Being (BS=0.69) and DASS-21 Depression (BS= 0.53) respectively. The plot for the bridge strength coefficient is reported in Figure 11. Bridge strength demonstrated excellent stability (BS-coefficient: 0.75).

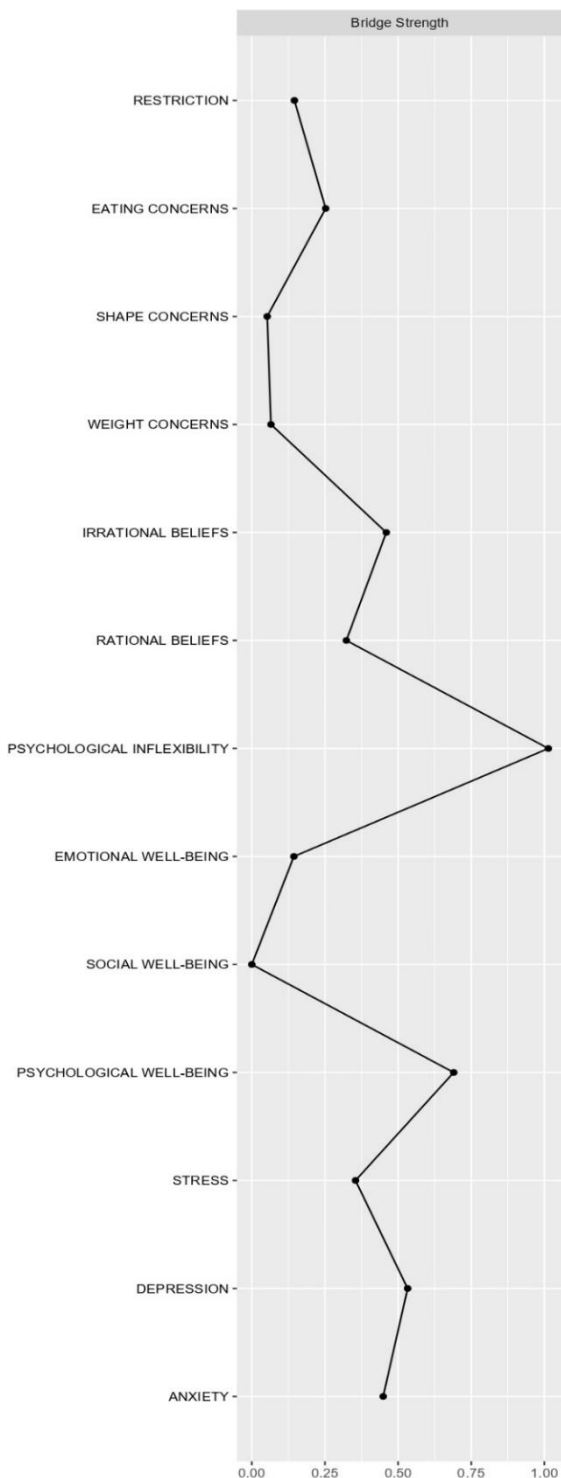


Figure 11 Bridge strength plot for the HC sample network

### 3.4.1.2. At-risk (RK) network model

The network model retrieved from the RK sample data can be observed in Figure 12.

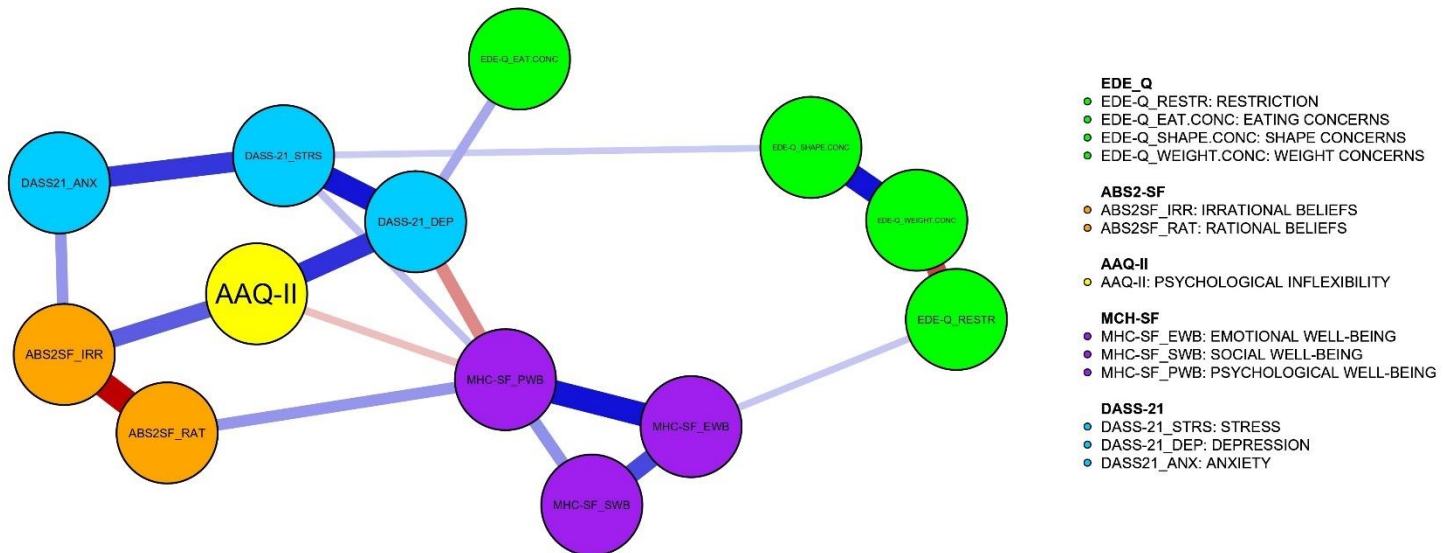


Figure 12 Network model for the RK study sample.

The RK network nodes with the higher STR coefficient were MHC-SF Psychological Well-Being (str=1.68), DASS-21 Depression (str= 1.38), and DASS-21 Stress (str= 0.81). The bootstrap test showed adequate network stability (CS-coefficient = 0.25). The plot for the strength centrality coefficient of all nodes is reported in Figure 13. The absolute sum of edges strength for the EDE-Q nodes cluster (EDE-Q\_STR) in this population was = 0.86; the absolute sum of edges strength for the MHC-SF nodes cluster (MHC-SF\_STR) in this population was = 1.07; the absolute sum of edges strength for the DASS-21 nodes cluster (DASS-21\_STR) in this population was = 0.89.



Figure 13 Node centrality plot for the RK sample network

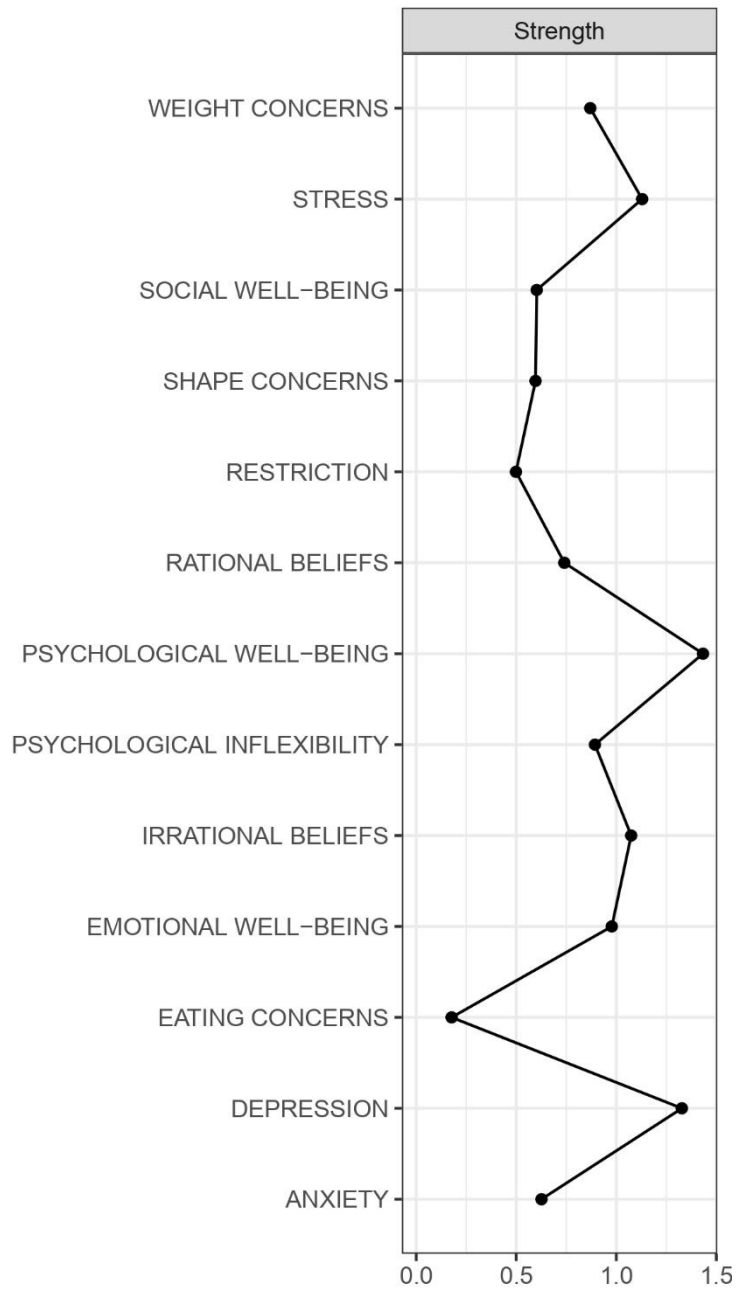


Figure 14 Strength centrality differences between RK network nodes. Black squares indicate a significant difference between the nodes on the X and Y axis.

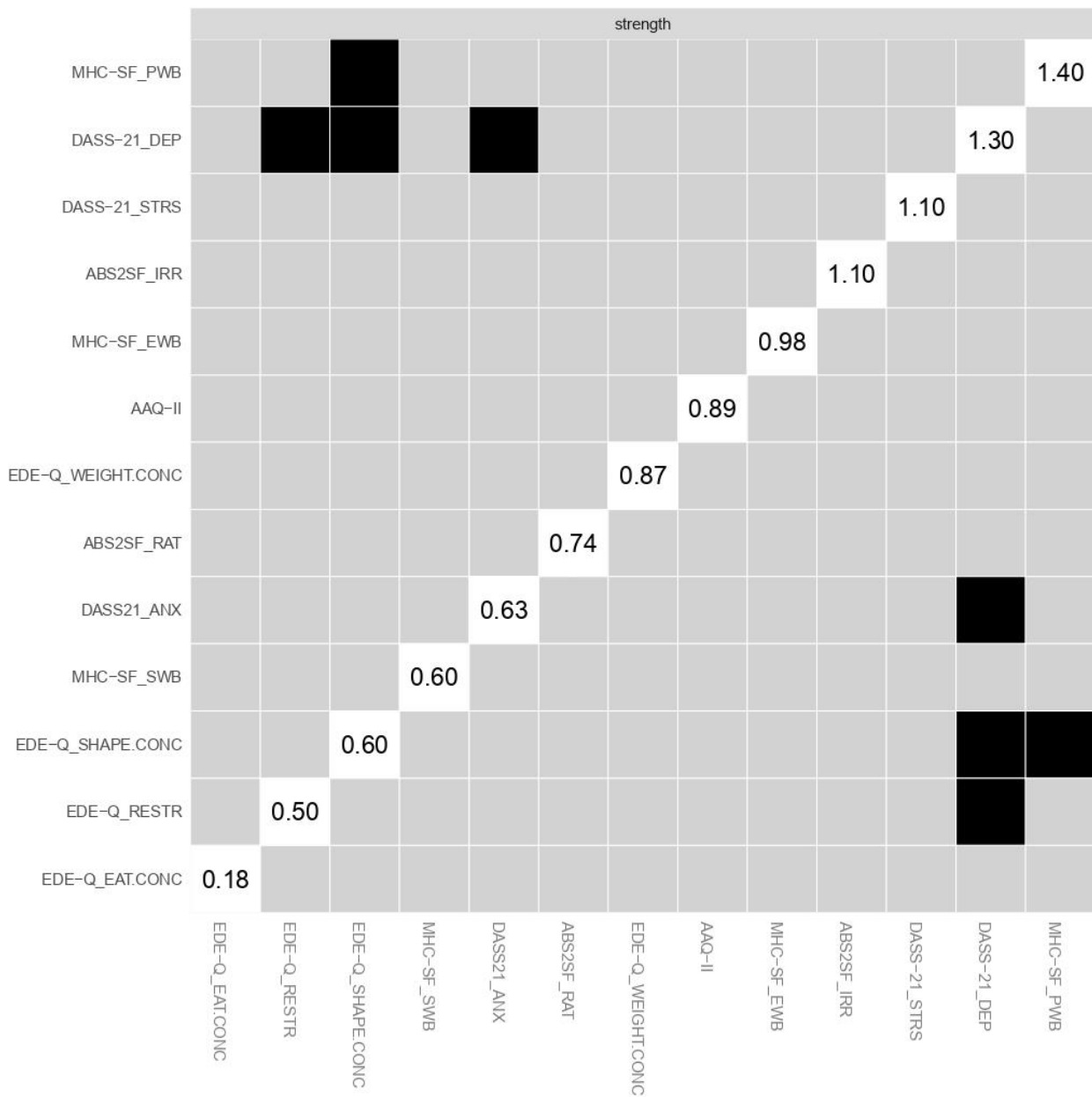


Figure 14 represents the table of significant differences in STR between the nodes of the network, Figure 15 reports significant differences between individual edges, and Figure 16 reports the bootstrapped confidence interval of the HC network edges.

Figure 15 Edge difference for all RK network edges. Black squares indicate a significant difference between the edges on the X and Y axis

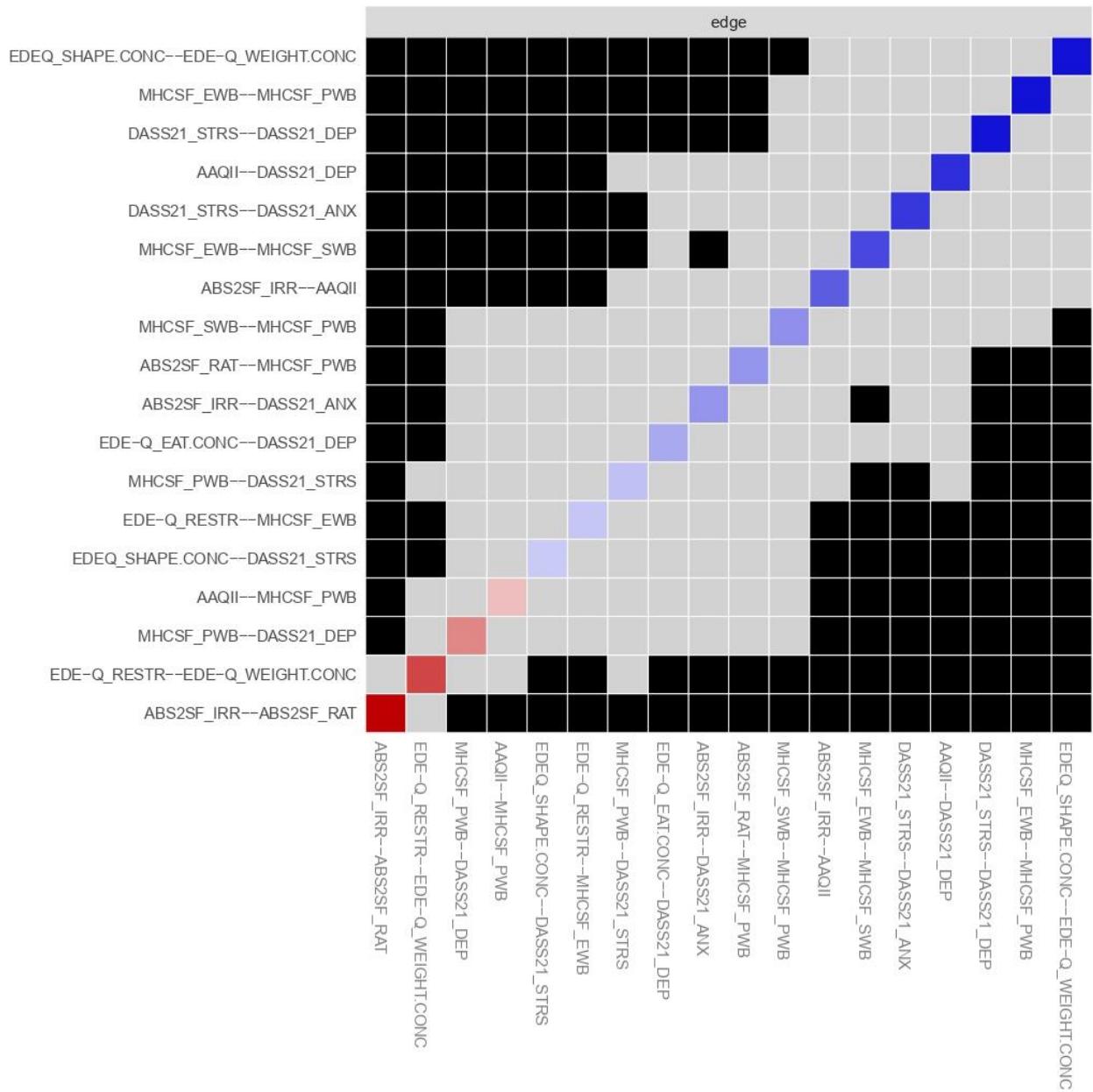
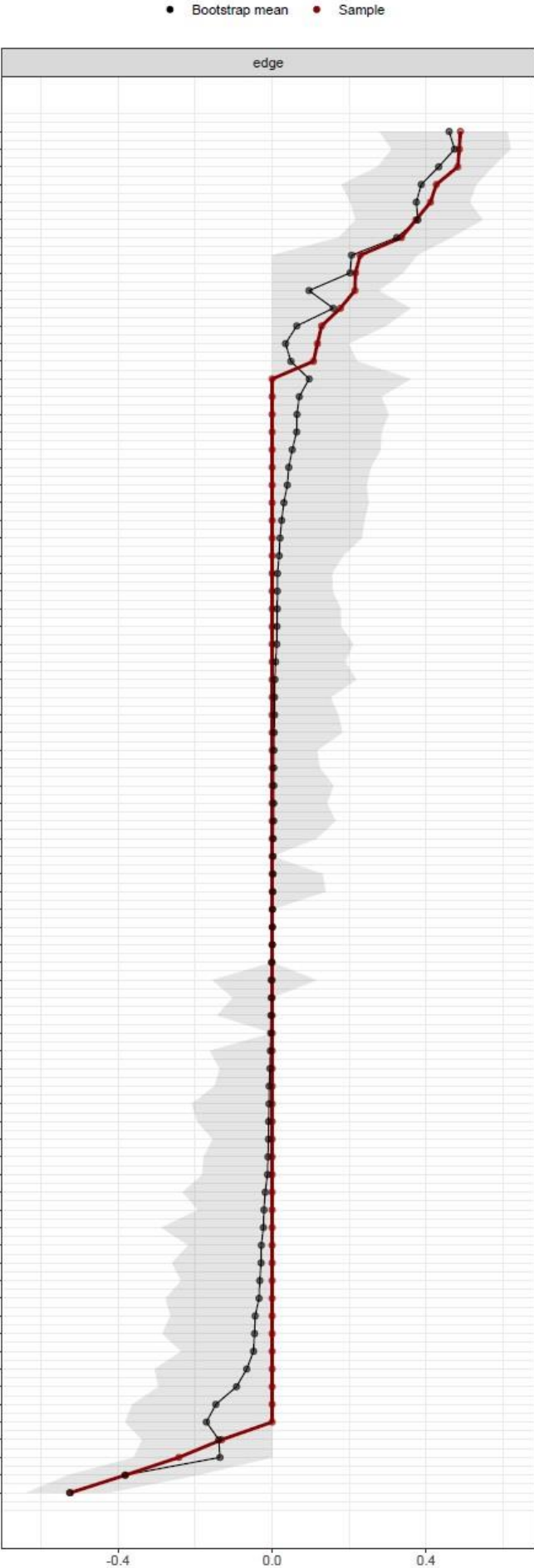
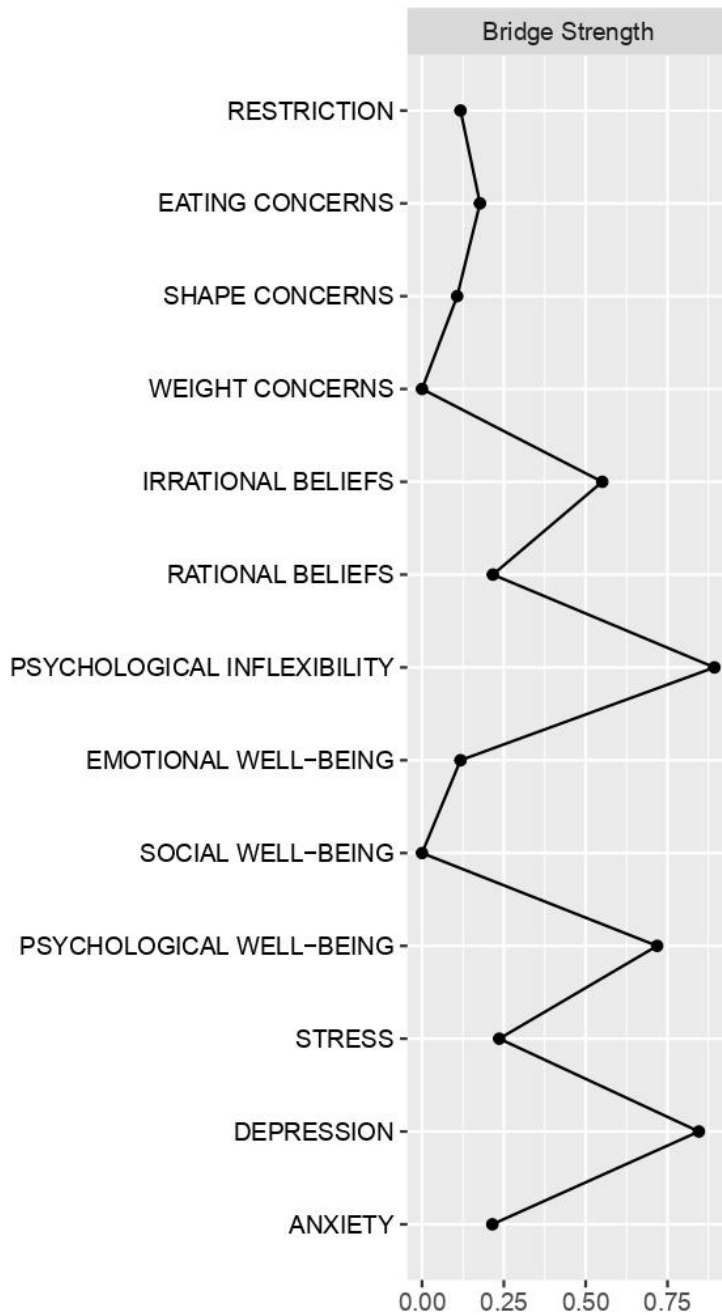


Figure 16 Bootstrapped confidence interval (BCI) of RK network edges.



Again, psychological inflexibility appeared as the node with the highest BS (BS= 0.89). The nodes with the second and third highest BS were DASS-21 Depression (BS=0.84) and MHC-SF Psychological well-being (BS= 0.71) respectively. The plot for the bridge strength coefficient is reported in Figure 17. Bridge strength demonstrated good stability (BS-coefficient: 0.51).

Figure 17 Bridge strength plot for the RK sample network



### 3.4.1.3 Acute population (AC) Network model

The network model retrieved from the AC sample data can be observed in Figure 18

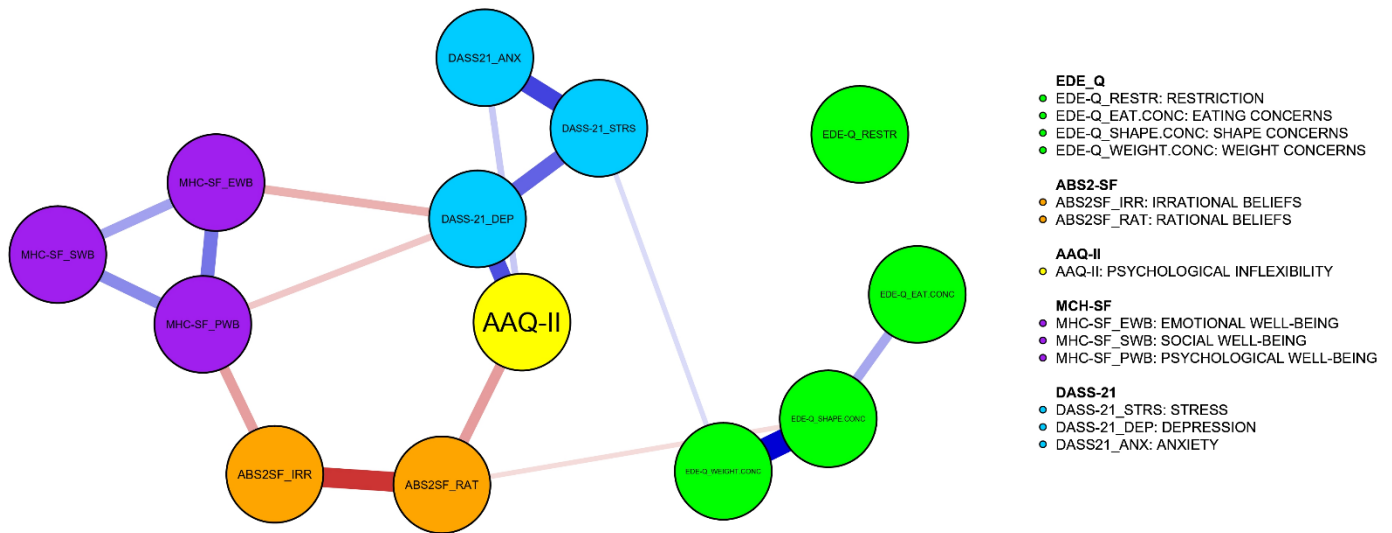
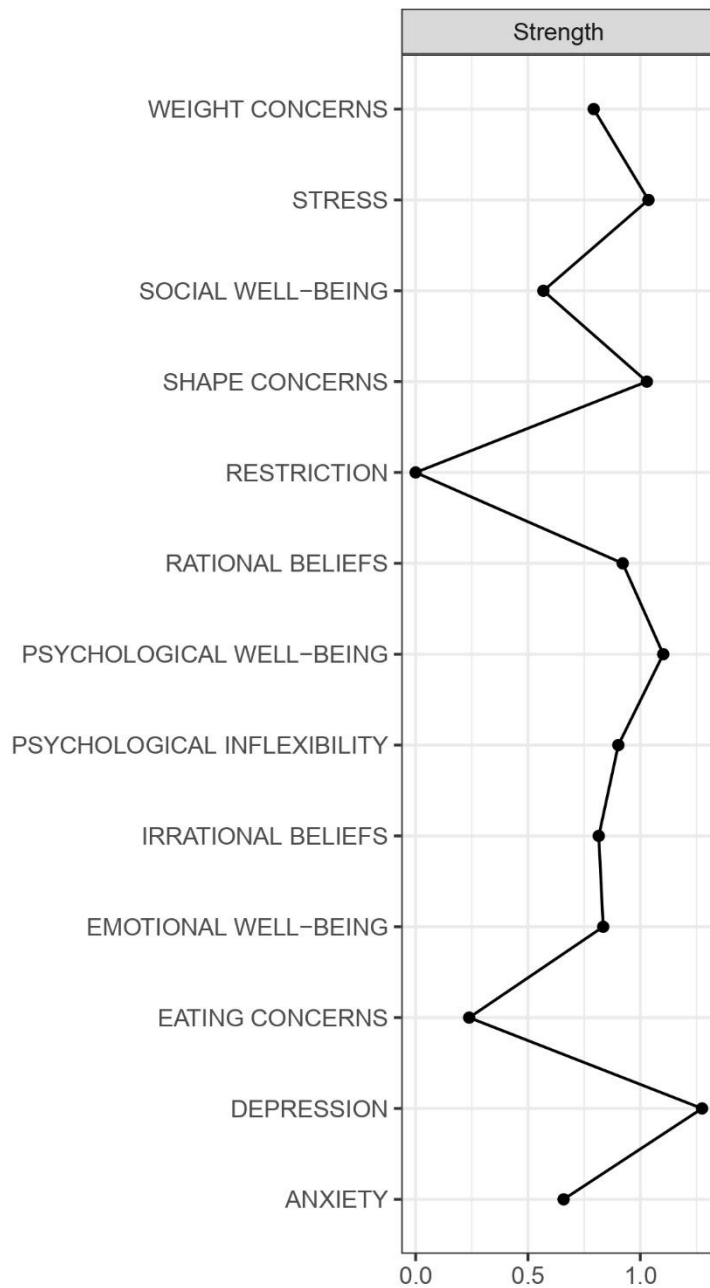


Figure 18 Network model for the AC study sample

The network nodes with the higher strength (str) coefficient were DASS-21 Depression (str= 1.40), MHC-SF Psychological Well-Being (str=0.91), DASS-21 Stress (str= 0.77). The bootstrap test showed good network stability (CS-coefficient= 0.51). The plot for the centrality of all network nodes can be seen in Figure 19. The absolute sum of edges strength for the EDE-Q nodes cluster (EDE-Q\_STR) in this population was = 0.92; the absolute sum of edges strength for the MHC-SF nodes cluster (MHC-SF\_STR) in this population was = 0.93; the absolute sum of edges strength for the DASS-21 nodes cluster (DASS-21\_STR) in this population was = 0.92.

Figure 19 Node centrality plot for the AC sample network



The table of significant differences in STR between the nodes of the network is presented in Figure 20, significant differences between individual edges are reported in Figure 21, and the bootstrapped confidence interval of the AC network edges is reported in Figure 22.

Figure 20 Strength centrality differences between AC network nodes. Black squares indicate a significant difference between the nodes on the X and Y axis

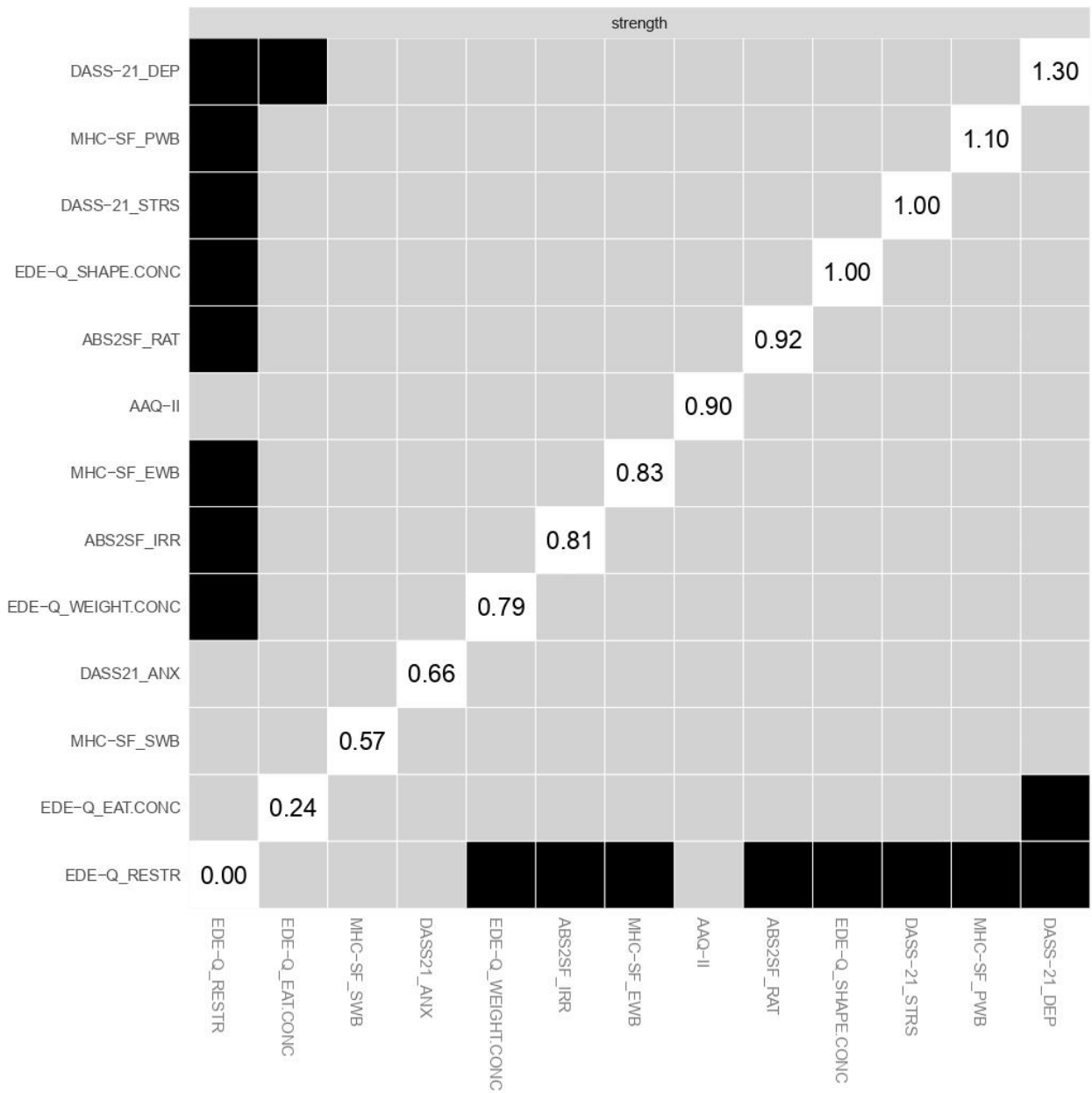




Figure 21 Edge difference for all AC network edges. Black squares indicate a significant difference between the edges on the X and Y axis

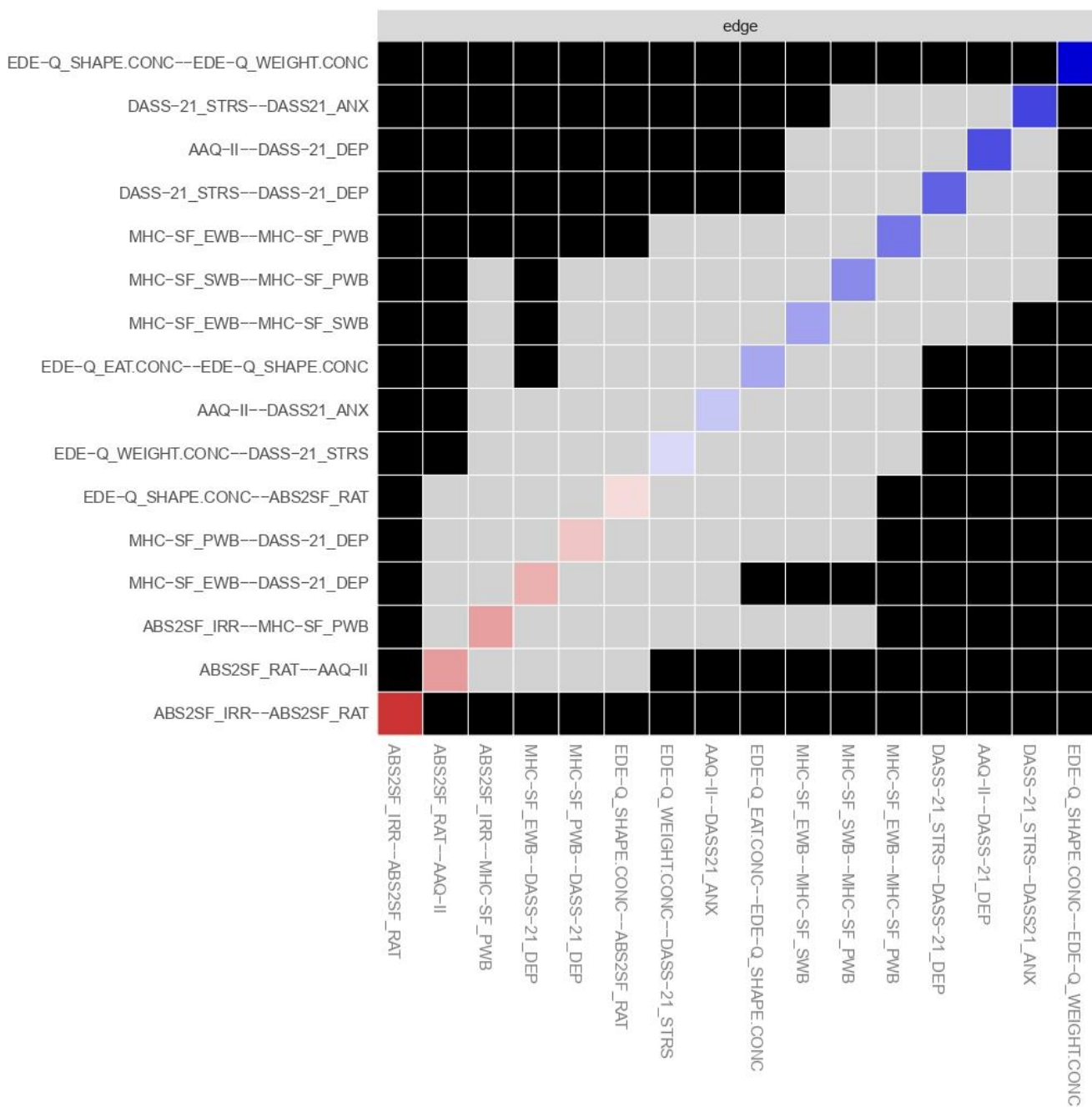
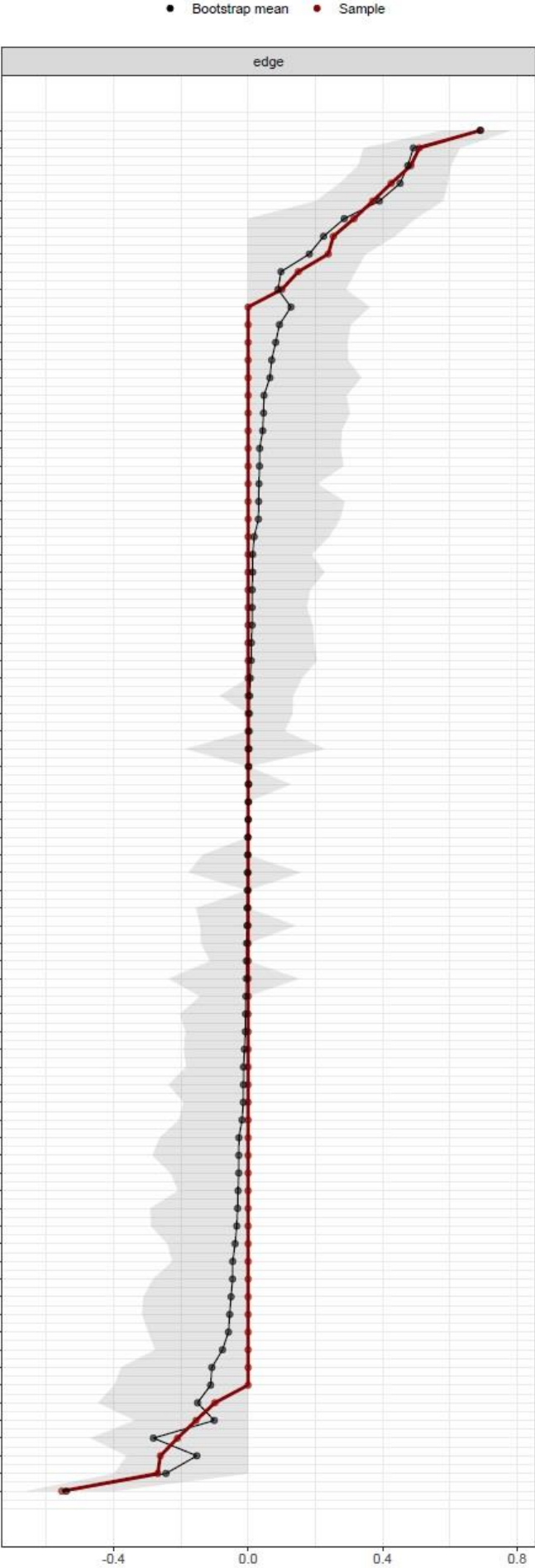
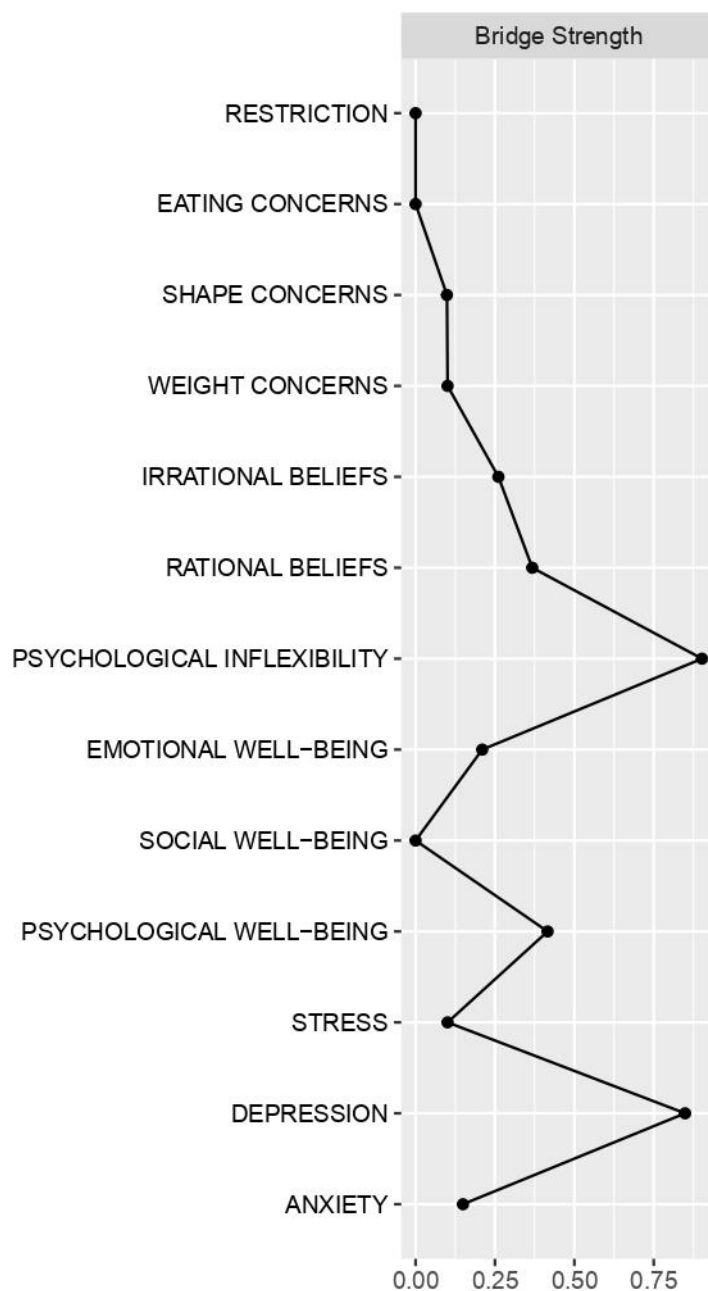


Figure 22 Bootstrapped confidence interval (BCI) of AC network edges.



As with the other bridge analyses, psychological inflexibility appeared as the node with the highest BS (BS= 0.90). The nodes with the second and third highest BS were DASS-21 Depression (BS=0.84) and MHC-SF Psychological Well-being (BS= 0.41) respectively. The plot for the bridge strength coefficient is reported in Figure 17. Bridge strength demonstrated adequate stability (BS-coefficient: 0.44).

Figure 23 Bridge strength plot for the AC sample network.



### 3.4.1.4. Clinical population (CP) Network model

The network model retrieved from the AC sample data can be observed in Figure 24

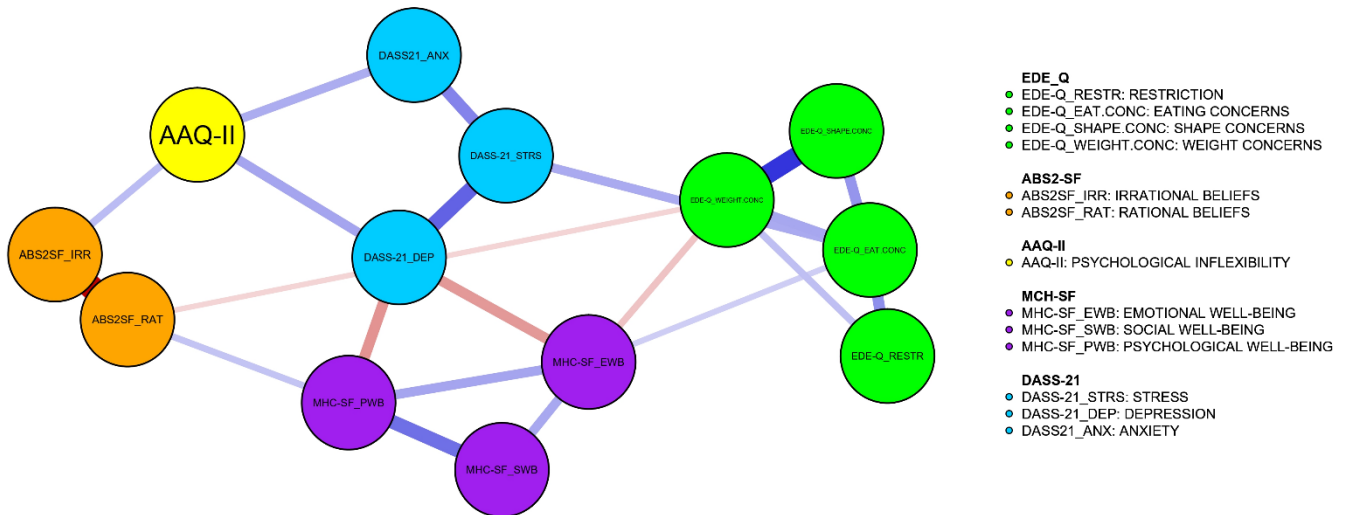
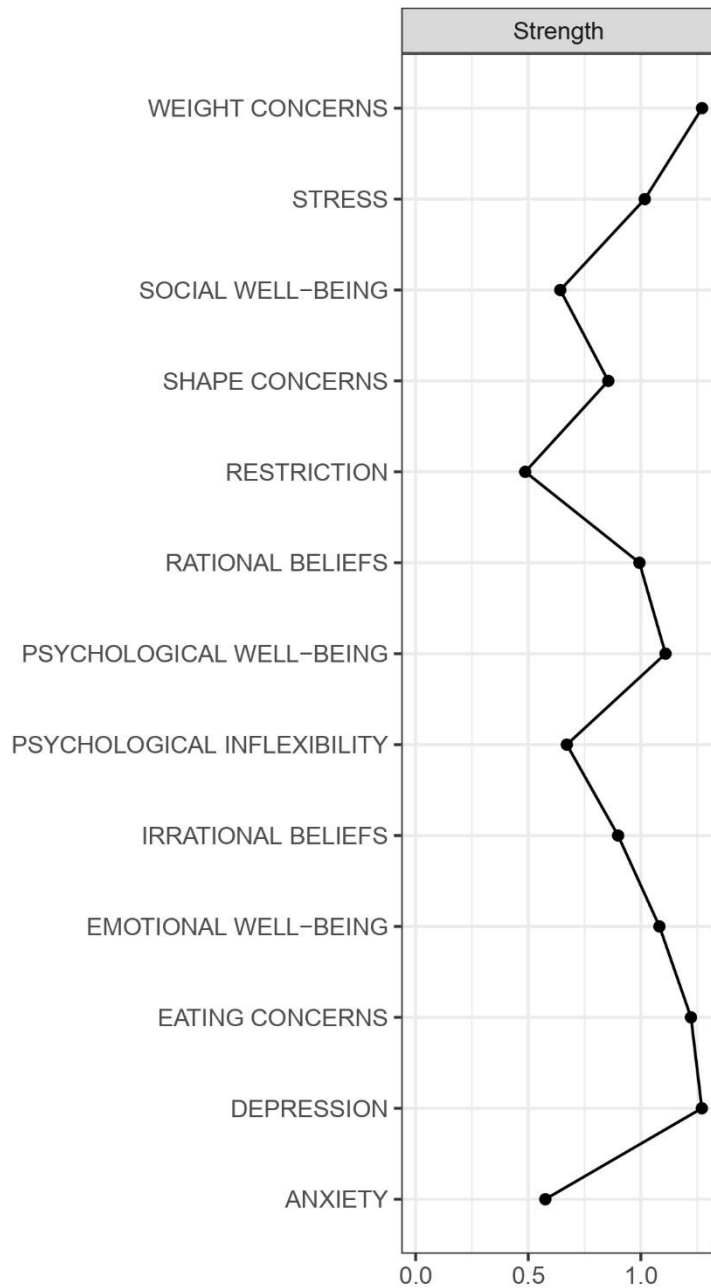


Figure 24 Network model for the CP study sample

The network nodes with the highest STR coefficient were EDE-Q Weight Concerns (str= 1.27), DASS-21 Depression (str= 1.26), and EDE-Q Eating Concerns (str= 1,08). The plot for the centrality of all network nodes can be seen in Figure 25. The absolute sum of edges strength for the EDE-Q nodes cluster (EDE-Q\_STR) in this population was = 1.57; the absolute sum of edges strength for the MHC-SF nodes cluster (MHC-SF\_STR) in this population was =0.88; the absolute sum of edges strength for the DASS-21 nodes cluster (DASS-21\_STR) in this population was = 0.77. The bootstrap test showed adequate network stability (CS-coefficient= 0.28).

Figure 25 Node centrality plot for the CP sample network



The table of significant differences in STR between the nodes of the network is presented in Figure 26, significant differences between individual edges are reported in Figure 27, and the bootstrapped confidence interval of the AC network edges is reported in Figure 28.

Figure 26 Strength centrality differences between CP network nodes. Black squares indicate a significant difference between the nodes on the X and Y axis

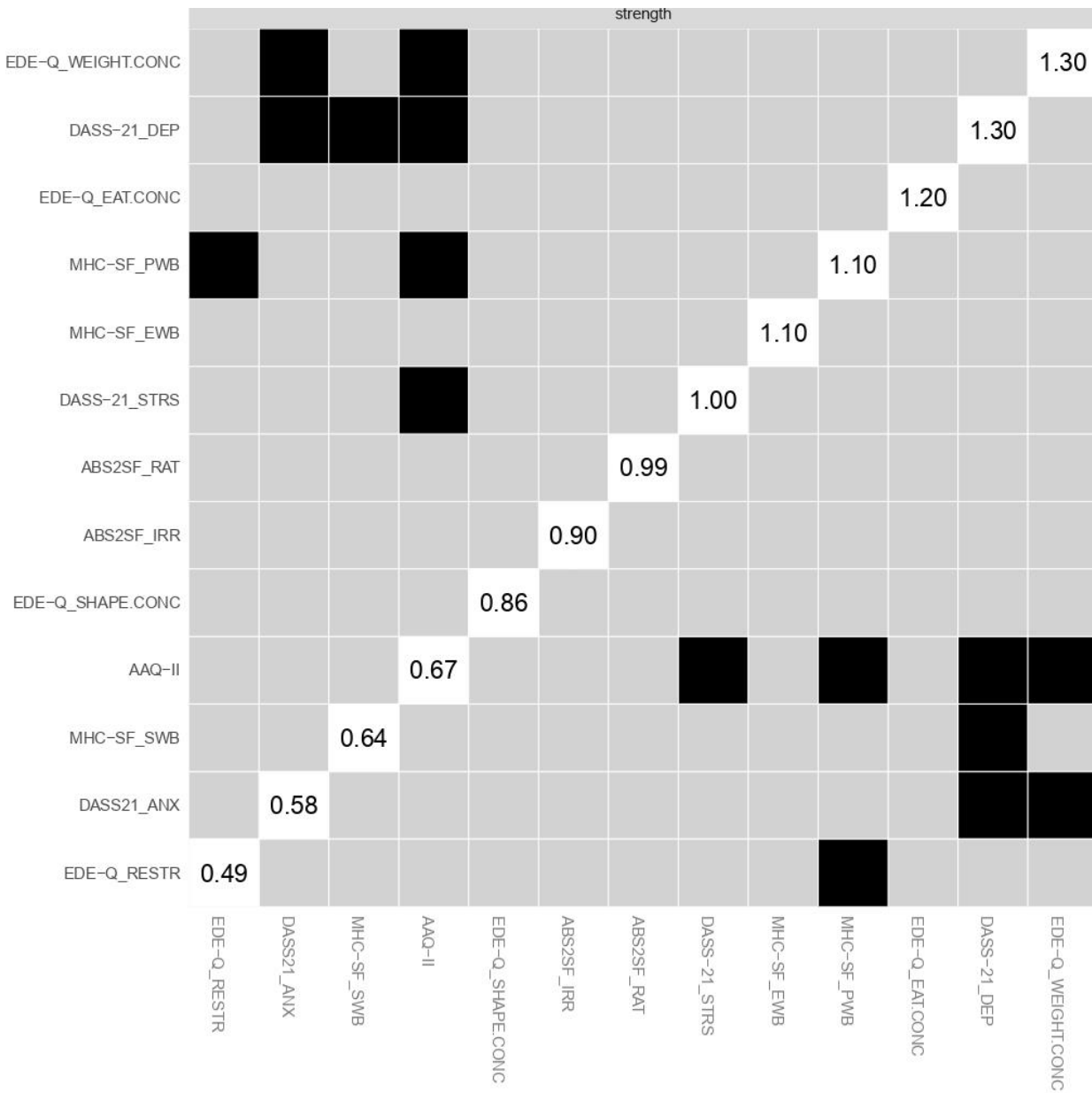


Figure 27 Edge difference for all CP network edges. Black squares indicate a significant difference between the edges on the X and Y axis

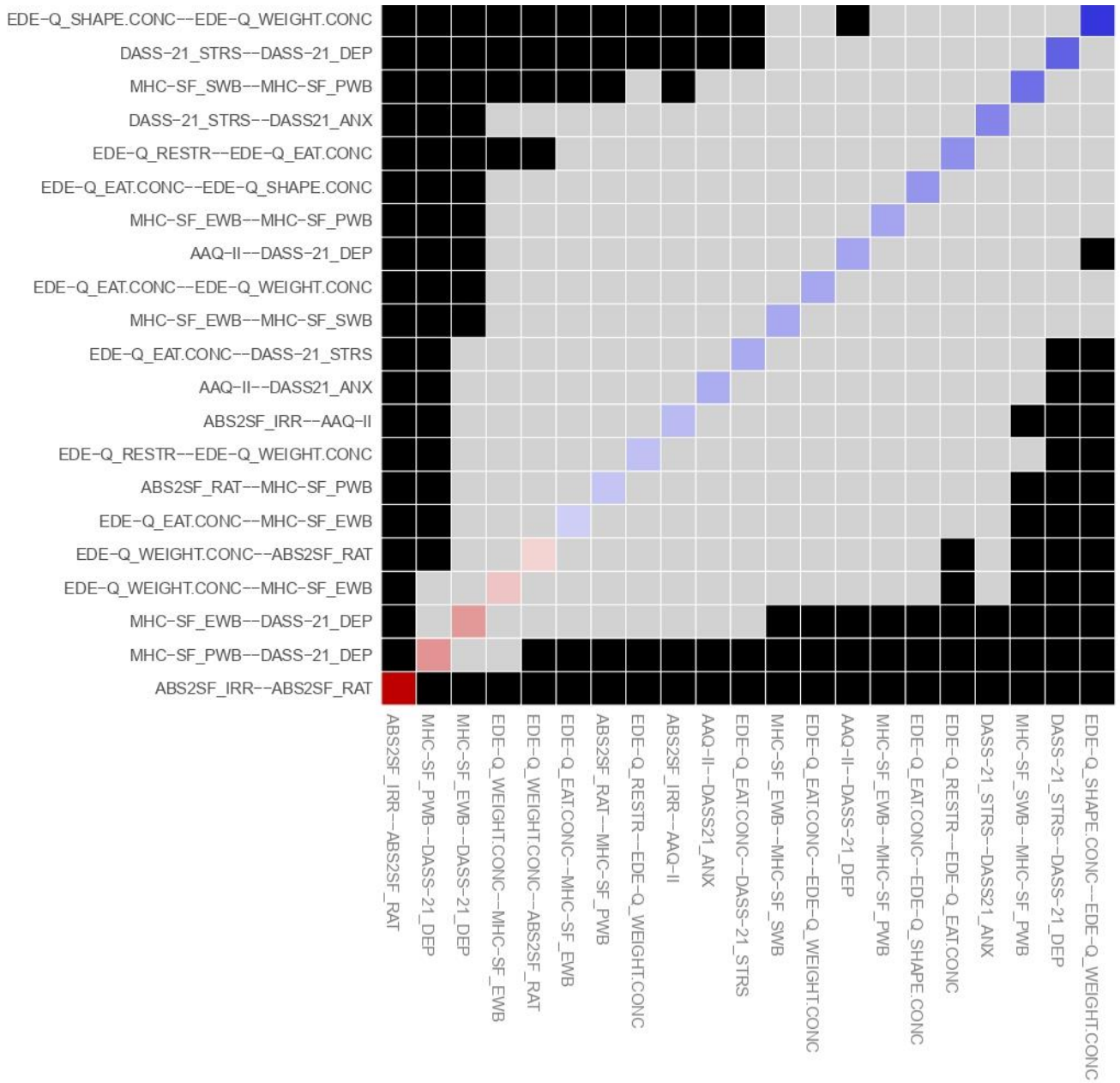
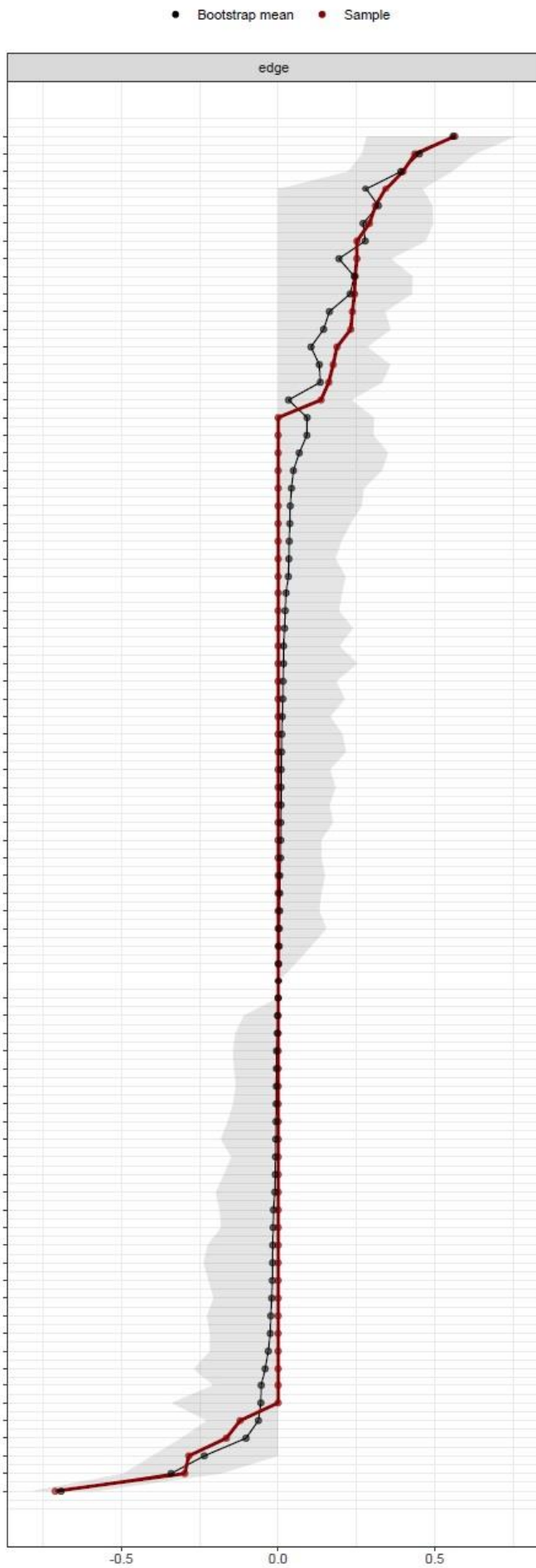


Figure 28 Bootstrapped confidence interval (BCI) of CP network edges.





The node with the highest BS in the network model retrieved from the data of the CP sample was DASS-21 Depression (BS= 0.83). The nodes with the second and third highest BS were AAQ-II Psychological Inflexibility (BS=0.67) and MHC-SF Emotional Well-being (BS= 0.58) respectively.

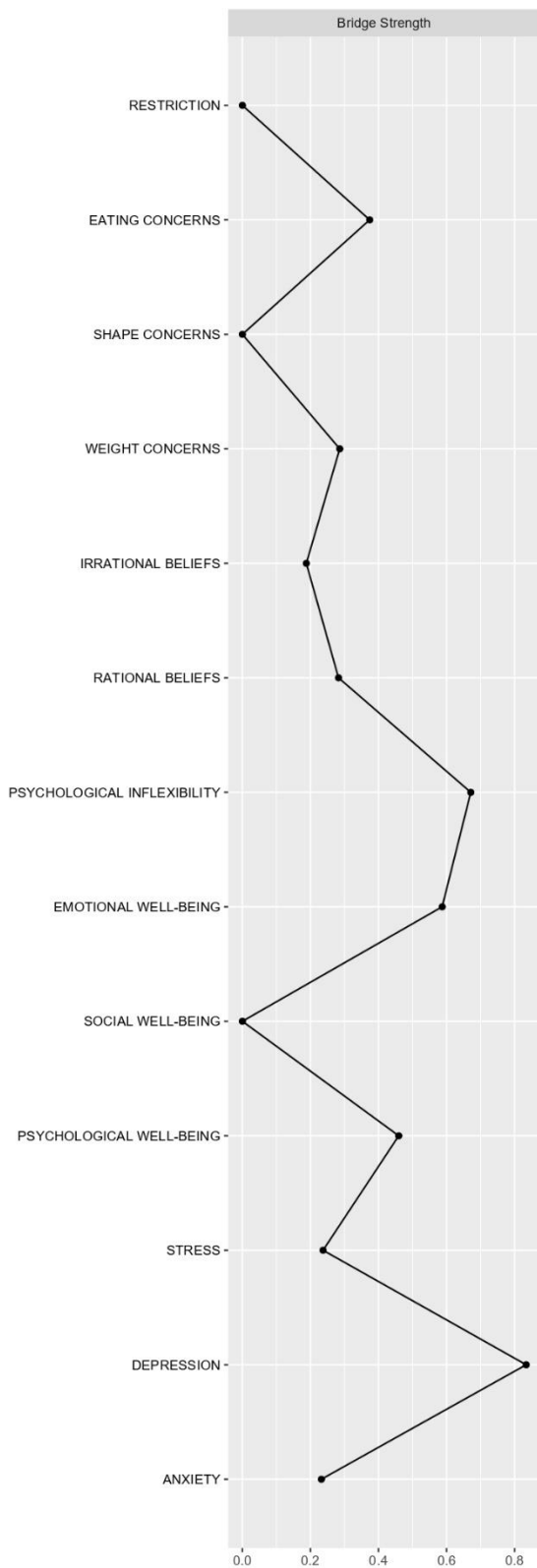


Figure 29 Bridge strength plot for the CP sample network

The plot for the bridge strength coefficient is reported in Figure 29. Bridge strength demonstrated adequate stability (BS-coefficient: 0.28).

### 3.4.2. Study 2

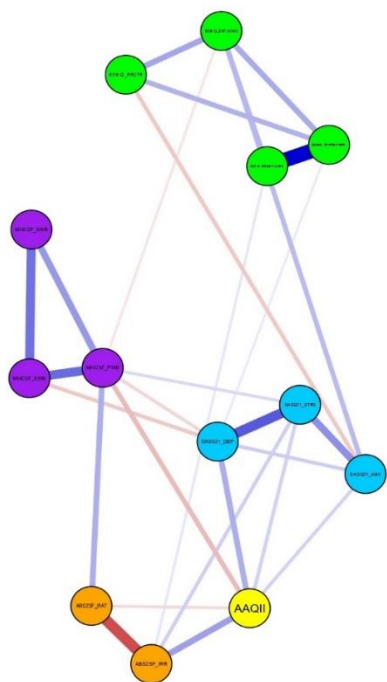
#### 3.4.2.1. Network comparison test of the 4 network models

A side-by-side representation of the four network models with the averaged layout for NetworkComparisonTest analyses can be seen in Figure 30.

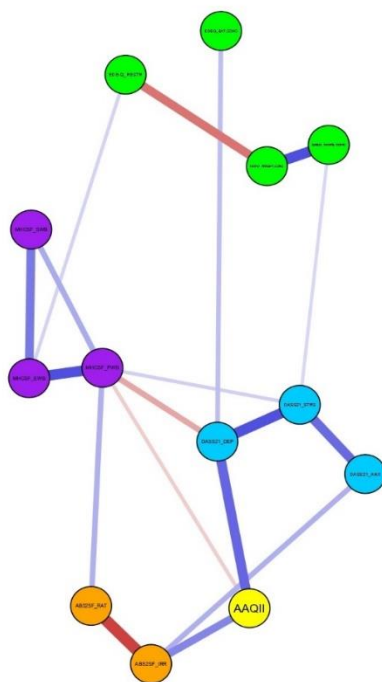
The network comparison test of the HC and RK network models showed significant differences in both the structure of the networks ( $M=0,38$ ;  $p < 0.05$ ) and global strength ( $S=0.82$ ;  $p < 0.05$ ), with the HC network resulting more tightly connected. Analysis of the differences between the individual edges highlighted significant differences in the edges connecting EDE-Q\_RESTR and EDE-Q\_WEIGHT.CONC (HC= 0; RK = -0.38;  $p < 0.01$ ), EDE-Q\_RESTR and MHCSF\_EWB (HC=0; RK = 0.11;  $p < 0.01$ ), EDEQ\_SHAPE.CONC and DASS21\_STRS (HC=0; RK=0,10;  $p < 0.05$ ), EDE-Q\_EAT.CONC and DASS21\_DEP (HC= 0; RK: 0.17;  $p < 0.05$ ), and ABS2SF\_IRR and DASS21\_ANX (HC= 0; RK= 0.21;  $p < 0.05$ ). Analysis of the absolute strength of edges between clusters showed differences only between the EDE-Q nodes clusters (HC= 1,55; RK = 0,86), while nodes clusters between MHC-SF nodes and DASS-21 nodes showed identical absolute strength values (1.07 and 0.89 respectively).

Figure 30 Network models represented with averaged layout as recommended for NCT testing (see Fried et al., 2022).

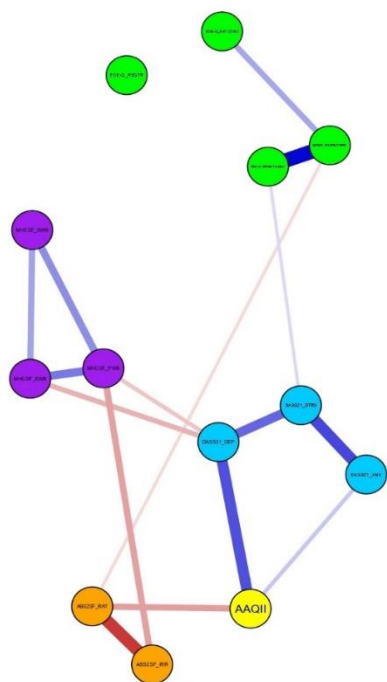
General Population



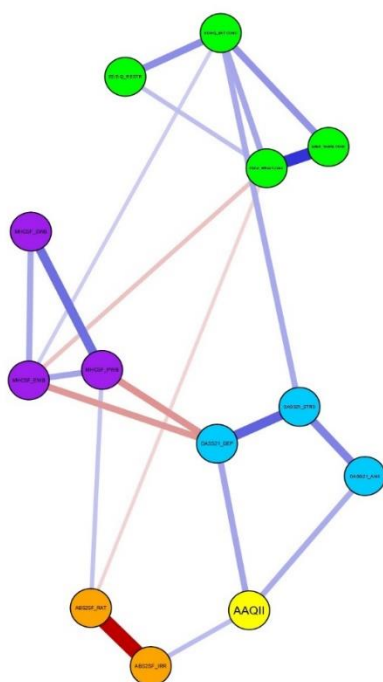
At risk Population



Acute Population



Clinical Population



- EDE-Q
- ABS2-SF
- AAQ-II
- MHC-SF
- DASS-21

The network comparison test of the RK and AC network models did not show significant differences in either the structure of the networks ( $M=0.38$ ;  $p=0.26$ ) or global strength ( $S=0.38$ ;  $p=0.31$ ). Analysis of the differences between the individual edges highlighted significant differences in the edges connecting EDE-Q\_RESTR and EDE-Q\_WEIGHT.CONC ( $RK=-0.38$ ;  $AC=0.00$ ;  $p<0.01$ ), EDEQ\_SHAPE.CONC and EDE-Q\_WEIGHT.CONC ( $RK=0.48$ ;  $AC=0.69$ ;  $p<0.05$ ), ABS2SF\_IRR and MHCSF\_PWB ( $RK=0.00$ ;  $AC=-0.26$ ;  $p<0.05$ ), EDE-Q\_EAT.CONC and DASS21\_DEP ( $RK=0.17$ ;  $AC:0.00$ ;  $p<0.01$ ), and ABS2SF\_IRR and DASS21\_ANX ( $RK=0.21$ ;  $AC=0.00$ ;  $p<0.05$ ). Analysis of the absolute strength of edges between clusters showed minor differences between the EDE-Q nodes clusters ( $RK=0.86$ ;  $AC=0.92$ ), the MHC-SF clusters ( $RK=1.07$ ;  $AC=0.93$ ), and the DASS-21 clusters ( $RK=0.89$ ;  $AC=0.92$ ).

The network comparison test of the AC and CP network models did not show significant differences in the structure of the networks ( $M=0.65$ ;  $p=0.31$ ) but did show significant differences in global strength ( $S=0.96$ ;  $p<0.05$ ), with the CP network resulting more tightly connected. Analysis of the differences between the individual edges highlighted significant differences in the edges connecting ABS2SF\_RAT and AAQ-II ( $AC=-0.26$ ;  $CP=0.00$ ;  $p<0.01$ ), EDEQ\_EAT.CONC and MHC-SF\_EWB ( $AC=0.00$ ;  $CP=0.13$ ;  $p<0.01$ ), ABS2SF\_IRR and MHCSF\_PWB ( $AC=-0.26$ ;  $CP=0.00$ ;  $p<0.01$ ), EDE-Q\_EAT.CONC and DASS21\_STRS ( $AC=0.00$ ;  $CP=0.23$ ;  $p<0.05$ ), and AAQ-II and DASS21\_DEP ( $AC=0.48$ ;  $CP=0.25$ ;  $p<0.05$ ).

The comparison of the RK and CP network models did show significant differences for the network structure ( $M=0.55$ ;  $p<0.01$ ) but not for global strength ( $S=0.57$ ;  $p=0.15$ ). Analysis of the differences between the individual edges highlighted significant differences in the edges connecting EDE-Q\_RESTR and EDE-Q\_WEIGHT.CONC ( $RK=-0.38$ ;  $CP=0.17$ ;  $p<0.01$ ), ABS2SF\_IRR and ABS2SF\_RAT ( $RK=-0.52$ ;  $CP=-0.71$ ;  $p<0.05$ ), EDE-Q\_WEIGHT.CONC and MHCSF\_EWB ( $RK=0$ ;  $CP=-0.16$ ;  $p<0.05$ ), and EDE-Q\_EAT.CONC and DASS21\_DEP ( $RK=0.17$ ;  $CP=0$ ;  $p<$

0.05), MHCSF\_EWB and DASS21\_DEP (RK = 0; CP = -0.28;  $p < 0.05$ ), and ABS2SF\_IRR and DASS21\_ANX (RK= 0.21; CP= 0;  $p < 0.05$ ).

## Chapter 4. Discussions

The present study sought to explore the differences in ED symptomatology, cognitive characteristics and psychopathological symptomatology commonly associated with ED between the general population, a population at prodromal/heightened risk of ED onset, a population with clinically relevant ED symptomatology, and a clinical ED population. The goal of the study was to observe whether there might be significant differences in the relevance of specific psychological dimensions, configuration of symptoms, and their reciprocal influences that might help to differentiate populations at different stages of ED. To do so, individual network models were developed and analysed for each of these groups, and strength centrality indexes and stability of the networks were estimated. Subsequently, the individual network structures were compared for differences in the edges, the global strength of all edges, and the strength of specific edges between the various pairs of networks.

### Study 1

The network model estimated from the HC sample showed greater strength centrality for the nodes representing psychological well-being, body shape concerns, and symptoms of depression. The psychological well-being scale of the MHC-SF is based on Ryff's psychological well-being model (Ryff, 1989) and encompasses six specific domains that contribute to the development of optimal functioning and the fulfilment of one's potential: autonomy, self-acceptance, a sense of continued growth and development, the belief that life is purposeful and meaningful, quality relationships with others, and the capacity to master effectively one's environment. The relevance of this aspect in this particular population is in line with other scientific works highlighting the prominent role of positive functioning in a healthy and growth-oriented lifestyle (Ruini, 2017; Ryff, 1989), and in decreasing vulnerability to psychiatric conditions (Fava & Tomba, 2009). Higher levels of psychological well-being have also been associated with a higher quality of life even in presence of mental health

conditions (Bergsma et al., 2011) and with a healthier lifestyle in the general population (Fava et al., 2017). Shape and body image concerns resulted in the second strongest node in the network. Shape concerns have long been considered a characteristic typical of ED, and while that remains true, it has also been observed how such preoccupation largely affects the general population at all ages (Olson et al., 2020). Body dissatisfaction has been associated with a higher risk of depression (Paans et al., 2018), and can be linked to common occurrences such as media exposition, especially through social media, or one's own perception of other's expectations (Barnes et al., 2020; Bennett et al., 2020; Voelker et al., 2015). Despite the relevance in the general population, it is difficult to establish the cause that might led to this construct to have such a prominent role in the network – granted the considerable gap in strength centrality between psychological well-being and shape concerns; it is possible that the relevance of shape concerns in an healthy sample is the result of the voluntary response sample bias, so that individuals especially interested or concerned in eating and body-related issues were more attracted to the study and data collection procedures. Symptoms of depression were the third strongest node in the HC sample network. This result is aligned with epidemiological data about the pervasiveness and frequency of the depressive symptoms in the general population (Hapke et al., 2022; Musliner et al., 2016; Vos et al., 2012). It is important to note, however, that only a minority of individuals exhibiting depressive symptomatology will actually develop clinically relevant and persistent symptoms (Musliner et al., 2016). Furthermore, the high centrality of depressive symptomatology might be a result of the high relevance of body shape concerns, as the two have a well-established connection, and the first represents a significant risk factor for the second (Kamody et al., 2018; Satghare et al., 2019).

The node with the highest bridge strength in the healthy population network model was the one representing psychological inflexibility, defined as the rigid dominance of psychological reactions over chosen values and contingencies in guiding action (Hayes et al., 2006). Interestingly, psychological inflexibility presented direct associations with all groups of nodes except the

disordered eating domain. This is in line with the purported influence of psychological flexibility in avoiding negative internal thoughts, feelings, and physical sensations by controlling the behaviour, at the expense of more meaningful actions (Guerrini Usubini et al., 2022), and its mediating role between positive functioning and anxiety and depressive symptoms (Guerrini Usubini et al., 2022). Psychological well-being and depressive symptomatology appeared respectively as the second and third strongest bridge nodes in the network. Again, the prominence of positive psychological functioning is not surprising as such aspect has been well documented as playing a key role in reducing the risk of psychopathology and maintaining euthymia (Fava & Guidi, 2020; Guidi & Fava, 2020; Ruini, 2017). The presence of depression as a bridge node, on top of being a highly central node, begs the question about its role in this population. A possible interpretation is that depression acts as the intermediary between underlying cognitive processes, such as irrational beliefs or psychological inflexibility, and disordered eating, a relationship well documented in the literature (Levinson et al., 2017; Martin et al., 2019; Monteleone & Cascino, 2021). As such, its prominence in this population could be the byproduct of voluntary response sample bias. Another possible explanation is the transversal and multifaceted nature of what we define as depressive symptomatology (Fried, 2015), and its connection with a large number of aspects of cognitive, positive and negative psychological functioning (Fava et al., 2007). Considering the psychological and cognitive dimensions included in this study, it does not surprise that depression appears at the intersection of those aspects.

The network model from the RK sample exhibited high centrality for well-being dimensions and transdiagnostic factors such as low mood and chronic stress. This is in line with previous findings highlighting the relevance of well-being dimensions in the assessment and clinical management of ED in clinical populations (de Vos et al., 2021; Tecuta et al., 2023; Tomba et al., 2014; Tomba et al., 2019). Furthermore, previous studies reported similar levels of quality of life in ED outpatients and healthy controls (Mond et al., 2005; Tomba et al., 2014); it is, therefore, possible that the high



relevance of positive functioning observed in this group of participants reflects the egosyntonicity and lack of insight characteristic of the early stages of an ED (Roncero et al., 2013; Vitousek et al., 1998). On the other hand, positive functioning could represent a buffer against exacerbation of symptoms, thus acting as a protective factor, as already observed in the literature (Fava & Tomba, 2009). In light of these considerations, future studies might want to further explore the role of psychological well-being in populations at risk of ED, and evaluate whether to intervene preferentially on psychological well-being to test its efficacy in improving prevention intervention outcomes. The relevance of transdiagnostic symptoms is also in line with both previous network studies (de Vos et al., 2021) and established ED literature (Monteleone & Cascino, 2021; Tomei et al., 2022). The role of depression in the onset of ED is still unclear, and the debate about whether low mood precedes or follows disordered eating is still open (Hilbert et al., 2014; Vervaet et al., 2021). The relevance of depressive symptomatology in individuals with subthreshold ED manifestations observed in this study is consistent with the theories assuming depression precedes ED, but no inference about causality between the two can be made due to the nature of the study. Much like for depression, the relevance of chronic stress in this population is a well-documented phenomenon in the onset of ED: clinical studies indicate an association between stress and ED onset and symptom expression (Hardaway et al., 2015). Notably, nodes representing ED-specific symptomatology appeared at the periphery of the network: this result is in line with the outcomes reported by a review of network studies on ED populations (Monteleone & Cascino, 2021), highlighting how ED symptoms were often among the less relevant nodes of their respective networks. This would seem to support Fairburn's model of ED, in which the actual behavioural and cognitive component related to eating and body shape is secondary to other psychological dimensions such as self-esteem, mood, or frustration tolerance (Fairburn et al., 2003).

Psychological inflexibility represented the main bridge node in the network from the RK sample of this study. Psychological inflexibility and clinical perfectionism have been observed to be

associated with disordered eating and worry about body shape and weight (Masuda et al., 2010; Morton et al., 2020). In this study, psychological inflexibility acted as bridge between irrational beliefs and depression, which was in turn strongly associated with stress. Considering the literature on the role of psychological inflexibility in ED and psychopathology in general (Masuda et al., 2011; Merwin et al., 2011), it is possible that a rigid cognitive style might exacerbate the impact of irrational beliefs on mood and psychological well-being, two highly central nodes, thus helping the propagation of negative triggering events to the rest of the network. This conceptualization of the interactions of the triad psychological inflexibility – depression – psychological well-being finds further support in the latter two dimensions' role as the second and third most relevant bridge nodes.

Similar considerations to those made for the RK population for node centrality apply to the network obtained by the AC sample. Interestingly, despite the more severe symptomatology compared to the RK sample, ED symptoms are still relatively in the periphery of the network. In this population, depression was the most central node of the network, followed by psychological well-being and stress. The unclear relationship between depression and ED onset has already been discussed above. The exponentially increased relevance of negative affective symptomatology as ED symptoms become more severe would seem to support the observations of Rojo and colleagues (2006) in which symptoms of depression had a direct effect on ED severity; a relationship also reported in a literature review by Calvo-Rivera and colleagues (2022), in which the authors commented how cumulative evidence supports the direct influence of depressive symptomatology on ED manifestations. Again, psychological well-being seems to play an important role in the psychological network of this population, and more specifically appears to mediate the relationship between irrational beliefs and depressive symptomatology. This is consistent with the results of the study by Tecuta and colleagues (2023) and further supports the idea that the influence of cognitive maladjustment on psychopathology, including ED, might be mitigated by promoting and maintaining a positive attitude towards oneself and a positive general evaluation of one's life (Fava & Tomba,

2009; Ryff, 2014). Interestingly, the strength centrality of psychological well-being would seem to be determined by the numerosity of its edges rather than their individual strength, possibly suggesting a widespread influence of such dimension on the rest of many other aspects considered in the network. Considering the importance given to well-being by patients with ED (de Vos et al., 2018; Tomba et al., 2014; Tomba et al., 2019), these results confirm the relevant role of this construct in the clinical picture of individuals with clinically relevant ED symptoms. In this network model, as for the previous one, stress appears to be the mediator between general psychopathology and ED-specific manifestations, warranting the same considerations made for the network of the RK sample.

In this network, as for the previous ones from ED populations, psychological inflexibility represented the main bridge node, followed by depression and psychological well-being. Similar considerations apply here to the RK population: psychological inflexibility has been associated with disordered eating and worry about body shape and weight (Masuda et al., 2010; Morton et al., 2020). In this population, differently from the RK one, psychological inflexibility acted as bridge between rational beliefs and depression. This shift in the conditional association from irrational to rational beliefs might be explained by the increased severity of the psychiatric symptomatology. As the disorder progresses and the adherence to strict rules becomes more and more important, it might be the case that psychological inflexibility starts exerting its influence directly on the rational cognitive processes of the individual, rather than affecting the irrational ones. Indeed, previous studies observed greater irrational beliefs endorsement in clinical populations compared to healthy controls (Tecuta, Gardini, Schumann, et al., 2021); the results obtained in this study might explain the direct influence of psychological inflexibility in “suppressing” rational beliefs, allowing the irrational ones to surface more powerfully. Similarly, as for the RK sample, the effect of a rigid cognitive style might here influence rational rather than irrational beliefs, exerting however similar effects on mood and psychological well-being, the two strongest nodes in the network. This conceptualization of the

interactions of the triad psychological inflexibility – depression – psychological well-being follows the same pattern as in the RK sample and similarly is supported by their role as bridge nodes.

The results from the network analysis of data from the CP sample underline a shift to a more prominent ED symptomatology. Together with the exacerbation of the condition, it is possible to observe how ED-specific symptoms such as weight concerns and eating concerns become more central, together with negative affective symptomatology. The increased centrality of ED-specific symptomatology in this sample is consistent with the previous conceptualization of full-blown severity (Grange & Loeb, 2007; Treasure et al., 2015), and reflects a well-established condition warranting clinical attention and treatment. Compared to the other network models presented, it is interesting to note how the ED-specific manifestations presented by this population are more oriented towards metrics of eating conduct, such as body weight or preoccupation around calories of foods eaten, eating in front of others, or feelings of guilt regarding eating. This change might represent the efforts of the individual in maintaining the disorder: once the disordered eating conducts are established, the person might progressively become more and more preoccupied about maintaining or further decreasing their body weight, as well as become preoccupied about being in contexts where they can't adhere to their dietary rules. The high strength of depressive symptomatology in this population does not surprise either, and the presence of depressive symptoms in established ED cases has been well documented in the literature, and comorbidity of ED and depressive symptoms has been historically linked to physiological starvation (Keys et al., 1950), a common self-imposed state in many ED patients.

While the nodes for depression and psychological inflexibility are the strongest and second strongest bridges respectively, it is interesting to see how emotional well-being is the third strongest bridge node, as opposed to psychological well-being as in the other networks. This result might be explained by the increased relevance of depression in the network from this population. Indeed, emotional well-being represents positive affect and satisfaction with life (Keyes et al., 2008), a

dimension that appears at the opposite end of the spectrum from depression. It is possible then that the increased centrality of depressive symptomatology is also reflected in the increased relevance of its opposite.

## Study 2

The network comparison test between the healthy controls and at-risk populations showed significant differences in both the structure and global strength of the network. Interestingly, the network from the general population appeared more tightly connected than the one from the at-risk sample, in contrast with the idea that tighter networks reflect higher psychopathology (Fried et al., 2017). An analysis of the individual node clusters revealed that the tighter connectivity of the network from the healthy controls was mainly due to the stronger connections between nodes representing ED symptoms. These results might appear counterintuitive, but can be explained in light of the function and insight of the individual into the dynamics that govern the interactions between ED symptoms (Gorwood et al., 2019). A decreased awareness of how ED symptoms influence each other is associated with a higher risk of ED and lower help-seeking behaviours, and in fact, in the early stages of the disorder, symptoms of ED are often evaluated positively by the individual (McAndrew, Menna & Oldershaw, 2022; Ali et al., 2020). Consistently with these considerations, the sample at risk of ED presented stronger edges between restricted eating, weight concerns and shape concerns, as well as between eating concerns and depression, and irrational beliefs and anxiety. Of particular interest is the positive edge connecting restricted eating and emotional well-being, which resulted in significantly stronger in the at-risk population. This seems to reflect the theoretical assumption that has restricted eating as a self-regulatory behaviour associated with positive affect, enacted to counterbalance the negative feelings associated with the idea of eating (Fairburn et al., 2003) – an association which was confirmed in the present study. These results appear more in line with the theoretical assumptions of network theory concerning psychopathology, and it is possible that the higher global connection showed by healthy controls might be explained by stronger associations

between positive functioning dimensions in this population, reflecting better psychological adjustment.

The network comparison test between the population at risk and the population with acute symptoms showed no significant differences in either the structure or the global strength of the network. A comparison of the individual edges between the two networks, however, showed that the population at risk presented a stronger negative association between restricting and weight concern. This result might be explained in light of restriction conducts ameliorating concerns about weight, and in general acting as a gateway to acute ED (Allen et al., 2012). This group of participants also exhibited a stronger relationship between depressive symptomatology and worries about food, coherently with the idea that dietary rules might be first enacted to counter negative feelings until the individual loses control over such conducts (Sander et al., 2021). On the other hand, the population with acute ED symptomatology showed a stronger negative association between irrational beliefs and psychological well-being, reflecting the greater severity of general psychopathology of this population (Tecuta, Gardini, Schumann, et al., 2021), and between shape concerns and weight concerns, possibly reflecting the progressive shift towards disordered eating behaviours as more focused toward perceived external appearance.

The network comparison test between the acute population and the clinical population showed no significant differences in the structure of the network, but a significant difference in global strength between the two models. In the clinical group, eating concerns were more strongly associated with emotional well-being and stress. These two associations can be explained by the crystallization of disordered eating behaviours and their influence over positive functioning and distress. As the ED takes hold, habits regarding dieting and avoidance of eating in front of others become more and more stable, and their modification causes significant distress to the person (Tomba et al., 2023). The network from the acute population, on the other hand, presented stronger edges between psychological inflexibility and rational beliefs and depressive symptomatology. This possibly reflects

a more rigid cognitive scheme in this population, associated with lower mood and with egosyntonicity of the ED symptomatology, which has been observed to influence treatment-seeking behaviours even in the presence of severe symptomatology (Ali et al., 2020; Gregertsen et al., 2017).

#### 4.1. Limits and conclusions

The results of this study should be interpreted in light of some important limitations. First of all, despite the efforts to collect high-quality data, the self-report nature of the questionnaires and the recruitment procedures might have potentially introduced a selection bias in the study sample. Secondly, it is important to note that female participants vastly outnumber their male counterparts, an imbalance which does not represent the Italian general population distribution. Another relevant element potentially introducing a bias in the sample is the possible lack of insight of participants regarding their ED symptomatology, a relatively common and clinically relevant feature which affects this population's symptom reporting and help-seeking behaviours (Gorwood et al., 2019). Furthermore, it is possible that topological overlap might have influenced the quality of the results. While the decision of keep all the nodes in the model despite some of them overlapping was a deliberate one, it is nonetheless probable that some nodes might share statistical properties even if conceptually dissimilar, or might be more clinically than statistically valid, as in the case of the EDE-Q (Fairburn & Beglin, 1994). Finally, the criteria applied to distinguish the various groups according to their ED severity was not conducted using an *ad hoc* instrument. While the categorization of the various populations analyzed in this work was based on the approach successfully used in previous works (Melisse et al., 2021; Bryant et al., 2021), and using reference values from validation studies on the Italian population (Calugi et al., 2017), the results of this work should be interpreted in light of the potential bias in the groups' selection procedure.

Despite these limitations, the results of the present study have some interesting implications. The results of the individual network analyses and the comparison between the different models

further support the idea that ED is a complex category. One aspect that consistently emerged across all groups is the relevance of established transdiagnostic elements, such as irrational beliefs, affective and anxiety symptomatology, and psychological inflexibility, at all degrees of intensity of ED symptoms. Furthermore, less studied transdiagnostic dimensions, such as rational and irrational beliefs or positive functioning, emerged just as strongly and demonstrated to characterize and influence in different ways the various stages of intensity of ED symptomatology. Indeed, it would seem that a potential prodromal stage of ED might be characterised by high sensitivity to fluctuations in well-being and mood, and by the reciprocal influence of well-being dimensions and ED cognitive contents. As the ED-specific symptoms worsen, so does the pervasiveness of mood symptomatology, and the reciprocal influence between ED cognitive aspects and between irrational beliefs and well-being, possibly identifying an acute stage. While the relevance of psychological dimensions outside of symptoms strictly related to ED has been exhaustively discussed elsewhere (Monteleone & Cascino, 2021), the present work provided novel insight into how less studied aspects involved in ED might influence the clinical presentation, and consequently impact on clinical interventions. (Tomba et al., 2023). While it was beyond the scope of this exploratory project to establish clear criteria to identify the different stages of ED, the data presented can, nonetheless, indicate a possible direction for future research endeavours.



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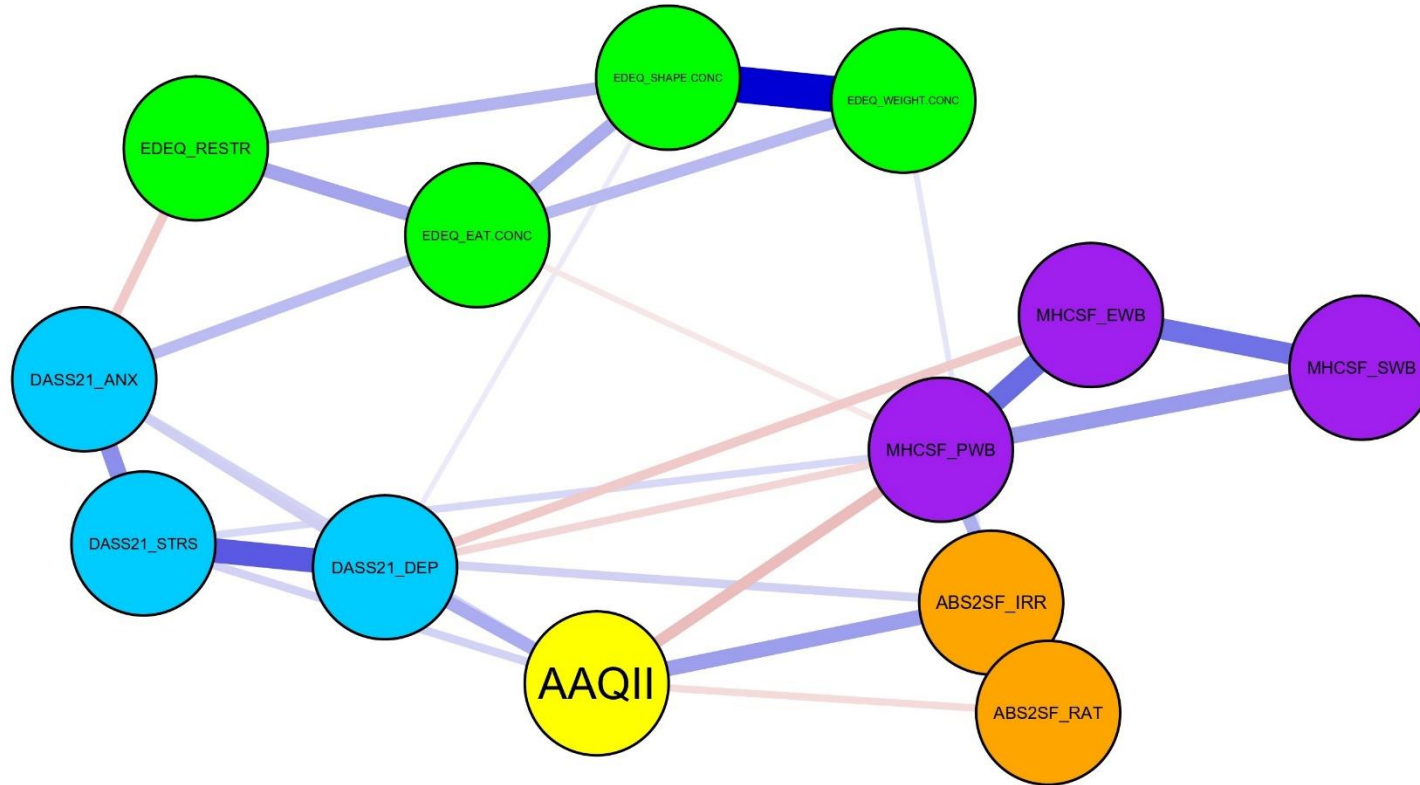
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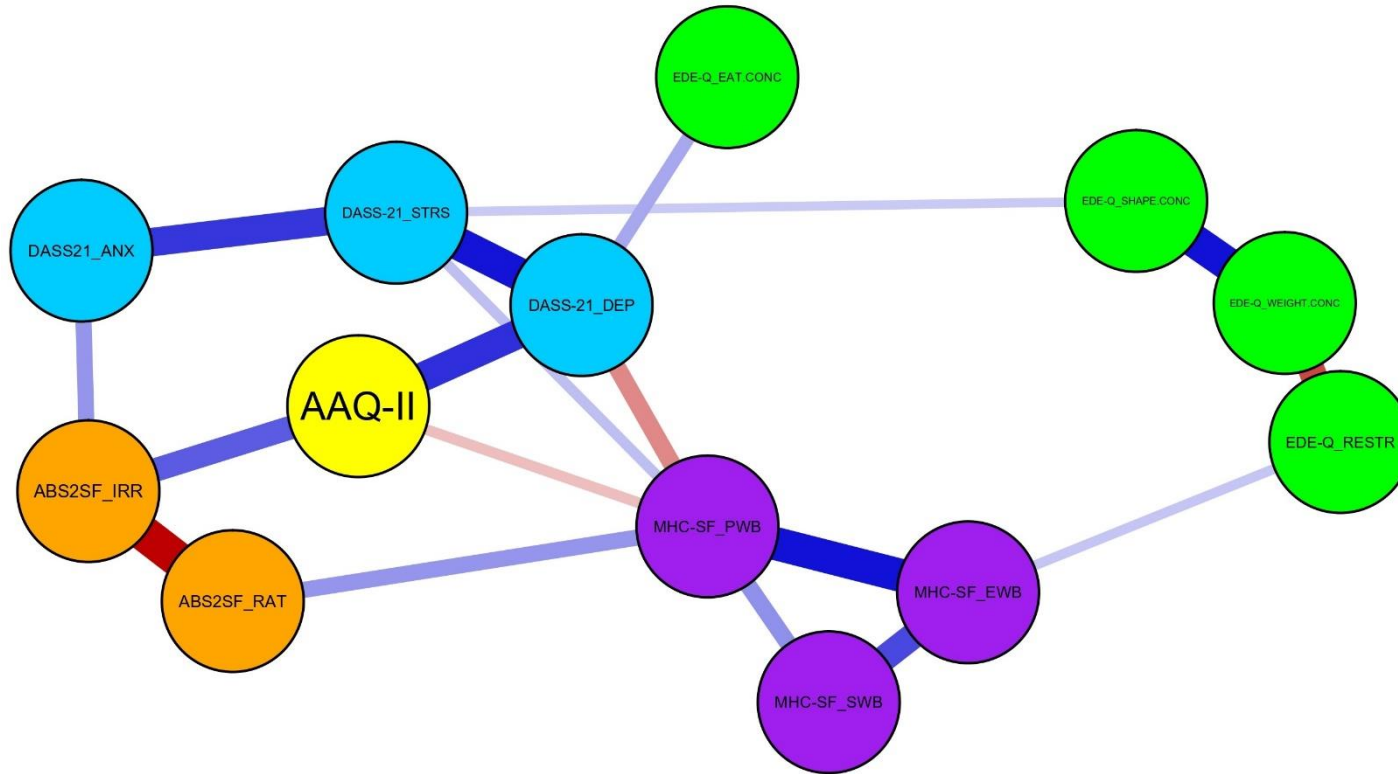
# Appendix

Network model from the healthy control (HC) sample



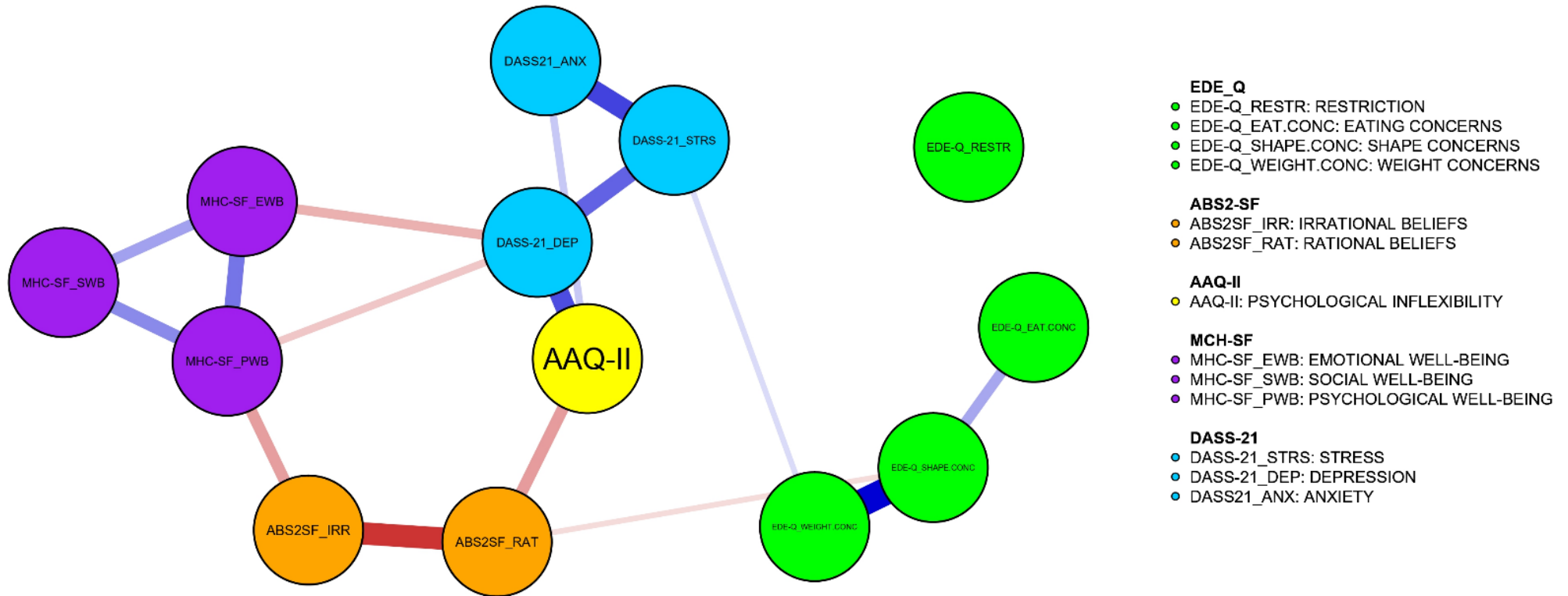
- EDE\_Q**
  - EDEQ\_REST: RESTRICTION
  - EDEQ\_EAT.CONC: EATING CONCERNS
  - EDEQ\_SHAPE.CONC: SHAPE CONCERNS
  - EDEQ\_WEIGHT.CONC: WEIGHT CONCERNS
  
- ABS2-SF**
  - ABS2SF\_IRR: IRRATIONAL BELIEFS
  - ABS2SF\_RAT: RATIONAL BELIEFS
  
- AAQ-II**
  - AAQII: PSYCHOLOGICAL INFLEXIBILITY
  
- MCH-SF**
  - MHCSF\_EWB: EMOTIONAL WELL-BEING
  - MHCSF\_SWB: SOCIAL WELL-BEING
  - MHCSF\_PWB: PSYCHOLOGICAL WELL-BEING
  
- DASS-21**
  - DASS21\_STRS: STRESS
  - DASS21\_DEP: DEPRESSION
  - DASS21\_ANX: ANXIETY

Network model from the at risk (RK) sample

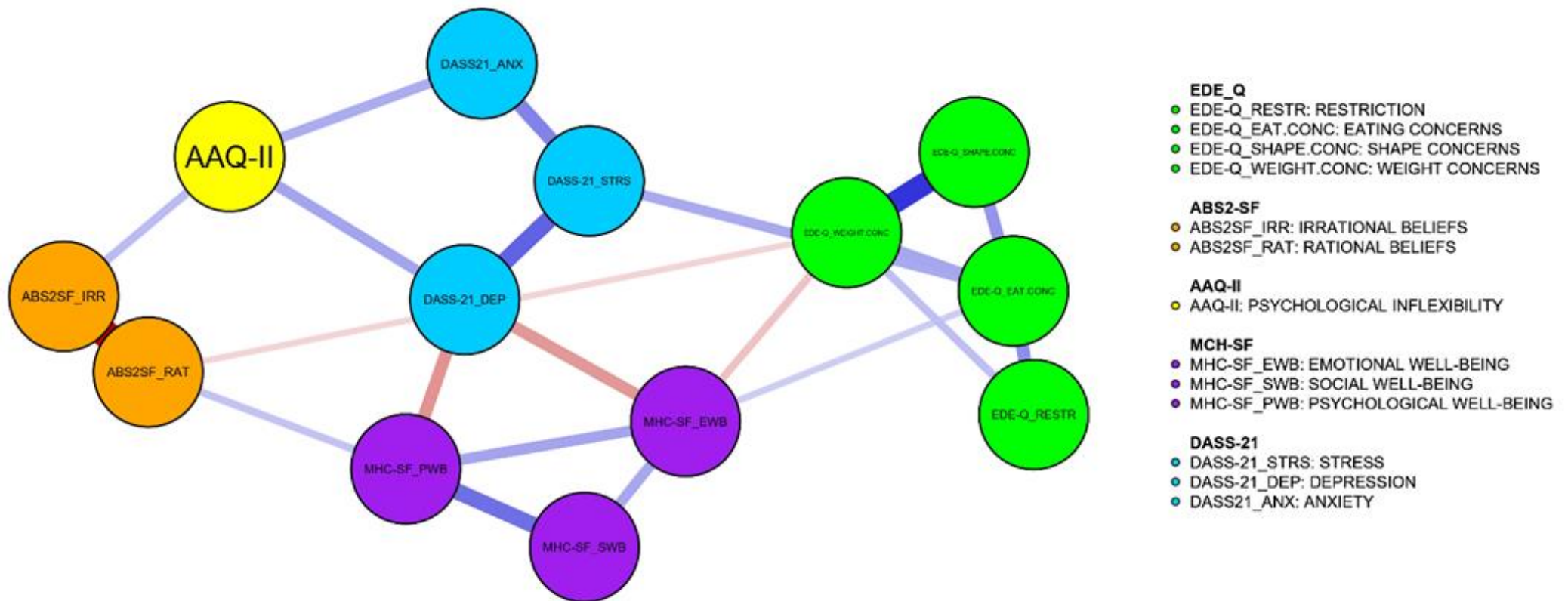


- EDE\_Q**
  - EDE-Q\_RESTR: RESTRICTION
  - EDE-Q\_EAT.CONC: EATING CONCERNS
  - EDE-Q\_SHAPE.CONC: SHAPE CONCERNS
  - EDE-Q\_WEIGHT.CONC: WEIGHT CONCERNS
- ABS2-SF**
  - ABS2SF\_IRR: IRRATIONAL BELIEFS
  - ABS2SF\_RAT: RATIONAL BELIEFS
- AAQ-II**
  - AAQ-II: PSYCHOLOGICAL INFLEXIBILITY
- MCH-SF**
  - MHC-SF\_EWB: EMOTIONAL WELL-BEING
  - MHC-SF\_SWB: SOCIAL WELL-BEING
  - MHC-SF\_PWB: PSYCHOLOGICAL WELL-BEING
- DASS-21**
  - DASS-21\_STRS: STRESS
  - DASS-21\_DEP: DEPRESSION
  - DASS21\_ANX: ANXIETY

Network model from the acute population (AC) sample

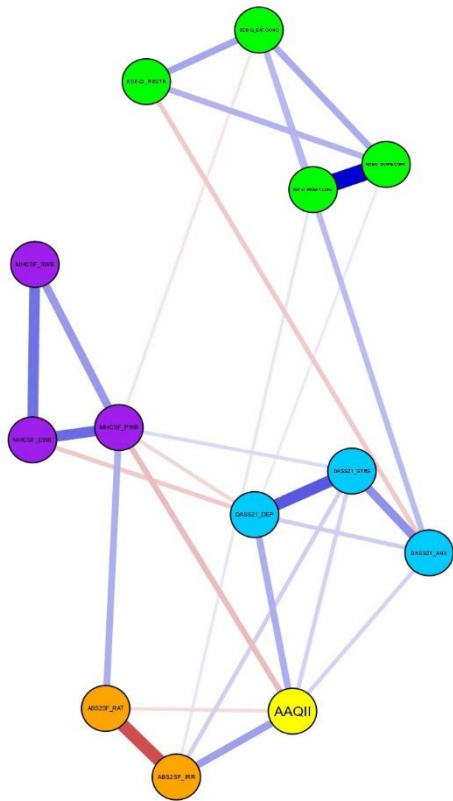


Network model from the clinical population (CP) sample

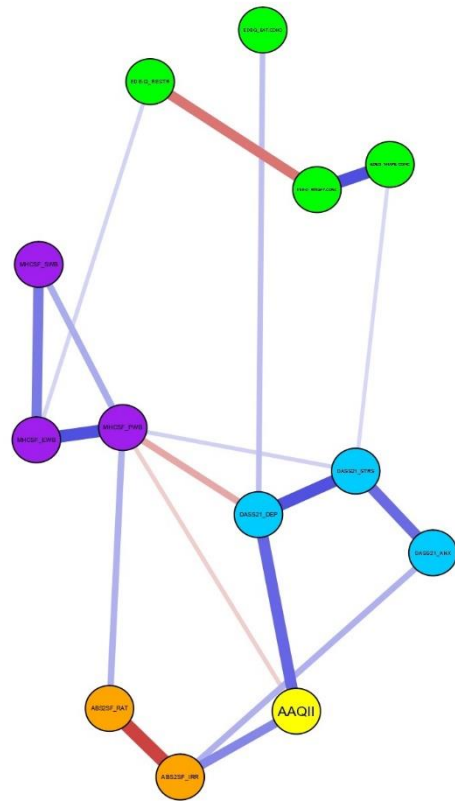


Network models represented with averaged layout as recommended for NCT testing (see Fried et al., 2022)

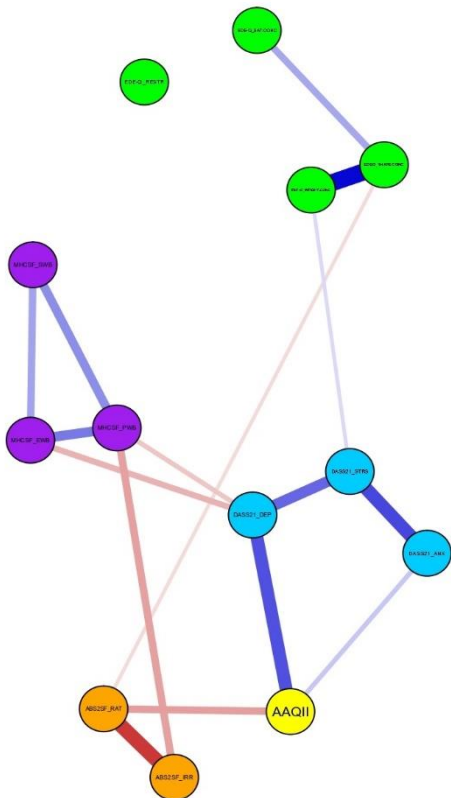
General Population



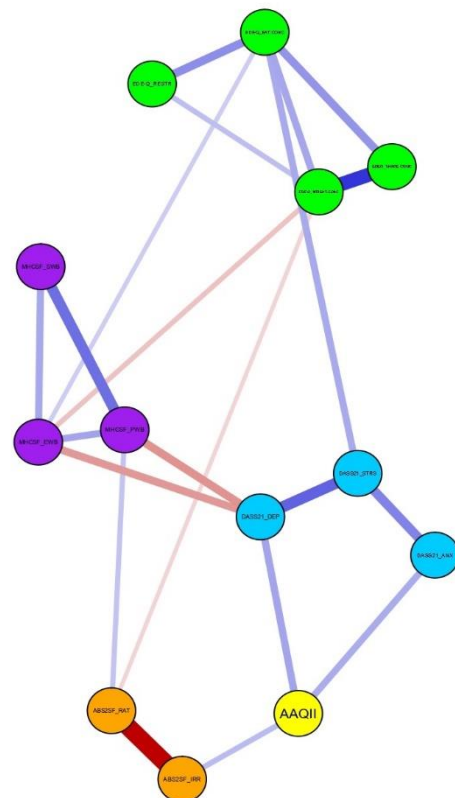
At risk Population



Acute Population



Clinical Population



- EDE-Q
- ABS2-SF
- AAQ-II
- MHC-SF
- DASS-21