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Eating Disorders and Autism: A Network Approach

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Eating Disorders (EDs) and autism spectrum disorder (ASD) have several overlapping symptoms that may inform our understanding of both disorders. Increased knowledge of the overlap of EDs and ASD can improve the treatment of EDs in those with ASD. This paper explores the overlap of ED and ASD symptoms, and evaluates the results of a study that used network analysis to investigate pathway and core ED and ASD comorbidity symptoms.

Eating Disorders

Eating disorders are life altering and potentially fatal disorders characterized by severe disturbances in eating behaviors and attitudes towards body shape and weight (Treasure et al., 2020, Schmidt et al., 2016). The Diagnostic and Statistical manual of Mental Disorders- 5th Edition (DSM-5; American Psychiatric Association [APA], 2013) includes anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and other specified feeding or eating disorder (OSFED).

AN and BN are eating disorders characterized by distorted perceptions of one's weight and shape, internalization of the thin-ideal, and persistent extreme weight control behaviors (Klein et al., 2021, Hay, 2020, Neale & Hudson, 2020). AN is distinct from BN as it is a condition of self-starvation (Hay, 2020) and has a Body Mass Index (BMI) requirement for diagnosis (APA, 2022). BN involves a cycle of binge eating followed by compensatory behaviors such as self-induced vomiting, fasting, and other compensatory behaviors (APA, 2022). BED is characterized by recurrent episodes of binge eating without compensatory behaviors, and a feeling of a lack of control over ones eating and distress regarding the binge eating (APA, 2022). Unlike AN and BN, the DSM-5 criteria for BED does not include body

dissatisfaction (APA, 2022), however, a large body of research has found that those with BED do exhibit significant overevaluation of their shape and weight (e.g., Yiu et al., 2017).

Unlike other the EDs, OSFED includes a heterogenous group of ED presentations (APA, 2022). OSFED is diagnosed when symptoms of an ED are present and causing significant distress or impairment but do not meet full diagnostic criteria for AN, BN, or BED. OSFED diagnosis includes specifications such as: atypical anorexia nervosa (AAN), subclinical BN and BED, purging disorder (PD), and night eating syndrome (NES; APA, 2022). Research finds OSFED to be more prevalent than any other ED, and a 2020 study found OSFED and unspecified feeding or eating disorder (UFED) to be about 2.5 times more common than AN, BN, or BED (Mitchison et al., 2020).

A study done in 2019 found EDs to have a lifetime weighted mean prevalence of 8.4% in woman and 2.2% in men (Galmiche et al., 2019). Recent research prompted by the COVID-19 pandemic suggests that these rates have increased, with a recent analysis finding hospitalizations due to EDs increased by 48% following the start of the pandemic (Devoe et al., 2022).

Risk factors for developing an ED include thin-ideal internalization, concern about one's weight, and personality factors like perfectionism and negative emotionality (Culbert, Racine, & Klump, 2015). While people with EDs do seem to interact with healthcare providers more frequently than the general population, the vast majority of them do not receive diagnosis or treatment for their ED (Hart et al., 2011). Along with this, a 2020 study found that across eating disorders, 62-70% of people who received inpatient treatment for ED and 35% of those who received outpatient treatment still showed eating disorder symptoms or met full diagnostic criteria at a long-term (10-20 years) follow-up (Van Hoeken & Hoek, 2020).

The mortality rate of EDs is also remarkably high, with one 2021 study of people receiving ED treatment in a hospital finding the all-cause mortality rate for this group to be five times higher than expected based on mortality rates of the Ontario population, and another study finding the hazard ratio when adjusted for age and sex to be 4.5 for those with EDs when compared to those without EDs (Iwajomo et al., 2021; Pedram et al., 2021). The high prevalence of EDs, rate of relapse after treatment, and mortality rate make it clear that more information on the nature and presentation of EDs is incredibly important to improve treatments and reduce the risks associated with EDs.

Autism

ASD is a neurodevelopmental disorder generally diagnosed in early childhood (APA, 2013). ASD is characterized by restrictive and repetitive patterns of behavior, and persistent difficulties in social interactions and social communication (Milner et al., 2019). A 2021 study found that approximately 1 out of every 100 children worldwide are diagnosed with ASD (Zeidan et al., 2022).

The limited research regarding autism in adults suggests that autistic children do not grow out of their autism as adults. A 2020 study found that in a population of people who received a diagnosis equivalent to an ASD diagnosis as a child either continued to qualify for a diagnosis of ASD as adults, or continued to have higher self-reports of autistic behavior than those who had never received a diagnosis of ASD (Horwitz et al., 2020). This is important as it suggests that most adults who had been diagnosed with autism as children maintain at least some degree of autistic traits into adulthood, meaning that their autism continues to impact their life, and would reasonably impact their experience of an ED if they develop one.

Gender Discrepancies in Diagnoses

Prevalence of ASD diagnoses have a high male to female ratio at approximately 4:1 (Fombonne, 2009). However, recent research suggest that ASD may be underdiagnosed in women, potentially due to the use of solely or majority male samples in ASD research, along with the stereotyping of ASD as a male disorder (Milner et al., 2019). This research also suggests that autistic women may differ in their presentation of ASD when compared to autistic men (Wood-Downie et al., 2021). For example, autistic women are more likely to use camouflaging to disguise autistic traits to compensate for the social difficulties associated with autism (Wood-Downie et al., 2021).

Alternatively, EDs are much more commonly diagnosed in women than in men, and current research suggests that EDs may be underdiagnosed in men (Strother et al., 2012). Research suggests men may have a harder time understanding their behaviors as potentially linked to or symptoms of an ED, partially due to the misconception that EDs are mainly an issue for women (Räisänen & Hunt, 2014). This issue is compounded by healthcare providers who often lack training regarding ED, and female-centric medical treatment guidelines for ED that reinforce the idea that women are the group most impacted by EDs (Ganson, Murray & Nagata, 2021; Golden et al., 2015; Girz, Robinson, & Tessier, 2014). Along with this, many men feel shame about their ED, causing them to postpone seeking treatment, or conceal their diagnosis from others (Björk, Wallin, & Pettersen, 2012). Consequently, shame can lead to men only receive treatment for an ED when it has become very serious.

Co-Occurring EDs and Autism

Research consistently finds elevated rates of ASD symptoms in those diagnosed with AN (Westwood & Tchanturia, 2017; Koch et al., 2015). It is unclear if this association is due to a greater likelihood of developing AN in those with ASD, or if there are other mitigating factors.

Oftentimes in the research examining ASD symptoms, data is not collected on the developmental history of participants, making it unclear if they had demonstrated autistic traits as a child or prior to their development of AN (Westwood & Tchanturia, 2017). Although the majority of research is unclear, a small number of studies have found that those with an ED diagnosis were at elevated risk of receiving an ASD diagnosis, and that those with a primary diagnosis of ASD were at elevated risk of receiving an ED diagnosis; research has also demonstrated an association between ASD and BED (Koch et al., 2015; Nickel et al., 2019; Numata et al., 2021). Though there is currently no research exclusively investigating connections between ASD and BN, or ASD and OSFED, research examining transdiagnostic EDs and ASD consistently finds heightened autistic traits or rates of autism (Vagni et al., 2016; Gesi et al., 2017; Huke et al., 2013).

Importantly, recent research has found ED treatment to be less effective for those with ASD or ASD symptoms when compared to treatment for ED patients that do not display autism symptoms (Nielsen et al., 2015). Multiple studies show that symptoms of ASD were correlated with worse outcomes and reduced recovery from AN (Nielsen et al., 2022; Nielsen et al., 2015). Further, in a qualitative study of their experiences receiving ED treatment, autistic women reported multiple issues, largely centering around providers not recognizing their ASD or ASD symptoms. This lack of understanding of ASD lead to providers failing to modify the ED treatment for these women, resulting in them being unable to effectively engage in or access treatment for their ED (Babb et al., 2021). The lack of understanding and failure to modify treatment may connect to the medical model of autism, where providers see autism as a disorder to be cured, similar the ED that they are there to treat. This is opposed to the neurodiversity model, which posits ASD as a part of the natural variation of humans, and something to be

accommodated and respected instead of punished or corrected (Schuck et al., 2021). Understanding the potential link between ASD and ED is crucial to developing effective, inclusive ED treatments for those with co-occurring EDs and ASD.

The Current Study

A network analysis approach is a valuable way to investigate the links between ED and ASD. Network analysis has been popularized in mental health research in part by the network theory of psychiatric illnesses (Borsboom, Cramer, & Kalis, 2019). This theory posits that instead of psychiatric problems and symptoms being caused by a currently unknown biological pathway, symptoms directly influence each other (Borsboom, Cramer, & Kalis, 2019). This approach illuminates the direct relationships between individual symptoms of each disorder to allow for the identification of bridge symptoms between the disorders and relationships between symptoms (Cramer et al., 2010). Bridge symptoms—called bridge nodes in the context of a network analysis—are the symptoms from one disorder that are calculated through the network analysis to be the most connected to the symptoms of the other disorder. Understanding bridge nodes can deepen our understanding of comorbid ASD and ED, and potentially become the basis for future research into causal relationships between the disorders.

Despite the values of using a network analysis to investigate the link between EDs and ASD, only a minimal amount of research does so. The first study using network analysis to look at the relationship between ASD and AN in people with a diagnosis of AN found the bridge symptoms between the two disorders to be; "I have good self-confidence" (reverse coded), "How concerned have you been about other people seeing you eat?", and " How uncomfortable have you felt about other people seeing your shape or figure" (Kerr-Gaffney et al., 2020). Along with the network analysis looking at AN and ASD in 2020, another used a network analysis to

identify common symptoms across all ED diagnoses and found that symptoms associated with body checking and dissatisfaction was one of the strongest nodes connecting all EDs, indicating that all EDs have common symptoms tying them together (Forbush, Siew, & Vitevitch, 2016). The 2020 paper provides valuable evidence of connections between ASD and AN, and the 2016 paper shows connections between each individual ED diagnosis; supporting further exploration of the connections between ASD and EDs. To date, no network analyses have investigated transdiagnostic ED symptoms in relation to ASD symptoms, and the 2020 study looking at ASD and AN was done in a clinical population, further limiting applicability.

As such, the goal of this study was to fill this gap of knowledge about the relationship between ASD and ED symptoms, by building on the findings of the 2020 paper. This study used a network analysis to investigate the relationship between ED and ASD symptoms, broadening to scope to disordered eating symptoms of all types, beyond diagnosed AN.

Research Questions

What are the relationships between symptoms of disordered eating and autism in college students? What are the bridge symptoms that connect disordered eating and autism symptoms?

Hypotheses

1. A network analysis will demonstrate a relationship between autism and ED symptoms, and find that there are bridge symptoms between the disorders.
2. Autism and ED bridge symptoms will revolve around one's relationship with food and social relationships, similar to the findings in a 2020 paper that found bridge symptoms to be struggling to eat around others and with self-confidence (Kerr-Gaffney et al., 2020).

3. By using a larger sample and scope, we may also find bridge symptoms relating to food rules connecting ED and ASD. For example, having limited foods one can eat, or limited ways they can be prepared.

Method

Participants and Procedures

Participants were undergraduates over the age of 18 who consented to completing the survey online on Qualtrics. Participants answered self-report questions about their thoughts, feelings, and behaviors. The study included 3 adherence checks to ensure the participants were paying attention (e.g., "choose always"). Students who participated were able to receive extra credit in courses for their voluntary participation. The procedures were approved by the university's Institutional Review Board.

Of the original 198 participants who completed the measures, 10 had to be excluded due to failure to complete the survey, meaning that they either stopped after the consent, or in the case of one participant, filled out most of the survey, but none of the AQ or EDE-Q questions (7 participants), age below 18 (1 participant), or failure to pass the three validity checks (2 participants). The remaining 188 participants were included in analyses.

Measures

Demographics. Participants were asked to report their age, gender, race, ethnicity, sexual orientation, and year in college. Participant ages ranged from 18-43 with their mean age being 22.11 years old. The majority of participants reported being White (86%), cisgender women (68%), and straight/heterosexual (73.22%). 55% of participants were in their first year of college, 17% were sophomores, 15% were juniors, 9% were seniors, and 2% were in their 5th year or beyond.

Autism Symptoms. The Autism Spectrum Quotient-Short (AQ-S) is a 28-item self-report measure used to assess the prevalence of traits associated with autism in an adult with normal intelligence. The AQ-S is a shortened version of the Autism Spectrum Quotient and is highly correlated with the full-length AQ (Hoekstra et al., 2011). The AQ-S uses a 4-point Likert type scale ranging from 1- 'definitely agree' to 4-'defintely disagree' on most questions and the inverse on questions 3, 5, 8, 11, 19, 21, and 22, which were reverse scored. Participants can score anywhere from 28-112, with 70 points being the more stringent cut-off point for distinguishing autistic traits (Hoekstra et al., 2011). The AQ-S has sensitivity and specificity values of .94 and .91, respectively (Hoekstra et al., 2011). Cronbach's alpha for the AQ-S in this study was 0.70, indicating acceptable reliability.

Eating Pathology. The Eating Disorder Examination Questionnaire 7 (EDE-Q7) is a seven-item questionnaire used to measure the severity of ED pathology derived from the 28-item EDE-Q (Aardoom et al., 2012; Fairburn & Beglin, 1994; Grilo et al., 2015,). The seven items ask participants to report the frequency of cognitions and behaviors over the last 28 days using a seven-point Likert scale, with higher scores indicating greater pathology. The EDE-Q7 has 3 subscales: dietary restraint (3 items), shape/weight overevaluation (2 items), and body dissatisfaction (2 items). The alpha level in this study was between 0.84 and 0.95 for the subscales, indicating good to excellent reliability.

Analytic Plan

Determining an appropriate sample size for a network analysis is difficult, as few guidelines exist and traditional methods like power analyses are not possible. Currently, the best and most consistent recommendation is to use the largest sample size possible and ensure the

network is stable (Levinson et al., 2018; Epskamp, Borsboom, & Fried, 2018). As such, this study aimed to collect a sample of at least 300 participants for the research given the number of items included in the network analysis.

This network was analyzed using custom scripting in the program R and the statistical packages *qgraph, bootnet,* and *networktools* made for R (R Core Team, 2022; Epskamp et al., 2012; Epskamp, Borsboom, & Fried, 2018; Jones, 2022). To identify my nodes, the goldbricker function of *networktools* was used to evaluate the 28 items in the AQ-short and the 7 items in the EDE-Q7 and determine what individual items from the questionnaires measure the same construct. After finding the pairs of items that measure the same construct, one of the two items was dropped from the network analysis. This is important as the network analysis assumes that each node in the network represents a distinct construct, so nodes that measure the same construct will artificially alter the results of the network analysis. Then *qgraph* was used to run a least absolute shrinkage and selection operator (LASSO) with a tuning parameter set at 0.25, and use this to estimate a partial correlation network with weighted edges (Friedman, Hastie, & Tibshirani, 2008). The LASSO method and parameter of 0.25 are used to limit the number of spurious relationships with the goal of only seeing the most important empirical relationships in the network (Hevey, 2018). LASSO was also used to ensure network stability by improving the specificity and obtain a more interpretable network (Epskamp, & Fried, 2018).

To further ensure network stability, the tool *bootnet* was used to preform bootstrapping—a process of repeated estimation of the model under sampled data to estimate the accuracy of the edge-weights or other statistics of interest. This provided 95% bootstrapped confidence intervals (CIs) and correlation stability (CS) coefficients. The CIs indicate sampling variation, with smaller intervals allowing for the easy estimation of the strength of edges, and edges with larger

intervals being hard to estimate. The CS coefficient checks the stability of expected influence (EI) and bridge EI if the network was reassessed with a subset of the current sample, indicating the proportion of the sample that could be dropped while maintaining a correlation >0.7 with the original sample (Epskamp, & Fried, 2018). Finally, the central nodes of the network and bridge symptoms were identified by calculating EI and bridge EI for each node in *networktools*.

Results

Missing values made up 0.18% of all questionnaire data, with 10 of the 19 missing values in the AQ-28 questionnaire, and 9 in the EDE-Q questionnaire. As these missing values made up 0.18% and 0.21% of the data in each scale, respectively, this missing data was replaced with the item mean, as recommended by Parent (2013) when less than 5% of an item is missing.

The mean score on the AQ-28 was 67.04, with nearly a quarter of all participants scoring above 70, the most stringent cut-off for distinguishing autistic traits on the AQ-28 (Hoekstra et al., 2011). The mean scores on the EDE-Q short subscales were 1.87 on the restraint subscale, 2.64 on the shape/weight overevaluation, and 2.97 on the body dissatisfaction subscale. These scores are all within one standard deviation of the mean of the expected score in a non-clinical population (Machado et al., 2020).

The data cleaning and analyses were done in R, and the R code can be found in the Supplementary Materials. To identify the nodes, the goldbricker function of *networktools* was used. This function evaluated all items in both questionnaires and determined what items from each questionnaire measure the same construct. Once the pairs of items that measure the same construct were found, the *net_reduce* command was used to drop one of the two items in each pair. Thus, one node measuring each construct remained. This step is important as the network

analysis assumes that each node in the network represents a distinct construct, so nodes that measure the same construct will artificially alter the results of the network analysis.

The R package *qgraph* was used to run a least absolute shrinkage and selection operator (LASSO) with a tuning parameter set at 0.25, allowing for the estimation of a partial correlation network with weighted edges (Friedman, Hastie, & Tibshirani, 2008). The LASSO method and parameter of 0.25 are used to limit the number of spurious relationships with the goal of only seeing the most important empirical relationships in the network (Hevey, 2018). Using LASSO ensures network stability by improving the specificity of the edges in the network, obtaining a more interpretable network (Epskamp, & Fried, 2018).

Network estimation and accuracy

The network of EDE-Q short and AQ-28 responses is shown in Figure 1, and the questions included in the network are shown in Tabel 1. Red nodes represent nodes from the AQ questionnaire, and blue nodes represent nodes from the EDE-Q short questionnaire. Green edges represent positive associations between nodes and red edges represent negative ones. The network found no edges shared between the EDE-Q and the AQ-28.

The expected influence (EI) correlational stability (CS) coefficient was found to be 0.596, and the bridge EI CS was found to be 0.516. According to a publication by Epskamp, Borsboom and Fried (2018), to be interpreted these coefficients must be above 0.25, and will ideally be above 0.5. As both the EI CS coefficient and bridge values are above 0.5 they can be interpreted. Bridge EI values are plotted in Figures 2. The strongest bridge EI1 and EI2 values were AQ question 12 and EDE-Q question 23 on both graphs. Along with this, the skew and kurtosis of the average AQ-28 scores was found to be roughly normal, but the EDE-Q scores were found to be somewhat right skewed, indicating that lower scores on the measure were more common. This

confirms what was seen in the mean scores of each measure, as the AQ-28 mean score of 66 is close to the middle possible score on the measure, but the EDE-Q subscale scores are all significantly below the middle possible scores on the subscales.

Discussion

This study hypothesized that the network analysis would find a connection between ASD symptoms and ED symptoms as they are represented by questions on the EDE-Q short and AQ-28. The specific expectation was finding connections between nodes relating to individuals' relationships with food and social relationships, and a possible relationship between eating behaviors and rigid systems/rules.

The results did not support the study hypotheses, as no edges were found between nodes on the different questionnaires. This lack of edges connecting the measures of ED symptoms and measures of ASD symptoms differs some from previous research, where a prior network analysis found bridge symptoms between ASD and AN that related to self-confidence, social situations with food, and others' perceptions of their body (Kerr-Gaffney et al., 2020). This prior study differed in its use of an exclusively clinical population, use of social responsiveness scale 2 instead of the AQ-28 to asses ASD, and smaller sample size. These differences may account for the difference in findings, as the change in measures may have altered somewhat the symptoms being assessed in the network, and the smaller sample may mean that some of these findings may be spurious (Constantin & Cramer, 2018). The lack of connection found between ASD and ED symptoms in this study may be due to the use of a non-clinical population. Most of the literature investigating the link between ASD and ED is done using clinical populations with diagnosed ED's, often while they are in treatment (Kerr-Gaffney et al., 2020; Vagni et al., 2016; Gesi et al., 2017). When compared to clinical populations, non-clinical populations have been shown to

replicate the network structure seen in clinical populations, but have less density than the networks of clinical populations (Vanzhula et al., 2019). The discrepancy seen here between this network made using a non-clinical populations and prior networks of clinical populations may be due to this difference in network density, as lower density results in less network connectivity, seen in this network.

Another possible contributor to the study's findings may be the scope of the study. Unlike the prior network analysis looking for AN-ASD connections, this study was not only done with a non-clinical population, but it also looked at transdiagnostic ED symptoms. While prior research as shown some correlations between ED and ASD broadly, the literature finding the strongest and most consistent associations has been research looking at AN and ASD specifically (Kerr-Gaffney et al., 2020; Westwood & Tchanturia, 2017). This may mean that there is not a strong connection between ASD and ED symptoms broadly. Research investigating this link between AN and ASD has frequently found social difficulties to be an important shared connection, and have a few explanations for this potential link. One prominent theory is that ASD behaviors are so common in those with AN because they are an epiphenomenon, meaning they are caused by the AN (Westwood & Tchanturia, 2017). This explains why parents of these patients often do not report ASD behaviors in early childhood, and why the behaviors are so common in clinical populations, but it does not explain why the ASD behaviors persist after recovery from AN (Kerr-Gaffney et al., 2020; Westwood & Tchanturia, 2017). Another hypothesis is that due to the cultural conception of ASD as a "male disorder", parents under-recognize and under-report ASD behaviors in childhood. This also explains the lack of reported ASD in childhood, and explains why the ASD behaviors continue after recovery from AN (Kerr-Gaffney et al., 2020; Westwood & Tchanturia, 2017).

Along with this, while the study had a broadened scope, the network analysis used only 7 items assessing for ED symptoms, and it is possible that the other items on the longer version of the EDE-Q that had stronger connection to the ASD symptoms measured in the AQ-28. While the items in the network do asses relationship with food and restriction, not all questions relating to food and restriction were included, and future research may benefit from a more expansive ED symptom network.

Finally, the lack of findings in this study may indicate that in a non-clinical population there is not a connection between ASD symptoms and ED symptoms. This would be a case for further research into both non-clinical and clinical populations, to investigate other possible reasons for the heightened rates of ASD symptoms in clinical ED populations. The lack of connection between ASD and ED in non-clinical populations may suggest a lurking variable as the cause of connection in clinical populations. This lurking variable could be the social difficulties associated with both ASD and ED- particularly AN, as these social difficulties would raise the scores on measures of ASD in ED populations, giving the appearance of elevated rates of ASD.

Strengths and Limitations

This study was one of the first to use a network analysis to examine possible relationships between ASD and transdiagnostic ED symptoms and helps to lay the groundwork for future studies using network analysis to evaluate connections between disorders. This is important because while the evidence of direct connections between the two disorders is still minimal, the evidence that having autism negatively impacts the efficacy of ED treatment is clear (Nielsen et al., 2015). Autistic people consistently report less success in ED treatment (Babb et al., 2021). It is crucial that more research is done looking at the interactions between ASD and ED's and that

methods of ED treatment are developed that work for Autistic people. The development of these new treatments must include Autistic people and Neurodiverse people, and must embrace neurodiversity instead of try to cure it (Schuck et al., 2021). If researchers and clinicians want to be able to treat and prevent ED's they need to reject a vison of sameness and embrace the diversity of the human experience. This study is important because it contributes to the research of ASD and ED symptoms, and can be used as a base for future work that will improve the care that autistic people with EDs receive.

This study has several limitations. First, the population of 188 is smaller than recommended for the number of nodes being evaluated. As this network includes 28 nodes the literature recommends between 300-500 participants to achieve reasonable network accuracy (Constantin & Cramer, 2018). The small population limits the accuracy and reliability of the network, lowering the replicability of this finding. Along with this small population, the next limitation that must be considered is the change from the planned use of the EDE-Q to the EDE-Q 7. The change was done to try and preserve network accuracy by removing the total number of items that will be transformed into nodes. The final limitation is the population demographics of the study, as the population is college students, the majority of which are white, cisgender, and women.

In conclusion, the goal of this study was to address the lack of research into the relationship between ASD and transdiagnostic ED symptoms, and provide support for future research into more effective treatments of ED in autistic people. The study is one of the first to look ASD and transdiagnostic ED using a network analysis. Future research should focus on investigating the link in clinical and non-clinical populations of a larger size, and on working

with autistic people with ED's to identify where they report overlap between their disorders and what issues they report in ED treatment.

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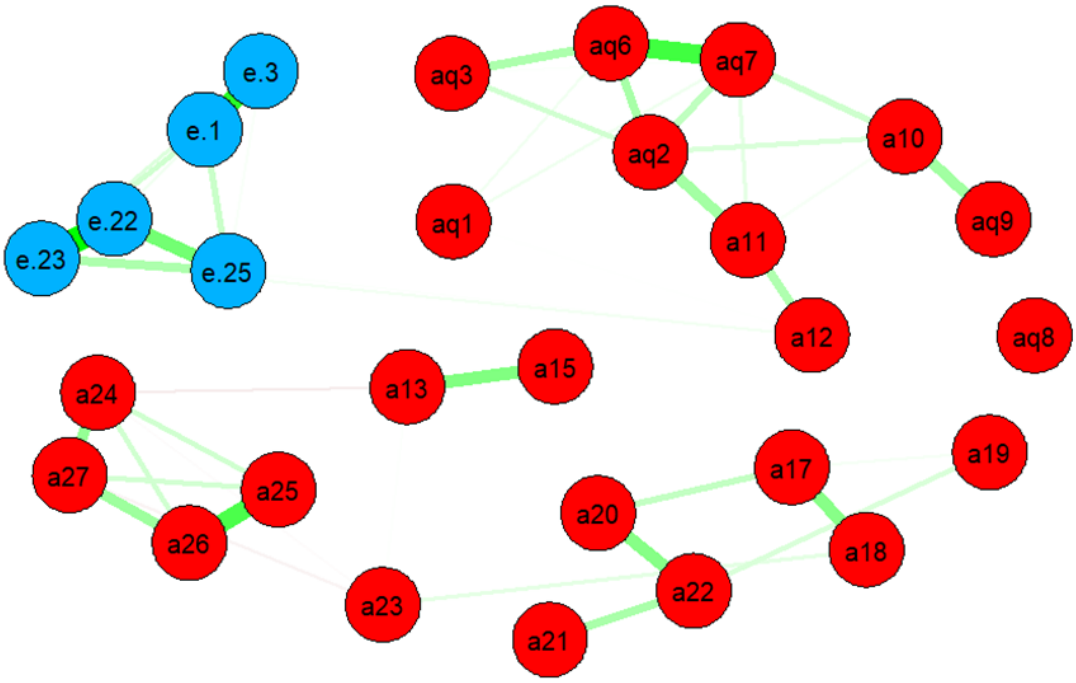

Figure 1. The network of EDE-Q short and AQ-28 responses

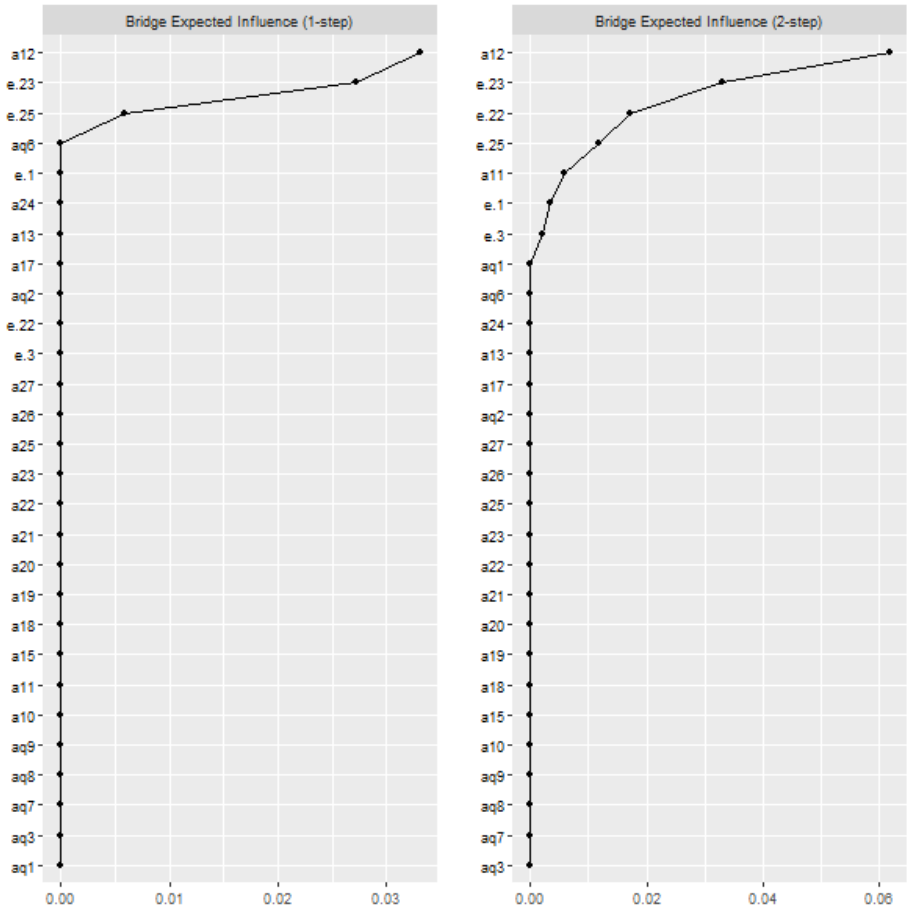

Figure 2. Bridge Expected Influence Coefficients

Tabel 1

Item numbers and associated questions in the network analysis

ITEM NUMBER	QUESTION
EDE-Q1	Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)?
EDE-Q3	Have you tried to exclude from your diet any foods that you like in order to influence your shape or weight (whether or not you have succeeded)?
EDE-Q22	Has your weight influenced how you think about (judge) yourself as a person?
EDE-Q23	Has your shape influenced how you think about (judge) yourself as a person?
EDE-Q25	How dissatisfied have you been with your weight?
AQ1	I prefer to do things with others rather than on my own
AQ2	I find social situations easy
AQ3	I would rather go to a library than to a party
AQ4	I find myself drawn more strongly to people than to things
AQ5	I find it hard to make new friends
AQ6	I enjoy social occasions
AQ7	I enjoy meeting new people
AQ8	I prefer to do things the same way over and over again
AQ9	It does not upset my if my daily routine is disturbed
AQ10	I enjoy doing things spontaneously
AQ11	New situations make me anxious
AQ12	I frequently get strongly absorbed in one thing
AQ13	I can easily keep track of several different people's conversations
AQ14	I find it easy to do more than one thing at once
AQ15	If there is an interruption, I can switch back very quickly
AQ16	Trying to imagine something, I find it easy to create a picture in my mind
AQ17	Reading a story, I can easily imagine what the characters might look like
AQ18	I find making up stories easy
AQ19	Reading a story, I find it difficult to work out the character's intentions
AQ20	I find it easy to work out what someone is thinking or feeling
AQ21	I find it difficult to imagine what it would be like to be someone else
AQ22	I find it difficult to work out people's intentions
AQ23	I find it easy to play games with children that involve pretending
AQ24	I usually notice car number plates or similar strings of information
AQ25	I am fascinated by dates
AQ26	I am fascinated by numbers
AQ27	I notice patterns in things all the time
AQ28	I like to collect information about categories of things

Appendix 1

```
#--------NETWORK-------#
library(igraph)
library(bootnet)
library(qgraph)
library(psych)
library(networktools)
library(ggthemes)

data2 <- as.data.frame(data[c(1:28,29,31,32,43,44,48,50)]) # data for
analysis

gb2 <- goldbricker(
  data2,
  p = 0.05,
  method = "hittner2003",
  threshold = 0.25,
  corMin = 0.5,
  progressbar = TRUE
)

reduced_edsrs2 <- net_reduce(data = data2, badpairs = gb2,
method="best_goldbricker")
corMat <- cor.smooth(reduced_edsrs2)
#png("network.png",width=1000,height=600)
graph2 <- qgraph(corMat,
                 graph = "glasso",
                 sampleSize = nrow(reduced_edsrs2),
                 layout = "spring",
                 tuning = 0.25,
                 cut=0,
                 groups= list(c(1:18,23:26,28), c(19:22,27)),
                 posCol="green",negCol="darkred",
                 colorblind_pal()) # max = 0.46
graph2$Edgelist

wm2 <- getWmat(graph2)
sum(wm2[upper.tri(wm2, diag=F)]!=0)  #95 edges (95 missing)
sum(abs(wm2[upper.tri(wm2, diag=F)])) /2 #4.625315

centralityPlot(list(EBIC0.25=graph2))
cor(reduced_edsrs2, use= "complete.obs")
groups <- list(AQ = c(1:18,23:26,28), EDEQ = c(19:22,27))

#plot expected influence centrality
```

```
#png("EIcentrality.png",width=1000,height=600)
centralityPlot(graph2, include = "ExpectedInfluence", orderBy =
"ExpectedInfluence")

#plot bridge metrics
b <-  bridge(graph2, communities = groups)
#png("EICSbridge.png",width=600,height=600)
plot(b, order = "value", include = c("Bridge Expected Influence (1-
step)", "Bridge Expected Influence (2-step)"))

#Estimates network for bootstrapping
Network_reduced <- estimateNetwork(reduced_edsrs2, default = 'cor')

#Bootstraps, nonparametric
Results_bridge_ei_nonparameteric <- bootnet(Network_reduced,
default='EBICglasso', type='nonparametric', nBoots = 1000, nCores = 8,
statistics = c('bridgeExpectedInfluence', 'edge', 'strength',
'expectedInfluence'))

# Plot bootstrapped edge CIs:
plot(Results_bridge_ei_nonparameteric, labels = TRUE, order =
'sample',statistics = 'edge')

# Plot significant differences (alpha = 0.05) of edges, expected
influence, expected bridge influence
plot(Results_bridge_ei_nonparameteric, "edge", plot =
"difference",onlyNonZero = TRUE,
     order = "sample")
plot(Results_bridge_ei_nonparameteric, "bridgeExpectedInfluence", plot
= "difference")

# Case-drop bootstrap. This is needed to compute cs-coefficients and
stability
Results_case_bridge_ei <- bootnet(Network_reduced, nBoots = 2000,
nCores = 10,
                                  type = "case", default='EBICglasso',
statistics = c('bridgeExpectedInfluence', 'edge', 'strength',
'expectedInfluence'))
# Plot centrality stability:
plot(Results_case_bridge_ei, statistics =
c('bridgeExpectedInfluence','expectedInfluence'))

# Compute CS-coefficients:
corStability(Results_case_bridge_ei)

big<- which(graph2[["Edgelist"]]$weight>0.20)
```

```
graph2[["Edgelist"]]$to[c(big)]
graph2[["Edgelist"]]$from[c(big)]
```