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Using Network Analysis to Identify Central Symptoms of Adolescent Depression

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Abstract

Objective: Experiencing depression symptoms, even at mild to moderate levels, is associated with maladaptive outcomes for adolescents. We used network analysis to evaluate which symptoms (and associations between symptoms) are most central to adolescent depression.

Method: Participants were part of a large, diverse community sample (N = 1,409) of adolescents between the ages of 13-19 years old. Network analysis was used to identify the most central symptoms (nodes) and associations between symptoms (edges) assessed by the Children's Depression Inventory (CDI). We also evaluated these centrality indicators for network robustness using stability and accuracy tests, associated symptom centrality with mean levels of symptoms, and examined potential differences between the structure and connectivity of depression networks in boys and girls.

Results: The most central symptoms in the network were self-hatred, loneliness, sadness, and pessimism. The strongest associations between symptoms were sadness-crying, anhedonia-school dislike, sadness-loneliness, school work difficulty-school performance decrement, self-hatred-negative body image, sleep disturbance-fatigue, and self-deprecation-self-blame. The network was robust to stability and accuracy tests. Notably, symptom centrality and mean levels of symptoms were not associated. Boys and girls' networks did not differ in levels of connectivity, though the link between body image and self-hatred was stronger in girls than boys.

Conclusions: Self-hatred, loneliness, sadness, and pessimism were the most central symptoms in adolescent depression networks, suggesting these symptoms (and associations between symptoms) should be prioritized in theoretical models of adolescent depression and could also serve as important treatment targets for adolescent depression interventions.

Keywords

adolescent; depression; network analysis

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Adolescents who experience depression have more costly and frequent physical health problems (Wright et al., 2016), attend school less frequently (King & Bernstein, 2001), and are at increased risk of attempting suicide (Lewinsohn, Rohde, & Seeley, 1994). It has been estimated that 11.7% of adolescents experience major depressive disorder (MDD) before they turn 19 (Merikangas et al., 2010), and the number of adolescents who have experienced MDD in the past year has increased approximately 23% from the year 2005 to 2014 (Mojtabai, Olfson, & Han, 2016).

Even among adolescents who do not meet criteria for MDD, endorsing more symptoms of depression is associated with decreased quality of life (Bertha & Balázs, 2013), increased functional impairment, and increased suicidality (Balázs et al., 2013). Higher levels of depression symptoms in adolescence also predict increased risk of MDD and suicide attempts in adulthood (Fergusson, Horwood, Ridder, & Beautrais, 2005), indicating that depression in adolescence is associated with important clinical outcomes even when symptoms are not at clinically significant levels.

One of the major challenges for understanding depression during adolescence (and in general) is the heterogeneous collection of symptoms (Olbert, Gala, & Tupler, 2014; Zimmerman, Ellison, Young, Chelminski, & Dalrymple, 2015), leading to a highly variable presentation of symptoms. For example, in a study of 3,703 outpatient adults with MDD, 48.6% of the 1,030 potential symptom profiles were only endorsed by one individual, and the most common symptom profile was endorsed only by 1.8% of the outpatients (Fried & Nesse, 2015a). This heterogeneity can be consequential, as different symptoms of depression can be associated with as little as 0.7% impairment of general functioning (hypersomnia) to over 20% impairment of general functioning (sad mood) in adults with MDD (Fried & Nesse, 2014). This work clearly suggests that some symptoms of depression may be more important than others, at least in terms of predicting co-occurring impairment.

The presence of certain depression symptoms (e.g., sad mood, social withdrawal, loss of self-esteem) also appears to be more strongly related to overall sum scores of depression, while other symptoms (e.g., weight gain, irritability, insomnia) can have limited associations with sum scores of depression in adults with MDD (Faravelli, Servi, Arends, & Strik, 1996). This pattern of results extends to non-adult samples as well; children and adolescents endorsing a sadness or low mood symptom are more likely to have conduct problems, while those who endorse worrying and somatic symptoms are not (Maasalo, Wessman, & Aronen, 2017). Therefore, some symptoms may be of more importance to depression than others, and solely relying on sum or mean scoring that gives equal weight to all symptoms may be misleading when trying to determine depression severity (Fried & Nesse, 2015b).

However, so far, traditional approaches to studying psychopathology implicitly assume symptoms of mental health disorders to be manifestations of an underlying disease (Brown & Barlow, 2005; Insel, 2014). For example, feelings of sadness and self-hatred are seen as outcome features of an underlying disease, namely depression, in the same way the pustules, fever, and headache are outcome features of the smallpox virus (Fried, 2015). For this assumption to be correct, feelings of sadness and self-hatred should only co-occur at the same time that someone is experiencing depression. Consequently, one would expect

individuals who are not experiencing depression to exhibit no significant association between feelings of sadness and self-hatred. In fact, symptoms such as these continue to demonstrate robust associations with one another in adults even in the absence of a major depressive episode (Borsboom, 2008; Santos, Fried, Asafu-Adjei, & Ruiz, 2017). This suggests that a latent factor model of depression may not fully capture the complexity of the depressive phenomenon.

More recently, the network approach to psychopathology supposes that the disorder is constituted of co-occurring symptoms and their tendency to causally reinforce one another (Borsboom & Cramer, 2013; Boschloo et al., 2015). Therefore, symptoms are not outcome factors of an underlying disease; symptoms and the associations between them are the disease itself (McNally et al., 2015). Hence, certain symptoms, defined as *nodes*, may have strong causal links with one another, relate significantly to other symptoms in the network, and serve as a bridge between two other symptoms in the network. Similarly, specific links between two symptoms, defined as *edges*, may be substantially stronger and more influential than other edges. A strong edge in a network can be thought of as strong association between two symptoms that are likely either to occur or non-occur simultaneously. In other words, in the context of such an important edge, the activation/presence of one symptom predicts the activation/presence of the other symptom (i.e., pathological network), but also the deactivation/absence of one symptom predicts the deactivation/symptom of the other one (i.e., healthy network).

Analyzing depression symptoms from the perspective of network analysis allows us to go beyond the current mean level of symptoms and understand which symptoms might be particularly central to experience of depression (Borsboom, 2017). Indeed, network approaches to depression can better prospectively identify which adults in a community sample will develop MDD over 6 years than traditional mean symptom level approaches (Boschloo, Borkulo, Borsboom, & Schoevers, 2016). Moreover, influential symptoms and the related edges may have an outsized impact on clinical risk, trajectory, and outcomes (Hofmann, Curtiss, & McNally, 2016), though more investigation, especially in developmental periods where depression prevalence increases, is needed. Nevertheless, symptom centrality and symptom mean levels appear to measure different aspects of depression, as they are often weakly associated (Yang et al., Under Review), and mean levels of symptoms can change without changes in the network structure.

To highlight which symptoms may be most influential in the depressive network, three major centrality indices are usually examined: *betweenness*, *closeness*, and *strength*. Betweeness quantifies how efficiently a certain node connects other nodes, while closeness indicates how easily the flow of information reaches all the other nodes starting from a specific node. Finally, strength represents how well connected with the rest of the network a node is. Taken together, these indices point out which nodes are the most central and more likely to exert influence on the rest of the network (Valente, 2012).

When strength of symptoms is used to identify central symptoms, network analyses in adult, clinical samples have identified sadness (Beard et al., 2016; Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016; Santos et al., 2017), loss of interest (Bringmann, Lemmens,

Huibers, Borsboom, & Tuerlinckx, 2015), and fatigue (van Borkulo et al., 2015) as central symptoms. In contrast, few depression symptom network analyses have been conducted in adolescents. The only previous network analysis study examined a community sample of 10–12 year olds who reported on emotional and behavioral problems (Boschloo, Schoevers, van Borkulo, Borsboom, & Oldehinkel, 2016). Importantly, that study primarily examined the patterns of overlap and non-overlap between symptoms in different problem domains (e.g. between internalizing and externalizing disorders) and did not directly examine different indices of symptom centrality. Thus, the central symptoms of adolescent depression have not yet been identified.

There are reasons to believe that depression symptom networks may be different in adolescents compared to adults. Adolescents are not just smaller adults or larger children (Crosnoe & Johnson, 2011), as they are experiencing a cascade of social and biological transitions that could impact their experience of depression and its symptoms (Crone & Dahl, 2012). Therefore, assuming adolescents have similar central symptoms of depression to adults or preadolescents without empirical investigation would be unwise. Further, other symptom networks (e.g. PTSD) differ across developmental periods (Russell, Neill, Carrión, & Weems, 2017), and specific features of depression can differ across the lifespan (Hammen, Garber, & Ingram, 2001). For example, self-hatred, or low self-esteem, is more common in adolescents than adults (Carlson & Kashani, 1988). Hence, investigating depression symptom networks in adolescence can help elucidate which depression symptoms may be particularly important during this transitional phase of development.

These adult samples were also primarily drawn from clinical populations, which in some cases can limit the generalizability of findings to non-clinical populations (Kukull & Ganguli, 2012). There is some evidence to suggest, however, that the network structure will be similar in a non-clinical sample. For example, there is evidence that network structures do not differ based on whether one meets the criteria for diagnosis or not, though whole network connectivity at clinical vs. non-clinical mean levels of symptoms may differ (Santos et al., 2017). So, although it is important to examine adolescent depression networks in a non-clinical sample directly, we might still expect some overlap in network structure with adult, clinical samples.

Finally, no network analysis (in adults or adolescents) to our knowledge has examined whether the network of depression symptoms differs across sexes, an investigation that might be especially important in an adolescent sample. Gender differences in depression mean levels are not evident in children, but girls display much higher mean levels than boys starting in adolescence (Hankin et al., 2015). In fact, the lifetime prevalence of MDD in adolescent girls is nearly twice the lifetime prevalence in boys (Merikangas et al., 2010), and this disparity continues into adulthood (Kessler et al., 2005). There may be differences in the depression networks between sexes that help explain this increased vulnerability for girls.

The present study will examine item-level depression symptoms using network analysis among a large sample of adolescents recruited from several high schools in the southwestern United States. We will evaluate which symptoms and associations between symptoms are most influential in the network of depression symptoms and evaluate how these symptoms

relate to their current mean level. We will also compare the symptom networks of adolescent boys and adolescent girls.

Method

Participants

The sample was racially diverse (Race/Ethnicity: 36.62% White/Caucasian, 26.33% Hispanic, 7.38% Asian, 2.63% Black, 0.92% Middle Eastern, 1.84% Other, 14.55% Multi-Racial, 9.73% missing) adolescents (Age 14.35 ± 0.66 years old; range: 13 – 19 years; < 16 years = 96.3% and ≥16 years = 3.7%); with approximately even sex representation (52.8% female, 45.85% male, 1.35% missing). Socioeconomic status of the sample as indexed by maternal education was diverse as well (9.08% did not finish high school, 10.07% high school or equivalent, 7.95% some college courses but no college degree, 4.68% associate's degree, 22.36% bachelor's degree, 13.63% master's degree, 5.18% lawyer, doctor, or PhD, 8.58% don't know, 18.47% missing).

Participants were universally invited and self-selected into the study, drawn from United States high schools in primarily urban and suburban areas, and were contacted during one of their classes in school. The participants were informed of the opportunity to participate in the study by members of the research team, given a packet explaining what the study would involve, and given another packet for their parent to consent and the student to assent.

Students were offered a $5 gift card if they returned the form to the researchers, regardless of whether affirmative consent and/or assent was given. Students who returned forms indicating consent from a parent and assent from the student then completed the assessments in school using an online platform. Students also completed other assessments not relevant to the current study. Students assented to the research again while accessing the online platform. Parental consent and student assent rates, which were completed on the same form, were both on average 58.9%. The study was approved by the IRBs of all relevant institutions.

Measure

Depressive symptoms were evaluated with the Children's Depression Inventory (CDI; Kovacs, 1985). The CDI is a 27-item self-report questionnaire investigating a variety of cognitive, emotional, physiological, and interpersonal symptoms of depression, such as sadness, low self-esteem, sleep disturbance, and social withdrawal. Upon request of the local Institutional Review Board, suicidality (i.e., item #9) was not assessed. All items were measured on a 3-point Likert scale, ranging from 0 (i.e., "*I do most things OK*") to 2 (i.e., "*I do everything wrong*"). The CDI is widely used in research and clinical practice with children and adolescents, and it is a valid and reliable measure for depressive symptoms (for a review, see Klein, Dougherty, & Olino, 2005).

Statistical Analysis

We first inspected mean, standard deviation, kurtosis, and skewness of all the CDI items. Then, considering that the optimal way to model (skewed) trichotomous items in network

analysis is still under debate (Fried et al., 2015), we dichotomized the single item-level values to signify the absence (0) and presence (1) of depressive symptoms. Item values reporting "0" were recoded as absence of the symptom, whereas values reporting either "1" or "2" were recoded as presence of the symptom. The thirteen positively worded items present in the CDI were reversed prior the dichotomization. By doing so, it was possible to estimate an Ising model (van Borkulo et al., 2014).

In network analysis, each variable of the model is represented as a *node*, while the association between two nodes is shown as an *edge*. In the context of psychopathological networks, green edges indicate positive associations, whereas red edges indicate negative associations (Epskamp et al., 2012). Moreover, thickness and saturation of the edges signifies their magnitude. In our study, the network nodes were displaced in accordance to the force-directed placement algorithm (Fruchterman & Reingold, 1991), whereby nodes that are stronger and/or more connected are preferentially located in the center of the network, while less strong and/or less connected nodes are shown in the periphery. For the visualization of the all network, we used the *R*-package qgraph 1.4.3 (Epskamp et al., 2012).

In the context of binary nodes, it is possible to estimate network models with the Ising model (van Borkulo et al., 2014). Ising model can be conceived as a series of pairwise associations among binary variables, after controlling for all the other associations. More technically, it is a probabilistic model in which the joint distribution over K binary variables (i.e., CDI items) is represented using pairwise association parameters (i.e., edges) and threshold parameters related to the marginal probability of endorsement of any individual item.

In addition, as implemented by van Borkulo and colleagues (2014), the Ising model integrates *l*1-regularized logistic regressions with model selection based on extended Bayesian Information Criterion (i.e., eLasso based on the Extended Bayesian Information Criterion or EBIC; Ravikumar, Wainwright, & Lafferty, 2010). Based on sample size, this penalty approach shrinks small edge coefficients to zero, thus allowing more conservative and sparse networks (i.e., many edges are absent).

In sum, eLasso Ising model leads to two main benefits: 1) the edge between two nodes cannot spuriously be due to a third node (i.e., conditionally dependence) and are more likely to be present in the population; 2) small/negligible edges are shrunken to zero, thus determining a network model that is sparser and easier to interpret. The binary network was fitted using the *R*-package IsingFit 0.3.1 (van Borkulo et al., 2014).

To highlight which symptoms may be most influential in the depressive network, we computed three major centrality indices. *Betweenness* indicates the number of shortest paths connecting any two symptoms, while *Closeness* is computed as the inverse of the sum of the total length of all the shortest paths between a specific node and the rest of the network. Finally, *Strength* amounts to the sum of the absolute weights of the edge connecting the node to all the other nodes (Valente, 2012).

It is also crucial to determine the robustness of psychopathological networks. To do so, we thoroughly investigated the accuracy and stability of our Ising model. We adopted a two-fold

approach: i) centrality stability and bootstrapped difference test for centrality indices; ii) edge accuracy and bootstrapped difference test for edges (Epskamp, Borsboom, & Fried, 2017).

First, centrality stability refers to the order stability of centrality indices (e.g., betweenness, closeness, and strength), after subsetting an increasing amount of observations (i.e., *m out of n bootstrap*; Chernick, González-Manteiga, Crujeiras, & Barrios, 2011). Centrality stability was operationalized with the correlation stability coefficient (i.e., *CS-coefficient*). CS-coefficient represents the maximum proportion of cases that can be dropped to still obtain a 95% probability that the correlation of the ranking between the original network and the case-subset network will amount to 0.7 (very large effect; Cohen, 1977). Epskamp and colleagues (2017) recommend only interpreting centrality indices with a CS-coefficient above 0.25, but preferentially above 0.5. Then, significant differences among centrality indices were computed as 95% non-parametric bootstrap confidence intervals (CIs; 1000 bootstrap samples) of the difference between each pair centrality indices. If zero is included in CI, the two nodes do not differ.

Second, edge-weights accuracy was estimated with with 95% bootstrap CIs. Larger CIs indicate reduced precision in the estimation of the edges, whereas narrower CIs imply a more trustworthy network. It is important to note that bootstrap CIs should not be used for testing the difference of each edge from zero, as regularized edges that are different from zero are strong enough to be considered in the model. Significant differences among edges were estimated as CIs of the difference between each pair of edges. If zero is included in CI, the two nodes do not differ. All the analyses described above were performed by means of the *R*-package bootnet 1.0 (Epskamp et al., 2017)

Then, we calculated Spearman's rank-order correlation between centrality indexes and both mean and standard deviation for each symptom. With the latter approach, we could test whether symptom centrality could potentially be due to the items' differential variability (Fried & Nesse, 2014). With the former analysis, it was possible to investigate whether or not the most central symptoms are also rated as the most severe.

Finally, in accordance to van Borkulo and colleagues (in submission), we also tested three specific differences between symptom networks in males and females. First, we tested whether males and females show different global strength in the depressive symptoms (i.e., invariant global strength hypothesis). Global strength is operationalized as overall network connectivity, namely the absolute sum of all the edges of the network. Second, we tested whether depression symptoms in males and females are characterized by a different network structure (i.e., invariant network structure hypothesis). The metric of interest is the maximum (absolute) element-wise difference among all the possible edges. Third, we investigated the possible difference at level of each specific edge between males and females, after applying the Holm-Bonferroni correction for multiple comparisons (i.e., invariant edge strength hypothesis).

All three of these network comparison tests rely on a similar permutation-based procedure. The specific metric of interest computed on the original (unpermutated) was tested against

the same metric computed on 1000 permutated data sets, where group membership was repeatedly and randomly rearranged across cases. The permutated data sets served as reference distribution under the null hypothesis and the *p*-value indicates the proportion of test statistics that are at least as extreme as the observed statistics (for further details, see van Borkulo et al., submitted). All the tests were performed with the *R*-package NetworkComparisonTest 2.0.1 (van Borkulo & Millner, 2016)

Results

Descriptive statistics

Mean, standard deviation, skewness, kurtosis, and frequency of the CDI symptoms are reported in Table 1. The overall mean and standard deviation of all symptoms is $M = 0.48$, $SD = 0.24$. The symptoms of fatigue and school work difficulty had the highest mean ratings, whereas the symptoms of getting into fights and disobedience had the lowest mean ratings. The sample's diversity in race/ethnicity and socioeconomic status (as indexed by maternal education) is representative of the population from which the participants were drawn.

Depressive symptoms network estimation, centrality stability tests, and strength comparison

First, we checked the level of item informativeness (i.e., standard deviation of the item) (Table 1). Importantly, the CDI item measuring the tendency of engagement into fights showed an extremely skewed distribution (skewness = 13.14), with 91.56% of participants reporting not getting into fights at all. Moreover, when compared to the mean level of informativeness of the CDI items (0.60 ± 0.11), the item *fights* (i.e., item #27) was very poorly informative (i.e., < 2.5 standard deviations). For all these reasons, this item was excluded from the subsequent analysis, and with the exclusion of the suicidal ideation item 25 items were included in the subsequent analyses[1].

The network of depressive symptoms, as estimated with the Ising model, is shown in Figure 1, while Figure 2 shows the network's centrality indexes, namely strength, betweenness, and closeness. We then investigated the stability in the order of the symptoms across the three centrality indices considered. While betweenness reported low stability (i.e., *CS-coefficient* = 0.206), closeness showed an acceptable level of stability (i.e., *CS-coefficient* = 0.283). Strength reported an excellent level of stability (i.e., *CS-coefficient* = 0.672). That is, by dropping approximately 67% of the observations, the order of the symptoms in terms of strength still correlated 0.7 with the original order. In other words, the ranking of the symptoms as indexed by strength is to be taken as strongly robust and trustworthy. Hence, we primarily focused on interpretation of symptom strength in the subsequent network analysis.

In terms of strength, *self-hatred* was statistically stronger from the majority of the other symptoms. *Loneliness*, *sadness*, and *pessimism* also appeared to be statistically stronger than

[1]All the analyses reported did not substantially change, when the item "fights" was included.

the majority of the symptoms in the network (Figure 3). Thus, these four symptoms appeared to be particularly important and central for understanding depression symptom networks in this sample. In contrast, several symptoms appeared to be rather marginal, such as *reduced appetite*, *indecisiveness*, and *somatic concerns*.

Accuracy and edge comparisons

The analysis of the accuracy of 300 edges, as implemented by means of non-parametric CIs, suggested that the precision of the edges was acceptable, with smaller CIs indicating more accurate estimation of the edges (Figure S1). Consequently, the parameters represented in the network model can be considered as reasonably accurate. Moreover, the analysis revealed that the strongest edges (e.g., *sadness-crying*, *anhedonia-school dislike*, *sadness-loneliness*, *school work difficulty-school performance decrement*, *self-hatred-negative body image, sleep disturbance-fatigue*, *self-deprecation-self-blame*) were statistically different from the majority of the other edges (Figure S2).

Symptoms mean levels, variability, and association with strength centrality index

In the whole sample, the depressive symptoms with the highest mean levels (i.e., third tertile) were *fatigue*, *school work difficulty*, *indecisiveness*, *irritability*, *low self-esteem*, *sleep disturbance*, *negative body image*, *pessimistic worrying*. It is notable that items' mean levels were unrelated to symptoms strength ($r_s = 0.028$). Further, symptom strength and symptom standard deviation were also not correlated ($r_s = -0.005$). Taken together, these findings imply that high centrality of symptom is unrelated to mean level of the symptom and variability.

Network and symptoms mean levels comparisons between sexes

Figure 4 shows the network built on the CDI symptoms across males (n = 646) and females (n = 744). While the test for network connectivity differences showed no statistical difference (global strength difference = 1.40; females global strength = 38.31, males global strength = 36.91; $p = 0.76$), the two networks were significantly different in terms of structure (maximum difference = 1.16; $p = 0.011$). To understand this finding, we checked the statistical difference at level of each single edge, between males and females, after applying the Bonferroni-Holm correction. The only edge to be statistically different was the *self-hatred-negative body image*, which was stronger in females than in males (1.50 – 0.34; $p < 0.001$).

Despite only a single network structure difference between sexes, there were numerous sex differences in mean levels of individual symptoms. Compared to males, females reported higher mean levels of *sadness*, *pessimism*, *pessimistic worrying*, *self-hatred*, *crying*, *irritability*, *indecisiveness*, *negative body image*, *fatigue*, *reduced appetite*, *somatic concerns*, *loneliness*, *lack of friendship*, and *low self-esteem* (Table S1). Males did not report higher mean levels in any of the CDI symptoms. In line with the findings in the whole sample, symptom strength was unrelated to items' variability in females ($r_s = 0.013$) and males ($r_s = 0.18$). Further, strength was not correlated with symptoms mean levels in either sexes ($r_s = -0.11$ in females and $r_s = 0.16$ in males). These results suggest that network symptom strength and mean levels of symptoms are largely independent in this sample.

Discussion

The results of our network analyses reveal, in an ethnically and socioeconomically diverse sample of adolescents, the most central depression symptoms and strongest associations between symptoms in adolescent depression. These results were robust to stability and accuracy tests, and provide a novel framework for understanding adolescent depression.

Self-hatred, the most central symptom in our adolescent depression network as indexed by strength, may be of particular relevance to adolescent depression because adolescence is a key time for identity development (Dahl, 2004). Forming a positive identity during this developmental period may be critical to preventing negative mental health consequences (Neff & McGehee, 2009). Indeed, increased negative self-referent thinking is emerging as a potent risk factor of depression risk in adolescents, as measured behaviorally (Connolly, Abramson, & Alloy, 2016) and with brain activity associated with the processing of negative self-referent information (Speed, Nelson, Auerbach, Klein, & Hajcak, 2016).

Further, the association between self-hatred and body image also appears to be particularly important for depression in adolescence, especially for girls. The edge connecting self-hatred and body image concerns is one of the central edges of the network, indicating that the association between these symptoms is a core feature of adolescent depression across sexes. Still, while the network of boys and girls displayed identical levels of overall network connectivity, there was a stronger edge between self-hatred and body image concerns among girls. Besides, girls also reported higher mean levels of these two specific symptoms. Taken together, these results may indicate an area of the network that has a higher chance of being pathological. Notably, interventions targeting body dissatisfaction in both sexes can decrease depressive symptoms (Bearman, Stice, & Chase, 2003), while it is still unclear whther such interventions may weaken the association between self-hatred and body image concerns, perhaps specifically in girls.

Loneliness was also an important symptom in the depression network. This may be especially true as adolescents move further through puberty, as neural and hormonal changes associated with puberty also appear to drive a need for affiliation with others (Sisk & Foster, 2004). Indeed, chronic feelings of loneliness appear to originate in childhood and adolescence (Ernst & Cacioppo, 2000). The central edge connecting loneliness and sadness suggests that the association between these two symptoms, while each central on their own, further reinforces the importance of these two symptoms.

It is interesting that several of the central symptoms are consistent with Beck's descriptive cognitive model of depression (Beck, 2002), where negative thoughts about the self (i.e., self-hatred), the world (i.e., loneliness), and the future (i.e., pessimism) along with negative mood (i.e., sadness) conspire to exacerbate each other and maintain negative feelings and symptoms (Beck, 2002). Pessimism about the future is an important feature of Beck's model and, consistent with the current study, recent empirical evidence indicates that pessimism may be a stronger predictor of change in depression symptoms for younger rather than older individuals (Armbruster, Pieper, Klotsche, & Hoyer, 2015). Higher pessimism in adolescents is related to higher depression symptoms at any given moment (Piña-Watson & Abraído-

Lanza, 2017), and also predicts who is less likely to respond to interventions designed to prevent major depressive episodes (Weersing et al., 2016).

As the central symptoms of adolescent depression correspond well with Beck's (2002) model of depression, examining whether interventions based on that model improve symptoms central to adolescent depression could be a generative process for both theoretical understanding and clinical application. Evidence-based treatments for adolescent mental health difficulties, including depression, tend to reduce symptomatology more for the problem area targeted by the treatment than for other types of symptoms (Weisz, Jensen-Doss, & Hawley, 2006). Given that CBT interventions tightly correspond with the central depression symptoms in adolescence, additional work, perhaps using extant datasets, should examine whether CBT interventions change central depression symptoms or associations between them.

There is also some evidence that while current pharmaceutical and talk-therapy approaches may decrease mean levels of depressive symptoms in adults, they do not change node centrality or connectivity relative to control groups (Snippe et al., 2017). While this research is in early stages, a change in mean level but not in network connectivity could help explain the high relapse or recurrence rate of MDD following treatment in adolescents (Kennard, Emslie, Mayes, & Hughes, 2006). In keeping with this, our study provides a network framework useful for evaluating whether current treatments effectively target central elements of depression and a roadmap of desirable symptom targets for future interventions (Fried, 2015).

A related important direction will be to examine the temporal dynamics of activating and deactivating central symptoms and associations. For example, treatments that deactivate a central symptom or association could lead to spreading deactivation of other less central elements and be more effective in treating the disorder (Hofmann et al., 2016). On the other hand, perhaps treatments that target deactivating central symptoms or associations too exclusively will be less effective, as their strong associations with other symptoms could lead to greater risk of subsequent reactivation. So, while interventions that specifically target self-hatred, loneliness, sadness, and pessimism might efficiently and effectively reduce depression symptoms, increase likelihood of recovery, and possibly reduce the risk of relapse, these possibilities remain open empirical questions.

The lack of a association in this sample between strength of a symptom and the mean level of the same symptom indicates that network analysis provides valuable information about symptoms above and beyond mean-level approaches. For example, girls in our sample had higher mean levels than boys on 14 symptoms but did not exhibit large structural differences in their depression networks. This relative similarity of structure implies that while mean-level expression might be different for adolescent boys and girls, their depression experiences share most central features. Therefore, work that further addresses how the underlying structure and current mean level expression of depression in adolescents differs could provide value for understanding sex differences in depression and perhaps other areas of theoretical interest as well.

Given the cross-sectional nature of our data, future prospective designs that specifically attend to the network structure of adolescent depression could provide further unique insights. Longitudinal designs with adolescents could elucidate whether self-hatred influences body image concerns, body image concerns influences self-hatred, or whether this is a reciprocal association. For example, adults in treatment who completed the BDI-II depression inventory weekly over 11 weeks demonstrated mutually reinforcing associations between the depression symptoms assessed over time (Bringmann et al., 2015).

One limitation, common to all network analyses, is that if two nodes (symptoms in this case) of a network are closely related they could be measuring the same construct (i.e., topological overlap). In such cases there is the potential for misinterpreting a network (Fried & Cramer, 2016). In our study, the central associations between school work difficulty-school performance decrement and self-deprecation-self-blame may be examples of this phenomenon, as there is significant overlap in the wording and response options in these items, and these associations should therefore be interpreted cautiously. For this reason, these associations were not strongly emphasized in the findings from the current study. The other most central edges, sadness-crying, anhedonia-school dislike, sadness-loneliness, self-hatred-negative body image, and sleep disturbance-fatigue, appear appropriate for substantive interpretation.

Another potential limitation of these analyses is the relatively low mean level of depression symptoms. There is some preliminary evidence that symptom network connectivity, though not necessarily structure, differs between clinically and non-clinically depressed populations. For instance, the structure of depression symptom network in pregnant women did not differ depending on whether the women had MDD or not, but women with MDD did display stronger network connectivity (Santos et al., 2017). Therefore, the connectivity of our network analysis may not fully generalize to clinical adolescent samples, as connectivity between clinical and non-clinical samples may differ. On the other hand, there is some evidence that more depressed populations might have less connected depression networks. High depression sum scores require up to six latent factors to adequately fit the data (Fried, et al., 2016), an indication of a less connected network (Kruis & Maris, 2016). So, especially in relation to connectivity analyses, further investigation using similar network techniques that involves adolescents recruited from clinical settings may be warranted.

Also, these results provide some evidence that the central features of depression in adolescence have some overlap with central features of adult depression (e.g., sadness) but also have central features potentially unique to adolescence (e.g., pessimism). One limitation, however, is that, for the purpose of comparison, there is only one other network analysis of depression in exclusively non-clinically diagnosed persons.(Boschloo, Borkulo, et al., 2016) known to these authors, and in this study, participants were all adults. Additionally, the perceived differences in network structure between adolescents and adults could be due to differences in which symptoms of depression are assessed; there are some items (e.g., school difficulty) present on the CDI that do not appear on the adult depression scale used in the adult sample. While it is not uncommon for depression scales to have minimal content overlap even within developmental periods (Fried, 2017), having different types of content in scales can affect which symptoms and associations between them end up

being more central in network models (Fried & Cramer, 2016). Therefore, any interpretations of symptoms unique to adolescence as compared to other developmental stages should be preliminary.

Also, these results come primarily from adolescents below 16 years of age, who are typically earlier in pubertal development. The network structure could differ based on pubertal development, as risk for depression generally increases throughout puberty even after controlling for age (Angold, Costello, & Worthman, 1998). Pubertal status, but not age, better predicts females showing higher mean levels of depression symptoms than males (Angold et al., 1998); so, network comparisons among girls and boys may have shown more differences if more adolescents later in puberty were included.

Overall, these results indicate that the most central symptoms in adolescent depression are self-hatred, loneliness, sadness, and pessimism. Several of these symptoms had strong associations with other symptoms (e.g., sadness-crying, sadness-loneliness, self-hatred-negative body image), suggesting that these symptoms may be of central importance to the experience of depression in adolescence. This knowledge could inform a more nuanced understanding of depression at this particular stage of development and may provide a framework for evaluating interventions that target adolescent depression. The increased association between self-hatred and body image concerns among adolescent girls seems important and novel, and may inform new theoretical considerations for sex differences in depression, especially considering that sex differences in depression tend to emerge at this point in development (Nolen-Hoeksema & Girgus, 1994).

Continuing to employ the most up to date network analytic techniques, like the accuracy and stability tests employed in this study, with longitudinal data sets in clinical and non-clinical samples across development will also facilitate greater understanding of both adolescent depression networks and their clinical utility. A network approach to adolescent depression may help us further our understanding of both how to properly characterize this heterogeneous disorder while also helping reduce its impact on this particularly at-risk population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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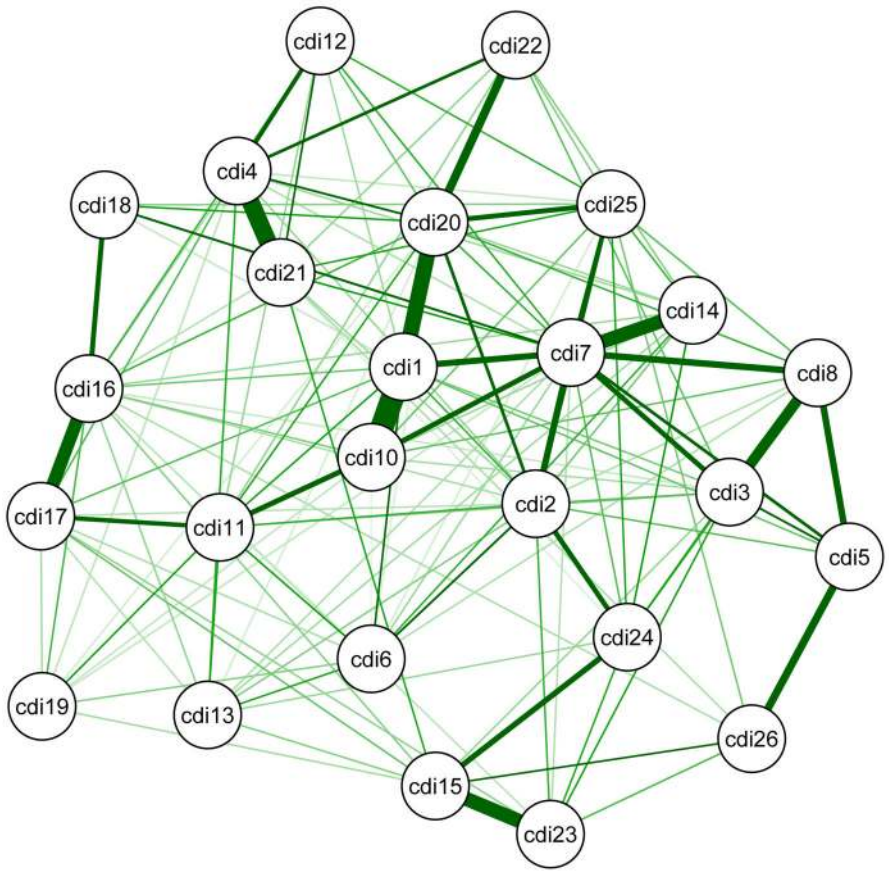

cdi1: Sadness
cdi2: Pessimism
cdi3: Self−deprecation
cdi4: Anhedonia
cdi5: Misbehavior
cdi6: Pessimistic worrying
cdi7: Self−hatred
cdi8: Self−blame
cdi10: Crying
cdi11: Irritability
cdi12: Social withdrawal
cdi13: Indecisiveness
cdi14: Negative body image
cdi15: School work difficulty
cdi16: Sleep disturbance
cdi17: Fatigue
cdi18: Reduced appetite
cdi19: Somatic concerns
cdi20: Loneliness
cdi21: School dislike
cdi22: Lack of friendship
cdi23: School performance decrement
cdi24: Low self−esteem
cdi25: Feeling unloved
cdi26: Disobedience

Figure 1.
Estimated network model for dichotomized depressive symptoms in the total sample (n = 1409).

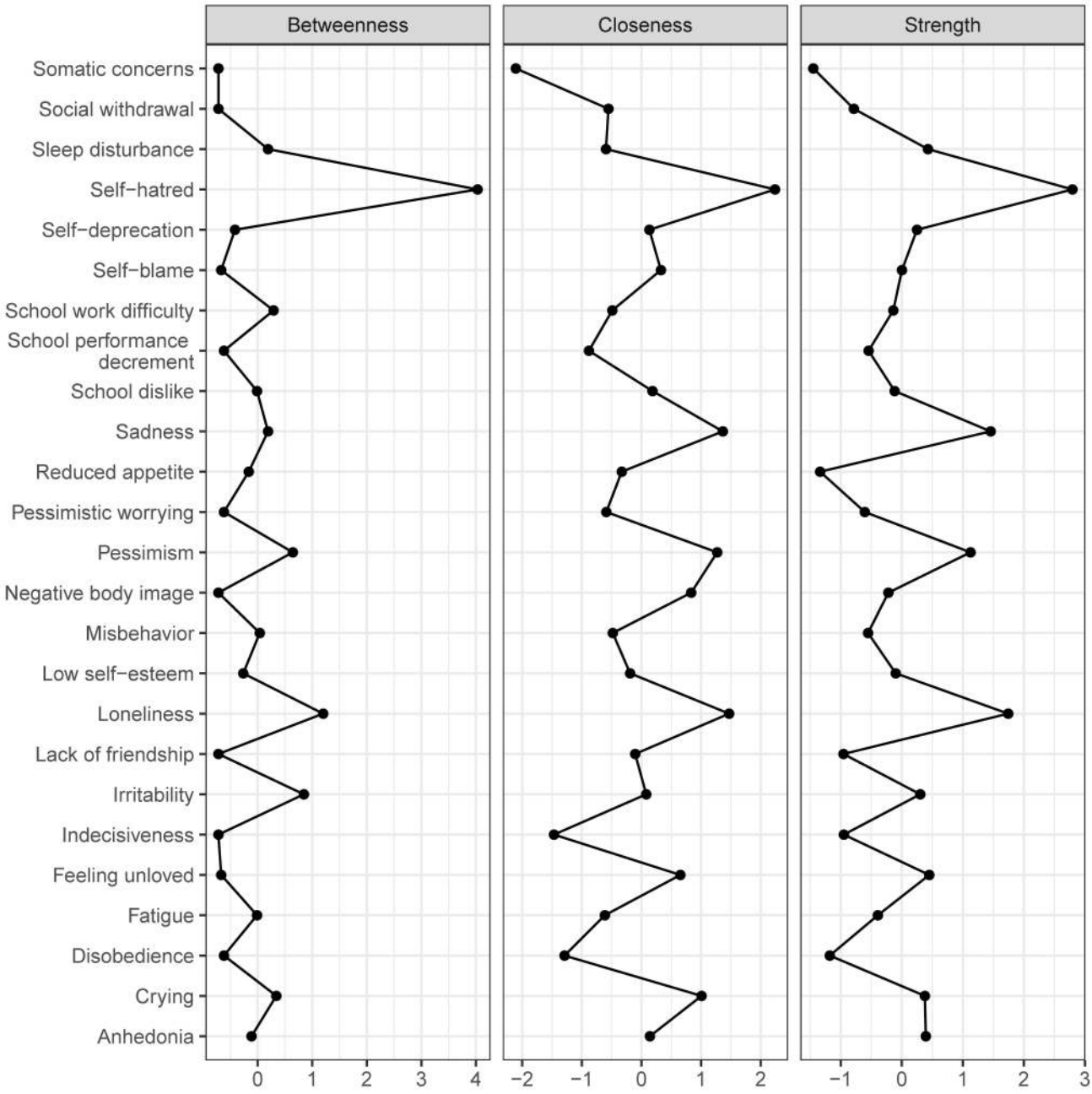

Figure 2.
Centrality indices of depressive symptoms, shown as standardized values *z*-scores.

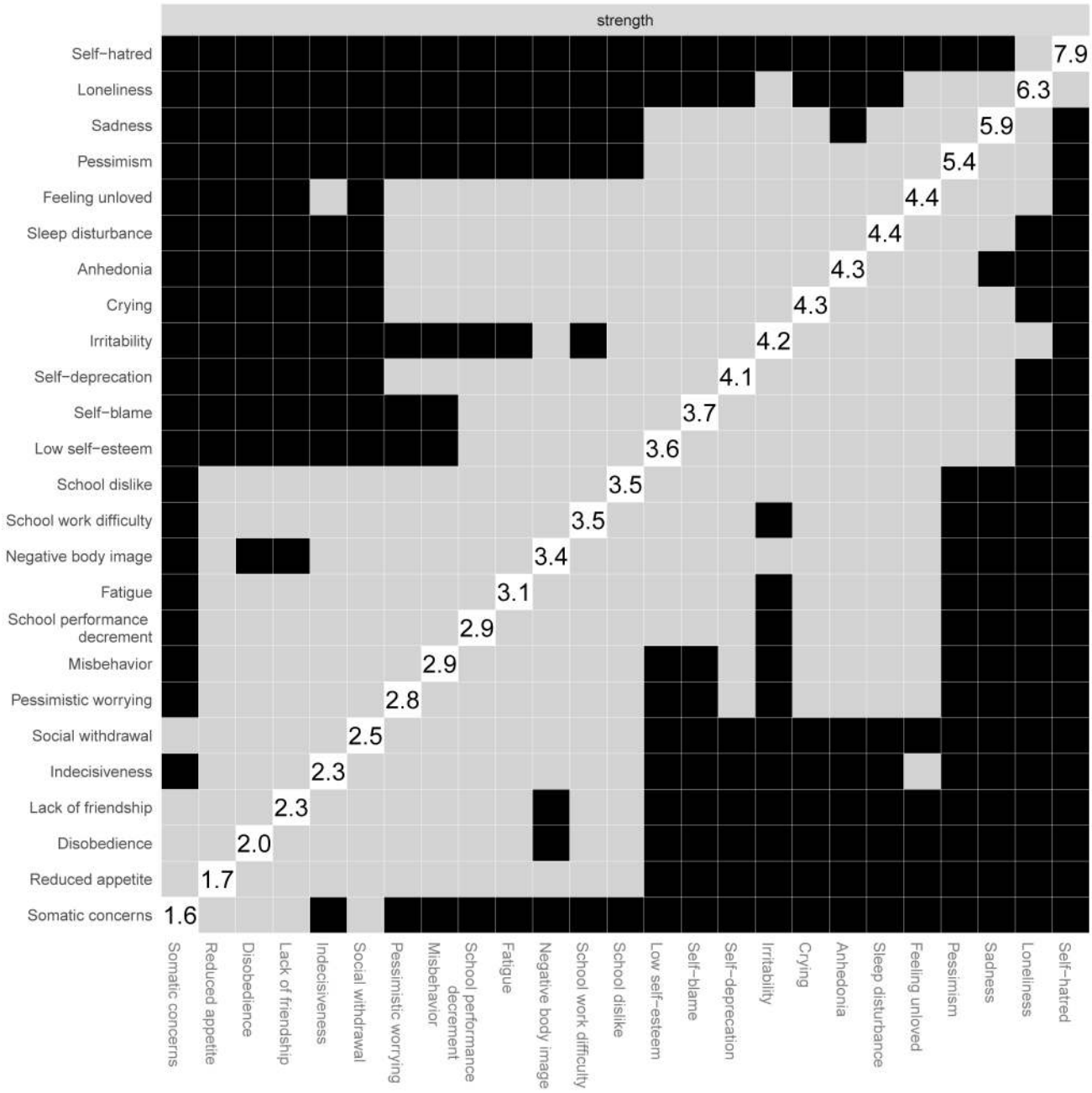

Figure 3.

Non-parametric bootstrapped difference test for strength. Gray boxes indicate no difference between nodes, while black boxes indicate significant difference ($\alpha = 0.05$). Values reported in the diagonal represent the strength values of each node.

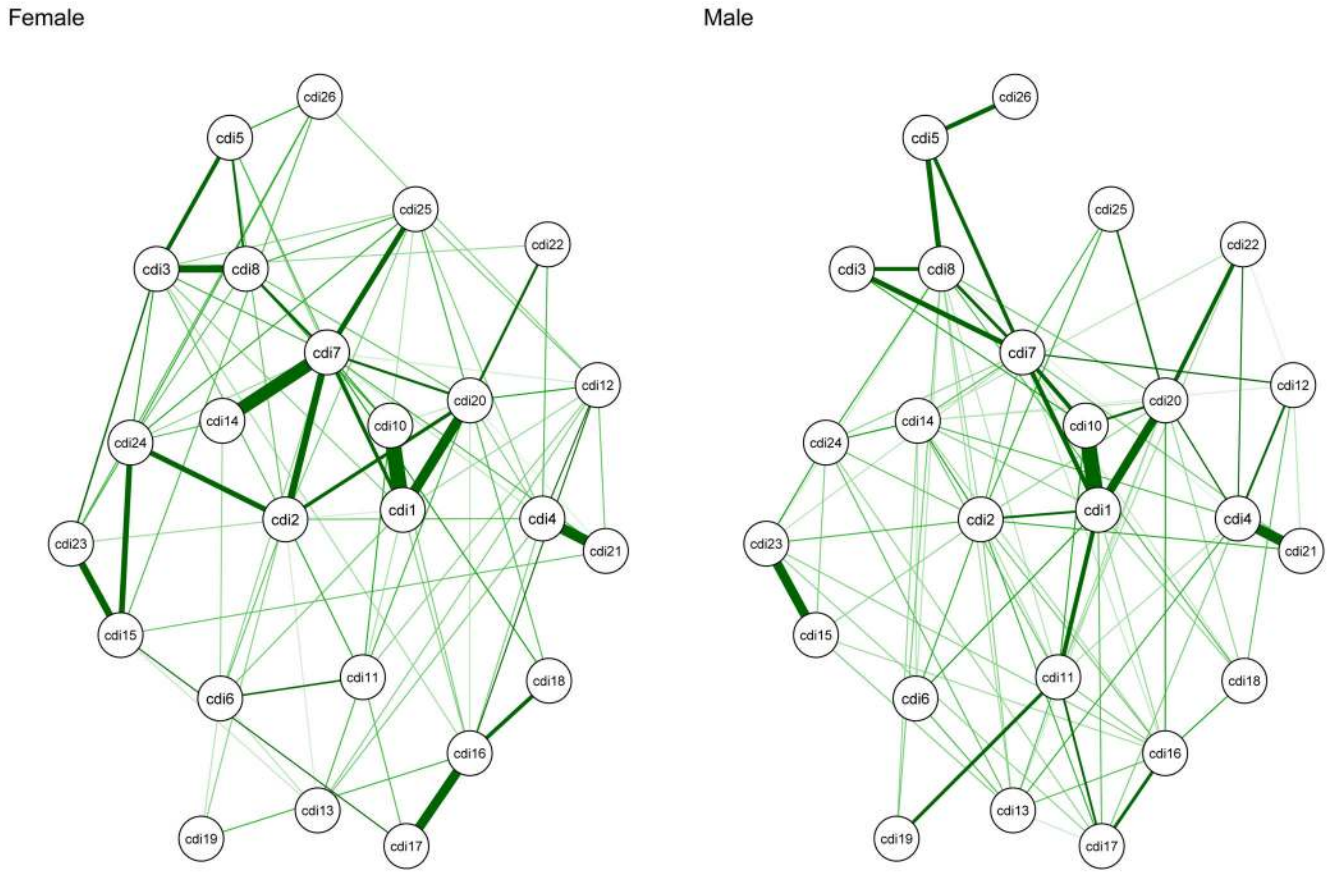

Figure 4.

Estimated network model for dichotomized depressive symptoms in males (n = 646) and females (n = 744)

Table 1.

Mean, standard deviation, minimum, maximum, skewness, and kurtosis, and frequency of the CDI Symptoms (n = 1409)

Symptoms	CDI #	Mean	St.Dev	Min	Max	Skewness	Kurtosis	% Absence ("0")	% Presence ("1")
Sadness	1	0.33	0.55	0	2	1.38	0.94	70.33	29.67
Pessimism	2	0.48	0.57	0	2	0.68	−0.54	55.50	44.50
Self-deprecation	3	0.27	0.50	0	2	1.62	1.72	75.51	24.49
Anhedonia	4	0.49	0.54	0	2	0.43	−1.02	52.95	47.05
Misbehavior	5	0.21	0.46	0	2	2.07	3.58	81.12	18.88
Pessimistic worrying	6	0.57	0.63	0	2	0.64	−0.56	50.53	49.47
Self-hatred	7	0.35	0.60	0	2	1.49	1.10	71.26	28.74
Self-blame	8	0.35	0.54	0	2	1.24	0.55	68.20	31.80
Crying	10	0.32	0.61	0	2	1.73	1.74	75.59	24.41
Irritability	11	0.80	0.75	0	2	0.34	−1.15	39.96	60.04
Social withdrawal	12	0.29	0.51	0	2	1.51	1.34	73.46	26.54
Indecisiveness	13	0.82	0.62	0	2	0.14	−0.53	29.67	70.33
Negative body image	14	0.61	0.69	0	2	0.67	−0.70	50.25	49.75
School work difficulty	15	0.93	0.78	0	2	0.12	−1.36	34.14	65.86
Sleep disturbance	16	0.62	0.74	0	2	0.73	−0.84	53.23	46.77
Fatigue	17	1.08	0.75	0	2	−0.13	−1.23	24.84	75.16
Reduced appetite	18	0.35	0.63	0	2	1.61	1.31	73.81	26.19
Somatic concerns	19	0.45	0.63	0	2	1.08	0.06	62.38	37.62
Loneliness	20	0.48	0.64	0	2	1.00	−0.11	60.25	39.75
School dislike	21	0.56	0.59	0	2	0.51	−0.65	48.83	51.17
Lack of friendship	22	0.38	0.55	0	2	1.08	0.16	65.15	34.85
School performance decrement	23	0.47	0.67	0	2	1.09	−0.06	62.67	37.33
Low self-esteem	24	0.73	0.72	0	2	0.46	−0.99	43.22	56.78
Feeling unloved	25	0.24	0.49	0	2	1.89	2.76	78.57	21.43
Disobedience	26	0.23	0.46	0	2	1.84	2.51	79.06	20.94
Fights	27	0.09	0.32	0	2	3.58	13.14	91.56	8.44