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Network analysis of depression in prostate cancer patients: Implications for assessment and treatment

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Abstract

Objectives: Many prostate cancer patients also suffer from depression, which can decrease their life satisfaction and also impede recovery from their cancer. This study described the network structure of depressive symptomatology in prostate cancer patients, with a view to providing suggestions for clinical interventions for depressed patients.

Methods: Using a cross-sectional design, 555 prostate cancer patients completed the Patient Health Questionnaire-9 (PHQ-9).

Results: Network analysis and multidimensional scaling indicated that anhedonia was the most central symptom for these men, and that several sets of depression symptoms were closely associated with each other. These included anhedonia-depressed mood; sleeping problems-fatigue/lethargy; and suicidal ideation-low self-worth-depressed mood. Other depression symptoms such as appetite problems, concentration problems, and motor problems, were less well-related with the remainder of the network. Patients receiving treatment for reocurring prostate cancer (PCa) had significantly higher PHQ9 scores than patients undergoing their initial treatment, but no major differences in their network structures. Implications for clinical practice were derived from the relationships between individual depression symptoms and the overall depression network by examining node predictability.

Conclusions: The use of total depression scores on an inventory does not reflect the underlying network structure of depression in PCa patients. Identification and treatment of the central symptom of anhedonia in PCa patients suggests the need to adopt specific therapies that are focussed upon this symptom.

KEYWORDS
anhedonia, cancer, depression, network, oncology, prostate

1 | BACKGROUND

Between 15% and 22% of prostate cancer (PCa) patients suffer from comorbid major depression.[1,2] These depressed PCa patients also have higher likelihoods of emergency room visits, hospitalisations, outpatient visits, and excessive risk of death.[3] Consequently, recommendations for routine screening and treatment of depression in these men have been made,[3] but the heterogeneity of depression,[4] and the limited treatment efficacy for depression in the wider population (about 34% from singular treatments, rising to 74% when treatments are combined)[5] argue for a more 'individualised' method of assessing depression. For example, the difference in treatment efficacy across patients with different depression symptom profiles[6] and subtypes of depression (e.g., depression with anxiety, melancholic features, with atypical features and with mixed features)[7] challenges the 'one size fits all' model of some treatment approaches for these men.[8]

Several studies have been made of the nature of depression in PCa patients for example,[9–11] using various methods of classifying subgroups of symptoms of depression. One hitherto unused method of identifying the underlying structure of depression symptomatology in PCa patients is by 'network analysis',[12] that describes the causal interplay between symptoms that may also include feedback loops of those symptoms.[12] This information could hold implications for the design and delivery of symptom-focused treatments. Previous network analyses of depression in non-PCa samples have provided valuable insights into the ways that the nine symptoms of Major Depressive Disorder (MDD) are related, with implications for focussed treatment of those symptoms.[13,14] To extend understanding of the nature of depression in PCa patients, with a view to developing more focussed and effective treatments, this study applied network analysis to the depression symptoms experienced by a sample of PCa patients.

2 | METHOD

2.1 | Participants

PCa patients from treatment centres in south-east Queensland participated in the study. All these men had biopsy-proven prostate cancer and were attending either for treatment or follow up after previous treatment. No patients were on active surveillance, and patients with all other stages of prostate cancer were included. Treatments included radiotherapy and/or surgery and hormone therapy when required. Other inclusion criteria were: (i) the diagnosis of prostate cancer was proven histologically, and (ii) all of the treatment options were properly considered by patients via discussion with their GP, a radiation oncologist and a urologist. Unwillingness to participate in the study was the only exclusion criterion.

2.2 | Measures

As well as a questionnaire about their age, PCa status, past and present treatments, relationship status, and date of their first diagnosis, the PCa patients completed the Patient Health Questionnaire-9 (PHQ-9) for how they felt at the time. All these questionnaires were in English.

The Patient Health Questionnaire-9 (PHQ-9)[15] is based upon the diagnostic criteria for MDD as they are in the current DSM-5-TR.[7] The PHQ9 has specificity and sensitivity above 95%.[15] Possible total scores on the nine items of the PHQ9 range from 0 to 27, with cutoff points of 1–4 (signifying a rating of 'none'), 5–9 ('Mild'), 10–14 ('Moderate'), 15–19 ('Moderately severe') and 20–27 ('Severe').[16] The PHQ-9 has been used in previous studies of depression in cancer patients[17]

2.3 | Procedure

One thousand PCa patients were invited to participate in a 'study about how you feel' by completing questionnaires they received from reception staff, and returning them to the second author's treatment site personally or via post. Of these, 555 (55.5%) returned useable data. Approval for this study was received from the UnitingCare Health Human Research Ethics Committee (Approval number 2013.32.104) in accordance with the Helsinki Declaration of 1964 and confirmed in 2013. Written informed consent was obtained from all participants.

2.4 | Statistical analyses

Network analyses were performed using RStudio.[18] The regularised partial correlation network was estimated using the EBICglasso procedure with a default Extended Bayesian Information Criterion (EBIC) tuning parameter of 0.5.[19] The network structure, node (i.e., PHQ-9 items) centrality estimates, and accuracy and stability of the network and its properties (i.e., centrality, edge weights) were computed using the *bootnet* package.[20] Node centrality estimates serve as an indicator of the relative importance of nodes (depression symptoms) within the overall network structure; nodes that are more central are those that are more highly interconnected with other nodes.[21] Confidence intervals and significant difference tests for the centrality estimates and for edge-weights were computed with bootstrapping of 2500 samples.

The network structure was visualised using Multidimensional Scaling (MDS), which facilitates interpretation of the distance between nodes (i.e., items, symptoms), such that nodes that are in closer proximity to one another are more closely related,[22] and this was generated using the *qgraph* package.[23] Communities (i.e., clusters of symptoms within the network structure) were investigated to extract additional information about how depression symptoms relate to one another, as this has seldom been performed in network analyses of depression so far,[24] and was done here with the *igraph* package[25] using the *spinglass* community-detection algorithm.[26]

In order to detect the presence of possible group differences in overall depression scores, network structure, and associated network properties that might occur due to the treatment stage, patients

undergoing their initial treatment were compared to patients receiving treatment for reocurring PCa, and those in remission (Table 1) via ANOVA. Differences in network structure and properties were examined with the *NetworkComparisonTest* package (NCT).[27]

Finally, node predictability (i.e., a measure of how well a given node can be predicted by its neighbouring nodes) was computed using the *mgm* package.[28] Reporting standards for network analysis described in Burger at al.[29] were followed. Where appropriate, Bonferroni corrections were made to the acceptable p value to reduce the likelihood of family-wise error.

3 | RESULTS

The upper section of Table 1 describes the background data for the sample. No data are available regarding the men who did not choose to participate, although the entire sample was recruited from the same patient pool. Shown at the bottom of Table 1, the current sample's distribution of depression severity was very similar to that reported by Kroenke et al.[15] for their sample of 474 participants who did not have a formal diagnosis of depression. The PCa sample data presented here may be said to represent a non-clinically-depressed subsample of the wider population, although with some members who reported severe-moderate, and severe distress, which is consistent with the findings reported by Hinz et al.,[17] who found a mean PHQ-9 score of 4.0 for their sample of 640 PCa patients. By applying the Bonferroni-corrected of $p = 0.05/6 = 0.008$ value to the results of a Pearson and Spearman correlation analysis of the patients' age, relationship status, time since diagnosis, current, and past treatments, and cancer status with their PHQ-9 score, no significant relationships were identified (all $p > 0.013$).

3.1 | Descriptive statistics

Floor effects were observed in PHQ-9 items, in that the means and the standard deviations for each of the 9 items were highly correlated with one another ($r = 0.95$, $p < 0.001$). Scores on motor problems and suicidal ideation were highly skewed in this sample (Supplementary Table S1), but means and standard deviations of these two items were similar to those reported in the general population sample by Hartung et al.[14] The relatively lower endorsement of these two items, plus the overall degree of skewness of all items, was consistent with other network analyses on the PHQ-9.[30,31]

3.2 | Network structure

Figure 1 presents the network structure of the 9 PHQ-9 items for the sample. The low *stress-1* value of 0.18 indicates that the distance between nodes was highly interpretable.[22] As such, it can be concluded that: (1) anhedonia was closely linked with depressed

TABLE 1 Background and PHQ-9 data for sample of 555 patients with prostate cancer

Variable	Sample characteristics	
Age	M = 68.07 years (SD = 6.51 years), range = 48–85 years	
Relationship status		
With wife/partner	88.8%	
Widowed	4.3%	
Divorced/separated	3.8%	
Never married/partnered	3.1%	
Time since diagnosis	M = 40.63 months (SD = 40.41 months), range = 1–195 mo.	
Treatments received		
Radiotherapy	8.8%	
Surgery	43.4%	
Hormone therapy	5.2%	
Combinations	33.4%	
Surveillance	9.2%	
Current treatment		
Radiotherapy	33.1%	
Surgery	2.1%	
Hormone therapy	20.5%	
Combinations	15.0%	
Surveillance	29.3%	
Present status		
Cancer still present, undergoing treatment	59.4%	
In remission (no signs)	26.9%	
Cancer reocurring after previous treatment	13.7%	
PHQ-9	Current sample	Kroenke et al. (2001)
	M = 2.8 (SD = 3.7)	M = 3.3 (SD = 3.8)
Depression severity		
Minimal	76.8%	73.4%
Mild	17.3%	19.6%
Moderate	4.3%	4.9%
Moderately severe	1.2%	1.7%
Severe	0.4%	0.4%

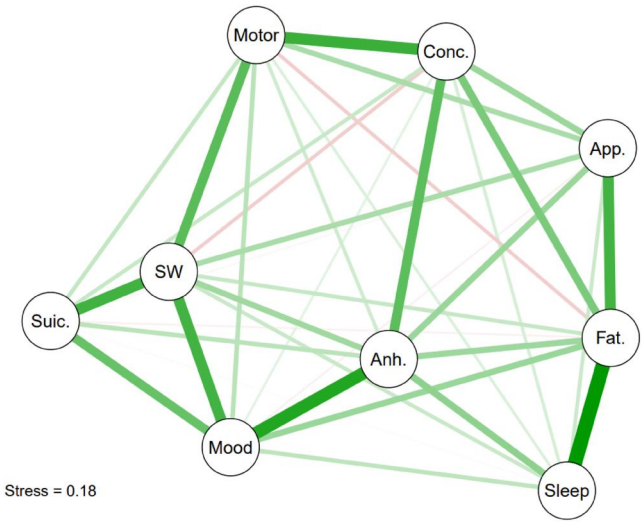

FIGURE 1 Network Structure of Depression for the Overall Sample. Green lines represent positive correlations between nodes, red lines represent negative correlations. The darker and thicker a line, the stronger the correlation. $N = 555$. Multidimensional Scaling (MDS) using spline transformation provided a visual network structure of depression, measured by the 9 items of the PHQ-9. Anh., Anhedonia; App., Appetite problems; Conc., Concentration problems; Fat., Fatigue/lethargy; Suic., Suicidal ideation; SW, Low self-worth

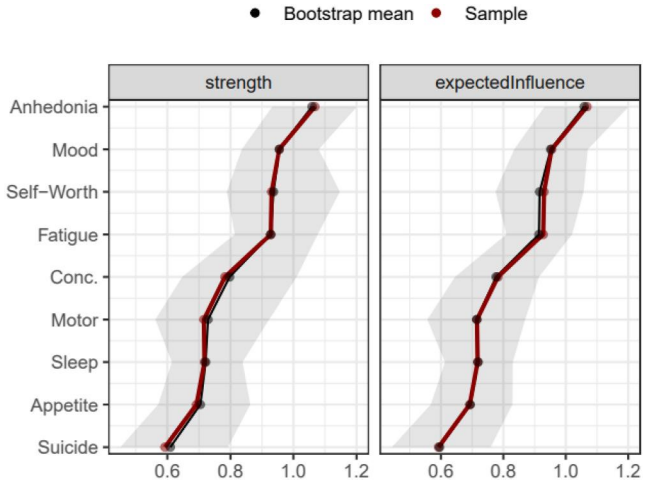

FIGURE 2 Strength and Expected Influence Centrality Estimates and Associated 95% Confidence Intervals for the Overall Depression Network. $N = 555$. 95% confidence intervals were generated with bootstrapping using 2500 samples (represented with grey shaded boundaries surrounding the obtained sample estimates). Conc., Concentration problems

mood; (2) sleeping problems and fatigue/lethargy were strongly associated with each other; (3) suicidal ideation was most strongly related to low self-worth and depressed mood; and (4) appetite problems, concentration problems, and motor problems were more distal to the remainder of the network. Network density was 0.833, such that 30 of 36 possible edges were connected in the network, with a mean weight of 0.103, indicating that all 9 PHQ-9 items were strongly connected with each other.

3.3 | Node centrality

The strength and expected influence centrality estimates and their associated 95% confidence intervals are presented in Figure 2. The stability of the centrality estimates was acceptable (both the *CS*-coefficient of the strength centrality estimates [0.52]), and the expected influence centrality estimate (0.60) exceeded the 0.5 cutoff suggested by Epskamp and Fried.[19] By performing difference tests of the centrality estimates, it was found that anhedonia was significantly more central than most other symptoms in the network (see Supplementary Figure S1). The next most central symptoms were depressed mood, fatigue/lethargy, and low self-worth.

3.4 | Edge weight accuracy

The raw values and associated 95% confidence intervals for edge-weights are presented in Figure 2. The strongest statistical associations between symptoms were between sleep problems and fatigue/lethargy, and between anhedonia and depressed mood (see Supplementary Figure S2), indicating that these were the most reliable and robust symptom associations in the network. These statistical analyses confirm the interpretation of associations between PHQ-9 items that were suggested in Figure 1, (i.e., that patients who had sleep problems frequently also had problems with fatigue or lethargy, and patients who experienced anhedonia also frequently experienced depressed mood). The associations between suicidal ideation and low self-worth/depressed mood suggested in Figure 1 were not found to be robust by this analysis. Associations between other symptoms were not as reliable.

3.5 | Group comparisons

A one-way ANOVA indicated that there was a significant difference between the PHQ-9 total scores of the three patient groups defined in Table 1 (Present Status) $F(2, 544) = 4.79$, $p = 0.009$, $\eta^2 = 0.017$. A Tukey *post hoc* test showed that patients who were receiving treatment for reocurring PCa ($M = 3.96$, SD = 4.00) were significantly more depressed than patients receiving their initial treatment for PCa ($M = 2.53$, SD = 3.79), $p = 0.007$, 95% CI (0.33, 2.54) Other *post hoc* comparisons were non-significant (all $p > 0.05$).

A *NetworkComparisonTest*[27] indicated that the overall network structure was invariant across (a) the active treatment and remission groups ($M = 0.33$, $p = 0.27$), (b) the active treatment and reocurring groups ($M = 0.29$, $p = 0.71$), and (c) the remission and reocurring groups ($M = 0.39$, $p = 0.30$). Additionally, global strength was invariant across (a) the active treatment and remission groups ($S = 0.82$, $p = 0.08$), (b) the active treatment and reocurring groups ($S = 0.15$, $p = 0.77$), and (c) the remission and recurring groups

($S = 0.67$, $p = 0.33$). The only major difference between the groups was that appetite problems were significantly more central (strength $p = 0.02$, expected influence $p = 0.01$) to their network for those PCa patients who were receiving initial treatment than for those PCa patients who were in remission. However, consistent with the overall sample, anhedonia remained the most central symptom in all three groups.

3.6 | Node predictability

Across the entire sample, depressed mood ($R^2 = 0.58$), low self-worth ($R^2 = 0.53$), anhedonia ($R^2 = 0.52$), and fatigue/lethargy ($R^2 = 0.49$) had the greatest predictability. Inferences made from the data and accompanying visual plot include: suicidal ideation may be relatively and more confidently predicted by depressed mood and low self-worth than other items in the depression network; anhedonia and depressed mood are related and predictive of one another; and fatigue/lethargy is likely to be predicted by sleep problems.

4 | DISCUSSION

Several new findings emerge from this first report of the network analysis of depression symptoms in PCa patients. First, visual interpretation of the network structure of the nine PHQ-9 items for the sample of 555 PCa patients indicated that several of the depressive symptoms were closely linked (anhedonia and depressed mood; fatigue/lethargy and sleeping problems; and suicidal ideation and low self-worth/depressed mood). Other symptoms of depression, such as appetite problems, concentration problems, and motor problems, were less closely connected to the core symptoms of the network. Overall, all 9 PHQ-9 items were strongly connected with each other. Second, anhedonia was significantly more central than most other symptoms in the network, followed by depressed mood, fatigue/lethargy, and low self-worth. This means that if a PCa patient were to experience the symptom of anhedonia, it is likely that he would also suffer from each of the other symptoms of depression. Third, anhedonia was not only the most central (i.e., most highly interconnected) symptom, 52% of its variance was predicted by neighbouring nodes. Given the relative strength of the anhedonia-depressed mood edge, much of this variance would likely be attributable to the association between these two nodes. As such, because of it being the most highly interconnected node in the network, plus its association with depressed mood, targeting anhedonia for treatment would likely reduce the patients' depressed mood and, subsequently, overall levels of depression.[21]

Although anhedonia was found to be the central node in the depression symptom network found here, it is not as well-known as the more global notion of 'depression = sad mood' in the general community, despite being one of the two key symptoms required for a diagnosis of MDD.[7] Defined as loss of interest and/or loss of pleasure in activities which were previously enjoyed by the individual, anhedonia has been conceptualised as a withdrawal response to ongoing uncontrollable stress,[32] which fits with the experiences of a person diagnosed with a life-threatening illness such as PCa. There is some evidence that anhedonia is negatively correlated with activation of the key brain structures that are involved in reward processes, such as the nucleus accumbens, basal forebrain, and hypothalamus,[33] and may result from interference in the dopamine system, which in turn blunts the reinforcing effects of naturally occurring rewarding stimuli such as food, water or sex, leading to behavioural extinction of motor responses designed to access those stimuli—i.e., a loss of interest in pleasurable activities, or anhedonia.[34]

4.1 | Clinical implications

Because anhedonic patients have difficulty in making the effort to obtain the rewards they desire, the most promising psychotherapy might be Behavioural Activation,[35] which is relatively directive, and therefore may help to overcome the patient's motivation deficits that are a characteristic of anhedonia. Additionally, antidepressant medication designed to re-establish dopamine stores may prove effective, and some recent data suggest that ketamine may prove efficacious with anhedonia.[36] Transcranial magnetic stimulation may also be useful.[37,38]

When considering the possible causal factors that may have contributed to PCa patients experiencing anhedonia, only 52% of the variance in anhedonia can be explained by neighbouring nodes in the network, and so it is likely that other factors were also influencing anhedonia. Some of these factors could include worries about one's PCa status, side effects of PCa treatment, individual levels of resilience, etc. Such factors may be implicated in the causality of depression, and therefore individual depression symptoms cannot be fully explained by other depression symptoms in the network, but must also partly be explained by external variables.

These data present a different network than reported in some previous studies based on data from a large sample of depressed patients, that did not find anhedonia was central in the network.[13] However, in their study of 4020 cancer patients, Hartung, et al.[14] did find that anhedonia was the most central symptom of MDD on PHQ9 data. These findings suggest that PCa depression may differ to that experienced by the rest of the population, and confirms the need to develop focussed and individualised treatments for these men.

4.2 | Study limitations

The usual limitations on generalisability of these findings apply, including the selective and voluntary nature of the sample, geographical and cultural restrictions, temporal limitations (no ongoing data were collected), and the use of a self-report of depression rather than clinician interviews. Some caution must be exerted regarding the lack of significant differences between three subgroups reported under 'Group Comparisons', due to the high network density

observed and the limited sample size for the treatment reoccurring group.[27,39] Future research with larger sample sizes would help in confirming these findings, as would studies that tracked any changes in the depression networks of PCa patients from the time of first diagnosis, through to the end result of treatment, and at follow-up, particularly in deciding which aspects of depression were most likely to require treatment at which stage of the PCa patient's journey.

4.3 | Conclusions

Notwithstanding these limitations, these first data regarding the network of depression symptoms in a sample of PCa patients suggest that anhedonia may be central to the patient's overall depression, and that there are significant associations between some other symptoms of depression in these men. There is also evidence that PCa patients' depression may differ in its network structure to that reported in the general population. As such, the application of 'one-size-fits-all' global treatment models that are focussed upon the total score on a depression inventory may be less efficacious than treatments that are aimed at the specific symptoms exhibited by these men, such as the central symptom of anhedonia.

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CONFLICT OF INTEREST

None of the authors has any conflict of interest to declare.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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REFERENCES

1. Watts S, Leydon G, Birch B, et al. Depression and anxiety in prostate cancer: a systematic review and meta-analysis of prevalence rates. *BMJ Open*. 2014;4(3):e003901. https://doi.org/10.1136/bmjopen-2013-003901
2. Jayadevappa R, Malkowicz S, Chhatre S, Johnson JC, Gallo JJ. The burden of depression in prostate cancer. *Psycho-Oncolo*. 2012;21(12):1338-1345. https://doi.org/10.1002/pon.2032
3. Fervaha G, Izard J, Tripp D, Rajan S, Leong DP, Siemens DR. Depression and prostate cancer: a focused review for the clinician. *Urologic Oncol Seminars Orig Invest*. 2019;37(4):282-288. https://doi.org/10.1016/j.urolonc.2018.12.020
4. Ostergaard SD, Jensen SOW, Bech P. The heterogeneity of the depressive syndrome: when numbers get serious. *Acta Psychiatr Scand*. 2011;124(6):495-496. https://doi.org/10.1111/j.1600-0447.2011.01744.x
5. Rush A, Trivedi M, Wisniewski S, et al. Acute and longer-term outcomes in depressed outpatients requiring one or several treatment steps: a STAR* report. *Am J Psychiatr*. 2006;163(11):1905-1917. https://doi.org/10.1176/ajp.2006.163.11.1905
6. Kaiser T, Volkmann C, Volkmann A, Karyotaki E, Cuijpers P, Brakemeier EL. Heterogeneity of treatment effects in trials on psychotherapy of depression. *Clin Psychol Sci Pract*. 2022;29(3):294-303. https://doi.org/10.1037/cps0000079
7. APA. *Diagnostic and Statistical Manual of Mental Disorders 5th Ed Text Revision*. American Psychiatric Association; 2022.
8. Sharpley C, Christie D, Bitsika V. Depression and prostate cancer: implications for urologists and oncologists. *Nat Rev Urol*. 2020;17(10):571-585. https://doi.org/10.1038/s41585-020-0354-4
9. Sharpley C, Bitsika V, Christie D. "The worst thing was…": prostate cancer patients' evaluations of their diagnosis and treatment experiences. *Am J Men's Health*. 2018;12(5):1503-1509. https://doi.org/10.1177/1557988318772752
10. Zhu L, Ranchor A, van der Lee M, Garssen B, Sanderman R, Schroevers MJ. Subtypes of depression in cancer patients: an empirically driven approach. *Support Care Cancer*. 2016;24(3):1387-1396. https://doi.org/10.1007/s00520-015-2919-y
11. Adam S, Thong M, Martin-Diener E, et al. Identifying classes of the pain, fatigue, and depression symptom cluster in long-term prostate cancer survivors—results from the multi-regional Prostate Cancer Survivorship Study in Switzerland (PROCAS). *Support Care Cancer*. 2021;29(11):6259-6269. https://doi.org/10.1007/s00520-021-06132-w
12. Borsboom D, Cramer A. Network analysis: an integrative approach to the structure of psychopathology. *Annu Rev Clin Psychol*. 2013;9(1):91-121. https://doi.org/10.1146/annurev-clinpsy-050212-185608
13. Fried E, Epskamp S, Nesse R, Tuerlinckx F, Borsboom D. What are 'good' depression symptoms? Comparing the centrality of DSM and non-DSM symptoms of depression in a network analysis. *J Affect Disord*. 2016;189:314-320. https://doi.org/10.1016/j.jad.2015.09.005
14. Hartung T, Fried E, Mehnert A, Hinz A, Vehling S. Frequency and network analysis of depressive symptoms in patients with cancer compared to the general population. *J Affect Disord*. 2019;256:295-301. https://doi.org/10.1016/j.jad.2019.06.009
15. Kroenke K, Spitzer R, Williams J. The PHQ-9: validity of a brief depression severity measure. *J Gen Intern Med*. 2001;16(9):606-613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x
16. Spitzer R, Kroenke K, Williams J, et al. Validation and utility of a self-report version of PRIME-MD: the PHQ primary care study. *J Am Med Assoc*. 1999;282(18):1737-1744. https://doi.org/10.1001/jama.282.18.1737
17. Hinz A, Mehnert A, Kocalevent R, et al. Assessment of depression severity with the PHQ-9 in cancer patients and in the general population. *BMC Psychiatr*. 2016;16(1):22. https://doi.org/10.1186/s12888-016-0728-6
18. RStudio Team. *RStudio: Integrated Development Environment for R. 2022.07.1+544 Ed*. RStudio, PBC; 2022.
19. Epskamp S, Fried E. A tutorial on regularized partial correlation networks. *Psychol Methods*. 2018;23(4):617-634. https://doi.org/10.1037/met0000167
20. Epskamp S, Borsboom D, Fried E. Estimating psychological networks and their accuracy: a tutorial paper. *Behav Res Methods*. 2018;50(1):195-212. https://doi.org/10.3758/s13428-017-0862-1

21. Bringmann L, Albers C, Bockting C, et al. Psychopathological networks: theory, methods and practice. *Behav Res Ther*. 2022;149:104011. https://doi.org/10.1016/j.brat.2021.104011
22. Jones PJ, Mair P, McNally RJ. Visualizing psychological networks: a tutorial in R. *Front Psychol*. 2018;9:1742. https://doi.org/10.3389/fpsyg.2018.01742
23. Epskamp S, Cramer A, Waldorp L, Schmittmann VD, Borsboom D. qgraph: network visualizations of relationships in psychometric data. *J Stat Software*. 2012;48(4). https://doi.org/10.18637/jss.v048.i04
24. Malgaroli M, Calderon A, Bonanno G. Networks of major depressive disorder: a systematic review. *Clin Psychol Rev*. 2021;85:102000. https://doi.org/10.1016/j.cpr.2021.102000
25. Csardi G, Nepusz T. The igraph software package for complex network research. *InterJournal Complex Syst*. 2006:1695.
26. Yang Z, Algesheimer R, Tessone CJ. A comparative analysis of community detection algorithms on artificial networks. *Sci Rep Nat Publ Group*. 2016;6(1):30750. https://doi.org/10.1038/srep30750
27. van Borkulo CD, van Bork R, Boschloo L, et al. Comparing Network Structures on Three Aspects: A Permutation Test. 2017. https://doi.org/10.1037/met0000476
28. Haslbeck JMB, Waldorp LJ. mgm: estimating time-varying mixed graphical models in high-dimensional data. *J Stat Software*. 2020;93(8). https://doi.org/10.18637/jss.v093.i08
29. Burger J, Isvoranu A, Lunansky G, et al. Reporting standards for psychological network analyses in cross-sectional data. *Psychol Methods*. 2022. Advance online publication. https://doi.org/10.1037/met0000471
30. Wasil AR, Venturo-Conerly KE, Shinde S, Patel V, Jones PJ. Applying network analysis to understand depression and substance use in Indian adolescents. *J Affect Disord*. 2020;265:278-286. https://doi.org/10.1016/j.jad.2020.01.025
31. Osborn TL, Campbell S, Ndetei D, et al. Network Analysis Reveals Central Symptoms of Adolescent Depression and Anxiety in Subsaharan Africa. 2020. https://doi.org/10.31234/osf.io/dv6c9
32. Willner P, Muscat R, Papp M. Chronic mild stress-induced anhedonia: a realistic animal model of depression. *Neurosci Biobehav Rev*. 1992;16(4):525-534. https://doi.org/10.1016/s0149-7634(05)80194-0
33. Keller J, Young C, Kelley E, Prater K, Levitin DJ, Menon V. Trait anhedonia is associated with reduced reactivity and connectivity of mesolimbic and paralimbic reward pathways. *J Psychiatr Res*. 2013;47(10):1319-1328. https://doi.org/10.1016/j.jpsychires.2013.05.015
34. Salamone J, Cousins M, Snyder B. Behavioral functions of nucleus accumbens dopamine: empirical and conceptual problems with the anhedonia hypothesis. *Neurosci Biobehav Rev*. 1997;21(3):341-359. https://doi.org/10.1016/s0149-7634(96)00017-6
35. Kanter J, Callaghan G, Landes S, Busch AM, Brown KR. Behavior analytic conceptualization and treatment of depression: traditional models and recent advances. *Behav Analyst Today*. 2004;5(3):255-274. https://doi.org/10.1037/h0100041
36. Cao B, Zhu J, Zuckerman H, et al. Pharmacological interventions targeting anhedonia in patients with major depressive disorder: a systematic review. *Prog Neuro Psychopharmacol Biol Psychiatr*. 2019;92:109-117. https://doi.org/10.1016/j.pnpbp.2019.01.002
37. Bersani F, Minichino A, Enticott P, et al. Deep transcranial magnetic stimulation as a treatment for psychiatric disorders; A comprehensive review. *Eur Psychiatry*. 2012;28(1):30-39. https://doi.org/10.1016/j.eurpsy.2012.02.006
38. Wang X, He K, Chen T, et al. Therapeutic efficacy of connectivity-directed transcranial magnetic stimulation on anticipatory anhedonia. *Depress Anxiety*. 2021;38(9):972-984. https://doi.org/10.1002/da.23188
39. Haslbeck J. Estimating group differences in network models using moderation analysis. *Behav Res Methods*. 2022;54(1):522-540. https://doi.org/10.3758/s13428-021-01637-y

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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