

Network Analysis Reveals Central Symptoms of Adolescent Depression and Anxiety in Sub-Saharan
Africa

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Abstract

Adolescent depression and anxiety—which are linked with many negative life outcomes—are prevalent around the world, particularly in low-income countries such as those in Sub Saharan Africa (SSA). We used network analysis to examine the topology, stability, and centrality of depression and anxiety symptoms. We analyzed data from a large community sample ($N = 2,192$) of Kenyan adolescents aged 13-18, using the Patient Health Questionnaire and the Generalized Anxiety Disorder Screener. We identified the central symptoms of the depression and anxiety symptom networks, and we compared the structure and connectivity of these networks between low-symptom and elevated-symptom sub-samples. Our findings indicate the most central depression symptoms were “self-blame” and “depressed mood”, while the strongest depression symptom associations were “self-blame” —“depressed mood” and “trouble concentrating” —“little interest/pleasure”. Similarly, the most central anxiety symptoms were “too much worry” and “uncontrollable worry”, while strongest anxiety symptom associations were “too much worry” —“uncontrollable worry” and “trouble relaxing” —“restlessness”. We found a statistical difference in the network structure between low-symptom and elevated-symptom adolescents. The low-symptom sample had higher network connectivity scores for both depression (global strength difference = 0.30; low-symptom = 0.49; high-symptom = 0.19; $p = .003$) and anxiety symptoms (global strength difference = 1.04; low-symptom = 1.57; high-symptom = 0.53; $p < .001$). This is the first report that uses network analysis techniques to identify central symptoms of adolescent depression and anxiety in SSA. Our findings illustrate how network analysis may inform understanding of psychopathology within cultures and suggest promising treatment targets.

Keywords: Depression, anxiety, network analysis, developmental psychopathology, Sub Saharan Africa

Network Analysis Reveals Central Symptoms of Adolescent Depression and Anxiety in Sub-Saharan Africa

Depression and anxiety symptoms are prevalent in adolescent populations all over the world (Patel & Stein, 2015) and account for nearly half of the overall burden disease (The Lancet, 2017). Adolescents suffering from depression, even at subclinical levels, experience an inferior quality of life (Bertha & Balázs, 2013), school attendance, and academic performance (King & Bernstein, 2001). They also report higher functional impairment and increased suicide rates (Balázs et al., 2013). This burden might be especially elevated in low-income countries, such as those in Sub-Saharan Africa (SSA), where risk factors for mental disorders (like poverty) are particularly high (Dashiff et al., 2009), limited options exist for help-seeking (Patel, Flisher, Hetrick, & McGorry, 2007) and stigma around psychiatric syndromes deters help-seeking (Ndeti et al., 2016). Given the minimal research on adolescent mental health syndromes in SSA coupled with the youthful nature of SSA countries (where mean age is 19.4; United Nations, Department of Economic and Social Affairs, Population Division, 2019), there is a pressing need for adolescent mental health research with this population.

The scarce studies with SSA youths show a relatively high prevalence of depression and anxiety symptoms. A recent study with 658 school-going adolescents in Kenya revealed that about 1 in 3 youths endorsed moderate-to-severe depression and anxiety symptoms (Osborn, Venturo-Conerly, Wasil, Schleider & Weisz, 2019), consistent with other findings of prevalence rates with Kenyan youths (Khasakhala et al., 2012; Ndeti et al., 2008). In neighboring Tanzania, 40% of secondary school students in a community sample of 700 endorsed depression and anxiety symptoms (Nkuba et al., 2018), and in Nigeria, 21.2% of youths in a sample of 1,713 school-going adolescents reported depression symptoms (Fatiregun & Kumapayi, 2014). This high prevalence of adolescent depression and anxiety might be due to the efficacy and appropriateness of the existing measurement tools that are commonly used. Most are primarily Western tools that have not extensively investigated with SSA youths. Insofar as the psychometric properties of these tools remain under-investigated, epidemiological studies in SSA remain

handicapped as these instruments are likely to either under- or over-report prevalence rates of youth depression and anxiety (Ndetei et al., 2008; Osborn et al., 2019).

Beyond this, the heterogeneity of symptoms within diagnostic tools for depression and anxiety (Olbert et al., 2014; Zimmerman et al., 2015) presents a challenge in understanding the significance of individual symptoms. For example, using a standard self-report depression questionnaire, we calculated sum-scores to determine if an individual meets the clinical threshold for depression. However, due to symptom heterogeneity, loss of information is inevitable and potentially profound if these assessment instruments are used outside their cultural context—as is often the case in SSA. Simply relying on sum-scores of equally-weighted symptoms might present a misleading picture of the prevalence and experience of depression and anxiety (Fried & Nesse, 2015).

One way of expanding our understanding of youth depression and anxiety in SSA might lie in complementing the traditional models of diagnoses with the increasingly popular network framework of psychopathology. Network analysis can be used as a tool to visualize psychiatric constructs as graphs—a disorder is represented as a dynamic system made up of nodes (*symptoms*) and edges (*the psychometric associations between pairs of symptoms*). Since these associations between symptoms are not explicitly present in a dataset, we estimated them by computing the partial correlations between all symptoms, such that each edge depicts the association between a pair of nodes while controlling for all other associations. If two nodes share an edge, they have a relationship—either positive or negative. On the other hand, if two nodes do not share an edge, they are conditionally independent given all the other nodes. Thus, a main advantage of the network perspective is that it provides a way to analyze the importance of symptoms and the interconnections between them (Contreras et al., 2019).

Using this approach, a mental disorder is not a discrete entity but rather a network of interrelated symptoms. The network perspective departs from the traditional disease model by abandoning the assumption that mental health disorders arise from an underlying latent disorder (Insel, 2014). Instead, “disorders” are construed as the interaction of unique and non-overlapping variables. For example,

depression is not some latent abnormality in the brain that gives rise to the anhedonia, sadness, and guilt; rather, it is the interaction of these symptoms that *is* depression. Even though prevalence studies that use self-report questionnaire scores to diagnose depression can shed important light into the prevalence of youth depression and anxiety in SSA, complementing them with network analyses of symptoms allows us to uncover the associations among symptoms and identify which symptoms are central to these disorders (Borsboom, 2017).

Although the network approach is potentially beneficial to understanding the psychopathology of youth psychopathology, it has only recently been used in clinical research among adults, in which *little interest/pleasure*, *low energy*, and *sad mood* have emerged as the central symptoms of adult depression (Beard et al., 2016; Fried et al., 2016; Santos et al., 2017). Limited studies have investigated the network of symptoms in adolescent depression and anxiety (McElroy et al., 2018; Mullarkey et al., 2019; Ventura-Conerly et al., 2019; Wasil et al., 2020). A recent study with a community sample of American adolescents revealed that the central depression symptoms were *self-hatred*, *loneliness*, *sadness*, and *pessimism* (Mullarkey et al., 2019). This contrast with central symptoms in adult depression might indicate that psychiatric symptom networks may be different between adolescents and adults. One such study—a recent longitudinal study assessed the symptom-symptom associations between depression and anxiety from early childhood to mid-adolescence—found that depression and anxiety symptoms were highly interconnected and clustered together. Also, *anxious/fearful* and *unhappy/sad* were consistently ranked as the most central symptoms across development. The authors also reported an increase in connectivity between anxiety and depression symptoms across development, which might suggest that these symptoms potential reinforce each other (McElroy et al., 2018). Another recent study, with a community sample of American adolescents, revealed that the central depression symptoms were *self-hatred*, *loneliness*, *sadness*, and *pessimism* (Mullarkey et al., 2019). This contrast with central symptoms in adult depression might indicate that psychiatric symptom networks may be different between adolescents and adults.

In the present study, we used network analysis to examine item-level depression and anxiety symptoms in a large community sample of adolescents in Kenya, a low-income country in SSA. Understanding mental illnesses in school-going adolescents is particularly important because 80% of mental health caregiving is provided in school settings (Bussing et al., 2003) and many youths aged 13- to-18 in SSA countries attend school (Ministry of Education, Science and Technology, 2014). Specifically, we sought out to accomplish three main goals: (1) estimate depression and anxiety symptom network structures in an adolescent community sample in SSA, (2) identify the central symptoms of each network, and (3) compare the networks between an elevated-symptom subsample and low-symptom subsample; per network theory, symptoms should be strongly connected in elevated-symptom samples because stronger interconnectivity suggests that symptoms may reinforce each other contributing to the high levels of symptoms in elevated-symptom samples (Borsboom, 2017; McElroy et al., 2018). However, recent studies have offered mixed support for this, one study, for example, found that difference in network structure between elevated and low-symptom networks (Groen et al., 2019) and another found the network in an elevated-symptom sample to have lower global strength than a low-symptom sample (Funkhouser et al., 2020). These studies have called for investigation to see if this observation extends to other samples, we sought to test this possibility.

Methods

Participants and Procedures

Data were collected from four secondary schools in Nairobi and Kiambu Counties as part of an intervention study with adolescents in Kenya (Osborn, Venturo-Conerly, Wasil, Rodriguez, et al., 2019). Self-report data on depression, anxiety, and other health-related and socio-demographic variables were collected in their classrooms. All study procedures were approved by the Maseno University Ethics Review Committee (MUERC) before the start of data collection. All adolescents were eligible to participate if they consented and were enrolled in the participating schools. Parental consent and written informed consent/assent were obtained for all adolescents per research ethics procedures at MUERC.

The community sample of Kenyan adolescents ($N = 2,192$) used for the present study was recruited predominantly from urban schools ($N = 1,715$; 78.2%), while the rest were from rural schools. There was a slight female majority ($N = 1,246$; 58.3%), and the mean age was 15.21 ($SD = 1.14$). A significant proportion of participants met the clinical cutoff for moderate-or-severe depression or anxiety; 33.81% of participants ($N = 741$) met the criteria for depression and 31.30% ($N = 686$) met that of anxiety. See Table 1 for full demographic information and descriptive statistics.

Measures

Depressive symptoms

Depressive symptoms were assessed using the *Patient Health Questionnaire* (PHQ; Kroenke & Spitzer, 2002), a 9-item self-report questionnaire used to assess the severity of depressive symptoms. For this study, we used the 8-item version, which excludes the suicidal item, but with which the same cutoffs can be used (PHQ-8; Kroenke & Spitzer, 2002). The PHQ has been shown to have adequate internal consistency ($\alpha = .89$), test-retest reliability and discriminant validity with North American samples (Kroenke et al., 2001). A previous study with Kenyan adolescents supports adequate consistency ($\alpha = .73$) and validity for the PHQ (Osborn et al., 2019). In our present study, Cronbach's alpha was .78.

Anxiety symptoms

Anxiety symptoms were assessed using the Generalized Anxiety Disorder Screener (GAD-7; Spitzer, Kroenke, Williams, & Löwe, 2006), a 7-item self-report questionnaire used to assess the anxiety symptom severity. Previous studies validate that the GAD-7 has adequate internal consistency ($\alpha = .92$) and criterion validity in samples of North American adolescents (Spitzer et al., 2006) and of Kenyan youths (Osborn et al., 2019). In our present study, Cronbach's alpha was .82.

Network Analysis

We performed all our analysis in *R*. First, we investigated the presence of redundant nodes, or overlapping symptoms, by checking pairs of nodes for high correlation ($r > .5$) and similar correlation patterns with all the other nodes via the *networktools* package in *R* (Jones et al., 2018; Levinson et al.,

2018). Second, we estimated the network models for the data and validated their accuracy and stability using the *bootnet* package in *R* (Epskamp, Borsboom, & Fried, 2018). Third, we plotted the network structures and computed the centrality measures using the *qgraph* package in *R* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). Fourth and lastly, we compared the network structures using the *NetworkComparisonTest* package in *R* (NCT; Borkulo et al., 2017).

Symptom Selection

The lack of variance in variables may lead to the misinterpretation of network structure. If two nodes represent the same construct, the edge between them depicts shared variance and not their association (Fried et al., 2017). Thus, these redundant pairs should not be included together in the network. In contrast, if two nodes represent independent constructs, we would expect their correlation patterns with other nodes to vary. We tested for the problematic presence of multiple symptoms representing the same underlying construct using the *goldbricker* procedure (Jones et al., 2018; Levinson et al., 2018), which checks each pair of nodes for a high correlation between them ($r > .5$) and similar correlation patterns with all the other nodes (under a given threshold proportion of significantly different correlations).

Missing data

Given that some participants did not answer every question; the data set includes some missing data. To avoid biases, inefficiencies, and incorrect estimates in our analysis, the data were imputed using the mean of each item.

Network estimation and accuracy

We estimated partial correlation networks for both depression and anxiety symptoms by computing a Gaussian graphical model (GGM) regularized with the graphical Lasso method (Borsboom & Cramer, 2013). This technique finds the best fitting model by penalizing, or shrinking, small edge values estimated in the network, and it helps address the multiple testing problem by controlling false-positive errors. Next, we tested the stability of each network using both nonparametric bootstrapping

(Epskamp et al., 2018). This process repeatedly resamples subsets of the data to calculate a confidence interval (CI) as the range of bootstrapped values from different sampling levels. We used CIs to assess the accuracy of edge-weights (e.g., a large CI indicates that it may be difficult to interpret the edge-weight).

Centrality and stability

The centrality of a node can be used to infer its influence, or structural importance, in the network. There are many indices used to estimate centrality: *betweenness*—how a node influences the average path between other pairs of nodes, *closeness*—how a node is indirectly connected to the other nodes, *strength*—how a node is directly connected to the other nodes, and *expected influence (EI)*—how a node is connected to the sum of all edge weights (Robinaugh et al., 2016). Since we aim to understand which symptoms should be most directly targeted in treatment, we focused on *strength* in our discussion. Next, we tested the stability of the centrality indices with case-dropping bootstrapping (Epskamp et al., 2018), which is the process of repeatedly estimating a model while dropping rows of the data (i.e., only observing subsets of the data). With this case-dropping bootstrap, we calculated a correlation-stability (CS) coefficient, which indicates the maximum proportion of the data that can be dropped while continuing to estimate centrality values that correlate highly ($r > .7$) with the network from the full sample. Scores .25 and .5 indicate benchmarks for adequate and good network stability, respectively (Epskamp et al., 2018).

Network comparisons

In addition to finding the core elements of each network, we also compared the network structures for subsets with high and low scores on psychiatric measures (Borkulo et al., 2017). NCT allows us to identify invariances in network structure and connectivity if any. Specifically, this algorithm takes in two networks estimated from cross-sectional data, repeatedly calculates test statistics, and determines whether the invariance hypotheses hold.

Results

Descriptive Statistics

Table 2 reports the mean, standard deviation, minimum, maximum, skewness, and kurtosis for the PHQ and GAD symptoms. The mean sum scores were below the moderate-to-severe cutoff for PHQ ($M = 7.96$) and GAD ($M = 7.47$). For PHQ, the maximum mean scores were for *self-blame* and *trouble concentrating* and the minimum for *psychomotor problems* and *appetite*. For GAD, the maximum mean scores were for *too much worry* and *uncontrollable worry* and the minimum for *restlessness* and *trouble relaxing*.

Depression Symptoms Network Structure

Network estimation, accuracy, centrality, and stability

Networks were constructed for depressive symptoms (see Figure 1). The bootstrapped CIs for the edge-weights were quite high, indicating that the network structure was accurately estimated. Notably, the PHQ symptoms were highly interconnected. The most consistently strong edges observed were between *self-blame* and *depressed mood*, *self-blame* and *trouble concentrating*, and *sleep problems* and *little energy*. The most central PHQ symptoms, according to strength as well as expected influence and betweenness, were *self-blame* and *depressed mood* (see Figure 3). The network structure also had high stability. The CS coefficient was high; 92% of the sample could be dropped before the correlation with original centrality values dropped below $r = .7$.

Network comparison between high-symptom and low-symptom subsamples

Figure 1 shows the PHQ symptom networks for low-symptom ($N = 1,453$) and high-symptom ($N = 739$) participants. The test for network connectivity difference suggests a statistical difference between the networks for participants with depression sum-scores above and below the clinical threshold (global strength difference = 0.30; low-symptom = 0.49; high-symptom = 0.19; $p = .003$). The networks did not vary significantly in structure (maximum difference = 0.13; $p = .257$). As shown in the centrality plots, depression symptom centrality index *strength* was similar but not identical for low-symptom and high-

symptom networks (see Figure 3). PHQ3 and PHQ4 were more central in low-symptom than high-symptom networks. Meanwhile, the rest were slightly more central for high-symptom participants.

Anxiety Symptoms Network Structure

Network estimation, accuracy, centrality, and stability

Networks were constructed for anxiety symptoms (see Figure 2). The bootstrapped CIs for the edge-weights were quite high, indicating that the network structure was accurately estimated. Notably, the GAD symptoms were highly interconnected. The most consistently strong edges observed were between *uncontrollable worry* and *too much worry* as well as *trouble relaxing* and *restlessness*. The most central GAD symptoms, according to strength, were *too much worry*, *uncontrollable worry*, and *nervousness* (see Figure 3). The network structure also had high stability. The CS coefficient was high; 92% of the sample could be dropped before the correlation with original centrality values dropped below $r = .7$.

Network comparison between high-symptom and low-symptom subsamples

Figure 2 shows the GAD symptom networks for low-symptom ($N = 1,507$) and high-symptom ($N = 685$) participants. The test for network connectivity difference suggests a statistical difference between the networks for participants with anxiety sum-scores above and below the clinical threshold (global strength difference = 1.04; low-symptom = 1.57; high-symptom = 0.53; $p < .001$). The networks also did not vary significantly in structure (maximum difference = 0.14; $p = .119$). As shown in the centrality plots, anxiety symptom centrality index *strength* was similar but not identical for low-symptom and high-symptom networks (see Figure 3). GAD1, GAD3, GAD4, and GAD5 were more central in low-symptom than high-symptom networks. Meanwhile, the rest were slightly more central for high-symptom participants.

Discussion

Our study investigated depression and anxiety symptom networks in a large community sample of Kenyan adolescents—a largely understudied population in Sub Saharan Africa. Our results identify the central symptoms and calculate the strength of symptom associations in adolescent depression and

anxiety networks. This is, to the best of our knowledge, a first of its kind in SSA for both depression and anxiety. The present study is also the first report of anxiety symptom networks in a community sample of adolescents around the world. Taken together, these results—which were robust to statistical and accuracy tests—expand our understanding of adolescent depression and anxiety in an understudied population through a novel network framework.

Adolescent Depression Symptom Networks

In the current study, *self-blame* was the most central symptom of adolescent depression as indexed by *strength*, followed closely by *depressed mood*. The importance of *self-blame* is not surprising given prominent theories—including Beck’s cognitive theory of depression—regard *self-blame* as a primary feature of depression (Beck, 2002; Peterson, Schwartz, & Seligman, 1981). Studies have also shown that *increased negative self-referent thinking* is a potent risk factor of depression in adolescents (Connolly et al., 2016; Speed et al., 2016). A recent network analysis project with a similarly large community sample ($N = 1,409$) of adolescents in North America found *self-hatred* to be the most central symptom of depression (Mullarkey et al., 2019). The study used the Child Depression Inventory (CDI; Kovacs, 1992) to assess for depression, which measures both *self-blame* and *self-hatred*. Both *self-blame* and *self-hatred* capture the DSM symptom category of *feelings of worthlessness, excessive or inappropriate guilt* (American Psychiatric Association, 2013), which our findings support as a core symptom of depression in diverse populations around the world. Additionally, our finding of *depressed mood* as a central symptom of adolescent depression does not come as a surprise since it is generally considered a hallmark of depression; per DSM-5 criteria, either *depressed mood* or *little interest/pleasure* is required for a diagnosis of depression (American Psychiatric Association, 2013).

We found the association between *self-blame* and *depressed mood* to be particularly important in adolescent depression; these symptoms shared the strongest edge. This observation reinforces our current understanding of depression, particularly when using Beck’s cognitive model (Beck, 2002): *self-blame* (negative thoughts about self) is linked to *depressed mood* (sadness) in a mutually reinforcing system that

exacerbates and maintains symptoms of adolescent depression. This strong link also suggests that cognitive behavioral therapy (CBT) targeting cognitive processes associated with *self-blame* might be effective for adolescent depression given CBT potentially weakens the association between *self-blame* and *depressed mood*. Another relationship in adolescent depression that is worth special consideration is the strong association between *trouble concentrating* and *self-blame*. Though limited studies have investigated this link, we posit that the unique nature of the Kenyan education system might have a role to play in this association, which appears to be potentially pathological. Studies have shown that the Kenyan adolescents face increased psychosocial pressure to succeed academically—due to familial sacrifices to pay school fees and the “do or die” mentality associated with exam success (upon which future education and career opportunities are hinged)—which exacerbate depression symptoms (Osborn et al., 2019; Yara & Wanjohi, 2011). As a result, we suggest that the association between *trouble concentrating* and *self-blame* in depression amongst Kenyan adolescents might help us understand academic functioning and achievement in this population and future studies should explore this possibility. Perhaps interventions targeted towards improving academic achievement reduce depression symptoms and vice-versa. A recent trial of an in-school group-based intervention with high-symptom Kenyan adolescents showed that subjects assigned to the intervention group reported both a reduction in depression symptoms and improvements in academic achievement (Osborn, Wasil, Venturo-Conerly, Schleider & Weisz., 2019; Osborn et al., 2019). Future studies that elucidate how academic achievement (in the high-pressure context of Kenya and similar SSA countries) relates to this link can potentially be of high clinical utility. Such interventions might focus on the target associations identified in this study.

We found a statistical difference in the networks for participants with low- and high- symptom depression networks. Interestingly, the low-symptom network was more strongly connected (global strength = 0.49) than the high-symptom network (global strength = 0.19). This finding adds to recent findings, such as those by Groen et al., 2019 and Funkhouser et al., 2020, that found the networks in elevated-symptom samples to have lower global strength than a low-symptom sample. Our findings are

contrary to what network theory would suggest; per network theory, stronger interconnectivity suggests that symptoms may be reinforcing each other and should contribute to a high symptom levels, such as those in elevated-symptom samples (Borsboom, 2017; McElroy et al., 2018). Our findings, when considered together with other recent findings that have shown stronger interconnectivity in low-symptom networks, suggest that further, and perhaps urgent, research is required to examine the differences, if any, between networks of low-symptom and elevated-symptom samples.

Adolescent Anxiety Symptom Networks

The most central symptom of adolescent anxiety was *too much worry* (worrying too much about different things), followed closely by *uncontrollable worry* (not being able to stop or control worrying). This is not surprising because persistent, excessive, and irrepressible worry across different things is a hallmark of anxiety disorders (American Psychiatric Association, 2013). To the best of our knowledge, no network analysis has examined the central symptoms of adolescent anxiety in a community sample. A recent network analysis study with a community sample of trauma-exposed adults found *uncontrollable worry* to be one of the central symptoms of anxiety (Price et al., 2019). Given that *worry* appears to be a defining symptom of adolescent anxiety, treatment techniques that address the processes underlying pathological *worry* might be effective. Indeed, CBT for anxiety has demonstrated large effects on the reduction of *worry* (Covin et al., 2008; Hanrahan et al., 2013).

We found the strongest association between *uncontrollable worry* and *too much worry* in the anxiety symptom network; that two types of *worry* are highly linked does not come as a surprise. Another strong link in the network of adolescent anxiety was the relationship between *trouble relaxing* and *restlessness*. As anxiety is marked by streams of thoughts on wide-ranging topics that are saturated with emotional ambiguity about perceived negative outcomes (Hirsch et al., 2019; Hirsch & Mathews, 2012), adolescents with anxiety are likely to exhibit automatic emotional-processing biases that favor negative information and, thus, magnify the association between *trouble relaxing* and *restlessness*. As anxiety is marked by streams of thoughts on wide-ranging topics that are saturated with emotional ambiguity about

perceived negative outcomes (Hirsch et al., 2019; Hirsch & Mathews, 2012), adolescents with anxiety are likely to exhibit automatic emotional-processing biases that favor negative information and, thus, magnify the association between *trouble relaxing* and *restlessness*. Emotional processing biases such as interpretation bias (i.e. interpreting ambiguous situations to be threatening) and attention bias (i.e. selective attention to threatening information) might cause an almost consistent state of negative arousal in adolescents with anxiety that appears as restlessness and the limited ability to relax.

As with the adolescent depression networks, we found a statistical difference in the networks for participants with low- and high- symptom anxiety networks. Similarly, the low-symptom network was more strongly connected (global strength = 1.57) than the high-symptom network (global strength = 0.53). These findings, like those with the depression networks, bolster the call for a further examination of the network differences between low- and high-symptom networks.

Study Limitations and Future Directions

One limitation of our study is that even though we have a moderate mean level of depression and anxiety symptoms (when compared to Mullarkey et al., 2019). For example, some evidence suggests that symptom network connectivity might differ between clinical and nonclinical samples (Santos et al., 2017). As such, it is possible that the network connectivity that we report here might not be generalizable to clinical adolescent populations. Another limitation is that our chosen measures (PHQ-8 and GAD-7) might miss certain symptoms of depression and anxiety, risking incomplete or inaccurate conclusions (Jones et al., 2017). Despite their limitations like comorbidity, these diagnostic questionnaires are widely accepted and used. We might also lose some information since our data is cross-sectional and, thus, we can only draw conclusions on contemporaneous associations and temporal relationships between symptoms are precluded. Data will need to be collected over certain times intervals to analyze temporal dynamics.

An important strength of our study is that it is the first of its kind with a large community sample of adolescents ($N = 2,192$) from a low-income country. First, adolescence is a critical period for the

development of psychiatric disorders (Crews et al., 2007; Goldbeck et al., 2007). Investigating this age range can shed light on the nature and onset of depression and anxiety, which, in turn, can help improve the future quality of life. Especially in youthful Kenya where the median age 19.4 years, studying adolescence is key, which brings us to the second characteristic of our sample: the realities of life in Kenya, a low-income country. Poverty is a known risk factor for mental disorders (Najman et al., 2010), as well as inferior quality of life and superior impairments. There is a pressing need to study depression and anxiety in SSA to better understand which symptoms most critically need attention to ameliorate living conditions. Finally, our data were collected from a community (not clinical) sample. As this is the first network analysis for depressive and anxiety symptoms with a community sample of adolescents in SSA, future studies are required to replicate these findings both with other communal samples and in clinical samples as well. These future studies, together with our findings, will help expand our understanding of adolescent depression and anxiety in understudied populations—high utility information that can inform treatment options.

Furthermore, future studies of a cross-cultural nature can help us understand cross-cultural differences in symptom centrality and associations between symptoms. It is possible that symptom networks are different across cultures, and such research will help explain the nature of these differences if any. Finally, additional studies can help inform treatment by identifying targets. For example, as the central symptoms of adolescent depression appear to be *self-blame* and *depressed mood*, interventions targeting these symptoms might be effective for treating adolescent depression. Similarly, interventions targeting *too much worry* and *uncontrollable worry* might be effective for treating adolescent anxiety. Future studies should investigate these possibilities.

Conclusion

Our study examines item-level depression and anxiety symptoms in a large community sample of school-going adolescents in Kenya using network analysis. Our results indicate that the most central symptoms of adolescent depression are *self-blame* and *depressed mood*, which suggests that these

symptoms might be of significant importance in the experience of adolescent depression. We also find strong associations between certain symptoms, particularly *self-blame—depressed mood* and *self-blame—trouble concentrating*, which might be useful links to target in clinical intervention. Similarly, we find the most central symptoms of adolescent anxiety to be *too much worry and uncontrollable worry*, which supports that *worry* is the core symptom of anxiety. We also find anxiety symptoms to have strong associations, particularly *uncontrollable worry—too much worry* and *trouble relaxing—restlessness*, which suggests that these links might be of significant importance in the experience of adolescent anxiety. Overall, these results could be helpful in expanding our understanding of adolescent depression and anxiety in understudied populations. Cutting edge network analytic techniques, like those employed in this study, supplement standard cross-sectional analyses of adolescent depression and anxiety (such as sum-scores) and, together with longitudinal studies, can help provide information of high clinical utility that can expand on our current sparse understanding of adolescent depression and anxiety in SSA contexts.

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Tables and Figures

Table 1

Participant demographic information

Characteristic	N (%)	M (SD)
Age		15.21 (1.14)
Gender		
Female	1246 (56.80)	
Male	890 (40.60)	
School type		
National	1715 (78.2)	
Rural (sub-county)	476 (21.7)	

Note: For more information regarding school type, see Ndetei et al., 2008.

Table 2

Mean, Standard Deviation, Minimum, Maximum, Skewness, and Kurtosis of PHQ and GAD symptoms.

Variable	M	SD	Min	Max	Skewness	Kurtosis
PHQ-8 Symptoms:						
PHQ-1: Little interest/pleasure	1.13	1.00	0	3	0.67	-0.56
PHQ-2: Depressed mood	0.95	0.96	0	3	0.86	-0.18
PHQ-3: Sleep problems	1.00	1.08	0	3	0.76	-0.73
PHQ-4: Little energy	0.89	0.92	0	3	0.89	-0.02
PHQ-5: Appetite problems	0.83	1.04	0	3	1.02	-0.26
PHQ-6: Self-blame	1.25	1.13	0	3	0.42	-1.22
PHQ-7: Trouble concentrating	1.20	1.08	0	3	0.50	-1.00
PHQ-8: Psychomotor problems	0.71	0.99	0	3	1.24	0.32
GAD-7 Symptoms:						
GAD-1: Nervousness	1.1	1.01	0	3	0.63	-0.66
GAD-2: Uncontrollable worry	1.26	1.20	0	3	0.42	-1.12
GAD-3: Too much worry	1.31	1.26	0	3	0.38	-1.18
GAD-4: Trouble relaxing	0.89	0.73	0	3	0.92	-0.22
GAD-5: Restlessness	0.60	0.42	0	3	1.47	1.28
GAD-6: Irritability	1.08	0.98	0	3	0.67	-0.82
GAD-7: Feeling afraid	1.16	1.07	0	3	0.57	-0.92

Note: PHQ-8 = Patient Health Questionnaire. GAD-7 = Generalized Anxiety Disorder Screener.

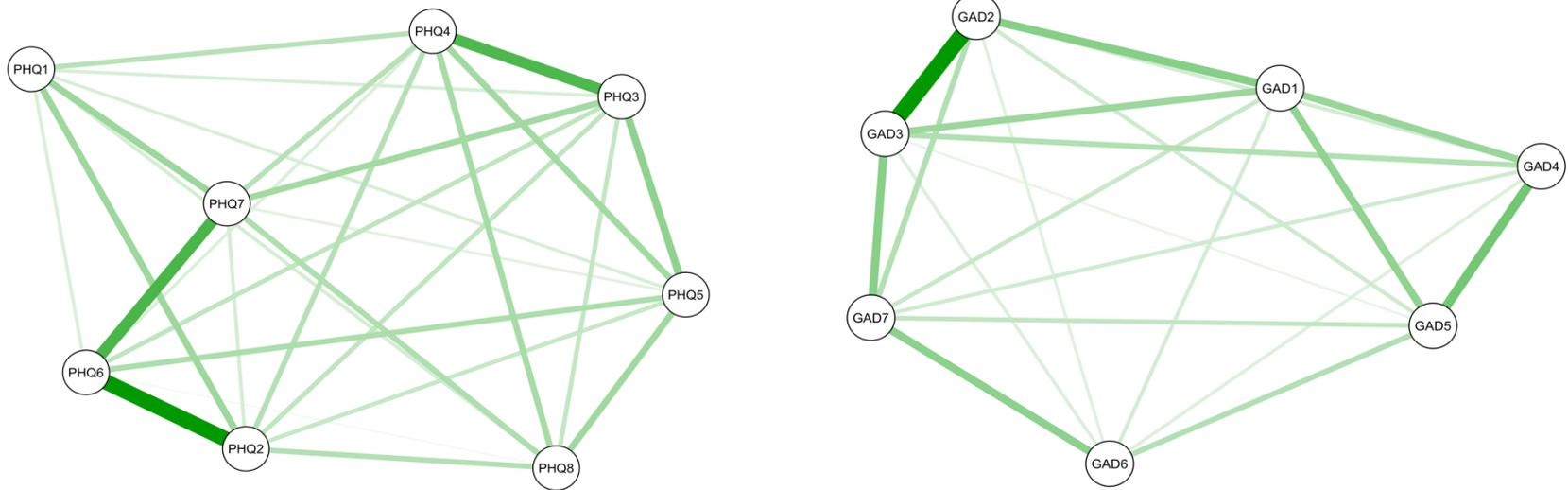


Figure 1. Regularized partial correlation network for depression symptoms (left) and anxiety symptoms (right) for all participants.

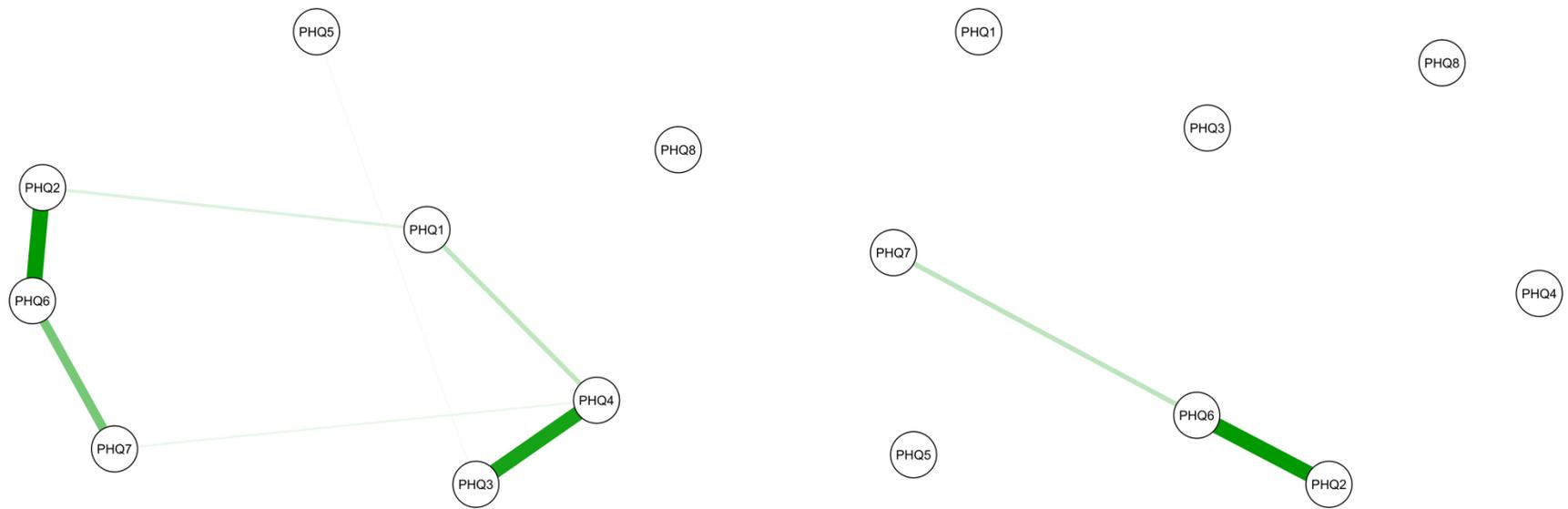


Figure 2. Regularized partial correlation network for depression symptoms of the low-symptom subsample (left) and the high-symptom subsample (right).

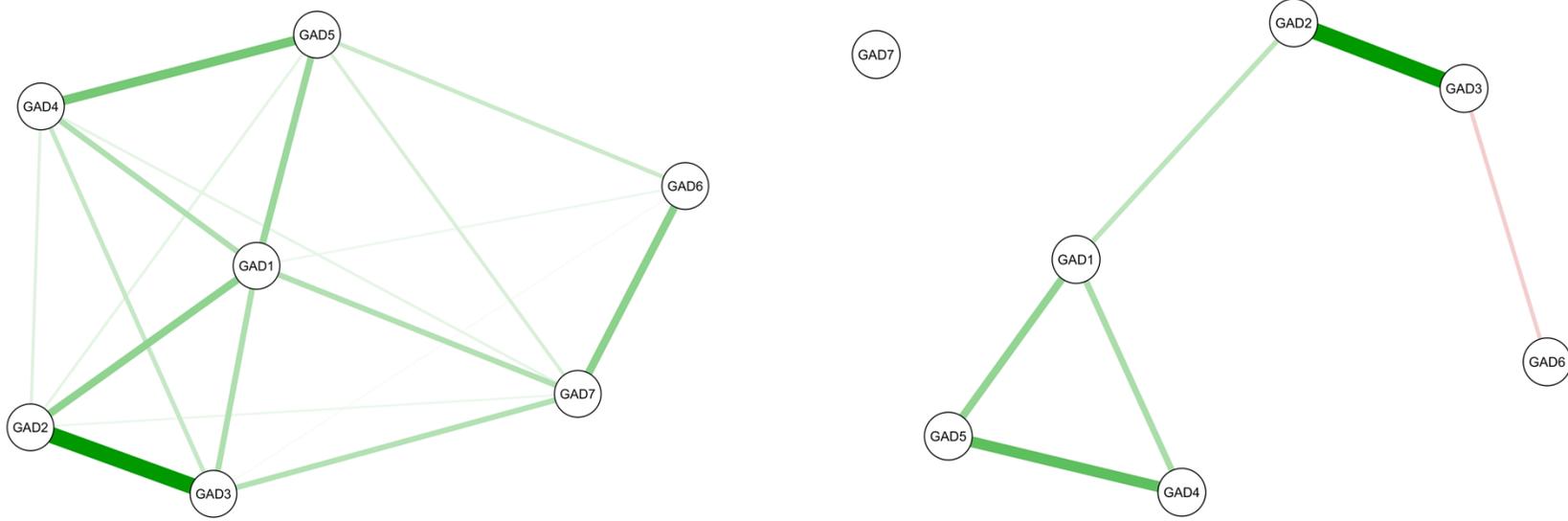


Figure 3. Regularized partial correlation network for anxiety symptoms of the low-symptom sub-sample (left) and the high-symptom subsample (right).

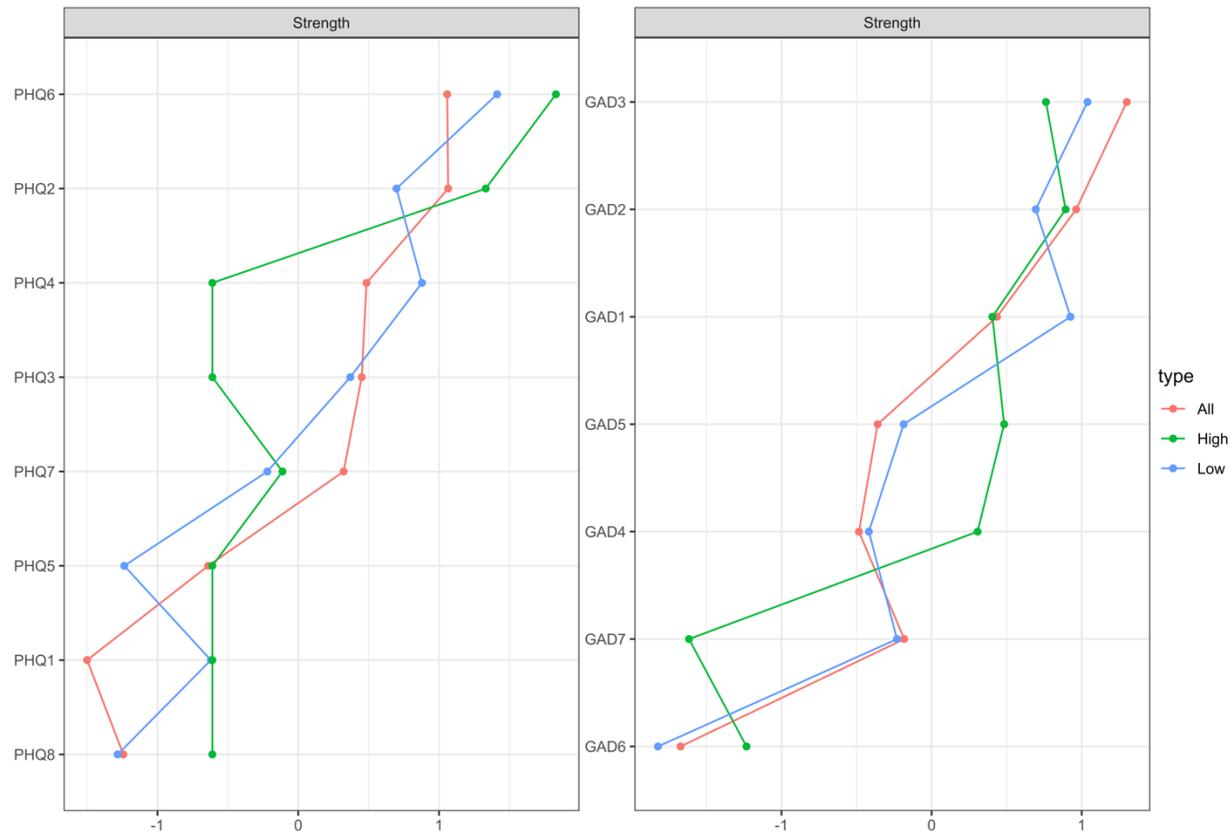


Figure 4. Centrality indices (strength, expected influence, closeness, betweenness) of each PHQ and GAD symptom.