A NETWORK APPROACH TO EARLY LIFE ADVERSITY, SLEEP DISTURBANCE, AND DEPRESSIVE SYMPTOMS

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A THESIS

Submitted in partial fulfillment of the requirements
for the degree of Master of Arts
in the Department of Psychology
in the Graduate School of
The University of Alabama

TUSCALOOSA, ALABAMA

2022

ABSTRACT

Experiences of early life adversity (ELA) engender a variety of adverse health outcomes, such as obesity, the metabolic syndrome, and major depressive disorder. Compared to individuals without exposure to adversity, individuals with ELA experience a greater likelihood of sleep disturbance. Associations between early adversity and sleep disturbance may explain increased risk for depressive symptoms in individuals with ELA. However, the bidirectional nature of the relationship between sleep disturbance and major depression presents a challenge in unraveling the temporality of these associations. Moreover, the underlying affective, somatic, and interpersonal factors that influence sleep disturbance in individuals with ELA are poorly understood. To address this gap, we employed a nomothetic network analytic approach to assess ELA-specific differences between the presentation of comorbid sleep disturbance and depressive symptoms. To this end, data from the second wave of the National Survey of Midlife Development in the United States study (MIDUS2, N=1255, 56.8%=Female) were used to generate ELA-specific networks of sleep disturbance and depressive symptoms. Individuals exposed to ELA experienced greater comorbid somatic, cognitive, and interpersonal symptoms of depression compared to those without ELA (M = 0.15; $p < 0.05$). Moreover, we found that depressive symptoms were more severe among those with ELA, relative to those without (S = 0.24; 1.4 vs 1.1, $p < 0.01$). Bridged network analysis indicated that there was increased sleep disturbance and depressive symptom comorbidity among those with ELA, relative to those without ELA (S = 0.54; 4.1 vs 3.6, $p < 0.01$). In adherence with the NIH's Research Domain Criteria, our findings underscore the utility of

novel statistical approaches to identify transdiagnostic mechanisms of risk that may result from environmental exposures. Statistical approaches which uncover these transdiagnostic mechanisms of risk may serve to develop trauma-informed interventions, thereby serving to assuage the public health burden of ELA.

LIST OF ABBREVIATIONS AND SYMBOLS

a	Cronbach's alpha - statistic representing internal consistency of a measure
F	Computed F statistic resulting from an F test
p	Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value
r	Correlation stability coefficient of networks
t	Computed value of t test
M	Computed test statistic resulting from a network invariance test
S	Computed test statistic resulting from a network strength invariance test
E	Network edge statistic
N	Number of participants in a group or sample
η^2	Eta squared, a measure of effect size in an analysis of variance
ε^2	Epsilon squared, a measure of effect size in a Kruskall-Wallis rank sum test
X^2	Chi-squared test statistic derived from a Chi-square test for independence
<	Less than
=	Equal to
$	Dollar amount
%	Percent
SD	Standard deviation

ACKNOWLEDGEMENTS

First, I would like to thank my mother and father, for their immeasurable sacrifice and their journey to the United States, which ultimately facilitated my capacity to pursue a fulfilling life of learning. Second, I'd like to thank my primary advisor and committee chair, Dr. Matthew R Cribbet for his undying patience, support, and mentorship throughout my time at the University of Alabama. It was him who introduced me to network analysis and ultimately inspired a rekindling of my love for applied statistical analysis. I'd also like to thank my committee members, Drs Mengya Xia and Jason DeCaro, for their brilliant contributions and insight during the development of my thesis and related work.

I am also grateful to the Department of Psychology at the University of Alabama for the wonderful opportunities I have been exposed to throughout my training. These experiences continue to inspire and propel me to pursue my endeavors. Finally, to my siblings, friends, and mentors external to the University of Alabama – *thank you* - for your unconditional support, guidance, and perspective as I navigate a rigorous program. It was you all who kept me grounded in moments when I experienced uncertainty.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Contemporary analytical approaches, such as network analysis, use symptom-level data to better understand both individual variation in psychopathology and variation in psychological symptoms that may co-occur across disorders (Borsboom & Cramer, 2013). To this end, network analysis allows researchers to model the covariance between symptoms of psychological disorders, thereby granting a foundation to assess comorbid relationships after a common cause or exposure (Fried et al., 2017). Traditional conceptualizations of psychopathology hold that underlying constructs, or latent variables, are made manifest through symptoms. For instance, the underlying latent construct of depression may lead to hypersomnia, anhedonia, and social withdrawal. The network framework provides an alternative approach, wherein psychological disorders are not characterized as an underlying latent construct but are instead comprised of the dynamic relationships between the symptoms of the disorders themselves (Borsboom & Cramer, 2013).

Within the network framework, symptoms are modeled as *nodes* and the relationships between symptoms are characterized as *edges*. Edges may be quantified as either odds ratios or correlation coefficients. The system of edges and nodes is known as a network and may represent a constellation of symptoms. A single network informs us how strongly symptoms are related to all others within its respective network. The magnitude of the association between a single symptom and all other symptoms within its network is called *centrality*. Because some symptoms may be more strongly associated with all others in its network, symptoms can be

compared in terms of their centrality – with more central symptoms exhibiting stronger associations to all other symptoms in its network and less central symptoms exhibiting weaker associations to all other symptoms in its respective network.

Not only does the network approach to psychopathology provide a method to quantify the most central symptoms within a single constellation of symptoms, but this framework also quantifies the magnitude of association between symptoms in one network and symptoms in a separate network. This concept, *bridge centrality*, describes the association between a single symptom in one network and all other symptoms in a separate network (Jones, Ma, & McNally, 2019). Like measures of centrality, different symptoms can be compared on the grounds of their bridge centrality, unveiling symptoms which strongly co-occur across psychological disorders (i.e., potential mechanistic targets for transdiagnostic interventions).

In addition to measures of centrality and bridge centrality, network statistics can be compared between groups who vary on a particular characteristic, thereby granting further insight into how the relationship between symptoms within and across networks differ between strata. For instance, individuals with varying socioeconomic positions might be compared to examine how symptoms of depression differ at various levels of socioeconomic position. In network analysis, these differences might present as differences in network structure (i.e., the manifestation of symptoms is different between groups) and differences in network strength (i.e., the strength of correlation between symptoms is different between groups).

In short, network analyses provide a framework to model relationships within and between networks of symptoms without assumptions of a latent construct. This alternative approach to examining psychopathology has demonstrated promise across studies and

disorders (Borsboom & Cramer, 2013; Elliott et al., 2019). For instance, in a network analysis conducted by Levinson and colleagues (2017), the symptoms which link Bulimia Nervosa to symptoms of depression and anxiety were explored. Results of this study showed that fear of weight gain was a central symptom of Bulimia Nervosa. Furthermore, the study found that sensitivity to physical sensations was most related to symptoms of depression and anxiety. However, the utility of network analyses is not limited to eating disorder research and has been demonstrated across various facets of psychopathology and psychology (Borsboom & Cramer, 2013).

That is, the network perspective provides insight to specific constellations of symptoms that teeter across diagnostic categories. Knowledge of comorbid symptoms that cut across diagnostic categories may elucidate the presence of shared biological mechanisms between mental disorders (Guloksuz et al., 2017). Further, investigations using the network perspective may aid in discriminating symptoms of sleep disturbance that may impact the prognosis of some mental disorders, but not others. For instance, impaired sleep has been implicated in depression, bipolar disorder, generalized anxiety disorder, post-traumatic stress disorder, and schizophrenia (Krystal, 2012). Further, sleep problems are included in the diagnostic criteria for all these disorders and improved prognosis of these disorders has been shown after treatment of these sleep problems (Krystal, 2012). Nevertheless, it remains unknown *which* symptoms of sleep disturbance are affecting the prognosis of specific disorders. Thus, this underscores the potential clinical and descriptive contribution of network approaches in discriminating sleep disturbance symptoms specific to mental disorders, which share hallmark symptoms with other mental disorders - such as depression.

To this end, exploration of existing data using network frameworks allows us to investigate criticisms that target the validity of predominant nosological systems (i.e., DSM-V and ICD-10), while propelling future research towards the identification of transdiagnostic elements of psychological functioning – as described in the National Institutes of Health's Research Domain Criteria (Insel et al., 2010). Specifically, cross-sectional network analysis grants researchers the capacity to examine specific symptom-to-symptom relationships, after controlling for all other symptoms (Fried & Cramer, 2017).

Further, regularization techniques often employed in network analyses decreases the possibility of false positive associations between these networks. When examining multiple disorders (i.e., using bridge centrality statistics), these methods provide sensitive, data-driven information regarding associations between clusters of symptoms *across* diagnostic categories, potentially unveiling underlying biological mechanisms shared between disorders. The contribution of these techniques is two-fold. That is, this methodology serves to explore the DSM-V's emphasis on reliability over validity of pathological processes which underlie psychological disorders (Regier et al., 2013), while unveiling potential shared biological and psychosocial processes which underlie the development of mental disorders that have high prevalence of comorbidity with others.

Network Analysis: Applications in the field of ELA, Sleep Disturbance and Depression

Historically, exposure to early life adversity (ELA) has been shown to precede both sleep disturbance (Kajeepete et al., 2015) and depressive symptoms (Merrick et al., 2019), independently. However, there is little work examining the impact of ELA on the *comorbidity* of depression and sleep disturbance at the symptom level. The dearth of research examining sleep and depression symptom comorbidity is incongruent with the widespread prevalence of exposure to early life adversity and its contribution to the development of depression. Indeed,

prevalence estimates obtained from samples in the United States of America (USA) demonstrate that approximately 61% of adults have experienced at least one instance of early life adversity (Finkelhor et al., 2009; McLaughlin et al., 2012; Gilbert et al., 2015; Merrick et al., 2019). Early life adversity (ELA) is defined as potentially traumatic events, including but not limited to physical or emotional neglect, parental loss (Bifulco et al., 1997; Marquez et al., 2021), poverty (Hughes & Tucker, 2018), sexual assault or abuse, and exposure to violence (Keane et al., 1989; Kilpatrick et al., 1989; Felitti et al., 1998; Merrick et al., 2019). ELA has been linked to negative mental and physical health outcomes (Anda et al., 2009; Dube et al., 2009; Felitti et al., 1998), including obesity (Alvarez et al., 2007; Marquez et al., 2021), cardiovascular disease (Dong et al., 2004), and depression (Chapman et al., 2004). Depression bears an incremental economic burden that increased from $236 billion in 2010 to $326 billion from in 2020 (Greenberg et al., 2021). Most notably, twenty-one million (population attributable fraction of 44.1%) cases of depression are associated with exposure to ELA, a preventable risk factor (Merrick et al., 2019). These findings suggest that up to 44% of incident cases of depression may be prevented by addressing exposure to ELA and the downstream behavioral outcomes of ELA.

Although there is much work examining ELA as an antecedent to depression and sleep disturbance, previous research has often conceptualized sleep disturbance as a symptom of depression or as a mechanism on the pathway from ELA to depression (Franzen & Buysse, 2008; Fang et el., 2019). These theoretical conceptualizations have resulted in the utilization of mediation and multilevel analyses to understand causal associations between symptoms of sleep disturbance and other psychological disorders (Franzen & Buysse, 2008; Fang et al., 2019).

However, traditional statistical frameworks - like mediation, multiple regression, and perhaps even multilevel analysis - may not be equipped to model the reciprocal relationships between sleep disturbance and depressive symptoms (Fried & Cramer, 2017). Moreover, traditional methods have yet to model the ELA-specific differences in the reciprocal relationships between sleep disturbance and depressive symptoms.

To address this gap, we employ a network analytic framework to examine the comorbidity between sleep disturbance and depression *at the symptom-level.* Moreover, we examine how these comorbid relationships differ in those exposed to ELA and those who have not experienced ELA. In direct contrast to previous research examining the temporality of the relationships between depression and sleep disturbance, we employ network analysis to explore the symptom-level *comorbidity* between symptoms of sleep disturbance and depression.

To this end, we employ network analysis to test three hypotheses. First, we hypothesize that ELA-specific differences in network structure and density will be present in symptoms of sleep disturbance. Second, we hypothesize that ELA-specific differences will be present in network structure and density of depressive networks. Finally, given the high levels of comorbidity in sleep disturbance and depression (Fang et al., 2019), we hypothesize that ELA- specific differences in sleep disturbance networks will coincide with ELA-specific differences in depressive networks.

Not only will this study provide insight to the specific elements of sleep disturbance that co-occur with symptoms of depression, but this study's network approach will grant insight into the differential presentation of comorbid sleep disturbance and depression symptoms between those with exposure to ELA and those without. This so-called hybrid

approach (Fried & Cramer, 2017) will not only serve to discern symptoms of sleep disturbance related to the prevention and prognosis of depression, but it will serve to grant understanding of co-occurring symptoms after exposure to a common cause, namely ELA. Given the known comorbidity between sleep disturbance and depression (Fang et al., 2019; Freeman et al., 2020) as well as the cross-sectional nature of the data, this study's use of network analysis will prove advantageous traditional methods (i.e., mediation frameworks and cumulative risk modeling). In contrast to traditional methodology, this network analysis will explore symptom-level ELA-specific differences in sleep disturbance and depression comorbidity, thereby elucidating affective and cognitive factors specific to individuals with ELA that may lead to the disruption of sleep and mood. While previous studies have examined the temporality of sleep disturbance and depression, our exploration of co-morbid symptomatology will contribute a novel understanding of how shared biological mechanisms (e.g., HPA and SNS dysregulation) manifest as symptoms at the population level. Accordingly, findings from this study will grant insight to transdiagnostic mechanisms of risk that underlie both sleep disturbance and depression. Above and beyond the prevention of depression, such knowledge may also inform future research investigating shared biological mechanisms of risk between sleep disturbances and depression and may assist in the discrimination of sleep disturbance symptoms which are specific to depression.

METHODS

Participants

Americans were recruited (N=1255) in 2004 and 2006 during the second wave of the original MIDUS study. These individuals participated in follow-up from the initial wave of MIDUS, which collected data on the sociodemographic, psychosomatic, behavioral, and physical health characteristics of 7108 Americans in 1995. During the second wave of data collection, participants completed phone interviews consisting of sociodemographic, psychosomatic, and medical history questionnaires. Participants also completed two mailed self-administered questionnaires along with a phone-administered cognitive battery. Further details regarding recruitment and sample characteristics are provided in a previous study (Brim et al., 2004).

Measures

Early Life Adversity (ELA). Early life adversity was assessed using the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994). The CTQ is a retrospective self-report questionnaire that assesses the frequency of adversity across 5 subscales consisting of physical abuse, physical neglect, emotional abuse, emotional neglect, and sexual abuse. Question types vary across subscale and are summed to create subscale scores. The CTQ includes items like *when I was growing up, I did not have enough to eat.* Responses range from *never true* (1) to *very often true* (5). Subscale scores range from 5-25. The CTQ has demonstrated good internal and intraclass reliability (α=0.79-0.94; intraclass=0.88). Subscale-specific cut-offs were used to categorize individuals into those with *no level of abuse/neglect, low levels of abuse/neglect,*

moderate levels of abuse/neglect, and *severe levels of abuse/neglect.* Given that cut-off scores are highly sensitive in their classification of lower thresholds of abuse and neglect (sensitivity ranging 79%-89%; Bernstein et al., 1994), clinical cut-offs were used to separate individuals with no histories of trauma (non-ELA) and individuals with histories of trauma (ELA).

Individuals who experienced any form of trauma at low-to-severe levels were all considered to have previous exposure to ELA. All other individuals (i.e., those who endorsed experiencing *no* abuse or neglect) were included in a non-ELA group.

Sleep Disturbance. The *Pittsburgh Sleep Quality Index* (PSQI) was used to measure symptoms of sleep disturbance (Buysse et al., 1989). The PSQI is a 19-item measure that assesses sleep quality and disturbances as a function of seven component scores (i.e., sleep latency, duration, efficiency, disturbances, daytime dysfunction, perceived sleep quality, and use of medication for sleep). The PSQI includes items like, *During the past month, how often have you had trouble sleeping because you cannot get to sleep within 30 minutes.* Responses range from *Not during the past month (0)* to *Three or more times a week (3).* The PSQI component scores are derived using the first 9 items of the scale. Component scores are derived from items 1-9 in the instrument. Seven-node networks were generated from the seven component scores and were examined for stability, centrality, and global strength (Borsboom & Cramer, 2013).

Depressive symptomatology. Depressive symptoms were assessed using the *Center for Epidemiological Studies Depression Scale* (CES-D; Lewinsohn et al., 1997). The CES-D is a 20- item scale that ascertains proximal symptoms of depression. The CES-D includes items such as *I felt fearful, I felt lonely,* and *I felt sad.* Items are scored in a 0-3 Likert-type scale,

with higher scores indicating a greater frequency of depressive symptoms within the last week. Scores are summed to obtain composite scores. Composite CES-D scores can range from 0 to 60, such that higher scores indicate a greater frequency of depressive symptomatology. The CES-D has high internal consistency and good measures of sensitivity and specificity (Lewinsohn et al., 1997). In this sample, measures of internal consistency suggested very good reliability (alpha=0.89). Items of this subscale were utilized to generate networks of depressive symptomatology within domains of positive affect (range (R): 0-12), depressive affect (R: 0-20), somatic (R: 0-18), and interpersonal functioning (R: 0-6). Networks of depressive symptomatology were assessed for stability, centrality, and global strength (Borsboom & Cramer, 2013).

STATISTICAL ANALYSIS

Descriptive Statistics

All analyses were conducted with statistical software *R* (https://cran.r-project.org/). In particular, the packages 'bootnet', 'ggplot2', 'networktools', and 'NetworkComparisonTest' were used for network analyses. Univariate analyses included appropriate measures of central tendency (i.e., median for non-normal variables and means for normally distributed variables) for continuous variables and proportions for categorical variables in both the overall sample and the sample stratified by ELA status.

Bivariate analyses included Pearson χ^2 tests to assess for differences in categorical variables between groups. ELA-specific differences in normally distributed variables were assessed using one-way analysis of variance. Differences in non-normally distributed variables were assessed using Kruskall-Wallis rank-sum tests with comparisons of median and inter- quartile range statistics. Zero order correlations were conducted to examine the potential for multicollinearity (Berry et al., 1985). All analyses were considered significant at an alpha level of 0.05.

Network Analysis

Separate networks were generated to model sleep disturbance and depressive symptoms in ELA groups versus non-ELA groups. As described in previous studies (Borsboom & Cramer, 2013; Fried & Cramer, 2017; Elliot et al., 2019), correlation stability coefficients (test statistic: *r*) were calculated to examine the fit and stability of these networks. Correlation stability

coefficients were derived by assessing the sameness of centrality statistics calculated from bootstrapped subsets of the original sample. That is, different iterations of network centrality statistics were calculated using subsamples of the original data. For instance, a subsample generated from 25% of the data was compared to a subsample generated using 95% of the sample. Networks were considered interpretable insofar as their correlation stability coefficients were above a cut-off of $r > 0.5$.

To examine which symptoms or nodes are most central to each network, measures of expected influence were calculated. Expected influence is defined by the absolute value of all correlations between one node in a network and all other nodes in its respective network. As described in previous research (Elliot et al., 2019), these measures account for negative as well as positive relationships between symptoms. Therefore, expected influence would be appropriate to model the heterogenous valence of relationships between symptoms of depression and sleep disturbance.

To explore whether symptoms of sleep disturbance are linked to differential presentations of depressive symptomatology, we also calculated measures of bridge expected influence (Jones, Ma, & McNally, 2021). Bridge expected influence is the absolute value of all correlations between one node and all other symptoms within and outside of its respective network. Thus, this estimate quantifies the correlation between one symptom in a single network (e.g., sleep disturbance) and all other symptoms in a separate network (e.g., depression).

To assess for ELA specific differences in the presentation of symptoms of sleep disturbance and depression, network invariance tests as well as global strength invariance tests were conducted using the R package NetworkComparisonTest (van Borkulo et al., 2017).

Briefly, these tests are conducted by first estimating the test statistic from observed data. Then, a reference distribution is generated by obtaining test statistics from permutations of the observed data. Finally, the observed test statistics (*M* for network invariance; *S* for global strength invariance) are compared to the derived distribution and a significance value is obtained.

Network invariance tests measure the extent to which network structures (i.e., presentation of symptoms) differ between groups, while global strength invariance tests measure the extent to which the network connectivity or density (i.e., strength of relationships between symptoms) differs between groups. Significant findings in network invariance tests were followed up by edge invariance tests to assess which symptom relationships were driving significant differences in network structure. Edge invariance tests were corrected for false discovery rates using the Benjamini & Hochberg correction (Benjamini & Hochberg, 1995).

RESULTS

Descriptive Statistics and Correlation Analyses

Most of the sample (N=713; 56.8%) was female. On average, individuals were 54 years old at the time of data collection. Among 1255 individuals, 781 (62.2%) reported experiencing ELA. Those without ELA were, on average, older than those with ELA (F (1,1244) = 26.23; 55 versus 54, $p < 0.05$, $\eta^2 = 0.003$). Individuals with ELA reported significantly more median measures of depressive affect (Kruskal-Wallis $X^{2\ =}$ 99.90; 1.0 versus 0.0, $p < 0.05$, $\varepsilon^2 = 0.06$), lower median measures of positive affect (Kruskal-Wallis $X^{2\ =}$ 80.70, 9.0 versus 11.0, $p < 0.05$, $\varepsilon^2 = 0.05$), and significantly more somatic complaints (Kruskal-Wallis $X^{2\ =}$ 103.31, 4.0 versus 2.0, $p < 0.05$, $\varepsilon^2 = 0.06$) relative to those without ELA. Overall, individuals with ELA demonstrated significantly greater overall measures of depressive symptoms (F (1,1244) = 110.5; 8.0 versus 4.0, $p < 0.05$, $\eta^2 = 0.08$) and significantly larger average global measures of sleep disturbance compared to those without ELA (F (1,1170) = 35.83; 6.7 versus 5.4, $p < 0.05$, $\eta^2 = 0.03$).

Pearson correlations were conducted between depressive symptoms and sleep disturbance to assess for potential multicollinearity. Using a cut-off of 0.8 (Berry et al., 1985), results of correlation analyses were indicative that it was unlikely that network estimates would be affected by multicollinearity. Descriptive statistics are presented in **Table 1** and correlation analyses are shown in **Table 2**.

Network Analysis - Stability Coefficients

Regularized partial correlation networks were generated for sleep, depression, and both sleep and depression in samples stratified by ELA. Thus, a total of 6 networks were generated. In networks generated for those with ELA, networks of depressive symptoms had measures of expected influence stability and strength stability that were 0.75. Similarly, networks of sleep disturbance symptoms had measures of expected influence stability and strength stability that were 0.75. In bridged networks including both clusters of depressive and sleep disturbance symptoms generated for those with ELA, both strength and expected influence stability coefficients were 0.75. Network models for groups with ELA are depicted in **Figures 1-3**.

In networks generated for those without ELA, networks of depressive symptoms had measures expected influence stability and strength stability that were 0.75. For this group, networks of sleep disturbance symptoms had expected influence and strength coefficients of 0.59. In bridged networks for this group, expected influence and strength stability coefficients were 0.67. Network models for groups without ELA are depicted in **Figures 4-6**. Given that all networks had stability coefficients above 0.5, measures for all 6 networks were deemed interpretable.

Network Analysis - Between-Group Differences in Symptomatology

Using the NetworkComparisonTest package in R (van Borkulo et al., 2017), the network invariance and global strength invariance hypotheses were tested in sleep, depression, and bridged sleep and depression networks. The former hypothesis holds that the structure of networks is identical between groups. That is, there are no between-group differences in the presentation of symptoms. The global strength invariance hypothesis holds that there are no between-group differences in the strength of the relationship between

symptoms. Comparison tests of sleep disturbance networks indicated that the structure and strength of networks did not differ significantly by ELA ($M = 0.11$; $S = 0.05$; ps > 0.05).

Network comparison tests indicated that there were statistically significant between-group differences in depressive network structure ($M = 0.15$; $p < 0.05$). Moreover, those with ELA had greater global strength measures compared to those without ELA, suggesting that symptoms of depression present differently and are more strongly correlated in those with ELA ($S = 0.24$; 1.4 vs 1.1, $p < 0.01$). Specifically, depressive networks for those with ELA demonstrated symptoms of depressive affect that were more strongly correlated with lower positive affect ($E = 0.12$; $p < 0.01$) and interpersonal problems ($E = 0.07$; $p < 0.05$), relative to non-ELA networks of depressive symptoms. Additionally, in those with ELA, lower positive affect was more strongly related to interpersonal problems, relative to those without ELA ($E = 0.04$; $p = 0.01$).

When assessing for ELA-specific differences in bridged network structure and strength (both sleep disturbance *and* depression networks), it was found that network structure did not differ between groups ($M = 0.11$; $p > 0.05$). However, there were statistically significant between-group differences in global strength measures, with those with ELA demonstrating greater global strength ($S = 0.54$; 4.1 vs 3.6, $p < 0.01$). These findings suggest that although the joint presentation of sleep disturbance and depressive symptoms may not differ by ELA, the strength of association between all symptoms of sleep disturbance and all symptoms of depression are larger in those with ELA.

Network Analysis - Overall and Within-Group Centrality Measures

To assess which symptoms were most correlated with all other symptoms in a respective network, measures of expected influence were calculated in all 6 networks.

In depressive symptom networks generated for those with ELA (**Figure 1**), somatic complaints were found to be significantly more related to all other symptoms of depression. In those with ELA, measures of subjective sleep quality were significantly more associated with all other symptoms of sleep disturbance (**Figure 2**). In those with ELA, somatic complaints were most related to all other symptoms of depression and symptoms of sleep disturbance (**Figure 3**).

In depressive symptom networks generated for those without ELA (**Figure 4**), depressive affect was most associated with all other symptoms of depression, this finding was not significant. In networks of sleep disturbance, those without ELA had measures of subjective sleep quality that were most closely related to all other measures of sleep disturbance (**Figure 5**). In bridged networks, those without ELA had measures of sleep disturbance that were most closely correlated with all other symptoms of both sleep disturbance and depression (**Figure 6**).

DISCUSSION

In this secondary data analysis, we found that there were no statistically significant ELA- specific differences in sleep disturbance networks. These findings are incongruent with previous polysomnographic findings that show disrupted non-REM sleep and increased REM sleep in individuals with ELA (Insana et al., 2012; Lewin et al., 2019). Given that one of these studies did not control for depression (Insana et al., 2012) and the other used a convenience sample of military veterans (Lewin et al., 2019), this incongruency might indicate that ELA-preceded depressive symptoms may be driving differences in sleep architecture in human and animal models. However, in a one-way ANOVA, there were significant differences in global PSQI score between those with and without ELA (as shown in **Table 1**). It is likely that the global PSQI may be less reflective of actual sleep parameters and more related to a negative cognitive point of view, general dissatisfaction, or general pessimistic thinking (Grandner et al., 2006).

Despite the lack of differences in sleep disturbance networks, the presentation of self-reported depressive symptoms and the magnitude of association between depressive symptoms differed significantly between participants with ELA compared to participants without ELA. Specifically, participants who experienced ELA exhibited greater measures of depressive network connectivity, relative to those without ELA. In corroboration with previous research (Kendler, Karkowski, & Prescott, 1999), our finding suggests that individuals with ELA experience symptoms of depression that are not only more severe but are also more strongly

correlated relative to those without ELA, suggesting that symptoms of depression were more likely to co-occur in those with ELA, relative to those without ELA.

In addition to differences in network density, we also found marked ELA-specific differences in depressive symptom profiles. Distinctively, those with ELA had symptoms of depressive affect that were more strongly linked to lower positive affect and interpersonal difficulties relative to those without ELA. Moreover, networks generated for those with ELA showed stronger relationships between lower positive affect and interpersonal problems. Given known relationships between ELA and difficulties with intimacy, the development of insecure attachment, and maladaptive interpersonal strategies (Hyu Jung Huh et al., 2014), these findings might suggest that increased depressive network connectivity (i.e., increased depressive symptom severity) among those with ELA might be explained by an unwillingness to appropriately seek social support that may aid in ameliorating depressive symptoms.

Furthermore, when we assessed depressive and sleep disturbance networks together, we found that both sleep and depression networks were more densely connected in those with ELA relative to those without ELA. This finding suggests that those with ELA may have symptoms of depression and sleep disturbance that are more likely to co-occur when compared to individuals without ELA. Despite this difference in network density, we did not find a statistically significant difference in the structure of these bridged networks. This might indicate that ELA- specific differences in the magnitude of co-occurring sleep disturbance and depressive symptoms may be driven by differences in the density of depressive networks. Alternatively, these findings might indicate that ELA-specific differences in the co-occurrence of sleep disturbance symptom may be dependent on depressive symptoms. Future studies

might assess these by employing intensive longitudinal data - such as experience sampling – to assess whether acute disruptions in sleep are preceded by changes in mood.

Taken together, these ELA-specific differences suggest that individuals with ELA may not only be more likely to experience comorbid sleep disturbance and depression, but they may also present with different depressive symptomatology when compared to those without ELA. Specifically, there were significant ELA-specific differences in the relationships between depressive affect and lack of positive affect, lack of positive affect and somatic complaints, and somatic complaints and interpersonal complaints. These differences suggested that the relationships between the previously mentioned symptoms were stronger in populations exposed to ELA, relative to those without ELA. Together, these findings suggest that these symptoms are more likely to co-occur in those with ELA when compared to those without this exposure.

Several limitations of our methods and findings warrant discussion. First, cross-sectional data were used to examine ELA-specific differences in comorbid symptoms between depressive and sleep disturbance networks. Because of this, we were unable to examine the temporality of the associations between depressive and sleep disturbance symptoms. The use of cross-sectional data limited our ability to draw causal inferences regarding the comorbid relationships between sleep disturbance and depressive symptoms. That is, by aggregating symptom measures across people with and without ELA, the inferences that were drawn from this study are purely nomothetic in nature and do not apply to single individuals (Goloksuz et al., 2017; Fried & Cramer, 2017). Future studies might employ idiographic network approaches to not only examine sleep disturbance and depression comorbidity at the individual level, but they may also explore this comorbidity

while examining the temporality of these relationships.Furthermore, the cross-sectional assessment of sleep disturbance and depressive symptom data may not be representative of long-term sleep habits and mood. Replication of these analysis on longitudinal data may provide information regarding the direction of association between symptoms of sleep disturbance and depression. Knowledge of the direction of these relationships may elucidate the temporal structure of symptom clusters, thereby further uncovering targets for interventions and may inform optimal timing for intervention.

Additionally, because our study employed data from participants at middle age, we may have been limited in our assessment of depressive symptoms due to the lower prevalence of depression at mid-life (Sutin et al., 2014). Future studies exploring similar aims might employ data that includes participants at a broad range of ages, as opposed to only including participants at midlife. Given that our network analysis was nomothetic in nature, populations with greater base rates of depression – such as groups in young adulthood (Sutin et al., 2014) – may exhibit different network structures from those at middle age. Moreover, our study may have also been limited due to the retrospective appraisal of ELA. Previous research illustrates that retrospective assessments of ELA may be prone to recall biases and may disagree from prospective assessments of this exposure (Baldwin et al., 2019). Thus, our findings in network structure and density may differ from those found in studies that assess ELA using prospective measures.

Future studies might explore the influence of measurement type (i.e., prospective versus retrospective) on network structure and density.

Beyond the use of cross-sectional data, our use of component scored sleep disturbance data is another important limitation. Specifically, the components of the PSQI (Pittsburgh

Sleep Quality Index) are scored on a 0-3 scale. The use of a 0-3 scale may have reduced the amount of variability in sleep disturbance data, thereby limiting our capacity to detect statistically significant differences in network structure and density. Further studies might employ raw data from the PSQI or explore data collected from daily sleep diaries. Such data may provide more granularity in network estimates and may uncover detailed correlations between symptoms that may not have been uncovered in our analyses.

Taken together, these findings have important implications for future work on informing treatment and intervention research among adults who have experienced ELA. Specifically, our findings underscore the importance of tailored transdiagnostic interventions for depression for trauma exposed individuals, but that also include a sleep intervention component. Viable treatment approaches might include sedative antidepressants, Cognitive Behavioral Therapy (CBT) for depression, combination therapy, sleep restriction therapy, and deep brain stimulation. In particular, CBT for insomnia (CBT-I) has been shown to be effective in improving depression and insomnia symptoms in patients with comorbid depression and insomnia (Manber et al., 2008). It is likely that tailored CBT-I interventions for individuals exposed to ELA may assuage the downstream effects of ELA on executive function and emotion regulation (Tyrka et al., 2013), such as increased propensity for risky health behaviors (Nusslock & Miller., 2016; Alvarez et al., 2007; Marquez et al., 2021). These transdiagnostic interventions may ultimately reduce the burden of adverse health outcomes in those with ELA.

Conclusions

The objective of this study was to ascertain the differential presentation in sleep disturbance and depressive symptoms between individuals with ELA and individuals without

ELA using network analysis. Consistent with our hypotheses, we illustrated an ELA-specific differential presentation of depressive symptomatology. Moreover, we found that sleep disturbance and depressive symptoms co-occurred more strongly in individuals with ELA relative to those without ELA. We also demonstrated that our findings derived from bridged networks may have been partially attributable to differences found in depressive network strength and structure. These findings underscore the importance of trauma-informed transdiagnostic interventions for depression which simultaneously target sleep disturbance in individuals with ELA.

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TABLES

Table 1. Overall and Stratified Descriptive Statistics

	Overall	ELA			
		No	Yes	Test Statistic	*p*-value
N	1255	474	781		
Age (mean (SD))	54 (11.7)	55 (12.5)	53.95 (11.2)	F (1, 1253) = 4.96	0.026
Sex = n female (%)	713 (56.8)	254 (53.6)	459 (58.8)	X^2 = 3.02	0.082
CESD - Depressive Affect (median [IQR])	1.00 [0.00, 3.00]	0.00 [0.00, 1.00]	1.00 [0.00, 4.00]	KW X^2 = 99.90	<0.001
CESD - Positive Affect (median [IQR])	10.00 [8.00, 12.00]	11.00 [9.00, 12.00]	9.00 [7.00, 11.00]	KW X^2 = 80.70	<0.001
CESD - Somatic Complaints (median [IQR])	3.00 [1.00, 5.00]	2.00 [1.00, 4.00]	4.00 [2.00, 6.00]	KW X^2 = 103.31	<0.001
CESD - Interpersonal (median [IQR])	0.00 [0.00, 1.00]	0.00 [0.00, 0.00]	0.00 [0.00, 1.00]	KW X^2 = 66.54	<0.001
CESD - Total (median [IQR])	7.00 [3.00, 12.00]	4.00 [1.00, 8.00]	8.00 [4.00, 15.00]	F (1,1170) = 35.83	<0.001
CTQ - Emotional Abuse (median [IQR])	6.00 [5.00, 9.00]	5.00 [5.00, 6.00]	8.40 [6.00, 12.00]	KW X^2 = 428.90	<0.001
CTQ - Physical Abuse (median [IQR])	6.00 [5.00, 7.00]	5.00 [5.00, 6.00]	7.00 [5.00, 9.00]	KW X^2 = 247.80	<0.001
CTQ - Sexual Abuse (median [IQR])	5.00 [5.00, 5.00]	5.00 [5.00, 5.00]	5.00 [5.00, 8.00]	KW X^2 = 233.11	<0.001
CTQ - Emotional Neglect (median [IQR])	9.00 [6.00, 13.00]	6.00 [5.00, 7.00]	11.00 [9.00, 15.00]	KW X^2 = 637.21	<0.001
CTQ - Physical Neglect (median [IQR])	6.00 [5.00, 8.00]	5.00 [5.00, 5.00]	7.00 [5.00, 9.00]	KW X^2 = 366.66	<0.001
CTQ - Minimization/Denial (median [IQR])	0.00 [0.00, 1.00]	1.00 [0.00, 2.00]	0.00 [0.00, 0.00]	KW X^2 = 234.80	<0.001
PSQI - Global (mean (SD))	6.23 (3.68)	5.41 (3.37)	6.72 (3.77)	F (1,1170) = 35.83	<0.001
ELA = n (%)	781 (62.2)				

IQR=Interquartile Range; CESD=Center for Epidemiological Studies-Depression Scale.
CTQ=Childhood Trauma Questionnaire; PSQI=Pittsburgh Sleep Quality Inventory; Differences in non-normal variables were assessed using Kruskal-Wallis rank-sum tests (KW; with median comparison), differences in normally distributed variables were assessed using analysis of variance

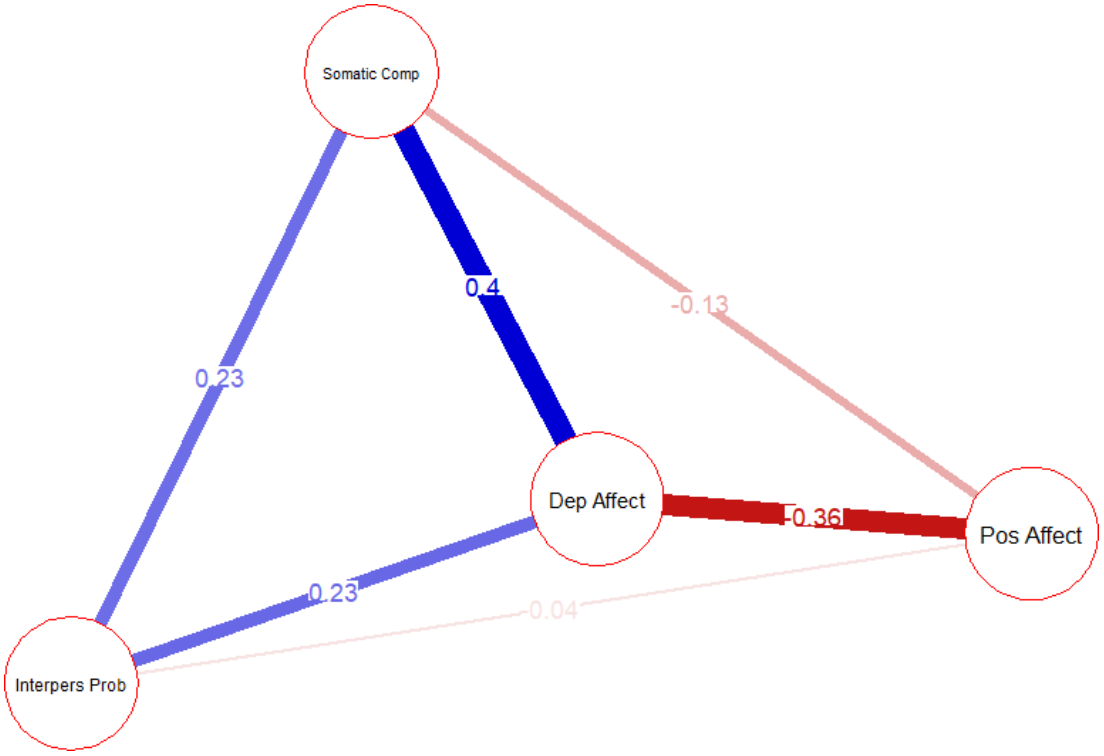
Table 2. Pearson Correlations for Sleep Disturbance and Depressive Symptom Measures

	Depressive affect	Positive affect	Somatic complaints	Interpersonal problems	Sleep duration	Sleep disturbances	Sleep latency	Daytime dysfunction	Sleep efficiency	Sleep quality	Medication use
Depressive affect	1.000	**-0.562**	**0.656**	**0.512**	**0.285**	**0.259**	**0.155**	**0.178**	**0.283**	**0.197**	**0.364**
Positive affect	/	1.000	**-0.456**	**-0.323**	**-0.241**	**-0.169**	**-0.122**	**-0.175**	**-0.231**	**-0.147**	**-0.328**
Somatic complaints	/	/	1.000	**0.439**	**0.449**	**0.343**	**0.228**	**0.224**	**0.354**	**0.245**	**0.491**
Interpersonal problems	/	/	/	1.000	**0.187**	**0.144**	**0.136**	**0.115**	**0.151**	**0.075**	**0.272**
Sleep duration	/	/	/	/	1.000	**0.432**	**0.425**	**0.302**	**0.395**	**0.216**	**0.383**
Sleep disturbances	/	/	/	/	/	1.000	**0.272**	**0.274**	**0.376**	**0.293**	**0.235**
Sleep latency	/	/	/	/	/	/	1.000	**0.376**	**0.199**	**0.104**	**0.266**
Daytime dysfunction	/	/	/	/	/	/	/	1.000	**0.140**	**0.128**	**0.161**
Sleep efficiency	/	/	/	/	/	/	/	/	1.000	**0.217**	**0.298**
Sleep quality	/	/	/	/	/	/	/	/	/	1.000	**0.168**
Medication use	/	/	/	/	/	/	/	/	/	/	1.000

Note: All coefficients above are Pearson product-moment correlations. Bold text indicates statistical significance at $p < 0.05$

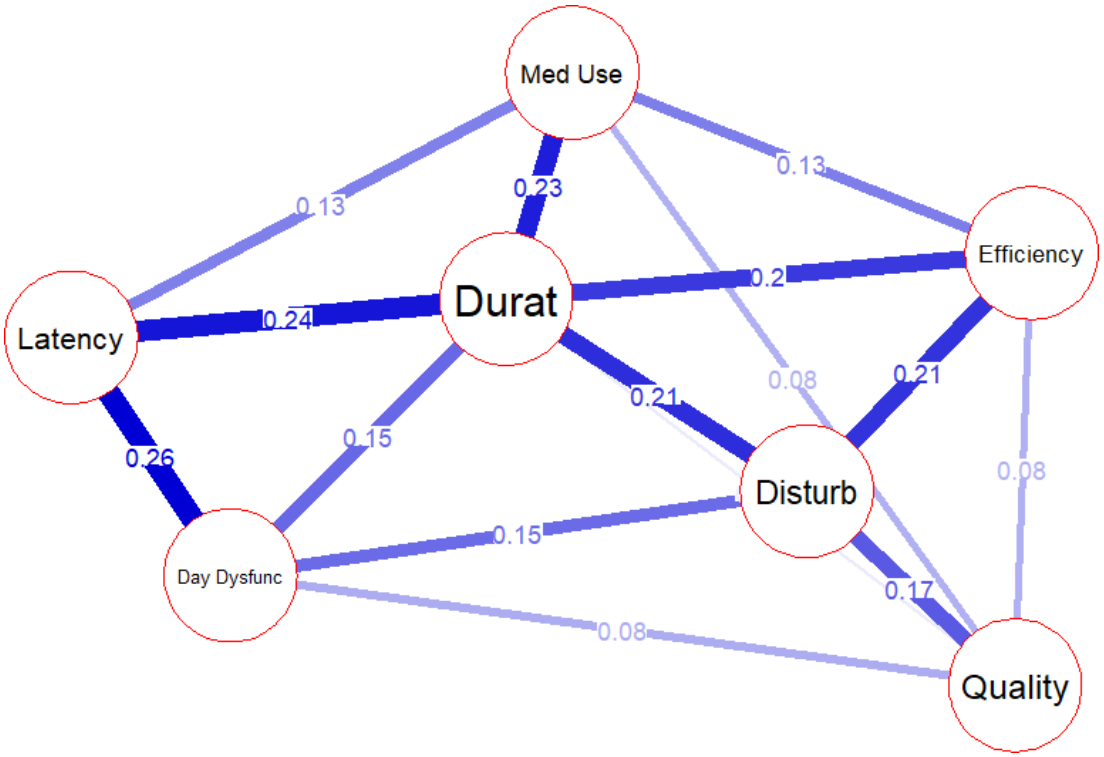
FIGURES

Figure 1. Depression Symptom Network in MIDUS2 Participants with Exposure to ELA



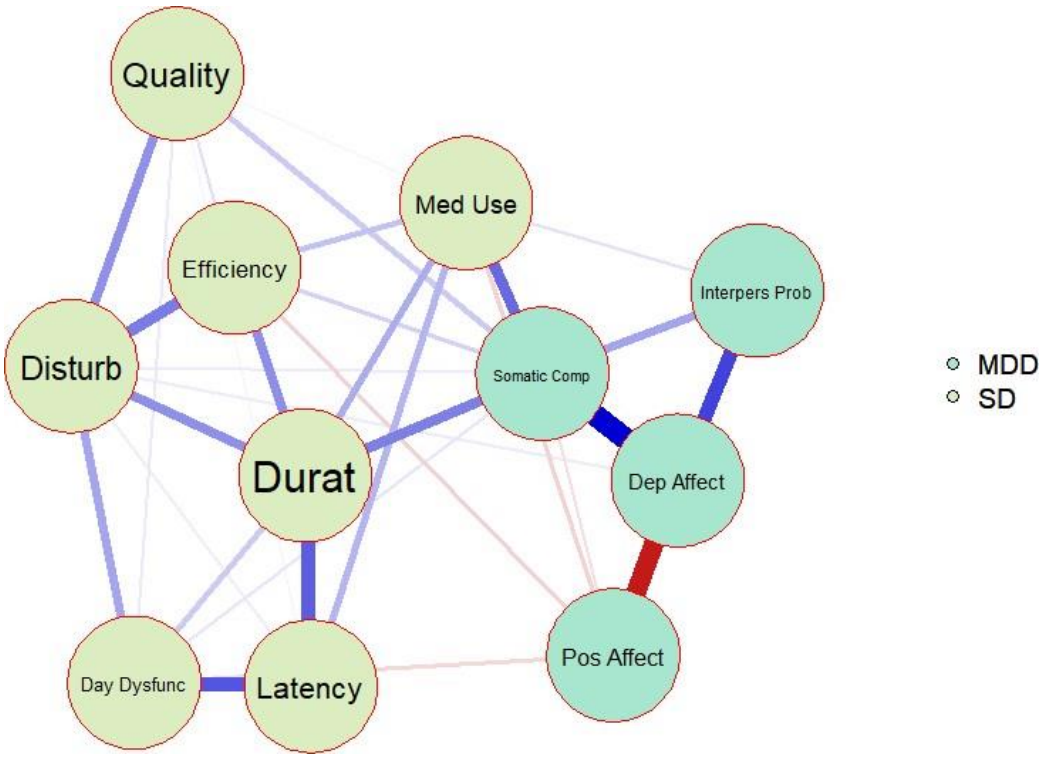
Notes: Somatic Comp = Somatic Complaints; Pos Affect = Positive Affect; Dep Affect = Depressive Affect; Interpers Prob = Interpersonal Problems. Blue lines denote positive regularized partial correlations and red lines denote negative regularized partial correlations.

Figure 2. Sleep Disturbance Symptom Network in MIDUS2 Participants with Exposure to ELA



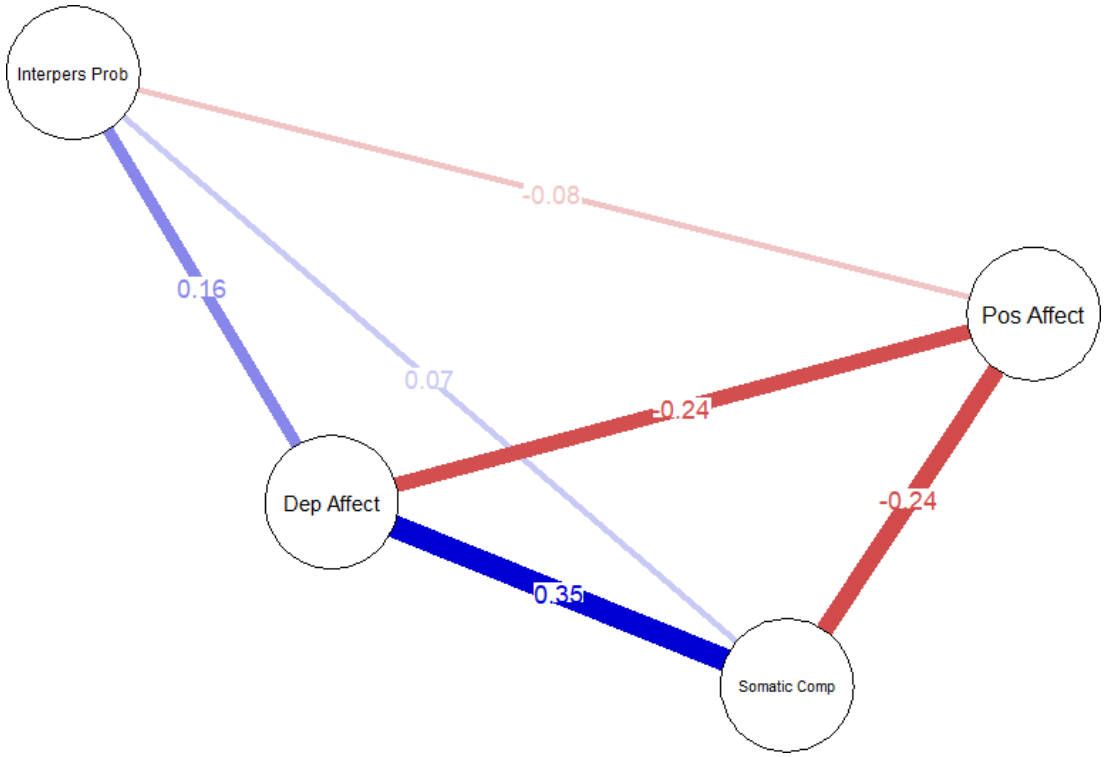
Notes: Quality = Subjective Sleep Quality; Latency = Sleep Latency; Durat = Sleep Duration; Efficiency = Habitual Sleep Efficiency; Disturb = Sleep Disturbances; Med Use = Use of Sleep Medications; Day Dysfunc = Daytime Dysfunction. Blue lines denote positive regularized partial correlations and red lines denote negative regularized partial correlations.

Figure 3. Bridged Depression and Sleep Disturbance Symptom Network in MIDUS2 Participants with Exposure to ELA



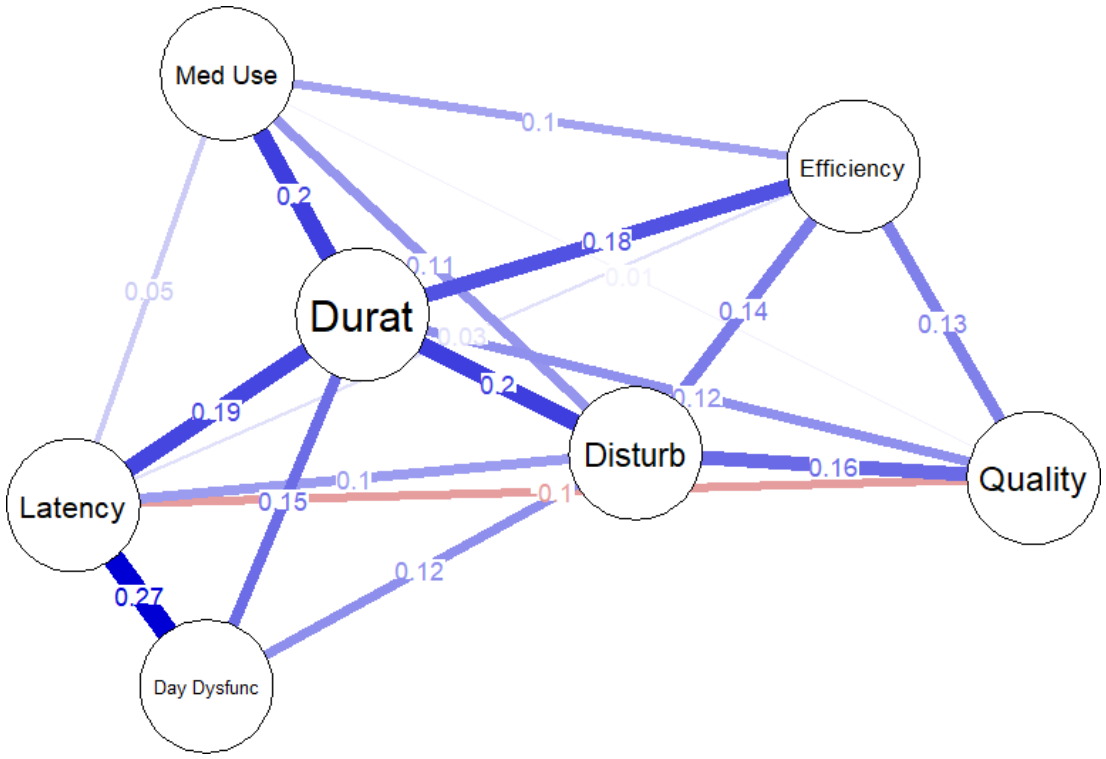
Notes: Somatic Comp = Somatic Complaints; Pos Affect = Positive Affect; Dep Affect = Depressive Affect; Interpers Prob = Interpersonal Problems. Quality = Subjective Sleep Quality; Latency = Sleep Latency; Durat = Sleep Duration; Efficiency = Habitual Sleep Efficiency; Disturb = Sleep Disturbances; Med Use = Use of Sleep Medications; Day Dysfunc = Daytime Dysfunction. Blue lines denote positive regularized partial correlations and red lines denote negative regularized partial correlations. *Edge weights omitted for ease of representation.*

Figure 4. Depression Symptom Network in MIDUS2 Participants without Exposure to ELA



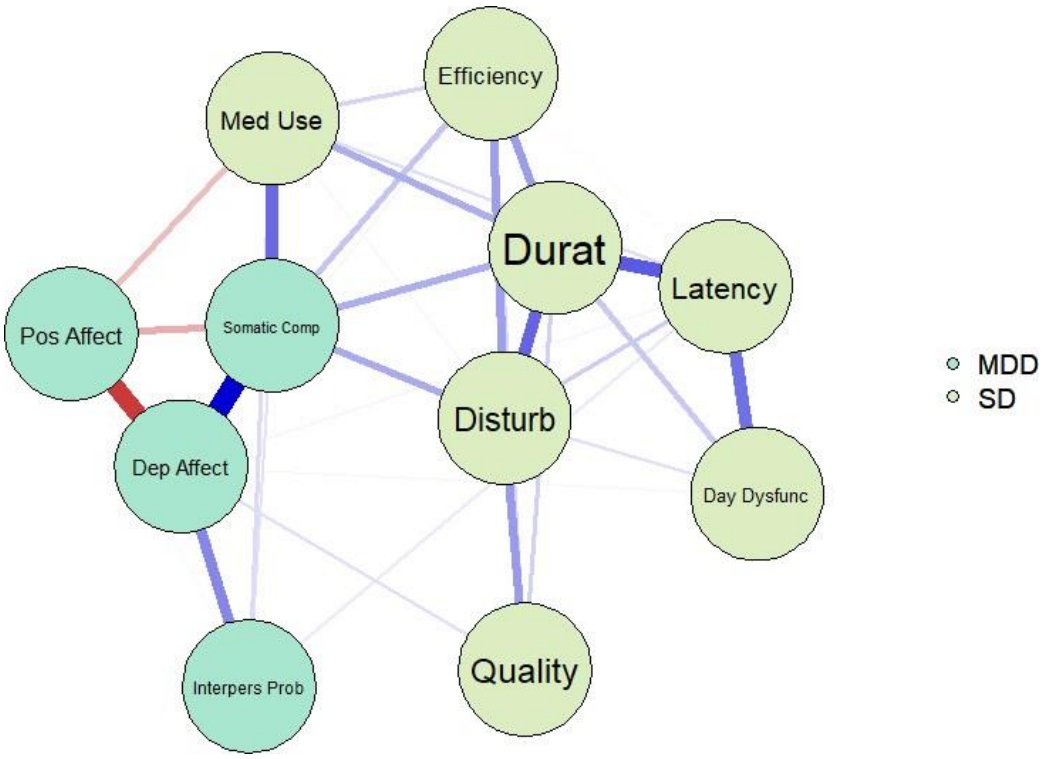
Notes: Somatic Comp = Somatic Complaints; Pos Affect = Positive Affect; Dep Affect = Depressive Affect; Interpers Prob = Interpersonal Problems. Blue lines denote positive regularized partial correlations and red lines denote negative regularized partial correlations.

Figure 5. Sleep Disturbance Symptom Network in MIDUS2 Participants without Exposure to ELA



Notes: Quality = Subjective Sleep Quality; Latency = Sleep Latency; Durat = Sleep Duration; Efficiency = Habitual Sleep Efficiency; Disturb = Sleep Disturbances; Med Use = Use of Sleep Medications; Day Dysfunc = Daytime Dysfunction. Blue lines denote positive regularized partial correlations and red lines denote negative regularized partial correlations.

Figure 6. Bridged Depression and Sleep Disturbance Symptom Network in MIDUS2 Participants without Exposure to ELA



Notes: Somatic Comp = Somatic Complaints; Pos Affect = Positive Affect; Dep Affect = Depressive Affect; Interpers Prob = Interpersonal Problems. Quality = Subjective Sleep Quality; Latency = Sleep Latency; Durat = Sleep Duration; Efficiency = Habitual Sleep Efficiency; Disturb = Sleep Disturbances; Med Use = Use of Sleep Medications; Day Dysfunc = Daytime Dysfunction. Blue lines denote positive regularized partial correlations and red lines denote negative regularized partial correlations. *Edge weights omitted for ease of representation.*