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Unique longitudinal relationships between symptoms of psychopathology in youth: A cross-lagged panel network analysis in the ABCD study

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Background: The network theory suggests that psychopathology may reflect causal relationships between individual symptoms. Several studies have examined cross-sectional relationships between individual symptoms in youth. However, these studies cannot address the directionality of the temporal relationships hypothesized by the network theory. Therefore, we estimated the longitudinal relationships between individual internalizing, externalizing, and attention symptoms in youth. **Methods:** Data from 4,093 youth participants in the Adolescent Brain Cognitive Development (ABCD) study were used. Symptoms were assessed using the Brief Problem Monitor, which was administered at three time points spaced six months apart. Unique longitudinal relationships between symptoms at T1 and T2 were estimated using cross-lagged panel network modeling. Network replicability was assessed by comparing this network to an identically estimated replication network of symptoms at T2 predicting symptoms at T3. **Results:** After controlling for all other symptoms and demographic covariates, depressed mood, inattention, and worry at T1 were most predictive of other symptoms at T2. In contrast, threats of violence and destructiveness at T2 were most prospectively predicted by other symptoms at T1. The reciprocal associations between depressed mood and worthlessness were among the strongest bivariate relationships in the network. Comparisons between the original network and the replication network (correlation between edge lists = .61; individual edge replicability = 64%–84%) suggested moderate replicability. **Conclusions:** Although causal inferences are precluded by the observational design and methodological considerations, these findings demonstrate the directionality of relationships between individual symptoms in youth and highlight depressed mood, inattention, and worry as potential influencers of other symptoms. **Keywords:** Comorbidity; continuity; symptomatology; developmental psychopathology; etiology.

Introduction

Psychopathological symptoms in youth are prevalent, impairing, and associated with increased risk for the onset of psychopathology in adolescence and adulthood (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Psychopathologies in youth and adults have typically been conceptualized as latent variables indicated by individual symptoms (Schmittmann et al., 2013), and studies of the structure of psychopathology largely suggest that youth psychopathology can be categorized into latent internalizing, externalizing, and attention disorders/syndromes (Haltigan et al., 2018). Research has examined the homotypic (i.e., a disorder predicting itself at a later time point) and heterotypic (i.e., a disorder prospectively predicting another disorder) continuity of diagnoses or latent psychopathology factors, which may identify potential causal relationships between disorders that may reflect mechanisms underlying comorbidity (e.g., Shankman et al., 2009; Shevlin, McElroy, & Murphy, 2017; Snyder, Young, & Hankin, 2017). However, growing evidence suggests that individual symptoms may have different courses (van Eeden, van Hemert, Carlier, Penninx, & Giltay, 2018) and distinct genetic and environmental etiologies (e.g., Keller, Neale, & Kendler, 2007; Myung et al., 2012). Extending this evidence supporting the differential validity of individual symptoms, the network theory of psychopathology suggests that psychopathology is the result of causal relationships between individual symptoms (Borsboom, 2017). Additionally, diagnostic comorbidity may result from causal relationships between symptoms of two disorders ('bridging edges') or shared symptoms (e.g., concentration difficulty in both major depressive disorder and generalized anxiety disorder) that relate to other symptoms of both disorders ('bridge symptoms'; Cramer, Waldorp, van der Maas, & Borsboom, 2010). An important implication of the network theory is that previous studies of the homotypic and heterotypic continuity of diagnoses or latent factors may have studied psychopathology at the wrong level (Fried, 2015).

Psychopathology network models have aimed to identify the putative causal relationships between symptoms by estimating the unique relationship between each pair of symptoms ('edges') after

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statistically controlling for all other variables in the network (e.g., partial correlations). Although the majority of psychopathology network analyses have focused on psychopathology in adults, several studies have used cross-sectional network modeling to examine the unique relationships between symptoms in youth and/or adolescents. Findings from transdiagnostic symptom network analyses suggest that symptoms within the same domain (e.g., internalizing) tend to cluster together graphically. However, boundaries between domains are not distinct and individual symptoms differ in the quantity and strength of their within-domain and cross-domain connections (Boschloo, Schoevers, van Borkulo, Borsboom, & Oldehinkel, 2016; Rouquette et al., 2018), highlighting the importance of utilizing symptom assessments that cut across diagnostic boundaries. These studies have also provided novel information about the relationships between specific symptoms. For instance, sleep disturbance may not be directly related to depression symptoms, but rather indirectly connected to depressive symptoms via worry (Boschloo et al., 2016). In addition, investigations of symptom centrality (e.g., the number and strength of connections between a symptom and its neighboring symptoms) have found that feeling 'unhappy/sad' and 'anxious/fearful' were most central in a network of internalizing symptoms (McElroy, Fearon, Belsky, Fonagy, & Patalay, 2018), and inattention was the most central symptom in a network of internalizing, externalizing, and attention symptoms and prosocial behaviors (Rouquette et al., 2018). It is therefore important to incorporate symptoms from multiple different domains of psychopathology in symptom network models to better understand the unique and potentially causal relationships between symptoms.

However, these studies in youth (and most psychopathology network analyses more broadly) estimated undirected networks from cross-sectional data, which cannot provide any information about the direction of relationships. For example, if two symptoms are strongly connected in an undirected network, it is possible that one symptom leads to the other symptom, in which case the first symptom may potentially cause the second and represent a viable target for intervention. However, it is also possible that the second symptom leads to the first symptom, in which case intervening on the first symptom would have no effect on the second symptom. Intervening on the first symptom would also have no impact on the second symptom if a third unmeasured variable affects them both (i.e., a common cause) or if a common effect of both (i.e., a collider) is in the network, inducing a conditional dependence. As centrality measures are based on the estimated relationships between symptoms, it is impossible to discern whether a central symptom causes other symptoms or is caused by other symptoms from an undirected network. Because of their inability to discern the direction of relationships, undirected cross-sectional networks can only provide limited insight into the *temporal* and *causal* relationships hypothesized by the network theory. Longitudinal data are therefore necessary (but not sufficient) to model the temporal relationships between symptoms.

The present study therefore examined the unique longitudinal relationships between individual symptoms from the three major symptom clusters often found in youth – attention, externalizing, and internalizing – using cross-lagged panel network (CLPN) modeling. CLPN modeling was developed to apply a network approach to the modeling of temporal effects between individual elements of a construct in panel data (Rhemtulla, Cramer, van Bork, & Williams, 2019) and is well suited for identifying the temporal relationships between symptoms hypothesized by the network theory. In addition to modeling and interpreting individual edges between symptoms, we computed symptom centrality indices to identify which symptoms were most central in terms of prospectively predicting, and being predicted by, other symptoms. Based on findings from previous transdiagnostic cross-sectional network analyses (Boschloo et al., 2016; McElroy et al., 2018; McElroy, Shevlin, Murphy, & McBride, 2018; Rouquette et al., 2018), it was hypothesized that depressed mood, inattention, anxiety, and oppositional defiant disorder (ODD) symptoms would be among the most central in predicting other symptoms. As there is ongoing controversy regarding the replicability of network models (e.g., Forbes, Wright, Markon, & Krueger, 2019; Funkhouser et al., 2020) and symptoms were assessed at three time points, we examined the replicability of the CLPN by comparing the results of a CLPN using T1 symptoms to predict T2 symptoms to an identical model using T2 symptoms to predict T3 symptoms.

Methods

Participants

This study used data collected from youth at the six-month ('T1'), twelve-month ('T2'), and eighteen-month ('T3') follow-up assessments of the Adolescent Brain Cognitive Development (ABCD) study (data release 2.0; NDAR-https://doi.org/10.15154/1503209). The ABCD study consists of a nationally representative sample of over 11,000 youth aged 9 or 10 at enrollment. A detailed description of the design and recruitment approach of the ABCD study is available elsewhere (Garavan et al., 2018). Briefly, youth participants and their parents were recruited from elementary schools within the catchment areas of the 21 ABCD study research sites, which encompassed over 20% of the United States population of youths aged 9 or 10. Multistage probability sampling was used to yield a sample that closely approximated national sociodemographics. This design and recruitment approach aims to minimize systematic sampling biases and maximize the generalizability of inferences drawn from the sample to the population. Schools were selected based on gender, race and ethnicity, socioeconomic status, and urbanicity. Written

informed consent was obtained from all participants, and the rights of participants were protected under the local IRBs. Additionally, the demographics of the accumulating sample were monitored throughout recruitment, and recruitment strategies were adjusted to reduce observed deviations from demographic targets. To examine predictive relationships between symptoms assessed six months apart, we extracted a subsample of youths who completed assessments at T1 and T2 (N = 4,093). A subsample of youths completed assessments at T2 and T3 (N = 1,583) was extracted to examine network replicability. When extracting each subsample, one sibling was randomly selected from families with multiple youth participants to avoid nonindependence of observations. The mean follow-up interval between assessments was 192.5 days (SD = 39.9) for T1 and T2, and 167.3 days (SD = 36.0) for T2 and T3. Demographic characteristics for each subsample are reported in Table 1.

Symptom assessment

This study used data from the youth-report Brief Problem Monitor (BPM; Achenbach, 2009), which assesses symptoms over the past week using 19 items drawn from the Child Behavior Checklist, Teacher Report Form, and Youth Self-Report (Achenbach & Rescorla, 2001). Items are rated as 0 ('not true'), 1 ('somewhat true'), or 2 ('very true') and are categorized into three domains (Attention, Internalizing, Externalizing). We used youth-report BPM data in the primary analyses because the youth-report BPM was administered at all three follow-ups for which data are available (Barch et al., 2018), while parent- and teacher-reported symptoms were only assessed at T2. However, we also conducted sensitivity analyses examining the extent to which results were sensitive to rater (youth versus parent) at T2, which are reported in Appendix S1 and Figures S1–S5. Youth-, parent-, and teacher-reported symptoms at T2 were weakly to moderately correlated across raters (median cross-informant correlation = .19; maximum = .43). In line with recommendations (Rhemtulla et al., 2019), two pairs of conceptually similar items were collapsed[1] prior to performing the CLPN analyses and missing data (≤1.0% of responses in both subsamples) were imputed using random forest imputation implemented via the *missForest* R package (Stekhoven & Buhlmann, 2012). Consistent with item-level analyses of other youth assessment measures such as the CBCL (Boschloo et al., 2016; Deutz, Geeraerts, van Baar, Deković, & Prinzie, 2016; McElroy et al., 2018), there was low endorsement of 2 ('very true') for some items. Therefore, items were dichotomized to indicate the presence ('somewhat true' and 'very true' = 1) or absence ('not true' = 0) of symptoms in line with common practice for item-level analyses of the CBCL (Achenbach & Rescorla, 2001; Boschloo et al., 2016; McElroy et al., 2018). Symptom endorsement rates at each time point are presented in Table 2.

Table 1 Demographic characteristics of each subsample

Characteristic	T1 → T2 Subsample (N = 4,093)	T2 → T3 Subsample (N = 1,583)
Age at T1 (M, SD)	10.45 (0.62)	–
Age at T2 (M, SD)	10.97 (0.63)	10.99 (0.61)
Age at T3 (M, SD)	–	11.45 (0.61)
Gender (% Female)	1916 (46.8%)	726 (45.9%)
Ethnicity (%)		
White	2408 (58.8%)	989 (62.5%)
Black	353 (8.6%)	102 (6.4%)
Hispanic	822 (20.1%)	302 (19.1%)
Asian	106 (2.6%)	36 (2.3%)
Other	404 (9.9%)	154 (9.7%)

T1 = six-month follow-up. T2 = twelve-month follow-up. T3 = eighteen-month follow-up.

Data analysis

The CLPN was estimated using a series of nodewise logistic regression models to compute autoregressive (i.e., the coefficient for a symptom at T1 predicting itself at T2 after controlling for all other symptoms at T1 and covariates) and cross-lagged (i.e., the coefficient for a symptom at T1 predicting a different symptom at T2 after controlling for all other symptoms at T1 and covariates) effects. Age at T1, sex, ethnicity (white versus nonwhite), and indicators of parental demographics and socioeconomic status (combined income, highest education level, and marital status) were included as covariates. There are many methods for network model selection, and the specificity and sensitivity of each method is a topic of ongoing investigation (e.g., Williams, Rhemtulla, Wysocki, & Rast, 2019). However, a recent simulation study comparing the performance of several regularization parameter selection criteria found that cross-validation had the highest sensitivity and lowest specificity across a variety of conditions (Wysocki & Rhemtulla, 2019). Because the present study did not interpret most individual edges or network density, regression coefficients were regularized using LASSO with 10-fold cross-validation tuning parameter selection to shrink small regression coefficients to exactly zero. The use of cross-validation means that the estimated CLPN is likely to contain the edges that make up the true population network, but may also contain some false positive edges and overestimate network density.

To increase interpretability, the coefficients of the logistic regressions (i.e., edge weights) were converted from log odds to odds ratios (ORs) by exponentiating the coefficients. Thus, an edge weight greater than 1 indicates a positive relationship, an edge weight below 1 reflects a negative relationship, and an edge weight that is exactly 1 indicates no relationship. Regularized regressions were computed using the *glmnet* package in R (Friedman, Hastie, & Tibshirani, 2010). The *qgraph* package was used for network plotting (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012), and node placement was determined using an algorithm that places more strongly connected nodes closer together (Fruchterman & Reingold, 1991). Identical layouts and the maximum value of the edge weights were imposed on each network plot to facilitate visual comparisons across networks.

Symptom centrality in undirected networks can be estimated using expected influence (EI), which is computed by summing the values of the edges connected to each symptom (Robinaugh, Millner, & McNally, 2016). EI provides a summary level index of the number, strength, and sign of the relationships between a given symptom and all other symptoms in the network, but as previously mentioned, it does not indicate the direction of relationships. As the CLPN is a directed network estimated from longitudinal data, we parsed directionality by separately calculating cross-lagged *out-EI* (i.e., the sum of the values of *outgoing* edges connected to a symptom) and *in-EI* (i.e., the sum of the values of *incoming* edges connected to a symptom). Additionally, we sought to identify bridge symptoms by calculating bridge EI (i.e., the sum of a symptom's edges to symptoms from other domains; Jones, Ma, & McNally, 2019). Although utilizing the three BPM domains is an obvious approach to categorizing symptoms, relying on factor analytically derived domains would be inconsistent with the network theory's assertion that symptom covariance is due to direct and causal relationships between symptoms rather than a latent factor. Therefore, we used the package *igraph* (Csárdi &

Table 2 Symptom labels and endorsement rates at each assessment

			T1 → T2 Subsample		T2 → T3 Subsample	
			T1	T2	T2	T3
Item	Label	Short Name	Endorsement	Endorsement	Endorsement	Endorsement
I act too young for my age	A1	behavioral immaturity	1184 (29.2%)	1089 (28.3%)	435 (29.0%)	407 (25.8%)
I fail to finish things I start	A2	poor task completion	1386 (34.0%)	1358 (33.9%)	523 (33.6%)	475 (30.1%)
I have trouble concentrating or paying attention/I am inattentive or easily distracted[a]	A3	inattention	2506 (61.3%)	2432 (60.4%)	972 (62.8%)	906 (57.3%)
I have trouble sitting still	A4	hyperactivity	1849 (45.2%)	2297 (56.6%)	901 (57.2%)	683 (43.2%)
I act without stopping to think	A5	impulsivity	1794 (44.0%)	1721 (42.6%)	666 (42.6%)	579 (36.6%)
I argue a lot	E1	argumentativeness	2215 (54.2%)	2118 (52.8%)	841 (54.3%)	810 (51.2%)
I destroy things belonging to others	E2	destructiveness	152 (3.7%)	165 (4.1%)	71 (4.5%)	43 (2.7%)
I disobey my parents/I disobey at school[a]	E3	disobedience	933 (22.8%)	979 (24.4%)	415 (26.8%)	291 (18.4%)
I am stubborn	E4	stubbornness	1459 (35.8%)	1667 (41.8%)	638 (41.5%)	583 (36.9%)
I have a hot temper	E5	irritability	1194 (29.2%)	1423 (35.2%)	567 (36.4%)	441 (27.9%)
I threaten to hurt people	E6	threats of violence	165 (4.0%)	209 (5.1%)	88 (5.6%)	51 (3.2%)
I feel worthless or inferior	I1	worthlessness	493 (12.1%)	426 (10.6%)	170 (11.0%)	131 (8.3%)
I am too fearful or anxious	I2	fear	1186 (29.1%)	1037 (25.8%)	366 (23.5%)	387 (24.5%)
I feel too guilty	I3	guilt	520 (12.7%)	534 (13.2%)	188 (12.0%)	143 (9.0%)
I am self-conscious or easily embarrassed	I4	social anxiety	1526 (37.3%)	1588 (39.4%)	642 (41.1%)	592 (37.4%)
I am unhappy, sad, or depressed	I5	depressed mood	719 (17.6%)	591 (14.6%)	251 (16.0%)	185 (11.7%)
I worry a lot	I6	worry	1561 (38.2%)	1643 (40.5%)	646 (40.9%)	516 (32.6%)

[a]Two pairs of items were collapsed based on conceptual similarity (see Footnote 1). T1 = six-month follow-up. T2 = twelve-month follow-up. T3 = eighteen-month follow-up.

Nepusz, 2006) to implement the spinglass algorithm, an algorithm that empirically identifies communities of symptoms in a network (Reichardt & Bornholdt, 2006).

The accuracy and stability of edge weights was estimated using two bootstrapping approaches implemented by the R package *bootnet* (Epskamp et al., 2018). First, edge weight accuracy was estimated by computing 95% confidence intervals (CIs) around each edge weight value via nonparametric bootstrapping using 1,000 iterations. Second, we used case-drop bootstrapping to estimate correlation stability (CS) coefficients to determine the stability of the rank-order of centrality indices. Edge weights difference tests and centrality difference tests were calculated to test whether differences between edge weights or node centralities were statistically significant (see Epskamp et al., 2018 for a detailed description of these methods). To evaluate network replicability, we used identical methods to estimate a replication network of T2 symptoms and covariates predicting T3 symptoms. Similarities between the two networks were evaluated using (a) the correlation between edge lists, which provides a global measure of network similarity, (b) the percentage of individual edges in one network that replicated in the other network (i.e., OR > 1 or OR < 1 in both networks), (c) correlations of centrality indices between networks, and (d) consistency in the most central symptoms.

Results

Accuracy and stability of network parameters

Bootstrapped confidence intervals around edge weights were small to moderate (see Figure S6). The rank-order of out- and in-EI had moderate to strong stability in the T1 → T2 network (CS coefficients = .44 and .51), but bridge EI was not stable (CS coefficient = 0; see Figure S7). Therefore, we only interpreted out-EI and in-EI. Edge weight difference tests and centrality difference tests are presented in Figures S8 and S9.

Network inference

The CLPN is plotted as a directed network (see Figure 1), in which arrows represent temporal pairwise relationships between symptoms controlling for all other symptoms at T1 and covariates. Edge weights are presented in Tables S1 and S2. All autoregressive edges were present (see Figure S10), and autoregressive edges (mean OR = 3.16) were substantially stronger than cross-lagged edges (mean OR = 1.17). Because the plotting algorithm determines path thickness relative to the strongest path, autoregressive edges were excluded from Figure 1 to make the cross-lagged edges more visually interpretable. Additionally, there were 225 estimated cross-lagged edges (208 [92.4%] with OR > 1). As plotting all cross-lagged edges would reduce interpretability, weaker edges (arbitrarily defined as odds ratios within 1 ± .35) were excluded from Figure 1. A plot that includes weaker edges is provided in Figure S11.

The spinglass algorithm identified three communities that were identical to the symptoms listed in the three BPM domains, with the exception that impulsivity (A5) was placed in a community by itself (see Table S3). Given the near perfect agreement between the BPM domains and the communities

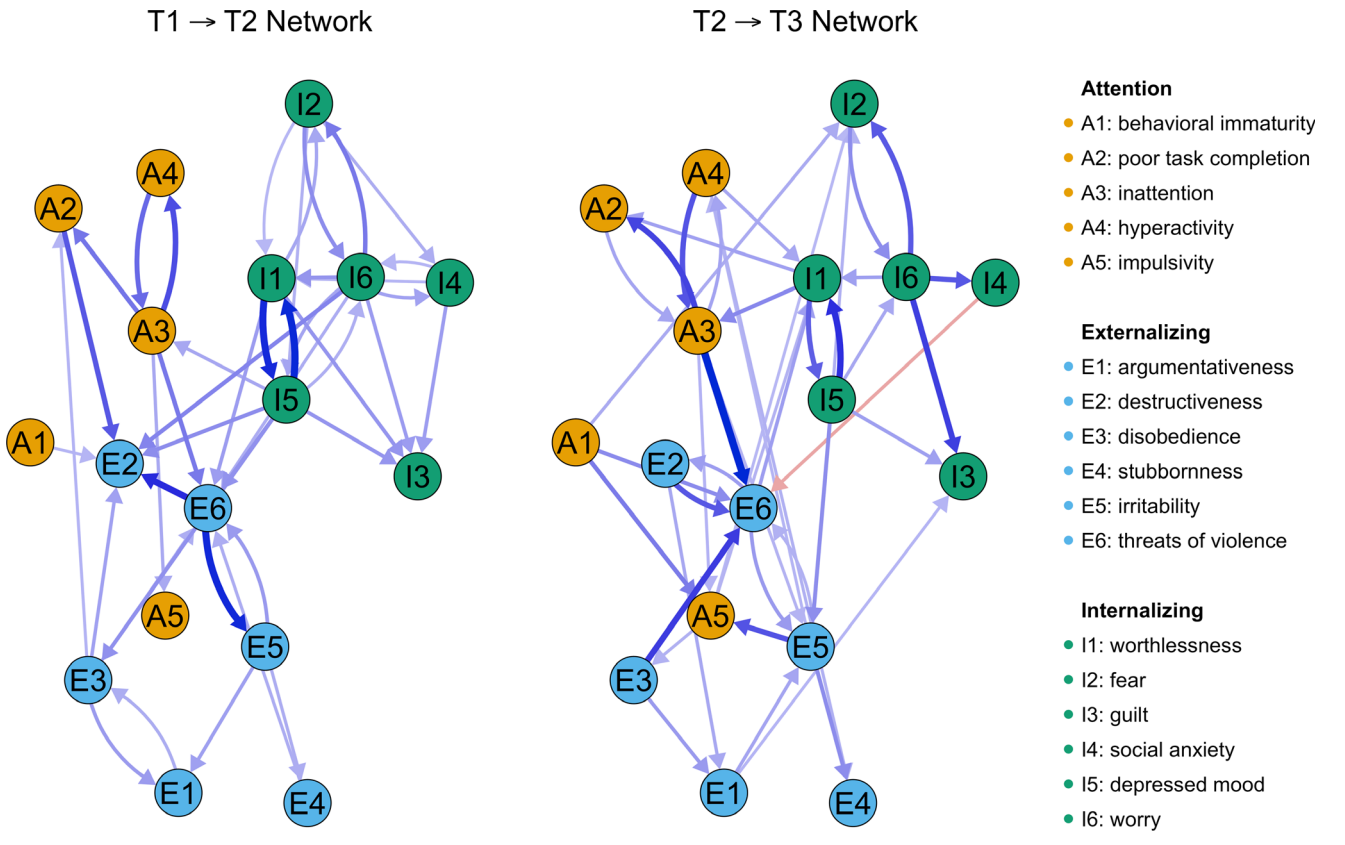

Figure 1 The cross-lagged panel networks for T1 → T2 (left) and T2 → T3 (right). Arrows represent unique longitudinal relationships. Blue edges indicate positive relationships (i.e., odds ratios greater than 1), and red edges indicate negative relationships (i.e., odds ratios less than 1). Edge thickness represents the strength of the odds ratio such that thicker edges represent stronger relations. Autoregressive edges, weaker edges (i.e., odds ratios within 1 ± .35), and covariates were excluded from the plot to ease visual interpretation. [Colour figure can be viewed at wileyonlinelibrary.com]

identified by the spinglass algorithm, we used the BPM domains to group symptoms.[2] Symptoms generally clustered together in three regions reflecting the attention, externalizing, and internalizing domains, and the strongest cross-lagged edges tended to be between symptoms within the same domain. However, symptoms were highly interconnected (see Figure S11).

The three strongest cross-lagged edges were depressed mood (I5) → worthlessness (I1; OR = 2.20), its reciprocal (i.e., worthlessness → depressed mood; OR = 2.16), and threats of violence (E6) → irritability (E5; OR = 2.17), and these edges were significantly stronger than 92.4%, 91.1%, and 89.7% of other edges, respectively (see Figure S9). The strongest bridging edges (i.e., edges between symptoms of two different domains) were poor task completion (A2) → destructiveness (E2; OR = 1.82) and inattention (A3) → threats of violence (E6; OR = 1.66), which were the sixth and tenth strongest edges in the network and significantly differed from 50.4% and 14.7% of the other edges, respectively. The comorbidity of internalizing and externalizing symptoms has been a topic of interest in developmental psychopathology (e.g., Shankman et al., 2009), and the six strongest edges bridging these domains were from internalizing symptoms (specifically, worry or depressed mood) to externalizing symptoms (destructiveness, threats of violence, or disobedience).

Centrality estimates are plotted in Figure 2. Depressed mood (I5) had the highest out-EI and had significantly greater out-EI than 11 of the 16 other symptoms in the network (see Figure S9). Inattention (A3) and worry (I6) also had high out-EI, and both symptoms had significantly higher out-EI than 9 other symptoms. Destructiveness (E2) and guilt (I3) had the lowest out-EI values, and their out-EI was significantly lower than five and seven other symptoms, respectively. In-EI estimates revealed that threats of violence (E6) and destructiveness (E2) had the highest in-EI, meaning they were strongly prospectively predicted by other symptoms. These symptoms had significantly greater in-EI than 8 of the 16 other symptoms. The symptom with the lowest in-EI was stubbornness (E4). Its in-EI was significantly lower than that of 13 other symptoms, suggesting it was minimally predicted by other symptoms.

Network replicability

Network replicability was examined by comparing the T1 → T2 network to a T2 → T3 network. As expected due to the smaller size of the T2 → T3 subsample, there were fewer cross-lagged edges in

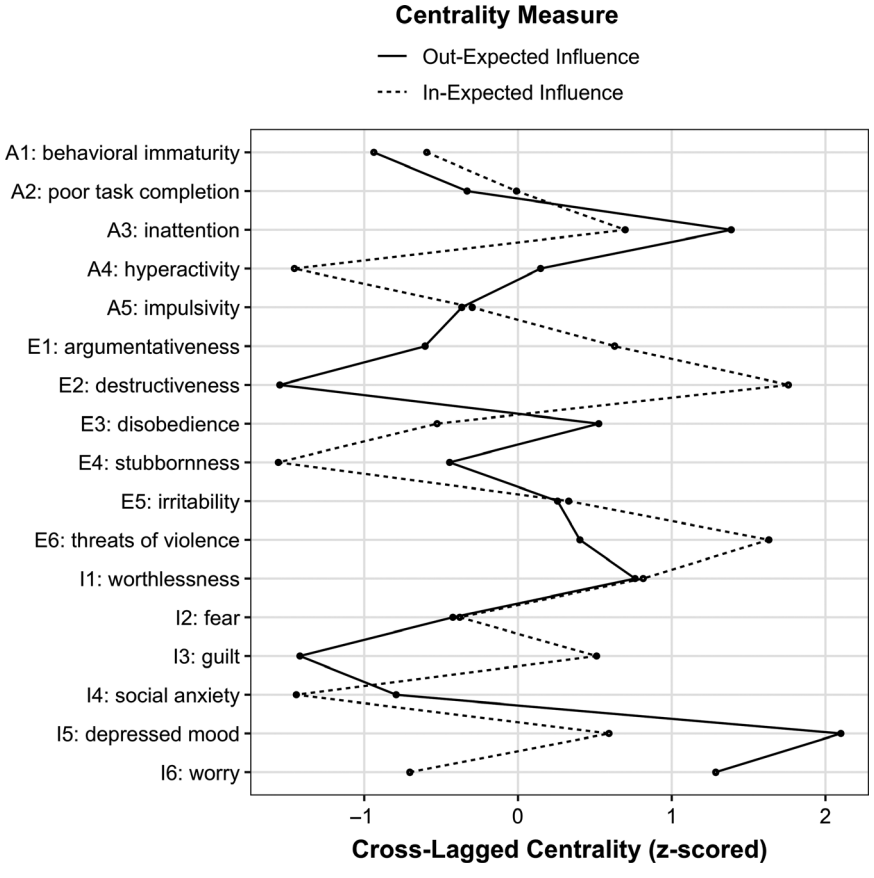

Figure 2 Symptom centrality estimates in the T1 → T2 network. Larger values reflect greater centrality.

the T2 → T3 network (167) than the T1 → T2 network (225). The communities detected in the T2 → T3 network largely mapped onto those of the T1 → T2 network, with the exceptions that impulsivity and argumentativeness were placed in the externalizing community, and destructiveness and threats of violence were placed in the attention community (see Table S3). Edge lists were moderately correlated between networks (r = .61). One hundred fifty-five individual edges replicated across networks (i.e., ORs > 1 or ORs < 1 in both networks), which represents 84.2% of the edges in the T2 → T3 network and 64.0% of the edges in the T1 → T2 network. The strongest edges in the T1 → T2 network (depressed mood → worthlessness, worthlessness → depressed mood, and threats of violence → irritability) were the second, eleventh, and eighteenth strongest edges in the T2 → T3 network, respectively. The stability of the rank-order of out-EI, in-EI, and bridge EI was below recommended cutoffs in the T2 → T3 network (CS coefficients = .13, .21, and 0, respectively; see Figure S7; Epskamp et al., 2018), precluding the interpretation of centrality rank-order in this network and cross-network centrality comparisons.

Discussion

This study used a network approach to model unique longitudinal relationships between individual internalizing, externalizing, and attention symptoms in youth. The use of network modeling of longitudinal data allowed for the estimation of directional and unique relationships and establishment of temporal precedence. Symptoms were highly interconnected in the resulting network, but there was substantial heterogeneity in individual symptoms' centrality, even when comparing symptoms from the same domain. This heterogeneity highlights the importance of examining the relationships between individual symptoms rather than composite sum-scores or diagnoses (Fried, 2015). Modeling these temporal relationships offers an opportunity to improve understanding of the etiology of psychopathology in youth, which in turn could highlight potential targets for early intervention.

A community detection algorithm identified three communities of symptoms that were nearly identical to the BPM domains, thus validating the factor structure of the BPM. As one might expect given this result, the BPM's factor structure, and findings from cross-sectional network studies in youth (e.g., Boschloo et al., 2016), symptoms from the same domain tended to cluster together graphically. Similarly, cross-lagged edges tended to be stronger between symptoms from the same domain as compared to different domains. Despite this tendency, several interesting relationships between individual symptoms emerged. For example, the relationship between depressed mood and worthlessness, and

its reciprocal, were among the strongest edges in the network. This strong bidirectional relationship (which, importantly, remained after statistically adjusting for all other symptoms and replicated in the T2 → T3 network) is consistent with the hopelessness theory of depression, which posits that low self-worth – when perceived as global and stable – causes hopelessness, which then causes other symptoms including depressed mood and decreased motivation (Abramson, Metalsky, & Alloy, 1989). The hopelessness theory also provides an explanation for the reciprocal path, as depressed mood may in turn increase negative cognitions such as thoughts of worthlessness. Further work incorporating elements of negative cognitive style in psychopathology networks may help to elucidate these relationships and identify additional connections between depressive symptoms (especially depressed mood) and components of negative cognitive style (Bernstein et al., 2019).

This study extends prior cross-sectional work by parsing the extent to which symptom centrality was driven by edges *to* other symptoms versus *from* other symptoms. Depressed mood had the highest out-EI in the present study, meaning that it most strongly predicted other symptoms six months later after adjusting for all other symptoms at T1 and demographic and socioeconomic status covariates. The high out-EI of depressed mood is consistent with findings from cross-sectional network analyses in both youth and adults (Funkhouser et al., 2020; McElroy et al., 2018). However, temporal network models have yielded mixed results regarding the centrality of depressed mood. A CLPN analysis of depressive symptoms and components of negative cognitive style reported that depressed mood had high out-centrality (Bernstein et al., 2019), whereas a depression and anxiety symptom network estimated from intensive longitudinal data collected from treatment-seeking adults indicated that depressed mood had low out-centrality (Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017). Different time lags may explain these discrepancies. Inattention had the second highest out-EI in this study. This finding is consistent with prior studies in which inattention was highly central in cross-sectional networks of ADHD symptoms (Martel, Levinson, Langer, & Nigg, 2016) and transdiagnostic emotional and behavioral symptoms in youth (Boschloo et al., 2016; Rouquette et al., 2018). Worry had the third highest out-EI. Although worry or generalized anxiety disorder (which is characterized by excessive worry) were among the most central nodes in cross-sectional networks of internalizing symptoms (Beard et al., 2016) or internalizing and externalizing disorders (McElroy, Shevlin, et al., 2018), Fisher et al. (2017) reported that worry was among the least central symptoms in a network estimated from intensive longitudinal data. It remains unclear whether this inconsistency in the centrality rank-order of worry is due to differences in sample characteristics, methodological differences (e.g., different time lags), differences in network estimation, sampling variability, or some other factor.

The highest in-EI estimates (i.e., the sum of longitudinal associations into a particular symptom) were for threats of violence and destructiveness, both of which are in the externalizing domain and symptoms of conduct disorder. Interestingly, the two strongest bridging edges in the network were from attention symptoms (inattention and poor task completion) to these two symptoms. Furthermore, the strongest edges bridging the internalizing and externalizing domains were from depressed mood or worry to these two externalizing symptoms or disobedience. It is possible that these bridging edges underlie externalizing symptoms' comorbidity with attention and internalizing symptoms.

Although longitudinal network modeling can reveal the direction of potentially causal relationships, there are several unresolved methodological issues related to network modeling of longitudinal data. One important issue in longitudinal studies of psychopathology more generally is that it is unclear what time lags are appropriate or optimal to capture relationships between symptoms. The present study used a six-month time lag, and it is possible that relationships between symptoms occur at shorter or longer time periods. Parameter estimates can vary as a function of the time interval between measurements (Gollob & Reichardt, 1987), and differences in time lag may explain why some of the results from the present study differed from those of previous intensive longitudinal network analyses. The present study provides insight into predictive and potentially causal relations between symptoms across a six-month lag, but results may have been different if a different time lag had been used. Researchers should therefore consider the expected time lag when deciding sampling frequency. For example, if causal effects occur over the course of minutes or hours (e.g., impulsivity → destructiveness, perhaps), more frequent sampling would be required. The possibility that the time lag of causal effects may differ across pairs of symptoms and/or individuals should also be considered. Although not yet implemented in the context of CLPN model estimation, continuous time modeling, which generates effect estimates for any arbitrarily selected time interval based on the assumption that causal effects accumulate continuously (Voelkle, Oud, Davidov, & Schmidt, 2012), offers one option to address the issue of time-interval dependency. Approaches that aim to identify and estimate optimal time lags (e.g., the lag at which X maximally predicts Y) such as differential time-varying effect modeling offer another potential solution (Jacobson, Chow, & Newman, 2019).

It is also important to note that CLPN models conflate within- and between-subject effects (Rhemtulla et al., 2019), which can result in biased

parameter estimates when there are stable individual differences in the variables being modeled. One way to disentangle between- and within-subjects effects is through the random-intercept cross-lagged panel model (RI-CLPM), which fits a latent factor to the repeated measurements of a variable to remove time-invariant individual differences (Hamaker, Kuiper, & Grasman, 2015). Alternative approaches have also been suggested (e.g., fitting a CLPN after mean-centering each participant's data on each variable; Rhemtulla et al., 2019), but further methodological work is needed to assess the ability of these methods to separate within- and between-subjects effects.

The directional edges modeled in the present study elucidate the direction of causality if edges reflect causal relationships (as hypothesized by the network theory). However, there are other possible interpretations of these directional relationships. For example, edges may reflect spurious relationships due to an unmeasured common cause (e.g., a latent variable) that influences both the predictor and the outcome. Although controlling for autoregressive effects alleviates this possibility to some degree, this possibility is important to consider because different theoretical explanations of symptom etiology translate to different implications for intervention and prevention. If a network model is the true data-generating mechanism and symptom X causes and maintains symptom Y, an intervention that reduces X will cause a decrease in Y (assuming certain assumptions such as acyclicity are met). In contrast, if a common cause model reflects the true data-generating mechanism, an intervention targeting X will have no impact on Y and the intervention must instead target the common cause, which will lead to reductions in both Y and X. Network models and latent variable models are statistically equivalent (i.e., every network model can be represented as an equivalent latent variable model, and vice versa; Kruis & Maris, 2016), meaning that comparison of model fit is insufficient to identify the true model. More rigorous research is needed to determine the relative validity of each theory – for example, experimental manipulation of symptoms will be particularly helpful for identifying causal relations.

Taken together, these findings suggest that symptom-level longitudinal studies can provide insight into risk factors for specific symptoms and potential etiological pathways of psychopathology in youth. These pathways may also explain the high rates of comorbidity between disorders or higher-order latent factors of psychopathology. For example, our findings suggest that previously observed relationships from ADHD to conduct disorder (e.g., Loeber, Green, Keenan, & Lahey, 1995) may be specific to (or stronger for) certain ADHD symptoms (e.g., inattention, task completion difficulty) and externalizing behaviors (e.g., threats of violence, destructiveness). Psychopathology in youth is strongly associated with risk for psychopathology in adolescence and adulthood (Costello et al., 2003; Klein, Shankman, Lewinsohn, & Seeley, 2009), and further research examining whether the observed relationships are causal as well as identifying underlying mediators will help to highlight more specific (and possibly novel) targets for preventative interventions.

Strengths and limitations

Several strengths of this study are worth noting. First, we used a state-of-the-art modeling technique (CLPN modeling) to model autoregressive and cross-lagged effects. The utilization and modeling of longitudinal data allowed for the estimation of a directed network and the identification of temporal effects between symptoms. Many extant psychopathology network analyses have focused on cross-sectional data, and we join others in recommending that increased attention be devoted to longitudinal network modeling (Guloksuz, Pries, & van Os, 2017). Second, the use of a large, population-based sample allowed for sufficient power to detect relationships between symptoms and avoided issues related to Berkson's Bias, a form of selection bias that occurs when relationships in a subpopulation (e.g., a clinical sample for which a clinical severity cutoff was an inclusion criterion) differ from those in the general population. Berkson's Bias can pose a problem for the generalizability of psychopathology networks (Hoffman et al., 2019; de Ron, Fried, & Epskamp, in press), and the rigorous design and recruitment strategy of the ABCD study combined with its large sample size avoided this issue. Third, many network analyses have exclusively focused on symptoms of one or more DSM disorder(s). Reliance on the DSM perspective of psychopathology limits the ability of network analyses to cumulatively produce a framework that provides a better understanding of psychopathology than existing classification systems (Guloksuz et al., 2017). Examining heterogeneous sets of symptoms (such as those in the BPM) that cut across traditional diagnostic categories is much more likely to improve understanding of symptom etiology. Lastly, although unstable centrality rank-order in the T2 → T3 network precluded the evaluation of centrality replicability, we were able to examine the replicability of symptom communities and network edges.

Despite these strengths, the findings of this study should be interpreted in light of several limitations in addition to the considerations discussed above. First, the youth-report BPM was not normed for nine- or ten-year-old children and the average ages at T1, T2, and T3 were 10.45, 10.97, and 11.45, respectively. Although the manual for the BPM states that 'many children younger than 11 may be able to complete the BPM' (Achenbach, McConaughy, Ivanova, & Rescorla, 2011, p. 2), this may have influenced results, especially for the T1 → T2 network. Second, we used a self-report measure of youths'

symptoms in our primary analyses because symptom data were not collected from parents or teachers at the six- or eighteen-month follow-ups of the ABCD study. Sensitivity analyses in which youth-reported symptoms at T2 were replaced with parents' report of youths' symptoms generally suggested that results were moderately sensitive to who assessed the youth's symptoms at T2 (see Appendix S1 and Figures S1–S5). Cross-sectional correlations of individual symptoms across raters at T2 were weak to moderate, which may explain differences between networks estimated from T2 symptom data from different informants (youth versus parent). Future studies may benefit from incorporating ratings from multiple informants at multiple time points. Third, although we included demographic variables and indicators of socioeconomic status as covariates in the CLPN model to mitigate confounding from these sources, we did not consider so-called 'external field' factors (e.g., stressful life events) that might cause symptoms or moderate their temporal interrelationships. The possibility that important covariates may be missing from the model remains an unresolved issue in network modeling and other multivariate statistical methods (e.g., multiple regression), and future research should extend recent studies examining the role of variables such as environmental risk factors and/or genetic risk in adults (Hasmi et al., 2017) to youth and adolescent samples. Fourth, nodes were assessed using individual items from factor analytically derived scales, and internal consistencies of each domain (after combining tautologically overlapping items; see Footnote 1) were moderate to high (Kuder–Richardson Formula 20 [KR-20] coefficients = .61–.77). Although using moderately to highly correlated items from factor analytically derived scales as nodes in a psychopathology network is common practice, this approach is not ideal for studying conditionally independent relationships and individual items are less reliable than aggregates of multiple items (Cicchetti & Prusoff, 1983). We therefore recommend that network analyses assess each node using multiple items whenever possible.

Conclusion

This study's application of network modeling to panel data from a large, population-based cohort of youth identified unique longitudinal relationships between symptoms within and across symptom domains. We found strong reciprocal effects between depressed mood and worthlessness. Disruptive behaviors (i.e., threats of violence, destructiveness) were most predicted by other symptoms six months earlier, and depressed mood, inattention, and worry were most predictive of other symptoms six months later. Network parameters (e.g., individual edges, most central symptoms) were moderately replicable in the T2 → T3 replication network. Although causal inferences are precluded by this study's observational design and methodological considerations, further research should elucidate the nature and underlying mechanisms of the relationships from depressed mood, inattention, and worry to other symptoms and evaluate the viability of intervening on these specific symptoms.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Sensitivity analyses examining the impact of T2 symptom rater.
Table S1. Adjacency matrix of the T1 → T2 cross-lagged panel network.
Table S2. Adjacency matrix of the T2 → T3 cross-lagged panel network.
Table S3. Communities detected in each network.
Figure S1. Confidence intervals around edge weights in networks using parent-reported symptoms at T2.
Figure S2. Centrality stability in networks using parent-reported symptoms at T2.
Figure S3. Network plots of models using parent-reported symptoms at T2.
Figure S4. Edge weight difference tests in networks using parent-reported symptoms at T2.
Figure S5. Centrality difference tests in networks using parent-reported symptoms at T2.
Figure S6. Confidence intervals around edge weights.
Figure S7. Centrality stability.
Figure S8. Edge weight difference tests.
Figure S9. Centrality difference tests.
Figure S10. Autoregressive edges.
Figure S11. The cross-lagged panel networks (including all cross-lagged edges).

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Key points

- Individual symptoms may causally interact in a network, but studies examining these relationships in youth have primarily been cross-sectional, precluding directional inferences.
- Using cross-lagged panel network modeling, we found that depressed mood, inattention, and worry were the strongest unique, prospective predictors of other individual symptoms.
- The reciprocal associations between depressed mood and worthlessness were among the strongest bivariate relationships in the network.
- Network parameters (e.g., individual edges) were generally moderately replicable.
- This study identified depressed mood, inattention, and worry as central prospective predictors of other symptoms, and further work examining whether these relationships are causal will elucidate whether these specific symptoms may be good targets for prevention and/or treatment.

Notes

1. Items 4 ("I have trouble concentrating or paying attention") and 14 ("I am inattentive or easily distracted") were collapsed, and the collapsed symptom was labeled inattention (A3). Items 7 ("I disobey my parents") and 8 ("I disobey at school") were collapsed and labeled disobedience (E3).
2. Given the poor stability of bridge EI when BPM domains were used to categorize symptoms, we also tested the stability of bridge EI in networks in which symptoms were categorized according to the communities identified by the spinglass algorithm. Grouping symptoms using the spinglass algorithm did not affect bridge EI stability in any of the networks (all CS coefficients = 0).

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