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Unique and predictive relationships between components of cognitive vulnerability and symptoms of depression

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Data Availability

Data will be made available in accordance with the NIMH data sharing agreement.

Abstract

BACKGROUND: Cognitive vulnerability theories of depression outline multiple, distinct inferential biases constitutive of cognitive vulnerability to depression. These include attributing negative events to internal, stable, and global factors, assuming that negative events will lead to further negative consequences, and inferring that negative events reflect negative characteristics about the self. Extant research has insufficiently examined these biases as distinct, limiting our understanding of how the individual cognitive vulnerability components interrelate and confer risk for depression symptoms. Thus, we conducted exploratory network analyses to examine the relationships among the five components of negative cognitive style and explore how components may differentially relate to depressive symptoms in adolescents. METHODS: Participants completed measures of negative cognitive style twice over a two-year period. We estimated Graphical Gaussian Models using contemporaneous data and computed a cross-lagged panel network using temporal data from baseline and 2-year follow-up. RESULTS: Results reveal interesting structural dynamics among facets of negative cognitive style and depressive symptoms. For example, results point to biases towards stable and future-oriented inferences as highly influential among negative cognitive style components. The temporal model revealed the internal attributions component to be heavily influenced by depressive symptoms among adolescents, whereas stable and global attributions most influenced future symptoms. CONCLUSIONS: This study presents novel approaches for investigating cognitive style and depression. From this perspective, perhaps more precise predictions can be made about how cognitive risk factors will lead to the development or worsening of psychopathology.

Keywords: cognitive vulnerability, cognitive style, hopelessness theory, depression, network analysis

Depression is a serious personal and public health concern in the United States. Nearly one-fifth of Americans experience major depression in their lifetime and many more suffer from subthreshold symptoms (Hasin et al., 2018). For many, depressive episodes are chronic and recurrent and rates of depression are increasing in younger cohorts (Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Ritschel et al., 2013). Understanding its etiology remains a clinical research priority.

Over the past four decades, there has been enthusiasm and empirical support for cognitive vulnerability theories of depression risk, including the reformulated learned helplessness theory of depression (Abramson, Seligman, & Teasdale, 1978) and its revision, the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989). These theories posit that individuals at the greatest risk for depression are characterized by certain ways of explaining the causes and consequences of negative events. Specifically, these individuals typically attribute the causes of negative events to internal (vs. external), stable (vs. temporary), and global (vs. specific) factors; assume that negative events will lead to further adverse consequences; and infer that negative events reflect negative characteristics about the self. This pattern of making internal, stable, and global attributions and assuming negative future consequences and negative inferences about the self is broadly referred to as negative cognitive style. Having an overall negative cognitive style prospectively predicts the first onset and recurrence of major depression (Alloy et al., 2006; Mac Giollabhui et al., 2018).

The five components of negative cognitive style—internality, stability, globality, assuming negative consequences, and inferring negative personal characteristics/low self-worth— are partly distinguishable. For example, they are only moderately intercorrelated, particularly in children and adolescents (Abela, Parkinson, Stolow, & Starrs, 2009; Calvete,

Villardón, & Estévez, 2008; Gibb & Abela, 2008), and items assessing each component are more strongly related to each other than to items assessing other components (Hewitt, Foxcroft, & MacDonald, 2004). Among adolescents, although the different components of cognitive style are correlated significantly, they remain distinct, as multifactor models provide better fits than single latent factor models (Hankin & Abramson, 2002).

Yet, in practice, there is a disconnect between theory and measurement. Although hopelessness theory proposes distinct cognitive style components (e.g., internal vs. external, stable vs. temporary), tests of the theory frequently operationalize cognitive style as a single composite score across components (for review, see Liu, Kleiman, Nestor, & Cheek, 2015). Other approaches, such as the *weakest link* (Abela & Sarin, 2002), operationalize cognitive style as an individual's highest component score (i.e., their "weakest link"). That is, it does not matter from which component the high score comes; a person with a score of 7 on the globality dimension is deemed at equal risk as a person with a score of 1 for globality, but 7 for internality, and a person with a score of 7 on both globality and internality. These approaches do not treat the components as meaningfully distinct and preclude examination of how components interact. Accordingly, the goal of this study is to ascertain how the components of negative cognitive style differentially interrelate and heighten depression risk.

Some studies have examined individual components, testing the relationship between a single component and a depression outcome measure, with and without adjusting for other components, in children (e.g., Abela, 2001; Abela & Sarin, 2002), adolescents (e.g., Abela, 2001; Abela & Seligman, 2000; Calvete, Villardón, & Estévez, 2008), and adults (e.g., Abela, Aydin, & Auerbach, 2006; Stone, Gibb, & Coles, 2010). As findings regarding whether individual components uniquely contribute to vulnerability are mixed, we use novel methods to explore the

empirical distinguishability of these components and their clinical significance. We build on prior work by considering cognitive style components simultaneously, as systems of inferential thoughts that can interact with each other and influence clinical outcomes uniquely and together. We explored these topics in a large sample of adolescents. Early symptoms or indicators of depression tend to emerge and noticeably rise during adolescence (Alloy et al., 2012; Hankin et al., 1998; Twenge & Nolen-Hoeksema, 2002). Furthermore, the mean age of depression onset is decreasing in younger cohorts towards late adolescence (Zisook et al., 2007). Thus, improved methods of identifying risk and delivering targeted, early interventions during this vulnerable period could have substantial public health impact.

The present study

What are the relationships between the components of negative cognitive style? It seems likely that developing a high propensity for one component of negative cognitive style could increase one's likelihood of exhibiting another and that certain components could contribute more than others to overall cognitive vulnerability. Advances in network analysis techniques allow us to simultaneously model relationships between all cognitive style components. This approach can generate hypotheses regarding which components are most strongly related to each other. Given that relationships between components have not been examined in this manner, we had no specific a priori hypotheses about these relationships. However, stability, globality, and negative consequences may be more strongly interrelated as they involve generalizing the occurrence of a negative event over time and space, and internality and self dimensions may be more strongly interrelated as they both involve self-blame.

How do the components of negative cognitive style relate to the symptoms of depression? Incorporating symptoms into network models can identify which components may

afford the greatest overall depression risk as well as specific symptom pathways that could mediate this effect. Because network analyses can simultaneously examine multiple relationships among correlated variables, it is ideally suited to address these questions. Given that overall negative cognitive style is not just contemporaneously associated with depression, but also prospectively predicts it (Alloy et al., 2006; Gibb & Alloy, 2006; Mac Giollabhui et al., 2018), we examined contemporaneous and temporal connections between networks of negative cognitive style components and depressive symptoms.

Method

Participants

Participants were adolescents recruited as part of the Adolescent Cognition and Emotion (ACE) project at Temple University for an 8-year longitudinal study on the onset and course of depression (see Alloy et al., 2012 for additional information). The average age of the sample at baseline was 12.57 years (*SD*=.90). The sample was approximately 53% female, 47% Caucasian, and 53% African American.

Procedures

Adolescents completed repeated measures of cognitive vulnerability and depressive symptoms. Six-hundred eight adolescents with complete data were included in baseline (Time 1; T1) analyses. Time 2 (T2) for the current study is approximately 2 years later (mean=2.14, *SD*=.24, range=1.78–2.78). Adolescents who provided complete data at this timepoint were included in T2 analyses (*N*=222). As our network analysis methods (described below) do not allow for missing data, this drop in sample size is not unexpected. Data were drawn from the larger, long-term ACE project, which followed adolescents and caregivers in socioeconomically diverse areas of Philadelphia. With long follow-up periods, participants' missing data from one

or more appointments is common and indeed more than 70% of participants who completed the baseline assessment completed at least one other follow-up assessment. However, because the difference in sample sizes between T1 and T2 is notable, we examined demographic and clinical differences between participants who did and did not complete the T2 assessment in full. There were no significant differences in scores on any of the T1 symptom or cognitive style measures. Compared to adolescents who only were included at T1, those who were included in T1 and T2 were significantly younger at baseline ($p<.05$) and more likely to be African American ($p<.01$).

Measures

Adolescent Cognitive Style Questionnaire—Modified (ACSQ-M; Alloy et al., 2012; Hankin & Abramson, 2002). The ACSQ-M is a modified version of the Adolescent Cognitive Style Questionnaire (Hankin & Abramson, 2002), including appearance-related as well as achievement and interpersonal domains. It is designed to assess the components of negative cognitive style in adolescents. Participants read 12 vignettes about negative events (e.g., "You take a test and get a bad grade.") and are asked to imagine a potential cause for each event. Participants rate the cause of an event in terms of (1) internality (caused by the person vs. environment), (2) stability (to what extent the cause of the problem is temporary vs. persistent), and (3) globality (to what extent the cause of the problem will affect one area vs. many areas of one's life). In addition, the participant also rates: (4) how likely the event is to lead to other negative future consequences, and (5) to what extent the event occurring implies negative characteristics about the self. Scores for each component of negative cognitive style, reflected in each of the five repeated questions, are constructed by taking the average across all 12 vignettes, which use a 1–7 scale. Higher scores indicate a more negative bias (e.g., greater tendency to interpret events as being caused by the person, rather than the environment). Internal consistency

was acceptable across all five component scales at T1: internality (α=.77), stability (α=.83), globality (α=.83), negative future consequences (α=.86), and negative self-worth implications (α=.88).

Children's Depression Inventory (CDI; Kovacs, 1985). The CDI assesses 27 affective, behavioral, and cognitive symptoms of depression. Respondents rate items on a 0 to 2 scale and total scores range from 0 to 54. To simplify the models, increase interpretability, and maximize power, we combined highly similar items according to the designated 5 component scales (Kovacs, 1992): Negative/Depressed Mood (α=.82), Ineffectiveness (α=.69), Anhedonia (α=.78), Negative Self-Esteem (α=.81), and Interpersonal Problems (α=.75).

Analyses

Contemporaneous Network of Cognitive Style Components. To explore the relations among the five components of cognitive style, we estimated a Graphical Gaussian Model (GGM) with baseline data (T1), using the R package *qgraph* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). We included as nodes the five ACSQ variables (internal, stable, global, consequences, self-worth). The edges (i.e., connections between nodes) signify associations between items, controlling for the effects of other items in the network. A graphical LASSO (Least Absolute Shrinkage and Selection Operator) was used to regularize the GGM; this approach increases the sparsity of a graph and reduces the likelihood of false positives being included as edges (Friedman, Hastie, & Tibshirani, 2011). Additionally, within the *qgraph* package, an extended Bayesian Information Criterion (EBIC) model selection procedure (Foygel & Drton, 2011) identified the best fitting network (γ=.5). In the resulting undirected network, nodes represent cognitive style variables and edges between these variables reflect pairwise regularized partial correlations.

To assess the stability and accuracy of edge weights, we calculated 95% confidence intervals (CIs) for each network via non-parametric bootstrapping (1,000 bootstraps) in the R package *bootnet* (Epskamp et al., 2018). The resulting sampling distributions are plotted in descending order of observed edge weights. This approach does not test whether each edge is significantly different from zero; this is accomplished through the initial graphical LASSO. Instead, we can be most confident in edges whose CIs do not include zero. We also conducted non-parametric bootstrapped difference tests for edges (Epskamp et al., 2018) that identify significant differences among the edge weights.

We estimated centrality metrics for the variables included in the graphical LASSO network. These metrics quantify the importance of each node within the network. We computed one- and two-step expected influence indices for each node as they handle positive and negative edges well (Robinaugh, Millner, & McNally, 2016). *One-step expected influence (EI1)* is the sum of edge weights directly attached to a node. *Two-step expected influence (EI2)* includes pathways that pass through a neighboring node as well and estimates direct and indirect influence. This network reflects cross-sectional data and is undirected, meaning that the direction of influence cannot be determined.

All T1 analyses were conducted on the full sample (N=608). However, the pattern of results did not change when conducted for the reduced sample (i.e., participants with complete T1 and T2 data).

Contemporaneous Network of Cognitive Style and Depression Items. A second graphical LASSO network included baseline depression symptoms. Because the aim of this analysis was to clarify the relationships between ACSQ items and depressive symptoms, we focus on *bridge* expected influence (*EI*). *Bridge EI* isolates the relationship between a given node

(e.g., globality) and nodes from outside communities (e.g., depression symptoms). Resulting centrality estimates from the *bridge* function of the R package *networktools* (Jones, 2018) include *one- and two-step bridge EI* (*bridge EI1*; bridge *EI2*). *Bridge EI1* comprises edge weights between a certain node and nodes in the other community. *Bridge EI2* further includes the secondary influence of this node via indirect connections. Normalized (z-scored) *bridge EI* values are presented in plots. Nodes with high *bridge EI* may be most likely to activate a nearby community or be activated by nodes from a nearby community, and consequently, spread activation within its own community (Heeren, Jones, & McNally, 2018; Jones, Mair, Riemann, Mugno, & McNally, 2017).

Temporal Networks. Finally, we examined ACSQ and CDI (depression) data collected at baseline (T1) and at follow-up (T2). To explore prospective associations between cognitive style components and depression symptoms, we computed a cross-lagged panel network, an approach developed by Rhemtulla, van Bork, and Cramer (2018). This approach examines the effects of individual items at T1 on all other items at T2, controlling for auto-regressive effects (i.e., regressing each variable at T2 on itself at T1). Both within-timepoint (undirected) and between-timepoint (directed) associations are considered.

As outlined by Rhemtulla et al. (2018), we first computed within- and between-timepoint unstandardized coefficients and auto-regressive coefficients with regularized regressions. Penalized maximum likelihood with a LASSO penalty is applied to estimate a sparse network structure, reducing the number of false positives. The *glmnet* package (Friedman, Hastie, & Tibshirani, 2010) was used to calculate regressions and the *qgraph* package (Epskamp et al., 2012) was used to plot all graphs. We then quantify the overall effects of each node. As cross-lagged (i.e., from T1 to T2) edges are directed, we can estimate summary statistics for both *in-*

prediction and *out-prediction*. *In-prediction* refers to the proportion of variance for a given node at T2 that is accounted for by variables at T1. *Out-prediction* refers to the effect a given node at T1 has on variables at T2. Both for in-prediction and out-prediction, three statistics are calculated: *overall*, which includes all variables, *cross-lagged*, which excludes the auto-regressive path of the node of interest, and *cross-construct*, which excludes paths connecting nodes within the same community.

Results

See Table 1 for ACSQ and CDI items and their node labels used in all figures.

Contemporaneous Network of Cognitive Style Components

We first examined the relationships among cognitive style components alone. Figure 1 presents the graphical LASSO network, which visualizes regularized partial correlations among the five ACSQ nodes (T1). The strongest connection was between ACSQ2-*stable* and ACSQ4-*consequences*, r=.44. Additional strong connections were ACSQ4-*consequences*—ACSQ5-*self-worth*, r=.40, and ACSQ2-*stable* —ACSQ3-*global*, r=.30. Stability analyses reveal that most edges were stable and that the largest partial correlations (i.e., strongest edges) were significantly greater than most others (see Supplemental Figure S1). For example, the ACSQ2-*stable*—ACSQ4-*consequences* association is statistically significantly greater than all other edges, with the exception of ACSQ4-*consequences*—ACSQ5-*self-worth*. Additionally, the ACSQ4-*consequences*—ACSQ5-*self-worth* association is significantly larger than all smaller observed associations, with the exception of ACSQ2-*stable* —ACSQ3-*global*. These stability analyses increase confidence in the interpretability of results.

Finally, centrality metrics appear in Figure 1. ACSQ2-*stable* (*EI1*=.89, *EI2*=1.7), and ACSQ4-*consequences* (*EI1*=1.08, *EI2*=1.97) were most influential. ACSQ1-*internal* had the lowest expected influence (*EI1*=.50, *EI2*=.90).

Contemporaneous Network of Cognitive Style and Depression Items

Figure 2 shows the relationships between components of cognitive style and depression symptoms. The strongest edges between communities include ACSQ5-*self-worth*—CDI4-*self-esteem*, *r*=.12, ACSQ5-*self-worth*—CDI3-*anhedonia*, *r*=.07, ACSQ1-*internal*—CDI1-*depressed mood*, *r*=.07, ACSQ4-*consequences*—CDI1-*depressed mood*, *r*=.05, and ACSQ4-*consequences*—CDI5-*interpersonal problems*, *r*=.05. ACSQ items with the greatest bridge expected influence were ACSQ5-*self-worth* (*bridge EI1*=.21, *bridge EI2*=.40) and ACSQ4-*consequences* (*bridge EI1*=.10, *bridge EI2*=.27). From the depression community, CDI1-*depressed mood* (*bridge EI1*=.16, *bridge EI2*=.34) and CDI4-*self-esteem* (*bridge EI1*=.13, *bridge EI2*=.27) had the greatest bridge expected influence. Stability analyses (Supplemental Figures S2-3) indicate strong stability for expected influence metrics and modest stability for edge weights. Consequently, the relative order of magnitude of observed edge weights surviving regularization, particularly for cross-community edges, should not be assumed to be replicable.

We reran both contemporaneous networks with T2 data and found similar patterns of results. The strongest edges and estimates of influence largely replicated across time points (See Figures S4 and S5 in Supplemental Materials).

Temporal Networks

Figure 3 shows the cross-lagged panel network. Edges represent cross-time effects and denote the direction of prediction with arrows. First, ACSQ2-*stable*, ACSQ4-*consequences*, ACSQ5-*self-worth*, CDI1-*depressed mood*, and CDI3-*anhedonia* have high auto-regression

coefficients. By contrast, ACSQ1-*internal* does not and appears to be heavily influenced by CDI3-*anhedonia*, B=.28, CDI2-*ineffectiveness*, B=.10, and CDI4-*self-esteem*, B=.10. Other strong connections include ACSQ2-*stable* predicting CDI4-*self-esteem*, B=.16, and ACSQ3-*global* predicting CDI1-*depressed mood*, B=.13, and CDI5-*interpersonal problems*, B=.10. Importantly, in this initial model, some of the strongest connections were between nodes from the same construct. For example, ACSQ5-*self-worth* strongly predicted ACSQ3-*global*, B=.24, and CDI1-*depressed mood* strongly predicted CDI2-*ineffectiveness*, B=.48, and CDI4-*self-esteem*, B=.34.

Figure 4 shows cross-lagged and cross-construct estimates of in-prediction and out-prediction. Full summary statistics appear in Supplemental Table S1. Cross-lagged estimates suggest that ACSQ1-*internal*, CDI2-*ineffectiveness*, and CDI4-*self-esteem* are not drivers in the network, given very high in-prediction values but very low out-prediction values. Instead, CDI1-*depressed mood*, CDI3-*anhedonia*, and to a lesser extent ACSQ5-*self-worth* appear to be the biggest influencers, with high out-prediction and low in-prediction estimates. Although informative, these estimates are limited in describing the effects of ACSQ components on depression symptoms because of the strong interconnections between CDI items. Hence, cross-construct estimates are especially informative; they suggest that ACSQ1-*internal* is heavily influenced by depression items (i.e., high in-prediction values). CDI1-*depressed mood* and CDI4-*self-esteem* were the depression items most influenced by cognitive style overall. Additionally, ACSQ2-*stable* and ACSQ3-*global* most influence depression nodes and CDI3-*anhedonia* most influences ACSQ nodes.

Discussion

The hopelessness theory outlines five components of cognitive vulnerability: internality, stability, globality, negative future consequences, and inferred negative personal characteristics/low self-worth. Although it distinguishes among these components, in practice, they often have not been measured as distinct. Network approaches offer novel ways to model cognitive style in a manner more consistent with the overarching hopelessness theory.

What are the relationships between the components of cognitive style?

These data reveal interesting structural dynamics among these facets of cognitive style. For example, centrality indices point to biases towards stable attributions and beliefs about negative future consequences as the most influential. Importantly, a person's most severe component (i.e., highest absolute score or "weakest link") is not necessarily the most important to target, as its severity could be a consequence of other, more influential factors. Hence, developing a component of cognitive style with high expected influence (e.g., stable) may be riskier than developing one with low expected influence (e.g., internal) as the former is more likely to activate other nodes than is the latter.

How do the components of negative cognitive style relate to the symptoms of depression?

Contemporaneous models of cognitive style and depression reveal how cognitive style components might uniquely impact emotional health. For instance, among adolescents, attributions related to the self were linked to low self-esteem and anhedonia and negative future-oriented interpretations to depressed mood. In many ways, these pairings are not surprising. For example, it makes sense that negatively interpreting situations in terms of one's self-worth or negative personal characteristics would lower self-esteem. The magnitude of associations between individual cognitive style and depression nodes were small, an observation that encourages caution in interpreting results. However, these connections survived controlling for

all other nodes in the network (i.e., partial correlations). Hopelessness theory already predicts that components (e.g., self dimensions) will be differentially affected by life events occurring in a closely related domain (e.g., affecting self-worth) (Abramson et al., 1989; Hankin & Abramson, 2001). Results suggest that components also could differentially affect specific depressive symptoms.

Expected influence indices suggest additional, testable hypotheses. Prognostically, the presence of a particular cognitive style component could signal risk for specific depressive symptoms or clusters. This could help clarify the etiology of depression or depressive sub-types (e.g., hopelessness depression). Additionally, presence of certain symptoms may imply that a certain cognitive style component is driving psychopathology and that targeting this upstream factor might alleviate symptoms and prevent their recurrence. This line of research could encourage the development or use of targeted treatment strategies. For instance, interventions designed to decrease stable attributions and increase growth mindsets improve mental health among adolescents (Schleider & Weisz, 2016). However, these networks were largely undirected and estimated from cross-sectional data. As such, causality cannot be verified. Additionally, current models cannot account for interactions between cognitive style components and the occurrence of stressful events. Rather the estimated edges help generate causal hypotheses that need to be verified through other methods.

The temporal network offers insights into how cognitive style and depression may coevolve over time in adolescence. The large difference in in- and out-prediction between nodes, such that the nodes that have the strongest in-prediction are not the same nodes as the ones that have the strongest out-prediction, further highlights the value in data that allow for inferring edge direction. Interestingly, whereas the development of a stable or global cognitive style predicts

future depressive symptoms, having an internal cognitive style appears heavily influenced by experiences of depression. This is consistent with findings that the internal component had low expected influence in both contemporaneous networks. Perhaps mood symptoms, particularly during impressionable adolescent years, instigate or strengthen beliefs that negative experiences are one's own fault. This is consistent with conceptualizations of the hopelessness theory that remove the internality dimension from calculation of negative cognitive style composite scores because it was thought to be more relevant in exacerbating depression (through decreased self-esteem) among those already depressed (Haeffel et al., 2008).

Additionally, among depressive symptoms, low self-esteem and low mood may be critical bridges. That is, negative cognitive styles (particularly stability or globality, respectively) may be most likely to lead to these symptoms and, when activated, these symptoms could trigger downstream activation of other symptoms. Notably, these symptoms were strong bridges in the contemporaneous network as well. This is consistent with research showing low self-esteem in adolescence predicts risk for depression in adulthood (Orth, Robins, & Roberts, 2008; Orth, Robins, Trzesniewski, Maes, & Schmitt, 2009). Additionally, the degree to which one's self-worth is sensitive to external feedback predicts depression in adolescents (Burwell & Shirk, 2006). Taken together, evidence for bidirectional effects between the networks of negative cognitive style components and depressive symptoms supports more nuanced hypotheses about feedback effects between cognitive vulnerability and chronic depression.

Limitations

Results could change with more observations and additional nodes (e.g., other symptoms, behavioral tasks, biomarkers). Furthermore, data were drawn from a community sample, potentially limiting generalization to clinical samples. However, cognitive style component

scores were largely normally distributed and included the full range of possible scores. Studying individuals with widely varying vulnerability scores is important as negative cognitive style may precede the onset of depression during adolescence.

Analyses are exploratory and hypothesis-generating. As much of the data were cross-sectional and resulting networks undirected, we can only speculate about causality; we must acknowledge that numerous possible explanations exist, including bidirectional relationships and third variables. Research with time-series and experimental methods is needed to support the hypotheses that follow from the analyses in this paper. Replication of these results and extensions to the individual level also are necessary before interventions based on these data are implemented. Additionally, although the cross-lagged panel network offers a compelling lens into a potential causal structure, results may be specific to the two-year time lag. For those vulnerable to depression, it is common for symptoms to emerge and worsen during the period from early to middle adolescence (Cole et al., 2002), which is the period reflected in this study. According to the hopelessness theory, cognitive styles develop and problematically interact with stressors (i.e., negative events that individuals interpret in problematic ways) to increase vulnerability over time. Thus, it is possible that two years is an appropriate window within which to see the emergence of particular cognitive styles and their negative effects as stressful events unfold or accumulate for individuals. Indeed, Mac Giollabhui et al. (2018) found that negative cognitive styles combined with stressful events to predict first onset of major depressive episodes among adolescents over this time interval. However, we do not know the ideal time lag at which to study these relationships, or if the time lag is even uniform across people, particularly in the absence of measures of intervening stressors.

Cognitive styles often stabilize during adolescence and persist thereafter (Hankin, 2008; Hankin et al., 2009). This work only tells us, however, that adolescents with high composite scores tend to become adults with high composite scores. We do not know whether relationships among components and between components and depression symptoms are also stable from adolescence to adulthood. Cognitive styles appear to be clinically relevant across the lifespan, but they may not convey risk in the same way during different phases. Future studies should more rigorously examine how and when components meaningfully differ and predict disorder onset as well as formally test whether a network or components-based approach bears greater predictive validity than additive or weakest link scores.

Conclusions

We present novel approaches for understanding cognitive style and its role in the etiology and maintenance of depression. Results suggest that the components of cognitive style are distinct and interacting, both within a system themselves and in their influence on depressive symptoms. From this perspective, more precise predictions could be made about how, when, and for whom risk factors will lead to the development or worsening of psychopathology. Substantial evidence demonstrates the perils of negative cognitive style; new hypotheses from this line of work may clarify this phenomenon and foster development of targeted interventions.

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Table 1. Node labels and item descriptions

ACSQ		CDI	
Node	**Item**	**Node**	**Item**
ACSQ1	Internal	CDI1	Negative/Depressed Mood
ACSQ2	Stable	CDI2	Ineffectiveness
ACSQ3	Global	CDI3	Anhedonia
ACSQ4	Consequences	CDI4	Negative Self-Esteem
ACSQ5	Self-Worth	CDI5	Interpersonal Problems

Note. ACSQ=Adolescent Cognitive Styles Questionnaire, CDI=Children's Depression Inventory.

Figure 1. Graphical LASSO comprising cognitive style items

Note. (A) Edges depict regularized partial correlations. The relative strength of a partial correlation is denoted with edge thickness (i.e., stronger correlations are depicted with thicker edges) and the sign is denoted with edge color (i.e., green indicates positive correlations and red indicates negative correlations). (B) Indices of centrality as normalized (*z*-scored) estimates. Higher values indicate a more central overall role in the network.

Figure 2. Graphical LASSO comprising contemporaneous measures of cognitive style and depression

Note. (A) Edges depict regularized partial correlations. Thickness of edges reflects relative strength of association. Maximum edge strength=.64. Communities highlighted in unique colors. (B) Indices of centrality considering only the relation between a given node and nodes in the other community. Higher values indicate greater influence.

Figure 3. Cross-lagged panel network

Note. Edges depict cross-time effects and arrows indicate the direction of prediction. Edge thickness reflects strength of the effects.

Figure 4. Cross-lagged panel network estimates of centrality

Note. Cross-Lagged (Column 1): In-prediction estimates for a given node at T2 by all *other* nodes at T1 (i.e., excludes auto-regressive path of a given node) and out-prediction estimates for a given node at T1 to all *other* nodes at T2. Cross-Construct (Column 2): In-prediction estimates for a given node at T2 by all nodes in the *other construct* at T1 (i.e., excludes any path connecting nodes from the same construct) and out-prediction estimates for a given node at T1 to all nodes in the *other construct* at T2. Larger values indicate greater centrality.