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Highlights

- Fixed mindsets show consistent associations with internalizing symptoms
- The unique contributions of mindsets to internalizing symptoms remains unclear
- We assessed unique contributions of mindsets to anxiety and depression symptoms
- Mindsets contributed little unique variance, as compared to hopelessness

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Contributions of fixed mindsets and hopelessness to anxiety and depressive symptoms:

A commonality analysis approach

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Abstract

Background. Fixed mindsets (beliefs that personal traits are unchangeable) show consistent associations with internalizing symptoms. However, the mindset-internalizing symptom link has previously been studied in isolation of other maladaptive cognitions that relate to internalizing symptoms. Thus, the unique contributions of mindsets to internalizing symptoms remains unclear. **Method.** We used commonality analysis (CA), which yields unique *and* shared effects of independent variables on an outcome, to assess unique contributions of *emotion* and *anxiety* mindsets to anxiety and depression

symptoms, relative to the contributions of hopelessness. Participants in two online studies (*Ns*=200, 430) self-reported depression and anxiety symptoms, hopelessness, and emotion and anxiety mindsets. **Results.** In Study 1, neither mindset type contributed unique variance to depression or anxiety beyond the contribution of hopelessness. In Study 2, emotion mindsets again explained no unique symptom variance. Anxiety mindsets uniquely contributed 2.0% and 6.5% of depression and anxiety variance, respectively—but far larger proportions of symptom variance (20.0%-60.9%) were contributed by hopelessness alone, variance shared by hopelessness and anxiety mindsets, and variance shared among hopelessness, anxiety mindsets, and emotion mindsets. **Limitations.** The cross-sectional design precludes causal conclusions, and the non-referred adult samples may limit generalizability. **Discussion**. Mindsets contributed little unique variance to internalizing symptoms beyond hopelessness. Interventions teaching growth mindsets have been shown to reduce internalizing problem in past studies. However, these interventions might not necessarily operate by shaping mindsets; rather, they may affect symptom change by shaping closely-linked maladaptive cognitions—like hopelessness—with stronger ties to internalizing distress.

Keywords: commonality analysis, emotion mindset, anxiety mindset, hopelessness, depression, anxiety

To navigate the social world, humans rely on *guiding cognitions* to interpret and respond to interactions and experiences. Whether or not these cognitions are accurate, they shape our responses to everyday events, particularly those involving adversity. As such, contemporary models emphasize cognitions' roles in the onset, maintenance, and course of depression and anxiety (Beck, 2002; Disner et al., 2011; Lewinsohn et al., 2001). Numerous maladaptive cognitions have been implicated in the etiology of depression *and* anxiety, including negative attributional style (Fresco et al., 2006; Luten et al., 1997), hopelessness (Starr and Davila, 2011; Strohmeier et al., 2016), interpretation biases (Everaert et al., 2018; Stuijfzand et al., 2018), and psychological inflexibility (Kashdan and Rottenberg, 2010). Recently, increased attention has focused on another kind of guiding cognition—implicit theories, or *mindsets*—

with possible relevance to internalizing psychopathology (for a review, see Schleider et al., 2015). Mindsets are core assumptions about the malleability of personal traits (Molden and Dweck, 2006). Whereas individuals with a growth mindset view personal traits (e.g. intelligence, likeability, anxiety, or sadness) as inherently malleable and changeable through effort, those with a fixed mindset view these attributes as fixed and thus unchangeable through effort (Dweck and Leggett, 1988; Molden and Dweck, 2006).¹ Individuals holding growth (versus fixed) mindsets tend to cope more adaptively and recover more rapidly following setbacks (Burnette et al., 2013; Erdley et al., 1997; Yeager et al., 2013), potentially because they view setbacks as opportunities for personal growth rather than indicators of permanent, personal deficits. Further, fixed mindsets of multiple attributes correlate with and predict higher levels of depression and anxiety in youth and adults (for reviews, see Schleider et al., 2015; Schleider and Schroder, 2018), and brief interventions teaching individuals to adopt growth mindsets have reduced internalizing problems in high-risk and community samples (Kneeland et al., 2016; Miu and Yeager, 2014; Schleider et al., 2019; Schleider and Weisz, 2016; Schleider and Weisz, 2018).

Thus, research identifies fixed mindset as a possible cognitive vulnerability factor for internalizing problems. However, the degree to which mindsets independently explain variance in these problems—and even change in emotional symptoms following "growth mindset" interventions—remains unclear. This is because fixed mindsets correlate with other well-established cognitive vulnerabilities for emotional problems, such as perceived control over behavior and emotion (Schleider & Weisz, 2016), perfectionism (Schroder et al., 2014), and problematic worry (Schroder et al., 2016). Indeed, construct overlap across cognitive vulnerability factors is a recognized challenge to identifying key contributors to internalizing distress (Abela and Sarin, 2002; Hong and Cheung, 2015). This overlap makes it difficult to

¹ In this article, as in much of the mindset literature, fixed and growth mindsets are discussed as dichotomies for the sake of convenience; in reality, people hold mindsets that lie along the continuous fixed-to-growth dimension (Dweck and Sorich, 1999), reflected in their operationalization as continuous rather than binary scores.

disentangle each factor's independent versus shared links with clinical outcomes. To clarify the potentially independent role of mindsets, we examined *independent* and *shared* contributions of two types of mindsets (of *emotion* and *anxiety*) to anxiety and depression symptoms, versus the independent contributions of hopelessness—an established risk factor for internalizing psychopathology (Alloy et al., 2016; Miranda and Mennin, 2007) that is conceptually similar to fixed mindsets. Indeed, measures of mindsets and hopelessness are both derived from learned helplessness theory (Dweck, 2017, 1975); therefore, directly assessing the unique and shared contributions of these constructs is all the more relevant. Emotion mindsets involve beliefs about whether emotions can be changed, especially through effort (Schroder et al., 2014; Tamir et al., 2007), while anxiety mindsets involve beliefs about whether symptoms of anxiety can be changed (Schroder et al., 2014). To overcome interpretive challenges created by construct overlap, we used commonality analysis (CA) to identify unique and shared links of mindsets and hopelessness to internalizing psychopathology. Beyond clarifying the role of mindsets in emotional distress, this study may offer a roadmap for using CA to parse independent and common contributions of correlated vulnerability factors to clinical outcomes.

Challenges to testing cognitive vulnerability models of internalizing problems. Equifinality and multicollinearity present challenges to modeling predictors of internalizing problems. Equifinality is the theory that multiple variables contribute to, and serve as distinct pathways toward, a given clinical outcome (Cicchetti and Rogosch, 1996). Given evidence reviewed above, equifinality is virtually certain in the etiology of depression and anxiety, suggesting the need to assess multiple predictors to gain a cohesive understanding of the onset and maintenance of anxiety and depression.

Acknowledging equifinality means relying on multivariate etiological models, leading to the challenge of *multicollinearity* in interpreting those models. Multicollinearity arises when at least two correlated predictors are assessed simultaneously in a linear regression – a type of statistical model that tests simultaneous predictors of an outcome. The adverse effect of multicollinearity on regression output

is well-recognized (Farrar and Glauber, 1967; Goodhue et al., 2017; Vatcheva et al., 2016). Problems due to multicollinearity include unstable, biased standard errors leading to unreliable *p*-values for predictors and untenable interpretations of effects (Hoffmann and Shafer, 2015; Mason et al., 1975; Tu et al., 2004). Linear regression aims to compare relative associations of various predictors to an outcome, but even modest multicollinearity can obscure computation of key independent effects due to the information that collinear predictors share. Multicollinearity among predictors is often unavoidably common in tests of cognitive models of depression and anxiety, particularly those aiming to differentiate effects of specific cognitive predictors (e.g., hopelessness; negative attributional style; fixed mindset, which often covary).

Addressing Multicollinearity: Challenges and Solutions. There are approaches to addressing multicollinearity. One such approach involves the creation of latent variables within structural equation models, in which multiple scores indexing various types of cognitive risk are loaded onto a single "cognitive vulnerability" factor. However, this approach precludes tests of whether specific factors relate differentially to outcomes. Likewise, other approaches (e.g., using partial least squares regression or factor analysis to generate and remove highly-correlated model components) result in some degree of lost predictive information by virtue of combining and removing various predictors. Thus, these approaches cannot identify *individual* key correlates and predictors, nor can they quantify shared variance among specific correlated predictors.

Commonality analysis (CA) is an effective but underutilized technique for determining individual predictors' contributions to an outcome. CA was developed in the 1960s (Newton and Spurrell, 1967) but remains infrequently used compared to regression, factor analysis, and SEM among clinical scientists (Kraha et al., 2012; for examples of CA applied in non-clinical social science research see Nimon et al., 2010). CA decomposes regression output, \Box^2 , into unique and common effects (Newton and Spurrell, 1967). *Unique effects* indicate how much variance is independently accounted for in the outcome by a single independent variable. *Common effects* indicate how much variance in the outcome is common to a

specific set of two or more predictors (Nathans et al., 2012). It establishes not only whether various predictor-outcome associations exist, but also the degree to which each predictor—and all possible predictor combinations—share common variance with that outcome. Thus, CA is one way of gauging the relative importance of multiple individual predictors to an outcome of interest (McPhee and Seibold, 1979).

Parsing the influence of individual vulnerabilities using CA or groups of vulnerabilities (versus latent variables or factors) may inform more precise etiological models. For example, CA has identified hopelessness as contributing more unique variance to depression in adults than other cognitive vulnerabilities such as rumination and dysfunctional attitudes (Marchetti et al., 2016). However, no studies known to these authors have directly examined the relative contributions of hopelessness and mindsets to psychopathology outcomes (Ford et al., 2018 controlled for hopelessness when predicting use of cognitive reappraisal, but not when evaluating whether emotion mindsets predicted depression symptoms).

Present study. Given the paucity of research on the independent links between fixed mindsets, hopelessness, and psychopathology, we examined the degree to which two types of fixed mindsets (emotion; anxiety) accounted for unique and shared variance in depression and anxiety symptoms relative to *hopelessness*, a well-established cognitive vulnerability for internalizing symptoms. Using CA, we examined these as sociations in two community samples of adults who are parents (recruited for a prior study; Schleider & Weisz, 2018). We expected both fixed mindsets and hopelessness to show bivariate associations to depression and anxiety sum-scores, but given the novelty of the specific research questions (regarding the magnitude of unique and shared associations), we had no specific hypotheses. Our specific objective was to evaluate how much variance in internalizing symptoms were unique to individual predictors vs. shared among those predictors. We also utilized a CA approach for individual depression symptoms (Fried and Nesse, 2015) given evidence that different depression symptoms may be

differentially related to certain risk factors (Fried et al., 2013). Beyond parsing the relative associations of mindsets and hopelessness to parents' internalizing psychopathology, a secondary aim of this study is to demonstrate a clinically-relevant application of CA: an underutilized, high-potential analytic technique for clinical science. Beyond this study, CA may be used to identify the most meaningful correlates, predictors, and mechanisms of psychopathology, overcoming many of the interpretive challenges that multicollinearity and equifinality present.

Methods

Participants

206 Amazon Mechanical Turk (mTurk) participants completed Study 1, which involved completion of a variety of self-report measures at one time point only (a cross-sectional study) online. Participants provided informed consent online, and were told they could withdraw from the study at any time. MTurk workers located in the United States with a >95% task approval rate for prior human intelligence tasks (HITs: online tasks which mTurk workers can complete in exchange for payment) were eligible. Participants were also required to be parents of one or more children ages 7–17; this inclusion criterion reflects the objectives of the original study for which these data were collected (procedure for identifying parent status, and unrelated previous analyses and study objectives, are detailed in Schleider & Weisz, 2018). There were no other inclusion or exclusion criteria.

MTurk participants consistently perform tasks similarly to laboratory participants (Hauser and Schwarz, 2016) and provide reliable, valid survey data on psychopathology (Chandler and Shapiro, 2016). Here, we excluded six participants who completed the study in an unusually short or long period, which can indicate inattention to questions and/or extended breaks (3 SDs above/below mean completion time, 22.13 min), resulting in a sample of 200 participants (49.5% male; *Mage* = 36.24 years, range = 24-64 years; 50.50% college graduates, 25.0% single parents, 70.40% Caucasian). 31.60% reported having

received mental health treatment at some point in their lives (of these participants, 13.80% had received psychotherapy, 43.08% medication, and 43.12% both types of treatment).

459 mTurk participants completed Study 2. Study 2, originally conducted for a separate project, involved a randomized, online trial of a single-session online intervention targeting parents of youths ages 7 to 17. For present purposes, we used baseline questionnaire data from participants, all of which were collected prior to intervention condition randomization. Inclusion and exclusion criteria were identical to Study 1, except for excluding 9 participants who exited the study prior to the randomization procedure, which was unrelated to present analyses (Schleider & Weisz, 2018). An additional 15 participants were excluded for completing the survey in an unusually short or long time (3 SDs above or below the mean completion time). Thus, analyses included 430 participants (44.70% male; Mage = 36.31, range 24-63 years; 75.80% Caucasian; 45.6% college graduates; 24.88% single parents). 40.00% reported having received mental health treatment at some point in their lives (of these parents, 21.76% had received psychotherapy; 33.54% medication, and 44.71% both types of treatment). All procedures, including the consent process in both samples, were approved by the appropriate institutional review board.

Procedure

Study 1 and 2 participants provided informed consent prior to data collection; the university's IRB approved all procedures. All participants completed a questionnaire battery including the following measures:

Demographic and treatment history questionnaire. This questionnaire asks about socioeconomic and demographic information (e.g., age, sex, number of children, marital status, educational attainment) and information about mental health treatment history (lifetime history of receiving psychotherapy, medication-based treatment, or both for mental health-related problems).

Mind-set measures. Participants' malleability beliefs about emotions and anxiety were assessed using previously validated measures of four items each (Schroder et al., 2014; Tamir et al., 2007). For each item, respondents indicate agreement or disagreement with a given statement (e.g., "The truth is, people have very little control over their emotions"; "Your anxiety is something about you that you cannot change very much"). The anxiety mind-set scale includes four statements worded in the fixed-minded framework (Schroder et al., 2014); the emotion mind-set scale includes two fixed-minded statements and two growth-minded statements (e.g., "Everyone can learn to change the emotions that they have"). After reverse-coding, higher summed scores on both scales indicate stronger fixed mindsets of anxiety or emotion. Both scales have shown strong internal consistency and construct validity (Schroder et al., 2014; Tamir et al., 2007). Studies suggest that mind-sets across domains are associated but psychometrically distinct (Schroder et al., 2014), such that (for instance) individuals can hold a fixed anxiety mind-set measures were $\alpha = .88$ and $\alpha = .94$, respectively in Study 1 and $\alpha = .85$ and $\alpha = .92$ respectively in Study 2.

Beck's Hopelessness Scale-4 (BHS-4). Participants' hopelessness were assessed using a previously validated 4-item self-report scale (Forintos et al., 2013). Respondents indicate agreement or disagreement with four statements (e.g., "My future seems dark to me"; "Things just won't work out the way I want them to"). Higher summed scores indicate higher hopelessness. The internal consistency and validity of the BHS-4 are similar to the full version of Beck's Hopelessness Scale (Yip and Cheung, 2006). Internal consistencies were $\alpha = .93$ in Study 1 and $\alpha = .92$ in Study 2.

Brief Symptom Inventory-18: Depression and Anxiety. Psychological distress was measured using the BSI-18, a self-report questionnaire measuring anxiety and depression (Derogatis, 2001). Respondents indicate on a 0-4 scale the extent to which they are troubled by each of five depression symptoms (*anhedonia, loneliness, feeling down, worthlessness, pessimism*) and six anxiety symptoms

(*nervousness, tension, suddenly scared, panic, restlessness, fear*). One item assessing suicidal ideation associated with depression was removed. The sum-score of each subscale yields a total depression score and a total anxiety score. The BSI-18 Depression and Anxiety subscales are widely-used psychiatric screening tools in clinical settings, and epidemiological studies and suggest adequate construct validity (Franke et al., 2017). Reliability was $\alpha = .90$ for Depression and $\alpha = 92$ for Anxiety in Study 1, and $\alpha =$.91 for Depression and $\alpha = .90$ for Anxiety in Study 2.

Analytic Plan

For each dependent variable in both studies, we first ran a multiple regression with hopelessness (BHS-4), anxiety mindsets, and emotion mindsets as independent variables. We then calculated the total amount of variance the entire model shared with the dependent variable (R^2) for each of X dependent variables (e.g. 59.7% R^2 for sum score depression in Study 1). We then conducted a commonality analysis using the *R* package *yhat* 2.0, which decomposes the total R^2 of the model into non-overlapping unique and common partitions. These partition scores add up to the total R^2 of the model, and include: unique variance in the dependent variable associated with hopelessness (U1); unique variance in the dependent variable associated with model associated with emotion mind-sets (U3); common variance in the dependent variable explained by hopelessness and emotion mind-sets only (C1); common variance in the dependent variable explained by anxiety mind-sets and emotion mind-sets only (C3); and common variance in the dependent variable explained by all of hopelessness, anxiety mind-sets, and emotion mind-sets (C4).

Given our focus on the relative contributions of hopelessness and mindset to psychopathology, our analyses focus on the partitions of unique variance (sections U1, U2, and U3). For our analyses, we scaled these partitions by converting them to the relative variance shared by the partition (adds up to 100% instead of R^2) and then multiplying them by the model R^2 value to improve interpretability. This

common practice allows us to estimate the amount of unique absolute variance each independent variable contributed to the dependent variable, accounting for the contributions of all other variables in the model. This procedure is carried about because it is possible for an independent variable to look very relatively important (e.g. uniquely sharing 50% of the variance in the dependent variable) while being unimportant in an absolute sense (the overall model shares 0.3% of the variance in the construct of interest, leading the "large" unique relative independent variable to only share 0.15% of the absolute variance in the construct of interest). Therefore, while multiple regression can include statistically significant independent variables with little practical relevance and no direct analysis of shared variance among independent variables, commonality analysis focuses on absolute variance shared and explicitly evaluates independent variables' unique and shared variance.

We also performed bootstrapping (1,000 bootstraps) around our partition estimates to determine their precision and examine whether certain variables shared more unique variance in each dependent variable than other independent variables. This bootstrapping procedure, however, cannot tell us whether a construct shares greater than 0 variance as unique partitions in commonality analysis cannot be less than 0. Instead, we can interpret the point estimate as effect sizes (Marchetti et al., 2018, < 1% negligible, > 1% small, > 9% moderate, and > 25% large, see 2016) and see whether the confidence intervals from the bootstrapping extend into the negligible range.

Results Study 1

Hopelessness, anxiety mind-sets, and emotion mind-sets accounted for 59.70% of the variance in sum-score depression in a multiple regression in this sample. Hopelessness was uniquely associated with 33.00% of that variance (95% CI: 21.21%, 45.81%), while anxiety mind-sets was uniquely associated with 0.60% (95% CI: 0.00%², 2.80%) and emotion mind-sets was uniquely associated with 0.00% (95%

 $^{^{2}}$ Here and throughout the paper we use 0.00 to denote that the point estimate or lower end of the confidence interval was 0 to at least three decimal places (i.e. 0.000). It is highly unlikely that the estimate is literally 0, but the

CI: 0.00%, 0.80%). Therefore, hopelessness shared significantly more unique variance in sum-score depression than anxiety or emotion mind-sets, which did not differ from one another. The confidence intervals for both anxiety and emotion mind-sets included negligible (<1%) values. We then directly examined the shared variance of emotion and anxiety mindsets with hopelessness while with sum score depression as the dependent variable. 98.3% of variance accounted for by emotion and anxiety mindsets was shared with hopelessness. This is direct evidence that hopelessness shares more unique variance with sum score depression and accounts for the vast majority of the variance mindsets share with the outcome.

Hopelessness, anxiety mind-sets, and emotion mind-sets accounted for 50.89% of the variance in sum score anxiety in a multiple regression in this sample. Hopelessness was uniquely associated with 28.20% of that variance (95% CI: 17.60%, 39.50%), while anxiety mind-sets was uniquely associated with 0.66% (95% CI: 0.00%, 3.80%) and emotion mind-sets was uniquely associated with 0.00% (95% CI: 0.00%, 1.30%). Therefore, hopelessness shared significantly more unique variance in sum score depression than anxiety or emotion mind-sets, which did not differ from one another. The confidence intervals for both anxiety and emotion mind-sets included negligible (<1%) values. We then directly examined the shared variance of emotion and anxiety mindsets with hopelessness with sum score anxiety as the dependent variable. 98.1% of variance accounted for by emotion and anxiety mindsets was shared with hopelessness. This is direct evidence that hopelessness shares more unique variance with sum score anxiety and accounts for the vast majority of the variance mindsets share with the outcome. The depression and anxiety results follow the same pattern whether or not participants have previously received mental health treatment (See Supplementary Material).

We then took a symptom-level approach to depression given its heterogeneous nature (Fried & Nesse, 2015) and the known instances of risk factors relating differentially to different symptoms using

statistical software used does not calculate enough significant digits to assess the contribution beyond the practical equivalent of 0.000.

regression-based (Beevers et al., 2018; Fried, Nesse, Zivin, Guille, & Sen, 2014) and commonality (Marchetti et al., 2018; Marchetti, Loeys, Alloy, & Koster, 2016) approaches. Hopelessness, anxiety mind-sets, and emotion mind-sets account for 29.60% - 57.84% of the variance in individual depression symptoms in multiple regressions in Sample 1. For all individual symptoms, hopelessness shared significantly more unique variance in sum score depression than anxiety or emotion mind-sets, which did not differ from one another and whose confidence intervals included negligible (<1%) values (Figure 2).

Results Study 2

Hopelessness, anxiety mind-sets, and emotion mind-sets accounted for 65.42% of the variance in sum score depression in a multiple regression in this sample. Hopelessness was uniquely associated with 39.84% of that variance (95% CI: 31.51%, 48.71%), while anxiety mind-sets was uniquely associated with 1.37% (95% CI: 0.40%, 3.10%) and emotion mind-sets was uniquely associated with 0.00% (95% CI: 0.00%, 0.50%). Therefore, hopelessness shared significantly more unique variance in sum score depression than anxiety or emotion mind-sets, which did not differ from one another. The confidence intervals for both anxiety and emotion mind-sets included negligible (<1%) values. We then directly examined the shared variance of emotion and anxiety mindsets with hopelessness with sum score depression as the dependent variable. 96.9% of variance accounted for by emotion and anxiety mindsets was shared with hopelessness. This is direct evidence that hopelessness shares more unique variance with sum score depression and accounts for the vast majority of the variance mindsets share with the outcome.

Hopelessness, anxiety mind-sets, and emotion mind-sets accounted for 48.58% of the variance in sum-score anxiety in a multiple regression in this sample. Hopelessness was uniquely associated with 22.79% of that variance (95% CI: 15.00%, 30.99%), while anxiety mind-sets was uniquely associated with 3.16% (95% CI: 1.20%, 6.30%) and emotion mind-sets was uniquely associated with 0.05% (95% CI: 0.00%, 0.80%). Therefore, hopelessness shared significantly more unique variance in sum score depression than anxiety or emotion mind-sets, which did not differ from one another. Confidence

intervals for emotion mind-sets included negligible (<1%) values, while anxiety mindsets' confidence interval indicated a small but significant unique contribution to sum-score anxiety. We then directly examined the shared variance of emotion and anxiety mindsets with hopelessness with sum score anxiety as the dependent variable. 90.1% of variance accounted for by emotion and anxiety mindsets was shared with hopelessness, indicating that hopelessness shares more unique variance with sum-score anxiety and accounts for the vast majority of the variance mindsets share with the outcome. The depression and anxiety results follow the same pattern whether or not participants have previously received mental health treatment (See Supplementary Material).

We then took a symptom-level approach to depression. Hopelessness, anxiety mind-sets, and emotion mind-sets accounted for 39.79% - 59.59% of the variance in each individual depression symptom in multiple regressions in Sample 1. Hopelessness shared significantly more unique variance in each individual depression symptom than anxiety or emotion mind-sets, which did not differ from one another and whose confidence intervals included negligible (<1%) values (Figure 3).

Discussion

Commonality analyses in two large adult samples revealed hopelessness consistently shared significantly more unique variance with anxiety, depression, and individual depression symptoms than emotion mindsets and anxiety mindsets. Hopelessness shared 22.79% - 39.84% more unique variance than emotion mindsets (medium-large effect sizes) and 19.63% - 38.47% more unique variance than anxiety mindsets (medium to large effect sizes) across all outcomes. Anxiety mindsets shared a substantive, though small (3.16%), amount of variance with anxiety in the larger sample, but hopelessness shared nearly 7 times as much unique variance (22.79%) with the same outcome. Emotion mindsets did not share substantive unique variance with any outcome in either sample. Further, most variance emotion and anxiety mindsets shared with the outcomes was also shared with hopelessness (90.1% - 98.3%): That

is, nearly all variance that mindsets shared with depression and anxiety were accounted for by concurrent hopelessness levels.

The origin and intended application of mindset theory (and mindset measures) might explain these results. Mindset theory was derived from the theory of learned helplessness (Dweck, 2017, 1975), a precursor to hopelessness theory (Abramson et al., 1989, 1978). However, the original measures designed to assess mindsets were developed in the contexts outside of clinical psychology, such as studies examining racial achievement gaps and intergroup conflict (Dweck, 2012); all subsequently-created mindset measures have followed these original templates. Measures primarily developed to predict outcomes such as odds of attempting more challenging math problems (Dweck and Leggett, 1988) might naturally share less unique variance with internalizing psychopathology than an assessment (i.e. hopelessness) with similar theoretical lineage developed for clinical science contexts.

Differences between the learned helplessness and hopelessness theories of depression may further explain the primacy of hopelessness, versus mindsets, for depression symptom variance. The learned helplessness theory of depression emphasized causal, negative attributions about the self and future consequences as key etiological processes in depression (Abramson et al., 1978). According to mindset theory's integration with the process model of emotion regulation, these negative, self-directed attributions result from fixed mindsets key affect outcomes, like internalizing symptoms, particularly following stressful situations (e.g., life transitions; Yeager, 2017). In contrast, hopelessness theory explicitly *de*emphasizes the direct role of negative, self-directed attributions in the etiology of depression (Abramson et al., 1989). In the hopelessness theory framework, negative attributions about the self and future consequences do not precipitate depression directly; rather, they exacerbate hopelessness, which in turn contributes to depressive symptoms and disorders. Our results for sum-score and symptom-level depression, where hopelessness consistently shares more unique variance than emotion and anxiety mindsets, fit with hopelessness theory rather than helplessness theory.

Original formulations of hopelessness theory primarily addressed the comorbidity of anxiety and depression rather than predicting anxiety directly (Maser, 1990). Early and more recent empirical work indicates hopelessness may increase risk for both anxiety and depression symptoms (Ahrens and Haaga, 1993; Fassett-Carman et al., 2019; Reardon and Williams, 2007). However, hopelessness does not predict anxiety symptoms in all circumstances (e.g. Waszczuk et al., 2016), and the theoretical process by which hopelessness leads to anxiety symptoms is less clear. Interestingly, in the present samples, hopelessness still shared far more unique variance with anxiety than emotion or even anxiety mindsets. Anxiety mindsets may have a small unique role to play in anxiety, but future investigations should explicitly test when anxiety mindsets provide information above and beyond hopelessness.

There are no studies known to these authors where interventions targeting emotion or anxiety mindsets directly improve anxiety or depression symptoms, though such interventions have been proposed in response to previous findings (Schroder et al., 2018, 2014); note that interventions targeting other mindset types, e.g. personality, have directly reduced internalizing distress (Schleider & Weisz, 2018). Three different interventions teaching emotion malleability beliefs have improved the perceived efficacy of psychotherapy in parents (Schleider and Weisz, 2018; Smith et al., 2018), school-related wellbeing in adolescents (Schleider and Weisz, 2018; Smith et al., 2018), and adaptive emotion regulation strategy use (e.g., increased acceptance and perspective-taking) in adults (Kneeland et al., 2016). The substantial overlap in variance shared with anxiety and depression between mindsets and hopelessness implies that interventions that effectively target emotion and anxiety mindsets may be able to effectively target hopelessness. Future work should assess whether interventions targeting emotion and anxiety mindsets might drive psychopathology-related outcomes.

Notably, these emotion mindset interventions were brief (as short as eight minutes) and scalable (administered online at no cost). Importantly, reducing interventions' length may not sacrifice

effectiveness; single session interventions have similar effect sizes for reducing psychopathology as multi-session interventions, implying hyper-brief treatment may in some cases be similarly effective as longer-term treatment (Schleider and Weisz, 2017). Future studies may directly test whether brief emotion mindset interventions can reduce hopelessness—and in turn, internalizing distress. Such studies will help elucidate the potential of brief interventions to mitigate a critical contributor to psychiatric risk.

These analyses have limitations. Their cross-sectional nature cannot establish causality, and other designs are better suited to evaluating to what extent hopelessness is a causal risk factor, relative to mindsets. However, the minimal unique variance shared between emotion and anxiety mindsets and internalizing psychopathology after accounting for hopelessness implies they might be less-than-ideal intervention targets for intervention, *unless* such interventions also targeted the variance those mindsets share with hopelessness. This possibility has yet to be tested and should be prioritized in future trials of these interventions. Analyses were also conducted in a non-selected sample. It is possible results would differ in a clinical sample, although present results did not differ by participants' mental health treatment histories. Finally, both samples in these analyses included only parents of children aged 7-17, so it is possible that these results do not generalize to non-parents or parents of children at different ages.

Using commonality analysis in these samples disentangled unique and shared variance among hopelessness, emotion mindsets, anxiety mindsets, and internalizing psychopathology. In the context of internalizing psychopathology, studying fixed mindsets separately from hopelessness puts us at risk for committing a version of the jangle fallacy – assuming two constructs contribute above and beyond one another because we give them different names. We can capitalize on the structure and scalability of mindset interventions to conduct well-powered studies that identify the relative contributions of potential intervention mechanisms (e.g. hopelessness, anxiety mindsets, emotion mindsets). The commonality analysis approach could be applied to other situations where the unique and shared variance for several predictors is of theoretical and clinical interest. For example, the relative and shared contributions of

psychological flexibility and interpretation, two potential risk factors for internalizing distress in youth, could be evaluated directly using this method (Everaert et al., 2018; Kashdan and Rottenberg, 2010). Therefore, using commonality analysis more often could pave the way to deeper theoretical understanding and better targeted clinical interventions.

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Contributions: MCM and JLS contributed to the research design. MCM takes responsibility for data analysis and JLS takes responsibility for data integrity. MCM and JLS take responsibility for interpretation of the results. JLS held responsibility for data collection. MCM and JLS drafted the paper, and all authors provided critical revisions. All authors approved the final version of the paper for submission.

Limitations: The cross-sectional nature of these analyses cannot establish causality, and other designs are better suited to evaluating to what extent hopelessness is a causal risk factor, relative to mindsets. However, the minimal unique variance shared between emotion and anxiety mindsets and internalizing psychopathology after accounting for hopelessness implies they might be less-than-ideal intervention targets for intervention, *unless* such interventions also targeted the variance those mindsets share with hopelessness. This possibility has yet to be tested and should be prioritized in future trials of these interventions. Analyses were also conducted in a non-selected sample. It is possible results would differ in a clinical sample, although present results did not differ by participants' mental health treatment histories.

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FIGURE 2



FIGURE 3

