YOUTH COMORBIDITY AS A FUNCTION OF AFFECT, COPING, AND ANXIETY AND DEPRESSION SYMPTOMS

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ABSTRACT OF THE THESIS

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Youth presenting with comorbid anxiety and depressive symptomatology are at higher risk for functional impairment and poorer clinical outcomes compared to youth meeting criteria for either disorder alone (Costello, Mustillo & Erkanli, 2003; Garber & Weersing, 2010). The tripartite model of anxiety and depression, which stipulates that both disorders share convergent and discriminant features of positive and negative affect, complements current models of comorbidity. Likewise, research on coping mechanisms has pointed to coping responses that are common to both anxiety and depression (rumination, avoidance; McLaughlin, & Nolen-Hoeksema, 2011; Chu et al., 2016; Roelofs et al., 2009) and others that are specific to either depression (disengagement; Evans et al., 2014) or to anxiety (physiological arousal, escape; Hedtke, Kendall, & Tiwari, 2009). However, little research has looked at both affective trait disposition (tripartite theory), coping styles to stress, and clinical severity as a way to understand the unique and common mechanisms that underlie anxiety and mood diagnoses. In order to determine whether such mechanisms were present, demographics, coping, affect and anxiety/depression symptoms were entered as predictors in a two-step binary logistic regression used to

predict comorbid depression. The first set of analyses included demographics, affect and coping as predictors while the second set of analyses added anxiety and depression symptoms to rule out the possibility that comorbidity was related to clinical severity alone. Problem-solving (OR = -.19, 95% CI, .69-1.0) socioeconomic status (OR = -.22, 95% CI, .69-.95) and depression symptoms (OR = .21, 95% CI, 1.12-1.37) were significant in predicting comorbidity. Results did not indicate any predictive effects of affect on comorbid anxiety-depression. Findings from the study suggest that problem-solving and socioeconomic status may be important targets when considering treatment planning.

TABLE OF CONTENTS

I. Introduction

Despite recent advances in refining and implementing evidenced-based psychological treatments (EBPTs), lifetime prevalence rates report that approximately 32% of youth (aged 13 to 18) meet criteria for an anxiety disorder and 14% meet criteria for a mood disorder (Merikangas et al., 2010). Female youth are twice as likely to experience depression compared to male youth. Further, the presence of mood disorders increases with age, particularly among youth between the ages 13-14 and 17-18 (Merikangas et al., 2010). Similarly, female youth are at an increased risk for anxiety disorders compared to male youth, with the prevalence increasing with age. Additionally, 10-15% of youth who meet criteria for a principal anxiety disorder also meet criteria for a depressive disorder. Further, 25-50% of youth meeting criteria for a principal depressive disorder also meet criteria for an anxiety disorder (Axelson & Birmaher, 2001; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Cumming, Caporino & Kendall, 2014). Compared to youth who meet criteria for an anxiety disorder only ("pure anxiety") or depressive disorder only ("pure depression"), the prognosis for youth with comorbid anxiety and depression is worse. Specifically, youth with comorbid anxiety and depression are at higher risk of relapse/recurrence (Cartwright-Hatton, 2006), longer duration of condition, increased suicide attempts (O'Neil & Kendall, 2012), and greater functional impairment (e.g., difficulties in accomplishing developmentally congruent tasks across major life domains; Garber & Weersing, 2010). These findings strongly suggest that our understanding of the etiology and interacting features of comorbid

anxiety-depression is still in its nascent stages and warrants further investigation. Affect, coping and clinical severity (e.g., depression and anxiety symptoms) and their relationship to comorbidity is also not fully understood. A more complete picture of this relationship would help with identifying warning signs and prospective treatment targets for youth at greater risk for meeting criteria for both disorders. The current study seeks to determine which coping, affective, clinical severity and demographic factors predict the expression of comorbid anxiety-depression among youth.

Anxiety and mood symptoms and disorders are major source of distress and impairment among youth (Costello et al., 2003) and their comorbidity is more the rule rather than the exception (Chu, Merson, Zandberg & Areizaga, 2012), co-occurring at rates of up to 75% in some samples (Garber et al., 2010; Sørensen, Nissen, Mors & Thomsen, 2005). However, this high rate of comorbidity is not symmetrical. Up to 50% of youth who meet criteria for a depressive disorder also meet criteria for an anxiety disorder and up to 15% of youth with a primary anxiety disorder also meet for a concurrent depressive disorder (Axelson et al., 2001; Costello et al., 2003). As such, youth with primary depressive disorders tend to experience comorbid anxiety more often than do those with primary anxiety disorders who also have comorbid depression (Garber et al., 2010)

Several factors might account for this imbalance. First, subsyndromal levels of symptoms often have not been assessed in studies of comorbidity. Specifically, children diagnosed with anxiety disorders may have concurrent depressive symptoms even if they do not meet full criteria for a depressive diagnosis. These subthreshold symptoms may explain the link between anxiety and subsequent depressive disorders in adolescence.

Likewise, subthreshold depressive symptoms have been found to be a more reliable predictor of subsequent depressive disorders than symptoms of either Separation Anxiety Disorder or Social Anxiety Disorder (Keenan, Feng, Hipwell & Klostermann, 2009).

Additionally, anxiety disorders exhibit heterogeneous features relative to depression, so comorbidity is contingent on what anxiety symptoms and disorders are assessed (Avenevoli, Stolar, Li, Dierker, & Merikangas, 2001). For instance, Panic Disorder does not predict future depression, however, SOC and GAD are more associated with depression in adolescence (Bittner et al., 2007). Thus, although anxiety disorders are highly comorbid, combining them collectively likely distorts the strength and direction of the relationship between particular anxiety and depressive disorders.

Third, the level of comorbidity varies by age and developmental period. Anxiety tends to be more present in childhood whereas depression emerges in adolescence and adulthood (Woodward & Fergusson, 2001). Further, youth with comorbid anxiety and depression tend to be older than those with either disorder alone (Merikangas et al., 2002). This phenomenon may be explained by structure and differentiation of affect across development (Garber et al., 2010). For example, younger youth (3rd graders) were more likely to exhibit a unified construct of either disorder whereas older youth (6th graders) were more likely present with features consistent with tripartite model (e.g., negative affect; Cole, Truglio & Peeke, 1997). Thus, higher rates of comorbid anxiety and depression tend to be present among adolescents compared to children (Ollendick, Shortt & Sander, 2005).

Finally, although the co-occurrence of anxiety and depressive disorders is well established (Garber et al., 2010), the literature is less definitive about the sequential

nature of anxiety and depression, that is, which disorder precedes the other. Anxiety is likely to influence the expression of depression and vice versa among individuals with comorbid anxiety and depression (Bittner et al., 2007). However, most studies have focused on anxiety as the predictor and depression as the outcome. Evidence suggests that anxiety symptoms and disorders in childhood precede the onset of depression in adolescence and young adulthood (Chaplin, Gillham, & Seligman, 2009; Keenan & Hipwell, 2005). Alternatively, there is accumulating evidence that depression may precede the onset of anxiety symptoms and disorders (Cummings, Caporino, & Kendall, 2014; Gallerani, Garber, & Martin, 2010).

Collectively, these characteristics highlight the complexity of comorbid anxiety-depression and relationships between the two disorders that are still not fully understood. Several factors make understanding comorbidity particularly relevant for treating behavioral and emotional problems in youth.

Comorbidity's Impact on Functioning and Implications for Treatment

Given that half of all lifetime disorders begin by the age of 14 (Kessler et al., 2008), approaches that improve prevention and intervention are warranted. Comorbidity among children and adolescent populations is higher than among adults, where both within-class (e.g., multiple anxiety diagnoses) and across-class comorbidity (e.g., diagnosis of anxiety and conduct disorder) are common (Angold, Costello, & Erkanli, 1999; Merikangas et al., 2010). Functional impairment is also higher among youth with comorbid anxiety-depression than either disorder alone. For instance, youth presenting with both disorders are more likely to suffer from recurrence, longer duration, increased suicide attempts, less favorable response to treatment and increased utilization of

treatment services (Ezpeleta, Domenech & Angold, 2006; Garber et al., 2010). Research is limited, however, on youth comorbidity treatment studies. Most investigations have focused exclusively on treating either anxiety (Walkup et al., 2008) or depressive (March et al., 2004) disorders. In order to address this gap, transdiagnostic (Barlow et al., 2017; Chu et al., 2016; Chu, Hoffman, Johns, Reyes-Portillo, & Hansford, 2015; Kennedy, Tonarely, Sherman, & Ehrenreich-May, 2018) and modular (Chorpita et al., 2017; Weisz et al., 2012) treatments have been offered as a means to target both disorders concurrently and have demonstrated reasonable efficacy and effectiveness. Understanding the mechanisms that underlie complex comorbidity may help develop treatments that address these gaps.

Affective Processes

Affective processes play an influential role in the expression of positive and negative emotions, which have been strongly linked to anxiety and depressive psychopathology. A seminal study led by Clark and Watson (1991) aimed to determine whether specific overlapping symptoms and features of anxiety and depression represented a new distinct class of disorder not fully captured as pure anxiety or pure depression. Findings from the study led to the development of the tripartite model of anxiety and depression, which stipulates that both disorders share convergent features of Negative Affect (NA) and discriminant features in Positive Affect (PA), which is more associated with depression, and Physiological Hyperarousal (PH), which is more associated with anxiety. Clark and Watson (1991) define NA as representing "the extent to which a person is feeling upset or unpleasantly engaged rather than peaceful, and encompasses various aversive states including upset, angry, guilty, afraid, sad, scornful,

disgusted and worried" (p.321). Conversely, PA is the "extent to which a person feels a zest for life and is most clearly defined by such expression of energy and pleasurable engagement like active, delighted, interested enthusiastic and proud" (p.321). Interestingly, despite their opposite sounding labels, both mood dimensions are largely independent of one another and exhibit specific correlational patterns with other variables (Watson & Pennebacker, 1989). The results of their study strongly support that anxious and depressive symptoms share the nonspecific component of NA, which includes general affective distress and other negative symptoms. Both syndromes are further distinguished by PH, which is anxiety-specific, whereas the absence of PA is depression-specific.

Extensions of this model suggest that NA and PA may represent temperamental risk factors for anxiety and depression (Watson, Clark & Harkness, 1994). Additionally, other research on the self-reported symptoms of negative emotion has generated further evidence supporting a three-factor model (Lovibond & Lovibond, 1995). This research has fostered similar lines of inquiry into better understanding the temperamental characteristics of children. Specifically, Lonigan, Carey, and Finch (1994) found that in a clinical child sample that low PA predicted depression in children but did not predict anxiety. Another investigation by Joiner, Cantanzaro and Laurent (1996) tested the structure of the tripartite model of emotion in child and adolescent psychiatric inpatients. Exploratory factor analysis of the anxiety and depression self-report measures supported the three factors and were conceptually congruent to the tripartite model. Similarly, Chorptita, Albano and Barlow (1998) conducted an investigation among 216 children

with anxiety and depression and found the three-factor model most effectively categorized the sample clinical characteristics.

The latent structure of emotional disorders also suggests that there are higher order factors derived from negative and positive affect that determine the expression of anxious and depressive psychopathology. From an etiological perspective, the tripartite theory posits that "triple vulnerability" consists of generalized biological vulnerability, generalized psychological vulnerability and specific psychological vulnerability from early learning (Barlow, Allen, & Choate, (2004). A generalized biological vulnerability consists of non-specific contributing factors to the development of anxiety and negative affect (e.g., anxious or negative affective temperament; behavioral inhibition, family history of mental illness). Additionally, early life experiences also contribute to either a more generalized or specific psychological diathesis that can lead to anxiety and other negative affect states (Chorpita & Barlow, 1998). Collectively, these findings replicate the unique role of absent PA in predicting depression, high PH in predicting anxiety, and NA being common to both sets of disorders.

Valid and reliable scales for measuring PA and NA have been developed for both adults (Positive and Negative Affect Scales; PANAS; Watson, Clark and Tellegen, 1988) and for children (Positive and Negative Affect Scales for Children; PANAS-C; Laurent, Cantanzaro, Potter & Joiner, 1999). Although follow-up evidence supports the factorial validity of the adult and child versions, both the PANAS and PANAS-C do not contain scales for PH, a core affective feature among individuals meeting criteria for an anxiety disorder.

In pursuit of an alternative measure that sufficiently captures the full range of psychometric indicators within the tripartite model (e.g., NA, PA and PH), the Affect and Arousal Scale (AFARS) was developed (Chorpita, Daleiden, Moffitt, Yim, & Umemoto, 2000). The scale has displayed robust effects at measuring pure affective dimensions as opposed to symptom or disorder manifestations among a clinical youth population. Specifically, each core emotional factor was validated using exploratory factor analysis and replicated using confirmatory factor analysis. The AFARS has also been validated in follow-up studies (Chorpita, 2002) and has been developed for both parent and child reporters (Ebesutani, C., Okamura, K., Higa-McMillan, C., & Chorpita, B. F., 2011). Additionally, the AFARS has been shown to distinguish between anxiety and depression among youth ages 7 to 18. Chorpita et al., 2000). Taken together, research suggests that elements of the tripartite theory are uniquely associated with anxiety (high PH) or depression (low PA), and elements that are associated with both anxiety and depression (high NA). Thus, higher NA might be associated with greater comorbidity.

Coping and Responses to Stress

Research over the last two decades has attempted to more concretely define automatic and volitional responses to various stressful stimuli among clinical youth populations. Conner-Smith, Compas, Wadswith, Thomson & Saltzman (2000) developed the Responses to Stress Questionnaire (RSQ) initially as a conceptual model to include volitional efforts and involuntary responses to stressful life events or domains of stress. Specifically, the model emphasizes measuring a broad range of responses to stress that are voluntary or controlled coping responses and involuntary or automatic reactions. Adapting to various stressors involves cognitive, behavioral, emotional and physiological

responses. Involuntary responses, for example, may inhibit a child's ability to apply a voluntary coping response.

The model primarily attempts to distinguish the dimension of voluntary and involuntary responses to stress. Coping refers to the responses that are experienced as voluntary or controlled by the individual with conscious effort (Lazarus & Folkman, 1984). Voluntary coping attempts are within one's control, more behaviorally valanced, and are directed toward regulating cognitive, behavioral or physiological responses to a stressor. Involuntary responses to stress may include temperamentally based or conditioned reactions that are more cognitively valanced and may not be completely controlled, such as emotional and physiological arousal, intrusive thoughts and rumination, and emotional numbing (Conner-Smith et al., 2000).

Voluntary and involuntary responses to stress are further dichotomized by a second level of engagement or disengagement to a stressor (Tobin, Holroyd, Reynolds, & Wigal, 1989). Engagement responses are directed towards a stressor with approach behaviors (e.g., I try to think of different ways to change what is making me feel upset). Disengagement responses are directed away from a stressor and include avoidant behaviors (e.g., I try not to feel anything). Voluntary engagement and disengagement strategies are further distinguished by primary and secondary control strategies (Weisz, McCabe, Dennig, 1994). Primary control strategies are behaviorally focused on directly altering objective conditions (e.g., problem solving, emotional regulation). Secondary control coping strategies are more cognitively focused on adapting to the problem or situation (e.g., acceptance and cognitive restructuring).

All coping responses can be further broken down as adaptive or maladaptive. Adaptive coping responses are proactive coping behaviors that significantly reduce the negative impact of a stressor. Examples of these include problem solving, cognitive restructuring and acceptance. Contrastingly, maladaptive coping responses are ineffective coping behaviors or involuntary responses that fail to reduce the negative impact of a stressor and often exacerbate it. Examples of these include avoidance, rumination, and distraction. For the current study, the RSQ subscales of problem solving (PS), cognitive restructuring (CR), avoidance (AV) and rumination (RM) will be investigated. These subscales were chosen because each reflects an example of adaptive/maladaptive behavioral (primary) (PS, AV) and adaptive/maladaptive cognitive (secondary) (CR, RM) coping. Each of these constructs play a significant role in regulating emotion among youth.

Several coping and involuntary responses have been linked to anxiety and depression in youth. Problem solving (primary adaptive coping) is a behavioral coping strategy used for systematically identifying, troubleshooting and remediating a specific concern expressed by the patient. Research has shown that problem solving is particularly helpful among individuals struggling with Generalized Anxiety Disorder (Pawluk, Koerner, Tallon, & Antony, 2017) and has been an effective stand-alone treatment for anxiety and depression broadly (Zhang, Park, Sullivan, & Jing, 2018). Additionally, problem solving is a ubiquitous component among multiple evidenced-based protocols for treating anxiety and depression (Beidas, Podell, & Kendall, 2008; Weisz et al., 2005). Some theories hypothesize that problem solving produces secondary effects by increasing patient self-efficacy (Pawluk et al., 2017), and strengthens the effect of treatment.

Alternatively, deficits in problem solving have been shown to increase the risks of individuals developing depression and anxiety and may lead to other types of functional impairment (Masi, Favilla, Mucci, & Millepiedi, 2000; Scharfstein, Alfano, Beidel, & Wong, 2011).

Behavioral avoidance (primary maladaptive coping) refers to fear-inducing situations in which a person does not enter or leaves prematurely and has been identified as a significant maintaining factor among individuals with anxiety and depression (Chu, Skriner, & Staples, 2013). Avoidance and escape maintain fear-related behaviors through negative reinforcement when an individual experiences stress-/anxiety-provoking situations or stimuli. Avoidance subsequently maintains anxiety because it restricts inhibitory learning (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014), fails to activate fear networks (Foa & McNally, 1996) and prevents opportunities to disconfirm negative beliefs (Salkovkis, 1991) in lieu of new alternative choices that may be more adaptive. Likewise, core depressive symptoms (e.g., isolation, withdrawal, anhedonia) are an analogue to avoidance in that they provide short-term relief but interfere with an individual's ability to address problems in the long run.

Learning theory assigns several impairing functions to avoidance. First, avoidance provides immediate relief through negative reinforcement and escape behaviors. Second, behavioral avoidance denies individuals the opportunity for positive experiences and contributes to a deprived environment (Ferster, 1973; Jacobson, Martell, & Dimidjian, 2001). Third, avoidance may exacerbate ruminative and self-focused thinking due to the removal of intrinsically reinforcing positive stimuli. Finally, avoidance engenders problematic functioning when an individual is unable to engage in major life domains

(e.g., school, work, family). Other mechanisms have also been implicated as significant contributors to anxious and depressive symptomatology.

Cognitive restructuring (secondary adaptive coping) is a cognitive coping technique that involves systematically identifying negative automatic thoughts (e.g., "I'm no good," "this will never end") and challenging the accuracy of those thoughts using competing or disconfirmatory evidence. The effects of cognitive restructuring have been used to counter emotional dysregulation (Campbell-Sills & Barlow, 2007). A core component of cognitive restructuring is cognitive reappraisal, which involves modifying the meaning of a stimulus or context that precipitates a particular emotion (Goldin et al, 2017). Cognitive reappraisal has been shown to modify the emotional reactions to stressful or anxiety-provoking situations and lead to greater psychological flexibility and emotional well-being (Gross, 2007). This technique is also particularly helpful for informing the structure of individualized exposures.

Rumination (secondary maladaptive response to stressor) is defined as the tendency to repetitively analyze one's problems, concerns and feelings of distress without taking action to make positive change (Nolen-Hoeksema, 1991; Watkins, 2008) and has been linked to multiple psychopathologies (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Ehring & Watkins, 2008; Watkins, 2011). More broadly, rumination is a cognitive-emotion regulation process that has been linked to symptoms and diagnoses of major depression (Aldao et al., 2010) as well as symptoms of anxiety, substance abuse, alcohol abuse and eating disorders (Aldao et al., 2010; Caselli et al., 2010; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Among anxiety disorders, rumination is more associated with diagnoses of Social Anxiety Disorder (Mellings & Alden, 2000) and symptoms of

Generalized Anxiety Disorder (Watkins, 2009).

Rumination has been shown to amplify the reciprocal relationship between cognition and mood, such that increases in negative mood exacerbates negative cognitions. Relatedly, rumination interferes with individuals' ability to use adaptive coping strategies. Problem-solving is particularly challenging, as higher rumination confers greater pessimistic thinking and difficulty abstracting alternate solutions (Donaldson & Lam, 2004; Lyubomirsky & Nolen-Hoeksema, 1995). This deficit in turn prevents problems from being remediated and leads to increased depressed mood and chronic stress (Pearson, Watkins, Mullan, & Moberly, 2010). Alternative coping approaches that include proactive responses, rather than reactive responses, have shown clinical benefits in regulating emotion among clinical youth samples.

Problem solving, avoidance, cognitive restructuring and rumination correspond to one or more components of Conner-Smith et al. (2000)'s factor sequence of responses to stress. This sequence is based off a multidimensional model of responses to stress (Compas, Connor, Osowiecki, & Welch, 1997; Compas, Connor, Saltzman, Thomsen, & Wadsworth, 1999). The model assesses a broad range of responses to stress including voluntary or controlled coping responses and involuntary or automatic reactions. Adaptation to stress requires cognitive, behavioral, emotional, and physiological responses, with involuntary responses capable of enabling or inhibiting a child's ability to apply voluntary coping responses. Of note, the effectiveness of the response cannot be separated from the actual stressor. Likewise, no specified responses to stress are assumed to be universally helpful or detrimental across situations. Identifying coping and

responses to stress that are related to comorbidity remains an area necessitating further exploration.

Coping and Comorbidity

Investigations into the relationship between coping responses and comorbidity are limited and varied, particularly in terms of how coping is defined (Compas et al., 2014). This has prompted the development of more reliable measures of coping and responses to stressful stimuli (Conner-Smith et al., 2000). Studies utilizing the Responses to Stress Questionnaire (RSQ) as a measure of coping and responses to stress have found that cognitive reappraisal/restructuring was associated with lower symptoms of anxiety and depression (Compas et al., 2014). In another study by Andriotti et al. (2013), secondary control coping was negatively associated with anxiety and depression symptoms. Similarly, Bettis et al. (2016) found that secondary control coping was a transdiagnostic correlate of symptoms of anxiety and depression in youth. That is, more adaptive secondary coping was negatively associated with comorbid anxiety and depression. These findings suggest that secondary coping may have a unique relationship to comorbidity. Beyond these few studies, however, the literature is limited on the how coping influences, or is influenced by, comorbid anxiety and depression. Collectively, these limited findings establish a link between specific types of coping and comorbidity.

Evidence that Coping and Affect Interact with Each other and Existing Gaps

Although coping (Wright, Banerjee, Hoek, Rieffe, & Novin, 2010) and affective processes (Laurent, Joiner Jr, & Catanzaro, 2011) have been extensively studied within clinical youth populations, little research has examined the relationship between both processes concurrently. In fact, examination of each construct has been sparse, as much

of the existing literature has focused on grouping both coping and affect together under the umbrella of emotion regulation (Compas et al., 2017). The relationship between affective traits and coping responses to stress have never been jointly investigated within the context of comorbidity. More specifically, research is limited as to whether emotion (expressed through positive and negative affective responses) uniquely transacts with coping styles to predict comorbid anxiety and depression. Understanding the relationship between how coping and response strategies interact with affect can enhance our understanding of processes that are associated with comorbid anxiety-depression. Ultimately, this information helps identify mechanisms that may assist in personalized treatment planning or establishing public health programming (e.g., consumer education, establishment of socio-emotional common core programs in schools), helping to identify early problems to prevent more disruptive long-term developmental outcomes.

Socioeconomic Status and Comorbidity

Literature on link between depression (Gilman, Kawachi, Fitzmaurice, & Buka, 2002; Miech and Shanahan, 2000; Roy-Byrne, Joesch, Wang, & Kessler, 2009; Twenge and Nolen-Hoeksema, 2002, Zimmerman and Katon, 2005), anxiety (Roy-Byrne et al., 2009; Lemstra et al., 2008), and SES is well-established. Individuals from lower income and lower education backgrounds are up to twice as likely to develop depression compared to their higher income and more highly educated peers. Likewise, lower SES predicts poorer depression treatment outcomes compared to individuals reporting higher income (Falconnier, 2010). Individuals from racial minority groups are also more likely to have higher anxiety and poorer treatment outcomes (Roy-Byrne et al. 2009). Among youth, lower SES is associated with greater anxiety and depression, along with poorer

educational development (Lemstra et al., 2008). Despite the evidence for lower SES's association with anxiety and depression individually, there is a dearth of information on the relationship between comorbid anxiety-depression and SES. Few studies have concretely studied the relationship of this unique group exclusively, which prompts the need for further investigations.

Anxiety and Depression Symptoms and Comorbidity

Dimensional approaches to assessing for anxiety and depression support the frequent cooccurrence of symptoms across development (Seeley et al., 2011). A substantial body of evidence also exists supporting the cooccurrence of these symptoms independently in youth, indicating that anxiety and depression represent similar yet distinct disorders (van Lang, Ferdinand, Oldehinkel, Ormel & Verhulst, 2005). High levels of comorbidity and symptom cooccurrence suggest that symptoms may share common correlates. Identifying shared and non-shared correlates would enhance our understanding of where symptoms of anxiety and depression converge and diverge. This distinction is especially important when assessing for comorbidity. Clinical severity symptoms are measured separately from binary diagnostic labels. As such, precisely determining whether comorbidity is a function of other independent predictors requires that anxiety and depression symptoms must be controlled for. Doing so establishes that comorbid anxiety and depression is indeed being predicted by affect and coping and are not just a function of clinical severity.

Current Study

The proposed study seeks to better understand whether comorbid anxiety-depression can be predicted as a function of specific coping styles, affective traits,

anxiety/depression symptoms, while controlling for demographics, using established literature as a guideline. First, high scores on NA and low scores on PA were hypothesized to significantly predict comorbid anxiety-depression while controlling for age, gender and SES, compared to the anxiety-only group. Second, higher scores on PS and CS and lower scores on AV and RM were hypothesized to be significantly related to comorbid anxiety-depression while controlling for age, gender and SES. That is, poorer PS and CS and more severe AV and RM were expected predict comorbidity compared to the anxiety-only group. Third, lower SES was hypothesized to be significant in predicting comorbidity, while controlling for affect, coping and anxiety and depression symptoms. That is, lower income families were expected to predict comorbid anxiety-depression anticipating significant main effects from specific coping and affective traits while controlling for age, and gender.

Accordingly, analyses will be conducted in a two-phase approach. In the first phase, analyses will establish which affective traits, coping responses and demographics have significant main effects in predicting comorbid anxiety-depression. In the second phase, analyses will include a depression and anxiety symptoms as a fourth variable.

II. Method

Participants

This non-experimental, cross-sectional study will utilize pre-treatment data collected from a clinical youth population (N = 181; ages 7-17 years; M=11.997; S.D.= 2.504; girls = 89) meeting criteria for an anxiety-disorder only (n = 104), and comorbid anxiety-depression (n = 77). Youth in the anxiety-disorder only group were permitted to meet multiple diagnoses for anxiety disorders. All youth assented to study participation and all parents provided written and verbal consent. The racial make-up of the population consisted of 72% Caucasian, 7.27% African American, 6.9% Asian American, 10.34% Latino and 3.44% who identified as Other. Socioeconomic status ranged from 1 (less than $5,000.00 annually) to 10 (greater than $150,000 annually) (M=$66,000.00, S.D.=$14,030).

Measures

Demographics. All treatment-seeking families provided basic demographic information. Age, total household income and gender will be included as covariates. Total household income was coded using a 0 to 10 scale (e.g., 0 = under $5,000, 10 = over $150,000) and gender was dummy coded (e.g., 0 = male, 1 = female).

The Anxiety and Related Disorders Interview Schedule for DSM-IV - Child and Parent Versions (ADIS-IV-C/P; Silverman & Albano, 2000). The ADIS is a semi-structured diagnostic interview consisting of independent but comparable parent and child interviews. Psychometrics for the ADIS-IV have shown good interviewer reliability (e.g., k=.98, parent interview; k=.93, child interview; Silverman & Nelles, 1988), retest reliability (e.g., r=.76, parent interview; Silverman & Eisen, 1992), sensitivity to

treatment effects (e.g., Albano, DiBartolo, Heimberg, & Barlow; Kendall et al., 1997) and interrater reliability (e.g., k=.92, principal diagnosis; k=.8-1.0, individual anxiety disorder, k=.65-.67, comorbid disorders; Lyneham, Abbot & Rapee, 2007). The child and parent interviews are conducted individually to derive parent-reported, child-reported, and composite (parent and child) diagnoses.

Affect and Arousal Scale (AFARS; Chorpita, Daleiden, Moffitt, Yim, & Umemoto, 2000). The AFARS is a 27-item child self-report measure of negative affect (NA), positive affect (PA) and physiological hyperarousal (PH). Youth respond to each item by indicating how often they have felt a particular way (e.g., "little things bother me") within the last week using a 4-point scale ranging from 0 (never true) to 3 (always true). Composite scores for each subscale range from 0 to 27. Psychometrics have demonstrated good internal validity and reliability among youth (e.g., α=.80, NA; α =.77, PA; α =.81, PHF; Chorpita et al., 2000).

Responses to Stress Questionnaire (RSQ; Conner-Smith, Compas, Wadswith, Thomson & Saltzman, 2000). The RSQ is a 57-item child self-report measure of six broadband (e.g., primary control engagement coping, secondary control engagement coping, primary control disengagement coping, secondary control disengagement coping, involuntary engagement, involuntary disengagement) and 19 narrow-band subscales assessing engagement coping, disengagement coping and involuntary responding. Primary Control Engagement Coping (PCEC) includes problem solving, emotion regulation, emotional expression narrowband scales. Secondary Control Engagement Coping (SCEC) includes positive thinking, cognitive restructuring and acceptance narrowband scales. Primary Control Disengagement Coping (PCDC) includes avoidance

and denial narrowband scales. Secondary Control Disengagement Coping (SCDC) includes wishful thinking and distraction narrowband scales. Involuntary Engagement (IE) includes rumination, intrusive thoughts, physiological arousal, emotional arousal and involuntary action narrowband scales. Involuntary Disengagement (ID) includes emotional numbing, cognitive interference, inaction and escape narrowband subscales. Each narrowband subscale (e.g., rumination, emotion regulation) is a composite sum of three individual items from the measure. Youth rate each item by indicating how often they engage in that type of thinking, behavior or feeling (e.g., "I get really jumpy when I feel upset") within the last month using a 4-point scale ranging from 1 (Not at all) to 4 (Always true). The RSQ has demonstrated good internal consistency (e.g., PCEC, α=.82; PCDC, α=.71; PCDC, α=.72; SCDC, α=.80 IE, α=.78; ID α=.85) and test-retest reliability (e.g., PCEC, α=.76; PCDC, α=.75; PCDC, α=.70; SCDC, α=.70; IE, α=.71; ID α=.74) among youth (Yao et al., 2010). These subscales will be used: avoidance, rumination, cognitive restructuring, and problem solving.

Revised Children's Anxiety and Depression Scale; Parent/Youth (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000). The RCADS is a 47-item scale, rated on a 4-point (Never to Always) scale, whose items correspond closely to DSM-IV-TR symptoms. The RCADS incorporates six subscales (e.g., SAD, SP, OCD, PD, GAD and MDD). Subscales demonstrate good factorial validity (associated with major anxiety and depressive disorders), internal consistency (SAD, α=.76; SP, α=.82; OCD, α=.73; PD, α=.79; GAD, α=.77; MDD, α=.76), one-week test-retest reliability, and good convergent and discriminant validity with established internalizing and externalizing symptom measures (Chorpita et al., 2000).

Procedure

Treatment-seeking families called a university specialty clinic and completed a no-cost diagnostic interview (ADIS-C/P, Silverman & Albano, 2000). Children/parents completed questionnaires as part of a larger intake assessment. ADIS-C/P interviews were conducted by psychology doctoral students trained to criterion. Informed consent was obtained from all study participants after being explained potential risks and benefits to treatment. Data were collected as part of a naturalistic, open trial treatment study from 2005 through 2017. All procedures were approved by the Rutgers Institutional Review Board.

III. Results

Data Cleaning and Assumptions

Missing values of all cases (38.7%, 70/181) were handled by using multiple imputation in Statistical Package for Social Sciences (SPSS). In this procedure, missing data are imputed by regression analyses using available baseline data. This regression analysis was then repeated five times and aggregated into a pooled output. Data were examined for normality violations using standard practice (Field, 2009). The Hosmer-Lemeshow test (Hosmer & Lemeshow, 1980) was conducted to determine goodness-of-fit. The test is based upon dividing the sample up according to their predicted probabilities and forming them into groups. High *p* values were reported from each iteration, including the pooled group, indicating a strong model fit. Independence of errors was assumed and data were not related.

Descriptive Statistics

One hundred and four participants met criteria for an anxiety-only diagnosis and 77 met criteria for comorbid anxiety-depression. The Anxiety/Comorbid score, coded 0 (no comorbidity) or 1 (comorbidity present) from N=181 participants, was .43 ± .50 (mean ± standard deviation). Age ranged from 7-17 years (M=11.997, S.D.=2.504) and socioeconomic status ranged from 1 (less than $5,000.00 annually) to 10 (greater than $150,000 annually) (M=$66,000.00, S.D.=$14,030).

Step 1: Hierarchical Binary Logistic Regression 1—Demographics, Affect and Coping

A hierarchical binary logistic regression was performed to assess covariate-adjusted prediction of comorbidity (no/yes) from youth demographics, affect and coping

(see Table 1). Comorbidity (no/yes) served as the dependent binary variable. Demographics (age, gender, socioeconomic status) were entered in block 1, affect (positive affect, negative affect, physiological hyperarousal) entered in block 2, and Demographics (age, gender, socioeconomic status) were entered in block 1, affect (positive affect, negative affect, physiological hyperarousal) entered in block 2, and coping (rumination, avoidance, cognitive restructuring, problem solving) in block 3. Block 1 accounted for 11.3% variance, block 2 19.4% variance, and block 3 25.2% variance. In the final model, significant effects were found for socioeconomic status (B = -0.17, OR = 0.85, p = 0.02) and problem solving (B = -0.23, OR = 0.8, p = 0.01) in predicting the Anxiety/Comorbid score.

Step 2: Hierarchical Binary Logistic Regression 2—Anxiety and Depression Symptoms, Demographics, Affect and Coping

A hierarchical binary logistic regression was performed to repeat analyses from Step 1, but to add anxiety and depressive symptoms to rule out the possibility that clinical severity alone was accounting for prediction of comorbidity (Table 2). Anxiety and depression symptoms were entered as block 1, demographics (age, gender, socioeconomic status) in block 2, affect (positive affect, negative affect, physiological hyperarousal) in block 3, and coping (rumination, avoidance, cognitive restructuring, problem solving) in block 4. Block 1 accounted for 26.4% variance, block 2, 33% variance, and block 3, 35 % variance. In the final model, a significant effect was found for depression symptoms ($p < .001$), socioeconomic status ($p = .01$) and problem solving ($p < .05$) in predicting comorbidity.

Discussion

This study sought to determine whether symptoms of depression and anxiety, coping and affect among anxious and depressed youth were predictive of comorbidity. In our first analysis, youth demonstrating greater problem solving (PS) skills or who were from higher income (SES) families were less likely to have comorbid anxiety-depression compared to children with poorer PS skills or from low SES families. A second set of analyses confirmed that problem PS and SES continued to predict comorbidity, even after controlling for anxiety and depression. This suggested that PS and SES are important in predicting diagnostically complex cases, beyond clinical severity alone.

Problem Solving

The present study offers findings to suggest that lower PS coping may put youth at greater risk for comorbid anxiety-depression. That is, youth who are less proficient in PS at baseline have a greater chance of meeting criteria for comorbid depression compared to youth who have greater strengths within this domain. These results are consistent with some findings about the predictive effects of PS on comorbid anxiety-depression among youth (Hoek, Schuurmans, Koot, & Cuijpers, 2009; Joiner Jr, Voelz, & Rudd, 2001). In the previous two studies, investigators examined the predictive effects of PS on youth and comorbid anxiety depression symptoms and diagnoses. Results were mixed, as Joiner et al. (2001) showed significant improvement in symptoms and diagnosis relative to WL control, but Hoek et al. (2009) showed no difference.

PS was negatively associated with comorbidity, whether controlling for anxiety/depressive symptoms or not. These findings suggest the PS is a critical skill that may help buffer youth from depression, or more complex clinical profiles, in general.

Specifically, PS may be more associated with youth depression than anxiety due to the syndrome's direct inhibition of goal-oriented behavior (i.e., anhedonia, avoidance, rumination). Deficits within this domain would likely precipitate depression symptoms. Efforts to strengthen PS in treatment may help youth combat depression by facilitating the generation of competing evidence that is incompatible with depressogenic schemas, which result in success experiences. This would be consistent with the results found in Joiner et al. (2001).

Socioeconomic Status

The current study found that SES was predictive of comorbidity while controlling for anxiety and depression symptoms, affect and coping. These findings lend further support to the existing literature that higher SES may be protective against comorbid and complex clinical presentations and that youth from lower SES backgrounds are generally worse off. The link between SES and comorbidity also suggests that youth lacking access to specific resources may be at higher risk for presenting with multiple disorders. For example, Mclaughlin et al. (2011) found that financial hardship was associated with elevated risk of initial disorder onset across multiple disorder classes—including anxiety and depression—and across multiple stages of development. Similarly, low parental education was associated with higher severity among youth mood, anxiety and other behavioral disorders. Although ameliorating components of SES is not a viable or traditionally prescribed treatment target, it is important for clinicians to be aware of under-resourced youth, which may indicate the need for more intensive services. For example, such information may be utilized by clinicians to anticipate treatment undermining factors related to income inequities (i.e., caregiver having less time to check

in with clinician due to work commitments, caregiver being unable to afford treatment, caregiver requiring more psychoeducation related to youth's treatment).

Depression Symptoms

Depression symptoms were also positively associated with comorbidity, while controlling for affect, coping and demographics. These findings suggest that depression symptoms alone may be an early indicator of potential comorbid anxiety and depression. Cummings, et al., (2014) describe a similar finding using a multiple pathways model for the onset and course of anxiety and depression. Specifically, one pathway includes youth with a diathesis for depression (elevated depression symptoms) leading to anxiety symptoms and diagnoses, but not necessarily depression diagnoses. Alternatively, Keenan et al., (2009) found that subdiagnostic symptoms of depression were more predictive of future depression compared to anxiety symptoms. These findings suggest that depression is often present at the symptom level but alludes detection at the diagnostic level. Therefore, youth meeting criteria for an anxiety disorder only may also present with subthreshold depression symptoms that are missed during a diagnostic interview. Youth exhibiting greater depression symptoms, relative to anxiety, appear to be at higher risk for comorbidity than anxious youth who also meet criteria for a depressive disorder (Merikangas & Avenevoli, 2002; Ollendick, et al., 2005). This is not surprising given the type of comorbidity under investigation is depressive disorders. Nonetheless, it does provide an early index for clinicians to use to identify clients potentially at risk for mood disorders. For example, administering an anxiety and depression symptom measure may be a quicker, more practical means of identifying youth in need of more intensive services compared to administering a full diagnostic

module. Indeed, this approach may save time and money in both fully-resourced and under-resourced settings.

We hypothesized that high negative affect (NA) and low positive affect (PA) would both be predictive of comorbidity, however, all affective predictors failed to reach significance in the final model. These results contradict two of our hypotheses, including well-established literature (Andreotti et al. 2013; Chorpita et al., 2000), that higher NA and low PA would be a strong predictor of comorbid anxiety-depression relative to physiological hyperarousal (PH). It is possible that our sample was not adequately powered enough to detect an effect of affect on comorbidity.

Limitations

First, our analysis only compared a comorbid anxiety-depression group to an anxiety-only group. A depression-only group would have been included in the analysis, however, the sample size was insufficient to establish adequate power and thus would have prevented us from making reliable interpretations. Including a depression group may have helped both determine whether transdiagnostic mechanisms were present and if our independent variables exhibited a unique predictive relationship with the "pure" depression group. Second, it would have been useful to include a non-clinical control group in order to determine whether the effect of the independent variables was not due to environmental factors. Third, symptoms and affective factors were assessed solely with youth self-report, leading to possible shared method variance, however, diagnostic status was assessed using clinician evaluation following youth and caregiver interviews.

Conclusion and Future Directions

The current study demonstrated that PS, SES and depression symptoms share a unique link with comorbidity while controlling for other covariates. These findings were consistent with the extant literature but offer new discoveries that highlight the importance of PS coping in predicting depression comorbidity. They also emphasize the role of family resources in influencing clinical complexity. Collectively, these findings may enhance the effectiveness of existing EBTs and broaden our understanding comorbid anxiety-depression psychopathology.

Problem-solving's negative relationship with comorbidity suggests that it may be an especially critical skill to cultivate and reinforce in treatment. Real world clinical implications for these findings would be custom-tailored PS components—within existing EBTs—that are calibrated to youths' depression symptoms. Specifically, additional time and sessions may be dedicated to improving PS skills for youth who possess PS deficits and are presenting with greater depression symptoms.

Likewise, SES and its negative relationship to comorbidity suggests that families with lower income, and perhaps lower education, are at higher risk for having children develop comorbid anxiety-depression. Clinicians could use this information to build in additional supports and resources into treatment for families under greater financial hardship. For example, it may be fruitful for clinicians to offer more parent sessions that focus on providing support for their child's mental illness and promote follow-through on treatment objectives. This would be in addition to providing updates about treatment progress and goals.

Depression symptoms may be a stronger indictor of comorbidity relative to anxiety symptoms given that they tend to be present at subclinical levels when anxiety is the principal diagnosis. As a result, those symptoms often fail to meet clinically significant diagnostic thresholds and may become lost when considering the entire clinical profile. Assessing for depression symptoms, in addition to assessing for diagnosis, may provide deeper insight into treatment planning for higher risk youth. An example of this may be tailoring treatment to target more transdiagnostic processes, such as avoidance (Chu et al. 2016) and rumination (McLaughlin & Nolen-Hoeksema, 2011) when depression symptoms are higher.

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Appendix

Table 1

Comorbidity Predicted as a Function of Demographics, Affect and Coping

Measure	B	S.E.	*P* Value	O.R.	95% C.I.
Gender	.32	.36	.37	.73	.36-1.46
Age	.14	.08	.06	1.16	1-1.34
Socioeconomic Status	-.17	.07	.03*	.85	.73-.98
Physiological Hyperarousal	.05	.06	.39	1.05	.94-1.17
Negative Affect	.09	.05	.09	1.09	.99-1.21
Positive Affect	-.04	.04	.29	.96	.87-1.04
Problem Solving	-2.22	.09	.01*	.8	.68-.95
Cognitive Restructuring	.14	1.0	.17	1.15	.94-1.4
Avoidance	.11	.09	.22	1.12	.93-1.35
Rumination	.03	.08	.67	1.03	.89-1.21

Table 2

Comorbidity Predicted as a Function of, Demographics, Affect, Coping and Anxiety and Depression Symptoms

Measure	B	S.E.	*P* Value	O.R.	95% C.I.
Gender	-.27	.38	.48	.77	.37-1.6
Age	.13	.08	.10	1.14	.98-1.34
Socioeconomic Status	-.22	.08	.01*	.81	.69-.95
Physiological Hyperarousal	.03	.07	.65	1.03	.91-1.17
Negative Affect	.04	.06	.49	1.04	.93-1.18
Positive Affect	-.02	.05	.64	.98	.9-1.07
Problem Solving	-.19	1.0	.05*	.83	.69-1.0
Cognitive Restructuring	.08	.11	.47	1.09	.87-1.36
Avoidance	.10	.10	.34	1.11	.9-1.36
Rumination	-.03	.09	.75	.97	.82-1.15
Depression Symptoms	.21	.05	<.001*	1.24	1.12-1.37
Anxiety Symptoms	-.03	.02	.07	.97	.94-1.0