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Comorbidity of Anxiety and Depression in Children and Adolescents: 20 Years After

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Brady and Kendall (1992) concluded that although anxiety and depression in youths are meaningfully linked, there are important distinctions, and additional research is needed. Since then, studies of anxiety–depression comorbidity in youths have increased exponentially. Following a discussion of comorbidity, we review existing conceptual models and propose a multiple pathways model to anxiety–depression comorbidity. Pathway 1 describes youths with a diathesis for anxiety, with subsequent comorbid depression resulting from anxiety-related impairment. Pathway 2 refers to youths with a shared diathesis for anxiety and depression, who may experience both disorders simultaneously. Pathway 3 describes youths with a diathesis for depression, with subsequent comorbid anxiety resulting from depression-related impairment. Additionally, shared and stratified risk factors contribute to the development of the comorbid disorder, either by interacting with disorder-related impairment or by predicting the simultaneous development of the disorders. Our review addresses descriptive and developmental factors, gender differences, suicidality, assessments, and treatment-outcome research as they relate to comorbid anxiety and depression and to our proposed pathways. Research since 1992 indicates that comorbidity varies depending on the specific anxiety disorder, with Pathway 1 describing youths with either social phobia or separation anxiety disorder and subsequent depression, Pathway 2 applying to youths with coprimary generalized anxiety disorder and depression, and Pathway 3 including depressed youths with subsequent social phobia. The need to test the proposed multiple pathways model and to examine (a) developmental change and (b) specific anxiety disorders is highlighted.

Keywords: comorbidity, anxiety, depression, children, adolescents

Anxiety and depression are common in youths (Chavira, Stein, Bailey, & Stein, 2004; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003), and each is associated with substantial functional impairment (e.g., Kendall et al., 2010) and future mental health problems (e.g., substance use, bipolar disorders; Lopez, Turner, & Saavedra, 2005; Weissman et al., 1999). Anxiety and depression often co-occur (i.e., Axelson & Birmaher, 2001; Essau, 2008) and their comorbidity is common in children and adolescents (Garber & Weersing, 2010).

Among youths with depression,[1] anxiety disorders are the most common comorbid mental health disorders, with comorbidity estimates ranging from 15% to 75% (Angold, Costello, & Erkanli, 1999; Avenevoli, Stolar, Dierker, & Merikangas, 2001; Yorbik, Birmaher, Axelson, Williamson, & Ryan, 2004). In youths with anxiety disorders, rates of comorbid depressive disorders are generally lower, ranging from 10% to 15% (Angold et al., 1999; Axelson & Birmaher, 2001; Costello et al., 2003). Garber and Weersing (2010) suggested several explanations for this imbalance. First, anxiety-disordered youths may present with subsyndromal depressive symptoms (Van Voorhees, Melkonian, Marko, Humensky, & Fogel, 2010) that are not captured by diagnostic interviews. Second, and important to this review, studies have combined anxiety disorders that are heterogeneous (e.g., panic disorder; social phobia), which obfuscates the relationship between specific anxiety disorders and depression. Third, anxiety has an earlier age of onset (e.g., Fichter, Quadflieg, Fischer, & Kohlboeck, 2010) and is overall more prevalent in childhood than depression, whereas depression is more prevalent in adolescence (Cohen, Cohen, & Brooke, 1993; Woodward & Fergusson, 2001); thus, the degree of comorbidity may vary based on age. Fourth, many studies have not differentiated between concurrent comorbidity (both disorders occurring at the same time) and sequential comorbidity (one disorder preceding or predicting the other).

Brady and Kendall (1992), in this journal, reviewed the research on the comorbidity of anxiety and depression in youths and concluded that although anxiety and depression are meaningfully linked, there are important distinctions, and additional research was needed to further understand these differences. Since that review, the number of studies on the topic has increased exponentially. For Brady and Kendall, a PsycINFO search of the phrases,

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[1] Throughout the article, the term *depression* is used to refer to symptoms and diagnoses of persistent depressive disorder (previously dysthymic disorder) and major depressive disorder.

"depression," "anxiety," and "comorbidity" among children and adolescents yielded 37 journal articles published between 1972 and 1992. The same search yielded over 650 journal articles published between 1992 and 2012. Given the information now available, and the fact that the field continues to struggle to understand the distinctive and overlapping features of these disorders (Belzer & Schneier, 2004), we updated the 1992 review. We examined the full literature, but cite illustrative studies. We considered all studies that advanced knowledge on the relationship between depression and specific anxiety disorders (i.e., generalized anxiety disorder, GAD; separation anxiety disorder, SAD; social phobia, SoP) in children and adolescents, particularly those regarding theoretical models.

We focused on GAD, SAD, and SoP (now social anxiety disorder) because they are commonly occurring anxiety disorders in youths (Beesdo, Knappe, & Pine, 2009; Benjamin, Beidas, Comer, Puliafico, & Kendall, 2011; Essau, Conradt, & Petermann, 1999) and their comorbidity with depression has been extensively studied. Other childhood anxiety disorders, such as panic disorder and specific phobia, differ from GAD, SAD, and SoP. We refer interested readers to studies of comorbidity among individuals with specific phobia and panic disorder (e.g., Burstein, Ameli-Grillon, & Merikangas, 2011; Choy, Feyer, & Goodwin, 2007; Roy-Byrne et al., 2000).

Although several theoretical models describe the co-occurrence of anxiety and depression in adults (e.g., Alloy, 1991; Alloy, Kelly, Mineka, & Clements, 1990; Barlow, 1991; Barlow, Chorpita, & Turovksy, 1996; Chorpita, Albano, & Barlow, 1998; Clark & Watson, 2006; Mineka, Watson, & Clark, 1998; Watson, 2005), there is a need for models that address their development in youths. The identification of developmental pathways, precursors, and protective factors has implications for prevention of further comorbidity and related impairment and complications to treatment (Silk, Davis, McMakin, Dahl, & Forbes, 2012). We hold a developmental psychopathology perspective (E. Cummings, Davies, & Campbell, 2000: Drabick & Kendall, 2010), considering principles of equifinality (different pathways or risk factors may lead to the same outcome) and multifinality (the same risk factor may lead to different outcomes) in examining the interactive processes that may lead to the emergence of psychopathology (Cicchetti & Toth, 2009). These principles apply to mood and anxiety disorders in youths, as they are commonly comorbid and may share similar risk and protective factors (Epkins & Heckler, 2011).

The advances since 1992 in our understanding of the comorbidity of anxiety and depression in youths are described and detailed in sections on theoretical models, descriptive and developmental variables, gender differences, suicidality, assessment, and treatment response, with a summary (see Table 1) provided to highlight both key findings and an agenda for future research. We review the literature relative to our proposed multiple pathways model. Pathway 1 describes youths with a diathesis for anxiety, with subsequent comorbid depression resulting from anxiety-related impairment. Pathway 2 refers to youths with a shared diathesis for anxiety and depression who experience both disorders simultaneously. Pathway 3 describes youths with a diathesis for depression, with subsequent comorbid anxiety resulting from depression-related impairment. Additionally, we suggest shared and stratified (i.e., unshared) risk factors (Klein & Riso, 1993) that play a role, either through interacting with disorder-related impairment or predicting the simultaneous development of the disorders. As evident following the review, these pathways are differentially applicable to specific disorders.

Comorbidity

Comorbidity typically refers to the presence of two or more distinct, co-occurring disorders in one person simultaneously (Klein & Riso, 1993). Brady and Kendall (1992) noted that the comorbidity of anxiety and depression may have been a result of the diagnostic system at that time, the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM–III–R*; American Psychiatric Association, 1987). Comorbidity within the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM–IV–TR*; American Psychiatric Association, 2000) continues, with some suggesting that comorbidity is "largely the product of a nosological system that classifies mental disorders categorically, presupposing discrete diagnostic entities or disease states" (Belzer & Schneier, 2004, p. 297). Indeed, comorbidity may be nothing more than an artifact of an imperfect diagnostic system. Although its merits are debatable, the literature considers the comorbidity of depression and anxiety within the context of the diagnostic system. The majority of studies since Brady and Kendall's (1992) review used *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) criteria. Although there were few changes to criteria for anxiety and depressive disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM–5*; American Psychiatric Association, 2013), changes in the required duration of symptoms (e.g., "fear, anxiety, *or* avoidance is persistent, *typically* lasting for 6 months or more") might slightly elevate comorbidity rates.

Since Brady and Kendall (1992), concepts of comorbidity have developed, with distinctions made between epidemiologic and clinical comorbidity (Kraemer, 1995). Epidemiologic comorbidity refers to the nonindependence of, or association between, two disorders in a population and is relevant to efforts to define the boundaries of each disorder and identify their etiologies. Clinical comorbidity refers to the presence of two disorders in one person and is relevant to discussions of prognosis, course, and treatment response. Clinical comorbidity can occur without epidemiologic comorbidity and vice versa. Although studies have examined epidemiologic comorbidity by assessing lifetime prevalence using mixed-age samples (vs. the simultaneous presence of more than one disorder), this approach has been criticized for inflating estimates of the association between disorders (Kraemer, Wilson, & Hayward, 2006). For this review, we highlight clinical comorbidity. However, we also report studies that describe the broader relationship between depression and anxiety (i.e., anxiety leading to subsequent depression and vice versa). In the multiple pathways model, comorbidity refers to the simultaneous presence of depression and anxiety, although onset of one disorder may precede the other.

Individuals who are generally comorbid (i.e., those with more than one concurrent diagnosis) have been found more likely than single-diagnosis individuals to use mental health services and to report more suicide attempts, periods of disability, greater life dissatisfaction, less job satisfaction, and less social stability (D. L. Newman, Moffitt, Caspi, & Silva, 1998). Comorbid individuals have more physical health problems and are less likely to attend

Table 1
Key Findings and Areas for Future Research Regarding Comorbid Depression and Anxiety in Youths

Topic	Current theories/hypotheses/ models	Key points	Recommended research agenda
Nature of comorbidity	Comorbid anxiety–depression is typically worse than anxiety alone but not worse than depression alone.	Comorbid depression in anxiety samples is less common than comorbid anxiety in depressed samples.	Address the nature of depressive comorbidity across ages and in specific anxiety disorders
		Among comorbid youths, severely depressed youths demonstrate severe anxiety, whereas severely anxious youths may not necessarily exhibit severe depression.	
		Comorbid youths are more impaired than purely anxious youths but no more impaired than purely depressed youths.	
		Comorbid depression may interfere with CBT for anxiety, but comorbid anxiety does not consistently interfere with CBT for depression.	
Theoretical models	Tripartite model: NA is responsible for anxiety–depression comorbidity.	Some studies support NA as a shared general distress factor (NA) in anxiety and depression.	Examine applicability of NA, PA, and PH across ages and in specific anxiety disorders
		PH and PA may not be specific to anxiety and depression, respectively.	
		PH may be linked to panic disorder.	
		Support for the tripartite model is inconsistent across developmental levels.	
		Variance explained by NA differs across anxiety disorders.	
	BIS/BAS: BIS responsible for anxiety–depression comorbidity.	High BIS related to anxiety.	Consider prevention informed by behavioral inhibition in young children
		Low BAS related to depression and may reflect anhedonia, explaining mixed findings.	
		BIS may also be related to depression.	
SAD and depression	SAD increases the risk of depression through common vulnerabilities and/or SAD-related impairment.	SAD has genetic links to panic, and depression is often present in youths with panic disorder.	Longitudinal studies that span the timeframe within which SAD and depression emerge
		SAD is related to parental depression.	Specifically address and examine panic disorder
			Determine whether SAD is associated with onset of depression in the short run as a result of SAD-related impairment
SoP and depression	SoP-related impairment leads to comorbid depression, particularly in adolescence due to heightened importance of peers.	SoP more commonly onsets before depression than vice versa.	Identify SoP before depression, and the potential of secondary prevention
		Core risk factors (e.g., genetics) interact with interpersonal risk factors (e.g., loneliness), leading to depression among SoP youths.	Examine symptom levels of both disorders
		Interpersonal risk factors with research support include peer alienation, peer victimization, avoidance of expressing emotion, and poor friendship quality.	Delineate interpersonal factors that link SoP and depression

Table 1 (*continued*)

Topic	Current theories/hypotheses/ models	Key points	Recommended research agenda
GAD and depression	GAD and depression are highly related but separate disorders.	GAD and MDD share genetic risk factors to a greater extent than with other anxiety disorders.	Investigate where comorbidity is an artifact of the diagnostic criteria
		GAD and MDD cross-predict each other more strongly than each disorder predicts itself over time.	Investigate criteria that distinguish the two disorders
		MDD and GAD do not reliably respond to the same medications.	Identify and study cases where GAD does not lead to depression and vice versa
		Stressful life events are more strongly related to MDD than to GAD.	
		High GAD–MDD comorbidity may be due to symptom overlap in the diagnostic criteria.	
Gender differences	Gender differences in cognitive style and relationship processes increase risk for both depression and anxiety.	Depression is more prevalent among adolescent girls than boys.	Use multi-informant approaches to lessen the impact of social desirability
	Depression symptoms may differ by gender, with girls showing more symptoms (e.g., somatic) that overlap with anxiety.	Anxiety is more prevalent among girls than boys in community samples but is comparable in diagnosed samples.	Given differences, consider gender more specifically in treatment approaches
		Rumination is more common among girls than boys and is associated with anxiety, depression, and comorbid anxiety–depression.	Observational studies to inform hypothesis-driven research
		Depression may develop secondary to anxiety in boys.	
Suicidality	Suicidality in anxious youths is a result of comorbid depressive symptoms.	There is an increased rate of suicidality among depressed youths; findings among anxious youths are mixed.	Differentiate across components/types of suicidality
		Other constructs related to anxiety and depression (e.g., emotion dysregulation, distress tolerance) may explain increased suicidality in anxious youths.	Examine associations of suicidality with specific anxiety disorders
			Consider the possibility that suicidality reported by anxious youths without depression may reflect intrusive thoughts

Note. CBT = cognitive behavioral therapy; NA = negative affect; PH = physiological hyperarousal; PA = positive affect; BIS = behavioral inhibition system; BAS = behavioral activation system; SAD = separation anxiety disorder; SoP = social phobia; GAD = generalized anxiety disorder; MDD = major depressive disorder.

college (D. L. Newman et al., 1998). Among adolescents, general comorbidity has been associated with greater overall impairment (Karlsson et al., 2006), academic difficulties, and suicide attempts (Lewinsohn, Rohde, & Seeley, 1995). Comorbidity (in general) may influence the clinical presentation of anxiety-disordered youths more than that of other disorders; Lewinsohn et al. (1995) found that comorbidity had the greatest effect on mental health treatment utilization, suicide attempts, academic problems, and conflict with parents for anxiety-disordered youths, compared to youths with other disorders.

Brady and Kendall (1992) found rates of overlap of anxiety and depression ranging from 16% to 62%, a large range that was attributed to the samples studied (J. C. Anderson, Williams, McGee, & Silva, 1987; Bernstein & Garfinkel, 1986; Carey, Finch, & Imm, 1989; Costello et al., 1988; Kovacs, Gatsonis, Paulaukas, & Richards, 1989; C. C. Strauss, Last, Hersen, & Kazdin, 1988). The studies reviewed (i.e., Carey et al., 1989; Mitchell, McCauley, Burke, & Moss, 1988; C. C. Strauss et al., 1988) reported greater levels of impairment among comorbidly depressed-anxious youths versus single diagnosis youths. However, methodological limitations and the paucity of studies limited the conclusions drawn in 1992.

Research has since confirmed that comorbid anxiety and depression are associated with greater impairment and symptom severity related to the primary diagnosis, and even more so when anxiety is the primary concern (e.g., Fichter et al., 2010; Franco, Saavedra, & Silverman, 2007; Masi, Favilla, Mucci, & Millepiedi, 2000; O'Neil, Podell, Benjamin, & Kendall, 2010; C. C. Strauss et al., 1988). O'Neil et al. (2010) found that among youths with a principal anxiety diagnosis, comorbid depression was associated with lower global functioning, poorer family functioning, more severe social anxiety, and greater severity of specific depressive symptoms (i.e., negative mood, ineffectiveness, and anhedonia, but not interpersonal problems or negative self-esteem).

Research on the impact of comorbid anxiety disorders on depression is mixed (Guberman & Manassis, 2011; Rohde, Clarke, Lewinsohn, Seeley, & Kaufman, 2001) and suggests that comorbid anxiety may not be associated with worsened functioning among depressed youths. For example, Guberman and Manassis (2011) found that anxious youths with comorbid depression evidenced greater anxiety severity than youths with anxiety alone, but depressed youths with comorbid anxiety did not evidence greater depression severity or family dysfunction than youths with depression alone. This finding is consistent with the notion that severely anxious youths are more prone to comorbid depression than non-anxiety-disordered youths, but not necessarily more severe depression. However, severely depressed youths appear to consistently demonstrate severe anxiety. A latent class analysis indicated that the vast majority of adolescents with high levels of depression also had high levels of anxiety but that the reverse was not true: adolescents with high levels of anxiety had either high or low levels of depression (Ferdinand, de Nijs, van Lier, & Verhulst, 2005). These results are in line with previous conclusions (Brady & Kendall, 1992) that depressed youths tend to report high levels of both depression and anxiety, whereas anxious youths tend to report high levels of anxiety but relatively low levels of depression.

Existing Theoretical Models

Several models have been proposed to explain the frequent co-occurrence of depression and anxiety. Seligman and Ollendick (1998) offered four explanations: (a) comorbidity between anxiety and depression in youths is due to overlap in definitions; (b) anxiety and depression represent two indicators of a single construct; (c) comorbidity is due to overlap in risk factors; (d) anxiety causes or puts youths at risk for depression. Similarly, Klein and Riso (1993) offered explanations for comorbidity, including comorbidity by chance, population stratification (i.e., separate risk factors associated with each disorder tend to co-occur in subsets of the population), one disorder serving as a risk factor for the other, and overlapping risk factors between the two disorders.

At the same time, the etiology of anxiety and depression may not be best captured by a single model (Issler, Sant'Anna, Kapczinski, & Lafer, 2004), and it is likely that more than one of Seligman and Ollendick's (1998) or Klein and Riso's (1993) explanations apply to the complex relationship between depression and the various anxiety disorders. The multiple pathways model builds on these explanations by applying them to comorbid depression and *specific* anxiety disorders in youths.

There is some evidence for distinct factors of depression and anxiety that are stable across time and gender (Boylan, Miller, Vaillancourt, & Szatmari, 2011) but the co-occurrence of disorders may be explained by a shared factor, negative affectivity (NA; Murphy, Marelich, & Hoffman, 2000; Watson, Clark, & Carey, 1988), particularly for depression and GAD (Trosper, Whitton, Brown, & Pincus, 2012). NA may involve "transdiagnostic factors," including neuroticism (Griffith et al., 2010), rumination (Hankin, 2008b; McLaughlin & Nolen-Hoeksema, 2011), and intolerance of uncertainty (Mahoney & McEvoy, 2012). Models that strive to explain comorbidities and have been tested include the tripartite model (Clark & Watson, 1991) and behavioral inhibition/activation (Gray, 1987, 1991). Newer models, including the quadripartite model (Watson, 2009), have not yet been empirically tested.

The Tripartite Model

The tripartite model (Clark & Watson, 1991) states that anxiety and depression share a general NA factor, whereas physiological hyperarousal (PH) is specific to anxiety and low positive affect (PA) is specific to depression. The shared NA factor links sadness/depression and anxiety/fearfulness, thus accounting for comorbidity between anxiety and depression. Brady and Kendall (1992) noted only a few studies supporting its application in adults (Watson et al., 1988) and youths (Finch, Lipovksy, & Casat, 1989). Since then, numerous studies have further examined the tripartite model in youths. Several studies have demonstrated support for the tripartite model in youths (Cannon & Weems, 2006; Cole, Truglio, & Peeke, 1997; Joiner, Catanzaro, & Laurent, 1996; Tully, Zajac, & Venning, 2009). Olino, Klein, Lewinsohn, Rohde, and Seeley (2008), for example, compared one- and three-factor models in adolescents followed up to 30 years of age. The one-factor model was an "internalizing" factor, whereas the three-factor model included depressive, anxious, and common factors. The three-factor model fit best, supporting the idea that depression and anxiety can be explained by a combination of shared and unique factors. Lonigan, Phillips, and Hooe's (2003) longitudinal study of youths in Grades 4–11 found that NA and PA were stable over time, related to each other, and provided a good fit to children's self-reported affect. Studying outpatient adolescents, Steer, Clark, Kumar, and Beck (2008) provided support for NA with their finding that a second-order factor (representing general symptom distress) best explained the common variance between anxiety and depression measures.

Other studies have suggested the tripartite model to be an imperfect fit to anxiety and depression. For instance, PH and PA may not be specific to anxiety and/or depression. An examination of parent- and child-reported anxiety and automatic arousal measures found PH in both anxiety and depression (Greaves-Lord et al., 2007). De Bolle, Decuyper, De Clercq, and De Fruyt (2010) found that PH and PA did not account for the unique aspects of anxiety and depression and that PH (rather than NA) was the shared factor. In a study of perceived arousal and hypothalamic-pituitary-adrenal (HPA)-axis functioning (an indicant of PH), perceived arousal was related to both depression and anxiety. Additionally, reactive HPA-axis functioning (high PH) was reduced in depressed youths (Dieleman, van der Ende, Verhulst, & Huizink, 2010). These findings suggest that PH is not unique to anxiety.

Still other studies have offered partial support for the tripartite model: PA, NA, and PH are not independent of each other. In a study that sampled elementary and high school boys and girls (Jacques & Mash, 2004), NA correlated positively with both depression and anxiety (consistent with the model). Contrary to the model, PA was negatively correlated with both depression and anxiety, and PH was positively correlated with both depression and anxiety. The tripartite model best fit data from high school girls. Using an elementary school sample, Lee and Rebok (2002) found that PA and NA scales were negatively correlated, cross-sectionally and longitudinally. In a study that sampled African American, urban youths in grades six through nine, the model was said to be supported, but the dimensions were highly correlated

(Gaylord-Harden, Elmore, Campbell, & Wethington, 2011; Lambert, McCreary, Joiner, Schmidt, & Ialongo, 2004).

Consistent with Jacques and Mash's (2004) findings that the tripartite model fit best for older girls, Ollendick, Seligman, Goza, Byrd, and Singh (2003) found that the two-factor model fit data from boys and younger (fourth grade) children. Anxiety and depression may be best differentiated among older girls. Nevertheless, findings examining whether the tripartite model applies across age groups are mixed. Cole et al. (1997) factor analyzed anxiety and depression measures administered to sixth graders and found both shared and unique factors, consistent with the model. However, constructs of anxiety and depression were indistinguishable in a third grade sample. Contrary to Cole et al.'s findings and the notion that depression and anxiety may differentiate with increasing age, Turner and Barrett (2003) found that a tripartite model provided a good fit to data across third, sixth, and ninth grade age groups.

One of our main concerns, that anxiety disorders are diverse (not one anxiety disorder), merits consideration in terms of the tripartite model and whether it applies to the various anxiety disorders (E. R. Anderson & Hope, 2008). In a sample of 1,578 school children, NA was positively related to anxiety and depression, and PA was negatively correlated with depression (as expected). However, PH was positively related to panic only (Chorpita, 2002). In relation to social anxiety, E. R. Anderson, Veed, Inderbitzen-Nolan, and Hansen (2010) found that low PA, high NA, and high PH characterized adolescents diagnosed with SoP, whereas adolescents with elevated symptoms of social anxiety (but not meeting diagnostic criteria) evidenced only high PH and high NA.

Thus, evaluations of the tripartite model have had varying results, possibly due to methodological and sampling differences. Nonetheless, a few conclusions can be drawn. First, anxiety and depression are difficult to discriminate in community samples with available measures but are more easily differentiated as symptomatology reaches diagnosable levels. Second, the model may not apply across developmental levels. Third, the model does not fully account for heterogeneity among anxiety disorders, with suggestions that PH only applies to panic. Research and a modified model (Chorpita, Plummer, & Moffitt, 2000; Laurent, Catanzaro, & Joiner, 2004) are needed to understand the relationship between anxiety and depression in youths. It is essential that such work recognize the heterogeneity among the anxiety disorders and call researchers to determine the unique components of each anxiety disorder.

Within the adult literature, concerns about the tripartite model are that PH does not capture the heterogeneity among the anxiety disorders (e.g., PH is found in panic, but not other disorders; Brown, Chorpita, & Barlow, 1998); and that the anxiety disorders show varying levels of overlap with depression. Alternative models, for adult disorders (i.e., Barlow, 1991; Barlow et al., 1996; Chorpita et al., 1998; Clark & Watson, 2006; Mineka et al., 1998; Watson, 2005), offer three factors that include (a) anxious apprehension, (b) fear, and (c) depression; with anxious apprehension (general distress or NA factor) a nonspecific component of both anxiety and depression, fear (autonomic arousal and panic) a specific feature of anxiety, and depression (anhedonia, low PA) a specific feature of depression. Mineka et al.'s (1998) model accounts for heterogeneity among the anxiety disorders by specifying that PH is a feature of panic only. Each anxiety disorder is proposed to have a unique component, though not yet identified, and the amount of variance attributed to general distress differs across specific anxiety disorders. Watson (2005) distinguished between fear and distress in his disorder-based model for *DSM*. Distress disorders (i.e., GAD, PTSD, MDD, & DD) contain a large amount of the NA component and the model implies that GAD and PTSD are more related to depression than they are to fear disorders (i.e., panic disorder, agoraphobia, SoP, and specific phobia). Watson's symptom-level quadripartite model (Watson, 2009) includes (a) high distress symptoms with limited specificity to depression versus anxiety (e.g., dysphoric mood in MDD), (b) high distress symptoms with greater specificity (e.g., suicidality in MDD), (c) low distress symptoms with greater specificity (e.g., psychomotor retardation in MDD), (d) low distress symptoms with limited specificity (e.g., insomnia in MDD).

These newer models, from the adult literature, improve upon the tripartite model by integrating research findings supporting the heterogeneity among the anxiety disorders and calling for researchers to determine the unique components of the anxiety disorders. Such work should include longitudinal studies and would inform child, adolescent, and adult literatures.

The Behavioral Inhibition/Behavioral Activation Systems

Gray (1987, 1991) proposed a model of the behavioral activation system (BAS) and the behavioral inhibition system (BIS), two brain systems that regulate approach and withdrawal behavior, respectively, in response to the environment. Activity in the left prefrontal cortex is hypothesized to be related to BAS functioning, and activity in the right prefrontal cortex is hypothesized to be related to BIS functioning, with the BIS responding to conditioned aversive stimuli (Davidson, 1998). Over time, the model was revised to include the fight-flight system (FFS; Gray, 1987), which refers to unconditioned aversive stimuli eliciting fight or flight responses. Gray and McNaughton (2000) revised the BIS/BAS (see Bijttebier, Beck, Claes, & Vandereycken, 2009; Corr, 2008), postulating that the BAS is responsive to all positive stimuli. The FFS was renamed the fight/flight/freeze system (FFFS) and proposed to manage reactions to aversive stimuli (i.e., fear), with the BIS managing goal conflicts (i.e., situations involving both reward and threat; anxiety). Although the BIS is proposed to underlie all anxiety disorders, the FFFS is proposed to relate specifically to panic disorder, SoP, and specific phobia (Kimbrel, 2008; Zinbarg & Yoon, 2008). Theory proposes that the BAS and BIS function independently; however, Corr (2002) suggested that the BIS and BAS jointly influence behavior.

BIS and BAS are considered relevant for understanding behavior in response to the environment. Individuals with BIS/BAS functioning at either extreme are at risk for psychopathology (Pickering & Gray, 1999). BIS activity is said to cause feelings of anxiety that lead to withdrawal behavior, whereas BAS activity produces impulsive behavior with little attention to negative consequences. Children with high levels of behavioral inhibition show lower thresholds for fear responding to unfamiliar stimuli (Kagan, Reznick, & Snidman, 1987). By definition, undercontrolled children have high BAS sensitivity, and inhibited children have high BIS sensitivity, although BIS applies across situations and behavioral inhibition applies to novel stimuli. In one study, Caspi, Moffitt, Newman, and Silva

(1996) classified children at 3 years of age and later assessed them via diagnostic interviews at age 21. Undercontrolled (i.e., defined as children who are irritable, impulsive, labile) and inhibited (i.e., defined as children who are socially reticent; distinct from behaviorally inhibited) children at age 3 were most at risk for psychopathology and suicide attempts 18 years later. Inhibited children were more likely than well-adjusted children to have depression but not anxiety.

Data on BIS/BAS were unavailable at the time of Brady and Kendall's (1992) review. A relationship between BIS sensitivity and anxiety symptoms has since been established (i.e., Campbell-Sills, Liverant, & Brown, 2004). Also, low BAS sensitivity has been linked to depression in clinical (i.e., Kasch, Rottenberg, Arnow, & Gottlib, 2002) and community samples (Beevers & Meyer, 2002) of adults. Among MDD adults, self-reported BAS sensitivity scores predicted depression at 6-month follow-up (McFarland, Shankman, Tenke, Bruder, & Klein, 2006). However, other studies have not found a relationship between BAS sensitivity and depression (i.e., S. L. Johnson, Turner, & Iwata, 2003; Jorm et al., 1998), and some have found a relationship between BIS sensitivity and symptoms of depression (i.e., Beevers & Meyer, 2002; Campbell-Sills et al., 2004). It can be argued that BIS sensitivity is a common factor to anxiety and depression, whereas BAS hypoactivity is specific to depression with anhedonic symptoms (Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil, 2007). Impaired reward sensitivity (i.e., anhedonia) is said to differentiate depression from anxiety (Pizzagalli, Jahn, & O'Shea, 2005).

In line with findings that high BIS sensitivity is associated with both anxiety and depression (Merikangas, Swendsen, Preisig, & Chazan, 1998; Muris, Meesters, de Kanter, & Timmerman, 2005), the model may apply to their comorbidity, although little research exists on this topic in youth samples. High BIS and low BAS may be a developmental precursor to comorbid anxiety and depression, and behavioral inhibition could represent a general risk factor for both depression and anxiety (e.g., S. L. Johnson et al., 2003; Schofield, Coles, & Gibb, 2009). Little research exists on this topic, particularly in child samples. In one study, Coplan, Wilson, Frohlick, and Zelenskis (2006) found that child reports of BIS sensitivity were associated with increased depressive symptoms, greater negative affect and social anxiety, and less positive reports of subjective well-being. BAS sensitivity was negatively associated with internalizing problems but was not related to subjective well-being. Children identified as avoidant (high in BIS and low in BAS based on median splits), reported more depressive symptoms, negative affect, and social anxiety; and less PA and subjective well-being than children who were not avoidant (Coplan et al., 2006). In a study of adults with SoP, retrospective reported BIS in childhood was associated with both increased severity of adult SoP symptoms and depressive comorbidity (Rotge et al., 2011). Although direct tests of BIS/BAS to anxiety–depression comorbidity in youths are required, this line of research is promising in light of the early identification of at-risk youths. We also note that revisions to BIS/BAS theory resemble the tripartite model, with BIS and BAS loading on the same factors as negative and positive affectivity, respectively (Jorm et al., 1998; Zinbarg & Yoon, 2008).

A Multiple Pathways Model

The multiple pathways model is consistent with models that emphasize the heterogeneity among the anxiety disorders (Brown et al., 1998) and with the distinction between "fear" and "distress" anxiety disorders (Watson, 2005), with distress disorders containing more overlap with depression. Additionally, we view these disorders from both categorical and dimensional perspectives, as the examination of symptoms versus diagnoses can change conclusions regarding the order of onset of anxiety and depressive disorders (e.g., Bubier & Drabick, 2009). For instance, a child may experience subsyndromal depressive symptoms, which precede and then exacerbate anxiety symptoms, contributing to a diagnosable anxiety disorder. The anxiety symptoms, in turn, exacerbate current levels of depressive symptoms, leading to a diagnosable depressive disorder. At the symptom level, depression preceded anxiety; at the level of disorder, anxiety preceded depression. Thus, dimensional approaches are necessary to capture subtle interactions between symptoms that would have otherwise been missed. Since Brady and Kendall's (1992) review, promising dimensional models have been suggested for youths with externalizing disorders (Walton, Ormel, & Krueger, 2011), internalizing symptoms in general (Markon, 2010), and anxiety and depression (Lahey et al., 2008). Further, item response modeling has been used to identify the range of severity assessed by various symptom measures, allowing the selection of instruments to be tailored to specific research questions (e.g., Olino et al., 2012).

By considering the developmental processes that lead to depression, anxiety, and their comorbidity, we learn more about the specific risk and protective factors. As opposed to examining already-comorbid individuals (adults), the multiple pathways model calls attention to the interactive processes associated with depression-anxiety onset among youths. We ask, "What differentiates those individuals progressing from anxiety to depression, depression to anxiety, or experiencing the two disorders simultaneously?" And, "What differentiates anxious-only and depressed-only individuals from depressed-anxious individuals?" (Cicchetti & Toth, 2009). We maintain that anxiety and depression are separate but meaningfully related constructs. We propose that anxiety and depression differ in their "relatedness" according to three different pathways to anxiety–depression comorbidity. Consistent with previous findings, our multiple pathways model acknowledges that comorbid anxiety and depression is not a singular construct; rather, this comorbidity differs based on the type of anxiety disorder. Additionally, the multiple pathways model addresses the developmental progression of one disorder to the other. The pathways propose a diathesis to primary depression, primary anxiety, or coprimary anxiety–depression. For those with a diathesis for only anxiety or depression, comorbidity arises from both direct and indirect influences, in line with equifinality principles (Cicchetti & Toth, 2009). These influences include risk factors resulting from impairment caused by the initial disorder. Additionally, shared and unique (but stratified) risk factors contribute to depression-anxiety comorbidity (Klein & Riso, 1993). These are a combination of social/environmental (e.g., school, peer, familial-related), biological (e.g., adrenogenic exhaustion), and behavioral (e.g., avoidance) factors. Other variables that are associated with anxiety (or depression) can also influence later depression (or anxiety).

Pathway 1 describes youths with a general diathesis (temperamental, biological, environmental) to anxiety. When anxiety is left untreated, anxiety-related impairment becomes a risk factor for comorbid depression. This anxiety-related impairment might interact with shared risk factors for the two disorders (e.g., cognitive biases, negative affectivity), leading to the onset of depression. For instance, Silk et al. (2012) suggested that sensitivity to social evaluative threat and alterations in reward processing are two potential vulnerabilities of anxious youths that could be associated with subsequent depression. From a diagnostic perspective, anxiety disorders that are characteristic of this pathway include SoP and SAD. Anxiety in Pathway 1 tends to be severe, rather than moderate or mild, whereas depression is mild in its presentation (relative to primary depression). For these youths, anxiety is the primary concern. Nonetheless, the presence of comorbid depression worsens the overall adjustment of these youths. Within this pathway, anxiety and depression represent separate but related conditions, with less overlap between depression (distress) and anxiety (fear-related disorders; Watson, 2005).

Pathway 2 consists of youths with the greatest shared diathesis for anxiety and depression. These individuals experience anxious and depressed feelings simultaneously in response to the same trigger. Youths experiencing comorbidity via this pathway most often show depression-GAD comorbidity. Anxiety is severe, whereas depression is moderate (relative to other forms of depression). These youths differ regarding whether anxiety or depression is identified as the principal concern (they are coprimary). Within this pathway, anxiety and depression have a high degree of overlap that is linked to worry, which is distinct from fear (e.g., Hofmann et al., 2005). This overlap may be, in part, an artifact of a diagnostic system in which anxiety and depression have shared symptoms. Given that anxiety and depression can be experienced simultaneously and their overlap is high (e.g., through the general experience of distress/anxious apprehension; Mineka et al., 1998; Watson, 2005), Pathway 2 includes many shared risk factors of anxiety and depression. Despite the shared diathesis, GAD and depression may not onset at the same time. As with all the pathways, it is possible that expression of depression and anxiety is influenced by age-related changes (e.g., pubertal processes; see reviews by Hyde, Mezulis, & Abramson, 2008; Reardon, Leen-Feldner, & Hayward, 2009). Additionally, reliance on categorical assessment may inflate the time interval between the onset of GAD and depression. We consider this pathway to contain the most shared risk factors.

The third pathway includes youths with a diathesis for depression, for whom depression-related impairment eventually becomes a source of anxiety. Such impairment can include poor social skills, peer victimization, or isolation resulting from depressive symptoms. Indirect influences link depression with subsequent anxiety among these youths. Although not yet extensively examined, the third pathway is the least common type of anxiety–depression comorbidity in youths. As such, we cannot rule out the extent to which comorbidity between these disorders occurs by chance (Klein & Riso, 1993), but there is some evidence of depression-related impairment linked to anxiety (e.g., Rudolph, Hammen, & Burge, 1994). Depression-anxiety comorbidity via this pathway occurs in older adolescents (and adults) and involves SoP and GAD. As with Pathway 1, anxiety and depression are separate but related conditions, with the anxiety more in line with a "fear" presentation (Watson, 2005).

Finally, the comorbidity of anxiety and depression may be explained, in part, by shared genes. In the context of the multiple pathways model, this shared diathesis is part of Pathway 2, which involves greater degrees of overlap between anxiety and depression. Brady and Kendall (1992) reviewed family history and twin studies and concluded, cautiously, that familial genetic patterns *distinguished* depression and anxiety. Since then, some genetic studies that have capitalized on technological advances point to *shared genetics* in anxiety and depression, which may explain their frequent comorbidity (see Axelson & Birmaher, 2001; Franić, Middeldorp, Dolan, Ligthart, & Bossmsa, 2010; Middeldorp, Cath, Van Dyck, & Boomsma, 2005; Williamson, Forbes, Dahl, & Ryan, 2005), although the findings from two studies suggest independent transmission of depressive and anxiety disorders (i.e., Avenevoli et al., 2001; Klein, Lewinsohn, Rohde, Seeley, & Shankman, 2003). Discrepant findings regarding the genetic transmission of anxiety/depressive disorders may be due to sampling or methodological differences (e.g., specific anxiety disorders included, early versus later-onset MDD, diagnosed cases versus population-based samples). Below, the multiple pathways model is applied to the research findings regarding anxiety–depression comorbidity.[2]

Descriptive and Developmental Variables

Longitudinal Research

Research findings suggested to Brady and Kendall (1992) that a temporal relationship existed between anxiety and depression, with anxiety generally preceding depression, but only one longitudinal study had been published at that time (Kovacs et al., 1989). An important advance in the last 20 years is the progress from cross-sectional and retrospective designs to prospective, longitudinal studies that sample community, high-risk, and diagnosed individuals, which have informed the multiple pathways model. Although there is some evidence that earlier depression predicts later anxiety among youths (consistent with the Pathway 3; Costello et al., 2003), the bulk of the data indicate that the onset of anxiety most often occurs before the onset of depression (consistent with Pathway 1; e.g., Avenevoli et al., 2001; Fichter et al., 2010; Kovacs et al., 1989). Further, comorbid anxiety (in particular, GAD and SoP) appears to be more prevalent among depressed youths with an earlier onset of depression (i.e., before age 15; Hammen, Brennan, Keenan-Miller, & Herr, 2008).

As our review makes salient, anxiety disorders are not unitary, and there is variation in the onset of specific anxiety disorders. Lewinsohn, Zinbarg, Seeley, Lewinsohn, and Sack (1997) found that specific phobia, SAD, overanxious disorder (currently GAD), and SoP more often preceded diagnosis of MDD, but that obsessive-compulsive disorder (OCD) and panic disorder were more likely to occur after the onset of depression. Essau (2003)

[2] Given that anxiety disorders in youths are highly comorbid (Kendall et al., 2010), the pathways are not mutually exclusive. It is possible, for example, that a child may have a shared diathesis for GAD and MDD and the risk for MDD is exacerbated by the onset of SoP as the child approaches adolescence, as anxiety-related impairment confers more risk for a depressive episode.

reported that SoP and specific phobias most often preceded depression but that agoraphobia, anxiety disorder not otherwise specified (NOS), and GAD occurred first only 50% of the time in their adolescent sample. Consistent with Pathway 1, SoP, in particular, has been found to precede depression for the majority of comorbidly anxious and depressed patients, with childhood and adolescent (vs. adult) onset of SoP (based on retrospective report) associated with greater severity of depression (Dalrymple & Zimmerman, 2011). Childhood (vs. adolescent) SoP onset was associated with a shorter time to MDD onset. Social anxiety in youths often goes along with poor social skills, low self-esteem, loneliness, and helplessness (Messer & Beidel, 1994; Rubin & Burgess, 2001) as well as cognitive vulnerabilities such as hopelessness (Gibb & Alloy, 2006), which predict later depression. Additionally, anxious adolescents have been found to engage in risky behavior (e.g., substance abuse or misconduct; possibly to gain acceptance from peers) that is associated with later depressive symptoms (Sears & Armstrong, 1998).

Anxiety that occurs first may leave the child more susceptible to depression (e.g., Avenevoli et al., 2001; Mathew, Pettit, Lewinsohn, Seeley, & Roberts, 2011). At the same time, research does not indicate a direct causal path from early anxiety to later depression among youths (Rice, van den Bree, & Thapar, 2004). Rather, a combination of factors associated with anxiety may lead to later depression. Anxiety's causal relationship to depression may be explained, in part, by cognitive models (i.e., Nolen-Hoeksema, 2000; Starr & Davila, 2012). Additionally, physiological models suggest that heightened arousal from sustained levels of anxiety may eventually cause the body to "shut down," or become depressed (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). In short, it is likely that a combination of biological, environmental, and cognitive factors associated with anxiety can contribute to later depression. Most longitudinal studies of community adolescents suggest that anxiety and depression are separate but related disorders. Among community adolescents, those identified as at risk for anxiety disorder (based on first-year anxiety symptoms) with high severity of depression or anxiety, were at risk for high severity of the other type of disorder (this did not hold for the low-risk group; Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2009). In a community sample, high rates of self- and parent-reported anxiety predicted self- and parent-reported depression over time (Cole, Peeke, Martin, Truglio, & Seroczynski, 1998). However, the reverse was not true; in fact, parents who reported that their child exhibited elevated depressive symptoms later reported decreased anxiety.

A longitudinal study of nonreferred girls suggested that *symptoms* of depression, SoP, and SAD were relatively stable and nonpredictive of each other from year to year. However, increases in levels of SoP in childhood did predict depressive *disorders* in early adolescence. Depressive symptoms were the strongest predictors of later depression, again suggesting that anxiety and depression can be separate but related (Keenan, Feng, Hipwell, & Klostermann, 2009). Olino, Klein, Lewinsohn, Rohde, and Seeley (2010) identified six distinct trajectories of anxiety and depressive symptoms among a community sample of adolescents followed until age 30, with each trajectory demonstrating a distinct relationship between anxiety and depression over time. Trajectories characterized by depressive disorders showed relatively low probabilities of anxiety disorders, whereas trajectories characterized by anxiety disorders showed relatively high probabilities of depressive disorders. Interestingly, depressive disorders that followed anxiety disorders tended to be less persistent than other forms of depression characterized in the study. It is important to note that this study followed individuals with relatively low rates of anxiety disorders, and changes in depressive/anxiety disorders were followed from adolescence (vs. childhood) to adulthood.

Finally, retrospective reporting of childhood sexual and physical abuse was associated with more severe anxiety symptoms and more frequent depressive comorbidity in an adult anxiety-disordered sample (Mancini, Van Amerigen, & Macmillian, 1995). Childhood trauma (i.e., retrospective reports of abuse or neglect) and recent life stressors were associated with the development of subsequent comorbidity (including depression) among anxious adults (de Graaf, Bijl, Ten Have, Beekman, & Vollebergh, 2004). However, comorbidity was relatively low (11%) over the 3-year period. In a longitudinal study of 816 youths, the combination of early exposure to stressors and a history of an anxiety disorder predicted increased depressive symptoms in response to low levels of current stress. The authors hypothesized that these variables interact to create a sensitivity/reactivity to stress, suggesting that anxiety disorder and stress sensitivity are markers of a vulnerability to depression that results from early exposure to adversity (Espejo et al., 2007). Hopelessness, on the other hand, may interact with stress to specifically predict depression (Gibb & Alloy, 2006; Hankin, 2008a).

It may be that depression and anxiety share a genetic pathway that is expressed differently depending on environmental stressors (Williamson et al., 2005). For example, anxiety disorders in youths are more strongly associated with parental overprotection and control, whereas depression in youths is more strongly associated with parental rejection and lack of warmth (Beesdo, Pine, Lieb, & Wittchen, 2010; Rapee, 1997). Additionally, stressful life events are often more closely linked to depression than anxiety (e.g., Pine, Cohen, Johnson, & Brook, 2002; S. C. Newman & Bland, 1994). Factors related to family and peers have also been studied in anxious and depressed youths, although the extent to which such interpersonal difficulties are causes versus consequences of these disorders remains unclear.

Specific Anxiety Disorders

Brady and Kendall (1992) did not encounter studies examining the relationship of depression to specific anxiety disorders in youths, but this area of research has greatly expanded since then, likely driven by the consensus that anxiety disorders are heterogeneous. In general, the number of types of fears experienced is associated with lifetime depression rates (Choy, Fyer, & Goodwin, 2007). Although co-occurring depressive symptoms are common across the anxiety disorders, the etiology of these relationships may differ by anxiety disorder. The research described already suggests mechanisms by which anxiety and depression could be related. It is possible that some anxiety disorders lead to increased rates of depressive disorders through their impact on the youth's environment (Pathway 1). For instance, avoidance of feared stimuli can lead to decreased engagement in pleasant activities and poorer interpersonal relationships, resulting in depression. At the same time, depression and other anxiety disorders may be associated through overlapping characteristics, such as cognitive style

(Pathway 2). Finally, one or more third variables (e.g., childhood adversity; genetics) could present a shared vulnerability to both depression and anxiety. These vulnerabilities interact with risk factors for depression related to anxiety-related impairment (or vice versa, Pathways 1 and 3), or they lead to the simultaneous comorbidity of anxiety and depression (Pathway 2). We suggest that each of these mechanisms varies by specific anxiety disorder, as research since the 1992 review indicates that depression-anxiety comorbidity varies depending on the specific anxiety disorder.

SAD and depression. Relatively little research has been devoted to the relationship between SAD and depression, perhaps because depression is more commonly comorbid with GAD and SoP (Verduin & Kendall, 2003). Some work suggests that persistent SAD can be a precursor to panic (Perugi et al., 1988; Savino et al., 1993), with which it may share a common genetic diathesis (Roberson-Nay, Eaves, Hettema, Kendler, & Silberg, 2012). In turn, panic disorder is associated with depression in youths and adults (Goodwin & Gotlib, 2004; Kessler et al., 1998; Roy-Byrne et al., 2000). Thus, children with SAD may be at risk for depression.

However, several studies with varying methodologies present mixed findings. A 7.4-year-follow-up of children with SAD showed that it was associated with later anxiety but not panic disorder/MDD specifically (Aschenbrand, Kendall, Webb, Safford, & Flannery-Schroeder, 2003). A study that used retrospective ratings of SAD also did not support a significant relationship with depression after adjusting for the effects of prior panic attacks (Hayward, Killen, Kramer, & Taylor, 2000). Foley, Pickles, Maes, Silberg, and Eaves (2004) reported that children with persistent SAD had a higher prevalence of depression at 18-month follow-up compared with children with transient SAD, after controlling for the presence of comorbid disorders. In another study, childhood SAD was a risk factor for depression within 1 year, but otherwise did not predict depressive symptoms/disorders (Keenan, Feng, et al., 2009). In line with this finding, Pine, Cohen, Gurley, Brook, and Ma (1998) found that SAD in adolescence was not significantly associated with depression in adulthood.

In contrast, Lewinsohn, Holm-Denoma, Small, Seeley, and Joiner (2008) found that adults who reported childhood SAD were more likely to have panic disorder and depressive disorders but not other anxiety disorders by age 30, after controlling for other disorders through age 19 (Lewinsohn et al., 2008). A study that followed offspring of adults with and without panic disorder and MDD found that childhood SAD increased risk for developing MDD (*odds ratio* = 3.2) but far less than it increased risk for developing panic disorder (*odds ratio* = 9.2) and agoraphobia (*odds ratio* = 9.1; Biederman et al., 2007). Inconsistencies in the findings may be due to the method and timing of assessments (e.g., relative to risk period for developing SAD or depression) and whether panic disorder is controlled.

Overall, SAD may be associated with increased risk for later depression (consistent with Pathway 1) but to a lesser degree than the other anxiety disorders. It is possible that increased risk for depression among youths with SAD is greatest in the short-term (i.e., closer in time to SAD). Consistent with Pathway 1, children with more severe forms of SAD are at greater risk for later depression based on SAD-related impairment (e.g., strain on peer and familial relationships, not participating in school and other activities). Future longitudinal research could examine whether panic symptoms mediate the relationship between SAD and depression. Additionally, studies finding that successful treatment of SAD prevents the onset of subsequent depression would be consistent with Pathway 1. Risk factors for depression that stem from SAD-related impairment include social isolation and withdrawal from activities, academic difficulties, and conflicted familial relationships. Studies that specifically test these risk factors would be informative and beneficial.

SoP and depression. In contrast to SAD, depression appears to be particularly linked to childhood SoP, especially the generalized subtype, both at the level of disorder (Chavira et al., 2004; O'Neil et al., 2010; Ranta, Kaltiala-Heino, Rantanen, & Marttunen, 2009) and symptoms (O'Neil et al., 2010; Tillfors, El-Khouri, Stein, & Trost, 2009). In a twin study, familial aggregation and common genetic factors were observed for major depressive disorder (MDD) and SoP (Mosing et al., 2009). Although shyness has not been related to increased levels of MDD among adolescents (Burstein et al., 2011), SoP in adolescence has predicted depression (i.e., onset in adolescence or early adulthood; Beesdo et al., 2007; Stein et al., 2001). Childhood-onset SoP may be associated with earlier onset of depression than adolescence-onset SoP (based on retrospective, self-report in treatment-seeking samples: Alpert et al., 1999; Dalrymple & Zimmerman, 2011). Likewise, childhood-onset MDD showed higher associations with comorbid SoP than adult-onset MDD (Alpert et al., 1999).

The co-occurrence of older age, severity, SoP, and depressed state was associated with lower rate of remission in the Child/Adolescent Anxiety Multimodal Study (CAMS; Ginsburg et al., 2011), and other studies have also linked comorbid mood problems and age to less favorable cognitive-behavioral therapy (CBT) outcomes among youths with SoP (Crawley, Beidas, Benjamin, Martin, & Kendall, 2008). It may be that depressed mood decreases motivation for change and/or makes it difficult to engage youths in treatment. Another possibility is that depression affects how youths with SoP cognitively process feedback during or following exposure to anxiety-provoking social situations, which appears to be a critical element of CBT (e.g., Peris, Compton, Piacentini, & the Child/Adolescent Anxiety Multimodal Study [CAMS] Research Group, 2013). If depression increases negative postevent processing (i.e., negatively valenced review of social situations in which inadequacies, mistakes, imperfections, and negative perceptions of the interaction are exaggerated; Rachman, Grüter-Andrew, & Shafran, 2000), exposure tasks may be less effective in producing new learning.

Among children followed longitudinally, those identified by their peers as anxious-solitary (i.e., interested in interacting with others but refrain due to anxiety) were likely to receive future diagnoses of SoP, dysthymic disorder, and MDD (Gazelle, Workman, & Allan, 2010). Interpersonal variables appear to play an important role in both disorders and may be a key link between depression and SoP in youths, particularly adolescents. Peer variables are salient in SoP, whereas family variables may be more salient to depression (Starr & Davila, 2008; Hutcherson & Epkins, 2009; H. S. Johnson, Inderbitzen-Nolan, & Schapman, 2005). At the same time, comorbid symptoms of SoP and depression among adolescent girls from a community sample were associated with increased peer and family alienation compared to girls with only symptoms of SoP or depression, although findings may have been affected by relatively low levels of psychopathology among par-

ticipants (Starr & Davilla, 2008). Girls' and mothers' reports of lower maternal acceptance and parental support were related to girls' depression but not SoP (controlling for depression) and girls' and mothers' reports of lower peer acceptance were related to girls' social anxiety but not depression (controlling for social anxiety; Hutcherson & Epkins, 2009). Peers can play a central role: early onset of depression can lead to poor social interactions and underdeveloped social skills (Rudolph et al., 1994), a path to SoP (Pathway 3). SoP has been found to be associated with peer victimization among adolescents (Ranta, Kaltiala-Heino, Rantanen, & Marttunen 2009), and youths with comorbid symptoms of depression and SoP experience peer victimization at even greater rates (Ranta, Kaltialia-Heino, Pelkonen, & Marttunen, 2009).

Cognitive biases, interpersonal styles, and coping styles link SoP and depression. Comorbid depression amplifies biases that are common in SoP (i.e., negative social events reflect negative self-characteristics; negative social events have long-term implications), whereas SoP alone has biases concerning negative evaluation by others (Wilson & Rapee, 2005). A study of late adolescents (undergraduates) found that interpersonal dependency, avoidance of expressing emotion, and lack of assertion were associated with symptoms of SoP, but only avoidance of expressing emotion was associated with depressive symptoms 1 year later (Grant, Beck, Farrow, & Davila, 2007).

Do coping strategies differentiate SoP and depression in youths? Within a community sample of school-aged children, six coping strategies were identified (Wright, Banerjee, Hoek, Rieffe, & Novin, 2010). Elevated depression symptoms were associated with decreased problem solving, distraction, and social support seeking, and increased externalizing (e.g., yelling or throwing something) over time. In contrast, elevated social anxiety symptoms were associated with increased social support seeking, distraction, and internalizing (e.g., worrying or feeling sorry for oneself), and decreased externalizing over time. Although, importantly, individual coping strategies did not predict later depression and/or anxiety, the findings allude to differential patterns of coping strategies in depression versus SoP.

The theoretical models can be applied to the links between SoP and depression. BIS/BAS theory suggests that behavioral inhibition (linked with social withdrawal; Rubin, Coplan, & Bowker, 2009) is more strongly related to social anxiety than to the other anxiety disorders (Chronis-Tuscano et al., 2009; Hirshfeld-Becker, 2010; Hirshfeld-Becker et al., 2007) and depression (Caspi et al., 1996). Tests of the tripartite model suggests that low PA is related to SoP in addition to depression (Clark & Watson, 2008; Chorpita, Plummer, & Moffitt, 2000; Dougherty et al., 2011), although more research is needed with youths (Epkins & Heckler, 2011). SoP most often precedes depression in youths (Pathway 1). The less common instance of depression preceding SoP may be attributed to poorly developed social skills due to early onset of depression and decreased motivation for social activities (Pathway 3). Future studies should examine interpersonal risk factors for depression as they relate to SoP. Based on data to date, interpersonal risk factors, namely, peer victimization, social withdrawal (due to anxiety), poor friendship quality, and maladaptive coping strategies are relevant to SoP as a precursor to depression (Pathway 1).

Appropriately, Epkins and Heckler (2011) used interpersonal risk as a model for the co-occurrence of social anxiety and depression in youths. Core risk factors (i.e., temperament; genetics; parental psychopathology) were proposed to interact with interpersonal risk factors centering on parents and peers (e.g., loneliness; maladaptive parenting; peer victimization) leading to depression. By emphasizing interpersonal variables as a pathway between SoP and depression, the model fits for a specific anxiety disorder. Even so, research linking core and interpersonal risk factors to SoP specifically (and not just anxiety in general) is lacking. Our Pathway 1 is consistent with Epkins and Heckler's findings. As with SAD, finding that successful treatment of SoP protects against onset of depression would be consistent with Pathway 1. Additionally, Pathway 1 suggests that youths with the situational subtype of SoP (who may be likely to demonstrate less impairment in interpersonal relationships than youths with generalized SoP) are considerably less likely to develop subsequent depression. Longitudinal studies of youths with risk factors for both depression and SoP would inform our understanding of the relationship of SoP and depression relative to Pathways 1 and 3.

GAD and MDD. Attention has been paid to whether GAD and MDD are separate disorders given the symptom overlap (i.e., insomnia; fatigue; difficulty concentrating). Longitudinal data suggest that GAD and MDD cross-predict each other more strongly than each disorder predicts itself over time (Copeland, Shanahan, Costello, & Angold, 2009; Moffitt, Caspi, et al., 2007; Moffitt, Harrington, et al., 2007; Pine et al., 1998). However, the pattern may not be consistent across developmental stages. In Copeland et al. (2009), only childhood depression predicted young adult GAD and only adolescent GAD predicted later depression. Further, childhood and adolescent GAD and MDD predicted different adult disorders, and young adult GAD and MDD were predicted by different childhood and adolescent disorders. The National Comorbidity Survey (N = 8,098 respondents, ages 15–54 years) found that nearly one third of all nonchance co-occurrences of GAD and a major depressive episode involved both disorders starting in the same year (Kessler et al., 2008). Within this study, the odds ratio for secondary major depression among individuals with GAD did not become higher with the passage of time since onset of GAD, contradicting the theory that MDD results from exhaustion in response to unremitting anxiety (Akiskal, 1985). According to Kessler et al. (2008), the cross-lagged associations between GAD and major depression are likely due to common causes and/or the effects of one disorder on the other. A recent study of the symptoms of adults with comorbid GAD-MDD versus MDD alone suggests that the high comorbidity rates are partly an artifact of the diagnostic system (Zbozinek et al., 2012).

According to meta-analyses by Hettema, Neale, and Kendler (2001) and Sullivan, Neale, and Kendler (2000), MDD and GAD each show modest familial aggregation and heritability. Family studies (Kendler, Davis, & Kessler, 1997; Mendlewicz, Papdimitriou, & Wilmotte, 1993; Reich, 1995; Skre, Onstad, Edvardsen, Torgersen, & Kringlen, 1994), twin studies (Kendler, 1996; Kendler, Gardner, Gatz, & Pedersen, 2007; Kendler, Neale, Kessler, Heath, & Eaves, 1992; Roy, Neale, Pedersen, Mathe, & Kendler, 1995), and transmission studies (which estimate prevalence rates in children whose parents are probands; Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002) indicate that MDD shares genetic risk factors with GAD, to a greater extent than with other anxiety disorders (Hettema, 2008). Using twin data, Kendler, Prescott, Myers, and Neale (2003) provided evidence that genetic risk pertains to internalizing and externalizing factors, with the inter-

nalizing factor further divided into "anxiety–misery" and "fear" factors. Anxiety–misery loads most strongly on GAD and MDD and "fear" loads most strongly on animal and situational phobias, with panic disorder falling between but closer to GAD and MDD.

Supporting the notion of separate disorders, GAD and MDD have been found to show neuroanatomical differences and distinct disruptions to neuroendocrine and neuropeptide systems (Martin & Nemeroff, 2010). Although both disorders respond to SSRIs, GAD but not MDD generally responds to anxiolytics (e.g., Schatzberg & Cole, 1978). There has been less research on environmental contributions to MDD and GAD specifically (vs. anxiety disorders generally). Stressful life events in adolescence predict adult MDD but not GAD (e.g., Pine et al., 2002). Adult studies suggest that stressful life events are related to both disorders but more strongly to MDD (e.g., S. C. Newman & Bland, 1994), with only partial overlap in the types of events preceding the onset of each disorder (e.g., Kendler, Hettema, Butera, Gardner, & Prescott, 2003).

Overall, findings support MDD as separate but more closely related to GAD than other anxiety disorders. Given that the median age of onset is much earlier for anxiety than depression (e.g., Kessler et al., 2005), it makes sense to think of these disorders as distinct in youths, although GAD typically does not precede depression in comorbid youths (e.g., Essau, 2003). We consider GAD–MDD comorbidity as exemplary of Pathway 2. In assessing GAD–MDD comorbidity, future studies need to attend to overlap in the diagnostic criteria. There are shared symptoms, but are there qualitative differences? For example, individuals with these diagnoses report difficulties with sleep and concentration, but are they experienced as different by GAD versus depressed individuals? Sleep interruptions may be due to worry in GAD versus restlessness in MDD. Diagnostic instruments lack detail and may need revision to address such symptom distinctions.

Gender Differences

Boy-girl differences, not reviewed in 1992 due to a dearth of published reports, have since received substantial attention. Not until early adolescence, when rates of depression in girls increase drastically, are gender differences in depression evident. At this time, depression occurs two to three times more often in girls than in boys (Hankin, 2009; Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006). Teen girls report higher levels of depression than boys, a finding that is amplified in clinical samples (i.e., Compas et al., 1997; De Bolle et al., 2010). Data on Age × Gender interactions for anxiety in youths are much less clear. Anxiety has been found to be more prevalent among girls in some community samples (Angold et al., 1999; Aune & Stiles, 2009; Axelson & Birmaher, 2001; Costello et al., 1996; Leikanger, Ingul, & Larsson, 2012) but not in diagnosed samples (i.e., Kendall et al., 2010). Girls report more fears than boys (Gullone, 2000), but they do not have higher rates of anxiety disorders (e.g., Kendall et al., 2010; Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008). There is some evidence of gender differences in temperamental fear as early as infancy (e.g., Halpern, Brand, & Malone, 2001). Although a meta-analysis suggested that these gender differences are negligible (Else-Quest, Hyde, Goldsmith, & Van Hulle, 2006), most of the studies had modest sample sizes and relied on parent-report measures of temperament. More recent studies using multiple informants have consistently shown significant gender differences in fear. Across three independent samples of preschool- and early elementary school-aged children, girls exhibited higher levels of fear than boys across maternal- and paternal-report measures as well as behavioral observations of temperament (Olino, Durbin, Klein, Hayden, & Dyson, 2013). In a twin study that used the same methods of assessment, girls showed higher levels of shyness than boys (Gagne, Miller, & Goldsmith, 2013). Evidence from the adult literature suggests that, compared with men, women are more likely to have comorbid anxiety-mood disorders (e.g., Angst, Merikangas, & Preisig, 1997).

Theories regarding gender discrepancies in anxiety and depression have been reviewed (Bell, Foster, & Mash, 2005; Hankin & Abramson, 2001; Hankin, Wetter, Cheely, & Oppenheimer, 2008; Rutter, Caspi, & Moffitt, 2003; Zahn-Waxler et al., 2006), and gender differences offer hints about the etiology of comorbid anxiety and depression. For instance, cognitive vulnerabilities such as rumination (more common among females) are common to depression and anxiety. McLaughlin and Nolen-Hoeksema (2011) found that rumination accounted for a large portion of the overlap between self-reported depression and anxiety symptoms among adolescents (and adults) and was also associated with elevated risk for the development of comorbid anxiety/depression symptoms over time. Corumination (excessive negative discussion of problems within a close relationship) is also common among adolescent girls (Rose, 2002) and is positively associated with symptoms of both depression and anxiety (Rose, Carlson, & Waller, 2007; Tompkins, Hockett, Abraibesh, & Witt, 2011), although negatively associated with social anxiety (Starr & Davila, 2009). Gender differences in relationship processes and interpersonal stressors, particularly in adolescence, may be linked with depression and anxiety, and warrant research (Rudolph, 2002).

Hankin (2009) supported a cognitive vulnerability-transactional stress model (Hankin & Abramson, 2001) among community youths: sex differences in depression and anxiety symptoms were partially explained by risk mechanisms, including rumination and a negative cognitive style, and exposure to stressors. Given that anxiety generally precedes depression, sex differences in anxiety have been hypothesized as a potential reason for sex differences in later depression (Zahn-Waxler, Shirtcliff, & Marceau, 2008). However, two retrospective reports (Parker & Hadzi-Pavlovic, 2001, 2004) suggested that female gender was independently related to subsequent depression when accounting for prior anxiety, although prior anxiety made a stronger contribution than gender (Parker & Hadzi-Pavlovic, 2001). Other studies suggest that the gender difference may be attributed to variations in the expression of depression. The "anxious" subtype of depression (i.e., high anxiety during mood episodes, negative self-evaluation, poor concentration) and "somatic" subtype of depression (i.e., sleep disturbance, pain symptoms) may be more prevalent among adult females than adult males (Halbreich & Kahn, 2007; Silverstein, 1999), but this finding has little support with youths (i.e., Nilzon & Palmerus, 1997).

Anxious boys, in some research, were more prone to developing later depression than girls (Gallerani, Garber, & Martin, 2010; Väänänen et al., 2011). Anxiety may be less socially acceptable for boys than girls, contributing to decreased social support and increased feelings of rejection.

Overall, the literature indicates that there are higher rates of anxiety, depression, and anxious-depressive comorbidity among

community samples of girls, particularly in adolescence. Boys and girls have comparable rates of anxiety disorders, but girls are more prone to common risk factors for anxiety and depression. Further research is needed to better understand biological (e.g., pubertal) as well as psychosocial factors that contribute to anxious-depressive comorbidity and gender differences over time. Within Pathways 1 and 3, gender differences can interact with risk factors associated with anxiety and depression comorbidity. Pathway 2 involves an anxious type of depression (two disorders are highly related), more often observed in females.

Suicidality

Youth suicide rates are a worldwide concern (Goldsmith, Pellmar, Kleinman, & Bunney, 2002). An increased rate of suicidality in depression, particularly MDD, has been well established (i.e., Liu et al., 2006). In contrast, the results of studies of the relationship between anxiety disorders and suicidality have been mixed, and not reviewed in 1992 due to the absence of data. In a recent study (O'Neil-Rodriguez & Kendall, 2012), 58% of youths diagnosed with an anxiety disorder reported suicidal ideation. Some studies have uncovered an association between anxiety and suicidality independent of depression (Boden, Fergusson, & Horwood, 2007; Foley, Goldston, Costello, & Angold, 2006; Ghaziuddin, King, Naylor, & Ghaziuddin, 2000; Goldston et al., 2009; Liu et al., 2006; Masi, Mucci, Favilla, & Millepiedi, 2001; O'Neil, Puleo, Benjamin, Podell, & Kendall, 2012; O'Neil-Rodriguez & Kendall, 2012), whereas a few others have not (Barbe et al., 2005; Esposito & Clum, 2002; Greene, Chorpita, & Aukahi, 2009; Ryan, Puig-Antich, Ambrosini, & Rabinovich, 1987; J. Strauss et al., 2000). It may be that emotion dysregulation (Garber, Braafladt, & Weiss, 1995; Keenan, Hipwell, Hinze, & Babinski, 2009; Silk, Steinberg, & Morris, 2003; Southam-Gerow & Kendall, 2000; Suveg, Hoffman, Zeman, & Thomassin, 2009) and distress tolerance (Daughters et al., 2009; Ellis, Fischer, & Beevers, 2010; Huang, Szabó, & Han, 2009; Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010) constructs related to both depression and anxiety, drive their associations with suicidality (Anestis, Bagge, Tulle, & Joiner, 2011; Nock & Mendes, 2008; Tamás et al., 2007; Zlotnick, Donaldson, Spirito, & Pearlstein, 1997). However, operational definitions of emotion dysregulation and distress intolerance have been inconsistent.

Why are the findings regarding the relationship between anxiety and suicidality mixed? Possible explanations have to do with severity of the disorder (e.g., in community vs. hospitalized youths), type of anxiety disorder, and the operational definition of suicidality (O'Neil-Rodriguez & Kendall, 2012). Although few studies have been adequately powered to examine suicidality in relation to each individual anxiety disorder, studies have supported specific relationships with GAD (Foley et al., 2006; Goldston et al., 2009; Masi et al., 2001) and SAD (Liu et al., 2006; Masi et al., 2001). The operational definition of suicidality (e.g., ideation; intent; capability of acting on intent) is especially relevant. For example, it is plausible that youths with an anxiety disorder show heightened suicidality because they experience and/or report *intrusive* thoughts about suicide, yet are not likely to experience suicidal intent. The content of suicidal thoughts experienced by depressed youths may differ and have associations with suicidal intent/behavior. Given the low base rate of suicidal behavior, large samples, as well as clear definitions of suicidality, are needed for this research.

It has been suggested that youths with both an anxiety disorder and a recent onset of a depressive episode are at particularly high risk for suicide attempts (Pawlak, Pascual-Sanchez, Raë, Fischer, & Ladame, 1999). Studies with youths have not distinguished among the anxiety disorders in examining their relationship to suicidality, but research with adults links generalized anxiety and social phobia, in particular, to high risk patterns of self-harm and suicide attempts (Chartrand, Sareen, Toews, & Bolton, 2012). Given the public concern that suicidality poses (Heron, 2007), research is need, but anxiety needs to be examined in terms of specific anxiety disorders rather than as a unitary disorder.

Assessment

To understand the pathways to anxiety and depression among youths, it is important to have accurate assessments. Brady and Kendall (1992) reviewed measures specific to anxiety or depression, relevant subscales of more general behavioral ratings scales, and structured interviews. At that time, anxiety and depression measures were highly correlated and lacked discriminant validity. We discuss discriminant validity and highlight the emergence of relevant symptom measures.[3] We emphasize that the purpose of assessment should guide the decision to measure symptoms versus disorders (Kraemer, Noda, & O'Hara, 2004), as well as the selection of specific symptom measures.[4]

Discriminant Validity of Anxiety and Depression Measures

Numerous clinician-rated, child self-report, and parent-report instruments are available (see Myers & Winters, 2002; Silverman & Ollendick, 2005). However, findings regarding the ability of the available measures to discriminate between anxiety and depression have been mixed. There is evidence that three measures, the Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997), the Screen for Anxiety and Related Disorders (SCARED; Birmaher et al., 1999), and the Pediatric Anxiety Rating Scale (PARS; Research Units on Pediatric Psychopharmacology Anxiety Study Group, 2002), tap depression less than other anxiety measures (e.g., Birmaher et al., 1999, 1997; Dierker et al., 2001; Wood, Piacentini, Bergman, McCracken, & Barrios, 2002), although this has not been studied

[3] Although not yet a focus for assessing anxiety and depression in youths, item response theory (IRT) merits consideration and application. IRT may be especially useful because it includes methods for scoring questionnaires that tap (a) the strength of a relationship of specific item responses to an underlying construct and (b) the degree to which a collection of questions measure one coherent construct (see Steinberg & Thissen, 2013). For example, one IRT study found that psychomotor agitation/retardation was more likely to be endorsed by adolescents with "pure" depression than by adolescents with depression and comorbid internalizing problems (Small et al., 2008).

[4] For example, there is some evidence based on item response modeling that the Beck Depression Inventory (BDI; Beck & Steer, 1993; Beck et al., 1961) assesses a larger range of depression symptom severity among adolescents than the Center for Epidemiologic Studies—Depression Scale (Radloff, 1977), providing more information at higher severity levels (Olino et al., 2012).

extensively. Total scores on the Revised Children's Manifest Anxiety Scale (RCMAS; C. R. Reynolds & Richmond, 1985) do not appear to discriminate between anxiety and affective disorders (see meta-analysis by Seligman, Ollendick, Langley, & Baldacci, 2004), but the Worry/Oversensitivity subscale of the RCMAS performs better (Lonigan, Carey, & Finch, 1994).

The Beck Depression Inventory (BDI; Beck & Steer, 1993; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) has discriminated between anxiety and depression in adults (Barrera & Garrison-Jones, 1988; Marton, Churchard, Kutcher, & Korenblum, 1991) and two of four factors, Negative Self-Attitude and Performance Difficulty, have discriminated depressed from anxious teenagers (Bennett et al., 1997). There is relatively little evidence supporting discriminative validity of other measures, such as the Children's Depression Inventory (CDI; Kovacs, 1985, 1992), the Reynolds Adolescent Depression Scale (RADS; W. M. Reynolds & Mazza, 1998), the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960; Warren, 1997). The Child Behavior Checklist (CBCL) does not differentiate between depression and anxiety, as the Anxious/Depressed subscale assesses negative affect more globally (Chorpita et al., 1998; Chorpita & Daleiden, 2002).

Measures Based on the Tripartite Model

Since Brady and Kendall's (1992) review, measures consistent with the tripartite model have been developed for youths.[5] These include the Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000), the Positive and Negative Affect Scale for Children (PANAS-C; Laurent et al., 1999), the Physiological Hyperarousal Scale for Children (PH-C; Laurent et al., 2004), the Negative Affectivity Self-Statement Questionnaire (NASSQ; Ronan, Kendall, & Rowe, 1994), and the Affect and Arousal Scale (AFARS; Chorpita, Daleiden, Moffitt, Yim, & Umemoto, 2000; Daleiden, Chorpita, & Lu, 2000). According to Silverman and Ollendick (2005), these measures have discriminated between anxiety and depression better than scales designed to target one or the other specifically. Considering the multiple pathways model, it is important to use measures of specific anxiety disorders that discriminate between depression and specific forms of anxiety, while also documenting any overlap of the two conditions. Although categorical assessments (i.e., structured diagnostic interviews) are required to document comorbidity (i.e., the presence of two disorders) and may be useful for research, they decrease power for hypothesis testing. Dimensional assessments (levels of severity) that tap risk for the comorbid condition may be preferred. To the extent that predictors of disorders may be different from predictors of symptoms (e.g., Gotlib, Lewinsohn, & Seeley, 1995), a combined approach to assessment is ideal for understanding comorbidity.

Treatment Response

The impact of comorbid anxiety and depression on treatment outcome among youths in Brady and Kendall's (1992) review was identified as a "major concern yet to be addressed" (p. 253). Although treatment response does not confirm or disconfirm an initial pretreatment diagnosis or causal hypothesis, it is of interest to consider the distinct and/or overlapping treatment effects with regard to anxiety and depression in youths. Since Brady and Kendall's review, studies have examined the effect of comorbidity on response to treatment of the target disorder(s). Identifying treatment moderators (e.g., comorbidities), or determining which children respond to which treatments, could greatly improve short- and long-term outcomes (Emslie, Mayes, Laptook, & Batt, 2003). If comorbid depression-anxiety predicts a poorer response to treatment for either disorder separately, then we are challenged to both (a) understand why/how comorbidity interferes with a favorable treatment outcome, and (b) enhance current or develop new interventions to meet the unmet needs. The majority of anxiety treatment studies have targeted multiple disorders: to the extent that the pathways to comorbidity are differentially related to specific anxiety disorders, larger and/or single-disorder samples would be needed to evaluate the impact of comorbid depression within the context of the proposed model.

Medication Treatment

Anxiety and depression have been reported to respond to selective-serotonin reuptake inhibitors (SSRIs; i.e., Brent et al., 2008; Hughes et al., 2007; Walkup et al., 2008), and studies suggest that anxiety and depression have overlapping functional and structural brain pathology (Nutt & Stein, 2006; see review by Cameron, Abelson, & Young, 2004). Research regarding the neurobiology of anxiety and depression is outside the current scope, but interested readers are referred to other articles (Beesdo, Lau, et al., 2009; Hettema, 2008; Lau et al., 2009; Morilak & Frazer, 2004; Thibodeau, Jorgensen, & Kim, 2006).

There is a small literature on medication response in youths with comorbid mood and anxiety disorders. For example, although studied in adults (Dunlop & Davis, 2008), few studies have examined benzodiazepine use in youths, and the available findings are mixed (Strawn, Sakolsky, & Rynn, 2012). Guidelines for

[5] The 47-item RCADS (Chorpita, Yim, et al., 2000) assesses SAD, SoP, GAD, panic disorder, OCD, and MDD in youths 6–19 years of age. Internal consistency estimates for the diagnoses range from .71 (SoP) to .83 (GAD), and retest reliability estimates range from .65 (OCD) to .80 (SoP). There is a parent version (RCADS-P; Ebesutani et al., 2010). Confirmatory factor analyses using clinic-referred and community/school samples have not supported combining MDD and GAD scales (Ebesutani et al., 2010; Ebesutani, Okamura, Higa-McMillan, & Chorpita, 2011; Watts & Weems, 2006; Weems, Zakem, Costa, Cannon, & Watts, 2005).

The 27-item PANAS-C assesses sensitivity to positive and negative stimuli over the past 2 weeks in children 8–14 years old. Fifteen items load on the NA subscale ($\alpha = .92$), and 12 items load on the PA subscale ($\alpha = .97$). The PANAS-C scales have demonstrated convergent and discriminant validity with scores on child self-reports of depression and anxiety (Laurent et al., 1999).

The 18-item PH-C assesses bodily manifestations of autonomic arousal in youths 12–17 years of age over the past 2 weeks. The PH-C is internally consistent ($\alpha = .97$). Three factors emerged representing PA, NA, and physiological arousal, and their intercorrelations were generally consistent with the tripartite model. Regression analyses showed that the PH-C added significantly to the prediction of anxiety after accounting for positive and negative affect.

The 27-item AFARS (Chorpita, Daleiden, et al., 2000), for use with 8- to 19-year-olds, has three subscales: NA, PA, and PH. Internal consistency estimates range from .77 to .81. Factor analyses identified NA and PA as relatively orthogonal, whereas physiological arousal was positively associated with negative affect.

benzodiazepine use, even for adults, cannot be established until further research is available (Sekula, DeSantis, & Gianetti, 2003).

SSRIs are frequently prescribed for child/adolescent anxiety disorders (Baldwin et al., 2005) and depression (Birmaher & Brent, 2002). Of interest, comorbid disorders (including anxiety) were not associated with fluoxetine outcomes among youths in three large trials (Cheung et al., 2010; Tao et al., 2009). The Research Units on Pediatric Psychopharmacology Anxiety Study Group (2002) found that depressive symptoms predicted poorer response to fluvoxamine among anxious youths, but none of the youths in the sample met criteria for MDD (Vitiello, 2003). In sum, SSRIs may be effective in reducing comorbid anxiety and mood symptoms but the current state of the research is too limited for a strong conclusion. At this juncture, if medication is pursued as a treatment option for anxious and depressed youths, combined treatment (CBT + medication) is the best option (March et al., 2004; Walkup et al., 2008).

Psychosocial Treatment

The most frequently studied psychosocial intervention for anxiety and/or depression in youths is CBT. Since Brady and Kendall (1992), gains have been made in three broad areas: (a) the differential effects of CBT on depression versus anxiety, (b) the impact of comorbidity on CBT outcomes, and (c) the impact of CBT for the principal disorder on comorbid symptoms.

The effects of CBT on depression versus anxiety. On the basis of the treatment literature, Chu and Harrison (2007) concluded that CBT has been comparably effective in producing symptom change in both anxiety and depression. However, relative to CBT for depression, CBT for anxiety has resulted in greater behavioral change and improved coping. Few studies examined physiological processes (although studies examining fMRI changes are underway), and studies are needed to examine whether CBT affects change through similar or different mediational pathways for anxiety versus depression. Many previous studies neglected to test mediation (Prins & Ollendick, 2003), but there are data indicating that anxiety and depression treatment outcomes are both at least partially mediated by changes in self-talk (e.g., Kaufman, Rohde, Seeley, Clark, & Stice, 2005; Kendall & Treadwell, 2007). That is, reduced negative self-talk mediates positive treatment outcomes, although temporal precedence has yet to be established. Corresponding to this, studies have supported the content specificity of negative self-statements, with anxious self-talk involving future-oriented questioning about harm, and depressive self-talk focused on loss and personal failure (e.g., Schniering & Rapee, 2004). Consistent with the tripartite model, which posits level of negative but not positive affect as common to anxiety and depression, change in negative but not positive self-talk has been implicated in anxiety reduction (Kendall & Treadwell, 2007). As Garber and Weersing (2010) noted, improved understanding of the mechanisms by which anxiety and depression are linked can be invaluable for identifying optimal treatment targets.

The impact of comorbidity on CBT outcomes. The impact of comorbid depression on CBT for anxiety (and vice versa) has been explored but findings are mixed. Several studies found that comorbid anxiety predicted poorer outcomes of depression treatment (Curry et al., 2006; Vostanis, Feehan, & Grattan, 1998; Young, Mufson, & Davies, 2006). Findings from a depression prevention program for adolescents (Interpersonal Psychotherapy-Adolescent Skills Training; Young & Mufson, 2003) showed that higher levels of comorbid anxiety symptoms were associated with slower rates of change in depressive symptoms. Upon further exploration, the authors determined that the effect of anxiety symptoms was apparent in the early weeks of treatment, but by postintervention, comorbid anxiety symptoms had no impact on depression outcomes. Perhaps, the presence of anxiety merely "delayed" intervention effects (Young et al., 2012). Similarly, there was largely no impact of comorbid anxiety on psychoeducational therapy for mood disorders (C. M. Cummings & Fristad, 2012), a school-based universal prevention program (Pössel, Seemann, & Hautzinger, 2008), or combined medication and psychotherapy for treatment resistant depression (Asarnow et al., 2009).

The presence of anxiety has predicted a more positive response to depression treatment for youths. Brent et al. (1998) reported that comorbid anxiety predicted end-of-treatment level of depression and that comorbidly anxious youths responded better to CBT than to a family therapy and a nondirective supportive therapy. Perhaps, as the authors suggested, this finding is related to the fact that youths with comorbid anxiety and depression share cognitive distortions (both targeted in CBT). Comorbid anxiety has also been associated with better treatment outcome for group and multifamily CBT for mood disorders (C. M. Cummings & Fristad, 2012; Rohde et al., 2001), but findings may be due to the comorbidly anxious patients' higher pretreatment depression scores, which provided greater opportunity for change. It is also possible that anxiety magnifies treatment effects among depressed youths due to the perfectionistic and social evaluation concerns that are part of anxiety contributing to child involvement and adherence.

What about the role of comorbid depressive symptoms on treatments for anxiety? Few studies have been sufficiently powered to answer this question, although some have examined the impact of nonanxiety comorbidity broadly (see Ollendick, Jarrett, Grills-Taquechel, Hovey, & Wolff, 2008). Two studies found that comorbid depressive symptoms and diagnoses were associated with poorer response to CBT for anxiety disorders generally (Berman, Weems, Silverman, & Kurtines, 2000), whereas another study did not replicate this finding (Rapee et al., 2013; Southam-Gerow, Kendall, & Weersing, 2001). Few studies have examined the impact of comorbid depression on the treatment of specific anxiety disorders. However, depressive symptoms have not predicted response to group CBT for SoP in youths (Kley, Heinrichs, Bender, & Tuschen-Caffier, 2012), nor have they moderated outcomes of social effectiveness therapy for children with SoP (Alfano et al., 2009). In some anxiety-treatment study designs, such as comparisons to medications where the medication arm may actually target both disorders, depressive disorders are an appropriate exclusion criterion (e.g., Walkup et al., 2008). Even so, excluding MDD cases limits conclusions regarding the role of depressive disorders on anxiety treatment outcomes.

The impact of CBT on comorbid symptoms. Although cases of diagnosed depression may not have been included, evidence from treatment-outcome studies indicates that CBT for anxiety often results in decreased depressive symptoms (e.g., Barrett, Dadds, & Rapee, 1996; Kendall et al., 1997; Kendall, Hudson, et al., 2008; Kendall, Safford, Flannery-Schroeder, & Webb, 2004; Manassis et al., 2002; Mendlowitz et al., 1999; Silverman et al., 1999). In terms of school-based interventions, a meta-analysis

found that anxiety interventions did not yield significantly greater reductions in depressive symptoms than controls, but only five studies have been reported (Mychailyszyn, Brodman, Read, & Kendall, 2012). The effect of depression treatments on anxiety symptoms has not been consistent. One meta-analysis, by Weisz, McCarty, and Valeri (2006), indicated that interventions for depression in children yielded significant reductions in anxiety symptoms (*effect size* = 0.39), though smaller in magnitude than reductions in depressive symptoms (*effect size* = 0.57). Interpersonal therapy has been associated with fewer panic/somatic and generalized anxiety symptoms, but not social anxiety symptoms compared to treatment as usual (Young et al., 2012). No reduction in comorbid anxiety symptoms was found in a study of treatment for mood-disordered youths (C. M. Cummings & Fristad, 2012).

It seems reasonable that youths with comorbid anxiety need treatments that specifically target fears through exposure tasks (Bouchard, Mendlowitz, Coles, & Franklin, 2004; Kendall et al., 1997, 2005). Given evidence that the onset of anxiety typically precedes the onset of depression, randomized clinical trials with extended follow-up assessments are needed to determine if treating anxiety prevents depression. The only study to date did not support this possibility (Benjamin, Harrison, Settipani, Brodman, & Kendall, 2013). Successful treatment of childhood anxiety was associated with reduced likelihood of substance use problems but not depressive disorders at 16-year follow-up. Enhancing anxiety treatment with strategies specific to the prevention of depression may prove beneficial.

Treating Comorbid Anxiety and Depression

Findings regarding both comorbidity and CBT outcomes have led investigators to directly address comorbid symptoms. Chu, Merson, Zandberg, and Areizaga (2012) proposed three approaches: the flexible implementation of single-target interventions, modular-based treatment, and transdiagnostic therapy. Interventions that target principal disorders can be implemented flexibly (e.g., Hudson, Krain, & Kendall, 2001; Manassis & Monga, 2001) to address related comorbidities. For example, "flexibility within fidelity" (Kendall & Beidas, 2007) involves adapting an empirically supported manual to suit a particular child while adhering to the treatment goals/approach (Kendall, Gosch, Furr, & Sood, 2008; see also Kendall, Kortlander, Chanksy, & Brady, 1992). Modular-based CBT includes core and supplemental modules that are selected to fit client needs based on decision making algorithms (Chorpita, Taylor, Francis, Moffitt, & Austin, 2004; Chorpita & Weisz, 2009). Finally, transdiagnostic therapy focuses on commonalities between the codiagnosed disorders (Kendall et al., in press).

Twenty years ago, Brady and Kendall (1992) raised the question of whether depression and anxiety benefit from similar treatment approaches or require differential intervention. At that time, transdiagnostic treatment approaches (e.g., Barlow, Allen, & Choate, 2004) had not been introduced. Presently, the Unified Protocol for the Treatment of Emotional Disorders in Youth (UP-Y; Ehrenreich et al., 2008; Ehrenreich, Goldstein, Wright, & Barlow, 2009) and Group Behavioral Activation Therapy (GBAT) for anxious and depressed youths (Chu, Colognori, Weissman, & Bannon, 2009) are two examples. By combining target problems, both UP-Y and GBAT hope to minimize the burden associated with training and supervision in community settings.

UP-Y, adapted from a transdiagnostic treatment for emotional disorders in adults (Barlow et al., 2004), focuses on ways in which youths experience and respond to multiple emotions. UP-Y has eight sections, five of which are required. Preliminary data support the feasibility of UP-Y (Bilek & Ehrenreich-May, 2012). GBAT, also referred to as the SKILLS Program, was adapted from adult protocols (Addis & Martell, 2004; Martell, Addis, & Jacobson, 2001) for use with adolescents in school settings. The program is based on the idea that behavioral activation is involved in the treatment of both anxiety and depression. The program consists of 10 weekly sessions, after which clients identify functional domains they would like to optimize. Pilot data suggest that the program is feasible and acceptable, but, as with the range of transdiagnostic approaches, randomized clinical trials are needed establish treatment efficacy. Additionally, evaluations of transdiagnostic prevention programs are needed. For example, EMOTION (Kendall, Stark, Martinsen, O'Neil, & Aroras, 2013), is a 20-session indicated prevention program, implemented in a group format, that integrates core components of empirically supported treatments for anxiety and depression in youths (*Coping Cat*: Kendall & Hedtke, 2006a, 2006b; *ACTION*: Stark, Schnoebelen, Simpson, Hargave, & Glenn, 2007, respectively).

Do the multiple pathways warrant differential treatment? For instance, we hypothesize that Pathway 2 comorbidity, with greater symptom overlap and simultaneous presentation of anxiety–depression, calls for concurrent treatment of anxiety and depression (e.g., a transdiagnostic approach). Pathway 1, with principal anxiety problem and depression as a complicating factor, may be best addressed with a flexible implementation of an anxiety treatment. Pathway 3 implicates a flexible implementation of a depression treatment, with residual anxiety addressed pending successful treatment of depression.

Conclusions and Future Directions

Research since the 1992 review has advanced our knowledge about comorbid anxiety and depression in youths (see Table 1). Although Brady and Kendall's (1992) conclusion that depression and anxiety were distinct but related disorders has been supported by the updated review, progress has been made delineating the overlapping and distinctive features of anxiety and depression. Further progress has been made describing the heterogeneity of the anxiety disorders (SoP, SAD, and GAD) and the unique relationship of each to depression.

Several studies suggest developmental trajectories of depression-anxiety comorbidity in youths, and these trajectories appear to vary depending on the subtype and onset of the disorders. Of the anxiety disorders, GAD demonstrates the most overlap with depression in terms of symptoms and risk factors. Although these two disorders have shared genetic risk and cross-predict each other, we must ask, "How much of the relationship is linked to symptom overlap between the two diagnoses?" Some researchers (e.g., Watson, 2005) suggest that GAD and depression be considered within a general category of "distress disorders," but we suggest that variations in the age of onset indicate that they are best considered distinct (but highly related) disorders in youths.

SAD and SoP are fear-related anxiety disorders (Watson, 2005) and demonstrate different associations with depression than GAD. In some instances, anxiety puts individuals at risk for depression, likely a result of interpersonal and cognitive processes. There is some indication that depression secondary to anxiety may be a less severe form of depression (Olino et al., 2010), perhaps different from more severe depression that occurs as a result of biological processes. SAD and depression may share risk factors (e.g., parental depression), or depression may onset following SAD-related impairment. Youths with SoP are more likely to become depressed, perhaps resulting from social isolation, low self-esteem, and potential peer victimization. However, the developmental pathway to depression from SoP is complex (Epkins & Heckler, 2011) and depression can precede SoP. Additional longitudinal studies beginning in early childhood, *with consideration given to the specific anxiety disorder*, will clarify the relationship between depression and anxiety and could have strong implications for the early identification of youths at risk for either disorder. Gender differences in depression and in anxiety, separately, have been studied, but less attention has been paid to gender and comorbidity. Data from community samples suggest that anxiety–depression comorbidity is more common in girls than boys. Some data suggest that suggest that anxious boys are prone to developing later depression. Future exploration of gender differences in the anxiety–depression comorbidity is warranted.

Theoretical models help explain the frequent comorbidity between anxiety and depression. Several models have been proposed, such as the Tripartite (Clark & Watson, 1991) and BIS/BAS (Gray, 1982) theories, yet findings have indicated shortcomings with both. For instance, the relationships between anxiety–depression and PA and PH are less straightforward than hypothesized. The role of the BIS/BAS and co-occurring depression-anxiety merits further study. Behavioral inhibition serves as an early marker of anxiety (and perhaps depression), a finding with important implications for prevention and early intervention (Hirshfeld-Becker et al., 2010). The models offer guidance and have merit, but there are research needs related to developmental change. Essential to advancements, future research must consider the heterogeneity among anxiety disorders when considering the co-occurrence/comorbidity of anxiety and depression: anxiety disorders are not all the same!

The multiple pathways model builds on previous models by emphasizing the heterogeneity of the anxiety disorders and their degree of overlap with depression, as well the developmental sequence of anxiety and depression. Consistent with the reported findings, we propose that anxiety–depression comorbidity is heterogeneous, not singular, and emerges from multiple pathways. Pathway 1 describes depressive presentations secondary to a principal anxiety disorder. The literature is consistent with Pathway 1 applying to SoP and SAD, both of which often precede the onset of a comorbid depression disorder (e.g., Fichter et al., 2010). Findings that depression secondary to anxiety is milder in presentation (Olino et al., 2010) also support Pathway 1. Pathway 2 involves depression and anxiety as coprimary, having the highest degree of overlap between the two conditions, and best fits the comorbidity of GAD and depression. In support of this pathway, the order of onset of these two disorders in comorbid youths is not consistent (Essau, 2003), and many studies have found that GAD may be more similar to depression than to the other anxiety disorders (e.g., Watson, 2005). Pathway 3 describes principal depression, with anxiety secondary. Pathway 3 includes SoP secondary to a more severe depression. This pathway has the least empirical support and may occur less frequently than Pathway 1 or Pathway 2. We cannot yet determine the extent to which Pathway 3 describes chance comorbidity of anxiety and depression (Klein & Riso, 1993), or whether undetected anxiety symptoms are present primary to the onset of a depressive disorder. The pathways all require empirical testing to further inform the interplay between depression and anxiety symptoms and disorders in youths.

The goal is for models of anxiety–depression comorbidity to be applied in a way that improves current assessment and intervention for comorbid anxiety–depression. Knowledge has advanced since 1992 regarding the impact of anxiety–depression comorbidity on treatment outcome, with some indications of secondary benefits for the nontargeted disorder. Transdiagnostic treatment (geared to anxiety and depression) has gathered interest and offers promise. Given the overlapping features of anxiety and depression, and the effects of one on the other, there may be cases where the successful treatment of one disorder (i.e., social anxiety) prevents or reduces the progression of the other disorder (Kessler & Price, 1993). The effective treatment of comorbid anxiety–depression depends on the relationship between them, with Pathways 1 and 3 calling for treatment of the principal disorder. Pathway 2, on the other hand, suggests the need for a transdiagnostic treatment.

We highlight specific areas for future research regarding the multiple trajectories of anxiety–depression among youths. For one, we need to further evaluate the risk factors. There are high-quality studies that examine the trajectories of anxiety/depression from adolescence to depression (e.g., Olino et al., 2010), but longitudinal studies beginning in early childhood are needed. These longitudinal studies could include analysis at the level of symptoms, as well as disorders, as otherwise undetected symptoms of one disorder may predict a diagnosis of another. Second, we highlight that anxiety disorders are heterogeneous and that advances are linked to specific anxiety disorders. Depression, too, can be heterogeneous (e.g., Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Small et al., 2008) and future work would be wise to consider such variation. For instance, it is possible to meet criteria for MDD through several symptoms (i.e., only five of nine MDD symptoms need be present). Diagnosed youths may exhibit varying symptom combinations, and future research should examine differing depressive profiles and how they are associated with anxiety in youths.

Third, development can inform us about the protective factors, and future research should examine depressed or anxious youths who do not develop comorbidity. These protective factors have preventative implications, especially in instances where comorbidity arises as a result of impairment from the primary diagnosis. Fourth, research addressing transdiagnostic factors (e.g., rumination, negative affectivity, neuroticism) of anxiety and depression is needed, as well as features that distinguish them (e.g., anhedonia). Fifth, research identifying specific genes (Franić et al., 2010), such as the serotonin transporter (5-HTTT; Verhagen et al., 2009) and dopamine receptor genes (DRD2; Hayden et al., 2010), would benefit from precise assessment of depression and anxiety and their comorbidity, as well as any direct and indirect influences on the expression of anxiety and depression (i.e., gene–environment interactions; Silberg, Rutter, Neale, & Eaves, 2001).

Finally, multiple pathways need to take into account other comorbidities (e.g., externalizing disorders) and comorbidity within the anxiety disorders. As the field shifts toward classification schemes that promote rapid translation of basic neurobiological and behavioral research to improved understanding of psychopathology and advances in treatment (e.g., Sanislow et al., 2010), integration of categorical and dimensional approaches to comorbidity will be critical. One can readily anticipate that the next 20 years will further advance our understanding of these impairing conditions, as well as inform intervention efforts, reducing the impact of anxiety and depression on society and those affected.

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