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Anxiety Disorders in Childhood: Casting a Nomological Net

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Empirical research highlights the need for improving the childhood anxiety disorder diagnostic classification system. In particular, inconsistencies in the stability estimates of childhood anxiety disorders and high rates of comorbidity call into the question the utility of the current *DSM* criteria. This paper makes a case for utilizing a nomological net[4] model for advancing the understanding of childhood anxiety disorders. In this article, we discuss measurement and assessment issues related to improving the childhood anxiety disorder diagnostic system and show how these issues can be addressed by employing the nomological net of childhood anxiety. Because employing the nomological net involves drawing from etiological process theories to facilitate classification and assessment, an integrative model of childhood anxiety disorders is presented as a tentative heuristic. Then evidence for the existing symptom sets is discussed in the context of how process theory mechanisms may be utilized to improve classification and assessment. Testable hypotheses are presented. Measurement, disorder definition, treatment, and policy implications are also discussed.

KEY WORDS: childhood anxiety; comorbidity; treatment; diagnostic classification.

Anxiety disorders are the most prevalent class of emotional disorders of childhood, can cause intense personal distress, and negatively impact children's school and social functioning (Bernstein & Borchardt, 1991; Bernstein, Borchardt, & Perwien, 1996; Silverman & Ginsburg, 1998). The scientific understanding of childhood anxiety disorders is largely a function of the ability to reliably and validly identify, classify, and measure these phenomena. Major advances in understanding and treating childhood anxiety disorders have resulted from utilizing the taxonomy provided by the *Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition* (*DSM-IV*; American Psychiatric Association, 1994). *DSM-IV* is a major accomplishment in scientific taxonomy. The current symptom lists that make up the diagnoses are the product of many individuals, years of research, current literature, and clinical experience (APA, 1994). However, researchers continue to question the validity of the childhood anxiety disorders (see Saavedra & Silverman, 2001). In this paper, we provide a model for revising and validating the childhood anxiety disorders diagnoses and for testing etiological theories of these disorders.

The *DSM-IV* diagnoses of childhood disorders are primarily descriptive, emphasizing observed or reported symptoms rather than underlying causal mechanisms. As a result, the disorders are grouped primarily on the basis of shared symptoms. Emerging research indicates that developmentally and theoretically based assessment of anxiety disorder symptoms may provide a more sensitive assessment than the *DSM-IV* criteria in children (e.g., Scheeringa, Zeanah, Drell, & Larrieu, 1995; Scheeringa, Peebles, Cook, & Zeanah, 2001; Scheeringa, Zeanah, Myers, & Putnam, 2003). Saavedra and Silverman (2001) and Dadds, James, Barrett, and Verhulst (2004) have recently reviewed the taxonomy of childhood anxiety disorders and concluded that there is a need

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[4]A nomological net can be briefly defined as the theoretical structure of a construct.

for continued research attention on the reliability and validity of the classification system. Their reviews highlight the need for additional conceptual work on the classification of anxiety disorders in children and adolescents. For example, inconsistencies in estimates of the stability of anxiety disorder diagnoses and in estimates of the long-term outcomes associated with *DSM* childhood anxiety disorder diagnoses strongly suggest the need for improved instrumentation and classification (e.g., Keller et al., 1992; Last, Hansen, & Franco, 1997; Last, Perrin, Hersen, & Kazdin, 1996; March, Leonard, & Swedo, 1995; Newman et al., 1996; Pine, Cohen, Gurley, Brook, & Ma, 1998, see Silverman & Ginsburg, 1998). Additional problems include high levels of comorbidity, poor agreement among reporters, as well as poor discriminant and predictive ability (Dadds et al., 2004). These problems may not be solved by additional research relying solely on the *DSM* diagnoses, because these problems may be endemic to the *DSM* definitions. The *DSM* diagnoses of childhood anxiety have been responsible for important advances in clinical decision making, understanding and comparing treatment outcome across studies as well as understanding the epidemiology and course of anxiety related problems in youth (see Dadds et al., 2004; Saavedra & Silverman, 2001), but this may be about as far as we can go with the current definitions.

The problem we attempt to address in this paper is how can we continue to benefit from the advances that the descriptive approach has provided while continuing to increase the utility of our understanding of what an anxiety disorder is. Our message is "let's not throw out the baby with the bath water," but we can and do need to improve our baby bath. To address problems of comorbidity, reporter agreement, discriminant and predictive validity, we argue that greater attention to etiological processes in the classification of childhood anxiety disorders is needed. In this paper, we first discuss conceptual and methodological considerations that may limit attempts to reliably and validly identify, classify, and measure childhood anxiety disorders. We then present an integrative biopsychosocial model of the etiology of the childhood anxiety disorders based on existing theory and empirical data as a tentative heuristic for further research. We then make the case for utilizing a nomological net model as a solution to the problems in classification and assessment.

Our proposed solution to classification can be understood by analogy to developmental psychopathology's view on etiology. Specifically, the field of developmental psychopathology has emphasized the utility of understanding etiology as an interlocking network of constructs and processes, as opposed to a single disease process or risk. We suggest approaching classification terms of the network of constructs that define a disorder, and by increased utilization of process theories and developmental modifications to the classification system. In the final sections of this paper we point out distinctions between a nomological net approach and purely etiological and descriptive approaches to classification (i.e., we try to show how to improve the baby bath without tossing the baby).[5] The implications of this approach to advancing the understanding of childhood anxiety disorders are discussed with regards to the evolution of the classification scheme, treatment, and policy implications.

GENERAL FACTORS INFLUENCING VALID CLASSIFICATION

Construct validity has been largely trivialized in the assessment of psychological disorders (Sechrest, Stickle, & Stewart, 1998). However, diagnoses are constructs, and as such the issues of construct validity apply equally to the *DSM* childhood anxiety diagnoses. As proposed by Cronbach and Meehl (1955), construct validity requires that for constructs and their measures to be valid, they must be incorporated into a theoretical structure, which they termed the "nomological net." An adequate nomological net must define childhood anxiety in several ways, both within and across studies (i.e., minimally, it must use multiple measures see Sechrest et al., 1998; Shadish, Cook, & Campbell, 2002). In practical terms, this means a clear definition and explication of the prototypical elements and processes of anxiety (e.g., symptoms of the disorder versus independent etiological factors), more specific observations about the relationships among these elements and processes, a better understanding of the influence of the source of information (e.g., parent, child, observation), and a clearer discrimination between the construct of anxiety and other important and associated conditions (e.g., depression). The problems with high comorbidity, poor reporter agreement, discriminant

[5] Although our focus is on the childhood anxiety disorders this review is drawn from a broad assessment literature and thus may have implications for other disorders of childhood.

and predictive validity of the current *DSM* anxiety disorders of childhood may result from inadequate inclusion of the prototypical elements and processes of anxiety implied by the nomological net in defining "what" an anxiety disorder in childhood is.

To advance the construct validity of childhood anxiety disorders, a delineation of the critical prototypical elements (i.e., diagnostic criteria and underlying processes) of childhood anxiety disorders is needed. More specifically, there is a need to know what the prototypical elements are that comprise useful definitions of the childhood anxiety disorders, a need to know if drawing from etiological models can facilitate improved classification, and a need to identify independent etiological and maintenance factors. In addition, the optimal measurement strategies of the prototypical elements and processes must be clarified.

Prototypical Elements

As noted, in the *DSM-IV*, the disorders are grouped primarily on the basis of shared symptomatology. This was adopted to improve reliability by focusing on observable descriptive phenomena (APA, 1994). To expand upon the point above, a major contributing factor to lowered diagnostic validity may result from the failure to adequately utilize data on etiological processes for forming categories (e.g., Clark, Watson, & Reynolds, 1995; Meehl, 1977; Meehl & Golden, 1982). In other words, the current symptom lists may not include important prototypical elements. In addition to limiting the classification scheme, the failure to include important elements may have treatment implications. Kanfer and Saslow (1969) suggested early on that the most important failing of pure descriptive classification is the lack of sufficient information to categorize behavior such that prediction of individual responses to particular situations can be made. Reliance on purely descriptive classification is thus limited because there is insufficient information about the putative processes that underlie the disorder to inform intervention efforts.

Research and theory suggest that utilizing etiological mechanisms to inform classification is likely to improve on the validity and stability of groupings, better explain observed heterogeneity, and decrease comorbidity of disorders that share symptoms (e.g., Andreason & Carpenter, 1993; Clark & Watson, 1991a, 1991b). An approach to classification utilizing proposed and empirically supported etiologic factors and developmental modifications might improve the validity of the childhood anxiety disorder taxonomy. For example, although anxiety and depression share general affective distress and other common symptoms, possibly contributing to diagnostic comorbidity and instability in diagnosis over time (Clark & Watson, 1991a, 1991b), underlying processes specific to each type of disorder may reliably distinguish them (e.g., Zinbarg, Barlow, Liebowitz, & Street, 1994). For example, physiological hyperarousal and tension appear to be specific to anxiety and absence of positive affectivity (anhedonia) specific to depression (Clark & Watson, 1991a, 1991b). Another example can be found in terms of developmental differences. For instance, some of the cognitive factors characteristic of adult anxiety may not be equally relevant for defining anxiety disorders in young children (e.g., certain types of worry are less common in young children, see Weems, Silverman, & La Greca, 2000).

Although an etiologic approach appears attractive, etiological classification schemes have also proven problematic in the past (APA, 1968, 1994). An important and complicating consideration from the field of developmental psychopathology is that anxiety disorders, like all developmental outcomes, are multidetermined (Vasey & Dadds, 2001). Additionally, a particular end state (e.g., anxiety disorder) may be reached from different risk factors and through different processes (i.e., "equifinality"; Cicchetti & Rogosch, 1996). Moreover, particular adverse processes, events and/or risk factors (e.g., exposure to traumatic stress, high anxiety sensitivity, behavioral inhibition) may not necessarily lead to the same outcome (e.g., anxiety disorder) in every individual (i.e., "multifinality"; Cicchetti & Rogosch, 1996). Thus, assessment of childhood anxiety and estimates of its stability and long term outcomes are also likely to be inconsistent if they focus solely on etiologic risk factors.

According to the developmental psychopathology model, youth who display disordered levels of anxiety do so as a result of a complex interaction of numerous causal mechanisms, including individual risk factors (e.g., high anxiety sensitivity, behavioral inhibition), problems in their immediate psychosocial context (family poverty, parental psychopathology, exposure to trauma), and problems in their broader psychosocial context (e.g., violent neighborhood). Additionally, these causal mechanisms typically operate in a transactional or multiplicative fashion with each other, rather than in an independent or additive fashion. For example,

a behaviorally inhibited child with high anxiety sensitivity may develop acute anxiety symptoms if exposed to traumatic stress. If such a child lives with parents who have limited skills in reducing the child's anxious responding, is in a high crime neighborhood, and is repeatedly exposed to challenging events, vulnerability to developing an anxiety disorder is compounded by the interaction of the parent and child, the child and the neighborhood, the parent and the neighborhood, and probably other factors as well (Stickle & Frick, 2002).

In sum, a failure to utilize etiological mechanisms and theory may contribute to poor validity. However, although utilization of risk factors and etiological mechanisms may improve estimates of stability and long-term outcomes, the mechanisms that lead to anxiety disorders may differ across individuals or subgroups of youth who exhibit anxiety. That is, the most important causal factors and how they interact to place a child at risk for anxiety disorders may differ across individuals or subgroups (i.e., equifinality). Such individual differences in etiology suggest an approach to assessment that does not rely solely on etiological mechanisms. Such an approach would limit the classification system because particular etiological factors may not necessarily lead to or be a problem for every child and a useful classification system needs to have a fairly high degree of consistency across individuals (Blashfield, 1989). The challenge is thus to have consistent, reliable diagnostic criteria, utilize etiological mechanisms to improve validity, and be able to distinguish symptoms and signs of the disorder from independent etiological factors. The following sections will focus on ways of utilizing etiological mechanisms to improve the validity of childhood anxiety disorder diagnoses. Such improvement is, however, intimately tied to the problem of method variance.

Optimal Measurement

An essential aspect of improving the diagnostic validity of childhood anxiety disorders is to attend to the problem of method variance. Method variance refers to variability in scores on a trait measure (e.g., anxiety) resulting from something other than the trait (Campbell & Fiske, 1959; Sechrest, Davis, Stickle, & McKnight, 2000). Inconsistencies in estimates of stability and functional outcome in research on childhood anxiety disorders may be clarified by information on informant, instrument and sample biases. For example, meta-analysis indicates that the average agreement among youth, parents, teachers, and clinicians is about $r = .25$ (Achenbach, McConaughy, & Howell, 1987). It is unclear to what degree low informant reliability results from situational differences in behaviors (Achenbach et al., 1987), method variance (Stickle & Blechman, 2002), inadequate assessment procedures (Sechrest et al., 1998; Stickle & Weems, in press), or poor construct validity (Sechrest et al., 1998, 2000).

An additional measurement problem results from sampling bias. For example, results from large epidemiological studies and samples of clinic-referred youth suggest that there is a high degree of comorbidity among anxiety disorders and comorbidity between anxiety and other disorders such as depression. However, different rates of comorbidity for the anxiety disorders in youth have been found depending on the sample. In general, results from clinic samples show higher rates of comorbidity than those from community samples (see Essau, 2003).

In sum, method variance can result from the characteristics of the informant, assessment situation, sample, or the assessment instruments themselves. Method variance has been observed to account for twice as much variance as is accounted for by particular traits in studies of childhood psychopathology (Stickle & Blechman, 2002). In fact, a majority of variance in measures in some studies of childhood psychopathology results from factors other than the behaviors of interest (Fergusson & Horwood, 1989; Stickle & Blechman, 2002). This lack of precision is troubling and underscores the need for improved measurement strategies. Research on the assessment and classification of child anxiety disorders will benefit from continued improvement in measurement and assessment strategies. Research detailing the characteristics of individual measures of anxiety and an accounting of how different modalities of assessment (e.g., self-report, parent report, interviews, behavioral observation, physiological measures) relate to each other is necessary to establish clarity about the relationships among the different elements of anxiety, the extent to which various elements are stable, and how these elements are associated both with initial impairment and with functional status over time.

FINDING SOLUTIONS TO VALIDLY AND RELIABLY CLASSIFYING CHILDHOOD ANXIETY DISORDERS

Drawing from the above considerations, we propose that advances in the understanding of childhood anxiety disorders will require studying

diagnostic symptoms in concert with the underlying constructs or "mechanisms" of the disorder process as well as the methods used to assess both symptoms and mechanisms. We have chosen the term mechanism to refer to constructs that are drawn from etiological process theories. We use the term symptoms to refer to the descriptive criteria in the *DSM*.[6] Mechanisms are constructs drawn from process theories, but all mechanisms may not necessarily be etiological. Some mechanisms may be etiological, markers of etiological risk, maintenance factors, may interact with other factors, or otherwise be clinically important to the definition of an anxiety disorder. The term mechanism is thus used to specify constructs that may be essential and more directly a part of what makes a childhood anxiety disorder a disorder. Symptoms and mechanisms are all part of a complex set of etiological processes. To clarify these distinctions consider the following hypothetical proposition. Genetic transmission of traits such as behavioral inhibition and trait anxiety may lead to an anxiety disorder. Genetic transmission of behavioral inhibition and trait anxiety is the etiological process; behavioral inhibition and trait anxiety would be considered specific mechanisms. Although we are arguing that the symptom sets are likely in need of modification and improvement, the current *DSM* criteria would constitute the symptoms in the above proposition.

Classification may be performed for a variety of purposes (Blashfield, 1989). In the case of utilizing the nomological net for childhood anxiety disorders, the purposes of classification involve improved identification of the disorders, a clearer delineation of the specific processes that affect their onset and course, clarity about how these processes affect functional status, and the extent to which aspects of those processes are mutable and make for effective prevention and intervention targets. A classifiable disorder should also have an ontological status that at least in part, transcends developmental, individual, and cultural boundaries. Attempts at improving the current classification of childhood anxiety disorders are thus faced with the difficult challenge of integrating process theory mechanisms without making the disorders completely contextual and individualized. This point can be clarified by thinking about the difference between a functional assessment and a diagnosis. A diagnosis is a heuristic that implies certain things about etiology, maintenance, and treatment, for example. A functional assessment, on the other hand, is a detailed individualized description that identifies the factors maintaining a behavior and suggests intervention points to change the behavior. A classifiable disorder must apply to more than an individual; it should characterize sets of individuals. Our suggestion is that the diagnoses can be improved with regard to their capacity for telling us certain things about etiology, maintenance, and treatment.

Our proposal draws on the recommendations of past theorists. Kanfer and Saslow (1969) and Noyes and Kolb (1963), for example, have recommended using multiple factors in diagnosis including genetic, psychological, and symptom level information. Kanfer and Saslow (1969) argued that diagnosis should be organized along three primary domains (1) etiology, (2) symptoms, and (3) prognosis. Kanfer and Saslow suggest that diagnosis by symptoms alone is limited because classification by symptoms may sometimes lead to a focus on a set of behaviors that is clinically irrelevant. For example, this approach can lead to a focus on diagnostic symptoms that do not interfere with functioning and have little to do with impairment (Kanfer & Saslow, 1969). There is evidence that this is true of the symptoms of PTSD in youth. Specifically, Carrion, Weems, Ray, and Reiss (2002) found that many of the *DSM* symptoms of PTSD were not associated with impairment in a sample of children aged 7–14 years who had been exposed to diverse traumas. Moreover, those meeting full criteria were not significantly more impaired than those who met partial criteria for PTSD. Related to this, although the *DSM* does suggest that there may be differences in the way symptoms are expressed in youth as compared to adults, there remains an assumption that although expressed differently, the same basic set of symptoms comprises the anxiety disorders in youth as in adults. However, a very different set of symptoms may be problematic (i.e., associated with poor outcomes) or chronic (i.e., have long term stability) in youth as compared to adults (see Scheeringa et al., 1995).

Kanfer and Saslow (1969) also recommended including classification by prognosis. Specifically, examining the extent to which assessment and tracking of the presence of diagnostic symptoms (and mechanisms) foretells functional impairment is an essential component in determining the utility of the symptoms and symptom sets. Prognosis is particularly important in terms of the development and evolution of

[6]We later use the term "etiological factors" to refer to independent etiological factors that are not a part of the definition of a disorder.

symptoms in childhood. Specifically, there is a consistent finding that anxiety disorders precede the development of depression. For example, Essau (2003) found that among youth (aged 12–17 years from an epidemiological sample) with comorbid anxiety and depression, social and simple phobias preceded the onset of depression. Such findings point to the importance of clearly delineating the evolution of anxiety symptoms over time so that better prognostic statements can be made.

In sum, past authors have made general recommendations for similar approaches to diagnosis (i.e., to include etiological factors such as biological, genetic, behavioral and learning factors, Kanfer & Saslow, 1969; Tharp & Wetzel, 1969). One of the reasons these suggestions have failed to significantly influence the field of diagnostic classification may be because of their generality. Applying these suggestions to specific sets of problems may facilitate the utilization of these ideas. The purpose of the next sections is to begin to delineate specific recommendations for improving the classification of anxiety disorders in youth. Another reason that these suggestions have failed to significantly influence the field of diagnostic classification is that there are difficulties in utilizing process theories in diagnosis. We attempt to address this by making distinctions between etiological process and more easily measurable mechanisms in the sections that follow.

An Integrative Model of Childhood Anxiety Disorders

Anxiety has been conceptualized as a response system that involves behavioral, physiological, and cognitive components (e.g., see Barlow, 1988; Lang, 1977). Worry, for example, has been defined as one of the cognitive components of anxiety in that it can be viewed as a normative cognitive response that prepares the individual to anticipate future danger (e.g., Barlow, 1988; Borkovec, Shadick, & Hopkins, 1991; Mathews, 1990; Weems et al., 2000). Fear, in contrast, has been viewed as a part of the biological response system that prepares the individual for escape (Mathews, 1990). Anxiety disorders are thought to be associated with quantitative and/or qualitative deviations in the normative mechanisms of the anxiety response system (Barlow, 1988; Vasey & Dadds, 2001). Constructs such as hyperarousal, negative affectivity, intense worry, intense fear, avoidance/withdrawal, trait anxiety, anxiety sensitivity, and cognitive distortions are thought to be some of the specific mechanisms of the anxiety disorder process in youth (Epkins, 1996; Gencoz, Gencoz, & Joiner 2000; Joiner & Lonigan, 2000; Kendall, 1994; Ollendick, 2000; Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999; Vasey & Dadds, 2001; Weems, Hammond-Laurence, Silverman, & Ginsburg, 1998; Weems, Berman, Silverman, & Saavedra, 2001; Weems, Silverman, Saavedra, Pina, & Lumpkin, 1999, Weems et al., 2000). That is, deviations in the experience of normal levels of these constructs are associated with anxiety disorders and anxiety related impairment.

The constructs noted above grew out of research on one or more specific theoretical processes. The theoretical processes implicated in the origins of problematic anxiety in youth have emphasized biology (e.g., genetics, temperament, psychophysiology), behavior (e.g., operant, observational, and respondent learning models), cognition (e.g., information processing, stimuli/event interpretation), interpersonal factors (e.g., attachment theory), and within a developmental psychopathology framework, the interaction of these factors in various contexts (parent-child relationship, family, home, school, community). These four domains are particularly relevant because treatment researchers have focused intervention efforts for childhood anxiety on biological systems (see Walkup, Labellarte, & Ginsburg, 2002), social family systems (see Ginsburg & Schlossberg, 2002), or cognitive and behavioral processes (see Kendall, 1994; Albano & Kendall, 2002).

Drawing from general models of developmental psychopathology (e.g., Bronfenbrenner, 1977; Cicchetti & Rogosch, 1996), anxiety disorders in childhood are hypothesized to result from a complex interplay of biological, cognitive, behavioral, and social influences. The various processes may be associated with normal anxiety (as well as anxiety disorders) and the processes may serve to exacerbate normal anxiety into an anxiety disorder (e.g., normal levels of anxiety paired with reinforcement of avoidance may result in anxiety problems). In addition, the processes may interact with each other to put an individual at risk for an anxiety disorder. For example, normal levels of anxiety paired with parental reinforcement of avoidance may lead to an avoidant cognitive style that may result in anxiety problems. Moreover, the processes may be avenues by which pathological anxiety (anxiety disorders) may return to normal levels of anxiety (i.e., they are the focus of interventions aimed to treat anxiety disorders). Finally, any one or any number of the

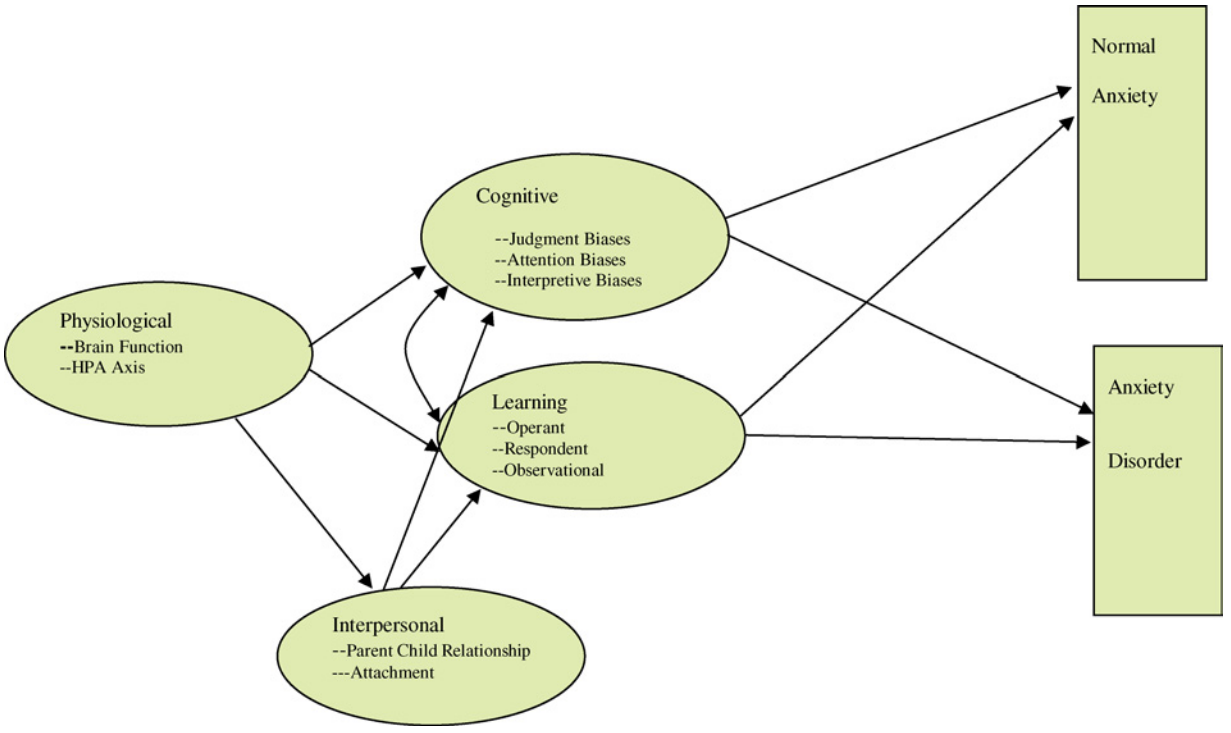

Fig. 1. A General schematic of theoretical processes forming the nomological net of childhood anxiety disorders. The figure illustrates a model of the general etiology of childhood anxiety disorders.

processes may be responsible for the etiology of a particular individual's anxiety disorder, and any one or any number of other processes may be effective in treating that anxiety disorder.

Figure 1 presents a general schematic etiological model as a tentative heuristic for the discussion that follows. Tentative, because the existing research literature does not allow exact statements about the interrelations among the various mechanisms (e.g., mediational and interactive processes among the mechanisms), and thus needs detailed modifications based on additional research. The model is heuristic because it is possible that different anxiety problems may have specific mediational and interactive processes (see Ollendick & March, 2004 for examples of etiological models for each of the anxiety disorders). The model applied to a specific disorder is presented in Fig. 2 (see also e.g., Kendall, Pimentel, Rynn, Angelosante, & Webb, 2004). The figures suggest *potential* temporal ordering of the processes. For example, that biological factors may appear early in the development of anxiety disorders, that the interpersonal or social aspects are likely to affect some of the other processes, and that the cognitive and learning factors are proximal to the development of anxiety disorder and may be responsible for some specificity in disorder development. The arrows leading to normal anxiety in Fig. 1 are intended to point out that these processes do not invariably lead to a disorder. For example, certain social, cognitive and learning factors may protect those with a biological risk. In other words, there are biological, social, cognitive, and behavioral protection factors as well, (see Vasey & Dadds, 2001). It is important to note that all paths will not be important for all individuals and that for some individuals certain factors may more important than others. This means that the path coefficients are likely to take on different values across individuals and subgroups, and that the relationships may also be altered by moderators such as gender, ethnicity, and age. Nevertheless, the general set of relationships should hold across individuals and groups.

The following sections provide a review of the specific theoretical processes that together compose the integrative model proposed above with an eye toward identifying useful information for improving classification. In the following sections we make the argument that the empirical evidence to date suggests that the processes help to distinguish levels

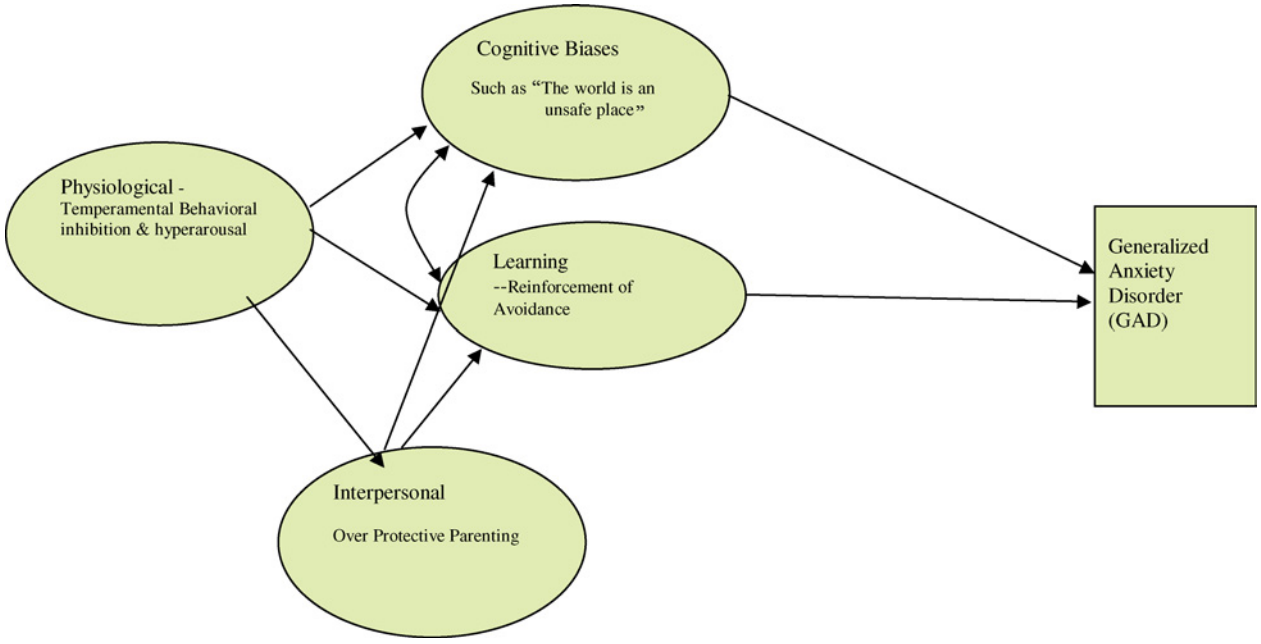

Fig. 2. Specific etiology of one individual's case of Generalized Anxiety Disorder.

of anxiety (e.g., high and low levels of anxiety, anxiety disordered youth from nondisordered youth) and that they are most likely important to understanding the etiology of childhood anxiety disorders (i.e., the evidence to date leads us to hypothesize that they are etiological). Casting a nomological net for childhood anxiety disorders involves drawing from each of the theories because a nomological model implies a foundation for the construct of childhood anxiety disorders in the processes from which they result.

Biological Theories

Genetic theories of childhood anxiety disorders posit that risk for anxious symptoms is transmitted genetically from parents to children. Cumulative research evidence suggests that there is a moderate genetic risk for symptoms of anxiety. Specifically, twin studies suggest that about 33% of the variance in measures of childhood anxiety can be accounted for by genetic influences. However, the genetic contribution may increase with age and may be greater for girls (see Eley, 2001 for a review). Genetic studies have also addressed the correlation between measures of anxiety and depression. Research conducted by Eley and colleagues also suggests a genetic basis for the comorbidity of anxiety and depression (Eley, 1997; Eley & Stevenson, 1999a, 1999b). Using 490 child twin pairs aged 8–16 years to examine the shared and specific etiological factors for anxiety and depression Eley and colleagues (Eley, 1997; Eley & Stevenson, 1999a, 1999b) found that all genetic variance was shared across the measures of anxiety and depression and that the shared genetic factor accounted for 80% of the correlation between anxiety and depression. Thus, biological accounts may help identify real (e.g., common etiology) versus artifactual (symptom overlap) comorbidity (Caron & Rutter, 1991) between anxiety and related disorders.

Unless molecular genetic research finds easily identifiable specific genes or sets of genes for anxiety disorders, genetic models can mainly inform classification efforts indirectly through related biological theories. However, family history may be an important factor to consider in the assessment of childhood anxiety disorders. Findings from family studies suggest that youth whose parents are diagnosed with an anxiety disorder are at risk for developing an anxiety disorder themselves (e.g., Berg, 1976; Moran & Andrews, 1985; Silverman, Cerny, Nelles, & Burke, 1988; Turner, Beidel, & Costello, 1987). For example, Turner et al. (1987) examined the prevalence of anxiety disorders (via semistructured interview and self-report measures) in the offspring (aged 7–12) of parents with an anxiety

disorder (agoraphobia or OCD) ($n = 16$), dysthymic disorder ($n = 14$), and normal controls ($n = 13$). Findings indicated that the children of parents who were diagnosed with an anxiety disorder were over two times as likely to meet *DSM-III* criteria for an anxiety disorder relative to the offspring of parents who were diagnosed with dysthymic disorder and seven times more likely than offspring of the normal controls.

In terms of biological theories, temperament theorists have drawn from genetic models and have posited the origins of anxiety problems in biological predispositions to react negatively to novel situations or stimuli (e.g., behavioral inhibition, see Biederman et al., 1990, 1993; Hirshfeld, Rosenbaum, & Biederman, 1992; Kagan, Reznick, & Gibbons, 1989; Kagan, Reznick, & Snidman, 1987; Kagan, Reznick, & Snidman, 1988; Rosenbaum, Biederman, & Gersten, 1988). Children characterized as behaviorally inhibited display many of the same behavioral, affective, and physiological characteristics as children with anxiety disorders. These characteristics include avoidance and withdrawal from novel situations, clinging or dependence on parents, fearfulness, and physiological hyperarousal (e.g., increased heart rate) when exposed to unfamiliar settings, people and objects (Kagan et al., 1987). Moreover, there is evidence that behavioral inhibition in infancy may be a risk factor for later anxiety disorders in childhood (Biederman et al., 1990, 1993).

Other biologically based theories emphasize the role of the brain function and neurochemistry. Functional neuroimaging (e.g., fMRI, EEG) studies suggest that normal threat assessment and emotional learning functions may involve differential hemispheric activation. For example, adults with PTSD have shown exaggerated activation of specific limbic regions such as the amygdala, but also deactivation of regions involved in cognitive functions, such as Broca's area (i.e., left inferior frontal gyrus), when compared to normal controls (Rauch, Shin, & Pitman, 1998). Electroencephalography (EEG) research has demonstrated increased right prefrontal and anterior temporal region activation in response to negative emotion, and predominately increased left prefrontal activation in response to positive emotion (Davidson, 1998). Davidson and colleagues (1998; Davidson, Marshall, Tomarken, & Henriques, 2000) have shown that increased left prefrontal activation is associated with the ability to suppress startle response to negative stimuli. Davidson and colleagues have also shown greater relative right prefrontal lobe activation in adults with social anxiety (Davidson et al., 2000). These findings have been replicated in infants (Davidson & Fox, 1989) and in samples of school age children aged 8–11 years diagnosed with anxiety disorders (Baving, Laucht, & Schmidt, 2002).

Similar to models of pathological anxiety which implicate differential brain activation are models emphasizing the hypothalamic–pituitary–adrenal (HPA) axis and amygdala in the pathophysiology of anxiety disorders. In these models, anxiety disorders result from an exaggeration of normal anticipatory anxiety. This exaggeration of normal anticipatory anxiety involves increased physiological reactivity (e.g., heightened sympathetic arousal), increased neuroendocrine activity (e.g., elevated levels of glucocorticods such as cortisol) and increased reflexive responsiveness to stimuli (e.g., exaggerated fear-potentiated startle). Research has begun to test these models in youth. Granger, Weisz, and Kauneckis (1994) investigated anxiety and neuroendocrine reactivity in a sample of clinic-referred children ($n = 102$, aged 7–17 years). Results indicated that youth who were high cortisol reactors in response to a social challenge ($n = 25$) compared to low cortisol reactors ($n = 25$) were more likely to report higher levels of social anxiety. Moreover, children diagnosed with PTSD have been shown to have increased urinary free cortisol levels after 24-hr urinary collection compared to matched controls (De Bellis et al., 1999) and have been shown to have increased diurnal salivary cortisol levels compared to matched controls (Carrion et al., 2002).

Biological models of anxiety implicate several constructs important to the nomological net of childhood anxiety. These include traits such as behavioral inhibition, trait anxiety, exaggerated fear-potentiated startle, cortisol reactivity, and physiological markers of arousal or relaxation (e.g., heart rate, heart-rate variability, skin conductance). Drawing these influences together, biological models suggest that a child may inherit traits such as behavioral inhibition or trait anxiety (Kagan et al., 1987). Such children are more likely to respond to potentially fearful situations (e.g., interaction with a stranger, strange situation, separation from mother, etc.) with heightened physiological reactions due to a lower threshold of reactivity in the amygdala and hypothalamus. Such evidence is largely based on differences in cortisol and norepinephrine levels in children behaviorally classified as inhibited and uninhibited. However,

animal studies utilizing selective breeding techniques have also supported these contentions (see Manassis & Bradley, 1994, for a review).

Biological models provide important contributions to the nomological net of childhood anxiety, however, other aspects are equally important. To illustrate, although there is evidence that behavioral inhibition in infancy may be a risk factor for later anxiety disorders in childhood, not all children with early childhood behavioral inhibition develop an anxiety disorder (Biederman et al., 1990, 1993). For example, behaviorally inhibited children have higher rates of each of the anxiety disorders compared to the uninhibited and healthy controls (28% of children with behavioral inhibition met criteria for anxiety disorders compared with 0% of uninhibited children and 0% of the healthy control children; Biederman et al., 1990). Yet, more than 70% of the children with early childhood behavioral inhibition did not develop an anxiety disorder (Biederman et al., 1990). Thus, understanding the other processes that combine with biological risk such as behavioral inhibition to produce anxiety disorders is needed.[7] Moreover, biological accounts do not currently have the specificity to alone inform classification efforts. As noted above, twin studies have indicated that genetic variance is largely shared across measures of both anxiety and depression (Eley, 2001). Moreover, the HPA axis and amygdala have been implicated in depression and conduct disorder as well as in anxiety disorders (Vasey & Dadds, 2001), and thus may be limited in helping to distinguish anxiety and depression without the help of other processes.

Behavioral Theories

Behavioral views have proposed respondent (classical or Pavlovian conditioning), vicarious (Bandura's modeling) and operant (Skinnerian conditioning) accounts of the acquisition of anxiety disorders. Limitations to early classical conditioning accounts involving direct aversive paring of stimuli have prompted theorists to posit multiple learning pathways to anxiety and phobic disorders. Rachman (1977) has posited three major pathways to anxiety. The first is through aversive conditioning. This pathway suggests that fear and anxiety in childhood can be acquired through the pairing of previously neutral stimuli with aversive, traumatic or subtraumatic stimuli or events (Wolpe & Rachman, 1960). The second is vicarious acquisition through observational learning or modeling. This pathway suggests that children may acquire fears by observing the actions of salient others such as parents, other caregivers, siblings, or friends (i.e., modeling, Bandura, 1982). The third pathway is through the verbal transmission of information. This pathway suggests that children may acquire fears by talking about fearful things with parents, caregivers, siblings, or friends. Ollendick, Vasey, and King (2001) suggest that there is a fourth major learning pathway to childhood anxiety problems and that is through operant conditioning processes. This account suggests that if a child learns to cope with normative anxiety and fear responses through avoidance of the anxiety or fear provoking stimuli, then normal anxiety responses may be maintained at high levels and can thus turn into problematic anxiety. Withdrawal from the stimuli may be negatively reinforced by reduction in levels of anxiety after withdrawing, or avoidance may be positively reinforced by caregivers through approval of avoidance behaviors. Considerable evidence exists to support these learning pathways in childhood anxiety (see Ollendick et al., 2001 for a review) and new classical conditioning models are responding to past criticisms.

Bouton, Mineka, and Barlow (2001), argue that criticisms of early learning models of anxiety disorders, particularly to panic and panic disorder, have been directed at limitations of conditioning theory that have been addressed by modern research. Recent research reveals a surprisingly complex learning process that requires a number of considerations not addressed by early accounts of classical conditioning. In particular, research shows that a conditioned response (CR) such as anxiety or fear is not an inevitable consequence of pairing a conditioned stimulus (CS) with an unconditioned stimulus (US). For example, Rachman's (1977) criticism of classical conditioning and learning theory in the etiology of anxiety disorders used the argument that air raids during World War II in London did not always cause an increase in anxiety disorders. Modern research indicates that potential conditioned stimuli (CS) might not cause fear conditioning if the potential CS were familiar rather than novel. Moreover, if the event (e.g., air raid) was experienced in the presence of

[7]This estimate may also be weak because of problems with the validity and reliability of the diagnostic assessment of anxiety disorders in youth.

safety cues that inhibited fear (e.g., bomb shelters, family), or if the raids were signaled by other cues such as sirens then conditioning could have been blocked (Bouton, 2001).

An additional criticism of classical conditioning in the etiology of anxiety disorders has been directed at the fact that fears appear to be disproportionately directed toward certain stimuli (e.g., spiders and snakes) and not others (e.g., dish towels). This fact appears to be addressed well by the concept of preparedness. That is, that some stimuli are especially salient and powerful signals because biological evolution has made them that way (Bouton, 2001; Bouton et al., 2001). For example, it has been demonstrated that monkeys are easily conditioned to fear snakes, but more resistant to conditioning fear with other cues such as flowers (Mineka, 1992). Drawing from advances in classical conditioning models of anxiety, an especially important aspect of learning theory and anxiety appears to be its role in conjunction with other etiological processes. The salience of interoceptive and exteroceptive cues may be heightened by certain cognitive mechanisms such as anxiety sensitivity (discussed later) or biological preparedness. Given a vulnerability to associate such cues with anxiety or panic, conditioned anxiety may potentiate future panic attacks, leading to a cycle of panic, fear of panic, heightened anxiety, and increased panic. Thus, it appears that fear conditioning is a complex process that is influenced by a range of various and possibly interacting vulnerabilities (Bouton et al., 2001).

Finally, it is important to note that the context within which learning occurs appears to have powerful and lasting effects not only on anxiety and anxious symptomology, but also on treatment response as well. Extinction plays a central role in treatment of anxiety related conditions. Extinction and counterconditioning do, in fact, reduce anxiety symptoms in youth (Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999). What basic research suggests they do not do, however, is to completely eliminate the original learning, which remains encoded and will return to behavior given the original context of the learning (the situation, mood, or state in which learning occurred), or if the current context is associated with the US (Bouton, 2000, 2001). Even conditioned responses that appear to have been eliminated by extinction can reappear once time passes before the CS is presented again (Bouton, 2001).

In sum, an integrative model of childhood anxiety disorders must include learning factors in order to understand how anxious responses develop, how these behaviors are maintained over time, lapse and relapse, and are most effectively prevented and treated (Bouton, 2000, 2001). Learning processes also imply important anxiety mechanisms such as avoidance and withdrawal.

Cognitive Theories

Cognitive and information processing models propose that understanding the processing of information at various stages in the cognitive system, such as encoding, interpretation, and recall can help to elucidate the etiology and maintenance of anxiety disorders. More specifically, these models of anxiety propose that anxious children have biased interpretation, judgment, and memory as well as attentional selectivity (Vasey & MacLeod, 2001). Each of these is explored below. In conjunction with biological and learning accounts, cognitive factors may foster or hamper learning acquisition, exacerbate biological predispositions, and maintain anxiety disorder symptoms.

Interpretive bias involves having disproportionately negative interpretations of ambiguous or potentially threatening stimuli or situations. Negatively biased cognitions are thought to be core processes in emotional problems such as anxiety disorders (Beck, 1976, 1985; Ellis, 1962). Catastrophizing is thought to be one such bias and involves expecting the worst possible outcome of an event or situation. Research indicates that interpretative biases such as catastrophizing are associated with self-reported anxiety in youth, although age may moderate the strength of this association (i.e., the association may be weaker in young children; Weems et al., 2001). Moreover, research has shown that clinically anxious youth presented with ambiguous vignettes, and then asked to explain what was happening in the story are more likely to provide interpretations indicating threat than are nonanxious controls (Barrett, Dadds, & Rapee, 1996; Chorpita, Albano, & Barlow, 1996). Catastrophic interpretations of threat and of anxiety sensations themselves are a form of negative thinking associated with anxiety disorders. For example, anxiety sensitivity refers to beliefs that anxiety related sensations have severe and negative consequences (Reiss, 1991). Anxiety sensitivity prospectively predicts the development of panic attacks in both young adult (e.g., Schmidt, Lerew, & Jackson, 1997; Schmidt, Lerew, & Jackson, 1999) and adolescent

samples (Hayward, Killen, Kraemer, & Taylor, 2000; Weems, Hayward, Killen, & Taylor, 2002).

Judgment bias involves negative and or lowered estimates of the individual's coping ability or style. Judgment biases in children refer to lowered expectations of their ability to handle threatening situations or to choose coping strategies such as avoidance that maintain anxiety. Research has suggested that perceived control over anxiety related events is significantly negatively correlated with self-reported anxiety symptoms and that youth with anxiety disorders report significantly lower perceived control about anxiety related events than nonreferred comparison participants (Weems, Silverman, Rapee, & Pina, 2003). Moreover, clinically anxious youth are more likely to choose avoidance responses when presented with vignettes depicting threatening situations (Bell-Dolan, Foster, & Christopher, 1995). What is not known is whether judgment biases predict the development of anxiety disorders.

Memory bias involves recalling disproportionately negative information about oneself, past situations or events. Again memory biases are similar to the other biases so far reviewed. The distinction we use for this review is that memory biases center on the recall or recognition of past experience or information. A variety of memory strategies have been employed and so the literature is characterized by diverse methodology. Some studies have examined the relation between general deficits in memory and anxiety, whereas others have examined bias in the recall of emotional information (e.g., positive, negative, neutral). Support for a general memory bias associated with anxiety disorders has been demonstrated (e.g., Moradi, Neshat-Doost, Taghavi, Yule, & Dalgleish, 1999; Pine, Wasserman, & Workman, 1999). In terms of emotional memory biases, Daleiden (1998), found that, controlling for depression, anxiety predicted memory biases for negative relative to neutral information for conceptual but not perceptual tasks, and predicted memory bias for positive relative to neutral on procedural tasks but away from positive relative to neutral on declarative tasks.

Selective attention involves predominantly attending to threatening stimuli when such stimuli are placed in a context with neutral or other nonthreatening stimuli. A large number of studies have supported the contention that youth with elevated anxiety show an attentional bias toward threat (see Vasey & MacLeod, 2001 for a review). For example, Vasey, Daleiden, Williams, and Brown (1995) examined biased attention in childhood anxiety disorders with a sample of children meeting diagnostic criteria for anxiety disorders ($n = 12$) and control subjects matched on age, gender, and intellectual ability who did not meet diagnostic criteria for any psychological disorder ($n = 12$). Results indicate that children with anxiety disorders demonstrated biased attention directed toward threatening stimuli (i.e., threat words) but that nonanxious controls did not show a bias away from threat words.

In sum, cognitive models emphasize biased interpretation, judgment, and memory as well as attentional selectivity in the etiology and maintenance of anxiety disorders in youth and so implicate several constructs important to the nomological net of childhood anxiety. A number of studies have shown that these cognitive processes are associated with anxiety and differentiate youth with anxiety disorders from nonanxious youth. Some longitudinal data suggest possible etiological roles, however, additional prospective research is needed to more firmly establish etiological versus concurrent associations.

Interpersonal/Contextual Theories

Interpersonal theories posit that factors (e.g., biological, learning, and cognitive) influencing the development of anxiety in youth do not occur in isolation. Rather, the social, interpersonal environment of the child determines how and which potential influences impinge on the child. Moreover, social contextual approaches posit that factors such as poverty, parental psychopathology, exposure to trauma, and exposure to violence can exacerbate vulnerability to developing an anxiety disorder. Although the specific mechanisms whereby interpersonal/contextual factors exert their influence on childhood anxiety are less clearly understood, there are models that may serve to inform the nomological net.

Attachment theory, for example, posits that the child's interactions with the environment are influenced by the underlying quality of the parent child relationship and that a number of factors influence the quality of the parent–child relationship (e.g., poverty, parental psychopathology). Attachment theory suggests that human infants form an enduring emotional bond with their caretakers (Bowlby, 1977; Cassidy, 1999). When the child's caretakers are responsive to their needs, this emotional bond can

provide a lasting sense of security that continues even when the caretaker is not present. However, an inconsistently responsive caretaker, a neglectful caretaker or some other disruption in the attachment bond can cause the child to become insecurely attached. These early attachment relationships are hypothesized to set a template for future relationships throughout the infant's development into childhood, adolescence, and adulthood (Hazan & Shaver, 1987). Children with insecure attachment have particular difficulty during separations from their parents. For example, children in the strange situation research paradigm, characterized with "ambivalent" attachment bonds, display intense protests upon separation and are hard to comfort or console upon the parent's return (Ainsworth, Blehar, Waters, & Wall, 1978).

The reaction of children with anxiety disorders such as Separation Anxiety Disorder (SAD) to separation from parents is strikingly similar to that reported of insecurely attached children in the strange situation (Ainsworth et al., 1978; Lyons-Ruth & Jacobvitz, 1999; Main & Solomon, 1990). For example, children with SAD protest desperately when separation is imminent, cry and become agitated during separation and then may act angrily or aggressively towards the parent upon return (Ollendick, Lease, & Cooper, 1993). Warren, Huston, Egeland, and Sroufe (1997) found that children ($n = 172$) classified as having anxious/resistant insecure attachment (assessed at 12 months of age) were more likely to have anxiety disorders at age 17, even when controlling for measures of temperament and maternal anxiety, than children classified with other types of attachment. Research on attachment theory has been criticized; however, because of poor stability and because a number of studies have failed to show long-term prediction (see Greenberg, 1999).

Parental control is also thought to influence childhood anxiety. For example, the presence of anxiety in either member of the mother-child dyad tends to elicit maternal overcontrol during interactions (Whaley, Pinto, & Sigman, 1999; Woodruff-Borden, Morrow, Bourland, & Cambron, 2002). Research conducted on mother-child interactions and anxiety consistently demonstrates higher levels of maternal control in anxious mother-child dyads as opposed to control dyads (see Silverman & Ginsburg, 1998). Empirical research also suggests that children's perceptions of maternal control may be as important as the mother's actual observed behavior and are convergent with observer ratings of maternal behavior. For example, Siqueland, Kendall, and Steinberg (1996) found that both children (aged 9–12 years) and observers rated mothers of clinically anxious children ($n = 17$) as significantly more controlling than nonanxious children and their mothers ($n = 27$).

In terms of a potential mediating role, Whaley et al. (1999) compared 18 clinically anxious mothers and their 7–14-year-old children to 18 non-anxious mothers and their children and found that anxious mothers were more psychologically controlling than non-anxious mothers during interactions with their child. Results indicated that the behaviors (e.g., psychological control) displayed by the anxious mothers during interactions with their child were stronger predictors of child anxiety than maternal anxiety. Although formal tests for mediation were not conducted in Whaley et al. (1999), the authors concluded that this finding makes it plausible to hypothesize that these types of parenting behaviors "may be potential mediators of the association between maternal and child anxiety" (pp. 834).

In sum, interpersonal models have demonstrated long-term prediction of anxiety disorders and may provide a context for differentiating normal separation protest from pathological separation anxiety. Although attachment theory provides a conceptual model of how interpersonal factors can spark or exacerbate anxiety problems, research is needed to replicate the predictive findings and clarify if or what type of insecure attachment is specifically predictive of anxiety problems before attachment theory can optimally inform the nomological net. Research examining the proposition that interpersonal distrust (e.g., expectations that other people will be untrustworthy and rejecting) may exacerbate potentially threatening events or foster negative anxiety sensations may be important in this regard (see Weems, Berman, Silverman, & Rodriguez, 2002).

UTILIZING PROCESS THEORY CONSTRUCTS IN THE ASSESSMENT AND CLASSIFICATION OF CHILDHOOD ANXIETY DISORDERS

Before discussing the use of etiological processes in improving classification and assessment some caveats about the etiology of childhood anxiety should be noted. Broadly, the hypothesized underlying mechanisms (i.e., indicators of the various

processes delineated above) may be responsible for the "disorder" (i.e., they may be concurrently responsible for the problem, maintain the problem, or may be etiological risk factors), or may be important markers or signs of the disorder. The current state of research does not currently allow conclusive causal statements to be made. Additional research is needed to extend models similar to those presented in Figs. 1 and 2. For example, research is needed to make the latent variables that compose the four major domains (i.e., biological, social, cognitive, behavioral) more explicit and their interrelations clearer. That is, we need to be able to specify the variables representing the major processes and to also include the indicators of these processes in a more formal structure of associations. However, we argue that there is enough information to let us begin to use process theories to improve classification. We suggest that this is necessary at this point because it is plausible that the reason we don't have clearly supported comprehensive etiological models of childhood anxiety is because the diagnoses are ineffective dependent variables (i.e., we may never have good comprehensive etiological models if the validity of the criterion for testing etiology is poor). For example, what if separation anxiety disorder is not an "outcome" but part of a developmental process that leads to chronic anxiety problems?" (e.g., see Westenberg, Siebelink, & Treffers, 2001 for relevant discussion)

Figure 2 suggests specific mechanisms hypothetically leading to Generalized Anxiety Disorder. Although each of the pathways are likely to be relevant for understanding anxiety generally, all of the pathways and indicators may not be equally relevant for everyone—that is the broad processes likely all apply in varying degrees, but the specific indicators of those processes will vary across individuals. The models presented in the Figures point out how utilizing the indicators of the processes thought to result in anxiety disorders (i.e., the mechanisms) may help specify important targets for intervention. Thus, classifying anxiety disorders around the mechanisms salient in the etiology and maintenance, as well as the symptoms, would be much more relevant to clinical purposes than the current *DSM* system. How utilizing mechanisms in the classification of anxiety disorders may help clarify issues of comorbidity, predictive and discriminant validity is discussed below and in subsequent sections.

A nomological understanding of childhood anxiety disorder assessment is not primarily descriptive or etiological. The processes discussed above form (in part) the hypothetical theoretical structure discussed by Cronbach and Meehl (1955) in their proposed desiderata for construct validation. However, the theoretical structure of anxiety disorders (i.e., nomological net) is comprised of numerous specific domains. As noted above, and drawing from Kanfer and Saslow (1969), we consider the two major subdivisions of these domains as (1) the symptoms of anxiety disorders (e.g., *DSM-IV* descriptive diagnostic criteria), and (2) the mechanisms of anxiety disorders (e.g., physiological responses, affect, behavior, and cognitive processes). Yet, the associations between hypothesized mechanisms and the current diagnostic criteria may take various forms. For example, they may have discriminative (e.g., distinguish depression from anxiety, or one type of anxiety disorder from another), predictive, or convergent associations.

At the most basic level, utilizing the nomological net suggests that tests of the validity of the childhood anxiety disorders should involve examining the relative stability, convergent and discriminant validity, and relative predictive ability of both the symptoms and the mechanisms to outcomes. In other words, the stability of anxiety disorders must be understood in terms of the *stability* of the symptoms that compose the anxiety disorder diagnostic criteria and also in terms of the *stability* of hypothesized mechanisms of anxiety. In addition, the long-term *outcomes* of anxiety disorders must also be understood in terms of the long-term *outcomes* of the symptoms that comprise the disorders as well as the mechanisms of anxiety. For example, if intense worry (assessed with a worry interview), which is theorized to be an important cognitive component of childhood anxiety disorders such as Generalized Anxiety Disorder, predicts negative outcomes in later life but the diagnosis of Generalized Anxiety Disorder does not (assessed with a *DSM*-based diagnostic interview), then there may be something wrong with the Generalized Anxiety Disorder diagnostic criteria for youth. For instance, the criteria may not tap intense worry or age related worries adequately, and thus greater or more developmentally accurate inclusion of the mechanism of worry in the diagnostic criteria is needed.

The approach we are suggesting, based on incorporating the nomological net, also implies a slightly different mind set from the typical approach to relating childhood anxiety disorders to their outcomes (i.e., anxiety → functional outcome). The

model proposes rethinking the typical sequences in anxiety disorder development and testing alternative hypotheses about the associations between mechanisms and symptoms. The typical model views a hypothesized mechanism leading to an anxiety disorder and then the anxiety disorder leading to for example, poor academic outcomes. However, the current state of empirical knowledge does not yet justify these types of tests alone, given the likelihood that current definitions of the childhood anxiety disorders are lacking in validity. Our view suggests also testing whether risk mechanisms-when added to the symptoms of anxiety disorder-better predict outcomes. If they do, then aspects of the mechanism may need to be incorporated into the definition of the anxiety disorder. Symptoms that are not predictive may need to be dropped from the definition or otherwise qualified in the definition of the disorder with regard to prognosis (cf. Kanfer & Saslow, 1969).

As discussed above, there is a need to know the prototypical elements that comprise useful definitions of the childhood anxiety disorders and a need to know if drawing from etiological models can facilitate useful classification. This may involve dismantling the symptoms and diagnoses in the *DSM* and then reconstructing them with new information. We propose a heuristic to guide conceptualizing how the mechanisms should be related to existing symptoms with regard to improving definitions of the anxiety disorders. Specifically, the associations should be additive with regard to classification. In other words, mechanisms that add to the prediction of functional outcomes or stability may need to be included in the definitions of the disorders. However, there is also a need to identify independent etiological factors. We propose that mechanisms that mediate or moderate the stability of symptoms and/or mediate or moderate the association of symptoms to outcomes may need to remain as separate constructs (i.e., should not included in the definition of the disorders). This does not imply that such factors are unimportant to assess. On the contrary, we argue that mediating and moderating factors need to be assessed in concert with diagnostic criteria to inform understanding of stability and prediction of long-term outcomes, as well as to inform prevention and intervention efforts.

In the next section we point out how various constructs drawn from processes theories (i.e., mechanisms) can help clarify issues of comorbidity, discriminant, and predictive validity.

UTILIZING THE CONTENT OF THE NOMOLOGICAL NET FOR IMPROVING CLASSIFICATION

A full review of the research literature on each of the potential components of the childhood anxiety disorders (or a full review of all potential components for that matter) is beyond the scope of a single review. Thus, the following is primarily intended to be illustrative of how a greater utilization of the mechanisms implied by the process theories may improve classification, how a nomological net model that draws from process theories of childhood anxiety disorders may be tested, and also to sketch the implications of testing the model. We start with a critical analysis of the *DSM* childhood anxiety disorder symptoms. We then examine specific mechanisms drawn from biological, behavioral, and cognitive process theories.

Symptoms

The symptoms that currently compose the anxiety disorders in *DSM-IV* have some empirical support from reliability studies of assessment instruments and factor analyses of anxiety disorder symptoms. Overall, there appears to be fairly good evidence to suggest that the *DSM-IV* anxiety disorder symptoms in childhood can be assessed with an adequate degree of short-term test–retest reliability. Saavedra and Silverman (2001) recently provided an extensive review of the diagnostic assessment of childhood anxiety disorders and concluded that although the validity of the classification system is in need of additional research and conceptual attention, the research to date suggests that the there is relatively better evidence for reliability of the current instruments used to assess the *DSM* symptoms than for validity. For example, test–retest reliability kappa coefficients of .78–.80 have been reported for present and lifetime diagnoses of generalized anxiety disorder/overanxious disorder, and Specific Phobia with the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; e.g., Kaufman et al., 1997; see Ambrosini, 2000).

Data from the Anxiety Disorders Interview Schedule for *DSM-IV*: Child and Parent Versions [(ADIS for *DSM-IV*; C/P; Silverman & Albano, 1996) and its previous edition (ADIS for *DSM-III-R* C/P; Silverman & Nelles, 1989)] also points to the reliability of assessing anxiety disorder symptoms

in youth. The ADIS has been the subject of several test retest reliability studies (e.g., Silverman & Nelles, 1989; Silverman & Rabian, 1995; Rapee, Barrett, Dadds, & Evans, 1994) each demonstrating good estimates of reliability for childhood anxiety disorders. Most recently, Silverman, Saavedra, and Pina (2001) found the ADIS for *DSM-IV* to have kappa's ranging from .80–.92 for 2–3 week test–retest reliability for separation anxiety disorder, social phobia, specific phobia, and generalized anxiety disorder.

It is important to consider, however, that reliability has been shown to vary by age and reporter. Some studies show poorer reliability for young children and some poorer reliability for adolescents (Silverman & Eisen, 1992; Silverman & Rabian, 1995). In terms of child versus parent report, child reports of social phobia tend to be less reliable than parents' reports of social phobia, and in general social phobia tends to have low reliability (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000 reported kappa = .25 for children and .54 for parents). Moreover, poor levels of agreement have been found across parents and children (Grills & Ollendick, 2003). Such findings may lead to the suggestion that the parent should be used for more reliable reports. However, this may be problematic in terms of validity. It should also be noted that levels of agreement between children and adult informants such as parents and teachers, are identical to levels of agreement between adult patients and other adult informants, when assessing psychopathology (Achenbach, Krukowski, Dumenci, & Ivanova, 2005). Results from meta-analysis on multiple informants in adult psychopathology suggest that past conclusions about children as less reliable than adults should be questioned.

Additionally, characteristics of the parent may influence how they rate their child's behavior. For example, research has suggested an association between parental depressive and anxious symptoms and parent overreporting (e.g., overestimating the severity) of their child's behavioral problems (Krain & Kendall, 2000; Frick, Silverthorne, & Evans, 1994). In terms of anxiety, Krain and Kendall (2000) found that, in a sample of 239 children aged 7–15 years diagnosed with a primary anxiety disorder, maternal and paternal depressive symptoms were related to overreporting their children's anxiety levels. Similarly, Frick et al. (1994) found that maternal anxiety was related to the mothers' ratings of childhood anxiety symptoms in a diagnostically diverse sample of 95 clinic-referred children aged 6–13 years.

A major question that has not been addressed is whether the symptoms of anxiety in youth are really taxonic as opposed to dimensional. If they are not dimensional, researchers really shouldn't expect stability of the disorders, but still could expect stability of certain symptoms. Symptom scores appear to have stronger reliability estimates than the disorders over 2–3 weeks (e.g., see Silverman et al., 2001). Taxometrics is a branch of applied mathematics that can be used to facilitate the classification of entities as it involves a set of statistical procedures that can be used to test the latent structure of constructs (Beauchaine, 2003; Meehl, 1995). Although we are aware of no studies that have been conducted with the childhood anxiety disorders, initial results suggest taxonicity for the construct of anxiety sensitivity in youth (Bernstein, Zvolensky, Weems, Stickle, & Leen-Feldner, in press). Finally, it is important to consider that, although there is evidence for the reliability of the anxiety disorder diagnoses of childhood, the utility of "reliability" is considerably minimized if validity is lacking in the definitions.

Existing factor analytic studies provide some support for the validity of the anxiety disorder symptom clusters. Spence (1997) examined the factor structure of anxiety disorder symptoms in two large community samples ($n = 698$) of youth (aged 8–12 years). Anxiety disorder symptoms were assessed with a 38-item self-report measure of anxious symptoms derived from clinical experience, existing assessment measures, and the *DSM-III-R* and IV criteria. Results supported a model of the structure of anxiety disorders in youth consistent with the *DSM-IV* diagnostic categories. More recently, Chorpita, Yim, Moffitt, Umemoto, and Francis (2000) examined the structure of the Revised Child Anxiety and Depression Scales that is an adaptation of the Spence Anxiety Scales (Spence, 1997). Chorpita, Yim, et al. (2000) modified the Spence scales to include depression and evaluated the factor structure of the anxiety disorder symptoms in a school sample of 1641 children and adolescents. The results suggested an item set and factor definitions that demonstrate a structure consistent with *DSM-IV* anxiety disorders and depression. However, if these item sets only comprise part of the prototypical elements of childhood anxiety disorders, the validity of the factor structure findings is compromised.

In sum, there is some evidence for the anxiety disorder symptoms as conceptualized in the

DSM-IV. As noted, however, the inconsistencies with regard to long-term stability and functional outcomes discussed above underscore the need for improvement in assessment and classification (see also Saavedra & Silverman, 2001). Limitations also include inadequate information on the taxonic status, sample variations, and informant bias. Drawing from the nomological net, we propose that advancing the understanding of the definition of anxiety disorders in youth requires that they be understood in the context of additional facets of the phenomena (i.e., the mechanisms of the disorder). We argue that the mechanisms discussed below are important to include in tests of the stability and long-term outcomes of childhood anxiety disorders as they point to facets that might need to be added to or used to modify the diagnostic criteria. The assessment of mechanisms also points to different modalities of assessment.

MECHANISMS

Biological

Negative Affectivity and Hyperarousal

Adequate inclusion of the concepts of negative affectivity and hyperarousal in assessment and classification may be essential for the childhood anxiety disorders. The tripartite model of anxiety and depression (Clark & Watson, 1991a) suggests that hyperarousal differentiates anxiety from depression and anhedonia (low positive affect, or PA) differentiates depression from anxiety, whereas generalized negative affect (NA) characterizes both depression and anxiety. Work has begun to examine this model in childhood. For example Laurent et al. (1999) developed a child version of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS-C was developed using a large sample of school children (grades 4–8; $N = 707$). The Negative Affect (NA) and Positive Affect (PA) scales demonstrated good convergent and discriminant validity with existing self-report measures of childhood anxiety and depression.

Joiner and Lonigan (2000) have examined part of the tripartite model in child and adolescent psychiatric inpatients (aged 7–17 years) using the PANAS-C. Results suggest differences among children in PA and NA were associated with depressive vs. externalizing diagnostic status. Depressive disorder diagnoses were associated with the combination of low levels of PA and high levels of NA. Changes over time in depression, but not in anxiety, also were associated with the combination of low PA and high NA consistent with theory. Less work has been done on the hyperarousal aspect of the model.

Chorpita and colleagues (Chorpita, Daleiden, Moffitt, Yim, & Umemoto, 2000; Daleiden, Chorpita, & Lu, 2000) have developed a self-report measure, the Affect and Arousal Scale for Children (AFARS; Chorpita, Daleiden et al., 2000). The AFARS assesses PA, NA, and physiological hyperarousal (PH). Data from 1,289 school-aged youth suggest the structural validity of the scale. Confirmatory factor analysis in an independent sample of 300 children and adolescents (age 6.2–18.2 years) suggested a factor structure consistent with the tripartite model.

The hyperarousal facet of the nomological net suggests an important component that should be considered in validation studies of the childhood anxiety disorders, but it also highlights an important modality for assessment (i.e., physiological arousal, startle reactivity). Including components in the nomological net such as hyperarousal thus helps to point toward additional procedures to include in the assessment of childhood anxiety disorders. Although research is needed to examine physiological indices of hyperarousal as conceptualized in the tripartite model, research does suggests that childhood anxiety disorders may be characterized by differences in physiological responding such as heart rate reactivity and cortisol levels (Beidel, 1991a; Carrion et al., 2002; Gunnar, 2001). Thus, in addition to self-report, parent report, teacher report, and interview methods of assessment, it may also be important to include physiological measures in the assessment of childhood anxiety. Research is needed to examine if such indices can be used to facilitate diagnosis.

Trait Anxiety

Theories concerning the origin of childhood anxiety problems often posit that anxiety or fear reactions are due to traits, biological predispositions, or temperamental differences. As noted, early behavioral inhibition does appear to be predictive of anxiety disorders in childhood (e.g., Biederman et al., 1990). In older youth, the concept of trait anxiety is a salient feature of anxious youth. Measures of trait anxiety show remarkable stability over time. For example, the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978) appears to be very stable across time. Reynolds

(1981) reported a 9-month test–retest correlation of .69 in a sample of 534 school children (grades 4–6). Ialongo, Edelsohn, Werthamer-Larsson, Crockett, and Kellam (1995) found that children's RCMAS scores in first grade were significant predictors of the children's fifth grade anxious symptoms in a sample of 684 school children. Weems and Silverman (1999) also found the RCMAS to be relatively stable ($r = .37$, $p < .05$) in a sample of 44 school children assessed 7 years apart (children were aged 7–12 years at time 1 and aged 14–18 years at time 2). Assessing behavioral inhibition and or trait anxiety may thus provide a good benchmark mechanism by which to gauge the relative stability of the symptoms of anxiety disorder as well as other core features of anxiety disorders over time.

Behavioral

Avoidance and Withdrawal

The concepts of avoidance and withdrawal probably have the strongest support for their role as a mechanism in childhood anxiety disorders. Research has demonstrated that behavioral techniques using gradual exposure (i.e., nonavoidance) are effective in treating anxiety and phobic disorders in children (Barrett et al., 1996; Kendall, 1994; Kendall et al., 1997; Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999; Silverman, Kurtines, Ginsburg, Weems, Rabian, et al., 1999). Moreover, research shows that childhood anxiety is associated with withdrawal from anxiety provoking stimuli, as well as withdrawal from activities and social interactions (Bell-Dolan et al., 1995; Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999).

This facet of the nomological net suggests an important component to consider in terms of the classification of anxiety disorders and also, as with hyperarousal, highlights an additional and important modality for assessment (i.e., behavioral observations). The Behavioral Avoidance Task (e.g., Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al., 1999; Beidel, 1991a, 1991b) has been used to assess avoidance of specific anxiety disorder-related stimuli and has been shown to differentiate youth with anxiety disorders from nonanxious controls (Beidel, 1991b). Including components in the nomological net such as avoidance thus helps to point toward additional avenues to include in the assessment of childhood anxiety disorders, as well as help to specify specific dysfunctional behavioral repertoires.

Cognitive

Control

Several investigators have suggested an important role of control in anxiety and anxiety disorders in youth (e.g., Capps, Sigman, Sena, Henker, & Whalen, 1996; see Chorpita & Barlow, 1998 for theoretical review). Research on the concepts of locus of control, learned helplessness and attributional style, self-efficacy, and perceived control suggests that control, in general, is important to understanding anxiety disorders in youth (see Chorpita & Barlow, 1998).

Barlow's (1988, 2002) model of anxiety suggests that a perceived lack of control over "external" threats (i.e., events, objects or situations that are fear producing for an individual) and/or control over negative "internal" emotional and bodily reactions are central to the experience of anxiety problems. That is, beliefs that anxiety related events and sensations are uncontrollable is part of what makes anxiety a "problem" for individuals with anxiety disorders. In other words, nonpathological anxiety in individuals who do not have anxiety disorders is differentiated from pathological anxiety by heightened levels of anxiety in response to the experience of threatening situations, but also by the belief that those events are uncontrollable. Empirical support exists for this conceptualization of control in youth (Weems et al., 2003).

Weems et al. (2003) investigated the role of control beliefs in childhood anxiety disorders using a developmentally modified version of Rapee, Craske, Brown, & Barlow (1996) Anxiety Control Questionnaire in a sample of 117 youth aged 9–17 years. Findings indicated that perceived control over anxiety related events was significantly negatively correlated with youth self-reported anxiety symptoms, and that youth with anxiety disorders ($n = 86$) reported significantly lower perceived control about anxiety than the nonreferred participants ($n = 31$). Moreover, results of logistic regression analysis indicated that perceptions of control over anxiety predicted anxiety disorder status even when controlling for existing measures of anxiety (i.e., RCMAS) and control (i.e., NSLOC). These results suggest that control beliefs specific to anxiety differentiate clinically anxious children from nonreferred children. However, they

also suggest that assessing anxiety control beliefs may add to understanding the clinical picture of anxious children by tapping an important aspect of anxiety disorders. Specifically, assessing control may help to specify dysfunctional interpretative repertoires that may be characteristic of anxiety disorders in youth.

Cognitive Errors and Attention Biases

As noted, a basic premise of cognitive models of psychopathology anxiety disorders stem from faulty or negative ways of thinking (Beck, 1976, 1985; Ellis, 1962). The patterns of faulty or negative thinking that characterize these emotional disturbances have been referred to as "cognitive errors" (e.g., catastrophizing, overgeneralization, personalizing and selective abstraction). Evidence suggests that anxiety symptoms in youth are associated with cognitive errors (e.g., Leitenberg, Yost, & Carroll-Wilson, 1986; Leung & Wong, 1998; Weems et al., 2001). Research also suggests that specific errors might differentiate anxiety and depression (Epkins, 1996; Weems et al., 2001). Including cognitive errors is important because, similar to control, assessing such errors may help to specify dysfunctional interpretative repertoires that may be characteristic of anxiety disorders in youth.

Assessing biased attention is another potentially useful way to get at important cognitive processes in youth. For example, Vasey et al. (1995) used a dot-probe detection task that measures the effect of threatening words on the direction of attention. Specifically, word pairs are briefly flashed on a computer screen and then a dot (i.e., the probe) is presented in the location of one of the words. Detection latencies are measured from the time of the disappearance of the words until the participant presses a button indicating detection of the probe. Biased attention toward threat is demonstrated by shorter detection latency when the dot appears in the location of the threat words versus neutral words, and biased attention away from threat is shown by longer detection latency when the probe appears in the place of the threat versus neutral words. As noted, results indicate that children with anxiety disorders demonstrate biased attention directed toward threatening stimuli (i.e., threat words). Such procedures suggest an additional modality for assessing cognitive processes in the assessment of childhood anxiety disorders. Research is needed to examine if such indices can be used to facilitate diagnosis.

Contextual

Inclusion of contextual or situational factors may also serve to define anxiety problems. However, because the specific mechanisms whereby interpersonal/contextual factors exert their influence on childhood anxiety are less clearly understood, the potential role of contextual influences in classification is not as certain. However, knowledge of how parental psychopathology, exposure to community violence, and different situations moderate the expression of anxiety problems may serve to clarify and improve classification. For example, the *DSM-IV* (1994, p. 414) suggests that to make the diagnosis of social anxiety disorder in children there must be evidence that the social anxiety occurs in peer settings, not just in interactions with adults. Dadds, Roth, Weems, Guastella, and McAloon (2004) found factor analytic support for this conceptual distinction but results predicting the development of anxiety problems suggest that the implication of predominantly having fear of social situations involving adults may need further empirical consideration. For females, shyness toward adults interacted with parental stress to predict anxiety disorders over and above that afforded by sociability, activity, and parental stress. For males, both shyness toward adults and children were unique in predicting anxiety disorders.

Other Anxiety Disorder Specific Constructs

In addition to the broad mechanisms implicated in anxiety disorders and those thought to differentiate anxiety from depression, several specific constructs are thought to be core features and either differentiate or predict specific anxiety disorders.

Anxiety Sensitivity

A growing body of research evidence has implicated anxiety sensitivity as a risk factor for panic attacks and panic disorder (e.g., Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997, 1999; see also Taylor, 1999). Anxiety sensitivity involves the belief that anxiety sensations (such as heart beat awareness, increased heart rate, trembling, and shortness of breath) have negative social, psychological, or physical consequences (Reiss, 1991).

Research in youth samples using the Childhood Anxiety Sensitivity Index (CASI; Silverman, Fleisig,

Rabian, & Peterson 1991) has demonstrated that anxiety sensitivity in children is related to fears, depression and negative cognitive errors (Weems et al., 1998, 2001; Weems, Hammond-Laurence, Silverman, & Ferguson, 1997). In addition, results from nonclinical samples suggest that anxiety sensitivity discriminates adolescents who report panic attacks from those who do not (Lau, Calamari, & Waraczynski, 1996). Levels of anxiety sensitivity differentiate children with panic disorder from children with other anxiety disorders (Kearney, Albano, Eisen, Allan, & Barlow, 1997). Finally, Hayward et al. (2000) investigated predictors of panic attacks in a large sample ($N = 2,365$) of adolescents (grades 9 through 12, mean age at study entry 15.4 years) who were followed over a 4-year period. Results indicated that anxiety sensitivity predicted the onset of panic attacks during the course of the study period. Improved attention to anxiety sensitivity in the classification of anxiety may help identify important dysfunctional interpretations.

Worry and Fear

The constructs worry and fear are not typically thought of as risk factors, per se, but as core features in the phenomenology of specific anxiety disorders (i.e., GAD and phobias) and also anxiety disorders generally. As such, they help to form the theoretical structure of childhood anxiety disorders.

Worry has been and continues to be a central component of several anxiety disorders as described by the *DSM-III-R* and IV (APA, 1987, 1994). Despite the prominent role of worry in the *DSM* description of GAD, empirical evidence regarding the role that worry plays in these disorders in youth has only recently begun to emerge (e.g., Chorpita, Tracey, Brown, Colluca, & Barlow, 1997; Muris, Meesters, Merckelbach, Sermon, & Zwakhalen, 1998; Perrin & Last, 1997; Weems et al., 2000). Overall, the results from these studies suggest that worry is a prominent and important feature among children with anxiety disorders and may differentiate types of childhood anxiety disorders. However, the types of worries that are salient show developmental differences. Specifically, older youth report more worries about performance, future events, and their appearance than younger children (Weems et al., 2000). Such developmental differences may point toward the utility of employing information on developmentally salient worries in diagnostic assessment.

In terms of fear, many fears are normative in childhood (e.g., see Ollendick, King, & Frary, 1989; Ollendick, Matson, & Helsel, 1985; Ollendick, Yang, King, Dong, & Akande, 1996; Ollendick, Yule, & Ollier, 1991), however, evidence has been garnered suggesting that assessing fears may be useful in clarifying the clinical phenomenology of anxiety disorders. Child as well as the parent-completed Fear Survey Schedule for Children—Revised (FSSC-R; Ollendick, 1983) subscale scores have been shown to be useful in differentiating specific types of phobias, although parents were more accurate for social fears, and children more accurate in death and dying related fears (Weems et al., 1999). However, fears may not be stable over time (Poulton, Trainor, Stanton, & McGee, 1997) and this may be because specific fears are tied to developmental milestones.

Developmental Differences in the Expression of Anxiety and Fear

Drawing upon data regarding normative emotional development as well as psychosocial developmental theory regarding critical developmental stages, both Westenberg et al. (2001) and Warren and Sroufe (2004) have presented models that posit specific age differences in the expression of the symptoms of childhood anxiety and phobic disorders. These models suggest that the predominant expression of anxious and phobic symptoms is tied to normative developmental periods and challenges. For example, based on their models regarding the sequence and timing of symptom expression for school aged youth (aged 6–17 years), one would hypothesize that separation anxiety symptoms and animal fears should be the predominant expression in youth around ages 6–9 years, generalized anxiety symptoms and fears concerning danger and death in youth 10–13 years, and social anxiety symptoms and social/performance related fears in adolescents around age 14–17 years.

The link between normal development and the symptoms of anxiety and phobic disorders can be understood as stemming from the idea that there are basic biological and behavioral predispositions to anxiety disorders. These predispositions give rise to undifferentiated "anxiety." The symptoms of specific anxiety disorders are shaped by various additional biological, cognitive, behavioral and social processes and thus the predominant expression of anxiety may be tuned, in part, to sequential developmental challenges in these domains. For example, children

aged 6–9 years have begun the process of individuation and are realizing their autonomy from parents. Along with this developing sense of self as separate from parents is the contrasting realization of dependence on parents. The developmental challenge is eventual secure self-reliance but this challenge likely gives rise to concerns about separation from or loss of parents. Similarly, youth aged 10–13 years are gaining insight into mortality and broader world concerns that may give rise to death and danger fears and the worries characteristic of generalized anxiety. Finally, the emerging social understanding and comprehension of adolescents may lead to a predominance of social and evaluative concerns in this age group (see Westenberg et al., 2001; Warren & Sroufe, 2004).

Epidemiological data in community samples on the age of onset of anxiety disorders is fairly consistent with the developmental predictions noted above with somewhat differing results across studies (see Costello, Egger, & Angold, 2004). In addition, research in clinical samples suggests that separation anxiety disorder is more common in children whereas social phobia is more common in adolescents. For instance, Weems et al. (1998) reported differences in the distribution of anxiety disorders with separation anxiety disorder more common children (aged 6–11 years) whereas social phobia was more common in older youth (aged 12–17 years) in a sample of 280 youth who met diagnostic criteria for anxiety disorders.

In general, symptoms of anxiety and phobias appear to diminish or decrease over time and at older ages in community samples (e.g., see Ollendick, King, & Frary, 1989; Ollendick, Matson, & Helsel, 1985; Weems et al., 2002). However, Chorpita, Yim, et al. (2000) found that separation anxiety symptoms decreased with school grade level whereas social anxiety symptoms increased with grade in a large school-based sample of youth ($N = 1641$) in grades 3 through 12. Research by Ollendick and colleagues (e.g., Ollendick et al., 1985; 1989) in normative samples also suggests higher rates of animal fears in children as compared to adolescents.

The salient classification implication of these developmental differences is that there may need to be a greater attention to developmental considerations in the diagnosis of childhood anxiety disorders. For example, diagnoses of separation anxiety and social anxiety may be more valid if they consider differences in the normative expression of anxiety and fear. For instance, diagnostic criteria for SAD in young children may require more symptoms or more information on the interference of symptoms to be developmentally valid with regard to differentiating a pathological state from a normative state.

IMPLICATIONS OF THE NOMOLOGICAL NET TO UNDERSTANDING CHILDHOOD ANXIETY DISORDER ASSESSMENT

Embedding the conceptualization of anxiety disorders in a nomological net implies greater inclusion of the mechanisms of the anxiety disorders and the use of multiple measures (across informants and modalities) in defining and testing the validity of the classification scheme. There are also specific implications for measurement, classification, treatment and policy. First, however, the model implies several testable hypotheses that we delineate below. Each of the hypotheses involves improved stability, incremental prediction of functional outcomes, and incremental discrimination of the disorders beyond existing symptom sets or diagnoses. Thus, initial tests of the model are relatively straightforward and build on the existing research on *DSM* anxiety disorders. We argue that utilization of the *DSM* information is important because at least part of a nomological net model is composed of symptoms as delineated in the *DSM*. However, we also suggest that the symptoms and diagnoses in the *DSM* may have to be dismantled and then reconstructed with new information.

1. The current *DSM* symptoms of childhood anxiety will have differential stability and differential associations with functional impairment. Identifying the specific diagnostic symptoms with low stability and low association with impairment will improve the clinical utility of the classification system. Research is needed to delineate the symptoms that are associated with impairment and long-term stability at different ages.
2. The additional assessment of biological mechanisms (such as indices of temperament, trait anxiety, and physiological response) should strengthen and clarify stability estimates by including trait like markers of the anxiety disorders.
3. The additional assessment of learning mechanisms (such as avoidance behavior, learning history) will add to the prediction of functional outcomes by including and detailing specific dysfunctional behavioral repertoires.

4. The additional assessment of cognitive mechanisms (such as cognitive errors, worry, anxiety sensitivity, control beliefs) will add to the prediction of functional outcomes and help in the discrimination of the disorders by including and detailing specific dysfunctional interpretative repertoires.
5. Integrating the assessment of mechanisms with the assessment of symptoms will improve estimates of the effects of method variance.

Measurement

One implication of the nomological net model is greater delineation of measurement bias. The degree to which various measures of anxiety may be biased is largely unknown. For example, it is not clear whether parents, clinicians, or youth most accurately account for the core factors (i.e., prototypical elements) in identifying problems and predicting outcomes (Epkins, 1995a, 1995b). Measurement theory assumes that measures are biased, though the degree or direction of bias in a single measure cannot usually be estimated. The usual solution to this problem is to use multiple measures, presumably biased in different ways and less biased on average than any single measure (Sechrest et al., 2000). We advocate the use of multiple measures not only to improve reliability, but also to begin developing estimates of the degree and direction of bias in measures used to assess childhood anxiety.

A nomological net model implies that a multitrait-multimethod approach to assessment will advance understanding of the degree to which the source of information influences the stability and predictive validity of disorder status. For example, one way that the understanding of childhood anxiety symptoms can be expanded is by examining the convergence or divergence of symptom expression across assessment modalities and informants. Extensive research and reviews have consistently demonstrated that agreement across informants is often low (Achenbach et al., 1987). Yet, the degree to which particular informants are biased and whether they are biased toward under- or overreporting remains unknown. We argue that improvement in this state of affairs requires examination of the correspondence of various informants not only to each other, but also to anxiety's mechanisms and to functional outcomes. It is impossible to know the degree and direction of bias unless there are criterion measures of anxiety disorders. Inclusion of mechanisms offers the possibility of validation of informants and instruments against potential criterion measures such as traits or anxiety disorder taxons.

Estimating the contribution of methods to the assessment of childhood anxiety disorders in combination with accuracy in prediction of functional impairment will provide initial estimates of the degree to which assessment methods bias identification of disordered youth concurrently and over time. It is expected that tests of the nomological net model that employ multiple measures can be used to inform decisions about which types of measures to use, estimates of the degree to which informants contribute to true and method variance, and recommendations for corrections to estimates associated with method, informant, and modality of assessment (e.g., informant report, observation, interview).

Modifications to the Classification Scheme

Another implication of the model and of greater inclusion of mechanisms in the assessment and identification of anxiety disorders is continued evolution of the childhood anxiety disorder criteria. Currently, a common diagnostic picture might be that the child meets criteria for Separation Anxiety Disorder and Generalized Anxiety Disorder. By adding the mechanisms to the assessment of anxiety disorders, the description of the problem is expanded to include other facets of the problem. In the above case, the diagnosis might be Anxiety disorder with features of separation distress and excessive worry, characterized by physiological hyper-arousal and negative cognitive errors. Greater inclusion of hypothesized mechanisms in defining what constitutes an anxiety disorder implies additional tests of the nomological net model. Specifically, greater inclusion of the mechanisms in the diagnostic criteria suggests examining the differential stability and outcome predictive ability of *DSM* symptoms alone, mechanisms alone, and composite variables of symptoms and mechanisms.

As noted, basic tests of the model involve examining the relative stability, convergent and discriminant validity, and predictive ability of the symptoms and the mechanisms to long-term outcome (e.g., the stability of anxiety disorders in the context of the stability of the mechanisms). In addition, the model also implies testing the stability and ability to predict functional outcomes of composite variables of what

we are calling symptoms and mechanisms. Drawing from the earlier example, a composite variable of intense worry, physiological response to worry, and diagnostic symptoms of Generalized Anxiety Disorder may better predict long term outcomes than either the symptoms or the mechanisms alone. Composing and analyzing composite variables of symptoms and mechanisms may be aided by the use of structural equation modeling of latent variables (Figueredo, McKnight, McKnight, & Sidani, 2000; Nunnally & Bernstein, 1994), rather than composites of symptom counts that are made up of different symptoms across individuals.

Treatment and Policy Implications

Another implication of the model and greater inclusion of mechanisms in the assessment and identification of anxiety disorders is that this approach should have greater relevance for clinical treatment planning. A classification system or standardized assessment that includes the mechanisms of the disorder should have greater relevance for treatment planning than the current system by including additional aspects of the problem that can be addressed by the therapist. The current system does not adequately include features of the disorder that might imply different treatments. Including mechanisms in the diagnostic assessment that are associated with the stability of the disorder and functional impairment will provide greater clinical information by delineating processes and facets that can be the targets of treatment and make prognosis more accurate.

There are a few interesting analogies that can be made from the field of medicine. The first are diagnoses of Irritable Bowel Syndrome (IBS), colitis, and Crohn's disease. Each of these problems has very similar descriptive symptoms (e.g., gastrointestinal discomfort, poor digestion, and diarrhea) yet different etiologies. If they were defined solely on the basis of these symptoms it is likely that they would be considered a single disorder with various etiologies and treatment would not be tailored to etiology. The opposite can also be true. Autoimmune dysfunction can result in thyroid dysfunction, rheumatoid arthritis, and skin diseases and thus treating the individual problems is missing the big picture. The field of childhood anxiety needs to ask if we are making these types of mistakes by focusing solely on descriptive criteria for classification.

Including the mechanisms of the disorder in the classification system has at least one very important policy implication. Currently third party funding for assessments do not include many forms of psychological testing because insurance payments are typically provided for diagnostic assessment and treatment alone (see Eisman, Dies, & Finn, 2000). Including mechanisms in the diagnosis of the disorder could serve to broaden the definition of diagnostic assessment. Such broadening of the diagnostic criteria might provide a broadening in the remunerable forms of assessment thereby allowing compensation for additional and clinically important testing.

CONCLUSIONS

Empirical research highlights the need for improving the childhood anxiety disorder diagnostic classification system and the techniques used to identify them. These problems may be addressed by using the nomological net of childhood anxiety. A nomological net understanding of childhood anxiety disorders is not primarily descriptive or etiological. It represents the theoretical structure of anxiety disorders and is comprised of numerous domains. The two major subdivisions of these domains are (1) the symptoms of anxiety disorders (e.g., *DSM-IV* descriptive diagnostic criteria), and (2) the mechanisms of anxiety disorders (e.g., physiological responses, affect, behavior, and cognitive processes). A nomological approach implies greater inclusion of the mechanisms of the anxiety disorders and the use of multiple measures (across informants and modalities) in defining, refining, and testing the validity of the classification scheme. There are also specific implications for measurement, classification, treatment, and policy.

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