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Worry and Generalized Anxiety Disorder: A Review and Theoretical Synthesis of Evidence on Nature, Etiology, Mechanisms, and Treatment

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Abstract

Generalized anxiety disorder (GAD) is associated with substantial personal and societal cost yet is the least successfully treated of the anxiety disorders. In this review, research on clinical features, boundary issues, and naturalistic course, as well as risk factors and maintaining mechanisms (cognitive, biological, neural, interpersonal, and developmental), are presented. A synthesis of these data points to a central role of emotional hyperreactivity, sensitivity to contrasting emotions, and dysfunctional attempts to cope with strong emotional shifts via worry. Consistent with the Contrast Avoidance model, evidence shows that worry evokes and sustains negative affect, thereby precluding sharp increases in negative emotion. We also review current treatment paradigms and suggest how the Contrast Avoidance model may help to target key fears and avoidance tendencies that serve to maintain pathology in GAD.

Keywords

Contrast Avoidance model; emotion dysregulation; interventions; mechanisms; risk factors; developmental factors

Defining and Delineating The Boundaries of Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) has been labeled the basic disorder in psychopathology (Barlow 1988) due to findings that its fundamental characteristics may reflect central processes of all emotional disorders. For instance, GAD at time 1 increases the likelihood of first onsets of mood disorders, panic disorder, posttraumatic stress disorder, and substance

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abuse disorders at time 2 (Ruscio et al. 2007). GAD is also frequently comorbid with other disorders, showing 12-month combined Axis I and Axis II comorbidity rates as high as 89.8% (Grant et al. 2005b). In addition, worry (the central feature of GAD) has been found to be a dimensional construct associated with depression, anxiety, and stress symptoms to an equal degree, leading some researchers to suggest that worry is an important core transdiagnostic process that cuts across current *Diagnostic and Statistical Manual of Mental Disorders* (DSM)-IV diagnostic boundaries (Kertz et al. 2012). Therefore, a better understanding of GAD and worry may help to inform our knowledge of other psychological problems in addition to providing information about GAD as a stand-alone disorder.

Clinical Features

Data regarding age of onset for GAD are conflicting. This may be due to substantial changes in the diagnostic criteria with each revision of the DSM. For example, whereas studies using DSM-III criteria (Am. Psychiatr. Assoc. 1980) suggest an earlier age of onset than other anxiety disorders (average age of 15.6 years; Angst et al. 2009), when DSM-IV criteria are employed (Am. Psychiatr. Assoc. 2000), average age of onset is in the early-to-middle 30s (Grant et al. 2005b).

Contrary to greatly changing age-of-onset estimates, retrospectively assessed prevalence rates have not changed substantially across revisions of the DSM. Lifetime prevalence of GAD is estimated at about 5.7%, and 12-month prevalence is 3.1% (Kessler & Wang 2008). However, in a recent prospective study, lifetime prevalence rates across all psychological disorders were doubled on average (e.g., lifetime GAD prevalence was 14.2% and past-year prevalence was 4.2%), suggesting that retrospective assessments may greatly underestimate diagnostic prevalence (Moffitt et al. 2010).

Women are more likely than men to be diagnosed with GAD (lifetime prevalence rates 5.3% versus 2.8%; Vesga-López et al. 2008), and 12-month prevalence rates are higher in white and Native American individuals but lower in Black, Asian, and Hispanic persons (Grant et al. 2005b).

Evidence of GAD as a Meaningful, Important Stand-Alone Disorder

There have been questions in the literature about whether GAD merits its own diagnostic category. Specifically, major depressive disorder (MDD) is one of the most commonly comorbid disorders with GAD, and these two disorders have a shared genetic origin (see Kendler et al. 2007). Such findings have led to suggestions that GAD and MDD should be combined under the umbrella of distress disorders. This notion is refuted by ample evidence that GAD is a valid diagnosis. For example, the core and associated symptoms of GAD co-occur sufficiently to demonstrate good construct validity (Brown et al. 1998). Further, Brown and colleagues (1998) demonstrated a distinct latent factor related to the symptoms of GAD. Order of onset between GAD and MDD also does not affect the progression of GAD, and GAD and MDD are equally likely to be temporally primary across an individual's life (Kessler et al. 2008), contradicting the theory that GAD is merely a prodrome for MDD.

It is also the case that GAD can be discriminated from MDD with respect to risk factors and adverse familial influences, temperament, life events, and environmental determinants

(Kendler et al. 2007, Moffitt et al. 2007). There are also distinctions between the disorders in attentional and memory biases (Goldberg 2008), biological characteristics (Martin & Nemeroff 2010), comorbidity (Kessler et al. 2008), and illness course (Brown 2007). Moreover, whereas pure GAD and pure MDD are similarly incapacitating, incapacity is considerably amplified when the two occur together, indicating that GAD is as much of an independent disorder as is MDD (Kessler et al. 2002). GAD has also demonstrated specificity with respect to other anxiety disorders, such as a stronger association with pain (Beesdo et al. 2009). Also, pure lifetime GAD (resulting in no additional diagnoses) occurs at the rate of 0.5% (Wittchen et al. 1994), indicating that it is not inconsequential.

Health, Disability, and Cost

There is sometimes a perception of GAD as a disorder that reflects the "worried well," suggesting that neither GAD in general nor its core feature of worry are particularly debilitating. However, this viewpoint is inconsistent with extensive data. For example, having pure GAD was associated with comparable disability to MDD but greater disability than pure alcohol and drug use disorders, nicotine dependence, other anxiety disorders, and personality disorders even after controlling for sociodemographics and any comorbid disorders (Grant et al. 2005a). Moreover, role impairment for GAD is equivalent to what has been reported for individuals with chronic medical conditions (e.g., autoimmune disease, arthritis; Alonso et al. 2011). In data from the World Health Organization, 56.3% of those with GAD were severely disabled (Kessler et al. 2009), and in the Epidemiological Catchment Area Study, 37% of participants with GAD were receiving some type of public assistance (Massion et al. 1993) with only about half of the total sample employed full time. GAD was also associated prospectively in young adults with a substantially increased probability of suicidal ideation and suicidal attempts, after controlling for comorbid disorders and stressful life events (Boden et al. 2007).

In addition to its association with role impairment, GAD has a major impact on physical health problems. For example, GAD represents an independent risk factor for coronary morbidity above and beyond comorbid depression (Martens et al. 2010). GAD prospectively increased the risk of all-cause cardiovascular mortality in Vietnam veterans (Phillips et al. 2009), and it substantially increased the rate of subsequent cardiac events in individuals with stable coronary artery disease (Martens et al. 2010). A three-item measure of worry and anxiety administered to healthy Dutch women predicted a 77% increased risk of premature mortality and cardiovascular death ten years later, after controlling for depression and other risk factors (Denollet et al. 2009). In initially healthy men, across 20 years there was a dose-response relationship between worry and nonfatal myocardial infarction and total coronary heart disease (Kubzansky et al. 1997). Such data have led to theories that it may not be stressful events per se, but rather perseverative thought that serves as the mechanism by which stress confers negative health outcomes (Brosschot et al. 2007).

In terms of cost to society, GAD has been credited as being among the uppermost disorders with respect to increased medical utilization as well as adverse impact on workplace performance (Hoffman et al. 2008). Individuals with GAD are also among the most common patrons of primary care, specialty clinic, and emergency room services, adding markedly to

the nonpsychiatric expense affiliated with anxiety disorders (Fogarty et al. 2008). Thus, when GAD goes untreated it is extremely expensive in terms of distress, incapacity, quality of life, and medical difficulties (Newman 2000).

Naturalistic Course

The naturalistic course of GAD is both chronic and fluctuating. For example, across a two-year period, only 39% of individuals who had been diagnosed with GAD at baseline in the Primary Care Anxiety Project achieved a full recovery. Additionally, 22% of those who had recovered experienced a partial relapse, and 30% had fully relapsed (Rodriguez et al. 2006). In a study comparing dimensional ratings of severity of GAD to categorical diagnoses across a 14-year period, whereas most participants appeared to have remitted when only the presence versus absence of diagnosis had been assessed, there was actually only a modest decrease in GAD severity (Ramsawh et al. 2009). Further, most participants continued to exhibit subthreshold symptoms along with significant impairment. With respect to whether GAD at time 1 predicts a greater likelihood of GAD (homotypic continuity) versus other disorders (heterotypic continuity) at time 2, there is both temporal consistency and temporal variability. For instance, across two years, GAD significantly predicted both homotypic and heterotypic prospective continuity at roughly equal rates in young adolescents (Ferdinand et al. 2007). Therefore, even when GAD may appear to be in remission, it may be the case that it remains at a subthreshold level or has simply been replaced with another disorder.

Recent studies have begun to examine GAD symptoms across shorter time periods using daily and within-daily assessments. This approach provides a better understanding of the ways in which GAD symptoms unfold naturalistically. Such studies have found that both worry and attempts at cognitive avoidance predicted subsequent increases in daily anxiety (Dickson et al. 2012) and that the physiological impact of worry during the day extends into sleep (Brosschot et al. 2007). Also, using analyses that accounted for idiographic variability across a two-week period, average GAD severity during that period was positively associated with rigid high-amplitude vacillations in anxiety levels within and across days (Fisher & Newman 2012, Newman & Fisher 2012). These data are consistent with the theory that individuals with GAD are highly reactive to internal and external triggers and tend to cope with such sensitivity by shifting intrapersonal experiences toward negativity. As described in the next section, this theory (called the Contrast Avoidance model), also accounts for a comprehensive range of mechanisms involved in the etiology or maintenance of GAD.

Mechanisms of The Psychopathology of Generalized Anxiety Disorder

Over the past two decades, an abundance of empirical studies have investigated a wide range of cognitive, affective, and neurobiological mechanisms associated with GAD. As suggested below, many of these mechanisms point to the central role of hyperreactivity and a fear of negative emotional shifts as well as the use of worry to prevent emotional contrasts that are perceived as unmanageable.

Cognitive, Affective, and Neurobiological Mechanisms

Individuals with GAD demonstrate cognitive and affective hyperreactivity— Relative to individuals with depression (Aldao et al. 2010), participants with GAD report greater perceived intensity of emotional experiences. Those with GAD also self-report more difficulty recovering from or managing a negative mood state, including experimentally induced emotion (Mennin et al. 2005), and were objectively rated as demonstrating more anxiety and sadness than controls in response to emotional disclosures by a confederate (Erickson & Newman 2007). Also, momentary assessment showed that compared to 9- to 13-year-olds without GAD, those with GAD were more emotionally reactive to negative events (Tan et al. 2012). Unsurprisingly, individuals with GAD describe feeling more threatened by and less in control of their emotions relative to nonanxious subjects (Llera & Newman 2010) and persons with social anxiety disorder (Turk et al. 2005).

Genetic and brain-imaging studies also characterize individuals with GAD as prone to negative emotional reactivity and partially explain this vulnerability. Specifically, GAD correlates strongly with trait negative affectivity or “neuroticism” (Brown et al. 1998), a proneness to emotional reactivity that is substantially heritable and accounts for much of the genetic variability of GAD (Hettema et al. 2006). Consistent with this vulnerability, functional magnetic resonance imaging studies of GAD cumulatively suggest hyperresponsivity of the amygdala, the limbic structure that mediates negatively valenced affective reactions, and fear in particular (Schienle et al. 2011). Relative to healthy controls, adults with GAD have demonstrated amygdalar activation to anticipation of aversive images (Nitschke et al. 2009) and to various neutral and conflict conditions (Etkin et al. 2010, Nitschke et al. 2009). Such hyperresponsivity corresponds to larger amygdala volumes observed in GAD patients compared to controls (Etkin et al. 2009). Thus, individuals with GAD appear to be vulnerable to emotional and neural hyperreactivity, which they report finding challenging to regulate. As a result of identifying such emotional difficulties, efforts have been made to try to understand the mechanisms responsible.

Worry as an experiential avoidance mechanism—One mechanism that has been implicated as a means to manage emotional reactivity is worry. In fact, a current notion in the GAD literature is that worry may be a cognitive mechanism employed in an attempt to master problems with emotional dysregulation and to feel more in control. To this end, Borkovec and colleagues' Cognitive Avoidance theory of worry (Borkovec et al. 2004) has played an important role in helping to inform our understanding of the relationship between worry and emotional dysfunction. According to this model, worry functions as a cognitive avoidance response to perceived internal and external threats and may reduce some aspects of emotional reactivity to otherwise unavoidable negative events. The Cognitive Avoidance model has been used as a foundation for subsequent theories, which have extended it to suggest that worry either helps people to avoid (or suppress) negative emotionality or is an unsuccessful attempt to do so. Nonetheless, there is substantial evidence that rather than avoid emotion, worry evokes and sustains negative emotionality. Such conflicting data led Newman & Llera (2011) to propose the Contrast Avoidance model, which argues that worry neither attempts to nor successfully facilitates avoidance of negative emotions, but instead serves a different function in the pathology of GAD. Below we review evidence supporting

the Contrast Avoidance model of worry, including studies showing that worry generates and prolongs negative emotionality.

The Contrast Avoidance model—The Contrast Avoidance model suggests that individuals with GAD engage in chronic worry because they prefer to experience a sustained state of distress as a way to be emotionally prepared for the worst possible outcome to various events (Newman & Llera 2011). As incongruous as it may seem that anyone would choose to maintain a state of chronic negativity, it may be understandable for individuals with GAD given their emotional sensitivities and vulnerabilities. Specifically, individuals with GAD report feeling more distraught than nonanxious controls when experiencing a sharp shift from a euthymic or relaxed state to one that is overwhelmingly negative (Llera & Newman 2011). This is referred to as a negative emotional contrast experience, and it is likely that individuals with GAD feel compelled to be emotionally braced for negative events at all times to avoid feeling even greater experienced disruption in response to emotional contrasts. The Contrast Avoidance model was partly informed by the Affective Contrast theory, which stems from cognitive psychology (Bacon et al. 1914). In support of this theory, the impact of an emotional experience is moderated by the state that precedes it, such that an unpleasant state is experienced as more unpleasant if preceded by a positive state and less unpleasant if preceded by a negative state (Dermer et al. 1979).

Given evidence that individuals with GAD are particularly reactive to emotional stimuli, it is likely that the experience of being caught off-guard by a negative event, which would provoke a sharp emotional contrast, is particularly threatening. Indeed, compared to moderate worriers, these individuals anticipate that negative events will be more costly for them and underestimate their ability to cope with such threats (Ladouceur et al. 1998). As such, generating a sustained negative emotional state may be preferable specifically because it reduces the possibility of experiencing a sharp increase in negative emotion if something bad were to happen. In the following sections we discuss the current research that may point to this model.

Worry leads to greater concurrent negative emotionality and physiological activation: The preponderance of studies that focus directly on the emotional impact of worry find that it evokes negative emotionality. For example, participants with and without GAD who experience a worry induction report greater negative emotionality than those in relaxation or neutral inductions, as well as compared to within-subject preworry baseline periods (Llera & Newman 2010, 2011). Also, trait worry is associated with increased sympathetic nervous system (SNS) activity, such as heightened cardiovascular activity, and decreased parasympathetic nervous system (PNS) activity, such as lower heart rate variability (Brosschot et al. 2007, Pieper et al. 2010). Similarly, experimentally induced worry results in increased SNS activity and decreased PNS activity relative to baseline and relaxation periods (e.g., Llera & Newman 2010, 2011), even above and beyond a cognitive challenge task (Hammel et al. 2011). Likewise, in a small sample of GAD patients, listening to personally relevant worry statements induced activation of the prefrontal and thalamostriatal brain regions, which are associated with response to threat (Hoehn-Saric et al. 2004); citalopram treatment for eight weeks decreased these activation patterns and self-

reported worry, implicating worry specifically (not just GAD status) in activation of key brain regions. Together, results strongly point to worry as a thought process that activates negative emotions, somatic systems, and neural networks.

Worry does not suppress subsequent negative emotionality or physiological activation: In a previous study using speech-anxious participants, worrying immediately prior to fearful imagery muted cardiovascular responding to the exposure, as compared to prior relaxation or neutral inductions, which facilitated responding (Borkovec & Hu 1990). Recently, these data were replicated with samples diagnosed with GAD (Llera & Newman 2010, 2011; Stapinski et al. 2010). However, these effects were present only when comparing negative emotionality during the stressor to the immediately preceding worry period, as opposed to a baseline period. When an initial baseline was used for comparison instead, there was no evidence that experimentally induced worry led to reduced reactivity in response to a subsequent stressor (Llera & Newman 2011, Stapinski et al. 2010). Further, Llera & Newman (2010, 2011) found no difference between preceding worry, relaxation, and neutral tasks with respect to absolute levels of negative affectivity during a subsequent stressor. Given that worry already leads to negative emotionality, these studies show that its only effect on emotional and physiological reactivity is that of preventing a further increase in negative emotionality between heightened levels during worry and subsequent emotional exposure periods.

Worry prolongs negative emotionality and physiological activation: As opposed to muting current or subsequent negative emotions, worry actually creates and sustains such emotionality. For example, when measuring absolute levels of emotionality across tasks, worry led to heightened negative emotionality relative to baseline (as compared to relaxation and neutral tasks), which was then sustained across negative emotional exposures (Llera & Newman 2011). Beyond in-the-moment measures of elevated negative emotionality, worry can prolong or sustain negative states even after the worry period has ended. For instance, during ambulatory monitoring, worry led to subsequent elevated physiological activity when participants were awake as well as asleep (Brosschot et al. 2007, Pieper et al. 2010) and was more significantly predictive of subsequent cardiovascular activity than were stressful events (Pieper et al. 2010). Furthermore, state worry in the evening predicted next-morning cortisol secretion independently of mood, anxiety, sleep, and stressors (Zoccola et al. 2011), and trait worry also predicted cortisol awakening responses (Schlotz et al. 2004), suggesting that worry prospectively contributes to hypothalamic-pituitary-adrenal axis activation. Together, these data support Brosschot and colleagues' proposition that worry prolongs the cognitive representation of a stressor along with its attendant emotional repercussions (e.g., Brosschot et al. 2007).

This viewpoint is consistent with findings of a heightened alertness to potential external threats in individuals with GAD (Waters et al. 2008). Further, those with GAD show strong reactivity to ambiguous or neutral stimuli as if they were negatively valenced stimuli (Olatunji et al. 2011), suggesting that their threat-detection system can become overresponsive to any potential danger. Consistent with this, whereas the central nucleus of the amygdala mediates rapid responses to acute stressors, the bed nucleus of the stria

terminalis (BNST) is associated with slower onset but longer-term sustained vigilance and anxiety; GAD patients demonstrated heightened BNST activation relative to controls in response to a task that could precipitate monetary loss (Yassa et al. 2012). Lastly, individuals with GAD exhibited higher mean SNS arousal (e.g., heart rate, skin conductance) in daily life and at rest relative to controls (e.g., Pruneti et al. 2010).

It is also possible that rather than contributing to muting of negative emotionality, the abstract verbal linguistic nature of worry (Stöber & Borkovec 2002) may be responsible for helping to prolong negative emotionality. For example, verbal-linguistic worry inductions facilitated more subsequent negative thought intrusions than imaginal worry inductions (Butler et al. 1995). In a treatment-seeking GAD sample, worry inductions led to higher skin conductance levels during subsequent reexposure to an anxiety stimulus after a delay period, whereas imaginal processing and relaxation conditions did not (Stapinski et al. 2010). Moreover, insomniac patients who were instructed to worry about an upcoming speech task reported longer sleep-onset latency and more anticipatory anxiety relative to those who engaged in imaginal processing of speech implications (Nelson & Harvey 2002). Together, this body of evidence supports a view of verbal-linguistic worry in which anxiety and negative emotionality are not only generated in the moment but also sustained across future experiences. In sum, data strongly point to the fact that worry creates and sustains negative emotion and does not lead to emotional avoidance or suppression.

Worry as a problematic compensatory strategy: Despite the negative impact of worry, those with GAD view it positively, particularly with respect to its ability to help distract them from more emotional topics (Borkovec & Roemer 1995). In light of self-reported and physiological evidence that worry does not lead to emotional avoidance, the Contrast Avoidance model proposes that individuals with GAD use worry to mute emotional contrast experiences as opposed to muting negative emotion per se. Thus, those with GAD prefer to anticipate all possible negative outcomes to future events and to take on a defensive negative intrapersonal stance because of their fear of emotional contrasts (i.e., sharp increases in negative emotion). As a means to avoid such experiences they worry to cultivate a sustained negative emotional state. In this way, worry serves to prevent negative emotional contrasts and thus may engender a sense of control over otherwise unmanageable internal experiences (Newman & Llera 2011). In line with this idea, individuals with GAD reported that worry prior to viewing emotional film clips was significantly more helpful than prior neutral or relaxation inductions in coping with the emotional impact of the clips (Llera & Newman 2011). However, the opposite was true for those without GAD. Healthy controls reported that relaxation or neutral inductions prior to the film clips helped them feel better able to cope with the impact of the clips compared to a worry induction. It does appear that individuals with GAD are somehow unable to regulate negative emotional contrasts, and that they use worry to generate a sustained negative state as a preemptive, compensatory strategy.

Recent neuroscientific research on monitoring and adapting to emotional conflict dovetails with these ideas. Conflict between emotionally valenced stimuli may be particularly relevant for individuals with GAD given their sensitivity to affective contrasts. To this end, Etkin and colleagues (2010) developed a paradigm to extend known functions of the anterior cingulate cortex (ACC) (monitoring for conflicts or mismatches between multiple sources of

information). Participants judge images of faces as happy or fearful while encouraged to ignore affectively congruent or incongruent words (“happy” or “fear”) superimposed on the images. “Emotional conflict” produced by incongruent stimuli (e.g., “happy” overlaid on a fearful face) is evidenced by slower reaction times in naming facial affect. However, if participants are able to more rapidly judge in-congruent stimuli after other incongruent stimuli than after congruent stimuli (Etkin et al. 2009), this suggests that “emotional conflict adaptation” has occurred, or specifically that emotional conflict (due to previous incongruency or contrast) triggers secondary regulatory processes that help individuals adapt to such conflict.

In this paradigm, both GAD and nonanxious groups demonstrated emotional conflict as well as faster reaction times to congruent stimuli when preceded by congruent (versus incongruent) stimuli (Etkin et al. 2010). However, only in nonanxious individuals was there a pattern of adaptation to emotional conflict. Further, only in these individuals was such adaptation associated with pregenual cingulate activation and inverse connectivity between pregenual cingulate and amygdalar activation. Because emotional conflict adaptation operates at an implicit level of which participants denied awareness, these findings cumulatively suggest that individuals with GAD are relatively unable to adapt to emotionally conflicting social stimuli (i.e., they do not get used to being surprised by contrasting emotional information), and their brains fail to automatically recruit medial prefrontal regulation of the amygdala. Lacking normal prefrontal cortical inhibition of amygdala reactivity, individuals with GAD are thought to utilize the explicit, compensatory prefrontal cognitive strategy of worry to disengage the amygdala (Etkin et al. 2009). Although both GAD patients and controls experience dorsal medial prefrontal cortex and ACC activation when exposed to worry sentences, this activation does not return to resting levels following a worry induction for GAD patients (Paulesu et al. 2010), providing further evidence of chronic worry as a problematic compensatory strategy. Thus, available research attests to ample, though not unambiguously unique, biological mechanisms of GAD.

In summary, emotional and neurobiological evidence supports the notion that individuals with GAD are more hyperreactive to threat and less able to cope with resulting negative emotional contrasts than those without GAD. Such evidence also points to worry as a compensatory mechanism that creates and prolongs negative emotionality, thereby reducing contrasting emotional experiences. In the next section, we review developmental, temperamental, and interpersonal risk factors associated with GAD—risk factors that may lead individuals to develop emotional sensitivities and corresponding compensatory mechanisms associated with fear of emotional contrasts.

Risk Factors for the Development of GAD

Environmental—The development of GAD is associated with unexpected negative life events (Nordahl et al. 2010), maltreatment (Moffitt et al. 2007), and loss (Kendler et al. 2003). Such adverse events can lead people to question their worldviews—that life is predictable and good things happen to good people. Even the presence of a one-time negative life event may lead individuals to become continuously anxious as a means to prepare for other potentially unpredictable events.

In addition to one-time events, as outlined below, those with GAD have increased likelihood of negative experiences that are more chronic in nature, including ongoing abuse, parental rejection, inconsistent parenting, and parental invalidation (Cougle et al. 2010, Moffitt et al. 2007). These chronic negative life events may also lead to the development of an aversion to emotional contrast due to inconsistency and lack of predictability about the timing of the next parental rejection, abusive episode, or negative parent-child interaction. As such, this may contribute to continuous negative affect being more comfortable than the shift from a euthymic state to a negative one.

Attachment and parenting style—Attachment has been considered a relational emotion regulation system (Bowlby 1973), and insecure attachment may be a potential risk factor in the development of GAD. Accordingly, insecure attachment and dysregulation of emotions, such as anxiety, go hand in hand as potentially important developmental risk factors.

Bowlby (1973) suggested that child anxiety stemmed from concern over parental availability. Ainsworth and colleagues (1978) identified two anxious, insecure attachment styles—anxious-avoidant and ambivalent/resistant styles. Anxious-avoidant infants were rejected by their mothers and avoided them upon reunion. It was theorized that children who experienced this rejection avoided relationships because of the anxiety experienced within them. Those with ambivalent/resistant attachment styles had inconsistent or intrusive parents, leading children to respond with anger, resistance, anxiety, and ambivalent behavior upon a reunion. Children with ambivalent/resistant attachment would likely experience fluctuations in affect in response to inconsistent parental responses. In line with the Contrast Avoidance model, children's uncertainty regarding whether their needs would be met, and the distress over resulting affective fluctuations, may lead those with ambivalent/resistant attachment to experience intrapersonal negativity continuously as a means to prepare for unexpected behaviors from their parents.

Only a few studies have examined the relationship between specific subtypes of attachment and GAD symptoms. For example, 12- to 14-year-olds who were ambivalently attached had higher GAD scores than those with secure attachment (Muris et al. 2000a) or those with secure or avoidant attachment (Muris et al. 2001). Avoidant and ambivalent attachment were also associated with worry severity in children (Brown & Whiteside 2008) and preadolescents (Muris et al. 2000b). Similarly, in the National Comorbidity Study, presence of GAD was positively associated with avoidant or anxious attachment and negatively associated with secure attachment (Mickelson et al. 1997). Similar results were found in one longitudinal study showing that twice as many children with either GAD or social phobia in late adolescence had been classified as ambivalently attached in infancy (12 months of age) compared to the number of infants classified as securely or avoidantly attached (Warren et al. 1997). Finally, a prospective study examined the degree to which new episodes of GAD could be predicted by preceding attachment patterns in adults. Angry-dismissive attachment (a subdivision of avoidant attachment, in which individuals are mistrustful of others, exhibit conflict and anger, and have little desire for close companionship) was more strongly associated with new GAD episodes than depression or social phobia (Bifulco et al. 2006). Although most of these data are cross-sectional, they suggest the possibility that both

ambivalent attachment and inconsistent parenting may play a role in the development of GAD.

Negative parenting behaviors have also been associated with GAD symptoms and worry. In community samples, samples of anxious children, and clinical samples of adults, perceived parental rejection was associated with offspring worry (Brown & Whiteside 2008, Muris et al. 2000b) and GAD symptoms (Cassidy et al. 2009, Hale et al. 2006). Similarly, retrospective reports of maternal and paternal coldness (Cassidy et al. 2009) were associated with adult GAD. In comparison with controls, individuals with GAD were also associated with more maternal role-reversal and enmeshment (Cassidy et al. 2009). Role reversal puts a child in the position of being the adult, providing comfort to the parent and being responsible for the parent's welfare. Given that children are ill equipped emotionally to take on such a role, this may lead them to experience negative affect within the parent-child relationship. Parental rejection, coldness, and lack of care may also cause interactions with parents to be emotionally difficult. However, given the association between ambivalent attachment, inconsistent parenting, and GAD, it is unlikely that every interaction with a parent will be characterized by maladaptive parenting, establishing a situation where children may never know when they are going to experience negative parenting and the associated shift from a positive or euthymic affect to negative affect. In the absence of mature emotion regulation strategies, insecurely attached children may develop an aversion to the associated affective contrasts and may feel more “comfortable” in a continuous state of anxiety.

In community samples of adolescents, perceived parental psychological control was also associated with GAD symptoms (Wijsbroek et al. 2011). When parents are psychologically controlling, they invalidate their child's emotions and experience and attempt to change them through such means as love withdrawal, isolating the child, and shaming the child. Such parental behaviors may undermine children's emotional development, thereby contributing to emotion regulation difficulties, negative contrast avoidance, and child anxiety symptoms. Child anxiety may also contribute to psychological control—leading parents to engage in psychological control as a means to try to regulate emotions for the child. Supporting this, adolescent GAD symptoms predict adolescents' report of parents' later psychological control (Wijsbroek et al. 2011). However, because this result was based on adolescent report, it is unclear whether this indicates that adolescent GAD contributes to greater parental psychological control or whether anxious adolescents simply perceive their parents as being increasingly controlling due to threat bias.

GAD symptoms in children and adolescents have also been associated with harsh parental discipline and parenting characterized by strict rules and high expectations (Shanahan et al. 2008) as well as parental overprotection (Beesdo et al. 2010, Nordahl et al. 2010). The combination of over-protection of the child and harsh discipline may impede children's development of autonomy and convey that they are incapable of handling challenging situations without parental intervention. If children internalize the message that they cannot cope with unanticipated negative events and the associated emotional contrast, their only perceived recourse may be to anticipate all possible negative outcomes to ensure that they will be emotionally prepared for whatever might occur.

Temperament—Temperament—innate, early differences in biological, behavioral, and emotional responsiveness to environmental stimuli—is related to GAD in young children (Rothbart 2007). Interestingly, such temperamental dispositions as behavioral inhibition may lead to difficulties in tolerating affective shifts. Children with behaviorally inhibited temperament, characterized by a low threshold to novelty, are thought to have increased SNS activity or decreased PNS activity, resulting in restraint, distress, and avoidance when exposed to novel stimuli (Kagan & Snidman 1999). As such, these children may experience large affective shifts between euthymic states and negative states, leading these contrasts to be quite uncomfortable and distressing. It is possible that individuals with temperamental styles characterized by high physiological arousal in response to novel stimuli may be especially likely to experience negative contrast avoidance. In fact, in a longitudinal study, the tendency to become aroused easily in mid-childhood was a pathway by which childhòod risk factors predicted the development of GAD symptoms (Karevold et al. 2009).

Interpersonal Factors in GAD

Related to temperament, personality disorders and maladaptive interpersonal processes have been associated with adult GAD (Newman & Erickson 2010). Such dysfunctional interpersonal processes may play a key role in the onset or maintenance of GAD via biased interpersonal cognitions and problematic behaviors (Newman & Erickson 2010).

With respect to problematic social cognition, interpersonal concerns are the most common worry topic (Roemer et al. 1997). Individuals with GAD are also exquisitely sensitive to interpersonal threats, as evidenced by self-reports of hypervigilance and sensitivity (Nisita et al. 1990). Objective measures have also confirmed biased attention toward threatening faces (Mogg et al. 2000). Further, GAD analogues exhibited bias toward perceiving others as attacking, ignoring, and controlling in a first-meeting encounter (Erickson & Pincus 2005). Worry also predicts perceiving coldness in others' interpersonal behavior during hypothetical scenarios, even when accounting for commonly comorbid symptoms of social anxiety and depression (Erickson et al. 2010). This hypervigilance and sensitivity may serve to maintain a negative affective state in order to prevent individuals with GAD from experiencing a startling negative affective shift associated with a negative interpersonal experience.

Such interpersonal sensitivity may also lead to problematic interpersonal behavior. GAD and worry are robustly linked to intrusive, exploitable, nonassertive, and cold interpersonal problems (Przeworski et al. 2011). One study found that even after controlling for depressive and social anxiety symptoms, individuals highest in worry in a mixed anxious-depressed clinical sample had the strongest negative interpersonal impacts (e.g., viewed as more hostile) as rated by significant others (Erickson et al. 2010). In addition, individuals with GAD appear to suffer interpersonally, as reflected in their greater likelihood of being separated or divorced (Whisman 2007), having disrupted family relations (Beesdo et al. 2010), and lacking close friendships (Whisman et al. 2000) compared to individuals with other disorders. Furthermore, GAD predicts low marital satisfaction even after controlling for a host of comorbid Axis I disorders (Whisman et al. 2000), further buttressing the idea that interpersonal disruption is central to GAD.

Beyond merely demonstrating cross-sectional links between negative interpersonal processes and GAD symptoms, some studies imply that these processes may play a role in the maintenance of GAD symptoms over time. Specifically, interpersonal problems (Crits-Christoph et al. 2005), partner hostility (Zinbarg et al. 2007), marital tension (Durham et al. 1997), and personality disorders (Ansell et al. 2011) predict lower likelihood of remission in GAD. Furthermore, changes in interpersonal problems correlated with changes in GAD symptoms (Crits-Christoph et al. 2005). Untreated interpersonal problems after cognitive behavioral therapy (CBT) also predicted worse GAD symptoms at follow-up assessment (Borkovec et al. 2002). Collectively, such studies suggest the possibility that interpersonal difficulties play a role in the maintenance of GAD over time, but they leave the mechanisms of their action in question.

Complicating our attempts to understand such mechanisms, psychotherapy studies have not fared well at ameliorating interpersonal problems or at reducing GAD symptoms by targeting interpersonal issues. For example, supportive-expressive therapy, which focused on gaining insight into core conflictual relationship themes, caused improvement for some interpersonal problems but not others (Crits-Christoph et al. 2005). Similarly, despite a deliberate focus on issues such as identifying interpersonal needs and behaviors employed to meet them, examining interpersonal impacts on others (including the therapy relationship), and learning more effective social behaviors, a recent trial that added an interpersonal treatment module to CBT did not lead to superior interpersonal outcomes in GAD symptoms relative to CBT plus supportive listening (Newman et al. 2011). Such null findings suggest that the mechanisms whereby interpersonal problems maintain GAD are poorly understood and not adequately targeted in extant therapy models, warranting new models. We suggest that the Contrast Avoidance model is a viable candidate to address this need. Specifically, it may be that individuals with GAD are using their interpersonal behaviors as a means to control their affect and to avoid affective shifts. If this is the case, then targeting tolerance of affective shifts may help individuals with GAD to be more flexible in their interpersonal behaviors.

The idea of interpersonal strategies to avoid negative emotional contrasts is novel in the context of GAD. However, it is reminiscent of the social-personality literature on the “consistency motive” or need to preserve a cognitive sense of predictability, which drives individuals high in this need to interpersonally reject positive feedback that is discrepant from their negative self-concept (Swann & Read 1981). This parallels the finding that persons with GAD prefer to maintain a predictably negative outlook by worrying as opposed to experiencing strong emotional shifts in response to threats when relaxed (Llera & Newman 2011), which is consistent with the Contrast Avoidance model.

For instance, individuals may hypothetically employ cold behaviors as a way to avoid negative emotional contrasts. Given that individuals with GAD “prefer to feel chronically distressed in order to prepare for the worst outcome” (Newman & Llera 2011, p. 375), some may adopt cold behaviors with the parallel logic that it is better to keep people at a distance than to allow oneself the vulnerability of warm behaviors and subsequently be confronted by conflict or rejection. This possibility is consistent with elevated levels of trait anger and hostility (Deschenes et al. 2012) and interpersonal conflicts (Judd et al. 1998) in GAD, as

well as a subset of GAD clients with problems related to interpersonal coldness (Przeworski et al. 2011) or, less frequently, (cold) schizoid and narcissistic personality disorders and antisocial traits (Vesga-López et al. 2008).

Analogously, submissive social behaviors may function strategically to prevent others from making negative (cold) responses, given that submission often serves the function of appeasing others and avoiding their hostility (Sloman & Gilbert 2000). Indeed, submissive (nonassertive, socially avoidant) problems characterize a sizeable portion of those with GAD (Przeworski et al. 2011). Similarly, avoidant personality disorder, typified by submissiveness, is the personality disorder most commonly comorbid in GAD (Dyck et al. 2001).

Alternatively, affiliative or warm behaviors may be utilized with the same avoidant motivation. Many individuals with GAD endorse interpersonal problems high in warmth—such as being intrusive, overly self-sacrificing, or exploitable (Przeworski et al. 2011). Individuals with GAD also endorse heightened empathy (Peasley et al. 1994). Given that negative affectivity (shared variance of worry, depressive symptoms, and social anxiety) predicts quarrelsome and unaffiliative social behavior, it is interesting to note that once the commonly comorbid symptoms of depression and social anxiety are controlled, worry predicts affiliative tendencies. Such tendencies include warm interpersonal traits, exploitable (warm-submissive) interpersonal problems, and high agreeableness and low quarrelsome social behavior, in a week of experience sampling (Erickson et al. 2010). In sum, it is possible that a desire to avoid a negative emotional contrast explains the relationship between worry and a diverse range of interpersonal problems.

Treatment for Generalized Anxiety Disorder

Accumulation of data on psychotherapy for GAD has lagged behind most other anxiety disorders and depression. Nonetheless, meta-analyses suggest that as the only empirically supported therapy for GAD, CBT reduces acute symptoms with maintenance sustained up to two years following treatment (Borkovec & Ruscio 2001). CBT packages are generally superior to nonspecific therapies and wait-list control conditions (Borkovec & Ruscio 2001). The positive impact of CBT also includes a reduction of most comorbid anxiety disorders, particularly when CBT has successfully reduced GAD symptoms (Newman et al. 2010).

In addition to psychotherapy approaches, psychopharmacological treatments have been recommended. Several different classes of drugs are currently used to address GAD symptoms, including antidepressants, benzodiazepines, and anticonvulsants, and most have demonstrated efficacy in placebo-controlled trials. Within classes of medications for GAD, the consensus across different guidelines is that selective serotonin reuptake inhibitor (SSRI) and serotonin-norepinephrine re-uptake inhibitor (SNRI) antidepressants should be considered the first line of treatment (Katzman et al. 2011). Nonetheless, despite some notable benefits, there are also drawbacks and limitations to medications (for a review of various medications and their impacts, see Newman et al. 2012), and there is no clear support for an additive effect when combined with CBT (Bond et al. 2002, Crits-Christoph et al. 2011).

Notwithstanding empirical support for GAD treatments, there is substantial room to improve its efficacy. Percentages of those who have attained clinically significant change in response to psychotherapy are lower than those typical for other anxiety disorders (Newman & Borkovec 2002). Thus, investigators have begun to examine predictors, moderators, and mediators of psychotherapy for GAD in order to target specific areas for improvement.

Predictors of Therapy Outcome

Studies examining predictors of treatment outcome have found that higher baseline self-reported anxiety (Butler & Anastasiades 1988) predicted higher anxiety after treatment with anxiety management training or behavior therapy as well as worse 10- to 14-year follow-up from cognitive therapy, analytic therapy, or anxiety management training (Chambers et al. 2004). On the other hand, higher assessor-rated severity predicted greater change in response to CBT for adults (Newman & Fisher 2010) and for a geriatric population with GAD (Wetherell et al. 2005), with no differences in posttreatment outcome. Also, higher assessor-rated severity predicted lower anxiety at posttreatment in response to anxiety management training (Butler & Anastasiades 1988). Presence of Axis I comorbidity has also been associated with worse outcome from cognitive therapy, analytic therapy, or anxiety management training (Chambers et al. 2004, Durham et al. 1997) as well as greater change in response to CBT or its components (Newman et al. 2010, Wetherell et al. 2005). Therefore, there is some conflicting evidence as to whether diagnostic comorbidity and symptom severity predict problematic outcome from psychotherapy.

More consistent predictors of negative outcome have included longer duration of illness (Biswas & Chattopadhyay 2001), having received previous psychiatric treatment (Durham et al. 1997), being unmarried (Durham et al. 1997), partner hostility (Zinbarg et al. 2007), higher marital tension (Durham et al. 1997), comorbidity with depression (Durham et al. 1997, Newman et al. 2010), and the tendency to interpret ambiguous information as threatening (Butler 1993). Moreover, as noted previously, interpersonal problems have predicted negative outcome from brief dynamic therapy (Crits-Christoph et al. 2005), and interpersonal problems remaining at posttreatment predicted lower posttherapy and follow-up improvement from CBT (Borkovec et al. 2002).

A number of process variables have also been examined as predictors of outcome for GAD from both CBT and dynamic therapies. Such studies have found negative outcome to be associated with resistance during session 1 (Westra 2011), lower first-session expectancy/credibility of therapy (Newman & Fisher 2010), lower homework compliance (Wetherell et al. 2005), therapists' negative emotional reactions to clients (Westra et al. 2012), and lower therapeutic alliance (Langhoff et al. 2008). Moreover, greater flexibility in anxiety symptoms across the therapy period as well as change from greater rigidity to greater flexibility in anxiety symptoms during therapy predicted better outcome from CBT (Fisher et al. 2011, Newman & Fisher 2012).

Moderators of Outcome

Researchers have also attempted to determine whether pretreatment variables discriminated between different forms of treatment in their efficacy. This endeavor might contribute to the

development of more personalized interventions. For example, a motivational interviewing adjunct may have been more effective for those with greater severity of GAD than was CBT without motivational interviewing (Westra et al. 2009). In another study, personality disorders predicted better outcome from brief Adlerian psychodynamic psychotherapy than from medications (SSRIs or SNRIs) (Ferrero et al. 2007). Finally, longer duration of GAD illness predicted better outcome from purely cognitive or purely behavioral treatments than from combined CBT, whereas shorter duration of GAD predicted better outcome from combined CBT than from its components (Newman & Fisher 2012). The latter result was explained as being due to those with longer-duration GAD having more entrenched symptoms and therefore needing a more focused and intense dose of a smaller number of treatment techniques compared to those with shorter-duration GAD, who benefitted most from having more technique options. Thus, clients with more challenging presentations (e.g., greater severity, more personality problems and chronicity) may fare better in therapies with an intensity and focus tailored to the individual.

Mediators of Outcome

Mediators have been examined as a means to try to understand mechanisms of change in response to psychotherapy for GAD. For example, the effect of adjunctive motivational interviewing on CBT outcome was mediated by change in client resistance (Aviram & Westra 2011). In addition, change in expectancy during the early part of therapy mediated the effect of baseline GAD severity on reliable change at posttreatment (Newman & Fisher 2010). Also, change in worry accounted for subsequent change in somatic anxiety to a greater degree in response to CBT compared to relaxation (Donegan & Dugas 2012). Moreover, the extent to which longer duration of GAD predicted better outcome from purely cognitive or purely behavioral treatments compared to combined CBT was mediated by the extent to which there was an establishment of greater flexibility in anxiety symptoms across the therapy period (Newman & Fisher 2012). Taken together, these results point to change in resistance, change in expectancy, change in worry, and change in rigidity of anxiety symptoms as potential mechanisms of change from CBT for GAD.

Areas of Improvement for Extant GAD Treatment

As mentioned previously, many clients with GAD do not demonstrate clinically significant, lasting improvement following traditional CBT (Borkovec & Ruscio 2001). Such data have led to various attempts to improve upon standard CBT by targeting specific mechanisms thought to be related to the maintenance of GAD. Such treatments have targeted emotional deepening or regulation (Mennin 2006; Newman et al. 2008, 2011), interpersonal problems (Newman et al. 2008, 2011), mindfulness (Roemer et al. 2008), intolerance of uncertainty (Dugas et al. 2005), attentional bias (Amir & Taylor 2012), and motivational interviewing (Westra 2011). However, there is currently no evidence that any of the newer treatments are superior to standard CBT. Previously reviewed research on moderators of outcome suggests that personalizing interventions may be one potential solution.

Another possibility is to examine a treatment that specifically targets contrast avoidance as a way to address maladaptive patterns of worry and their relationship to emotion and interpersonal behavior. Although worry reduction is a key focus in traditional CBT, the

Contrast Avoidance model would suggest that extant treatments aimed at challenging cognitive worry patterns are attempting to dismantle the cognitive defense without treating the underlying fears. Targeting contrast avoidance in treatment could be achieved via exposure to relaxation contiguous with emotional stimuli. Being in a relaxed state followed by a negative emotional contrast is a proxy for naturalistic contrasts, and therefore an appropriate exposure, which could lead to habituation over time. Such treatment could also include exposure to contrasting pleasant and unpleasant emotionally evocative images. Thus, the fear of negative contrast experiences could be addressed directly, with the effect of reduced dependence on worry as a defensive process. Also, as noted above, interpersonal problems seem to maintain GAD symptoms, so conducting functional analysis of clients' interpersonal behaviors (and ways they may serve to prevent emotional contrasts) may generate other opportunities for exposure or problem solving.

Conclusion

With its high level of prevalence and long course, GAD has been associated with substantial costs not only for the individual but also for families and society. Related to this disorder are elevated risks of comorbidity, medical problems, marital discord, and occupational difficulties. Considerable research has been conducted on diverse processes and risk factors potentially involved in the etiology and maintenance of GAD and, as argued in this review, the role of several of these maladaptive mechanisms can be explained, at least in part, by a new conceptualization of this disorder. Integrating empirical findings from different research domains, the Contrast Avoidance model proposes that (*a*) GAD is characterized by emotional hyperreactivity and discomfort with contrasting emotions (manifested through or associated with affective, cognitive, and neurobiological processes); (*b*) such difficulty with sharp negative emotional shifts is caused or maintained by biological, developmental, temperamental, and interpersonal vulnerabilities; (*c*) rather than avoid negative emotionality, worry evokes and sustains negative affect; (*d*) those with GAD use worry to shift their intrapersonal experiences toward negativity in an attempt to preclude sharp increases in negative emotion.

Avenues for Future Research

Following this synthesized review of the empirical literature on GAD, several questions remain. First is the question of whether the Contrast Avoidance model of worry is unique to GAD or whether core elements of this model could cross diagnostic boundaries. For example, a recent theory by Nolen-Hoeksema and colleagues (2008) posits a similar mechanism for depression in that rumination functions to engender a sense of helplessness regarding negative events, which is perceived by those with depression as less aversive than attempting to control an external situation and failing. In both disorders, a negative intrapersonal state is recruited as protection against an experience that is considered even more aversive, suggesting a possibility that similar underlying factors could mediate this process. For this reason, more research on the Contrast Avoidance model is warranted to identify elements and risk factors that are unique to GAD or those that might apply transdiagnostically. Further, risk factors associated with GAD (or other disorders) could be explored to the extent that they lead to contrast avoidant tendencies. This could include

behaviorally inhibited temperament in children and interpersonal affiliative or aggressive behaviors.

Future research should also be conducted to improve treatments for GAD. Whereas a substantial number of clients benefit from CBT, studies have also demonstrated that others fail to improve or fully respond. Moreover, recent efforts to improve upon CBT have not led to an increase of its efficacy. We propose that alternative treatments for GAD based on this new model of psychopathology should be developed and tested.

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Abbeviations

DSM
Diagnostic and Statistical Manual of Mental Disorders

Homotypic continuity
the prediction of the future likelihood of a disorder by the presence of the same disorder

Heterotypic continuity
the prediction of the future likelihood of a disorder by the presence of a different disorder

Contrast Avoidance model
the theory that individuals with GAD are hypersensitive to sharp shifts from positive/euthymic to negative emotional states and use worry to shift their intrapersonal experiences toward negativity in an attempt to preclude sharp increases in negative emotion associated with potential future threat

Amygdala
the limbic structure that mediates negatively valenced affective reactions, and fear in particular

Cognitive Avoidance theory
the theory that worry functions as a cognitive avoidance response to perceived threats and dampens some emotional reactivity to negative events

Affective Contrast theory
a theory in cognitive psychology stating that the affective valence of an experience is moderated by the valence of the preceding state

SNS
sympathetic nervous system

PNS
parasympathetic nervous system

Thalamostriatal brain region
a network of neurons originating in the thalamus and terminating in the striatum

Citalopram
a selective serotonin reuptake inhibitor classified as an antidepressant

BNST
bed nucleus of the stria terminalis

ACC
anterior cingulate cortex

CBT
cognitive behavioral therapy

SSRI
selective serotonin reuptake inhibitor

SNRI
serotoninnorepinephrine reuptake inhibitor

Summary Points

1. GAD is an important, stand-alone disorder that leads to significant impairment across multiple domains. These include role impairment, occupational problems, interpersonal and emotion regulation difficulties, and physical health problems, leading to substantial personal and societal cost.
2. A synthesis of data on the mechanisms of GAD indicates that it is a disorder associated with emotional and neurobiological hyperreactivity and a fear of negative emotional contrasts.
3. Evidence points to worry as a compensatory mechanism that creates and prolongs negative emotionality, thereby reducing contrasting emotional experiences.
4. We argue that this emotional and neurobiological dysregulation is caused or maintained by developmental vulnerabilities (environmental stress, insecure attachment, negative parenting behaviors), temperamental vulnerabilities (behavioral inhibition), and interpersonal processes (sensitivity to social threats and problematic interpersonal behaviors).
5. Although CBT is an empirically supported treatment for GAD, many clients fail to fully benefit from this treatment, and various attempts to modify standard CBT have not led to significant improvement. These realities suggest the need to reconsider maintenance factors for GAD, which have not been treated explicitly, such as avoidance of negative emotional contrasts.
6. Although worry reduction is a primary therapy outcome goal, current treatments that challenge worry patterns may attempt to dismantle the cognitive defense without treating the underlying fears. Therefore, treatments could be usefully explored that personalize intervention techniques and/or target behaviors and interpersonal patterns that maintain contrast avoidance.

Future Issues

1. Is the Contrast Avoidance model of worry unique to GAD or do core elements of this model cross diagnostic boundaries?
2. To what extent do risk factors associated with GAD (or other disorders) lead to contrast avoidant tendencies?
3. What is the relationship between contrast avoidant tendencies and the diverse interpersonal problems associated with GAD? For instance, do affiliative behaviors predict worry only when one believes that affiliation facilitates avoidance of negative emotional contrasts?
4. Could GAD therapy outcome be improved by investigating alternative treatments based on new models of psychopathology (such as the Contrast Avoidance model) and personalized interventions?