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Differences between trait fear and trait anxiety: Implications for psychopathology

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ABSTRACT

Fear and anxiety are poorly delineated in much of the clinical and research literatures. Although some theorists and researchers have posited explanations for how trait fear and trait anxiety differ, many others conceptualize the constructs as largely or entirely interchangeable. The primary goals of this review are to examine clinical conceptualizations and neurobiological studies of fear and anxiety, examine the animal and human literatures on the correlates of fear and anxiety, provide clearer definitions of these two constructs, and discuss their implications for psychopathology. A secondary goal is to evaluate content of self-report measures of trait fear and anxiety, and meta-analyze the relations between self-reported trait fear and anxiety. We found that existing measures share significant content overlap across constructs. Despite this overlap, our meta-analysis revealed only a moderate ($r = 0.32$) relationship between measures of trait fear and anxiety, with an even lower relationship ($r = 0.14$) when we examined trait fear measures operationalized in terms of harm avoidance. These findings suggest that fear and anxiety are largely distinct emotions, and that psychological disorders of trait fear and trait anxiety warrant classification in separate higher-order categories. Moreover, they suggest that future research should focus on deriving more content valid measures of trait fear and trait anxiety from the neurobiological and diagnostic literatures.

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Fear and anxiety are primitive emotions that function to preserve life. In healthy individuals, fear and anxiety can facilitate action in an effort to maintain safety and well-being (Lang, Davis and Öhman, 2000). Nevertheless, when these often adaptive states become characteristic of an individual, or trait-like, the resulting phenomena can turn physically (e.g., cardiovascular disease) and psychologically (e.g., agoraphobia) destructive. Currently, psychopathology marked by high levels of trait-like fear and anxiety reside largely under the umbrella category of *anxiety disorders* in the diagnostic nomenclature (American Psychiatric Association, 2000). Kessler, Chiu, Demler, and Walters (2005) found that the 12-month prevalence estimate for these common disorders was 18.1%. Moreover, these disorders account for approximately 32% of all mental health expenditures in the United States (DuPont et al., 1996).

1. Fear and anxiety: Conceptual and definitional confusion

Despite the severity and pervasiveness of anxiety disorders, the literature is only beginning to clarify the often poorly delineated relationship between trait fear and anxiety. Moreover, the definitional boundaries of trait fear and anxiety remain controversial. Although some theorists and researchers (e.g., Barlow, 2002) have posited explanations for how trait fear and anxiety differ, many others conceptualize the constructs as largely or entirely interchangeable (e.g., Beck & Emery, 2005). For example, Beck and Emery (2005, p. 9) wrote that, "Fear involves the intellectual appraisal of a threatening stimulus; anxiety involves the emotional response to that appraisal." Izard and Ackerman (2000, p. 260) wrote, "[Fear] is the key emotion in the anxiety pattern." Some prominent authors have even argued explicitly that fear and anxiety are essentially isomorphic; for example, Wolpe (1987) noted that he chose to use "'fear' and 'anxiety' synonymously in this paper because they are psychophysiologically indistinguishable" (p. 135).

Instead, and consistent with a growing body of research across multiple domains, we contend that there is now persuasive evidence that fear and anxiety are different emotions. We argue that their conflation in the literature has led to ambiguous and at times misleading findings. For example, there is a robust literature on the underpinnings of pathological fear in children (e.g., Hayden et al., 2007). Nevertheless, many of these studies used tasks designed to measure worry, hypervigilance, and rejection sensitivity as proxy measures of fear. As we will discuss later, these tasks appear primarily to assess anxiety, not fear.

The confusion is most prominent when investigators use harm avoidance (HA) measures, which can refer to either trait anxiety (e.g., Cloninger, Przybeck, & Svrakic, 1991) or trait fear (e.g., Tellegen, 1982). In research on psychopathy, a number of researchers have attempted to test etiological models positing a lack of fear as the core cause of this condition (e.g., Lykken, 1995) using either measures of trait anxiety or measures that are highly contaminated with trait anxiety. Consequently, some studies that appear to bear on the low fear hypothesis of psychopathy may be largely or entirely irrelevant to this hypothesis (Poythress et al., 2008).

Taken together, the conceptual confusion as to fear and anxiety may negatively impact the implications of existing research. The conflation of fear and anxiety, however, is not restricted to the clinical literature. For example, Crestani et al. (1999), in a study investigating the neural pathophysiology of anxiety in rodents, wrote that, "Anxiety states in humans are characterized by harm avoidance behavior and a bias for threat cues" (p. 833). As discussed later, the preponderance of evidence suggests that avoidance behaviors (that is, fight, flight, or freeze responses) are characteristic of fear, not anxiety. As another

example, when describing fMRI studies, Davidson (2002) wrote that "One common strategy for evoking anxiety among anxious patients in the laboratory is to present them with specific types of stimuli that are known to provoke their anxiety (e.g., pictures of spiders for spider phobics)" (p. 69). Yet spider phobics are presumably pathologically fearful of spiders and motivated to avoid them; therefore, pictures of spiders may instead provoke flight or freeze responses characteristic of fear, not anxiety. From these examples, it is evident that the relations between trait fear and trait anxiety and their definitional boundaries require clarification. Well-defined trait emotions, however, require clear operationalizations at the state level. Although the phenomena of state fear and anxiety ostensibly share many functional and structural similarities, they may encompass distinct emotional states.

In the animal literature, there is a rich discourse examining the biological underpinnings of state fear and anxiety. This literature is clinically relevant given that many of the same structures (e.g., amygdala) that mediate fear and anxiety in animals also mediate these emotions in humans (Blair, 2003). Moreover, as methodologists and philosophers of science have long noted, conclusions are strengthened to the extent to which they derive from maximally diverse sources of information (Shadish, Cook & Campbell, 2001). Several researchers have also recently examined the biological substrates of state fear and anxiety in humans. The findings from these literatures, to be discussed later, suggest that although there is some overlap, there are sharp differences in the patterns of brain activation. In healthy adults, complementary literatures have also noted differential external correlates of self-reported fear versus anxiety, such as pain perception (Rhudy & Meagher, 2000), whereby self-reported fear is inversely and self-reported anxiety positively related to pain perception. This finding is consistent with conceptualizations of fear as an emotion that motivates avoidance behaviors, wherein the organism experiences less physical pain during the fight, flight, or freeze response. This finding is also consistent with the conceptualization of anxiety as an emotion that motivates hypervigilance, wherein the organism exhibits heightened sensitivity to external stimuli during approach. Similarly, several studies using cognitive laboratory tasks have found evidence differentiating fear from anxiety (see Öhman, 2008 for a review).

Despite an abundance of biological and cognitive research separating fear from anxiety, definitions of the subjective human experiences of trait fear and anxiety remain contentious (Barlow, 2002). As Gray and McNaughton (2000) noted, however, the only direct means of understanding and defining the subjective experience of fear or anxiety is by self-report. Although preliminary efforts have been made to investigate the discriminant validity of self-reported trait fear and anxiety measures (e.g., Perkins, Kemp, & Corr, 2007), there are no comprehensive reviews of the relations between self-report measures of fear and anxiety, as well as potential moderators of this still murky relationship.

The primary goal of this article is to provide a broad overview of research on the clinical and biological correlates of fear and anxiety from both non-human animal and human literatures. In the case of the latter, we examine research on both "normal-range" personality and psychopathological disorders, especially anxiety disorders. We also examine implications of differences between fear and anxiety for the classification of psychopathological disorders and neuroscience research. A subsidiary goal of this paper is to evaluate the content of self-report measures of trait fear and anxiety and meta-analyze their relationships. As discussed later, the meta-analysis is not intended to provide a definitive evaluation of the relationship between self-reported trait fear and anxiety, as we later contend that many of the extant measures are fundamentally flawed. However, we conducted the meta-analysis not only to investigate the associations among these measures but to clarify potential sources of ambiguity inherent in these measures. To set the stage for our narrative review and meta-analysis, we first summarize the theoretical and biological literatures on fear and anxiety, highlighting the most pertinent writings and studies.

2. Conceptual distinctions between fear and anxiety

State emotions refer to affective adaptations to specific situations, whereas trait emotions refer to affective characteristics of a person across time and situation (Morrissette, Tull, Gulliver, Kamholz, & Zimering, 2007). Clinical conceptualizations of fear and anxiety are concerned primarily with trait emotions, whereas neuroscientific conceptualizations are concerned primarily with state. Although most neuroscience studies focus on emotion states, their findings are readily extrapolated to form trait operationalizations.

2.1. Clinical conceptualizations

In the clinical literature, there are several, in some instances competing, conceptualizations of fear and anxiety. Beck and Emery (2005), for example, posited that fear is a cognitive response to threat, whereas anxiety is an emotional response to fear. In other words, anxiety is the emotional byproduct of fearful cognitions. According to Beck and Emery, trait fear results from pervasive and persistent interpretations of stimuli as threatening. Trait anxiety, in contrast, encompasses the emotional responses to pervasive fearful cognitions. Accordingly, a trait fearful person would presumably be trait anxious.

Epstein (1972) suggested that fear is an emotional response that results from the interpretation of specific environmental cues as threatening and manifests itself in avoidance and escape behaviors. He posited that anxiety, in contrast, is a product of one of three different pathways. In the first, avoidance of a feared stimulus is disrupted, a phenomenon termed *unresolved fear*. Several theorists agree that the inability to avoid fearful stimuli leads to anxiety (e.g., Öhman, 2008). The second, according to Epstein, occurs when individuals overestimate the potential for threat in ambiguous situations. Because the threat is non-specific and future oriented, there are no clear avoidance options, leading to apprehension and indecision. Many contemporary clinical theorists (e.g., Barlow, 2002) define anxiety similarly. Some learning theorists (Zinbarg & Mineka, 2007) support this pathway by suggesting that pathological anxiety results from implicitly associating a benign feature of a dangerous experience with the danger itself. The third type occurs when an individual's environment and expectancies do not match. In terms of behavior, Epstein proposed that anxiety elicits hypervigilance in the face of uncertainty.

By Epstein's definitions, trait fear results from an individual being hypersensitive to several distinct environmental threat cues and avoiding situations involving those threats. Trait anxiety, in contrast, results from an inability to avoid a prolonged feared situation, overestimating the potential for threat across situations, or experiencing chronic mismatches between the environment and expectations. According to this model, trait fear and anxiety are largely unrelated, as the specificity of perceived danger cues and their proposed pathways differ.

In a review of the cognitive and psychophysiological literature on fear and anxiety, Öhman (2008) posited that fear occurs when individuals are actively coping with a perceived threat, whereas anxiety results from a threatening situation without an effective means of coping. Similar to Epstein's conceptualization of anxiety as *unresolved fear*, Öhman suggested that fear and anxiety share similar underlying processes and are differentiated based on perceived avoidance options. He also implied that trait fearful individuals who are concerned primarily with physical threat react more strongly than healthy individuals to physically threatening stimuli. Nevertheless, these individuals do not exhibit elevated trait anxiety as they actively avoid perceived threats. Conversely, individuals whose trait fear is concerned primarily with social threat also show elevated levels of

trait anxiety. Therefore, the relationship between trait anxiety and fear, according to Öhman's conceptualization, differs according to whether the fear is primarily physical or social (see also Lykken, Tellegen, & Katzenmeyer, 1973).

Tellegen (1982) conceptualized trait anxiety as aversive arousal in uncertain situations where avoidance does not seem possible. In contrast, he conceptualized trait fear as hypersensitivity to danger cues leading to avoidance behavior. Using these definitions, Tellegen found that trait fear (as assessed by a "Harm avoidance" scale) and trait anxiety (as assessed by a "Stress Reaction" scale) are separable and nearly orthogonal constructs in the development of the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982). Tellegen (1985) also found that, when using these definitions, trait fear loads on a higher-order Constraint factor, whereas trait anxiety loads on a higher-order Negative Emotionality factor (see also Watson, Clark, & Harkness, 1994). Individuals who score highly on the Constraint factor "convey caution, planfulness, a tendency to avoid danger, conventionality, and adherence to traditional values" (Tellegen, 1982, p. 36). Individuals who scored high on the Negative Emotionality factor, in contrast, "describe themselves as often stressed and harassed, prone to respond with strong negative emotions to everyday vicissitudes, and as enmeshed in adversarial relationships" (Tellegen, 1982, p. 35). The finding that measures of trait fear and anxiety load on two different higher-order factors of the "Big Three" personality dimensions suggests that fear and anxiety may relate to, and perhaps stem from, different etiological processes (Tellegen & Waller, 2007).

2.2. Neuroscientific conceptualizations

Compared with the clinical literature, the constructs of fear and anxiety are delineated more clearly in the neuroscience literature. Fear is commonly defined as an aversive reaction elicited by the perception of a specific threat stimulus, whether conditioned or not (e.g., Cooper & Guynn, 2006). Mammals display three behavioral responses to fear: fight, flight, and freezing in place (Gray & McNaughton, 2000). In most studies of fear reactivity, the threat to the animal or individual is physical (e.g., Berg & Davis, 1985). However, White and Depue (1999) suggested that specificity and degree of threat, not the type of threat, elicit fear. In other words, social situations may also induce fear in humans, as social harm occurs more often and can be equally as devastating as physical harm. Moreover, as Öhman (1986) noted, social threat can quickly escalate to physical harm.

Anxiety, in contrast, is commonly defined in the neuroscience literature as prolonged hypervigilance in anticipation of, or response to, a diffuse threat where danger is not clearly imminent (e.g., Macleod & Rutherford, 1992). As Blanchard and Blanchard (1990) noted, however, the perceived imminence of danger in both fear and anxiety is in part a cognitive construction. Therefore, individuals' perception of the specificity and imminence of danger may shape their emotional experience. In other words, perception of danger is subjective and can be influenced substantially by individual differences in personality.

Another theoretical distinction is the duration of arousal, whereby the fear response tends to be short lived and the anxiety response long-lived (e.g., Davis, 1998). Trait fear, by these definitions, results from an organism chronically engaging in fight, flight, or freezing behaviors due to perceiving specific environmental cues as threatening. Trait anxiety, in contrast, results from an organism being in a chronic state of hypervigilance due to the anticipation of a generalized threat. By these definitions, trait fear and anxiety are not necessarily related (see also Barlow, 2002).

Additionally, McNaughton and Corr (2004), based largely on the works of Jeffrey Gray (1982) and Blanchard and Blanchard (1990), posited a two-dimensional behavioral defense system, whereby fear and anxiety are distinct defensive responses. McNaughton and Corr posited that the certainty of threat is necessary but not sufficient for differentiating fear from anxiety. To McNaughton and Corr, the primary difference between fear and anxiety lies in the directional motivation of the behavior. They described fear as a defensive reaction during which fight/flight/freeze responses facilitate escape from threat. In contrast, they described anxiety as hypervigilance while approaching a potential threat, in the form of physical danger, failure, or loss of reward. Their theory holds that the anxiety system continually compares the current environment with goals and expectations. When conflicts arise between them (approach/approach, approach/avoidance, or avoidance/avoidance), the anxiety system interrupts behavior and places the individual in a heightened state of hypervigilance. In contrast with other neuroscientific conceptualizations, their operationalization of anxiety necessitates conflict arising from competition among available goals.

Taken together, the clinical and neuroscience literatures suggest several experiential characteristics of fear and anxiety (see Table 1). Although fear and anxiety appear to share some defining characteristics (e.g., negative valence), they also exhibit several discriminating characteristics (e.g., defensive direction).

3. Neuroscience research on fear and anxiety

Despite the relative consistency of neuroscientific conceptualizations, the substrates of fear and anxiety remain the subject of many research endeavors. Fear and anxiety are primitive states, which provide adaptive survival responses to threat (Porges, 1995). The brain structures implicated in fear and anxiety are similar across species, which allows investigators to extrapolate provisionally biological findings from non-human mammals to humans. Although there are species-specific fearful and anxious behaviors, translational research is consistently supported by complementary human studies. The majority of animal research in this area has been conducted using rodents (e.g., Nader & LeDoux, 1999), although some studies used non-human primates (e.g., Winslow, Noble, & Davis, 2007).

3.1. Animal studies

3.1.1. State fear

As Gray and McNaughton (2000) pointed out, the only feasible way to infer causal pathways in the neural circuitry related to fear and anxiety is through non-human animal experimentation. The human brain displays comparable connectivity to other mammals with analogous functioning in many areas implicated in fear and anxiety (Aggleton, 1992). Over several decades, neuroscientists have methodically pinpointed the structural and functional substrates necessary to induce fear- and anxiety-associated behaviors in animals, primarily through brain stimulation, lesion, and pharmacological blockade studies in rats.

Within these studies, the two most widely investigated behavioral responses include the freezing response and fear-potentiated startle reflex (Brown, Kalish, & Farber, 1951) acquired through classical (Pavlovian) conditioning (Fendt & Fanselow, 1999). A limitation of the freezing response is that it is limited by a zero baseline (Lang et al., 2000), meaning that it only occurs during a fear state. Therefore, it is unclear in pharmacological and lesion studies whether the effect of a treatment is due to blocking fear or blocking the animal's physical ability to freeze. A challenge of conditioning studies more broadly is clarifying whether the effects of interventions are due to fear, memory, or both (Walker & Davis, 1997). In other words, it is often unclear whether attenuated startle or freezing responses result from reduced fear *per se* or impaired memory of the fear-provoking stimulus resulting from the treatment.

Table 1
Experiential characteristics of fear and anxiety.

Dimension	Fear	Anxiety	References
Emotional valence	Negative	Negative	For example, Epstein (1972)
Temporal focus	Present-focused	Future-focused	For example, Tellegen (1982)
Duration of arousal	Phasic (brief)	Tonic (sustained)	For example, Davis (1998)
Defensive direction	Avoidance (escape)	Approach	For example, Epstein (1972); Gray and McNaughton (2000); McNaughton and Corr (2004); Mineka (1979); and Öhman (1993)
Specificity of threat	Specific	Diffuse	For example, Davis (1998); Lang, Davis, and Öhman (2000); and Macleod and Rutherford (1992)
Pain perception	Blunted	Enhanced	For example, Rhudy and Meagher (2000); and Tang and Gibson (2005)

3.1.1.1. Neurobiology. Despite these limitations, researchers have continued to isolate specific brain areas involved in fear conditioning. Blanchard and Blanchard (1972) implicated the amygdala in fearful responding by observing the failure of rats with amygdalar lesions to freeze when presented with a shock-associated CS. Later studies implicated the lateral nucleus (LA; e.g., LeDoux, Cicchetti, Xagoraris, & Romanski, 1990) and central nucleus (CeA; e.g., LeDoux, Iwata, Cicchetti, & Reis, 1988) of the amygdala. Whereas the LA is implicated only in the acquisition of fear, the CeA is implicated in the acquisition and expression of fear conditioned responses (Wilensky, Schafe, Kristensen, & LeDoux, 2006). In a review of the fear-potentiated startle reflex literature, Davis (2006) highlighted several studies implicating the CeA as the primary circuitry in the acquisition and expression of fear-potentiated startle in rats. Studies have also implicated the CeA in non-human primates (e.g., Kalin, Shelton, & Davidson, 2004). Overall, Davis (2006) concluded that the conditioned fear-potentiated startle reflex has a very short latency, is consistent, and deteriorates rapidly after the removal of the feared stimulus.

Fear-conditioning research also implicates prefrontal regions in the extinction of fear. Morgan, Romanski, and LeDoux (1993) demonstrated that lesions to the medial prefrontal cortex (mPFC) exerted no effect on fear conditioning but impaired extinction training in a freezing paradigm. Conversely, Gewirtz, Falls and Davis (1997) found that lesions to the mPFC had no effect on fear conditioning or extinction in a startle paradigm. Although these findings suggest that prefrontal circuits might have a differential involvement in freezing versus startle, Quirk, Russo, Barron, and LeBron (2000) found that ventro-medial prefrontal cortex (vmPFC) lesions exclusively impaired extinction recall. That is, vmPFC lesions did not affect initial extinction learning, but resulted in diminished recall of extinction on subsequent days. In a later study, Sierra-Mercado, Corcoran, Lebrón-Milad, and Quirk (2006) temporarily inactivated the vmPFC by infusing tetrodotoxin (a potent neurotoxin) prior to fear acquisition and prior to extinction. They found that inactivation prior to extinction led to diminished recall of extinction the following day, whereas inactivation prior to fear acquisition had no effect on recall. These findings suggest that the vmPFC may play a role in the maintenance of trait fear by impairing extinction memory for feared stimuli.

3.1.2. State anxiety

In the animal literature, state anxiety is often studied in rats using the open field test and the light/dark box. These tasks take advantage of rats' preference for enclosed, familiar spaces and dark environments. When given a choice between a darkened area and a brightly lit area, rodents will consistently choose the dark area (Crawley, 1981). However, anxiolytics reduce the preference for darkness (Onaivi & Martin, 1989). In one study investigating the startle response in a well-lit environment, rodents displayed increased startle responses (Walker & Davis, 1997). Consistent with several theories of anxiety, the light ostensibly represents a diffuse threat and uncertain environment characteristic of anxiety-inducing situations. A benefit of this paradigm over the fear-potentiated startle is that it does not rely on conditioning (Walker & Davis, 1997). Contrary to the extinction displayed in fear-potentiated startle paradigms, repeated exposure to the light without a US does not extinguish elevated responding. Moreover, the rapid dissolution characteristic of the fear-potentiated startle response is not observed in light-enhanced startle.

3.1.2.1. Neurobiology. Although lesions to the CeA diminished startle responses in fear-conditioning paradigms, they did not change the startle response in the light-enhanced paradigm (Walker & Davis, 1997). Conversely, lesions to the bed nucleus of the stria terminalis (BNST) fail to diminish startle responses in fear-conditioning paradigms (Hitchcock & Davis, 1991), but diminish startle in the light-enhanced paradigm (Walker & Davis, 1997). These findings suggest two separable pathways mediating the startle response in fear versus anxiety. However, the CeA and BNST share similar downstream connections (Davis, 2006).

In addition to the amygdala, Gray (1982) and Gray and McNaughton (2000) posited that the septum and the hippocampus are central to anxiety. Through several anxiolytic and lesion studies (see Gray, 1976), he demonstrated that anxiolytics and hippocampal lesions produce similar behavioral effects. Moreover, he found that among classical anxiolytics, the common electrophysiological "signature" involved the hippocampal theta rhythm. This rhythm refers to patterns of neuronal firing that produce a slow wave of approximately 5–10 Hz (McNaughton & Gray, 2000). In a review, McNaughton, Kocsis and Hajos (2007) found that manipulation of the theta rhythm is characteristic of all clinically effective anxiolytic agents. They also found that the same was not true for some other drug classes, such as antipsychotics. In support of this finding, studies have found that selective serotonin reuptake inhibitor (SSRI) injections into the hippocampus reduce anxious behaviors in rats (Degroot & Nomikos, 2005). Since Gray and McNaughton (2000)'s work, several studies have supported the role of the hippocampus in anxiety (e.g., Edinger & Frye, 2006).

3.1.3. Trait fear and trait anxiety

In addition to the voluminous literature investigating state fear and anxiety in non-human animals, there is a large literature investigating trait anxiety. Conversely, there is little evidence for trait fear (Miller, Garner, & Mench, 2006). However, Aguilar et al. (2003) found trait anxiety (measured using exploration tasks) and fear (measured using avoidance tasks) factors independent of a conditioned fear factor using several behavioral tasks in rats.

Trait anxiety in mice and rats is typically studied using animals bred for either high or low levels of anxious behaviors. In non-human primates, these animals are usually selected based on their performance on unconditioned tests. In addition to different levels of trait anxiety, studies have found that different strains of rats and mice are differentially affected by anxiolytic drugs (e.g., Vendruscolo, Takahashi, Brüske, & Ramos, 2003). One limitation to this literature is that the behaviors associated with these tests are often referred to as "anxious/fearful," reflecting a failure to distinguish these two constructs (e.g., Williamson et al., 2003). With that caveat in mind, Kalisch et al. (2004) found that selectively bred trait anxious rats displayed reduced dorsomedial prefrontal cortex (dmPFC) oxygenation compared with nonanxious rats. De Wit, Yutaka, Balleine, and Dickinson's (2006) findings also support the role of the dmPFC in trait anxiety. They found that rats with intact dmPFC were able to come to a resolution in a goal-conflict paradigm, whereas rats with chemically inactivated dmPFC were not.

In one study not using selectively bred rats, Blundell and Adamec (2007) observed trait anxiety-like behaviors following the unprotected exposure of a rat to a cat for 5 min. In this paradigm, the rat is stalked and attacked (bitten and clawed). Following this encounter, rats display enhanced startle (Blundell, Adamec, & Burton, 2005) and anxious behaviors (Cohen et al., 2004) for several weeks, even in the absence of apparent danger cues. Blundell and Adamec (2007) found that the behavioral changes result from changes to NMDA receptors in several amygdala nuclei, the BNST, and the periaqueductal gray.

3.1.4. Implications of non-human animal neuroscience studies

Studies investigating the biological substrates of fear and anxiety in non-human animals provide several important implications for our understanding of the trait and state emotions:

(1) Autonomic arousal occurs during fear and anxiety states.
(2) Two distinct neural pathways appear to mediate fearful and anxious responding: The CeA is the primary circuitry in the expression of fear, whereas the BNST is the primary circuitry in the expression of anxiety.
(3) Conditioned fear responses extinguish in the absence of CS/US pairings, whereas unconditioned anxiety responses may not.
(4) The fearful response dissipates quickly, whereas anxiety promotes a sustained response.
(5) Fear occurs in situations of certain threat, whereas anxiety occurs in response to uncertain situations.
(6) Preliminary evidence suggests that the vmPFC is involved in the maintenance of trait fear, whereas the dmPFC is involved in the maintenance of trait anxiety.
(7) Overall, these findings support the biological differentiation of fear from anxiety in non-human animals.

3.2. Human studies

Although there are several advantages to studying the biological correlates of fear and anxiety in animals, human research also holds several advantages, including the ability to measure subjective levels of fear and anxiety, the ability to manipulate experimental paradigms with verbal instructions, and the ability to measure the effects of implicit as well as explicit stimuli. However, one disadvantage is that humans readily habituate to experimental threat cues (Klorman, 1974). Moreover, it is ethically impermissible to induce state fear comparable to that presumably induced in non-human animal studies.

3.2.1. State fear

Fear studies in humans typically involve fear-conditioning paradigms consistent with those in the animal literature (e.g., shocks or air blasts). Additionally, fearful responding is measured following presentation of unconditioned fear-inducing stimuli (such as fearful faces). A variety of techniques is used in these paradigms, including functional magnetic resonance imaging (fMRI), positron emission tomography (PET), fear-potentiated startle, and skin conductance responding (SCR).

Fear conditioning in humans consistently increases SCR (e.g., Grillon & Ameli, 2001) and fear-potentiated startle (e.g., Hamm & Vaitl, 1996). Moreover, research demonstrates that, unlike SCR, fear-potentiated startle response is an indicator of arousal and valence, specifically negative valence (Ruiz-Padial & Vila, 2007). Ruiz-Padial and Vila (2007) further found that increased fear-potentiated startle does not require the subjects' awareness of unconditioned fearful stimuli, suggesting that the acquisition of fear may occur as part of implicit (unconscious) processing. Jovanovic et al. (2006) elaborated on this point by finding that startle responses increased to a CS whether or not subjects were aware of it. However, the inhibition of the startle response in the presence of safety cues occurred only in the aware group. In addition to external cues, the human fear-potentiated startle increases when visualizing fearful stimuli (Vrana & Lang, 1990). Although these findings indicate similarities in human and animal fear conditioning, they suggest two implications for the human experience of fear that differ from those found in the animal literature. First, humans are capable of processing environmental fear cues outside of their awareness. Second, internal fear cues can generate human fear reactions.

Studies investigating central nervous system responding to fear-conditioning paradigms parallel the animal literature. Several studies have found increased blood flow in the amygdala using PET during the acquisition of fear conditioning (e.g., Furmark, Fischer, Wik, Larsson, & Fredrikson, 1997) and increased amygdalar activity using fMRI (e.g., Pine et al., 2001). Supporting Ruiz-Padial and Vila's (2007) findings, studies have found increased amygdalar activity in response to unconditioned fearful stimuli, whether or not the subject was aware of the stimuli (e.g., Whalen et al., 1998).

Consistent with the animal literature, several studies have implicated the vmPFC in the recall of fear extinction (see Sotres-Bayon, Cain, & LeDoux, 2006, for a review), whereby activation in the vmPFC is associated positively with extinction recall. Milad, Quinn, Pitman, and Orr (2005) also found that vmPFC thickness, most notably the medial orbitofrontal cortex, was positively associated with extinction recall. In a more recent study, Milad et al. (2007) found that both hippocampal and vmPFC activation were positively associated with extinction learning.

3.2.2. State anxiety

Like the state fear literature, the animal literature on state anxiety is broadly corroborated by human studies. In contrast to nocturnal animals, humans experience increased anxiety in response to darkness. In light of this fact, Grillon, Pellowski, Merikangas, and Davis (1997) found preliminary evidence that darkness enhanced the human startle reflex. Nevertheless, they also found that self-reported state anxiety did not correlate with the magnitude of this reflex. Moreover, subjects reported the dark and light conditions as equally unpleasant. One limitation to this study is that participants were presumably healthy undergraduates who volunteered to participate. Therefore, negative findings concerning self-reported anxiety may be due to a restricted range at the lower bounds of trait anxiety, although the authors did not report descriptive statistics for self-reported anxiety. A second limitation is that subjects displayed habituation to both light and dark blocks after the first trial. Despite their limitations, these findings suggest that dark spaces may potentiate human startle.

In contrast to fear studies, many studies investigating the neural bases of human state anxiety are equivocal. One primary limitation to these studies is that many use fear-related stimuli, such as fearful faces (e.g., Bishop, Duncan, Brett, & Lawrence, 2004) to elicit anxiety. A secondary limitation is the seemingly conflated interpretation of the data. For example, Straub, Schmidt, Weiss, Mentzel and Miltner (2009) monitored 16 healthy female participants using fMRI while the participants anticipated 4 levels (mild to strong) of electric foot shock. Following the paradigm, participants rated the level of anxiety they experienced on a 9-point Likert-type scale. They found that activation in the pregenual anterior cingulate cortex (ACC) was positive during moderate threat and negative during strong threat. They also found that self-reported anxiety was positively related to dorsal ACC activation during the anticipation of strong threat. The authors interpreted this finding as suggesting that activation of ACC areas is dependent on the level of anxiety and severity of threat, whereby moderate threat is associated with increased attentional avoidance and strong threat with hypervigilance. However, it is unclear whether the authors were measuring self-reported anxiety or self-reported fear, as participants were probably rating their subjective experience of aversive arousal. Moreover, the authors labeled brain activation during a fear-conditioning paradigm as “anxiety.”

3.2.3. Trait fear and trait anxiety

Some studies have explicitly investigated the physiological and neurobiological substrates of trait fear and anxiety in humans. White and Depue (1999) found that self-reported trait fear was inversely related to pupil dilation in response to the introduction of a norepinephrine (which facilitates global alarm functions) agonist, whereas trait anxiety was unrelated to pupil dilation. Additionally, they found that pupil dilation in response to darkness (an evolutionary threat, as noted earlier) was positively related to trait anxiety but unrelated to trait fear. They interpreted these findings as implying that trait fear and trait anxiety are different emotional dispositions.

In terms of central nervous system functioning, several early studies (e.g., Gur et al., 1988; Martinot et al., 1990) found mixed results as to lateralization of fear versus anxiety. Some authors have found that anxiety is right-lateralized (e.g., Gur et al., 1988), whereas others have found that anxiety is bilateral (e.g., Reiman et al., 1989). However, Heller, Nitschke, Etienne, and Miller (1997) suggest that these findings are equivocal because studies often recruited participants from anxiety disordered populations and failed to differentiate whether fear (e.g., specific phobia) or anxiety (e.g., generalized anxiety disorder) was the primary emotion underpinning the anxiety disorder of interest. To address ambiguity regarding the lateralization of trait anxiety, Heller and colleagues administered the trait scale of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) to an initial pool of 2203 participants. From this pool, they selected 20 highly anxious and 20 comparison participants. The experimental task consisted of measuring EEG while participants listened to 30-second, emotionally aversive narratives. The authors found that greater hemispheric asymmetry, specifically more left hemispheric activity, differentiated anxious from comparison participants.

Dien (1999) found that self-reported trait fear correlated with increased right-lateral blood flow in the frontal lobe during an object-spatial recognition task, whereas trait anxiety correlated with increased left-lateral blood flow in the frontal lobes. Funayama, Grillon, Davis, and Phelps (2001) found some support for Dien's findings with a double dissociation of the startle reflex in patients with right or left temporal lobe damage. Patients with right temporal lobe damage reacted appropriately to a task involving uncertainty of threat (i.e. state anxiety), but displayed impaired reaction to visual presentation of threatening stimuli (i.e. state fear). Patients with left temporal lobe damage, in contrast, reacted appropriately to the visual presentation of threatening stimuli (i.e., state fear), but displayed impaired reaction to the uncertainty task (i.e., state anxiety). Moreover, Morinaga et al. (2007) found that right prefrontal brain activity was associated with self-reported fear, but not anxiety, during a fear induction paradigm.

Engels et al. (2007) sought to differentiate neural activity associated with high trait fear (which they referred to as "anxious arousal") versus high trait anxiety (which they referred to as "anxious apprehension") during an emotional Stroop task. They prescreened a group of 1099 participants and selected 11 scoring low on trait fear but high on trait anxiety, 13 scoring high on trait fear but low on trait anxiety, and 18 scoring low on both trait fear and trait anxiety. Their results indicated that the trait fearful group exhibited more activity in the right inferior temporal gyrus for negative than neutral words, whereas the trait anxious group exhibited more activity in the left inferior frontal gyrus for negative than neutral words. Consistent with Dien (1999), their findings suggest that trait fear is characterized by increased right-lateral blood flow, whereas trait anxiety is characterized by increased left-lateral blood flow.

3.2.4. Implications of human neuroscience studies

In summary, human neuroscience studies permit several conclusions:

(1) Autonomic arousal occurs during both fear and anxiety states.
(2) Preliminary evidence for the lateralization of trait fear and anxiety, whereby fear is right-lateralized and anxiety left-lateralized.
(3) Impaired extinction but not acquisition of fear characterizes trait fear, supporting the conceptualization of trait fear as persistent harm avoidance behavior even when it is irrational.
(4) Enhanced arousal during the acquisition phase of fear-conditioning paradigms characterizes trait anxiety, supporting the conceptualization of trait anxiety as hypervigilance.
(5) Evidence suggests that fear and anxious responding can occur inside or outside of awareness, challenging the conceptualization of fear or anxiety as purely cognitively mediated.

4. Operationalizations of fear and anxiety: Consensus and disagreement

Clinical and neuroscientific theorists have offered many conceptualizations of fear and anxiety that converge on some points and diverge on others. Although biological similarities and differences do not necessarily imply experiential differences, the findings from this literature suggest subjective differences in the certainty of threat, direction of defensive response, and duration of experience. In defining fear and anxiety, it becomes necessary to focus on trait emotions rather than clinical disorders or syndromes.

4.1. Defining fear

By informing theoretical conceptualizations with biological findings, we can discern that state fear has several characteristics (see Table 1). Trait fear appears to result, in part, from an underactive extinction circuit. Therefore, avoidance responses persist even with evidence demonstrating the removal of the objective danger. Although extinction of irrational fears remains possible, the learning curve associated with extinction appears to be higher in trait fearful than in other individuals. In terms of awareness, repeated avoidance reactions to fearful stimuli presumably become automatic over time. Therefore, the trait fearful individual can ostensibly avoid specific threat stimuli outside of awareness and in some cases without recalling the associated danger.

In assessing self-reported trait fear, questions addressing avoidant behaviors (that is, the fight, flight, and freeze response) across situations are appropriate. It is not sufficient to ask whether an individual is fearful of specific stimuli without assessing avoidance behaviors. It is likely that individuals cannot discriminate anxiety from fear without appropriate instruction. In other words, individuals may endorse being fearful of aversive stimuli about which they are hypervigilant but do not necessarily avoid. For example, individuals may endorse a fear of flying. However, they may elaborate by stating that they are concerned that something bad will happen when they fly and consequently experience a prolonged state of aversive arousal. In this scenario, individuals are endorsing anxiety surrounding flying (manifested as hypervigilance and hyperarousal during flight), but not fear (avoidance) of flying. Therefore, "How likely are you to avoid flying on an airplane because of fear?" is probably a more valid trait fear item than "How much do you fear flying on airplanes?"

4.2. Defining anxiety

Theoretical and biological findings also point to several characteristics of state anxiety (see Table 1).Trait anxiety appears to result largely from a hypersensitive appraisal circuit. Therefore, persistent hypervigilance and prolonged hyperarousal seem to result from overestimating the potential for threat in ambiguous situations. In humans, this sustained state of hyperarousal manifests itself as apprehension, hypervigilance, and rumination.

In assessing self-reported trait anxiety, generalized questions addressing apprehension and hypervigilance across situations are probably most valid. Similar to fear questionnaires, it may not be sufficient to ask whether an individual experiences anxiety

regarding a specific stimulus or situation. An individual may ascribe anxiety to the aversive arousal experienced before avoiding a specific stimulus (i.e., fear). Therefore, items that assess stimulus non-specific and chronic arousal, such as "I am often worried that something bad will happen," should be most valid for trait anxiety questions.

5. Diagnostic categories of pathological fear and anxiety: Descriptive psychopathology and behavior-genetic findings

The current diagnostic system, the DSM-IV, combines disorders of fear and anxiety under the umbrella category of *anxiety disorders*. However, several clinical researchers have recently questioned the validity of this DSM-IV classification and proposed alternative models that are aligned more closely with research evidence (e.g., Krueger, 1999; Watson, 2005).

Krueger (1999) factor analyzed data from 8098 non-institutionalized participants ranging in age from 15 to 54. To investigate common "comorbidities" (covariations) among mental disorders, Krueger included generalized anxiety disorder (GAD), panic disorder (PD), major depressive disorder (MDD), alcohol dependency, substance dependency, social phobia (SoP), simple phobia (SiP; known as "specific phobia" in DSM-IV), agoraphobia (AG), antisocial personality disorder, and dysthymic disorder (DD) in his analyses. The best fitting model consisted of two correlated lower-order factors, Anxious-Misery (MDD, GAD, and DD) and Fear (SoP, SiP, AG, and PD), that loaded on a higher-order Internalizing Disorders factor. These findings suggest that the DSM-IV category of anxiety disorders is a heterogeneous mix of conditions, some marked primarily by anxiety and others marked primarily by fear. These findings also suggest that at least some mood disorders may primarily be disorders of anxiety or negative emotionality more broadly, not fear.

Watson (2005) reviewed the factor-analytic literature on structural models of DSM-IV diagnoses and proposed a revised structural model consisting of three factors. The Emotional Disorders factor comprised Bipolar Disorders, Distress Disorders, and Fear Disorders sub-factors; the Distress Disorders sub-factor comprised of MDD, DD, GAD, and Posttraumatic Stress Disorder; and the Fear Disorders sub-factor comprised PD, AG, SoP, and SiP. Consistent with Krueger (1999), Watson proposed separate factors for primarily fear-based versus anxiety-based disorders.

Behavioral-genetic studies have similarly pointed to alternative classification models consistent with a distinction between fear- and anxiety-based disorders. Kendler, Prescott, Myers, and Neale (2003) acquired twin diagnostic interview data from more than 5600 individuals in the Virginia Twin Registry using raters blind to co-twin diagnostic status. To increase the number of individuals meeting criteria for PD and GAD, Kendler and colleagues reduced the minimum duration of GAD symptoms from 6 months to 1 month and the minimum number of PD criteria for panic attacks to 2 within 30 min. Their results indicated that MDD and GAD loaded strongly onto an Anxious-Misery sub-factor, whereas animal phobias and situational phobias (e.g., fears of closed spaces, fears of elevators) loaded strongly onto a Fear sub-factor, similar to Watson's (2005) and Krueger's (1999) conceptualizations. Moreover, the two sub-factors again loaded on a higher-order Internalizing Disorders factor. Nevertheless, they found that PD loaded weakly on both the Anxious-Misery and Fear sub-factors, in contrast to Watson (2005) and Krueger (1999). That ambiguity aside, the collective findings point to different genetic underpinnings for primarily anxiety-based and primarily fear-based clinical conditions.

6. Evaluation of the content of self-report fear and anxiety measures

Self-report measures are the primary tools for assessing fear and anxiety in studies of anxiety disorders. There are several hundred self-report measures of fear or anxiety in the literature (see Antony, Orsillo, & Roemer, 2001). Many of these measures assess specific components of fear or anxiety (e.g., dental fear), some assess relevant clinical syndromes (e.g., agoraphobia), and others assess related phenomena (e.g., anxiety sensitivity, namely the fear of anxiety). Nevertheless, few measures assess trait fear or anxiety more broadly. Of the trait measures, the most commonly used fear measures are the Fear Questionnaire (FQ; Marks & Matthews, 1979) and the Fear Survey Schedule (FSS; Wolpe & Lang, 1964), and the most commonly used anxiety measures are the Manifest Anxiety Scale (MAS; Taylor, 1953) and the STAI — Trait Scale. One or more of these measures were used in 84% of the studies in the meta-analysis to be reported later.

6.1. Self-report measures of trait fear

6.1.1. Phobic fear measures

6.1.1.1. FQ. The FQ is a 24-item self-report measure designed to assess phobic fears. The first 17 questions assess the specific fears on a 9-point Likert-type scale, with values ranging from 0 (would not avoid it) to 8 (always avoid it). Item content ranges from fears of public transportation to the sight of blood. The last 6 questions are related to anxiety and distress on a 9-point Likert-type scale, with values ranging from 0 (hardly at all) to 8 (very severely troublesome). Their item content ranges from depression to dissociation.

The FQ has demonstrated adequate internal consistency, with a range of 0.71 to 0.83 across studies (Oei, Moylan, & Evans, 1991). It also demonstrated a 1-week test–retest reliability of 0.82 (Marks & Matthews, 1979); however, the elapsed time between tests was brief so subjects may have recalled their initial responses. Factor analytic studies of the fear items on the FQ typically find a three factor solution (physical, social, and agoraphobia; Oei et al., 1991), although a few authors have not confirmed this structure (Trull & Hillerbrand, 1990). Moreover, these studies do not support a unitary fear factor. Studies have also demonstrated that the FQ is sensitive to changes in fear following treatment (e.g., Marks & Matthews, 1979). Within the present meta-analysis, all studies limited their analyses to the 17 avoidance items.

By the definition of trait fear derived earlier, the first 17 items of the FQ are appropriate for assessing fear across a variety of situations. However, at least some of the FQ content may be outdated or culturally specific. For example, item 5 is "Traveling along by bus or coach." Although many fears appear to be timeless (e.g., sight of blood), the content of the FQ has remained the same for nearly 30 years and may not accurately reflect common phobic fears in today's society (e.g., radiation, computers, driving on freeways).

6.1.1.2. FSS. There are several versions of the FSS; the most popular are the FSS-II (Geer, 1965) and the FSS-III (Wolpe & Lang, 1964). The FSS-II is a 51-item scale developed for research, whereas the FSS-III is a 72-item scale developed for clinical settings (Antony et al., 2001). Questions on the FSS-II and FSS-III assess fears on a 7-point Likert-type scale from 0 (None) to 6 (Terror). In contrast to the FQ, the FSS requires subjects to rate the degree of fear they feel towards a broad range of potentially threatening stimuli.

The FSS-II and III have demonstrated adequate internal consistency ($\alpha > 0.90$; Arrindell, 1980). In terms of construct validity, studies have demonstrated a positive relationship between scores on the FSS-III and the magnitude of fear-potentiated startle to fearful stimuli (Cook, Davis, Hawk, Spence, & Gautier, 1992). Factor analytic studies of the FSS-III have yielded mixed results (e.g., Arrindell, 1980; Bates, 1971). However, two factors, blood/physical injury and social, are consistently found across studies. Similar to the FQ, these studies do not support a unitary fear factor.

Unlike the FQ, the FSS may not assess fears with adequate validity for two reasons. First, the Likert-type scale does not assess avoidance; second, some of the items (e.g., life after death and God) assess abstract,

diffuse, and unavoidable threats akin to anxiety-provoking stimuli. Among the items, 43/51 (84%) assess potentially fear-inducing (rather than strictly anxiety-inducing) stimuli. Using the FQ and FSS-III, Stravynski, Basoglu, Marks, Sengun, and Marks (1995) found evidence for the relative independence for the diagnoses of social, agoraphobic, and specific phobias using the FQ and FSS-III. There are no published studies suggesting that a unitary fear factor provides the best fit to self-reported fear measures. There are also no published studies of the correlations among FQ and FSS-III total and factor scores.

6.1.2. Harm avoidance measures

6.1.2.1. Activity Preference Questionnaire (APQ; Lykken et al., 1973). The APQ is a 74-item forced-choice measure designed to assess fear. Each item consists of two choices, one of which is unpleasant primarily because it is frightening or embarrassing (e.g., "Sleeping out on a camping trip in an area where rattlesnakes have been reported," "Having to blow your nose while in a group of strangers") and the other of which is unpleasant primarily because it is burdensome or onerous (e.g., "Sitting through a two-hour concert of bad music," "Having a gabby old woman sit down next to you on the bus"). Respondents are asked which of the two activities within each pair they would prefer. Within each item, the two choices were matched for social desirability by a panel of judges using a modified Thurstone scaling procedure. The internal consistency of the APQ ranges from 0.82 to 0.86 (Lykken et al., 1973).

6.1.2.2. MPQ — Harm avoidance Scale (HA). The MPQ-HA scale, which was modeled largely after the APQ (Lykken et al., 1973), has been used in a number of studies as a measure of fearfulness. This scale consists of 28 items; many of the items are in forced-choice format and juxtapose a potentially fear-inducing activity (e.g., "Being chosen as the 'target' for a knife-throwing act") with another that is less so (e.g., "Being sick to one's stomach for 24 hours"). Other items ask people how likely they would be to fear or avoid a potentially scary stimulus. A high score on HA indicates a preference for avoiding potentially hazardous situations. Like its "parent" measure, the APQ, the HA scale has adequate internal consistency; for example, across four samples (some including college students, others members of the community), Tellegen and Waller (2007) reported Cronbach's alphas ranging from 0.82 to 0.84.

6.1.2.3. Psychopathic Personality Inventory — Fearlessness Scale (PPI-F; Lilienfeld & Andrews, 1996). The PPI-F scale was designed to assess fearlessness or (reversed HA), considered by many to be a core attribute of psychopathy (Lykken, 1995). This scale consists of 19 items in a 1–4 Likert-type format and, like other PPI scales, was constructed using an iterative approach in which the results of successive exploratory factor analyses informed construct reformulation and item revision (Lilienfeld & Andrews, 1996). Each item assesses the relative absence of fear, typically in one or more specific situations or settings (e.g., "I would find the job of movie stunt person exciting"). Across two undergraduate samples, Lilienfeld and Andrews (1996) reported internal consistencies (Cronbach's alphas) of 0.86 and 0.88 for the PPI-F scale. Correlations between this scale and trait anxiety measures were reflected in direction in the analyses reported here given that this scale assesses reversed fear/HA.

6.1.3. Self-report measures of trait anxiety

6.1.3.1. MAS. The MAS is a 50-item true/false scale of trait anxiety derived from the Minnesota Multiphasic Personality Inventory (MMPI). The internal consistency of the MAS is typically high (α>0.9; King & Campbell, 1986). Using MAS data, Buss (1962) found two primary anxiety factors, physiological hyperreactivity and subjective feelings of apprehension. Fenz and Epstein (1965), however, found that a unitary anxiety factor accounted for nearly 75% of the variance in the scale.

Among the items on the MAS, 4/50 (8%) are explicit fear items (e.g., "I do not have as many fears as my friends") and 5/50 (10%) are related ambiguously to anxiety (e.g., "I cry easily"). Therefore, 41/50 (82%) appear to be explicit anxiety questions.

6.1.3.2. STAI-T. The STAI-T is a 20-item scale designed to assess pervasive feelings of anxiety. Items are rated by respondents on a 4-point Likert-type scale. The STAI-T has demonstrated acceptable internal consistency (α>0.85) and 1-month test/retest reliability (r>0.70) in adolescent, healthy adults, and military samples (Spielberger et al., 1983). Factor analytic studies (e.g., Bieling, Antony, & Swinson, 1998) suggest that a two-factor model, consisting of depression and anxiety, best fits the data. Bieling and colleagues found that the depression factor correlated more strongly with measures of depression than anxiety, whereas the anxiety factor correlated more strongly with measures of anxiety than depression. This finding and others suggest that the STAI-T is heterogeneous in content and indexes construct-irrelevant variance (Messick, 1995) stemming from mood disturbance.

Among the items on the STAI-T, 5/20 (25%) are explicit depression items (e.g., "I wish I could be as happy as others seem to be") and 1/20 (5%) are ambiguously related to anxiety (e.g., "I have disturbing thoughts"). Therefore, 14/20 (70%) are explicit anxiety questions. Despite the differences in format and item content, correlations between the STAI-T and MAS are high, ranging from 0.72 to 0.92 (e.g., Watson & Clark, 1984).

7. Meta-analysis of self-report fear and anxiety measures

Our meta-analysis sought to evaluate the relationship between scores on frequently used measures of self-reported trait fear and trait anxiety. Based on the clinical and neurobiological literatures, we generated the following hypotheses.

7.1. Hypothesis 1: Primary meta-analysis

As the preponderance of evidence from these literatures suggests that trait fear and trait anxiety are separable phenomena, we hypothesize that the relationship between self-reported trait fear and trait anxiety will be low or at best moderate. Although three author teams who conducted narrative reviews (Perkins et al., 2007; Watson & Clark, 1984; White & Depue, 1999) have reported that the correlations between trait fear and trait anxiety appear to be low, none has examined this association meta-analytically.

7.2. Hypothesis 2: Self-report measure as moderator

We also investigate the moderating effect of self-report measure. From our evaluation of the content of commonly used trait fear and anxiety measures, we found that some measures contain less unrelated or overlapping items than other measures. Therefore, we hypothesize that the relationship between fear and anxiety will be significantly weaker in the FQ versus the FSS as well as the MAS versus the STAI-T. Moreover, we hypothesize that the relations between trait anxiety and trait fear assessed using HA measures (e.g., MPQ-HA) will be significantly weaker than with trait fear assessed using specific fear measures (e.g., FQ).

7.3. Exploratory analyses: Age, gender, and psychiatric status as moderators

In addition to testing these two hypotheses, we conduct exploratory analyses investigating whether age, gender, and psychiatric population moderate the relation between trait fear and trait anxiety. Because we do not have access to the ages of participants in the studies, we investigate age as a categorical moderator with child

Table 2
Phobic fear and anxiety scale correlations in child samples.

Study	Measures	*N*	*r*	ES_{zr}
Byrne (2000)	FSS-C; STAI-C	300	0.50*	0.55
King, Gullone, and Ollendick (1992)	FSS-C-R; RCMAS	1524	0.53*	0.59
Muris et al. (2003) Sample 1	KFS; STAI-T	189	0.29*	0.30
Muris et al. (2003) Sample 2	KFS; STAI-T	163	0.59*	0.68
Muris et al. (1998)	FSS-C; SCRAD	120	0.44*	0.47
Ollendick (1983) Sample 1	FSS-C; STAI-C	99	0.51*	0.56
Ollendick (1983) Sample 2	FSS-C; STAI-C	118	0.46*	0.50
Peleg-Popko and Dar (2001)	CFI; CSAI	108	0.54*	0.60
Scherer and Nakamura (1968)	FSS-II; MAS-C	99	0.49*	0.54
Schmidt and Mallott (2005)	FNES; STAI-T	405	0.33*	0.34
Shore and Rapport (1998)	FSS-R; MAS-C	385	0.31*	0.32
Silverman, Fliesig, Rabian, and Peterson (1991)	FSS-C; STAI-T	81	0.57*	0.65
Walters (2001)	FQ-C; STAI-C	149	0.50*	0.55
Wilson and Hayward (2006)	FQ; STAI-T	2246	0.31*	0.32

Note. ES_{zr} = Fisher's Zr-transform; FNES = Fear of Negative Evaluation Scale. FQ = Fear Questionnaire. FSS = Fear Survey Schedule. FSS-C = Fear Survey Scale — Child Version. MAS-C = Manifest Anxiety Scale — Child Version. RCMAS = Revised Child Manifest Anxiety Scale. STAI-C = State-Trait Anxiety Inventory — Child Scale. STAI-T = State-Trait Anxiety Inventory — Trait Scale.
* $p<0.05$.

and adult categories. As the anxiety disorders of PD (e.g., Oei, Evans, & Crook, 1990) and GAD (e.g., Oei et al., 1991) are not necessarily associated with trait anxiety and trait fear, respectively, we conduct exploratory analyses to investigate whether the relationship is weaker in clinical than non-clinical samples. As we do not have specific diagnostic information for most participants, we investigate psychiatric versus community samples as a categorical moderator.

Table 3
Phobic fear and anxiety scale correlations in adult samples.

Study	Measures	*N*	*r*	ES_{zr}
Bates (1971)	FSS-III; MMPI-A	41	0.57*	0.65
Carleton, Collimore, and Asmundson (2007)	BFNE; SIAS	322	0.64*	0.76
Carsrud and Carsrud (1979)	FSS; MAAC	99	−0.07	−0.07
Dien (1999)	FSS; STAI-T	63	0.40*	0.42
Geer (1965)	FSS-II; MAS	205	0.42*	0.45
Grossberg and Wilson (1965)	FSS-III; MAS	505	0.46*	0.50
Hagopian and Ollendick (1996)	FQ; STAI-T	61	0.38*	0.40
Hersen (1971)	FSS-III; MAS	351	0.46*	0.50
Kent and Keohane (2001)	FNES; HADS-A	141	0.48*	0.52
Kilpatrick and McLeod (1973)	FSS-III-M; STAI-T	36	0.52*	0.58
Kilpatrick, Sutker, Roitzsch, and Mason (1975)	FSS-III-F; STAI-T	211	0.39*	0.41
Kocovski and Endler (2000)	FNES; EMAS-S	174	0.41*	0.44
Kogan and Edelstein (2004)	FSS-II; BAI	114	0.40*	0.42
Lang and Lazovik (1963)	FSS; MAS	13	0.81*	1.13
Lerner and Keltner (2001) Study 1	FSS-II; STAI-T	75	0.54*	0.60
*Lerner and Keltner (2001) Study 2	FSS-II; STAI-T	601	0.57*	0.65
*Manosevitz and Lanyon (1965)	FSS-III; MAS	46	0.27	0.28
*Mattick and Clarke (1998)	FQ-SP; STAI-T	42	0.51*	0.56
Mellon (2000)	FSS-GV; MMPI-A	696	0.49*	0.54
Perkins et al. (2007) Sample 1	FSS-III; STAI-T	141	0.21	0.21
Perkins et al. (2007) Sample 2	FSS-III; STAI-T	101	0.34*	0.35
Schroeder and Craine (1971)	FSS-III; MAS	107	0.56*	0.63
Stanley, Beck, and Zebb (1996) Sample 1	FQ; STAI-T	50	0.34*	0.35
Stanley et al. (1996) Sample 2	FQ; STAI-T	94	0.43*	0.46
Weeks et al. (2005)	BFNE; SIAS	148	0.38*	0.40

Note. ES_{zr} = Fisher's Zr-transform; BAI = Beck Anxiety Inventory. BFNE = Brief Fear of Negative Evaluation Scale. EMAS = Endler Multidimensional Anxiety Scale. FNES = Fear of Negative Evaluation Scale. FQ = Fear Questionnaire. FSS = Fear Survey Schedule. FSS-GV = Fear Survey Schedule — Greek Version. HADS-A = Hospital Anxiety and Depression Scale — Anxiety. MAAC = Multiple Affective Adjective Scale. MAS = Manifest Anxiety Scale. MMPI = Minnesota Multiphasic Personality Inventory. SIAS = Social Interaction Anxiety Scale. STAI-T = State-Trait Anxiety Inventory — Trait Scale.
* $p<0.05$.

Table 4
Harm avoidance and anxiety scale correlations in adult samples.

Study	Measures	*N*	*r*	ES_{zr}
Blankstein (1976)	APQ; STAI-T	101	0.05	0.05
Church (1994)	MPQ-HA; NEO-A	674	0.20*	0.20
Helfritz and Stanford (2006)	PPI-F; PAI-A	39	0.23	0.23
Hodges and Felling (1970)	SSQ-P; STAI-T	228	0.02	0.02
Lilienfeld and Andrews (1996)	PPI-F; MPQ-SR	110	0.06	0.06
Lykken et al. (1973)	APQ; STAI-T	25	−0.01	−0.01
Newman and Schmidt (1998)	MPQ-HA; WAS	207	−0.22*	−0.22
Poythress et al. (n.d.)	MPQ-HA; STAI-T	1473	−0.05	−0.05
Schmidt and Newman (1999)	MPQ-HA; WAS	217	−0.15	−0.15
Sellbom and Ben-Porath (2005)	MPQ-HA; MMPI-2 RCP	985	0.04	0.04
Uzieblo, Verscuerea, and Crombez (2007)	PPI-F; STAI-T	120	0.14	0.14
Waller, Lilienfeld, Tellegen, and Lykken (1991)	MPQ-HA; TPQ	1236	0.22*	0.22

Note. ES_{zr} = Fisher's Zr-transform; APQ = Activities Preference Questionnaire. MMPI-2 RCP = Minnesota Multiphasic Personality Inventory — Restructured Clinical Scale Psychasthenia. MPQ-HA = Multidimensional Personality Questionnaire — Harm avoidance Scale. MPQ-SR = Multidimensional Personality Questionnaire — Stress Reaction Scale. PAI-A = Personality Assessment Inventory — Anxiety Scale. PPI-F = Psychopathic Personality Inventory, Fearlessness Scale, reverse scored. SSQ-P = Stressful Situations Questionnaire — Physical Danger Scale. STAI-T = State-Trait Anxiety Inventory — Trait Scale.
* $p<0.05$.

8. Method

8.1. Literature search and selection criteria

We identified relevant studies primarily through a computerized database search of journal articles using Psycinfo and Medline with the constraints of human studies, peer-reviewed journals, written in the English language using the key words *anxiety*, *anxious*, *fear*, *harm avoidance*, *neurotic*, *panic*, *phobia*, *phobic*, and *self-report*. If the title of a paper included the search terms *fear* and *anxiety*, the entry was automatically included in an initial pool of potential studies. In secondary searches, we used the names of commonly used self-report measures of fear and anxiety as key words, including *Fear Questionnaire*, *Fear Survey Schedule*, *Manifest Anxiety Scale*, *Multidimensional Personality Questionnaire*, *Psychopathic Personality Inventory*, and *State-Trait Anxiety Inventory*. In addition to using databases, we inspected reference lists from retrieved articles and books to generate potential studies. Finally, as the relationships of interest were often buried within studies, we perused issues of a specialized anxiety journal, the *Journal of Anxiety Disorders*, from the publication dates 1987 (the first year of publication) through 2009.

Studies reporting within-measure correlations among subscales (e.g., MPQ-Stress Reaction and MPQ-Harm Avoidance, PPI-Stress Immunity and PPI-Fearlessness) were excluded from the meta-

Table 5
Gender and correlations between phobic fear and anxiety scales.

Study	Measures	Male			Female		
		N	*r*	ES_{zr}	*N*	*r*	ES_{zr}
Byrne (2000)	FSS-C; STAI-C	150	0.49	0.54	150	0.50	0.55
Geer (1965)	FSS; MAS	150	0.39	0.41	55	0.55	0.62
Grossberg and Wilson (1965)	FSS; MAS	203	0.42	0.45	302	0.45	0.48
Gullone and King (1992)	FSS-C; STAI-C	207	0.38	0.40	225	0.31	0.32
Hersen (1971)	FSS-III; MAS	160	0.42	0.45	191	0.52	0.58
Ollendick (1983) 1*	FSS-C; STAI-C	57	0.44	0.47	42	0.56	0.63
Ollendick (1983) 2*	FSS-C; STAI-C	51	0.32	0.33	67	0.50	0.55
Scherer and Nakamura (1968)	FSS-II; MAS-C	59	0.41	0.44	40	0.52	0.58

Note. ES_{zr} = Fisher's Zr-transform; FSS = Fear Survey Schedule. FSS-C = Fear Survey Scale — Child Version. MAS = Manifest Anxiety Scale. MAS-C = Manifest Anxiety Scale — Child Version. STAI-C = State-Trait Anxiety Inventory — Child Version. All correlations reached significance at $p<0.05$.

analysis. Because these subscales were derived from factor-analytically developed measures that were intended to yield largely independent dimensions, the inclusion of these studies could artificially weaken the relationship between trait fear and trait anxiety (cf., White & Depue, 1999, who included these correlations in their narrative review). In the case of the MPQ, its 11 lower-order scales were designed to be largely independent (Tellegen & Waller, 2007). In the case of the PPI, items that loaded highly on more than one lower-order factor were discarded in the process of subscale development (Lilienfeld, 1990). Hence, our analyses focused exclusively on correlations between fear and anxiety subscales derived from different omnibus measures of personality or psychopathology. These analyses represent a "stringent" test of our central hypotheses, as they exclude numerous studies in which trait fear and trait anxiety correlations are likely to be spuriously low.

8.1.1. Inclusion criteria

The following criteria were used to select studies for inclusion in the meta-analysis:

1. The study was based on data published in a peer-reviewed, English journal through December 2009.
2. The study included self-report measures of trait fear and trait anxiety (i.e., studies using measures of a specific fear or anxiety were excluded).
3. The study reported data that allowed the computation of an effect size of the relation between the measures. In several cases where these data were not reported, we contacted authors for their raw data.
4. The study was based on correlations between trait fear and trait anxiety measures derived from different broadband measures.

8.1.2. Search results

These search strategies generated a list of approximately 800 articles. These articles were retrieved and examined for inclusion criteria. Only 55 of the 800 articles reported association between self-reported fear and anxiety. Of the 55 articles, we excluded 9 from the meta-analysis for using measures tailored towards specific, rather than trait, fear or anxiety. An additional dataset containing the variables of interest (which have formed the basis for several publications, but in which the intercorrelations between trait fear and trait anxiety were not reported) was identified by correspondence with the authors (Poythress et. al, unpublished).

8.2. Data analytic strategy

In total, this search strategy yielded 47 articles suitable for the current meta-analysis (see Tables 2, 3, and 4). Of these articles, five included more than one sample. For Hypothesis 1, we aggregated 52 effect sizes ($N = 17{,}620$) for the meta-analysis. In exploratory analyses, these studies were further evaluated for use in categorical moderator analyses relevant to age, gender, and population. For the analysis of age, 12 studies ($n = 4340$) reported the relation in non-psychiatric child samples and 19 studies ($n = 3{,}628$) in non-psychiatric adults samples. Because no studies reported the relation in psychiatric child samples, we excluded psychiatric adult samples from this analysis. For the analysis of gender, eight studies reported the relation in male ($n = 1037$) and female ($n = 1072$) separately. For the analysis of population type, 7 studies ($n = 921$) reported the relation in psychiatric adult populations and 19 studies ($n = 3628$) reported the relation in non-psychiatric (and non-forensic) adult populations.

For Hypothesis 2, the moderator analyses investigated whether the relationships between widely used measures of trait fear and trait anxiety differed significantly across measures. In the first analysis, we compared the relations between trait anxiety and 3 different phobic fear measures: FQ (5 studies, $n = 2493$), FSS (24 studies, $n = 4607$), and BFNE (5 studies, $n = 1190$). Second, we compared the phobic fear (FQ, FSS, BFNE; 34 studies, $n = 8290$) measures' relations with anxiety measures, on the one hand, with those of the HA (PPI-F, MPQ-HA, APQ; 13 studies, $n = 6765$) measures, on the other. To account for the possible effect of anxiety measure, we conducted a secondary analysis investigating relations between the FQ (4 studies, $n = 2451$) and FSS (12 studies, $n = 2037$) with the STAI-T. In the second analysis, we compared two anxiety measures: STAI-T (11 studies, $n = 1829$) and MAS (7 studies, $n = 1305$). The FSS was the only fear measure used in the MAS studies, so we only used the 11 studies correlating the STAI with the FSS for the analysis to minimize systematic error.

8.2.1. Computing effect sizes

In the current meta-analysis, effect sizes index the relationship between self-report scores on measures of anxiety with those of self-report measures of fear. Within studies, the effect sizes were reported as Pearson correlations. Effect sizes for individual studies were estimated using the unbiased estimator Fisher's Z_r-transform (Hedges & Olkin, 1985), defined as $ES_{zr} = 0.5 \log_e [1 + r/1 - r]$. We used Microsoft Excel 2003 to compute all effect-size estimates.

8.2.2. Effect-size aggregation

The grand mean, as well as means for each level of a given categorical moderator, was computed by using an estimate of the inverse of each estimates sampling variance (Hedges & Olkin, 1985). This weighting scheme accords greater weight to more precise effect-size estimates (Matt & Cook, 1994). Upon averaging the weighted effect sizes, the overall effect size was converted back into a Pearson correlation for clarity of presentation.

8.2.3. Categorical moderator analyses

We compared weighted mean effect sizes from different levels of the same moderator with the Q test statistic. The Q statistic is a between-group homogeneity test, analogous to a pairwise comparison with two categories, derived from formulae in Hedges and Olkin (1985), and defined as $Q = \Sigma w_i (ES_i - ES)^2$ (Lipsey & Wilson, 2001). The Q statistic is distributed as a chi-square with $k - 1$ degrees of freedom, where k is the number of groupings. If the Q statistic exceeds the corresponding chi-square critical value, one can infer that the effect differs across groups (e.g., the relation is different in male than in female).

9. Results

9.1. Hypothesis 1: Primary meta-analysis

We computed 52 effect sizes from the studies in the target literature. Of these effect sizes, 39 (75%) were significantly positive, 12 (23%) were nonsignificant, and 1 (2%) was significantly negative. Tables 1, 2, and 3 display effect sizes and relevant study characteristics for each study included in the analysis. Results of the main meta-analysis are consistent with Hypothesis 1 and indicated a moderate relationship ($r = 0.32$) between trait fear and trait anxiety scales, with correlations ranging from -0.22 to 0.81. Fail-safe N calculations (Rosenthal, 1979) indicated that an additional 114 studies reporting null results would be required to reduce the overall correlation to 0.10, and 1612 studies to reduce the overall correlation to 0.01.

9.2. Hypothesis 2: Self-report measure as a moderator

9.2.1. Fear measure as a moderator

We computed the relations between trait anxiety and 3 measures of phobic fear using 34 effect sizes (5 BFNE samples, 5 FQ samples, and 24 FSS samples). Consistent with Hypothesis 2, our results indicated that the relation with trait anxiety is significantly different

($Q = 43.32$, $df = 2$, $p < 0.05$) between the BFNE ($r = 0.45$), FQ ($r = 0.32$), and FSS ($r = 0.45$). In subsidiary analyses using only one measure of anxiety, 16 effect sizes (4 FQ and 12 FSS) were computed measuring the relation with the STAI-T. Consistent with Hypothesis 2, our results indicated that the relation with trait anxiety is significantly weaker ($Q = 39.45$, $df = 1$, $p < 0.05$) for the FQ ($r = 0.32$) than for the FSS ($r = 0.48$).

We also tested whether the relations differed between HA measures (6 MPQ-HA, 3 PPI-F, 2 APQ, and 1 Stressful Situations Questionnaire — Physical Danger Scale (Hodges & Felling, 1970) sample) and physical phobic fear measures (FQ and FSS) with trait anxiety. As HA measures were only assessed in adult samples, we only used phobic fear studies in adults (4 FQ and 18 FSS) as a comparison. Consistent with Hypothesis 2, our results indicated that the relation between trait anxiety and HA measures ($r = 0.14$) is significantly weaker ($Q = 308.29$, $df = 1$, $p < 0.05$) than the relation between trait anxiety and phobic fear measures ($r = 0.46$).

9.2.2. Anxiety measure as a moderator

We used 19 effect sizes (12 STAI-T samples and 7 MAS samples) to test whether the relations with trait fear differed across anxiety measures. Contrary to Hypothesis 2, our results indicated that relations with the FSS are not significantly different ($Q = 0.22$, $df = 1$, $p = $ ns) in the STAI-T ($r = 0.48$) compared with the MAS ($r = 0.46$).

9.3. Exploratory analyses: Age, gender, and psychiatric status as moderators

9.3.1. Age as a moderator

Thirty-one effect sizes (12 child samples and 19 adult samples) were computed from the studies in the target literature. Our results indicated that the correlation between fear and anxiety measures is not significantly different ($Q = 2.38$, $df = 1$, $p > 0.05$) in adults ($r = 0.44$) and children ($r = 0.42$).

9.3.2. Gender as a moderator

Sixteen effect sizes (8 male and 8 female) were computed to test whether gender moderated the association between trait fear and anxiety (see Table 5). Our results indicated that the relationship is nonsignificantly different ($Q = 1.50$, $df = 1$, $p = $ ns) in male ($r = 0.41$) and female ($r = 0.46$).

9.3.3. Psychiatric status as a moderator

To test the moderating influence of psychiatric status on the relation between trait fear and anxiety, 26 effect sizes (7 psychiatric and 19 non-psychiatric samples) were computed. Our results indicated that the relation is nonsignificantly different ($Q = 0.16$, $df = 1$, $p = $ ns) between psychiatric ($r = 0.45$) and non-psychiatric ($r = 0.44$) populations.

9.4. Summary of our meta-analytic findings

In summary, the overall relation between measures of trait fear with measures of trait anxiety was moderate. The association was significantly weaker among measures of HA than measures of phobic fear. Among phobic fear measures, the relation of trait anxiety with the FQ was significantly weaker than with the FSS. The relation between measures of trait fear and trait anxiety was not moderated by age, gender, or psychiatric status.

There were several limitations to our meta-analysis. First, it is unlikely that the search strategy located all relevant studies. Although the strategy was intensive, correlations of interest were often buried within studies as secondary analyses. Therefore, even fairly comprehensive database searches are likely to overlook pertinent studies. Second, the effect sizes were derived from presumably flawed measures. Although error variance is inherent in all self-report measures, a portion of the error variances in these measures was probably systematic due to their flawed conceptualizations of trait fear and anxiety. Third, the data collected for the meta-analysis were not sufficient for testing the potential moderating effect of subtype of phobic fear (e.g., social versus physical injury) on the relation between trait fear and trait anxiety.

10. Discussion and implications

Our review points to three broad implications regarding the relation between trait fear and trait anxiety. First, a wealth of research across scientific disciplines suggests that trait fear and trait anxiety reflect different emotions with separable correlates. Second, these literatures suggest that avoidance behaviors across several situations characterize trait fear, whereas sustained hypervigilance and prolonged hyperarousal while approaching several situations characterizes trait anxiety. These literatures dovetail with our finding that the relation between trait fear and trait anxiety was especially weak when the trait fear measures emphasized avoidance (viz., HA). Third, results from the meta-analysis suggest that the subjective experiences of trait fear and trait anxiety are largely independent, although probably at least somewhat overlapping, phenomena.

Although the differentiation and operationalization of trait fear from trait anxiety remains a contentious topic among clinical theorists, the preponderance of research evidence we have reviewed demonstrates that the two emotions are separable and derived largely from different etiological substrates. First, the neurobiological literature suggests that state fear and state anxiety are differentiated primarily by activation in the CeA and BNST, respectively. More provisionally, this literature also suggests that trait anxiety is characterized by increased left frontal hemispheric activity, whereas trait fear is characterized by increased right hemispheric activity. Consistent with the neurobiological findings, diagnostic and behavior-genetic studies have found that disorders characterized by trait fear versus trait anxiety load on separable factors and differ in their genetic underpinnings.

The neurobiological literature further supports a behavioral distinction between state fear and state anxiety. Although both emotional states are experienced as aversive and motivate behavior, they push behaviors in opposite directions. State fear is an aversive emotional state during which an organism is motivated to escape a specific and imminent threat. The characteristics of state fear include short-lived arousal that quickly dissipates after the threat is avoided. Trait fear, therefore, is the persistent and pervasive experience of state fear across situations. In colloquial terms, a “scaredy cat” is a vivid description of a trait fearful individual, who avoids taking risks that most perceive as relatively benign but does not appear distressed when these risks are absent.

State anxiety, in contrast, is an aversive emotional state that occurs while an organism approaches an ambiguous and uncertain threat. The behavioral characteristics of state anxiety include sustained hypervigilance and hyperarousal, which persist even after the potential for threat is removed. Trait anxiety is therefore the persistent and pervasive experience of state anxiety across situations. In colloquial terms, a “worry wart” is a vivid description of a trait anxious individual, who does not avoid situations but remains persistently hypervigilant for potential threats (Barlow, 2002).

Our meta-analysis suggests that the subjective experiences of trait fear and trait anxiety are distinguishable. Nevertheless, one major limitation to this meta-analysis was the content overlap between self-report measures of trait fear and trait anxiety. Our finding from the investigation of self-report measure as a categorical moderator suggests that the relation is significantly weaker with reduced content overlap. This finding suggests that a refinement of self-report measures of fear and anxiety to reduce cross-contamination of constructs and construct-irrelevant variance (Messick, 1995) is

warranted. Specifically, self-report measures of trait fear should emphasize freezing and avoidance behaviors aimed at an array of specific threats, whereas self-report trait anxiety should emphasize hypervigilance, uncertainty, and hyperarousal. In this respect, measures of HA, which our analyses showed to be the most independent of trait anxiety, are presumably more valid for detecting trait fear than are measures of phobic fear, which our analyses showed to be moderately correlated with trait anxiety. Moreover, these measures should directly assess the degree that an individual displays a propensity for these traits, rather than measuring a clinical syndrome (e.g., the inclusion of depression items on the STAI-T).

10.1. Implications for diagnostic classification systems

Our literature review and meta-analysis lend some support to Epstein's (1972) clinical conceptualizations of trait fear and anxiety as separable phenomena with differing etiologies, whereas they call into question Beck and Emery's (2005) clinical conceptualization as slightly different aspects of a single underlying phenomenon. Although data from the meta-analysis do not provide unambiguous support for Tellegen's (1985) assertion that trait fear and trait anxiety are virtually orthogonal constructs, our moderator analyses suggested that inappropriate item overlap across scales inflated the relationship. Therefore, Tellegen's position cannot be falsified based on the present results. Moreover, our moderator analyses suggested that, consistent with Tellegen's position, measures of trait anxiety and HA are at best only when weakly correlated. Our results are also consistent with recent analyses suggesting that disorders of fear and disorders of anxiety warrant classification in separate higher-order categories.

Results from our meta-analysis point to the need to devise consistent operationalizations of trait fear and trait anxiety in the personality and clinical literatures, and to develop self-report measures consistent with these operationalizations. Because our review indicates that trait fear and trait anxiety are largely independent, it supports Krueger's (1999) and Watson's (2005) contention that the current diagnostic category of *anxiety disorders* is not justified on empiric grounds. Moreover, it suggests that studies investigating levels of trait fear and anxiety characteristic of mental disorders is warranted. With appropriate assessment measures, these empirical questions would be testable and clearer conceptualizations of the role of trait fear and anxiety in mental disorders would be possible.

10.2. Clinical implications

Anxiety disorders, along with impulse control disorders, are among the most commonly diagnosed mental disorders (DuPont et al., 1996). Given the gravity and prevalence of these disorders, research efforts investigating their pathogenesis, differential characteristics, and treatment are numerous. Although many of these studies use measures designed to assess symptoms rather than underlying personality features, there is sufficient evidence for the differentiation of the two emotion systems to suggest that including theoretically and empirically sound measures of trait fear and anxiety in these studies is warranted. Moreover, including measures of trait fear and anxiety in treatment outcome studies of anxiety disorders may help us better understand the mechanisms of change in psychotherapy (e.g., do changes in trait anxiety precede changes in trait fear or vice versa?).

10.3. Implications for neuroscience

These findings also bear important implications for neuroscience research related to the treatment of anxiety disorders. When using self-report measures based on questionable conceptualizations of trait fear and anxiety to study the neurobiological underpinnings of these emotional dispositions, research is unlikely to yield replicable findings across measures and studies. As Heller's et al. (1997) findings suggest, the conflation of the constructs can produce ambiguity in the literature on cerebral laterality and probably other psychophysiological phenomena. Therefore, a refined operationalization of trait fear and trait anxiety, along with construct measures deduced from these definitions, may better elucidate the neural underpinnings of these trait emotions.

10.4. General summary and future directions

Overall findings from our review and meta-analysis suggest that trait fear and trait anxiety are separable constructs. Nevertheless, many widely used self-report measures used to assess these constructs are largely inadequate in light of the existing clinical and neuroscience literatures. Therefore, studies aimed at developing more construct valid – and less construct contaminated – self-report measures of trait fear and anxiety are warranted.

In closing, our review suggests several fruitful directions for future research. First, studies should strive to develop self-report measures of trait fear and trait anxiety based on the well-developed neurobiological and diagnostic research literatures. Such work should help to ascertain the extent to which the low to moderate correlations between trait fear and trait anxiety measures are due to artifacts, such as method covariance or content overlap, as opposed to genuine substantive overlap. Second, studies should investigate the neurobiological, cognitive, and behavioral correlates of these measures to better ascertain their etiological underpinnings. Functional brain imaging studies examining the patterns of neural activation in fear versus anxiety, at both state and trait levels, are especially needed.

Third and finally, studies should investigate the relationships of both self-reported trait fear and trait anxiety with psychological disorders. Such work should help to ascertain which disorders currently housed within the broad anxiety disorders category are primarily disorders of trait fear as opposed to trait anxiety. Moreover, such work may bear implications for a number of conditions outside the current anxiety disorders category but in which abnormal (either high or low) levels of fear or anxiety play a prominent role, such as somatoform disorders, impulse control disorders, childhood disruptive disorders, and personality disorders. For example, although a number of authors (e.g., Lykken, 1995) have argued that psychopathy is characterized by low trait fear, this claim remains controversial, in part because the negative correlations between psychopathy and trait fear are often low in magnitude (Schmidt & Newman, 1999). Moreover, it is unclear which, if any, psychopathological syndromes are characterized by low trait anxiety (Watson & Clark, 1984). A clearer conceptual and empirical delineation between fear and anxiety will be an essential first step toward addressing these questions.

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