Anxiety and Decision-Making

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Although the everyday decision-making of clinically anxious individuals is clearly influenced by their excessive fear and worry, the relationship between anxiety and decision-making remains relatively unexplored in neuroeconomic studies. In this review, we attempt to explore the role of anxiety in decision-making with a neuroeconomic approach. We first review the neural systems mediating fear and anxiety, which overlap with a network of brain regions implicated in studies of economic decision-making. We then discuss the potential influence of cognitive biases associated with anxiety upon economic choice, focusing on a set of decision-making biases involving choice in the face of potential aversive outcomes. We propose that the neural circuitry supporting fear learning and regulation may mediate the influence of anxiety upon choice and suggest that techniques for altering fear and anxiety may also change decisions.

Key Words: Amygdala, anxiety, decision-making, fear conditioning, neuroeconomics, prefrontal cortex

The tendency to experience anxiety is a relatively consistent individual trait (1), suggesting that it has stable underlying neural substrates and may be an important factor driving behavioral variation in a variety of domains, including decision-making. In patients suffering from anxiety disorders, heightened anxiety interferes with the ability to adaptively function in everyday tasks, such as employment or social relations. Although it is clear for these patients that their pathological anxiety influences their daily decisions, a more nuanced understanding of the relationship between anxiety and decision-making is needed. Although anxiety has long been known to involve behavioral aberrations in the face of potential negative outcomes, the burgeoning field of neuroeconomics provides a structured approach to studying the computational and neurobiological mechanisms underlying this dysfunction. Neuroeconomic studies typically define mathematically the optimal or normative behavior in a decision-making task, allowing precise quantification of individual deviations from these norms. These parameters can then be used to probe the neural correlates of decision biases. Characterizing the specific decision biases occurring with anxiety may enhance our understanding of the consequences of individual variability in nonclinical trait anxiety as well as the nature of the dysfunction underlying anxiety disorders.

In this review, we build on behavioral and neuroeconomic principles of decision-making to explore the impact of anxiety on specific decision variables. We first review the neural systems implicated in fear and anxiety, which overlap with those highlighted in neuroeconomic studies of decision-making. We then discuss how anxiety-induced alterations in the brain circuitry of fear result in predictable cognitive biases that may influence later choices. We review the relatively few studies using behavioral economic paradigms to characterize how anxiety influences decision variables, speculating as to how the effects of anxiety on choice may arise from cognitive biases associated with anxiety and the neural circuitry implicated in fear. Finally, we discuss how techniques for altering fear and anxiety may also change decisions.

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Neurocircuitry of Fear and Anxiety

Fear and anxiety share many common cognitive and physiological properties; however, they can also be distinguished (2). Fear responses are elicited by specific stimuli and tend to be short-lived, decreasing once a threat has dissipated. Anxiety may be experienced in the absence of a direct physical threat and typically persists over a longer period of time. However, anxiety is commonly conceptualized as a state of sustained fear (3).

Studies of fear neurocircuitry highlight a network of brain regions enabling the adaptive expression of fear to potential threats and its inhibition and control with safety. Details of this circuitry have been investigated across species using classical fear conditioning as a model paradigm. During fear conditioning, a previously neutral stimulus, such as a tone, is paired with an intrinsically aversive stimulus, such as an electric shock, eliciting a range of automatic, unconditioned fear responses (4,5). After one or more tone-shock pairings, presentation of the tone alone is sufficient to elicit a fear response, the conditioned response, providing evidence of a learned association between the two stimuli. Once acquired, conditioned fear can be diminished via a number of techniques (6). In extinction, the tone is presented repeatedly without the shock, resulting in a gradual decrease in conditioned fear expression. Evidence that fear can return after successful extinction training after the passage of time (spontaneous recovery), changes in context (renewal), or stress (reinstatement) suggests the original fear memory is not erased with extinction but is rather inhibited during the expression of extinction learning (7). In humans, intentional cognitive strategies including emotional suppression, redeployment of attention, or cognitive reinterpretation or reappraisal of the significance of a stimulus may also be used to diminish fear (6).

The neurocircuitry supporting fear conditioning has been extensively investigated in animal models and humans (4,5,8) and highlights the central role of the amygdala in fear acquisition, storage, and expression. The amygdala is thought to be the site of association and memory storage for simple cued fear conditioning, with projections to the brainstem and hypothalamus mediating autonomic fear expression and projections to the ventral striatum mediating the use of actions to cope with fear (5). In addition, the hippocampus plays an important role in contextual modulation of fear, supporting the acquisition of fears to contexts and guiding the contextually dependent expression of fear (9). Although their specific roles are less clearly defined, the insula and dorsal anterior cingulate cortex are also proposed to modulate fear acquisition (10,11).

The inhibition or control of conditioned fear requires the ventromedial prefrontal cortex (vmPFC), which is necessary for the storage of extinction memory. During extinction retrieval, projections from the vmPFC to inhibitory interneurons within the amygdala diminish fear expression. After extinction, contextual information modulates

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the competition between the original fear memory and the new extinction memory (7). Projections from the hippocampus to the vmPFC and the amygdala appear to mediate this context-dependent expression of extinction (9,12). During the intentional, cognitive regulation of fear (and negative affect more generally) amygdala activation typically decreases, driven by increased activation of the dorsolateral prefrontal cortex (dlPFC) that, in turn, recruits the vmPFC–amygdala inhibitory pathway that mediates extinction retrieval (6,13,14). In short, the amygdala, the vmPFC, and the hippocampus collectively support the acquisition, storage, retrieval, and contextual modulation of fear acquisition and extinction (5,15).

Although anxiety can be distinguished from fear in a number of important respects (2), several prominent theories propose that dysregulation of the neurocircuitry implicated in the acquisition and modulation of conditioned fear may be critically involved in the etiology and maintenance of anxiety (16–18). Neuroimaging studies suggest that the circuitry involved in the learning and regulation of conditioned fear is systematically altered in trait anxious individuals and clinical populations. Trait anxiety is associated with heightened amygdala activation as well as elevated fear expression during fear acquisition (19,20). Anxiety also impairs extinction learning and retention (19–21) as well as regulation of emotional responses via intentional cognitive strategies (22,23). These deficits appear to stem from impairments in the prefrontal-amygdala circuitry that typically supports the regulation of fear expression. Anxious individuals exhibit reduced prefrontal activation during or before fear extinction (20,24) and require heightened prefrontal recruitment to successfully reduce negative emotion through cognitive reappraisal (25). Anatomical evidence suggests prefrontal inhibition of the amygdala is mediated primarily by a fiber tract from the vmPFC to inhibitory cells within the amygdala (26). Structural integrity of this vmPFC–amygdala pathway is inversely correlated with trait anxiety (27), suggesting that anatomically compromised inhibitory function contributes to heightened reactivity and impaired emotion regulation in anxiety. Finally, atrophy of the hippocampus in clinically anxious patients (28) suggests that contextual modulation of fear may also be altered in anxiety. Consistent with this hypothesis, clinically anxious individuals show increased generalization of conditioned fear to similar stimuli (29).

Additional brain regions may contribute to differences in emotional expression and awareness associated with anxiety. Although the amygdala clearly mediates cue-evoked phasic fear responses to threat-related stimuli, the bed nucleus of the stria terminalis, a region in the ventral basal forebrain referred to as part of the "extended amygdala," appears to support a more sustained state of arousal and vigilance characteristic of anxious individuals (3,30). Anxiety is associated with heightened perception of physiological bodily sensations, or interoception (31), which may increase the aversiveness of responses to threats (32). The insula appears to play a critical role in the representation of interoceptive information (33). Increased interoceptive awareness in anxious individuals appears to be mediated by altered insula reactivity and is thought to contribute to the maintenance of anxiety (31).

The neurocircuitry of fear and anxiety provides a basis for understanding how anxiety may alter decision-making. Neuroeconomic studies of decision-making have highlighted a network of brain regions including the striatum, amygdala, vmPFC, insula, and dlPFC (34) that are also implicated in the expression and control of fear (4–7). Although precisely how these shared networks jointly contribute to anxiety and decision-making is unclear, this overlap suggests the brain systems mediating fear and anxiety are intertwined with those underlying the computation of value and choice.

Cognitive Effects of Anxiety

Although it is not surprising that dysregulation of the fear conditioning neurocircuitry has robust effects on emotional processing in anxious individuals, recent neuroimaging research suggests alterations in cognitive processing typically observed in anxiety may share the same underlying neural substrates (35). A large body of research highlights two principal information-processing biases characteristic of anxiety: 1) a bias to attend toward threat-related information, and 2) a bias toward negative interpretation of ambiguous stimuli (36). Across a variety of tasks, anxiety is associated with a general pattern of faster response times when detecting a threat stimulus or identifying a target cued by a threat stimulus and slower response times when detecting a neutral stimulus or reporting neutral information in the presence of a threat stimulus (23,37,38). This attentional bias appears to reflect both facilitated detection of threat-related stimuli and difficulty in disengaging attention from negative stimuli, relative to neutral or positive stimuli (23).

For stimuli with more than one potential interpretation, anxiety is associated with a tendency toward a more negative perception. For instance, anxious individuals tend to interpret ambiguous emotional facial expressions (39), face–voice pairings (40), and homophones (e.g., "die/dye") (41) as more negative in valence than less-anxious individuals. When evaluating the outcome probabilities of ambiguous future life events, anxious individuals unrealistically judge negative outcomes to be more likely than positive ones (42–44). Studies indicating that patients with anxiety disorders report negative biases in the interpretation of disorder-related stimuli (45–47) suggest that this bias may be selectively applied to self-relevant information.

Biased attention to threat in anxious individuals is proposed to reflect both engagement of pre-attentive amygdala-dependent threat evaluation processes (48) and compromised prefrontal control mechanisms typically engaged during attentional competition and control (49). Consistent with this proposal, high trait anxiety is associated with increased amygdala activity to attended as well as unattended threat stimuli (50–52) and decreased prefrontal activation under conditions of attention competition (49,50), even in the absence of threat-related stimuli (35).

The amygdala and prefrontal cortex (PFC) also appear to contribute to the negative interpretation bias in anxiety. In healthy individuals, the magnitude of the amygdala blood oxygenation level dependent (BOLD) signal to ambiguous surprise facial expressions is positively correlated with the degree to which the expression is interpreted as negative as opposed to positive (53). Higher trait anxiety is associated with heightened amygdala BOLD responses during passive viewing of neutral faces (54) and a tendency to interpret neutral faces more negatively (47). A study in mice reporting greater amygdala responsivity and anxiety-like behavior in the context of temporally unpredictable neutral stimuli suggests the amygdala may play a more general role in mediating an anxiogenic response to ambiguity (55). In contrast, regions of the PFC appear to support intentional efforts to reinterpret negative stimuli more positively (13,56) as well as the automatic effects of positive contextual information on interpretation of ambiguous facial expressions (57).

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Collectively, these data suggest that amygdala hyperresponsivity while attending to, evaluating, and anticipating negative stimuli may heighten the cognitive and affective responses to potential threat in anxious individuals. Furthermore, PFC-dependent cogni-