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.

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, redployment 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...
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 (dPFC) 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 dPFC (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 brain 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-
tive and affective regulatory processes may be impaired in anxiety, reducing the ability to modulate these prepotent tendencies. Here, we review both empirical evidence and theoretical predictions of how these altered cognitive and affective processes in anxiety may influence economic decision-making.

Behavioral economic studies have demonstrated that actual human choice exhibits systematic violations of normative decision-making principles (58). Accounts for such deviations from “rational” choice propose that we employ simplified strategies, or heuristics, that guide our decisions (59). A large body of research suggests these choice heuristics and biases are informed by affective responses to and cognitive interpretations of aspects of the decision context (60–64). In the following, we describe a set of extensively studied decision-making biases involving choice in the face of potential aversive outcomes. Drawing upon behavioral evidence and theoretical models suggesting that anxiety alters decision making (60–64), we propose that the neurocircuitry involved in fear learning and regulation is recruited in these decision-making contexts and that alterations in this circuitry may mediate the influence of anxiety upon choice.

Uncertainty: Risk Aversion and Ambiguity Aversion

Across species, unpredictable stimuli elicit greater anxiety than predictable events (55,65). In a decision context, unpredictability or uncertainty may evoke threat-related information-processing biases and emotional responses in anxious individuals that systematically alter decision-making. In behavioral economics, two forms of uncertainty are distinguished that influence human decision-making in a nonoptimal manner. The first form, risk, refers to a choice in which there are multiple potential outcomes with known or calculable probabilities (66). In such situations, humans tend to be risk averse (67). For example, if given a choice between a certain gain of $50 and a lottery offering a 50% chance of winning $0 and a 50% chance of winning $105, many people would select the certain amount, despite the fact that the uncertain lottery has a higher expected value (the sum of the probability times the amount, for each potential outcome).

The amount of attention paid to an aversive choice option predicts its avoidance (68), suggesting that attentional bias toward potential adverse outcomes of risky gambles may cause anxious individuals to favor certain and safe alternatives. Accordingly, experimental evidence suggests anxiety is associated with heightened risk aversion (69–71). Measures of trait anxiety, worry, and social anxiety in healthy participants are all predictive of heightened risk aversion in the balloon analog risk task, in which subjects accumulate rewards on the basis of the degree of inflation of virtual balloons with variable explosion thresholds (71). Clinically anxious patients show greater risk aversion than control subjects on a scale assessing likelihood of risk-taking behavior (71). A study using an anxiogenic mood-induction paradigm in healthy participants found that heightened anxiety predicted greater risk aversion in both a hypothetical gambling task and a set of hypothetical everyday decision-making scenarios (70). Notably, when asked to make choices for a hypothetical other person, the risk aversion of subjects decreased, suggesting this bias depends upon the self-relevance of the threat posed by a potential risky outcome.

In addition to the cognitive biases shaping the evaluation of risky choices, altered physiological responses to risk may also contribute to risk aversion in anxious individuals. Several influential decision-making models propose that the appraisal of one’s affective response to a decision serves as information that guides the evaluation process (60–64,72). Consistent with this theory, an early study suggested heightened physiological arousal responses to risk foster behavioral avoidance of risk in favor of safer options (73). Thus, either heightened arousal to risky choice options or increased interoceptive awareness of arousal responses (or an interaction of the two) may lead anxious individuals to be more risk averse.

A second form of uncertainty, ambiguity, refers to a decision context in which there are multiple possible outcomes with unknown probabilities (74,75). For example, if faced with a choice of drawing a ball from an urn containing 10 red and 10 black balls or an opaque urn containing 20 mixed red and black balls of unknown proportion, subjects tend to prefer the former, regardless of whether the selection of a red or a black ball would be rewarded. This classic demonstration of ambiguity aversion, or preference for known versus unknown risk, is known as the Ellsberg paradox (75). This tendency poses a problem for normative decision-making models, because the opaque urn either contains a 50%-50% mix of red and black, in which case subjects should have no preference between the urns, or a greater number of black or red balls, in which case one’s preference should switch depending on which color is rewarded. In hypothetical ambiguous scenarios, anxiety is associated with elevated estimates of both the likelihood of occurrence and the subjective cost of negative events (63,76–78), suggesting anxious subjects are biased toward interpreting ambiguous decision contexts negatively.

Although our understanding of the neural systems supporting risk processing remains speculative, several neuroimaging studies examining decision-making under risk highlight contributions of the insula and PFC. Recent functional magnetic resonance imaging (fMRI) research implicates the insula as a key structure involved in the prediction of risk (79). The BOLD activation of the insula is correlated with behavioral measures of risk aversion (80,81), and individuals with insula lesions exhibit choice patterns indicative of impaired risk sensitivity (82). Studies using both fMRI and transcranial magnetic stimulation also suggest a role for the PFC, particularly the dIPFC, in the expression of risk attitudes. Activity in the dIPFC is positively correlated with risk aversion (83), and disruption of the dIPFC with transcranial magnetic stimulation increased risky choices (84), whereas enhancement of the dIPFC decreased risky choice (85). This suggests normative populations may exhibit an orbitofrontal cortex or striatum-mediated predisposition toward risk-seeking (81,86) that is regulated or inhibited by the dIPFC. In anxious individuals, altered insular responses may shift this prepotent tendency toward the avoidance of risk.

Studies examining the neural substrates of ambiguity processing highlight the roles of the amygdala and the PFC (87–91). Although attitudes toward risk and ambiguity are not highly correlated within individuals, these two forms of uncertainty share overlapping neural substrates (91). However, studies explicitly attempting to dissociate risk and ambiguity have distinguished the contributions of different regions. One such study reported higher activation in the amygdala during processing of ambiguous versus risky information (87). A second study reported an inverse correlation between activation in a lateral prefrontal region and ambiguity aversion (88), a finding that stands in apparent contrast with reports of a positive relationship between dIPFC function and risk aversion (83–85). A meta-analysis comparing studies of risky and ambiguous decision-making suggests risk processing is more dependent on activity in orbital prefrontal regions, whereas ambiguity processing preferentially recruits dIPFC (92).

Although risk and ambiguity aversion engage overlapping and distinct neural mechanisms, both engage circuitry that is also implicated in fear and anxiety. The tendency to interpret uncertain situations negatively is a key component of risk and ambiguity aversion.
as well as anxiety. For this reason, it is not surprising that risk and ambiguity may be particularly aversive to anxious individuals.

Framing Effects

Historical decision-making research suggests that willingness to take risk is additionally influenced by whether the certain alternative to a risky choice is stated as a gain or a loss (58). When faced with a sure option of keeping $20 of $50 or a gamble with a 60% chance of losing the entire $50 and a 40% chance of keeping the $50, individuals typically display risk aversion, choosing the certain option (despite their equivalent expected value). However, when faced with a certain loss of $30 versus the identical gamble, subjects are more likely to become risk-seeking and choose the gamble. This phenomenon is referred to as the framing effect.

Trait anxiety is associated with greater susceptibility to the framing effect (93). Consistent with an attentional bias toward loss in anxious individuals, the framing effect is purported to be driven by heightened “loss aversion” or increased sensitivity to loss versus gain (94). Thus, appraisal of the sure option as a decisive loss may evoke a reflexive avoidance response, driving the preference for the gamble, which only entails the potential for loss. However, if the sure option is seen as a gain, this option is preferred over the potential loss associated with the gamble. An examination of the neural substrates of the framing effect revealed that increased BOLD activation in the amygdala and decreased vmPFC activation predicted greater susceptibility to framing (95). As mentioned earlier, this pattern of brain activation also correlates with heightened conditioned fear expression (96) and anxiety-related attentional biases (50), suggesting a common underlying mechanism.

Loss Aversion

Loss aversion refers to the degree to which avoiding losses is more heavily prioritized than attaining equivalent gains (58). Although no studies have examined whether trait anxiety is predictive of individual differences in loss aversion, a recent study reporting that loss aversion correlates with the electrophysiological response to loss outcomes relative to gains (97) suggests heightened physiological responses to potential loss could contribute to loss aversion in anxious individuals. A study demonstrating decreased loss aversion in patients with amygdala lesions (98) as well as an fMRI study showing a correlation between amygdala activity at choice and degree of loss aversion (99) suggest a role for the amygdala in generating this increased sensitivity to loss. Because anxiety is also associated with heightened amygdala responses, one may expect greater loss aversion with increased anxiety. Neuroimaging data also implicate the striatum and lateral prefrontal regions in loss-averse choices (100).

The weighting of losses versus gains in decision-making may in part reflect how one interprets the impact of the loss. A recent study exploring the effect of perspective shifts on loss aversion found that changing one’s perspective in a series of choices to decrease the impact of each potential loss also decreased loss aversion as well as the arousal response to losses relative to gains (97). Examination of the neural systems mediating this effect implicates a circuit commonly observed in studies of cognitive fear regulation (6,13). In short, amygdala activation to losses decreases when a perspective shift decreases loss aversion. This cognitive perspective shift is accompanied by increased activation in the dIPFC, vmPFC, and striatum. Impairments in the ability of anxious individuals to use cognitive strategies to regulate emotions (22,23), perhaps due to difficulty engaging the underlying circuitry (25), may also hinder their use of perspective shifts as a means to decrease the impact of losses on decision-making.

Conclusions

The everyday decisions made by individuals suffering from anxiety disorders to avoid perceived threats can have a profound impact on their ability to function adaptively. However, relatively little is known about how anxiety influences specific decision variables. The extant literature explicitly examining the role of anxiety in decision-making is limited. In this review, we have attempted to explore this topic by examining the overlap in the neural systems mediating anxiety and decision-making and the potential impact of the documented cognitive biases associated with anxiety on decision tasks.

Across a range of neuroeconomic decision-making tasks, particularly those involving uncertainty or potential loss, a circuitry involving the amygdala, insular cortex, and PFC has been observed. The amygdala is a key component of the brain systems mediating fear and anxiety and their cognitive effects. At the same time, the PFC is critically involved in the control of fear, and decreased prefrontal engagement is observed in trait and clinical anxiety. We propose that this shared architecture may yield predictable effects of anxiety on decision-making. Specifically, anxiety increases the attention to negative choice options, the likelihood that ambiguous options will be interpreted negatively, and the tendency to avoid potential negative outcomes, even at the cost of missing potential gains. Importantly, the neural systems enabling one to alter these maladaptive decision processes may be more difficult to engage with high anxiety.

Behavioral economics often describes decision tendencies, such as risk or loss aversion, as if they were immutable individual characteristics. In this review, we suggest that such tendencies are influenced by trait anxiety, which is also thought to be relatively stable (1). However, recent research has focused on how individuals can alter fear or anxiety responses in specific circumstances. These studies (see 6 for a review) demonstrate that fear can be altered through a variety of techniques, including the formation of new extinction memories, the use of intentional cognitive regulation strategies, performing actions that limit exposure to fear-related stimuli, or through pharmacological or behavioral disruption of learned fear associations. This research on the control of fear suggests that modulation of anticipatory responses to potential aversive outcomes in a decision-making context may similarly enable flexibility in individual choice tendencies. In this review, we have discussed the influence of anxiety upon choice as though the response of anxious individuals to negative decision options is relatively consistent. However, this is almost certainly an overly simplistic perspective. Future neuroeconomic research examining how the regulation of anxiety influences our decisions will no doubt reveal a relationship between anxiety and choice that is both more malleable and more complex than our present understanding.

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