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A return to the psychiatric dark ages with a two-system framework for fear

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ABSTRACT

The past several decades has seen considerable progress in our understanding of the neurobiology of fear and anxiety. These advancements were spurred on by envisioning fear as emerging from the coordinated activation of brain and behavioral systems that evolved for the purpose of defense from environmental dangers. Recently, Joseph LeDoux, a previous proponent of this view, published a series of papers in which he challenges the value of this approach. As an alternative, he and colleagues propose that a 'two-system' framework for the study of responses to threat will expedite the advancement of medical treatments for fear disorders. This view suggests one system for autonomic and behavioral responses and a second for the subjective feeling of fear. They argue that these two systems operate orthogonally and thus inferences concerning the emotion of fear cannot be gleaned from physiological and behavioral measures; confounding these systems has impeded the mechanistic understanding and treatment of fear disorders. Counter to the claim that this view will advance scientific progress, it carries the frightening implication that we ought to reduce the study of fear to subjective report. Here, we outline why we believe that fear is best considered an integrated autonomic, behavioral, and cognitive-emotional response to danger emerging from a central fear generator whose evolutionarily conserved function is that of defense. Furthermore, we argue that although components of the fear response can be independently modulated and studied, common upstream brain regions dictate their genesis, and therefore inferences about a central fear state can be garnered from measures of each.

1. Introduction

Across phylogeny, organisms display characteristic responses to danger, allowing them to avoid predation and other dangers in their environment (Bolles, 1970; Fanselow & Lester, 1988). These responses entail both internal physiological changes including increased heart rate and respiration, and external behaviors such as fight and flight responses (Davis, 1992; Fendt & Fanselow, 1999; Perusini & Fanselow, 2015).

The mental health field has placed great interest in responses to danger (also referred to as defensive behavior) in an effort to understand fear and anxiety disorders, often conceptualized as the body's defensive response exceeding its adaptive function. Owing largely to the relative ease with which behavioral and physiological responses to threat can be evoked in model organisms, as well as the quantitative manner in which they can be measured, we now know a great deal about defensive circuits in the brain (Davis, 1992; Duvarci & Pare, 2014; Fanselow & LeDoux, 1999; Johansen, Cain, Ostroff, & LeDoux, 2011; Paré, Quirk, & Ledoux, 2004). This research has already provided us with the ability to predict the efficacy of therapeutic drugs, from benzodiazepines for the reduction of fear and anxiety (Fanselow & Helmstetter, 1988; File & Pellow, 1985; Hart, Sarter, & Berntson, 1998) to D-cycloserine for the augmentation of exposure therapy (Bouton, Vurbic, & Woods, 2008; Bowers & Ressler, 2015; Mataix-Cols et al., 2017; Woods & Bouton, 2006). In addition, studies of the ontology of defensive responses have provided us with information relevant to behavioral therapies; for example, understanding why exposure therapy is liable not to transfer beyond the therapist's office (Bouton, 2002, 2004; Bouton, Westbrook, Corcoran, & Maren, 2006).

In several recent and widely publicized papers, LeDoux and colleagues call into question the utility of using autonomic and behavioral responses to danger to make inferences about the associated subjective emotional states of fear and anxiety (LeDoux & Pine, 2016). They argue that autonomic and behavioral responses to threat are orthogonal to the subjective experience of fear (Fig. 1A). Therefore, the terms fear and anxiety should only be used in reference to subjective mental experience, and should be studied accordingly. They propose that the failure to distinguish the systems supporting fear and anxiety from those giving rise to the autonomic and behavioral responses to threat – their 'two-system framework' – is one of the reasons that 'progress has stalled in

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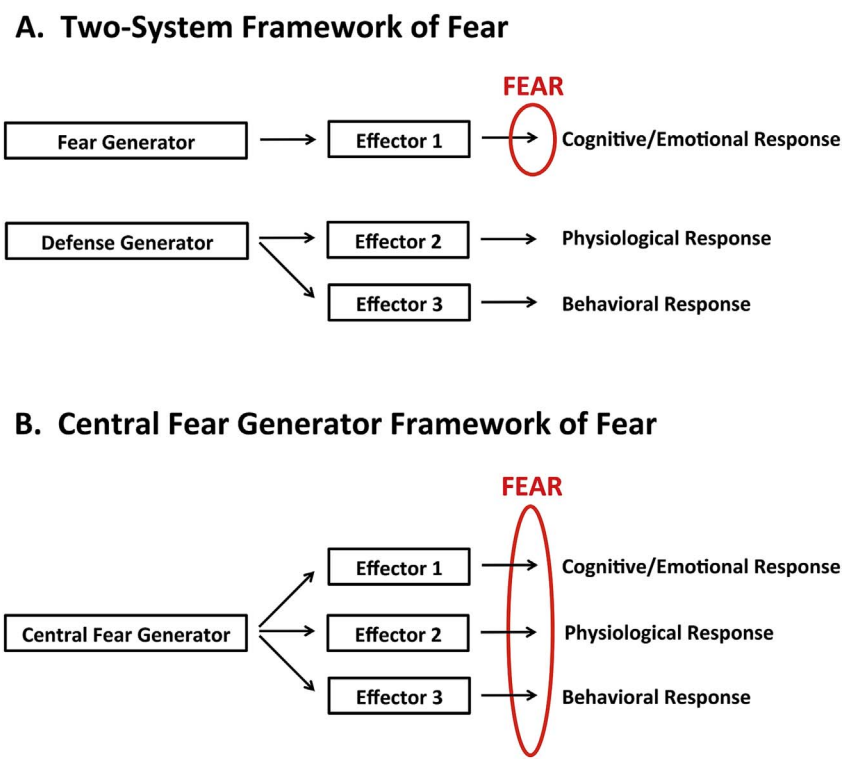

Fig. 1. Two opposing models for fear. A) The two-system framework proposed by LeDoux and Pine posits that the emotional experience of fear emerges from a distinct neuronal circuit than that which is responsible for the physiological and behavioral responses to threat. The term fear only refers to the subjective, cognitive/emotional experience in response to threat. B) The central fear generator framework advances that the various responses to threat (cognitive/emotional, physiological, and behavioral) emerge from a central neuronal circuit. Here, the term fear represents an integrated response.

treatment development for mental disorders' (LeDoux & Brown, 2017; LeDoux & Pine, 2016; LeDoux, 2017).

Here we contend with this view, and argue that the autonomic, behavioral, and cognitive-emotional responses to danger are best conceptualized as the unitary result of activation of a central fear generator (i.e. one-system).

2. The damage of a two-system framework

Before beginning, it is paramount to state that we are not writing this response only because we believe the two-system framework is theoretically troubled. Rather, we were compelled to do so because such a view has the potential to wreak havoc on progress in the field of mental health. Already the National Institute of Mental Health has broadcast one of these papers (NIMH., 2016), suggesting it has the potential to influence policy. Here are some notable problems:

First, if the subjective emotion of fear is orthogonal to its autonomic and behavioral counterparts, then all use of non-human animals to advance translation to the clinic in the study of fear can essentially be thrown out the window. Animals cannot tell us of their subjective emotional responses, and therefore they should not be used to study fear as an emotional experience. Sure, they can be used to study the physiological and behavioral responses to danger, but according to the two-system framework, this is moot, as an understanding of these responses would do little to lessen the subjective distress of patients. Any insights gained by the ability to probe specific neural circuits and test the efficacy of medications, as well as the environmental control that animal studies provide us with, would be gone.

Second, the inability to use physiological and behavioral measures to study fear does not merely apply to non-human animals. This must also hold true in humans, because across species the two-system framework holds that these measures do not predict the subjective experience of fear. Thus, experimental work examining behavioral and physiological responses in humans to assess fear would similarly need to be discarded.

Without physiological and behavioral indices of fear in human and non-human animals, we are left to study subjective responses. Of course, the reason the field moved away from subjective report is no mystery: they are often difficult to reliably quantify and subject to diverse response biases that can variably over-/under-estimate the subjective experience of fear. The demand characteristics of the situation may also influence self-report: for example, fear may be under-reported by a dedicated soldier and over-reported by someone wishing to persuade a physician to prescribe medications. Moreover, subjective report can only be captured from individuals capable of using language to communicate their subjective experience (because again, behavioral indicators are not reliable). This poses serious issues, as the study of emotional experience in young children, or adults with language disabilities, would be beyond scientific reach.

However inconvenient, if the two-system framework were correct, these would be the ramifications. Thankfully, we believe that there is little evidence that supports the two-system framework. Indeed, the vast preponderance of the literature, even that reviewed by LeDoux and and colleagues, clearly favors the central state view. In addition, upon scrutiny of the two-process framework we believe that it actually suggests that the subcortical circuits supporting defense are the unique and paramount circuits in driving fear.

3. The argument for a central fear generator

Not unlike previous models (Davis, 1992; Fendt & Fanselow, 1999; Johansen et al., 2011), we propose that fear is a coordinated reaction to danger involving autonomic, behavioral and cognitive responses emerging from a central fear generator. This central fear generator then recruits downstream effectors that control a restricted range of the response (Fig. 1B).

Traditionally, it has been assumed that the central generator of fear is the amygdala, because damage to the amygdala is able to gravely impact a multitude of defensive behaviors, and because plasticity within the amygdala is essential for fear learning to occur (Davis, 1992; Fanselow & LeDoux, 1999; Fendt & Fanselow, 1999; Maren, 2003, 2005; Rumpel, LeDoux, Zador, & Malinow, 2005). We largely agree with this assumption and the following discussion will focus heavily on evidence concerning the amygdala. Nevertheless, it is important to

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