Chapter 14

Neuroimaging of Emotion Dysregulation

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Abstract

Affective neuroscience, the study of neural mechanisms that give rise to emotional experiences in humans and animals, has a short but rich history. Almost three decades old, affective neuroscience has predominantly taken two theoretical approaches to understanding the brain bases of human emotions, and thus, two stances on the brain bases of emotion dysregulation. One approach, the traditional approach, argues that specific emotions are hardwired in human biology with specific neural underpinnings or signatures for said emotions. The second approach, a psychological constructionist approach, argues that each experienced emotion emerges not from a specific, dedicated anatomical circuit, but from an interplay of broad networks in the brain that are involved in general operations of the mind. In this chapter, we overview these two theoretical approaches with a specific focus on functional magnetic resonance imaging (fMRI) findings. We conclude with evidence suggesting how emotion dysregulation may arise, and link this work to clinical fMRI investigations of anxiety disorders. We close by suggesting future directions affective neuroscience may take to better understand processes underlying dysregulated emotions.

Introduction

Emotion dysregulation is a core feature of almost every major form of psychopathology across the lifespan (Beauchaine, 2015; Insel, 2014; Kring, 2008; Kring & Mote, 2016). It underlies maladaptive decision-making (Lee, 2013; Sharp, Monterosso, & Montague, 2012) and maladaptive interpersonal behaviors (Kring, 2008), and is often a source of distress for individuals who experience it (Bylsma, Taylor-Clift, & Rottenberg, 2011; Kashdan & Steger, 2006; Myin-Germeys et al., 2009). Understanding etiopathophysiologies of emotion dysregulation would therefore provide insight into mechanisms underlying myriad maladies.

For the past 25 or so years, human neuroimaging studies in the field of *affective neuroscience* have evaluated the functional neuroanatomical bases of emotions in attempts to identify processes that underlie emotion dysregulation. Much of this research follows the traditional model of emotion, which assumes largely discrete anatomical bases of particular emotion categories (e.g., fear), with the assumption that functional abnormalities of these anatomical structures (e.g., excessive amygdala activation) result in emotional dysregulation (e.g., anxiety). In this chapter, we discuss evidence for and against the traditional model, and offer a new approach—the theory of constructed emotion (TCE; Barrett, 2017a; Barrett, 2017b; Lindquist, 2013). In contrast to traditional approaches, the TCE suggests that each experienced emotion emerges not from a specific, dedicated anatomical circuit, but from an interplay of broad networks in the brain that are involved in general operations of the mind. Instances of each emotion category (i.e., fear, anger, happiness, and so forth) are represented by a pattern within these networks that is situation-specific and individually different. Taking a *constructionist* approach, we discuss evidence of how dysregulation within these networks may result in dysregulation of emotions.

We begin our chapter by describing terms and concepts. Next, we outline the two theoretical

approaches that have guided neuroimaging research on emotion for the past 25 years: the traditional, anatomically-given circuit-based view and the TCE. With these theoretical perspectives in mind, we discuss what affective neuroscience suggests about mechanisms underlying human emotion and its dysregulation. We conclude with evidence suggesting how emotion dysregulation may arise, and link this work to clinical investigations of anxiety disorders. Although we recognize there are myriad ways that emotion dysregulation affects well-being, behavior, and psychopathology, we focus specifically on these disorders for illustrative purposes. We close by suggesting future directions that affective neuroscience may take to better understand processes underlying dysregulated emotions, conceptually linking the constructionist approach to other recent attempts to identify basic processes that underlie multiple disorders (e.g., the Research Domain Criteria; RDoC approach; Insel, 2014).

Terms and Concepts

This chapter is about neuroimaging of emotion dysregulation, but we should first clarify how we use the terms neuroimaging, emotion, and dysregulation. In general, neuroimaging refers to any technology that allows researchers to create images of neural structure or function. In this chapter, we review the literature on human *functional neuroimaging*—technologies that generate images of neural processes related to mental states among live, waking human subjects. We focus specifically on functional magnetic resonance imaging (fMRI), which assesses changes in blood oxygenation to estimate blood flow to specific regions of the brain while humans experience mental states (e.g., fear). Blood flow reflects corresponding changes in activity (both excitation and inhibition) to neural populations in brain tissue (see Logothetis, 2008). Although there are other methods of functional neuroimaging (e.g., positron emission tomography [PET], electroencephalography [EEG], magnetoencephalography [MEG], functional near-infrared spectroscopy [fNIRS]), we focus on fMRI for two reasons. First, most recent neuroimaging

studies of human emotion use fMRI because it does not require injection of radioactive tracers (like PET) and affords good localization to subcortical and other structures (unlike EEG, MEG and fNIRS). Subcortical structures play a key role in most theories of emotion (e.g., Damasio et al., 2000; MacLean, 1949; Panksepp, 2016; Panksepp & Watt, 2011; cf. Kober, Barrett, Joseph, Bliss-Moreau, Lindquist, & Wager, 2008) and fMRI can image these deep brain structures—often with high spatial resolution (Satpute et al., 2013). In contrast, EEG, MEG, and fNIRS offer exceptional temporal resolution and are therefore better-suited for answering questions about "when" activation occurs during the experience of emotion. Temporal dynamics of neural processing are surely of interest to emotion researchers, and more research is needed in this area (see e.g., Heller & Casey, 2016; Lee, Lindquist, & Nam, 2017; Waugh, Hamilton, & Gotlib, 2010). As we note below, some recent evidence on network-properties of brain activation using fMRI combines spatial and temporal domains by examining sequences of regional brain activation during emotions.

Next, we define what we mean by "emotion," as the term is used differently among psychologists/neuroscientists, clinicians, and practitioners—and even among affective neuroscientists. We differentiate between what we call "emotions" and "affect." Whereas "emotions" describe discrete experiences of specific states, as named with words such as "sadness," "fear," and "anger," (often called "discrete emotions"; Keltner, Ekman, Gonzaga, & Beer, 2003), "affect" is used to describe more general feelings that range in positivity-negativity and activation-deactivation (see Barrett & Bliss-Moreau, 2009; Barrett, 2016; Lindquist et al., 2013; Russell, 2003). We consider affect as constitutive of emotions insofar as affect is a basic "ingredient" that underlies all emotional experience (i.e., all emotions can be described as having some degree of pleasantness-unpleasantness and activation-deactivation; Barrett & Bliss-Moreau, 2009; Lindquist, 2013; Lindquist et al., 2012; Lindquist et al., 2016; Russell, 2003).

Finally, we describe our definition of emotion dysregulation, which is largely but not fully consistent with the definition used throughout this volume (i.e., patterns of emotional experience and/or expression that interfere with goal-directed behavior; see Beauchaine, 2015). By some definitions, dysregulation is the opposite of regulation. Yet regulation is used differently throughout the emotion and affective neuroscience literature. In affective neuroscience, research on regulation often refers to explicit attempts by an individual to control or change his or her emotions (Gross & Thompson, 2007). There is a large body of imaging research on explicit regulation of emotion (see Ochsner & Gross, 2008; Buhle et al., 2014). This research focuses primarily on how effortful, "cognitive" control processes associated with prefrontal cortex (PFC) function can down-regulate functional activity of subcortical structures such as the amygdala, which—along with other subcortical structures—are canonically associated with emotion (e.g., Adolphs, 1999; Panksepp, 2016; Phelps & LeDoux, 2005). This literature is important, and is linked closely with emotion dysregulation in psychopathology across the lifespan (e.g., Beauchaine, 2015; Berking & Wupperman, 2012; Martin & Dahlen, 2005) and to other relational and social outcomes (e.g., Gross & John, 2003).

We use the term regulation in its broader sense to refer to normative emotional processes. We believe that before one studies explicit emotion regulation or impairments in related processes, it is important to understand neural processes that contribute normatively to emotions in healthy individuals across daily life[1]. We therefore spend most of this chapter discussing processes associated with regulated emotion, and use this as a jumping off point for exploring how those same processes may become dysregulated in anxiety disorders. Of course, emotion dysregulation is likely to be transdiagnostic across other disorders (Buchaine, 2015a, b; Buchaine & Zisner,

[1] We note that these processes change developmentally and that there is important variation across the lifespan. We focus herein on research from young, healthy adults with the caveat that there may be important differences in children and adolescents, and in middle and older age.

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Theoretical Perspectives: Competing Theories of Brain Bases of Human Emotions

Theories are much more than sets of explanations for how emotions operate—they are philosophical lenses through which observations are made, hypotheses are generated, studies are designed, and data are interpreted (Kuhn, 1977). At only 2-1/2 decades old, affective neuroscience is still a young discipline, and is still reliant on assumptions made since its inception (see also Kuhn, 1961). Thus, we begin by discussing traditional approaches to understanding brain bases of human emotion, outlining how these approaches began the study of emotion dysregulation among adults (and by extension, children). We then describe the more recent theory of constructed emotion (Barrett, 2017a; Barrett, 2017b; Lindquist, 2013), including its hypotheses regarding how emotion and dysregulation emerge.

The Traditional Approach

The traditional approach to understanding emotion has a long history in psychology, medicine, and neuroscience (see Barrett, 2017a; Barrett & Satpute, in press; Gendron & Barrett, 2009) and derives in part from the common assumption that our experiences of the world reveal underlying mechanisms (Ward, Ross, Reed, Turiel, & Brown, 1997). In the case of our emotions, since fear and sadness are experienced as distinct, we are likely to assume that these categories of emotion derive from different psychological and neural mechanisms. This inference—the idea that categories are distinct and the idea that they each have their own mechanism—is rooted in essentialism. Essentialism is the human tendency to ascribe dedicated causal mechanisms to "categories" of observable phenomena—in this case different emotions (see Gelman, 2009a; Gelman, 2009b). This translates into the belief that fear and sadness, for instance, must be associated with different feelings and behaviors and each derive from discrete mechanisms in the brain (see Lindquist et al., 2013).

In affective neuroscience, the traditional view is codified in basic emotion theory, which argues that emotions such as sadness, fear, and anger are categories of experience that exist across species as evolved mechanisms, are present at birth in humans, and are universal across cultures (see Barrett, 2006; Ekman & Cordaro, 2011; Panksepp & Watt, 2011; Tracy & Randles, 2011). The basic emotion perspective argues that at least some categories of emotional experience (fear, sadness, anger, happiness, disgust) evolved to motivate responses to survival-linked contexts throughout phylogeny (Ekman & Cordaro, 2011; Panksepp & Watt, 2011). These basic emotions are thought by some to constitute foundations for all human emotional experiences (including more complex "secondary emotions"), and cannot be broken down into constituent parts (Ekman & Cordaro, 2011; Panksepp, 1982; Panksepp & Watt, 2011).

Neural bases of emotions. Traditional theories of emotion sometimes suggest that each basic emotion category corresponds to a specific, inherited, and anatomically-dictated mechanism within the central nervous system (see Tracy & Randles, 2011). Over the years, this idea has taken multiple forms that manifest in the literature on emotion regulation and dysregulation.

Maclean (1949) famously proposed the "triune brain" concept, which is rooted in essentialist beliefs about emotion vs. reason that still shape—however implicitly—theory and interpretation of neural data. The triune brain concept localizes emotion categories, such as fear, to limbic and brainstem structures deep within subcortical regions (e.g., amygdala, striatum), and separates these processes from cognitive processes involved in cognition, planning, and self-regulation (functions associated with areas of the PFC). At the base of this three-part system lies the "reptilian" brain that generates "primitive" emotions (e.g., anger, fear). Above this system is the "visceral brain" that elaborates the "social" emotions (e.g., "contempt," "embarrassment," etc.). Finally, the neocortex, the top-most system, is typically the basis for cognitive functions and

attributed to regulatory processes.

Despite the legacy of the triune brain concept, research on brain evolution suggests that the triune brain concept is at best a heuristic; among mammals, there is no "limbic system" that is functionally separate from other parts of the brain (see Chanes & Barrett, 2016; Pessoa, 2008). Moreover, the mammalian brain did not develop in a linear, one-region-above-the-next fashion. Instead, as the human brain evolved, it fundamentally reorganized, with new connections between subcortical nuclei, limbic, paralimbic, and cortical tissue created across primate evolution (see Barbas, 1995; Striedter, 2005). Although debate on brain evolution is far from closed, most experts agree that the triune brain concept does not accurately describe functional neuroanatomy.

Nonetheless, the triune brain concept, in combination with basic emotion models (e.g., Ekman, 1992; Izard, 1992), facilitated initial hypotheses about emotion-specific subcortical brain structures within the subcortex and limbic tissue (Panksepp, 1998). It is beyond the scope of this chapter to review each of the subcortical, limbic, and paralimbic regions or circuits that have been linked to specific basic emotions (see Barrett et al., 2007, Lindquist et al., 2012, and Panksepp, 1998, 2016 for reviews). Instead we focus attention on the amygdala-fear link, which is the best-known and perhaps most historically influential hypothesis about the neural circuitry of emotion.

The amygdala was considered an essential part of a dedicated neural circuit involved in perception and experience of fear following early animal research demonstrating its role in neophobia (Klüver & Bucy, 1937), aversive conditioning ("fear learning"; Davidson & Irwin, 1999; LeDoux, Cicchetti, Xagoraris, & Romanski, 1990), extinction (Falls, Miserendino, & Davis, 1992) and freezing behavior (e.g., Blanchard & Blanchard, 1972). Together, these led early researchers to conclude that the amygdala is the brain locus of fear (LeDoux, 1995) or a

key part of an encapsulated, anatomically-defined subcortical circuit for this emotion (Davis, 1992; Fanselow, 1994; Panksepp, 1998; Tovote, Fadok & Lüthi, 2015).

The most compelling studies linking fear and amygdala activation are those in which amygdala lesions in animals, such as rats, abolish freezing behavior (e.g., Blanchard & Blanchard, 1972), a behavior commonly considered a fearful or defensive response. There are two concerns with this interpretation, one philosophical and the other empirical. First, linking adaptive behaviors in nonhuman animals with complex emotional experiences in humans can be problematic (LeDoux, 2012; 2013) as it cannot be verified whether non-human animal emotional behaviors are subjectively similar to conscious human emotions (see Barrett, 2017c for a discussion). More importantly, it is now empirically clear that the amygdala is not specific to defensive responses (e.g., Paré & Quirk, 2017). Rather, the amygdala is involved more generally in behavioral engagement "that governs transactions between mammals and their environments: whether or not to engage with (or disengage from) stimuli or situations" (Paré & Quirk, 2017, p. 6). That is, amygdala activation occurs during behavioral outputs to motivationally relevant stimuli (Amir, Lee, Headley, Herzallah, & Paré, 2015). This interpretation explains why the amygdala is also active during non-fear states such as reward (Baxter & Murray, 2002) and when dictating behavioral responses to non-aversive, yet uncertain stimuli (Herry et al., 2007).

Research with humans has also been invoked in ascribing the amygdala as the brain basis of fear. In humans, amygdala damage produces disruptions to both aversive conditioning (LaBar, LeDoux, Spencer, & Phelps, 1995), the recognition of fearful expressions (Adolphs, Tranel, Damasio, & Damasio, 1995; Adolphs, Damasio, Tranel & Damasio, 1996), and to some experiences of fear (e.g., in a haunted house; Feinstein, Adolphs, Damasio, & Tranel, 2011). However, more recent findings suggest these links are not consistent or specific enough to suggest that the amygdala is the brain basis of fear. For instance, the amygdala is not involved

consistently in “fear conditioning” and extinction across human neuroimaging studies; roughly half of studies reviewed failed to report amygdala activation (Sehlmeyer et al., 2009), suggesting a more complex role for the amygdala in learning about aversive stimuli than previously assumed. In the case of perceiving fearful facial expressions, complete amygdala lesions do not abolish accurate perceptions when patients are given the added instruction to look at the eyes of fearful faces (Adolphs et al., 2005). In addition, there are significant increases in amygdala activation following presentation of isolated fearful faces, but not when those faces are shown with complete bodies, which should presumably be an even clearer fearful signal (Poyo Solonas et al., 2017). Finally, patients with bilateral amygdala lesions are able to experience fear when deprived of oxygen, suggesting that all instances of fearful experience are not abolished by amygdala lesions (Feinstein et al., 2013). A meta-analysis of neuroimaging experiments in healthy humans identifies amygdala activation in roughly 30% of data points that induce fearful experiences (Lindquist et al., 2012), so it is not activated consistently during fearful experiences in humans. Rather, the amygdala is most consistently activated in fear inductions involving external (visual, auditory) stimuli as opposed to internally-generated states of fear (Lindquist et al. 2012). Nor is the amygdala specific to fear—it activates during other emotions (Lindquist et al., 2012) and during attention to motivationally relevant stimuli more generally (Cunningham & Brosch, 2012). Like the non-human animal findings (e.g., Paré & Quirk, 2017), these findings suggest that the amygdala is playing a more general psychological function and is not specific to the emotion category fear.

Finally, the traditional approach to emotion can be observed in recent neuroimaging findings that rely on multivariate pattern-based analyses (MVPA) based in machine learning to examine whether a pattern of brain activity across the whole brain can “diagnose” or serve as a “biomarker” of which emotion a person is experiencing (e.g., Kassam et al. 2013, Kragel &

LaBar, 2015; Saarimäki et al. 2015). These approaches move beyond simple 1:1 relations between specific brain regions and emotion categories to argue that neural circuits for emotion exist in complex patterns in the brain. It is easy to assume that identifying a pattern for a specific category reveals the neuroanatomical basis of that category, yet there are multiple problems with this interpretation (see Clark-Polner, Johnson, & Barrett, 2016). The first problem is statistical. A pattern that successfully distinguishes the members of one category from the members of another (at a significant level) is not a "signature" or "biomarker" but is instead a statistical summary of a sample of instances (instances of fear induced in one experiment) drawn from a greater population of instances (other instances of fear across other experiments, people, and time). Importantly, the patterns found across instances for, say, fear, do not replicate one another, meaning that there is not a stable "biomarker" for this emotion category (Clark-Polner et al. 2016). A second point is logical; MVPA analyses can differentiate the brain's representation of semantic categories such as "athletes" vs. "buildings" (Huth et al., 2012), but humans are less likely to essentialize the clearly socially constructed category of "athletes" than the body-based category of "emotions" (see Lindquist et al., 2013) and so scientists do not conclude that they have found "biomarkers" for categories such as "athletes." Finally, MVPA studies identify a functional pattern but do not say whether those brain regions are working together as a distinct circuit, or even whether those brain regions have functional connections to one another. It would be more compelling to identify networks of regions that have known anatomical connectivity and are consistently and specifically associated with the experience of specific emotions.

We (Touroutoglou et al., 2015) attempted to identify whether brain regions that showed consistent functional activation for specific emotions (from the Vytal & Hamann, 2010 meta-analysis) were each part of their own, emotion-specific anatomical network, or whether they formed broader networks that were not specific to emotions. We used resting state functional

connectivity, which reveals chronic functional connections between brain regions known to be undergirded by anatomical connections (i.e., white matter tracts measured through diffusion tractography in humans or in retrograde tracer injections in non-human primate brains) (Deco et al., 2011; Hermundstad et al., 2013; Pernice et al., 2011; van den Heuvel et al., 2009). Using brain regions that consistently activate for specific discrete emotions as seeds in resting state networks, we failed to reveal evidence for emotion-specific, anatomically-defined brain networks. For instance, a region of the left amygdala that was consistently associated with fear in the meta-analytic summary (Vytal & Hamann, 2010) did not form a fear-specific network. Rather, it was functionally connected to areas that form part of a broader "salience network" (cf., Seeley et al., 2007) identified across species (Touroutoglou et al., 2016). Activation within this salience network is generally associated with aversive states (e.g., Hayes & Northoff, 2011; Lindquist et al., 2016), attention (Menon & Uddin, 2010) and behavioral avoidance (Menon, 2011). Critically, we found that the regions that showed consistent activation across multiple negative emotions (fear, anger, disgust and sadness) were part of this anatomically-constrained salience network (Touroutoglou et al., 2015), underscoring the hypothesis that the salience network contributes to multiple types of emotional experiences.

In sum, the literature increasingly suggests that specific emotion categories do not map consistently and specifically to certain anatomical circuity. This finding is convergently revealed using neuroimaging in humans (for meta-analyses; Kober et al. 2008; Lindquist et al. 2012; Touroutoglou et al. 2015; Vytal & Hamann, 2010; Wager et al. 2015) and also in studies that use causal methods in both humans (e.g., using electrical stimulation; Guillory & Bjarski, 2014) and in non-human animals (see Barrett et al. 2007). Furthermore, and as mentioned previously, studies in non-human animals reveal anatomical circuitry supporting certain adaptive behaviors (for a review see Panksepp, 2004), but the interpretation that these circuits are the circuits for

complex categories such as human fear, disgust, anger, etc. is problematic (see Barrett et al. 2007; LeDoux, 2012; 2013). For instance, there are elegantly worked out circuits for escape (Vazdarjanova & McGaugh, 1998), freezing (LeDoux, 2007), fighting (e.g., offensive attack; Lin et al., 2011, defensive aggression; Motta et al., 2009) but the neural circuit for a behavior is not the neural circuit for an emotion per se (e.g., Barrett, 2012; Barrett, et al., 2007; LeDoux, 2012). A problem with this logic is that an animal might perform multiple behaviors when faced with a potential threat (i.e., a "fearful" situation): it might flee, freeze, or fight. This introduces the problem of having many fear circuits (e.g., Gross & Canteras, 2012) and poses an inductive problem for the science of emotion and psychopathology since it is unclear which fear circuit is the correct one to study when examining psychopathology. This said, it is likely that circuits for these adaptive behaviors form a more basic "element" in emotional experiences by combining with other more basic psychological "elements." This idea is consistent with the psychological constructionist account we discuss next.

Limitations with the traditional view aside, investigations of emotion dysregulation typically hew to these traditional assumptions about the brain basis of emotions by assuming that dysregulation derives from dysregulation of an emotion-specific brain area or circuit. For instance, much research focuses on increased reactivity within subcortical structures (e.g., the amygdala) or failures of the cortex to regulate such structures as the basis of emotion dysregulation (see Etkin & Wager, 2007). Other studies explicitly focus on dysregulation of a "fear circuit." Here we again focus our attention on the fear-amygdala link, which has led to over 150 publications examining the role of the amygdala in fear and anxiety. We are not arguing that the amygdala is uninvolved in the neural etiology of anxiety disorders; rather, we suggest that it is far from clear that amygdala dysregulation represents dysregulation in a "fear circuit" as such. Rather, the amygdala may be playing a more basic role that is general to multiple emotion

categories and is transdiagnostic across disorders, a point we delve into later.

Following the traditional model, emotion dysregulation arises in part from dysfunction within the "limbic system," including structures such as the amygdala in anxiety and from the failure of the cortex to regulate such structures. For instance, heightened amygdala activation is observed across patients with social anxiety disorder (Birbaumer et al., 1998; Furmark et al., 2004; Phan, Fitzgerald, Nathan & Tancer, 2006; Stein et al., 2002) and post-traumatic stress disorder[2] (PTSD; Rauch et al., 2000; Shin et al., 2006). Interestingly, heightened amygdala activation to negative stimuli is also observed in anxiety-prone individuals who are otherwise considered healthy (Stein, Simmons, Feinstein & Paulus, 2007). Consistent with the role of PFC in regulating the amygdala, diminished amygdala activity during emotion regulation tasks is associated with increased cortical thickness and greater white matter connectivity within prefrontal cortical areas (Foland-Ross et. al, 2010; Kim et al., 2011), suggesting that structural differences in the PFC predict reduced amygdala activity. In pathological anxiety, patients show reduced functional connectivity between the amygdala and frontocortical areas, such as the orbitofrontal cortex (Hahn et al., 2011). Patients with PTSD show a diminished ability to extinguish conditioned fear as compared to healthy controls, which is associated with weak recruitment of prefrontal cortical areas and reduced amygdala inhibition (Milad et al., 2009; Kolassa et al., 2007) and connectivity between the PFC and amygdala improves following successful treatment (see Clark & Beck, 2010 for a review).

Together, these findings suggest that subcortical and cortical structures, or some interplay between them, is important to regulated emotions. However, recent work suggests that the traditional approach may provide too narrow a lens for hypothesis testing and interpretation

[2] We recognize that the latest Diagnostic and Statistical Manual (DSM-5) no longer classifies PTSD as an anxiety disorder but instead as a "trauma- and stressor-related disorder." However, given the previous scientific conceptualizations of PTSD as an anxiety-related disorder and evidence for some shared neural circuitry between PTSD and anxiety (e.g., Etkin & Wager, 2007), we discuss PTSD throughout our chapter.

when it comes to understanding processes underlying emotional dysregulation. A recent meta-analysis (Sprooten et al., 2017) of 537 studies of case-control clinical examinations of mental disorders (including anxiety disorders, schizophrenia, bipolar disorder, major depressive disorder, and obsessive compulsive disorder) comprising observations derived from 21,427 participants calls the central role of the amygdala in anxiety disorders into question. The meta-analysis found some evidence for greater amygdala activation in anxiety disorders, but only when those studies that used region-of-interest (ROI) analyses were considered in the analysis. Regions of interest (ROIs) are a priori locations in the brain that are queried for activation during analysis, oftentimes using more lenient statistics. This is one manner by which traditional notions about brain function can influence the literature: Traditional assumptions about emotion-brain linkages cause researchers to look to the amygdala for what would otherwise be subthreshold activation, and when such activation is found, it is concluded that amygdala dysfunction is central to a disorder. However, when meta-analyzing studies that performed whole-brain analyses without an a priori focus and using stricter statistical thresholds, there was not a strong link between greater amygdala activity and anxiety disorders across the literature. In fact, the link between amygdala activity and other disorders (e.g., bipolar disorder), was relatively stronger when considering whole-brain analyses. In contrast, structures such as the thalamus and hippocampus, which are less frequently linked to anxiety, showed relatively greater activation across anxiety (Sprooten et al. 2017). Of course, these findings come from a single meta-analysis, but they converge with recent arguments that psychiatric disorders arise not from emotion-specific circuits, but from a set of common large-scale brain networks (Menon, 2011). This idea is articulated in a constructionist approach to mind-brain correspondence (Lindquist & Barrett, 2012).

Constructionism: Emotions as Emergent Phenomena

An alternative approach to understanding brain bases of emotion, and by extension, emotion dysregulation, is the TCE. The TCE is part of the broader class of "psychological constructionist" approaches to emotion (see Gendron & Barrett, 2009 for a historical review) and mind-brain correspondence more generally (Lindquist & Barrett, 2012). These approaches emerged in the history of psychology in attempts to explain failures of traditional approaches to account for existing empirical data on emotion (see Lindquist, 2013).

The TCE draws from patterns of data described in the literature on emotions, without appealing to essentialist assumptions. The TCE and psychological constructionist models before it (see Duffy, 1941; Hunt, 1941; James, 1890; Schachter & Singer, 1962; Wundt, 1897/1998, see Gendron & Barrett, 2009; Lindquist, 2013 for a historical review), recognize that emotions do not have their own specific behavioral action tendencies (Baumeister et al., 2010; DeWall, Baumeister, Chester, & Bushman, 2016), facial behaviors (Jack et al., 2012; Jack et al., 2016), peripheral physiological signatures (Siegel et al., in press), or causal mechanisms in the brain (Guillory & Bujarski, 2014; Lindquist et al., 2012; Wager et al., 2015). These approaches also recognize heterogeneity within each emotion category. For instance, although traditional approaches assume that "fear" names a set of instances that share a key set of features with a common causal mechanism, some instances of fear feel good and some feel bad (Wilson-Mendenhall et al. 2014), some involve an increase in heart rate and some involve a decrease (Kreibig, 2010), and some involve activity within the amygdala and some do not (Lindquist et al., 2012; Wager et al., 2008). Neural concomitants of different fear states (e.g., fear in a social context vs. fear in a physical context) are as dissimilar as neural concomitants of fear vs. anger (Wilson-Mendenhall et al., 2013). The TCE thus attempts to describe both similarities in objective measures (i.e., behaviors, facial expressions, peripheral physiology, brain activity; Barrett, 2006a, b; Barrett & Wager, 2006; Lindquist, 2013; Russell, 2003) that exist between

different emotion categories and differences that exist within emotion categories (Kreibig, 2010; Siegel et al., in press; Wilson-Mendenhall et al., 2013), all the while explaining why people subjectively experience emotions as relatively distinct from one another (e.g., people by and large experience fear as distinct from disgust; although see Barrett, 2006c for a discussion of individual differences).

The TCE achieves this by defining emotions as loose, fuzzy categories that are imposed by the minds' perceivers and emerge from a set of objectively measurable basic "elements" of the mind. One such element is *core affect*. Core affect is a product of the brain's attempts to maintain homeostasis by marshalling changes in the body in relation to external events (see Barrett, 2017a; Kleckner et al., 2017). Experientially, core affect can be described as feelings of activation or deactivation of the autonomic nervous system and feelings of pleasantness or unpleasantness (see Barrett & Bliss-Moreau, 2009; Barrett, 2016; Diener, Larsen, Levine, & Emmons, 1985; Duffy, 1957; Lindquist et al., 2013; Ortony, Clore, & Collins, 1988; Russell, 2003; Watson & Tellegen, 1985). Marshalling body changes to maintain homeostasis is experienced as activating, and depending on the context, sometimes unpleasant (e.g., in the face of a threat). In other cases, it might be experienced as activating and pleasant (e.g., in the case of pursuing a reward). All experiences have some core affective tone (Barrett & Bliss-Moreau, 2009; Craig, 2009; Russell, 2003) including but not limited to the experience of emotion categories. For instance, although fear is experienced as distinct from disgust, the TCE hypothesizes that each involves similar core affect (e.g., feelings of high activation and unpleasantness). Properties of core affect can construed as basic features of consciousness (Duncan & Barrett, 2007; Russell, 2005) and via reciprocal efferent and afferent projections between the brain and peripheral nervous system, core affect can act as a barometer that allows an organism to know whether it should approach or avoid something (Barrett & Bliss-Moreau,

2009; Russell, 2003).

The TCE predicts that the brain is always making meaning of core affect—sensations which are themselves ambiguous (Barrett, 2017a; MacCormack & Lindquist, 2017). To do so, the brain relies on the ongoing context outside the body, and prior experiences of which subjective experiences occurred in such contexts (Barrett, 2009; 2017a; Lindquist, 2013). A person thus experiences a specific emotion when he or she automatically and unconsciously makes meaning of (i.e., categorizes) their core affective sensations in given contexts (e.g., while delivering a speech) drawing on knowledge about specific emotion categories experienced in that context in the past (e.g., fear vs. excitement).

The second "element" in emotion is *categorization*. Categorization is the basic process(es) through which the brain uses prior experiences (i.e., memories, semantic and concept knowledge) to make predictions about the meaning of sensations in the present. Categorization is used to refine the meaning of all sensory information, whether those sensations are external to the body (as in visual perception; Barrett & Bar, 2009) or internal sensations of core affect (e.g., Lindquist & Barrett, 2008). The TCE thus shares much in common with predictive approaches to the mind (e.g., Clark, 2013; Friston, 2010; Hohwy, 2013; Lupyan & Clark, 2015) insofar as it hypothesizes that top-down information from prior experience is used to assign meaning to core affect, and produce discrete experiences of emotion (anger, fear, etc.). Consistent with the TCE, categorization yields subjective differentiation of emotions, even when the underlying physiology is not categorical. How people categorize their affective states drives self-reported emotional experiences (Jamieson et al., 2010; Kirkland & Cunningham, 2012; Lee et al., in press; Lindquist & Barrett, 2008; Oosterwijk et al., 2009; 2012), shifts physiology (i.e., whether someone is threatened vs. challenged; Jamieson et al., 2010; Kassam & Mendes, 2013; Oosterwijk et al., 2009), and alters observed patterns of brain activity during emotions (e.g.,

Lindquist et al., under review; Oosterwijk et al., 2012; Satpute et al., 2013a; 2013b; 2016; for a meta-analysis, see Brooks et al., 2017). Finally, the TCE hypothesizes that specific core affective sensations and category knowledge that are attended to and represented in any given instance alter ongoing emotional experiences.

The third "element" in emotion is *executive control.* Executive control refers to attentional resources that allow an individual to selectively enhance some information and suppress other information (Mack & Rock, 1998). It is hypothesized that executive control helps a person select aspects of core affective representations, category knowledge, or external sensory representations for conscious experience in a given instance, and to suppress other, less related representations. For instance, executive control processes appear to be involved in selecting category knowledge during emotion (Brooks et al., 2017; Oosterwijk et al., 2012). One hypothesis is that executive control allows the brain to unite together representations of core affect, category knowledge, and sensations from external perceptions (e.g., vision) into a unified experience of an emotion (see Lindquist, 2013, for a discussion). Consistent with this hypothesis, aspects of a brain network associated with executive control called the frontoparietal network show greater connectivity with the fusiform gyrus, an area involved in viewing faces, during intense anger experiences. These findings suggest that during anger, participants may draw on prior experiences of anger that involve seeing the faces of their enemies (Lindquist et al., under review). In such situations, regions associated with executive control are helping to select these representations in the service of creating the experience of anger.

Neural bases of emotions. Despite long-standing theoretical roots of psychological constructionist approaches to emotion, which began in the 19th Century (Dunlap, 1932; Harlow & Stagner, 1932; 1933; Spencer, 1855; 1894; Sully, 1892; see Gendron & Barrett, 2009 for a historical review), the TCE is the first such approach to be applied to understanding neural bases

of emotions (Barrett & Satpute, 2013; Lindquist et al., 2012; Lindquist & Barrett, 2012; Touroutoglou et al., 2015; for a neuroscience-based constructionist theory of memory, see Schacter, Norman, & Koutstaal., 1998; Schacter, Addis, & Buckner, 2007; for a constructionist theory of vision, see Bar, 2004). Indeed, its historical predecessors were articulated before modern brain imaging—as described herein—was available. Neuroscience findings bolster and refine behavioral and peripheral physiological work supporting the TCE. Rather than assuming that specific emotion categories, such as fear, map onto singular neural mechanisms (e.g., anatomical regions within the brain; Davis, 1992; or multivariate "fingerprints;" Saarimäki et al., 2015), the TCE hypothesizes that neural networks supporting more basic psychological processes such as core affect, categorization, and executive control contribute to emotional experiences.

There is growing evidence in the neuroimaging literature for the TCE hypotheses. As noted above, meta-analyses of neuroimaging studies on emotion reveal that emotion categories such as anger, fear, sadness, disgust, and happiness do not correspond to specific anatomical regions (Lindquist et al., 2012; Vytal & Hamann, 2010). Critically, these meta-analyses reveal that during instances of emotion, there is activity within and across a set of common networks that also activate during other psychological functions (e.g., memory, semantics, visual perception, etc; see Barrett & Satpute, 2013; Lindquist & Barrett, 2012). For instance, a meta-analysis using data-driven techniques to detect patterns of co-activation across neuroimaging studies of emotion revealed a set of six functional groups that span both the subcortex and cortex (Kober et al., 2008) (see Figure 7A-F). These functional groups did not correspond to specific emotions, but based on their functional neuroanatomy, appear to correspond to very basic psychological functions, many of which are predicted a priori by the TCE. Furthermore, these groups correspond to a set of "intrinsic" networks that are formed by the brain's structural architecture

(Deco et al., 2011), develop over infancy and early childhood (Gao et al., 2015; Pendl et al., 2015), are present across task domains (e.g., Habas et al., 2009; Seeley et al., 2007; Vincent et al., 2008) and exert constraints on information processing during emotion and cognition (Ciric et al., 2017; Cohen, 2017; Krienen et al., 2014; Yeo et al., 2011; Yeo, Krienen, Chee, & Buckner 2014).

The first group identified in our meta-analysis (Kober et al., 2008), referred to as the *core limbic*[3] *group*, comprised the amygdala, hypothalamus, ventral striatum and periaqueductal gray, all regions that contribute to visceromotor activation of the body via projections to the peripheral nervous system (Barrett & Simmons, 2015; Bujis & Van Eden, 2000). The second *lateral paralimbic*[4] *group* comprised the mid- and anterior insula and posterior orbitofrontal cortex (OFC), which receive afferent information via brainstem nuclei from the peripheral nervous system to represent visceromotor changes in ongoing experience (i.e., interoception; Craig, 2009; Kleckner et al., 2017). Together, this set of brain regions and their functional neuroanatomy correspond to the psychological element of core affect. Interestingly, many of these same regions constitute the intrinsic *salience network* (SN) (Seeley et al., 2007; Yeo et al., 2011). Activity within the SN during rest predicts self-reported arousal to evocative images (Touroutoglou et al., 2012), skin conductance responses (Kleckner et al., 2017), and reports of anxiety during scanning (Seely et al., 2007). This suggests functional relevance of this network to core affect.

The third group identified in our meta-analysis (Kober et al., 2008), referred to as the *medial posterior group*, comprised the posterior cingulate cortex (PCC) extending into primary visual cortex, regions associated with self-referential processing and vision. The fourth group was the *medial PFC group*, comprised of the dorsal medial PFC (dmPFC), the pregenual ACC (pgACC),

[3] In Latin, the word *limbic* means *border*. Herein, when we speak of a *limbic* group, we are referring to brain tissue primarily bordering or constituting the subcortex. Critically, we are not referring to notions of a *limbic system*, as outlined in the triune brain concept.

[4] *Paralimbic* tissue is 3-layer cortical tissue that abuts limbic tissue.

and rostral dorsal ACC (rdACC). These regions are associated with self-referential processing (Lou et al., 2004; Moran et al., 2006) and representation of value (Knutson et al., 2005; Levy & Glimcher, 2011). These regions often co-activate together across studies, particularly studies of autobiographical memory and semantics (Binder et al., 2009; Spreng, Mar, & Kim, 2009; Svoboda, McKinnon, & Levine, 2006), and are thought to collectively represent the brain's use of prior experiences to make meaning of internal and external sensations in the moment (Bar, 2011). Together, this set of brain regions and their functional neuroanatomy correspond to the psychological element of categorization. These same regions also constitute the intrinsic default mode network (DMN; Spreng, Mar, & Kim, 2009). Activity of the DMN during rest predicts self-reflection (Northoff & Bermpohl, 2004), emotion regulation (Wager et al., 2008), and planning future behaviors (Spreng et al., 2009). Thus, this network is involved in combining representations of prior experiences for use in thinking about the self, categorizing ongoing emotional experiences, and projecting oneself into the future.

Finally, the fifth group, referred to as the *cognitive/motor group*, comprised the right frontal operculum, bilateral inferior frontal gyrus (IFG), pre-supplementary motor area (pre-SMA), and left middle frontal gyrus. This set of brain regions and their functional neuroanatomy correspond to the psychological element of executive control[5] (Nee et al., 2007; Wager et al., 2004). Notably, these regions constitute the intrinsic executive control network known as the frontoparietal network (Corbetta & Shulman, 2002). This set of brain regions co-activate in response to top-down control that gates attention to sensory stimuli of potential behavioral significance (from the body, prior experiences, or the world) in the moment (Aron et al., 2004; Nee et al., 2007). Activity of the frontoparietal network during rest predicts response inhibition

[5] We note that executive control processes are typically associated with several brain regions, not only the ones we indicate here. In fact, regions from our *cognitive/motor group* emerged from data driven methods and were labeled with the network they best approximated.

(Nee et al., 2007), action selection (Corbetta & Shulman, 2002), and selection of semantic knowledge (Thompson-Schill et al., 1997).

The TCE suggests that networks mapping onto functions including core affect, categorization, and executive control functionally interact during the experience of emotions. Growing evidence is consistent with this hypothesis. For instance, Raz et al. (2012) used a network cohesion approach to examine the inter- and intra-network connectivity of the core limbic group observed in Kober et al. (2008) (roughly mapping on to aspects of the salience network) and the mPFC (part of the default mode network). There was increased functional connectivity between these two networks during an instance of sadness and participants' ratings of sadness additionally correlated with the degree of cohesion between these two networks. More recently, Raz and colleagues (2016) extended these findings to examine inter-network connectivity differences across different discrete emotions including anger, fear, and sadness. They examined connectivity within and between the SN and the DMN. Greater connectivity between dorsal parts of the SN (dorsal anterior insula, dorsal ACC) and aspects of the DMN extending into the subcortex (centromedial nucleus of the amygdala) was associated with more intense ratings of emotion during the experience of most instances of emotion. These findings provide some of the first evidence that networks associated with core affect and categorization interact during emotional experience.

If emotions emerge from dynamic functional coupling of intrinsic neural networks, then the novel prediction of the the TCE is that emotion dysregulation in the context of psychopathology originates from perturbations in the domain-general networks that contribute to emotions. Other recent accounts link psychopathology to dysregulation in intrinsic networks (Menon, 2011), but the TCE offers predictions at both psychological and neural levels. In the case of anxiety, it predicts that dysfunction in basic psychological processes that support emotion experience, such

as core affect, categorization and executive control, as manifested by dysfunction in the neural networks that support these functions.

Consistent with the TCE, preliminary evidence links anxiety disorders with alterations in functioning of the SN, DMN, and the frontoparietal network, which are hypothesized to support core affect, categorization, and executive control, respectively. Dysfunction in the anterior insula of the SN is thought to be a core feature of anxiety disorders (Paulus & Stein, 2006), suggesting an impairment in core affective processing, and in particular representation of afferent information from the viscera. Hyperactivity of the anterior insula of the SN is observed in individuals high in trait anxiety (Paulus & Stein, 2006; Stein et al., 2007) and individuals who score high on trait neuroticism (Feinstein, Stein, & Paulus, 2006). Neuroimaging work also shows decreased functional connectivity between the dACC—a region of the SN implicated in processing negative affect, pain, and cognitive control (Shackman et al., 2011)—and the amygdala, a subregion of the SN (Touroutoglou et al., 2012), among patients with generalized anxiety disorder at rest, relative to controls (Etkin et al., 2009). This suggests impairment in processing salient and potentially negatively valenced input. Furthermore, EEG studies report perturbed error-related brain activity in highly anxious individuals, specifically in the error-related negativity (ERN), a component of the event related potential (ERP) observed following the commision of an error in reaction-time tasks and linked to the ACC (Yeung, Botvinick, & Cohen, 2004). Studies demonstrate enhanced ERN in tasks involving neutral, non-emotional stimuli in patients with generalized anxiety disorder (Weinberg et al., 2010), obsessive-compulsive disorder (OCD; Xiao et al., 2011), and individuals with subclinical anxiety (Hajcak et al., 2003).These findings are consistent with increased functioning of the SN as a network important for detecting errors or conflict (Botvinick et al., 2001; Carter et al., 1999).

Other work links anxiety disorders with alterations in the DMN. For instance, patients with

anxiety relative to healthy controls show reductions in brain activation within the PCC and mPFC of the DMN while at rest, and while listening to emotionally neutral and threat-related words (Zhao et al., 2007). Critically, increased connectivity between parts of the DMN (e.g., PCC) and the right amygdala, a component of the SN, is associated with PTSD symptoms and predicts future PTSD (Lanius et al., 2010). These findings could be evidence that individuals with PTSD are more likely to conceptualize core affective sensations (e.g., a beating heart) as specific emotional experiences (e.g., fear) than healthy controls, who might experience these sensations as transient bodily feelings and not emotional per se. Other evidence points to relative differences in the functional connectivity within the DMN (including the ACC and mPFC) and SN (including the amygdala), with lesser functional connectivity in the DMN in PTSD but greater functional connectivity within the SN (Sripada et al., 2012).

Finally, many with anxiety disorders show altered function of the frontoparietal network and between this network and others. For instance, individuals high in trait anxiety show weaker functional connectivity relative to controls between regions of the SN (e.g., dACC), and the frontoparietal network (e.g., dlPFC) in an emotionally-neutral Stroop task (Basten et al., 2011). These results point to a general (non-threat related) impairment of attentional control in individuals high in trait anxiety. Similarly, aberrant functional connectivity patterns between regions of the frontoparietal network and the amygdala, a region within the SN (Touroutoglou et al., 2012), are observed in patients with generalized anxiety disorder (Etkin et al., 2009) and social anxiety disorder (Liao et al., 2010). Finally, patients with severe PTSD demonstrate impairments in disengaging the DMN and engaging the salience and frontoparietal networks during executive control tasks (Daniels et al., 2010). Together, these findings suggest that disorder might be characterized by a relative imbalance in processes linked to core affect vs. conceptualization vs. executive control.

Future Directions

The TCE charts a path forward for future investigations into the brain bases of human emotion and emotion dysregulation. It offers predictions not offered by traditional approaches. A first prediction is that dysregulation will emerge not only from activity within intrinsic networks, but also among these networks. The TCE predicts that anxiety may be more than a disorder circumscribed by an anatomically-defined fear circuit, but perhaps more related to abnormal variation within networks that support core affective, categorization, and executive control processes. A second prediction is that dysregulation of intrinsic networks is transdiagnostic. This prediction shares ideas in common with Insel et al. (2010) and Menon (2011). Here, we focused on anxiety disorders for illustration, and point out that dysregulation of intrinsic networks spans not only anxiety disorders, but other disorders as well. For example, dysregulation of the salience network is observed in major depressive disorder (Manoliu et al., 2014) and schizophrenia (Palaniyappan & Liddle, 2012). To understand how dysregulation in these networks occurs across disorders, we first need to understand how much variation underlies "regulated" function of these networks. Evaluating these predictions relies not only on changing theoretical frameworks for understanding emotions and mental illness, but on relatively new analytic techniques in neuroimaging.

Examining Interplay of Networks

One new direction prompted by the TCE is to evaluate neural networks that underlie regulated emotions and their interactions. As reviewed above, evidence suggests that emotions emerge from interactive effects of broad scale networks. Yet to date, very few studies have taken a network-based approach to examining neural bases of emotions (although see Lindquist et al., under review; Raz et al., 2012; 2016; Touroutoglou et al., 2015). Neuroimaging is increasingly examining the function of intrinsic networks in psychopathology, but this research began as

exploratory, and to our knowledge, there is no unified effort to understand how within-network or between network activity changes as a function of disorder and what mental implications of these changes are. The TCE offers such a framework. Of course, ongoing work must continue to validate the approach and link psychological levels of analysis (e.g. core affect) to neural levels of analysis (e.g., activity of the salience network). Novel statistical approaches are also necessary to reach these aims. To study functional dynamics of networks and how they change across different emotional states (dynamic functional connectivity; see Cohen, 2017), researchers must use advanced multivariate statistics. For instance, functional connectivity examines how time series of activation within different brain areas covary with one another to form complex networks. Directed functional connectivity approaches (e.g., Gates & Molenaar, 2012; Gates et al., 2014) further allow researchers to examine lagged and contemporaneous correlations between these brain area "hubs" and how these change across emotional states (e.g., anger vs. fear). For instance, we can examine how hubs of the salience network (e.g., insula, amygdala, dACC) change in connectivity within one another across anger and fear in healthy individuals. Similar approaches can be used to examine between-network co-variation across different emotional states. For example, we can examine how the salience network changes its connectivity with the default mode network during fear vs. anger. By understanding these dynamics and how they manifest among different healthy affective (and even non-affective) states, we can begin to understand regulated functions of the brain. Next, we can systematically explore how these network dynamics become dysregulated in mental disorder.

Variability in Emotions and Diagnostic Categories

A second new direction prompted by the TCE is to examine and model variability underlying emotions, both in regulated and dysregulated forms across diagnostic groups. Most human imaging research focuses on group-level mean brain activity without considering

individual differences in the circuitry that is associated with emotions. Statistical brain maps computed across groups of people often do not capture explanatory individual-level information about behavior or cognitions (Mueller et al., 2013) and inter-subject variability is often implicitly treated as noise rather than biologically informative features of brain organization (Zilles & Amunts, 2013). The social and/or environmental context in which a phenomenon is taking place is rarely modeled or even considered (Guloksuz, Pries, & van Os, 2017; Shankman & Gorka, 2015). These practices can lead to the false interpretation that a single process or set of processes is associated with a single emotion category, when in reality there may be different pathways to that emotion category across different people or even within the same person across instances. Growing neuroimaging research is consistent with the idea that there is heterogeneity—and even degeneracy (Price & Friston, 2002)—in the brain responses associated with emotions between people and within people across contexts. Degeneracy refers to the biological principle whereby different processes produce the same outcome. As an example of degeneracy, different brain patterns exist for the same emotion categories experienced across different contexts (e.g., Wilson-Mendenhall et al., 2011). Clinically, there is growing evidence that separate brain processes produce the same outcomes (e.g., anxiety symptoms) (Fisher, 2015; Gates et al., 2014; Price et al., 2017). Moving forward, we should recognize and model such sources of variation and degeneracy if we are to better understand how variation in brain processes leads to dysregulated emotions.

To study heterogeneity across categories (whether categories of regulated emotions such as fear and sadness or diagnostic categories such as anxiety and depression), researchers must use approaches that model different pathways to the same outcomes. Network-based sub-grouping approaches (e.g., Lane & Gates, 2017) arrive at group-level network solutions but also identify subgroups with different network patterns. We can examine how hubs of the salience network

(e.g., insula, amygdala, dACC) are connected differentially during fear in one context (e.g., social) vs. another (e.g., threat). Similar approaches can be used to examine between-network co-variation across different emotional contexts (e.g., how the salience network changes its connectivity with the default mode network during social fear vs. threat). We can even examine how different network patterns predict similar degrees of reported anxiety within a sample (e.g., Doyle, Lane, Brooks, Wilkins, Gates, & Lindquist, in prep).

Conclusion

In this chapter, we reviewed contemporary models of emotion and their implications for what goes awry in the brain during emotion dysregulation. We focused on neuroimaging approaches, which have promise for understanding emotion and emotion dysregulation in awake, emoting humans. After roughly 25 years of research, we have made substantial discoveries. We look forward to future research that uses sophisticated network-based approaches to search for neural processes that contribute to both healthy and dysregulated emotion.

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