Epilogue—The nature of emotion: *A research agenda for the 21st century*

Andrew S. Fox*

Department of Psychology and California National Primate Research Center, University of California, Davis, CA USA

Regina C. Lapate

Department of Psychology and Helen Wills Neuroscience Institute, University of California, Berkeley, CA USA

Richard J. Davidson

Center for Healthy Minds, University of Wisconsin—Madison, Madison, WI USA

Alexander J. Shackman*

Department of Psychology, Neuroscience and Cognitive Science Program, and Maryland Neuroimaging Center, University of Maryland, College Park, MD USA

***** A.S.F and A.J.S. contributed equally to this work; the order of authors was determined by a coin flip.

Please cite as:

Fox, A. S.*****, Lapate, R. C., Davidson, R. J. & Shackman, A. J.***** (*in press*). Epilogue—The nature of emotion: A research agenda for the 21st century. In Fox, A. S., Lapate, R. C., Shackman, A. J. & Davidson, R. J. (Eds.). *The nature of emotion: Fundamental questions* (2nd ed.). New York: Oxford University Press.

Address Correspondence to:

Andrew S. Fox (dfox@ucdavis.edu) –or– Alexander J. Shackman (shackman@umd.edu)

Emotion plays a central role in human experience and there is an abiding interest, among scientists, clinicians, and the public at large, in understanding the nature of emotion, identifying its biological underpinnings, and determining its contribution to other psychological processes, from cognition and decision-making, to health and disease. Over the past twenty-five years, methods for eliciting, assessing, and analyzing emotion have become increasingly refined (e.g., Coan & Allen, 2007; Dan-Glauser & Scherer, 2011; Kramer, Guillory, & Hancock, 2014; Samson, Kreibig, Soderstrom, Wade, & Gross, 2015; Soto & John, *in press*) and techniques for making sense of the underlying neurobiology have become more powerful and precise (e.g., Glasser et al., 2016; Kim, Adhikari, & Deisseroth, 2017; Urban & Roth, 2015; Woo, Chang, Lindquist, & Wager, 2017). The 92 essays that comprise *The Nature of Emotion* embody these exciting developments and make plain the important conceptual advances that have been made since the publication of the first edition in 1994. Despite this progress, it is clear that our understanding remains far from complete. Here, we outline a roadmap to the most important strategies and avenues for future research in the affective sciences. We begin by discussing two overarching conceptual issues that are likely to be of interest to all students of emotion. In the second section, we highlight the most important methodological challenges confronting the field and provide some specific recommendations for addressing them. Finally, in the third section, we describe several of the most exciting and potentially fruitful specific avenues for future research.

OVERARCHING CONCEPTUAL ISSUES

In this section, we describe two overarching conceptual issues: first, the need to develop a deeper understanding of the processes linking emotional antecedents to their responses and, second, the value and proper place of neurobiology in the study of emotion. We believe that both of these issues have far-reaching implications for all students of emotion.

Understanding the Links Connecting Emotional Stimuli and Responses

Once detected, emotion stimuli can spark a range of responses, including thoughts, behaviors, peripheral physiological changes, and, of course, feelings. Most of the contributors agree that emotions are functional, and involve this kind of complex, multi-componential response (Question 1), but there is no clear consensus on how emotional inputs—including foreground cues (e.g., predator), contextual cues (e.g., escape route), and interoceptive cues (e.g., fatigue)—are evaluated and used to select the most adaptive emotional response (Blanchard & Pearson, this volume; Moaz & Bar-Haim, this volume; Averill, 1983; Clore, 1994; Ekman, 1977, 1994b; Ekman & Cordaro, 2011; Ekman & Friesen, 1975; Frijda, 1994a, 1994b, 1994c; Lazarus, 1991, 1994a, 1994b; LeDoux, 1994, 2014; Levenson, 2011), although some valuable clues are just beginning to emerge (e.g., Fadok et al., 2017). In short, we still do not have solid answers to questions such as, *How does the organism determine the most appropriate feeling (e.g., anger vs. fear), titrate the intensity of the response (e.g., muttering under one's breath vs. overt physical violence), and select an appropriate suite of somatomotor responses (e.g., flight vs. defensive attack)? How does an individual "know" (i.e., predict) which response is most adaptive? How do these selection processes differ across emotions, antecedents, and contexts?*

The most influential contemporary theories of emotion include basic emotion models (e.g., Panksepp, this volume; Ekman & Cordaro, 2011), appraisal models (Ellsworth, this volume; Scherer, Schorr, & Johnstone, 2001), and constructivist models (e.g., Barrett, this volume). Each suggests a different mechanism by which emotions come to be and, although no theory has clearly emerged as a "winner," each approach has proved useful in its own right. Basic emotion models have provided a framework for cross-cultural and cross-species studies of emotions and helped to organize the field. The basic emotions model argues that emotions stem from an automatic appraisal process of distinct and universal antecedent events. Appraisal models, which have guided our understanding of emotion regulation, propose a substantial and elaborate appraisal process that dynamically unfolds over time and suggest that intermediate cognitions lead to an emotional response. Finally, constructivist models, posit that every emotional experience is different; that emotions are social constructs rather than natural kinds. They aim to explain the variety of emotional experiences by invoking principles of prediction—emotions are said to exist to make meaning of incoming sensations and fundamentally depend on an individual's conceptual representations of the world. Although the basic emotions model and the constructivist model are often pitted against each other, most of the contributors to *The Nature of Emotion* seem to acknowledge and, more importantly, make use of both perspectives. For example, Lee & Anderson suggest that *"Basic expressions may not be strong universal categories but the evidence for their functional origins provides a parsimonious, empirical account of their cultural consistency. … it is likely that these adaptive action tendencies were socially co-opted for communication, serving as anchoring sources of invariance in expression perception across cultures and contexts"* (Lee & Anderson, this volume). Likewise, Freeman proposes a dynamic model of emotion perception that links basic-emotion-like (bottom-up) cues with constructivist-like (top-down) influences that interact and stabilize onto particular percepts over time (Freeman, this volume).

A key challenge for the field will be to develop new theories that explain how emotions come to be. There is clear need for testable, biologically plausible hypotheses that address the complex mapping between emotional antecedents and their consequents. We hope that in the next edition the time will be ripe to ask, "*How are emotional stimuli linked to emotional responses?*"

The Conceptual Value of Neuroscience

One of the most striking developments since the initial publication of *The Nature of Emotion* (1994) has been the growing prominence of neuroscientific approaches to emotion or what has become popularly known as *affective neuroscience* (R. J. Davidson & Sutton, 1995; Panksepp, 1992, 1998). But what is the added value of studying the brain for understanding emotion? Can affective neuroscience really provide theoretical insights that go beyond the reach of more traditional measures—behavior, ratings, and peripheral physiology (Bradley, 2000; Bradley & Lang, 2000, 2007)? This is not an abstract or rhetorical question. Skeptics have questioned whether neuroscience can provide conceptually important evidence or adjudicate between alternative theoretical models (e.g., Coltheart, 2013) and hundreds of millions of research dollars have been spent on the assumption that it can. Many of the essays contained in the present volume say the answer is *Yes*. Taken together, they provide compelling evidence that studying the brain is useful for determining the nature, and not just the biological bases, of emotion.

Neurobiology has proven to be particularly helpful for unveiling otherwise hidden features of emotion (Questions 4 and 5; see also Adolphs, 2017). Neurobiological evidence teaches us, for example, that reward (or pleasure) is not a single, indivisible thing, but can instead be split into *wanting* (appetitive motivation, craving, and desire) and *liking* (hedonic pleasure and positive affect). Under normal conditions, wanting and liking are exceedingly difficult to tease apart using behavioral or self-report methods (Havermans, 2011). This challenge reflects the fact that wanting and liking are usually tightly coupled: humans and other animals are motivated to work harder for things that we enjoy more (e.g., an expertly prepared, gourmet dish vs. lukewarm porridge) and this appetitive drive rapidly declines once our desires are sated. Yet, two decades of careful study provides compelling evidence that wanting and liking reflect the operation of dissociable neural systems (Berridge & Kringelbach, 2015; Berridge & Robinson, 2016; Kringelbach & Berridge, 2012). Wanting critically depends on dopaminergic projections coursing from the brainstem to the nucleus accumbens, whereas liking reflects tiny 'hotspots' nestled within the accumbens and neighboring regions of the ventral striatum. In rodents, stimulation of these hedonic hotspots with opioids or cannabinoids—the endogenous neurochemicals underlying the pleasures of heroin and marijuana—selectively amplifies facial expressions of liking without altering wanting (i.e., willingness to work for reward). A similar story has emerged for fear, with mounting evidence that fear can be fractionated on neurobiological grounds into two or even three more basic constituents, loosely termed *panic*, *fear*, and *anxiety* (although see the section on nomenclature, below) (Adolphs, 2017; Davis, Walker, Miles, & Grillon, 2010; Shackman & Fox, 2016b). Beyond their implications for emotion theory (e.g., *How many emotions are there? Are emotions organized into families?*), these data have provided new insights into both the phenomenology and mechanistic underpinnings of substance abuse and anxiety disorders.

We encourage readers to reflect on the value of neuroscience as they peruse *The Nature of Emotion*. We urge the curious and the skeptical alike to read the responses to Questions 4 and 5 of this volume. Neuroscience generally does not provide easy answers: neurons in the same region often do entirely different things, the same neurons can be involved in entirely different processes depending on the context, and information is often stored across complex networks of neurons distributed throughout the brain (e.g., Berridge & Kringelbach, 2015; Fadok et al., 2017; Faull & Pattinson, 2017; Gungor & Paré, 2016; Janak & Tye, 2015; Namburi, Al-Hasani, Calhoon, Bruchas, & Tye, 2016; Pearson, Watson, & Platt, 2014; Pessoa, 2017; Reynolds & Berridge, 2008; Senn et al., 2014; Shackman & Fox, 2016a; Tovote et al., 2016; Xiu et al., 2014). Nevertheless, important progress has already been made. Affective neuroscience has revealed dissociable circuits—enabling us parse emotions into their more elementary constituents (e.g. liking vs. wanting, described above) and identified deep similarities between seemingly disparate psychological constructs, including emotion, pain, and cognition (Cavanagh & Shackman, 2015; de la Vega, Chang, Banich, Wager, & Yarkoni, 2016; Pessoa, 2017; Shackman, Salomons, et al., 2011).

Although neuroscience holds great promise for our understanding of emotion, neuroscience is not sufficient to gain insight into the nature of emotion. "Without well-characterized behavior and theories that can act as a constraint on circuit-level inferences, brains and behavior will be like two ships passing in the night" (Krakauer, Ghazanfar, Gomez-Marin, MacIver, & Poeppel, 2017, p. 484). Our biology can only be as strong as our behavioral models and assays. This need for scientific cross-talk is even more crucial when it comes to understanding the roots of emotional traits and emotional disorders, constructs that reflect bidirectional interactions between the brain and different kinds of psychosocial experiences, including parenting practices, social support, marital conflict, and socioeconomic adversity (Cramer et al., 2012a, 2012b; Kendler, 2012a, 2012b; Kendler & Halberstadt, 2013; Schwartz, Lilienfeld, Meca, & Sauvigné, 2016; Shackman, Tromp, et al., 2016; Zhang & Meaney, 2010). In short, scientists who view emotion through the lens of the neurobiology have much to learn from their non-neuroscientist colleagues. Likewise, students of emotion who do not themselves "do neuroscience" have much to gain by attending to the discoveries of affective neuroscience.

METHODOLOGICAL ISSUES AND RECOMMENDATIONS

In this section, we discuss some of the most important methodological challenges facing the affective sciences, and provide some recommendations for addressing them. Our hope is that these guidelines will prove useful to both producers of affective science—those charged with designing, executing, analyzing, interpreting, and disseminating research—as well as consumers and gatekeepers, including reviewers, editors, and funders. Wider adoption of these best practices would enhance the credibility, inferential utility, and practical significance of emotion research.

Open and Reproducible Affective Science

Understanding the nature of emotion demands reproducible research. Yet, the actual reproducibility of published research in the social and biomedical sciences is alarmingly low (Baker, 2016; Engber, 2017; Motyl et al., 2017; L. D. Nelson, Simmons, & Simonsohn, *in press;* Nuzzo, 2015; Palmers, 2016; Shrout & Rodgers, *in press;* Spellman, 2015; Tackett et al., *in press;* Yong, 2016) (but cf. Gilbert, King, Pettigrew, & Wilson, 2016). The resulting 'replication crisis' provides a critical opportunity for methodological reform and we would be remiss if we did not highlight a few of the most important recommendations and implications for emotion researchers (for detailed reviews, see Chambers, 2017; Munafò et al., 2017; Nichols et al., 2017).

Designing Emotion Research: Pre-Registration and Power. Addressing the CalTech graduating class of 1974, the Nobel laureate physicist Richard Feynman famously warned that:

"You must not fool yourself—and you are the easiest person to fool... If you've made up your mind to test a theory, or you want to explain some idea, you should always decide to publish it whichever way it comes out. If we only publish results of a certain kind, we can make the argument look good. [But] We must publish ***both*** *kinds of result"* (Feynman, 1974, p. 12).

Study pre-registration is a highly effective means of addressing Feynman's concern (Munafò et al., 2017). The pre-registration of basic study elements—general design and sampling plan, analytic approach, key outcomes and predictions—is a 'broad spectrum treatment' that inoculates against both cognitive biases (e.g., confirmation and hindsight biases) and questionable research practices (e.g., *p*-hacking, selective censoring, undisclosed data dredging, and flexible rules for terminating data collection). Although pre-registration has become standard practice in clinical medicine, it remains underutilized by emotion researchers, perhaps reflecting nagging doubts about stifling scientific creativity or 'extra paperwork.' Neither concern holds much water. Pre-registration does not preclude the possibility of serendipity—all analyses remain fair game (Poldrack, 2016). Pre-registration simply forces researchers to be transparent, with ourselves and with others, about which results were genuinely predicted and which are new discoveries (or *a posteriori* explanations) in need of replication. Once a study has been devised, pre-registration is comparatively painless using web-based tools maintained by the Open Science Foundation (https://osf.io) or other groups (e.g., https://clinicaltrials.gov).

Performing studies with inadequate statistical power represents another, perhaps more widely recognized, way to fool ourselves and a danger to reproducible science (Munafò et al., 2017; Poldrack et al., 2017). What is less well appreciated is that low power does not just increase the likelihood of false negatives ('null results'), it also increases the probability of false discoveries and produces overly rosy estimates of effect size (Button et al., 2013a, 2013b; Cohen, 1992; Yarkoni, 2009; Yarkoni & Westfall, *in press*), a lesson learned the hard way by geneticists over the past decade (Bastiaansen et al., 2014; Culverhouse et al., *in press;* Iacono, Vaidyanathan, Vrieze, & Malone, 2014; Okbay & Rietveld, 2015). Low power often reflects a lack of resources (e.g., funds, time, access to special populations), rather than a lack of knowledge; after all, software for computing power is freely available and power estimates are required for most grant applications. This challenge is not necessarily easily addressed, given resource constraints, but in many cases, can be circumvented by team science approaches, consortia, and other kinds of data sharing. These approaches are particularly well-suited to areas where the measures can be readily collected and quantified using standardized techniques (e.g., surveys of emotional traits, and anatomical MRI data).

Open Reporting and Sharing. In the same commencement address, Feynman underscored a second key principle, the need for full disclosure when communicating scientific research:

*"…the idea is to give **all** of the information to help others judge the value of your contribution; not just the information that leads to judgment in one particular direction or another"* (Feynman, 1974, p. 11).

Providing a complete and transparent report of the methods used in a particular study has a number of benefits. It enables other researchers to fairly evaluate the merits and limitations of the work, to more readily reuse the methods in their own work, and to unambiguously incorporate the results into narrative reviews and meta-analyses (Munafò et al., 2017). Public repositories and other new tools make it easy to go a step further and directly share data, code, and results (e.g., thresholded maps from brain imaging studies; http://neurovault.org). Shared data and results can be combined and reused in novel ways. Open source code can be examined, corrected, and refined in ways that are impossible with closed programs (Cox, Chen, Glen, Reynolds, & Taylor, 2017; Eglen et al., 2017; Eklund, Nichols, & Knutsson, 2016, 2017). Public sharing also makes it easier to recreate old experiments and analyses, even when the person who spearheaded the project has moved on (Eglen et al., 2017).

Although the replication crisis provides new urgency, the spirit motivating these four specific recommendations—pre-registration, power, reporting, and sharing—is not new. Skepticism, transparency, and communalism ('sharing') are all longstanding scientific values (M. S. Anderson, Martinson, & De Vries, 2007; Merton, 1942/1973) and concerns about statistical power have been voiced for more than a half-century (Cohen, 1962). What has changed is the development of a wide range of digital tools that make adoption easier than ever. Embracing all of these recommendations, or even one or two, promises to increase efficiency, enhance reproducibility, and accelerate our understanding of the nature of emotion.

Clear Nomenclature

Scientists have long recognized that the words we use to describe nature have the power to illuminate or to obfuscate. Francis Bacon, one of the original architects of the scientific method, wrote that the *"shoddy and inept application of words lays siege to the intellect in wondrous ways…words clearly force themselves on the intellect, throw everything in turmoil, and side-track men into empty disputes, countless controversies, and complete fictions"* (Barber, 2017, p. 500). Four hundred years later, Poldrack and Yarkoni made nearly the same point, arguing that *"many of the theoretical disputes that arise in psychology are…driven to a large extent by tacit differences in terminology that ramify as substantive disagreements"* (Poldrack & Yarkoni, 2016).

Understanding the nature and origins of emotion requires that researchers describe emotions in a clear and unambiguous way. Yet, as with other areas of psychology and neurobiology, this is rarely the case. Since the publication of the first edition of this volume, a growing number of commentators have highlighted problems with the language used to describe emotion perception (Schaafsma, Pfaff, Spunt, & Adolphs, 2015); emotional states (LeDoux, 2015; Shackman & Fox, 2016b); stress (Kagan, 2016a, 2016b; Koolhaas et al., 2011; Romero et al., 2015); emotional traits (Block, 1995; Morrison & Grammer, 2016; Nigg, 2017; Poldrack & Yarkoni, 2016; Sharma, Markon, & Clark, 2014); and emotional symptoms and disorders, such as anhedonia and depression (Zald & Treadway, 2017). The common refrain across these commentaries is that "seemingly harmless differences in nomenclature" (Zald & Treadway, 2017, p. 476) obscure important differences in neurobiology and phenomenology, impeding the development of cumulative knowledge.

This pervasive problem reflects several semantic sins, including the use of different terms to refer to the same idea (*jangle fallacy*; Thorndike, 1904) and a single term to refer to multiple ideas (*jingle fallacy*; Kelly, 1927). Take the case of *fear*. Naked and unadorned with information about antecedents or context (e.g., imminence of threat, possibility of escape), the word fear is insufficient for clear scientific communication. Like other English words for emotion, it can, and often is, used to refer to the *perception* of fearful or threatening stimuli, the *expression* of a range of defensive responses to threat, and the *experience* of fearful feelings, three processes that reflect overlapping but qualitatively distinct neural circuits (R. J. Davidson, 1993; LeDoux, 2012, 2014, 2015, *in press*). Among scientists and lay people, fear refers to a broad spectrum—or 'family' (http://atlasofemotions.org; Ekman, 1994a; Ekman & Davidson, 1994)—of subjective states, from mortal danger to existential angst, and there is ample evidence that these fearful states reflect partially dissociable neural systems (Blanchard, Griebel, & Blanchard, 2001; Calhoon & Tye, 2015; Fanselow & Lester, 1988; Fox & Shackman, *in press;* Kalin & Shelton, 1989; Mobbs et al., 2007; Shackman & Fox, 2016b). Preliminary attempts to name these systems have fallen prey to the sin of inconsistent usage. Inspired by evidence that fear, like reward and aggression[1], can be fractionated on etiological grounds into at least two kinds, a growing number of researchers draw a sharp distinction between *fear* (a phasic response to imminent danger) and *anxiety* (a sustained response in the absence of imminent danger; e.g., Barlow, 2000; Davis et al., 2010; LeDoux, 2015; Tovote, Fadok, & Luthi, 2015). In fact, a version of this dichotomy is now embodied in the National Institute of Mental Health's Research Domain Criteria (RDoC) as *Acute Threat* and *Potential Threat* (Kozak & Cuthbert, 2016)[2]. Confusion arises from the fact that lay people, scholars in other areas, the American Psychiatric Association's Diagnostic and Statistical Manual (American Psychiatric Association, 2013), psychometricians (Kotov et al., 2017), and even domain experts—at least in unguarded moments—use these terms interchangeably or inconsistently (Kagan, *in press;* Watson, Stanton, & Clark, *in press*). Even the seemingly more precise phrase *'diffuse, uncertain, or remote threat'* is used to refer to a bewildering array of paradigms, from 15 minutes of exposure to a unfamiliar arena in rodents to a several seconds of uncertain anticipation of an aversive image in humans (Shackman, Tromp, et al., 2016). The upshot is that English language words for emotion—anger, fear, disgust, joy, sadness and so on—and even more recently coined phrases, like 'uncertain threat,' can, and often do, refer to multiple phenomena (Wager, Krishnan, & Hitchcock, this volume; Barrett, 2017; Kagan, 2010) and as such represent a poor guide to the underlying mechanisms.

So what is to be done? There will always be a place for verbal shorthand and so some of these problems are inevitable, but we can easily do much better. We urge emotion researchers to:

- Be mindful of the jingle and jangle fallacies when considering the emotion literature

[1] Aggression can be split on functional and neurobiological grounds into systems involved in defensive, offensive (predatory), and conspecific aggression, with the latter including maternal aggression and resource competition (food, mates, and territory/shelter) (Adams, 2006; Berkowitz, 1993; R. J. Nelson & Trainor, 2007).

[2] See also https://www.nimh.nih.gov/research-priorities/rdoc/constructs/potential-threat-anxiety.shtml; https://www.nimh.nih.gov/research-priorities/rdoc/negative-valence-systems-workshop-proceedings.shtml.

- Eschew problematic redefinitions of everyday language
- Minimize or eliminate misleading single-word descriptors, such as fear, that lump together multiple processes and substrates.
- Beware of reification—*the index is not the construct* (Kagan, 1988; Kendler, 2016). Do not confuse 'fear' with potentiation of the startle reflex or freezing in the elevated plus-maze.
- Accurately specify the specific parameters of the antecedent stimuli, the context in which those stimuli are encountered, and the subsequent pattern of responses. Scientific constructs (e.g., gravity), gain strength when they are closely tied to empirical measurements (Marx, 1951).
- In some cases, as with stimuli or responses that vary in degree (e.g., physical imminence of threat, intensity of thermal stimulation, magnitude of monetary reward), it will be fruitful to develop and use computational models, which enable the use of unambiguous mathematical expressions that make precise predictions.

Rigor and Realistic Inference

Drawing strong inferences about emotions, their underlying neurobiology, and their consequences for cognition, decision-making, interpersonal processes, and other psychological processes, demands the use of carefully designed experimental paradigms, psychometrically sound measures, intelligent analytic choices, and sober interpretation of the results (Luck, 2005; Shackman et al., 2006; Tomarken, 1995). Tasks or conditions must be well equated for motor requirements and perceptual characteristics. In some cases, psychometric matching will be fruitful. Affect is notoriously fleeting and induction procedures typically fail to elicit the target affect in a subset of subjects (Shackman et al., 2006). Thus, it is imperative that the presence of the target emotion(s) be verified. Emotional states differ in their intensity as well as their persistence, which makes it crucial to understand the chronometry of affective responses (R. J.

Davidson, 1998; Shackman, Tromp, et al., 2016; Tracy, Klonsky, & Proudfit, 2014). Emotion measures often show weak coherence and, thus, it is useful to acquire separate measures of emotional experience, peripheral physiology, and behavior (Bradley & Lang, 2007; Ekman & Davidson, 1994; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005; Shackman et al., 2013). Investigators should be especially cautious when comparing responses, whether neural or behavioral, across tasks that markedly differ in duration or number of trials (i.e., in the variance of the read-out), as in paradigms where long blocks are compared to brief events.

Parametric manipulations of the emotion-eliciting stimuli (e.g., probability, duration, magnitude, or intensity) can be especially powerful. Strong claims of specificity require comparison with an appropriately arousing control condition. To draw strong inferences about disgust, for example, one needs to contrast the effects with those associated with an equally intense state of anger, fear, or joy. Likewise, strong claims about positive affect demand comparison with an equally potent state of negative affect. More generally, inferences about specificity or construct validity require comparison with a range of other tasks and conditions (Passingham, Stephan, & Kotter, 2002; Poldrack et al., 2017; Tomarken, 1995). Putative double dissociations need to be rigorously assessed by testing the appropriate interaction (e.g., Region x Condition, ERP Component x Condition, Emotion x Cognitive Task; Shackman, Maxwell, McMenamin, Greischar, & Davidson, 2011; Shackman et al., 2006; Somerville, Whalen, & Kelley, 2010). Investigators should be circumspect when interpreting unusually strong effects (Lakens, 2017). Concluding that a particular brain region is 'not involved' in something as complex and multi-dimensional as an emotion based on a significant difference in activity, a null test, or a single assay is completely unwarranted. For neurophysiological techniques, such as functional MRI (fMRI) and electroencephalography (EEG), trial-by-trial relations between neural signals and emotional experience provide some of the strongest and most direct links between the brain and emotion (Atlas, Bolger, Lindquist, & Wager, 2010; Cavanagh & Shackman, 2015; Lim, Padmala, & Pessoa, 2009). Machine-learning techniques, though not without their limitations (Paulus, 2015; Woo et al., 2017; Yarkoni & Westfall, *in press*), provide new opportunities for identifying patterns (e.g., neural activation, symptoms, behaviors) that robustly predict emotional states, traits, and disorders. Regardless of approach, it will be increasingly important to employ techniques that examine the generalizability of our findings (e.g., by cross-validating predictions *across* rather than within subjects). In summary, for optimal progress, we must be clear what we are studying, test our claims, and acknowledge the limitations of our research.

Emotions vary in important ways across sexes and cultures (e.g., Chiao, this volume; Shansky, this volume). Yet, scientists often make fundamental claims about the nature of emotion on the basis of data drawn from a narrow slice of this diversity. The vast majority of human studies rely on individuals drawn from Western, educated, industrialized, rich and democratic (WEIRD) societies—who represent perhaps as much as 80 percent of the participants in biopsychosocial research, but only 12 percent of the world's population (Henrich, Heine, & Norenzayan, 2010)—and laboratory studies of animals disproportionately rely on male rats and mice (Institute of Medicine, 2001). Whether the fruits of this work translate to 'everyone else' remains unclear. A similar concern applies to the issue of measurement context (Ekman & Davidson, 1994; LeDoux, 2014). The context in which an emotion is elicited can transform the emotion that is experienced (e.g., anger vs. fear), the way in which it is expressed (e.g., freezing vs. flight vs. defensive attack), and the brain circuits that subserve it (Blanchard & Pearson, this volume; Kagan, this volume; Barrett, 2017; Kagan, *in press;* Khalsa et al., 2016; LeDoux, 2014; Wager & Atlas, 2015). Yet, laboratory studies of emotion rely on a narrow range of measurement contexts (e.g., lying alone in a scanner). Students of emotion should be realistic about their conclusions and not assume that inferences derived from one context or group will necessarily prove universal.

Coordinated Cross-Species Research

Much of the neurobiological data reviewed in the *Nature of Emotion* comes from human EEG and imaging studies. The most critical limitation of imaging studies is that they are correlational and cannot address causation. Fully addressing this challenge mandates mechanistic studies in humans and animals, as well as coordinated cross-species research efforts. Animal models enable a degree of resolution, precision, and control that are nearly impossible to achieve in studies of humans. In contrast, human studies are essential for understanding the precise neural mechanisms supporting the subjective experience of emotions (D. J. Anderson & Adolphs, 2014; LeDoux, 2015) and for identifying the features of animal models that are conserved and, hence, most relevant to understanding human emotional disorders and, ultimately, to developing improved interventions for human suffering ('forward translation;' Birn et al., 2014; Hyman, 2016). Human studies also afford important opportunities for developing objective biomarkers of disease or disease risk (Woo et al., 2017) and for generating novel hypotheses that can be mechanistically assessed in animal models ('reverse translation').

In animal models, focal perturbation techniques can be combined with the same whole-brain imaging strategies routinely applied in humans, facilitating the development of integrated, bidirectional translational models (Borsook, Becerra, & Hargreaves, 2006; Casey et al., 2013; Desai et al., 2011; Fox et al., 2010; Grayson et al., 2016; Kalin et al., 2016). This combined approach is particularly valuable for determining whether changes in emotional behavior are mediated by alterations in the function of downstream regions, as occurs following OFC lesions in monkeys (Fox et al., 2010) or OFC damage in humans (Motzkin, Philippi, Oler, et al., 2015). Combining cognitive-behavioral, neurofeedback, neurostimulation, or pharmacological interventions with fMRI or EEG in humans can provide additional opportunities for understanding the neural mechanisms underlying the experience and expression of emotion (Duff et al., 2015; Lapate et al., *in press;* Paulus, Feinstein, Castillo, Simmons, & Stein, 2005; Schnyer et al., 2015; Sitaram et al., 2017). Similarly, imaging approaches can and should be applied to rare patients with circumscribed brain damage (Adolphs, 2016; Motzkin, Philippi, Oler, et al., 2015; Motzkin, Philippi, Wolf, Baskaya, & Koenigs, 2014, 2015; Spunt et al., 2015).

Once we establish candidate proximal causes for a particular emotion, we can begin to address some other crucial questions. For example, how specific is this circuitry (at the neuronal, voxel, or regional levels; Passingham et al., 2002; Pessoa, 2017; Woo et al., 2017; Zaki, Wager, Singer, Keysers, & Gazzola, 2016)? How is it related to more persistent affective phenomena, such as moods and emotional traits (Shackman, Tromp, et al., 2016)? Addressing specificity is particularly important for understanding how emotion is integrated into cognition, decision-making, and social processes and would provide valuable clues about why disparate features of emotion, such as vigilance and worrisome thoughts (Grupe & Nitschke, 2013) or anxiety and conflict monitoring (Cavanagh & Shackman, 2015), often co-vary. It would also help to address on-going debates about the degree to which emotions reflect specialized centers, domain-general modules, or some combination of these building blocks (Adolphs, 2017; Barrett, 2017).

Emotions in the Real World

Physiological studies of emotion have relied heavily on a limited number of well-controlled, but highly artificial manipulations—static faces, sounds, images, small monetary rewards, and so on—presented under unnatural conditions. Subjects may, for example, be instructed to passively view a randomized stream of positive and negative photographs while lying motionless in the cramped, dimly lit confines of an MRI scanner. By and large, these manipulations are far less arousing and much less engaging than the kinds of antecedents regularly encountered in daily life or those routinely used in animal models (Levenson, this volume; LeDoux, 2015; Levenson, 2011; Shackman et al., 2006). Although this approach has afforded a number of important insights, it is unclear how much of this knowledge is relevant to emotion in the real world—in the bedroom and the classroom, the boardroom and the bar.

Given the limitations of ambulatory measures of brain activity—there is no 'fMRI helmet' as yet—addressing this question requires integrating assays of brain function and behavior acquired in the scanner with measures of emotion and motivated behavior assessed under more naturalistic conditions in the laboratory (e.g., during semi-structured interactions; Creed & Funder, 1998; Laidlaw, Foulsham, Kuhn, & Kingstone, 2011; Perez-Edgar et al., 2010; Pfeiffer, Vogeley, & Schilbach, 2013) or in the field, a point also made by Ekman and Davidson in the first edition of *The Nature of Emotion* (Ekman & Davidson, 1994). Recent work combining fMRI with experience-sampling techniques underscores the potential of this approach for identifying the neural systems associated with naturalistic variation in mood and motivated behavior—a central goal of psychology, psychiatry, and the behavioral neurosciences (Berkman & Falk, 2013; Forbes et al., 2009; Heller et al., 2015; Lopez, Hofmann, Wagner, Kelley, & Heatherton, 2014; Wilson, Smyth, & MacLean, 2014). Prospective longitudinal designs would provide a valuable opportunity to discover the relevance of specific emotional circuits in the brain to the development of maladaptive mood and pathology-promoting behaviors, such as avoidance and social isolation (Admon et al., 2009; McLaughlin et al., 2014; Swartz, Knodt, Radtke, & Hariri, 2015).

The development of robust mobile eye trackers, the emergence of commercial software for automated analyses of facial expressions (Olderbak, Hildebrandt, Pinkpank, Sommer, & Wilhelm, 2014), and the widespread dissemination of smart-phone and other kinds of 'wearable' technology for adults and children afford additional opportunities for objectively, efficiently, and unobtrusively quantifying context, emotion, and motivated behavior *in vivo* (Gilmore, 2016; Gosling & Mason, 2015; Onnela & Rauch, 2016; Sano et al., 2015; Wrzus & Mehl, 2015). Combining these measures with laboratory assays of brain function and peripheral physiology (e.g., emotion-modulated startle) would provide novel clues about the neural systems supporting mood and motivated behavior in daily life, close to clinically and practically important end-points. This approach promises a depth of understanding that cannot be achieved using either animal models or isolated measures of brain function. Even in the absence of biological measures, these new tools promise important clues about the dynamics of emotional states in daily life (e.g., spill-over of mood across sequential contexts and assessments) and the social factors and coping behaviors that help govern the momentary expression of emotional traits (Shackman, Tromp, et al., 2016; Shackman et al., *in press;* Tambini, Rimmele, Phelps, & Davachi, 2017).

SPECIFIC CHALLENGES FOR FUTURE RESEARCH IN THE AFFECTVE SCIENCES

The 92 essays that make up the present volume tell us what we as a field know about the nature of emotion. Building on this foundation, in this third and final section of the Epilogue, we highlight some of the most important substantive areas for future research in the affective sciences.

The Integration of Emotion and Cognition

Emotion and cognition can seem quite different at first glance. Emotion is infused with feelings of pleasure or pain and manifests in readily discerned changes in the body, while cognition often appears devoid of these features. From classical times to the present, these apparent differences in phenomenological experience and peripheral physiology have led many scholars to treat emotion and cognition as categorically distinct, even oppositional, mental faculties (Barrett, 2017; de Sousa, 2014; Schmitter, 2014). A similar dichotomy pervades psychiatric nosology. But with the advent of new tools for noninvasively assaying the brain, the idea that emotion and cognition are different in kind began to dissolve (Okon-Singer, Hendler, Pessoa, & Shackman, 2015). At the time *The Nature of Emotion* was first published, in 1994, only a few commentators emphasized the neural integration of emotion and cognition (R. J. Davidson, 1994; Lazarus, 1991). A quarter century later, there is widespread agreement that emotion and cognition are deeply interwoven in the fabric of both the mind and the brain (Questions 1, 5, and 8). This should not be surprising—after all, the human brain did not evolve to optimize performance on laboratory measures of 'cold' cognition or to passively respond to experimental manipulations of 'hot' emotions. Our brain, like that of other animals, is the product of evolutionary pressures that demanded neural systems capable of using information about pleasure and pain, derived from stimuli saturated with hedonic and motivational significance, to regulate attention, learning, and behavior in the service of maximizing fitness.

Despite this progress, a number of important questions remain unresolved. For example, what is the functional significance of regions—like the anterior insula, mid-cingulate cortex, and dorsolateral prefrontal cortex—that are recruited by both cognitive and affective challenges (e.g., de la Vega et al., 2016; Shackman, Salomons, et al., 2011). Does this reflect a single invariant function or multiple functions (i.e., functional 'superimposition' or more dynamic kinds of multiplexing)? Does the function depend on the state of the larger networks in which these regions are embedded? Another general question concerns the relationship between emotions, on the one hand, and the kinds of functional circuits identified by cognitive neuroscience, on the other. How is threat-related vigilance, for example, related to the circuitry that underlies sustained attention (Davis & Whalen, 2001; Shackman, Kaplan, et al., 2016;

Shackman, Maxwell, et al., 2011)? How does rumination relate to short- and long-term memory systems? Addressing these questions is practically, as well as theoretically, important and would inform our understanding of psychiatric signs (e.g., threat over-generalization, aberrant reinforcement learning) and disorders (e.g., schizophrenia) that lie at the intersection of emotion and cognition.

Controlling Our Emotions

There have been tremendous advances in our scientific understanding of how *thinking* can exert bidirectional control over emotion (Questions 5, 7, and 8). Cognitive reappraisal is recognized as a means for deliberate emotion regulation and has had success as a core component of many treatments for emotional disorders (Gross, 2014). There is clear evidence that rumination, worry, and hopelessness can maladaptively promote and maintain negative affect and can contribute to the development of mood and anxiety disorders (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2013; Hyde, Mezulis, & Abramson, 2008; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, 2008). Despite this progress, it is clear that we have only scratched the surface of emotion regulation. A number of contributors discussed how thinking can generate and transform emotions. A keen example comes from Caroline Blanchard's story of how she "slept for nearly two years with a scarf tied around her neck after reading Bram Stoker's *Dracula* at an unwisely precocious age" (Blanchard & Pearson, this volume). This childhood anecdote highlights the power of cognition—often passed from person-to-person through story, conversation, and the written word—to trigger strong emotions and motivate enduring behavioral changes. We invite readers to reflect on what this implies about the limits (or lack of limits)—on our capacity to use our thoughts to control our emotions.

Thoughts can not only generate an emotional response, but they can also modulate the type of emotion that is provoked. For instance, thoughts can bridge between context and context-appropriate emotions. For example, as Maoz & Bar-Haim note, our feelings about a colleague running late to a meeting are inherently linked to our thoughts about her normal behavior, and can range from anger and frustration (if she is habitually late) to concern and apprehension (if she is habitually punctual) (Maoz & Bar-Haim, this volume). More generally, thoughts can transform the nature of an emotional stimulus. A delicious juice can quickly become disgusting following exposure to a perceived contaminant, such as a bug or bedpan (even after it has been sterilized!) (Rozin, Haidt, & McCauley, 2008). Moreover, this disgust-linked contamination changes across development, enabling older children to become disgusted by increasingly abstract ideas (e.g., a swastika or klansman's white hood) as their cognitive abilities mature (Rozin et al., 2008). These data underscore the intimate links between cognition and emotion, and reaffirm the ability of thoughts and ideas to control our feelings.

In short, our thoughts are linked to our emotions along the entire cascade of emotion processing and across a range of time-scales. A significant challenge for affective scientists is to develop a deeper understanding of precisely how these cognitive appraisals in emotional processing operate. For example, relatively little is known about the more elementary cognitive processes that underlie appraisal and appraisal tendencies (but cf. Scherer, 2009). What role do trait-like appraisal tendencies play in determining affective-style and to what extent are these processes susceptible to voluntary or habitual training? One hint that trait-like appraisal tendencies can change comes from the work of Carstensen and colleagues, who suggest that a changing appraisal of the subjective sense of time left on this planet mediates the increases in well-being seen during old-age (Carstensen, this volume). Reconceptualizing and elucidating the appraisal process to include these long-lasting appraisals can provide a framework for new, and potentially revolutionary, methods to manage and regulate emotions, and treat emotional disorders.

Influencing Others' Emotions

Emotion is profoundly social (Questions 9-10). Emotional experiences are routinely shared and dissected with friends and family (Rime, 2009) and social engagement tends to promote positive affect (Clark & Watson, 1988a, 1988b; Watson, 1988; Watson, Clark, McIntyre, & Hamaker, 1992). Most attempts at emotion regulation occur in social contexts (Gross, Richards, & John, 2006) and there is ample evidence that close companions play a vital role in coping with stress and regulating negative affect (Bolger & Eckenrode, 1991; Myers, 1999). Individuals differ in their ability to capitalize on these kinds of socio-emotional support (Shackman et al., *in press*) and those prone to loneliness and low levels of emotional intimacy are more likely to experience adverse outcomes, such as divorce and depression (Cacioppo, Grippo, London, Goossens, & Cacioppo, 2015; Editors, 2010; Kendler & Halberstadt, 2013; Shackman, Tromp, et al., 2016). In short, it is clear that other individuals can, and frequently do, influence our emotions in a variety of important ways (Coan & Sbarra, 2015; Zaki & Williams, 2013).

Of course, interpersonal processes are a two-way street, creating numerous opportunities for the bidirectional control of emotion. Parents and teachers, spouses and therapists, entertainers and politicians all leverage the capacity to strategically regulate the emotions of others to varying degrees, but we have only just begun to develop a scientific understanding of the underlying mechanisms (Reeck, Ames, & Ochsner, 2016). Intentionally managing others' emotions may depend on the same kinds of techniques commonly applied to the self-regulation of emotion, including situation selection and modification, distraction, cognitive reappraisal, and response modulation (Reeck et al., 2016). Recent work also highlights the possibility of deliberately cultivating states, such as compassion, that promote prosocial responses to others suffering and distress (Engen & Singer, this volume; Stellar & Keltner, 2014; Weisz & Zaki, *in press;* Weng et al., 2013). All of these regulatory strategies critically depend on accurately inferring others' emotions, often on the basis of limited or ambiguous information (Question 10). Because the very nature of perception can be susceptible to the influences of stereotypes and bias, it will be critical to develop techniques to overcome biases in active perception. Developing a deeper understanding of the underlying perceptual, computational, and neurobiological mechanisms that support interpersonal regulation of emotion is important and would guide the development of novel interventions focused on dyads (e.g., parents and their offspring) and other more complex social networks (Baucom, Belus, Adelman, Fischer, & Paprocki, 2014; Chronis-Tuscano et al., 2015; Kok & Singer, 2017; Marroquin, 2011).

Expanding the Physical Bases of Emotion

Scholars have long realized that it is problematic to consider emotion without taking signals from the body into account. Classical perspectives on physical embodiment focus on transient changes in the expressive muscles of the face, the heart, and the peripheral nervous system that prepare the body to adaptively respond to acute emotional challenges (Damasio & Damasio, this volume; Wood, Martin & Niedenthal, this volume; LeDoux, 2015). However, it has become clear that we need to expand these classical perspectives on embodiment. While our central nervous system evolved to emote (Fox, this volume), other bodily systems and organisms seem to have evolved the capacity to influence our emotions, something not addressed by existing theories of emotion. Future theories will need to account for the influence of the immune system as well as the many trillions of microorganisms that live on and within our bodies. Sickness behavior—which is characterized by outward signs that are similar to depression, including lethargy, apathy, and a desire to be alone—provides an excellent and relatively well-understood example of the immune system's role in governing mood and emotional behavior (Rosenkranz, this volume). Likewise, the curious case of the parasite *toxoplasma gondii* provides compelling evidence that microorganisms can dramatically influence motivated behavior (Rosenkranz, this volume; House, Vyas, & Sapolsky, 2011; Vyas, Kim, Giacomini, Boothroyd, & Sapolsky, 2007). *T. gondii* can only sexually reproduce in the cat gut. The problem is that they are regularly excreted and therefore require an intermediate host, such as a bird or rodent, to make their way back. After infecting their intermediate host, *T. gondii* alter the host's neural response to cat odor, changing their innate aversion to attraction and increasing the likelihood that a cat will eat the intermediate host. These kinds of observations tell us that the universe of interactions between emotion and the body is vastly larger, considerably more complex, and can play out over much longer spans of time than James and Lange probably had in mind. In particular, they highlight the need to expand embodied models of emotion to encompass a broader spectrum of peripheral biology, including the immune system and the body's microbiome.

Emotions and Decision-Making

Another major development since the initial publication of this volume has been the realization that affective processes are central to value-based decision-making. The observation that optimal and adaptive decisions rely on affective valuation rather than 'cold cognition' was an important advance for both affective and economic science. During this same span, a substantial body of evidence has amassed to indicate that the neural systems underlying emotion largely overlap with those supporting value-based decision-making; that emotion-altering lesions or drugs frequently change decision-making; and that task-irrelevant emotion can bias decisions (e.g., Engelmann & Hare, this volume; Knutson & Stallen, this volume; Murray, this volume; Tymula & Glimcher, this volume. These data dovetail with earlier observations that emotions help organisms choose adaptive behaviors that facilitate survival and reproduction. Determining exactly how emotions influence decisions can feed-back and inform our understanding of emotion and has potentially important implications for public health and public policy, with applications to obesity, addiction, and depression.

Looking to the future, it will be important to develop increasingly accurate models of choice that incorporate the biasing influence of emotional states, traits, and disorders. Choices often hinge on predictions about the value of potential outcomes. A number of contributors suggested that these economic valuation signals can be conceptualized as a form of affect, and that simulated emotion forms the basis of value based decision-making (e.g., Chang & Jolly, this volume; Engelmann & Hare, this volume; Hartley & Sokol-Hessner, this volume; Reber & Tranel, this volume). A chief strength of decision-making research has been the development of formal and precise mathematical models that underlie decision-making. These models have allowed researchers to tease apart the hidden processes that contribute to decision-making, and search for their neural and psychological correlates. For example, within the present volume specific decision-weights have been associated with particular aspects of emotion, including surprise (Friston, Joffily, Barrett, & Seth, this volume), guilt (Chang & Jolly, this volume), and arousal (Hartley & Sokol-Hessner, this volume). A major challenge for the field will be to understand the influence of emotions on the economic axioms of choice (Tymula & Glimcher, this volume). This will allow researchers to precisely articulate how current economic models are wrong and provide a clear guide for what adaptations to these models are necessary to best account for human behavior (e.g., decision-making under risk, fairness) (Fehr & Schmidt, 1999; Kahneman & Tversky, 1979).

The development of increasingly accurate models of decision-making will provide insight into why people make maladaptive and/or selfish decisions. Insight into how we assign value to food and drugs, for example, has promise for helping to clarify the etiology and treatment of addiction, a major public health issue. Likewise, developing a more comprehensive understanding of prosocial valuation, would guide potentially revolutionary developments of public policy focused on optimally incentivizing the global good and collective well-being (see the section on Plasticity, below).

The Development of Emotion and the Impact of Developmental Insults

There is widespread agreement that most, if not all, aspects of emotion change and mature throughout development, from gestation through late life (Question 14). Yet, the precise nature of many of these changes remains poorly understood. It will be necessary for affective scientists to disentangle often co-occurring processes (e.g., changes in 'emotional reactivity' vs. 'emotion-regulatory' systems; Crone & Pfeifer, this volume), in order to understand the mechanisms of developmentally relevant biological and psychological insults, and integrate emotional development with modern statistical learning theories.

Parsing the causal mechanisms underlying developmental changes is difficult because social, cognitive, and biological development often occur in tandem. Thus, a major challenge for affective developmental research is to develop tools and techniques to dissect the distinct contributions of changes in physical maturation (e.g., brain development, sex hormones), cognition (e.g., selective attention, cognitive control, working memory), environmental stressors (e.g., new school), and new social roles (e.g., dating). For example, does teenage emotional lability result from prefrontal-limbic imbalance or increased exposure to new and unknown situations (e.g., Crone & Pfeifer, this volume)? Presumably each has its own impact, but we will never know without intensive longitudinal assessments of neurobiology and cognition in the laboratory, coupled with ecological assessments of real-world emotion and socio-contextual information, alongside mechanistic studies of development in non-human animals.

There is ample evidence that biological and psychological insults (e.g., fetal exposure to maternal sickness, physical maltreatment, death of a spouse) can have enduring consequences for emotional health and well-being as we develop. Unfortunately, because we know preciously little about the specific mechanisms underlying the interaction between these developmental insults and the brain, we generally cannot predict how they will affect an individual, nor can we prescribe optimal development- or insult-specific interventions. Therefore, an important goal of developmental affective research will be to determine which components of emotion development are most susceptible to different kinds of biological and psychological insults, and determine the extent to which they are better conceptualized as temporary setbacks or a slowing of development.

Finally, it will be fruitful to integrate theories of emotion development with generalized statistical learning theory, which describes the process of using statistical regularities to learn about the world (Saffran, 2003). For example, when a child cries as her mother leaves, is it because the event is highly aversive or because it is negative and surprising? Friston and colleagues' theory of emotion, which suggests that emotions represent a kind of prediction error, implies that the development of emotion is intricately intertwined with a child's ability to accurately predict the world (Friston, Joffily, Barrett, & Seth, this volume). Thus, in the case of the child, the surprise may be more relevant than the aversiveness. As children develop better predictions about when food is coming and when parents will return, they may cease to express extreme emotions to these common events. This conceptual approach provides a unique set of hypotheses that may provide insight into the development of emotion early in life.

Feelings and Consciousness

Moving forward, there is a great need to distinguish *feelings*—that is, the subjective experience of emotion—from other components of emotion, such as alterations in peripheral physiology and action tendencies. Feelings are a fundamental component of the everyday experience of emotion and have intrinsic worth, as they form the basis of perceived well-being and largely define various forms of psychopathology. Subjective experience of feelings requires a conscious awareness of ongoing affective processing, and, importantly, conscious awareness of emotion-eliciting events has been demonstrated to influence neural and behavioral aspects of emotion processing (Question 12; LeDoux & Brown, 2017).

Developing a deeper understanding of the role of conscious awareness in emotion is important and promises to refine our thinking about the relations between emotions, feelings, and psychopathology (LeDoux, 2015). In particular, it will be critical to identify the relevant *types* of conscious awareness and each of their effects on emotional responding. For example, while a number of contributors suggested that the conscious awareness of emotion-eliciting events modulates neural responses and behavioral consequences of emotional processing (Question 12), it remains unclear which aspects of conscious awareness are essential: awareness of the emotion elicitor, awareness of the subsequent emotional response (interoception), and/or of the link between the two (contingency or source-of-emotion awareness).

Moreover, although it is widely assumed that conscious awareness of feeling states is critical for emotion regulation, and that increasing awareness of emotional triggers is a critical factor for distinct therapeutic approaches (e.g., psychotherapy and mindfulness), the necessity of conscious awareness *per se* in these processes has rarely been rigorously tested. For example, is decision-making more strategic when accompanied by conscious awareness of emotional events that influence the decision? Further, does awareness of peripheral-physiological aspects of an emotional response promote emotion regulation (e.g., by facilitating recovery or a return to baseline conditions)? Answering these and related questions will be essential for the development of well-informed theories of consciousness in emotion that have the potential to optimize cognitive-behavioral interventions.

Plasticity and Intervention

Classical theories of affective style, temperament, and personality would seem to suggest that the human brain is static, certainly once adulthood is reached. But it has become increasingly clear that, like a mature but growing tree, our brains are in constant motion. Throughout our lives, new synapses are being created, new neurons are being born, existing connections are being strengthened, and, ultimately, new thoughts are being generated (Goldsmith, this volume; McEwen, this volume). In parallel, evidence that emotional traits can, and do, change with time and training is beginning to accumulate (e.g., Roberts et al., 2017). Like the motor and visual systems, the emotional brain can change with experience. In the extreme, we can develop PTSD or learn to enjoy rollercoasters. More broadly, measures of dispositional traits only show moderate stability over long periods of time, and although a neurotic child is somewhat more likely to become a neurotic adult, they are not destined for this fate (Borghuis et al., *in press;* Fraley & Roberts, 2005; Hakulinen et al., 2015; Milojev & Sibley, 2017; Nye, Allemand, Gosling, Potter, & Roberts, 2016; Roberts & DelVecchio, 2000; Roberts & Mroczek, 2008; Roberts, Walton, & Viechtbauer, 2006) .

The mechanisms underlying this plasticity remain underexplored, making it a key challenge for the field. Although environmental influences, both good and bad, can modulate emotional traits (Shackman, Tromp, et al., 2016), the limits of these influences remain mostly unknown. Moving forward, it will be helpful for researchers to examine individuals exposed to more extreme cultural and environmental changes (e.g., adoption, emigration, enlistment).

Perhaps the most tantalizing prospect is our capacity to change in response to explicit interventions or training. Humans have learned to train our awareness, treat our anxieties, and practice being compassionate (e.g., R. J. Davidson & Harrington; Hofmann & Smits, 2008; Lutz, Slagter, Dunne, & Davidson, 2008). Scientists have observed that each of these interventions is associated with corresponding changes in neural function (e.g., Furmark et al., 2002; Tang, Holzel, & Posner, 2015; Weng et al., 2013). These data remind us that enduring changes in the mind necessarily reflect alterations in protein synthesis and expression in the brain. These neuroplasticity related processes are, in turn, related to individual differences in emotionality, and can be modulated by stress and exercise (e.g., R. J. Davidson & McEwen, 2012; Fox & Kalin, 2014; van Praag, Christie, Sejnowski, & Gage, 1999). In adult mice, for example, neurogenesis is associated with decreases in anxiety-like behavior, is increased with exercise, and is required for the behavioral effects of antidepressants (Pereira et al., 2007; Sahay & Hen, 2007; Santarelli et al., 2003). Developing a deeper understanding of the neural mechanisms that support different forms of emotional plasticity is important and would inform the development of new, biologically-motivated pharmacological, cognitive-behavioral, contemplative, and pharmacological interventions (R. J. Davidson & Kaszniak, 2015).

Finally, although we often think of interventions as treatments aimed at healing the sick, several contributors highlighted the possibility of emotional enhancements, the possibility of moving beyond baseline levels of emotional well-being (e.g., Fredrickson, this volume; Ryff, this volume). As we strive for a more peaceful and blissful world, a major challenge for affective scientists will be to develop training programs aimed at cultivating compassion and contentment.

CONCLUSIONS

Emotion is a core feature of the human condition, with important consequences for health, for wealth, and for wellbeing. Recent years have witnessed growing enthusiasm for understanding both the nature and the biological bases of emotion. The research contained in this volume provides a detailed survey of the current state of the affective sciences and highlights the important advances that have been made since the publication of the first edition, nearly a quarter century ago. Some of the fundamental questions originally posed by Ekman and Davidson have now been largely answered (e.g., "*Can emotions be regulated?*"), while others have proven more elusive ("*What is an emotion?*"). In some cases, there are now sufficient data to enable preliminary answers to entirely new kinds of questions ("*How are emotions organized in the brain?*"). Still, it is clear that most of the work, both empirical and conceptual, needed to understand the emotions and related affective phenomena remains undone. A major goal of *The Nature of Emotion* is to motivate the current and next generation of affective scientists to do the research and the thinking that will be required to address these outstanding questions, to develop new questions, and to generate more complete and useful theoretical models.

In this Epilogue, we have outlined some of the most important strategies and directions for future research in the affective sciences. At the broadest level, it will be important to develop a more complete understanding of the mechanisms linking emotional antecedents to their consequents. When appropriately grounded in behavior, neurobiological approaches promise to be especially valuable for addressing this and other key questions. The wider embrace of best practices—open and reproducible science, clear nomenclature, rigorous methods and realistic inferences—would increase the efficiency, reproducibility, and inferential utility of emotion research. An increased focus on coordinated cross-species models would accelerate our understanding of mechanism and has the potential to provide unique insights into the nature of emotion. Integrating laboratory assays of the brain and behavior with measures collected in the field using experience-sampling techniques, ambulatory physiology, and other kinds of smartphone-enabled and 'wearable' technologies would provide important clues about the neural systems supporting emotion and motivated behavior in daily life—a depth of understanding that cannot be achieved using animal models or isolated measures of brain function. In the final section of the Epilogue, we highlighted what we view as eight of the most exciting and potentially impactful avenues for the next generation of emotion research. In particular, we believe that there is a deep need for programmatic research focused on understanding the:

- Interplay and integration of emotion and cognition
- Self-regulation of our own emotions
- Social regulation of others' emotions
- Role of the immune system and microbiome in emotion regulation and dysregulation
- Development of emotion and the nature of developmental insults
- Nature of consciousness and its contribution to subjective feelings
- Origins of emotional plasticity and the processes underlying effective emotion interventions

Work to address these issues is a matter of practical as well as theoretical importance. Some of the most common, costly, and intractable mental illnesses—anxiety, depression, schizophrenia, substance abuse, autism, and so on—involve prominent emotional disturbances. Collectively, these psychiatric disorders impose a larger burden on global public health and the economy than cancer or cardiovascular disease (Collins et al., 2011; DiLuca & Olesen, 2014; Whiteford et al., 2013), underscoring the need to develop a deeper understanding of the nature and origins of emotion and the myriad ways in which emotional states and traits influence the way we think, feel, and behave.

To this end, elucidating the nature of emotion will require collaboration among researchers drawn from a range of disciplines, which extend far beyond affective psychology and affective neuroscience—from anthropologists and ethologists to social and cognitive psychologists, from economists to electrical engineers. Affective science is, by its very nature, interdisciplinary, and to address these fundamental questions, we will have to work together.

We hope that readers of this volume make a lasting impression on our scientific understanding of emotion, that they work to address the 14 fundamental questions that we have considered, and that they play a role in developing new ones. Ultimately, we hope that a deeper understanding of emotion will inform and accelerate the development of strategies for bringing contentment, compassion, and joy to the world.

ACKNOWLEDGEMENTS

This work was supported by the National Institutes of Health (AG051426, AT004952, DA040717, HD090256, MH018931, MH043454, MH100031, MH107444, MH113347); University of California, Davis; California National Primate Research Center; University of Maryland, College Park; Silicon Valley Community Foundation; and several gifts to the Center for Healthy Minds. Authors declare no conflicts of interest. We appreciate the assistance of Andy DeClercq, L. Friedman, and D. Tromp and critical feedback from L. Pessoa.

AUTHOR CONTRIBUTIONS

The authors collectively developed the overall structure of the review. A.S.F. and A.J.S. outlined and wrote the review. All the authors revised the review and approved the final version.

REFERENCES

Adams, D. B. (2006). Brain mechanisms of aggressive behavior: An updated review. *Neuroscience & Biobehavioral Reviews, 30*, 304-318.

Admon, R., Lubin, G., Stern, O., Rosenberg, K., Sela, L., Ben-Ami, H., & Hendler, T. (2009). Human vulnerability to stress depends on amygdala's predisposition and hippocampal plasticity. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 14120-14125.

Adolphs, R. (2016). Human lesion studies in the 21st century. *Neuron, 90*, 1151-1153.

Adolphs, R. (2017). How should neuroscience study emotions? by distinguishing emotion states, concepts, and experiences. *Social Cognitive and Affective Neuroscience, 12*, 24-31.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.).

Anderson, D. J., & Adolphs, R. (2014). A framework for studying emotions across species. *Cell, 157*, 187-200.

Anderson, M. S., Martinson, B. C., & De Vries, R. E. (2007). Normative dissoance in science: Results from a national survey of U.S. scientists. *Journal of Empirical Research on Human Research Ethics, 2*, 3-14.

Atlas, L. Y., Bolger, N., Lindquist, M. A., & Wager, T. D. (2010). Brain mediators of predictive cue effects on perceived pain. *Journal of Neuroscience, 30*, 12964-12977.

Averill, J. R. (1983). Studies on anger and aggression. Implications for theories of emotion. *American Psychologist, 38*, 1145-1160.

Baker, M. (2016). Is there a reproducibility crisis? *Nature, 533*, 452-454.

Barber, A. (2017). Francis Bacon. In M. Cameron, B. Hill & R. J. Stainton (Eds.), *Sourcebook in the history of philosphy of language. Primary source texts from the pre-Socratics to Mill* (pp. 497-502). New York: Springer.

Barlow, D. H. (2000). Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *American Psychologist, 55*, 1247-1263.

Barlow, D. H., Sauer-Zavala, S., Carl, J. R., Bullis, J. R., & Ellard, K. K. (2013). The nature, diagnosis, and treatment of neuroticism: Back to the future. *Clinical Psychological Science, 2*.

Barrett, L. F. (2017). *How emotions are made: The secret life of the brain*. New York: Houghton-Mifflin-Harcourt.

Bastiaansen, J. A., Servaas, M. N., Marsman, J. B., Ormel, J., Nolte, I. M., Riese, H., & Aleman, A. (2014). Filling the gap: Relationship between the serotonin-transporter-linked polymorphic region and amygdala activation. *Psychol Sci, 25*, 2058-2066.

Baucom, D. H., Belus, J. M., Adelman, C. B., Fischer, M. S., & Paprocki, C. (2014). Couple-based interventions for psychopathology: a renewed direction for the field. *Family Process, 53*, 445-461.

Berkman, E. T., & Falk, E. B. (2013). Beyond brain mapping: Using neural measures to predict real-world outcomes. *Curr Dir Psychol Sci, 22*, 45-50.

Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control*. Philadelphia, PA: Temple University Press.

Berridge, K. C., & Kringelbach, M. L. (2015). Pleasure systems in the brain. *Neuron, 86*, 646-664.

Berridge, K. C., & Robinson, T. E. (2016). Liking, wanting, and the incentive-sensitization theory of addiction. *American Psychologist, 71*, 670-679.

Birn, R. M., Shackman, A. J., Oler, J. A., Williams, L. E., McFarlin, D. R., Rogers, G. M., . . . Kalin, N. H. (2014). Evolutionarily conserved dysfunction of prefrontal-amygdalar connectivity in early-life anxiety. *Molecular Psychiatry, 19*, 915-922.

Blanchard, D. C., Griebel, G., & Blanchard, R. J. (2001). Mouse defensive behaviors: pharmacological and behavioral assays for anxiety and panic. *Neuroscience and Biobehavioral Reviews, 25*, 205-218.

Block, J. (1995). A contrarian view of the five-factor approach to personality description. *Psychological Bulletin, 117*, 187-215.

Bolger, N., & Eckenrode, J. (1991). Social relationships, personality, and anxiety during a major stressful event. *Journal of Personality and Social Psychology, 61*, 440-449.

Borghuis, J., Denissen, J. J., Oberski, D., Sijtsma, K., Meeus, W. H., Branje, S., . . . Bleidorn, W. (*in press*). Big Five personality stability, change, and codevelopment across adolescence and early adulthood. *Journal of Personality and Social Psychology*.

Borsook, D., Becerra, L., & Hargreaves, R. (2006). A role for fMRI in optimizing CNS drug development. *Nature Reviews. Drug Discovery, 5*, 411-424.

Bradley, M. M. (2000). Emotion and motivation. In J. T. Cacioppo, L. G. Tassinary & G. G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed., pp. 602-642). NY: Cambridge University Press.

Bradley, M. M., & Lang, P. J. (2000). Measuring emotion: Behavior, feeling, and physiology. In R. D. Lane & L. Nadel (Eds.), *Cognitive neuroscience of emotion* (pp. 242-276). NY: Oxford University Press.

Bradley, M. M., & Lang, P. J. (2007). Emotion and motivation. In J. T. Cacioppo, L. G. Tassinary & G. G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 581-607). NY: Cambridge University Press.

Button, K. S., Ioannidis, J. P., Mokrysz, C., Nosek, B. A., Flint, J., Robinson, E. S., & Munafo, M. R. (2013a). Confidence and precision increase with high statistical power. *Nature Reviews. Neuroscience, 14*, 585-586.

Button, K. S., Ioannidis, J. P., Mokrysz, C., Nosek, B. A., Flint, J., Robinson, E. S., & Munafo, M. R. (2013b). Power failure: why small sample size undermines the reliability of neuroscience. *Nature Reviews. Neuroscience, 14*, 365-376.

Cacioppo, S., Grippo, A. J., London, S., Goossens, L., & Cacioppo, J. T. (2015). Loneliness: clinical import and interventions. *Perspect Psychol Sci, 10*, 238-249.

Calhoon, G. G., & Tye, K. M. (2015). Resolving the neural circuits of anxiety. *Nature Neuroscience, 18*, 1394-1404.

Casey, B. J., Craddock, N., Cuthbert, B. N., Hyman, S. E., Lee, F. S., & Ressler, K. J. (2013). DSM-5 and RDoC: progress in psychiatry research? *Nature Reviews. Neuroscience, 14*, 810-814.

Cavanagh, J. F., & Shackman, A. J. (2015). Frontal midline theta reflects anxiety and cognitive control: Meta-analytic evidence. *Journal of Physiology, Paris, 109*, 3-15.

Chambers, C. (2017). *The seven deadly sins of psychology: A manifesto for reforming the culture of scientific practice*. Princeton, NJ: Princeton University Press.

Chronis-Tuscano, A., Rubin, K. H., O'Brien, K. A., Coplan, R. J., Thomas, S. R., Dougherty, L. R., . . . Wimsatt, M. (2015). Preliminary evaluation of a multimodal early intervention program for behaviorally inhibited preschoolers. *Journal of Consulting and Clinical Psychology, 83*, 534-540.

Clark, L. A., & Watson, D. (1988a). Mood and the mundane: Relations between daily life events and self-reported mood. *Journal of Personality and Social Psychology, 54*, 296-308.

Clark, L. A., & Watson, D. (1988b). Mood and the mundane: relations between daily life events and self-reported mood. *Journal of Personality and Social Psychology, 54*, 296-308.

Clore, G. L. (1994). Why emotions require cognition. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 181-191). New York: Oxford University Press.

Coan, J. A., & Allen, J. J. B. (2007). *Handbook of emotion elicitation and assessment*. NY: Oxford University Press.

Coan, J. A., & Sbarra, D. A. (2015). Social baseline theory: The social regulation of risk and effort. *Curr Opin Psychol, 1*, 87-91.

Cohen, J. (1962). The statistical power of abnormal-social psychological research: A review. *J. Abnorm. Soc. Psychol., 65*, 145–153.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*, 155-159.

Collins, P. Y., Patel, V., Joestl, S. S., March, D., Insel, T. R., Daar, A. S., . . . Stein, D. J. (2011). Grand challenges in global mental health. *Nature, 475*, 27-30.

Coltheart, M. (2013). How can functional neuroimaging inform cognitive theories? *Perspectives on Psychological Science, 8*, 98–103.

Cox, R. W., Chen, G., Glen, D. R., Reynolds, R. C., & Taylor, P. A. (2017). fMRI clustering and false-positive rates. *Proceedings of the National Acadademy of Sciences USA, 114*, E3370-3371.

Cramer, A. O. J., van der Sluis, S., Noordhof, A., Wichers, M., Geschwind, N., Aggen, S. H., . . . Borsboom, D. (2012a). Dimensions of normal personality as networks in search of equilibrium: You can't like parties if you don't like people. *European Journal of Personality, 26*, 414-431.

Cramer, A. O. J., van der Sluis, S., Noordhof, A., Wichers, M., Geschwind, N., Aggen, S. H., . . . Borsboom, D. (2012b). Measurable like temperature or mereological like flocking? On the nature of personality traits. *European Journal of Personality, 26*, 451-459.

Creed, A. T., & Funder, D. C. (1998). Social anxiety: from the inside and outside. *Personality and Individual Differences, 25*, 19-33.

Culverhouse, R. C., Saccone, N. L., Horton, A. C., Ma, Y., Anstey, K. J., Banaschewski, T., . . . Bierut, L. J. (*in press*). Collaborative meta-analysis finds no evidence of a strong interaction between stress and 5-HTTLPR genotype contributing to the development of depression. *Molecular Psychiatry*.

Dan-Glauser, E. S., & Scherer, K. R. (2011). The Geneva affective picture database (GAPED): a new 730-picture database focusing on valence and normative significance. *Behavior Research Methods, 43*, 468-477.

Davidson, R. J. (1993). Cerebral asymmetry and emotion: Conceptual and methodological conundrums. *Cognition & Emotion, 7*(1), 115-138.

Davidson, R. J. (1994). Complexities in the search for emotion-specific physiology. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 237-242). New York: Oxford University Press.

Davidson, R. J. (1998). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition & Emotion, 12*, 307-330.

Davidson, R. J., & Harrington, A. (Eds.). *Visions of compassion: Western scientists and Tibetan Buddhists examine human nature*. New York: Oxford University Press.

Davidson, R. J., & Kaszniak, A. W. (2015). Conceptual and methodological issues in research on mindfulness and meditation. *American Psychologist, 70*, 581-592.

Davidson, R. J., & McEwen, B. S. (2012). Social influences on neuroplasticity: stress and interventions to promote well-being. *Nature Neuroscience, 15*, 689-695.

Davidson, R. J., & Sutton, S. K. (1995). Affective neuroscience: the emergence of a discipline. *Current Opinion in Neurobiology, 5*, 217-224.

Davis, M., Walker, D. L., Miles, L., & Grillon, C. (2010). Phasic vs sustained fear in rats and humans: Role of the extended amygdala in fear vs anxiety. *Neuropsychopharmacology, 35*, 105-135.

Davis, M., & Whalen, P. J. (2001). The amygdala: vigilance and emotion. *Molecular Psychiatry, 6*, 13-34.

de la Vega, A., Chang, L. J., Banich, M. T., Wager, T. D., & Yarkoni, T. (2016). Large-scale meta-analysis of human medial frontal cortex reveals tripartite functional organization. *Journal of Neuroscience, 36*, 6553-6562.

de Sousa, R. (2014). Emotion. In E. N. Zalta (Ed.), *Stanford encyclopedia of philosophy*.

Desai, M., Kahn, I., Knoblich, U., Bernstein, J., Atallah, H., Yang, A., . . . Boyden, E. S. (2011). Mapping brain networks in awake mice using combined optical neural control and fMRI. *Journal of Neurophysiology, 105*, 1393-1405.

DiLuca, M., & Olesen, J. (2014). The cost of brain diseases: a burden or a challenge? *Neuron, 82*, 1205-1208.

Duff, E. P., Vennart, W., Wise, R. G., Howard, M. A., Harris, R. E., Lee, M., . . . Smith, S. M. (2015). Learning to identify CNS drug action and efficacy using multistudy fMRI data. *Sci Transl Med, 7*, 274ra216.

Editors, T. P. M. (2010). Social relationships are key to health, and to health policy. *PLoS Med, 7*, 2.

Eglen, S. J., Marwick, B., Halchenko, Y. O., Hanke, M., Sufi, S., Gleeson, P., . . . Poline, J. B. (2017). Toward standard practices for sharing computer code and programs in neuroscience. *Nature Neuroscience, 20*, 770-773.

Eklund, A., Nichols, T. E., & Knutsson, H. (2016). Cluster failure: Why fMRI inferences for spatial extent have inflated false-positive rates. *Proceedings of the National Academy of Sciences of the United States of America, 113*, 7900-7905.

Eklund, A., nichols, T. E., & Knutsson, H. (2017). Reply to Brown and Behrmann, Cox, et al., and Kessler et al.: Data and code sharing is the way forward for fMRI. *Proceedings of the National Acadademy of Sciences USA, 114*, E3374-3375.

Ekman, P. (1977). Biological and cultural contributions to body and facial movement. In J. Blacking (Ed.), *The anthropology of the body* (pp. 39-84). London: Academic Press.

Ekman, P. (1994a). All emotions are basic. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 15-19). New York: Oxford University Press.

Ekman, P. (1994b). Antecedant events and emotion metaphors. In R. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 146-149). New York: Oxford University Press.

Ekman, P., & Cordaro, D. (2011). What is meant by calling emotions basic. *Emotion Review, 3*, 364-370.

Ekman, P., & Davidson, R. J. (1994). Affective science: A research agenda. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 411-430). New York: Oxford University Press.

Ekman, P., & Friesen, W. V. (1975). *Unmasking the face*. Englewood Cliffs, NJ: Prentice-Hall.

Engber, D. (2017). Daryl Bem proved ESP is real. Which means science is broken. *Slate*. http://redux.slate.com/cover-stories/2017/05/daryl-bem-proved-esp-is-real-showed-science-is-broken.html

Fadok, J. P., Krabbe, S., Markovic, M., Courtin, J., Xu, C., Massi, L., . . . Luthi, A. (2017). A competitive inhibitory circuit for selection of active and passive fear responses. *Nature, 542*, 96-100.

Fanselow, M. S., & Lester, L. S. (1988). A functional behavioristic approach to aversively motivated behavior: Predatory imminence as a determinant of the topography of defensive behavior. In R. C. Bolles & M. D. Beecher (Eds.), *Evolution and learning* (pp. 185-211). Hillsdale, NJ: Erlbaum.

Faull, O. K., & Pattinson, K. T. (2017). The cortical connectivity of the periaqueductal gray and the conditioned response to the threat of breathlessness. *Elife, 6*.

Fehr, E., & Schmidt, K. M. (1999). A theory of fairness, competition, and cooperation. *Q J Econ, 114*, 817-868.

Feynman, R. P. (1974). Cargo cult science [1974 CalTech Commencement Address]. *Engineering and Science, 37*, 10-13.

Forbes, E. E., Hariri, A. R., Martin, S. L., Silk, J. S., Moyles, D. L., Fisher, P. M., . . . Dahl, R. E. (2009). Altered striatal activation predicting real-world positive affect in adolescent major depressive disorder. *American Journal of Psychiatry, 166*, 64-73.

Fox, A. S., & Kalin, N. H. (2014). A translational neuroscience approach to understanding the development of social anxiety disorder and its pathophysiology. *American Journal of Psychiatry, 171*, 1162-1173.

Fox, A. S., & Shackman, A. J. (*in press*). The central extended amygdala in fear and anxiety: Closing the gap between mechanistic and neuroimaging research. *Neuroscience Letters*.

Fox, A. S., Shelton, S. E., Oakes, T. R., Converse, A. K., Davidson, R. J., & Kalin, N. H. (2010). Orbitofrontal cortex lesions alter anxiety-related activity in the primate bed nucleus of stria terminalis. *Journal of Neuroscience, 30*, 7023-7027.

Fraley, R. C., & Roberts, B. W. (2005). Patterns of continuity: a dynamic model for conceptualizing the stability of individual differences in psychological constructs across the life course. *Psychological Review, 112*, 60-74.

Frijda, N. (1994a). Emotions are functional, most of the time. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 112-122). New York: Oxford University Press.

Frijda, N. (1994b). Emotions require cognitions, even if simple ones. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 197-202). New York: Oxford University Press.

Frijda, N. (1994c). Universal antecedants exist, and are interesting. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 155-162). New York: Oxford University Press.

Furmark, T., Tillfors, M., Marteinsdottir, I., Fischer, H., Pissiota, A., Langstrom, B., & Fredrikson, M. (2002). Common changes in cerebral blood flow in patients with social phobia treated with citalopram or cognitive-behavioral therapy. *Archives of General Psychiatry, 59*, 425-433.

Gilbert, D. T., King, G., Pettigrew, S., & Wilson, T. D. (2016). Comment on "Estimating the reproducibility of psychological science". *Science, 351*, 1037.

Gilmore, R. O. (2016). From big data to deep insight in developmental science. *Wiley Interdiscip Rev Cogn Sci, 7*, 112-126.

Glasser, M. F., Smith, S. M., Marcus, D. S., Andersson, J. L., Auerbach, E. J., Behrens, T. E., . . . Van Essen, D. C. (2016). The Human Connectome Project's neuroimaging approach. *Nature Neuroscience, 19*, 1175-1187.

Gosling, S. D., & Mason, W. (2015). Internet research in psychology. *Annual Review of Psychology, 66*, 877-902.

Grayson, D. S., Bliss-Moreau, E., Machado, C. J., Bennett, J., Shen, K., Grant, K. A., . . . Amaral, D. G. (2016). The rhesus monkey connectome predicts disrupted functional networks resulting from pharmacogenetic inactivation of the amygdala. *Neuron, 91*, 453-466.

Gross, J. J. (Ed.). (2014). *Handbook of emotion regulation* (2nd ed.). New York: Guilford.

Gross, J. J., Richards, J. M., & John, O. P. (2006). Emotion regulation in everyday life. In D. K. Snyder, J. A. Simpson & J. N. Hughes (Eds.), *Emotion regulation in families: Pathways to dysfunction and health*. Washington, DC: American Psychological Association.

Grupe, D. W., & Nitschke, J. B. (2013). Uncertainty and anticipation in anxiety: an integrated neurobiological and psychological perspective. *Nature Reviews. Neuroscience, 14*, 488-501.

Gungor, N. Z., & Paré, D. (2016). Functional heterogeneity in the bed nucleus of the stria terminalis. *Journal of Neuroscience, 36*, 8038-8049.

Hakulinen, C., Elovainio, M., Pulkki-Raback, L., Virtanen, M., Kivimaki, M., & Jokela, M. (2015). Personality and depressive symptoms: Individual participant meta-analysis of 10 cohort studies. *Depression and Anxiety, 32*, 461-470.

Havermans, R. C. (2011). "You Say it's Liking, I Say it's Wanting ...". On the difficulty of disentangling food reward in man. *Appetite, 57*, 286-294.

Heller, A. S., Fox, A. S., Wing, E., Mayer, K., Vack, N. J., & Davidson, R. J. (2015). The neurodynamics of affect in the laboratory predicts persistence of real-world emotional responses. *Journal of Neuroscience, 35*, 10503-10509.

Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world? *Behavioral and Brain Sciences, 33*, 61-83; discussion 83-135.

Hofmann, S. G., & Smits, J. A. (2008). Cognitive-behavioral therapy for adult anxiety disorders: a meta-analysis of randomized placebo-controlled trials. *Journal of Clinical Psychiatry, 69*, 621-632.

House, P. K., Vyas, A., & Sapolsky, R. (2011). Predator cat odors activate sexual arousal pathways in brains of Toxoplasma gondii infected rats. *PLoS ONE, 6*, e23277.

Hyde, J. S., Mezulis, A. H., & Abramson, L. Y. (2008). The ABCs of depression: Integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. *Psychological Review, 115*, 291-313.

Hyman, S. E. (2016). Back to basics: luring industry back into neuroscience. *Nature Neuroscience, 19*, 1383-1384.

Iacono, W. G., Vaidyanathan, U., Vrieze, S. I., & Malone, S. M. (2014). Knowns and unknowns for psychophysiological endophenotypes: integration and response to commentaries. *Psychophysiology, 51*, 1339-1347.

Institute of Medicine. (2001). *Exploring the biological contributions to human health: Does sex matter?* Washington, DC: National Academy Press.

Janak, P. H., & Tye, K. M. (2015). From circuits to behaviour in the amygdala. *Nature, 517*, 284-292.

Kagan, J. (1988). The meanings of personality predicates. *American Psychologist, 43*, 614-620.

Kagan, J. (2010). Some plain words on emotion. *Emotion Review, 3*, 221-224.

Kagan, J. (2016a). An overly permissive extension. *Perspect Psychol Sci, 11*, 442-450.

Kagan, J. (2016b). Why stress remains an ambiguous concept. *Perspectives on Psychological Science, 11*, 464-465.

Kagan, J. (*in press*). Brain and emotion. *Emotion Review*.

Kahneman, D., & Tversky, A. (1979). Prospect theory: An analysis of decision under risk. *Econometrica, 47*, 263-292.

Kalin, N. H., Fox, A. S., Kovner, R., Riedel, M. K., Fekete, E. M., Roseboom, P. H., . . . Oler, J. A. (2016). Overexpressing corticotropin-releasing hormone in the primate amygdala increases anxious temperament and alters its neural circuit. *Biological Psychiatry, 80*, 345-355.

Kalin, N. H., & Shelton, S. E. (1989). Defensive behaviors in infant rhesus monkeys: environmental cues and neurochemical regulation. *Science, 243*, 1718-1721.

Kelly, E. L. (1927). *Interpretation of educational measurements*. Yonkers, NY: World Book.

Kendler, K. S. (2012a). The dappled nature of causes of psychiatric illness: replacing the organic-functional/hardware-software dichotomy with empirically based pluralism. *Molecular Psychiatry, 17*, 377-388.

Kendler, K. S. (2012b). Levels of explanation in psychiatric and substance use disorders: implications for the development of an etiologically based nosology. *Molecular Psychiatry, 17*(1), 11-21.

Kendler, K. S. (2016). The phenomenology of major depression and the representativeness and nature of DSM criteria. *American Journal of Psychiatry, 173*, 771-780.

Kendler, K. S., & Halberstadt, L. J. (2013). The road not taken: life experiences in monozygotic twin pairs discordant for major depression. *Molecular Psychiatry, 18*, 975-984.

Khalsa, S. S., Feinstein, J. S., Li, W., Feusner, J. D., Adolphs, R., & Hurlemann, R. (2016). Panic anxiety in humans with bilateral amygdala lesions: Pharmacological induction via cardiorespiratory interoceptive pathways. *Journal of Neuroscience, 36*, 3559-3566.

Kim, C. K., Adhikari, A., & Deisseroth, K. (2017). Integration of optogenetics with complementary methodologies in systems neuroscience. *Nature Reviews Neuroscience, 18*, 222-235.

Kok, B. E., & Singer, T. (2017). Effects of contemplative dyads on engagement and perceived social connectedness over 9 months of mental training: A randomized clinical trial. *JAMA Psychiatry, 74*, 126-134.

Koolhaas, J. M., Bartolomucci, A., Buwalda, B., de Boer, S. F., Flugge, G., Korte, S. M., . . . Fuchs, E. (2011). Stress revisited: a critical evaluation of the stress concept. *Neuroscience and Biobehavioral Reviews, 35*, 1291-1301.

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., . . . Zimmerman, M. (2017). The hierarchical taxonomy of psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology, 126*, 454-477.

Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH research domain criteria initiative: Background, issues, and pragmatics. *Psychophysiology, 53*, 286-297.

Krakauer, J. W., Ghazanfar, A. A., Gomez-Marin, A., MacIver, M. A., & Poeppel, D. (2017). Neuroscience needs behavior: Correcting a reductionist bias. *Neuron, 93*, 480-490.

Kramer, A. D., Guillory, J. E., & Hancock, J. T. (2014). Experimental evidence of massive-scale emotional contagion through social networks. *Proceedings of the National Academy of Sciences of the United States of America, 111*, 8788-8790.

Kringelbach, M. L., & Berridge, K. C. (2012). The joyful mind. *Scientific American, 307*, 40-45.

Laidlaw, K. E., Foulsham, T., Kuhn, G., & Kingstone, A. (2011). Potential social interactions are important to social attention. *Proceedings of the National Academy of Sciences of the United States of America, 108*, 5548-5553.

Lakens, D. (2017). Impossibly hungry judges. *The 20% statistician*.

Lapate, R. C., Samaha, J., Rokers, B., Hamzah, H., Postle, B. R., & Davidson, R. J. (*in press*). Inhibition of lateral prefrontal cortex produces emotionally biased first impressions: A transcranial magnetic stimulation and electroencephalography study. *Psychol Sci*, 956797617699837.

Lazarus, R. S. (1991). Cognition and motivation in emotion. *American Psychologist, 46*, 352-367.

Lazarus, R. S. (1994a). Appraisal: The long and the short of it. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 208-215). New York: Oxford University Press.

Lazarus, R. S. (1994b). Universal antecedants of emotion. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 163-171). New York: Oxford University Press.

LeDoux, J. E. (1994). Emotional processing, but not emotions, can occur unconsciously. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion. Fundamental questions* (pp. 291-292). New York: Oxford University Press.

LeDoux, J. E. (2012). Rethinking the emotional brain. *Neuron, 73*, 653-676.

LeDoux, J. E. (2014). Coming to terms with fear. *Proceedings of the National Acadademy of Sciences U S A, 111*, 2871-2878.

LeDoux, J. E. (2015). *Anxious. Using the brain to understand and treat fear and anxiety*. NY: Viking.

Ledoux, J. E. (*in press*). Semantics, surplus meaning, and the science of fear. *Trends in Cognitive Sciences*.

LeDoux, J. E., & Brown, R. (2017). A higher-order theory of emotional consciousness. *Proceedings of the National Academy of Sciences of the United States of America, 114*, E2016-E2025.

Levenson, R. W. (2011). Basic emotion questions. *Emotion Review, 3*, 379-386.

Lim, S. L., Padmala, S., & Pessoa, L. (2009). Segregating the significant from the mundane on a moment-to-moment basis via direct and indirect amygdala contributions. *Proceedings of the National Academy of Sciences of the United States of America, 106*, 16841-16846.

Lopez, R. B., Hofmann, W., Wagner, D. D., Kelley, W. M., & Heatherton, T. F. (2014). Neural predictors of giving in to temptation in daily life. *Psychol Sci, 25*(7), 1337-1344.

Luck, S. J. (2005). Ten simple rules for designing ERP experiments. In T. C. Handy (Ed.), *Event-related potentials: A methods handbook* (pp. 17-32). Cambridge, MA: MIT Press.

Lutz, A., Slagter, H. A., Dunne, J. D., & Davidson, R. J. (2008). Attention regulation and monitoring in meditation. *Trends Cogn Sci, 12*, 163-169.

Marroquin, B. (2011). Interpersonal emotion regulation as a mechanism of social support in depression. *Clinical Psychology Review, 31*, 1276-1290.

Marx, M. H. (1951). Intervening variable or hypothetical construct. *Psychological Review, 58*, 235-247.

Mauss, I. B., Levenson, R. W., McCarter, L., Wilhelm, F. H., & Gross, J. J. (2005). The tie that binds? Coherence among emotion experience, behavior, and physiology. *Emotion, 5*, 175-190.

McLaughlin, K. A., Busso, D. S., Duys, A., Green, J. G., Alves, S., Way, M., & Sheridan, M. A. (2014). Amygdala response to negative stimuli predicts PTSD symptom onset following a terrorist attack. *Depression and Anxiety, 31*, 834-842.

Merton, R. K. (1942/1973). The normative structure of science. In R. K. Merton (Ed.), *The sociology of science: Theoretical and empirical investigations* (pp. 267-278). Chicago: University of Chicago Press.

Milojev, P., & Sibley, C. G. (2017). Normative personality trait development in adulthood: A 6-year cohort-sequential growth model. *Journal of Personality and Social Psychology, 112*, 510-526.

Mobbs, D., Petrovic, P., Marchant, J. L., Hassabis, D., Weiskopf, N., Seymour, B., . . . Frith, C. D. (2007). When fear is near: Threat imminence elicits prefrontal-periaqueductal gray shifts in humans. *Science, 317*, 1079-1083.

Morrison, F. J., & Grammer, J. K. (2016). Conceptual clutter and measurement mayhem: Proposals for cross-disciplinary integration in conceptualizing and measuring executive function. In J. A. Griffin, P. McCardle & L. S. Freund (Eds.), *Executive function in preschool-age children: Integrating measurement, neurodevelopment, and translational research* (pp. 327–348). Washington, DC: American Psychological Association.

Motyl, M., Demos, A. P., Carsel, T. S., Hanson, B. E., Melton, Z. J., Mueller, A. B., . . . Skitka, L. J. (2017). The state of social and personality science: Rotten to the core, not so bad, getting better, or getting worse? *Journal of Personality and Social Psychology, 113*, 34-58.

Motzkin, J. C., Philippi, C. L., Oler, J. A., Kalin, N. H., Baskaya, M. K., & Koenigs, M. (2015). Ventromedial prefrontal cortex damage alters resting blood flow to the bed nucleus of stria terminalis. *Cortex, 64*, 281-288.

Motzkin, J. C., Philippi, C. L., Wolf, R. C., Baskaya, M. K., & Koenigs, M. (2014). Ventromedial prefrontal cortex lesions alter neural and physiological correlates of anticipation. *Journal of Neuroscience, 34*(31), 10430-10437.

Motzkin, J. C., Philippi, C. L., Wolf, R. C., Baskaya, M. K., & Koenigs, M. (2015). Ventromedial prefrontal cortex is critical for the regulation of amygdala activity in humans. *Biological Psychiatry, 77*(3), 276-284.

Munafò, M. R., Nosek, B. A., Bishop, D. V. M., Button, K. S., Chambers, C. D., du Sert, N. P., . . . Ioannidis, J. P. A. (2017). A manifesto for reproducible science. *Nature Human Behaviour, 1*, 21.

Myers, D. G. (1999). Close relationships and quality of life. In D. Kahneman, E. Diener & N. Schwartz (Eds.), *Well-being* (pp. 374-391). NY: Sage.

Namburi, P., Al-Hasani, R., Calhoon, G. G., Bruchas, M. R., & Tye, K. M. (2016). Architectural representation of valence in the limbic system. *Neuropsychopharmacology, 41*, 1697-1715.

Nelson, L. D., Simmons, J., & Simonsohn, U. (*in press*). Psychology's renaissance. *Annual Review of Psychology*.

Nelson, R. J., & Trainor, B. C. (2007). Neural mechanisms of aggression. *Nature Reviews. Neuroscience, 8*, 536-546.

Nichols, T. E., Das, S., Eickhoff, S. B., Evans, A. C., Glatard, T., Hanke, M., . . . Yeo, B. T. (2017). Best practices in data analysis and sharing in neuroimaging using MRI. *Nature Neuroscience, 20*, 299-303.

Nigg, J. T. (2017). On the relations among self-regulation, self-control, executive functioning, effortful control, cognitive control, impulsivity, risk-taking, and inhibition for developmental psychopathology. *Journal of Child Psychology and Psychiatry, 58*, 361-383.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*, 400-424.

Nuzzo, R. (2015). How scientists fool themselves-and how they can stop. *Nature, 526*, 182-185.

Nye, C. D., Allemand, M., Gosling, S. D., Potter, J., & Roberts, B. W. (2016). Personality trait differences between young and middle-aged adults: Measurement artifacts or actual trends? *Journal of Personality, 84*, 473-492.

Okbay, A., & Rietveld, C. A. (2015). On improving the credibility of candidate gene studies: A review of candidate gene studies published in Emotion. *Emotion, 15*, 531-537.

Okon-Singer, H., Hendler, T., Pessoa, L., & Shackman, A. J. (2015). The neurobiology of emotion-cognition interactions: Fundamental questions and strategies for future research. *Frontiers in Human Neuroscience, 9*.

Olderbak, S., Hildebrandt, A., Pinkpank, T., Sommer, W., & Wilhelm, O. (2014). Psychometric challenges and proposed solutions when scoring facial emotion expression codes. *Behav Res Methods, 46*, 992-1006.

Onnela, J. P., & Rauch, S. L. (2016). Harnessing smartphone-based digital phenotyping to enhance behavioral and mental health. *Neuropsychopharmacology, 41*, 1691-1696.

Palmers, K. M. (2016). Psychology is in crisis over whether it's in crisis. *Wired*. https://www.wired.com/2016/03/psychology-crisis-whether-crisis/

Panksepp, J. (1992). A critical role for "affective neuroscience" in resolving what is basic about basic emotions. *Psychological Review, 99*, 554-560.

Panksepp, J. (1998). *Affective neuroscience. The foundations of human and animal emotions*. New York: Oxford University Press.

Passingham, R. E., Stephan, K. E., & Kotter, R. (2002). The anatomical basis of functional localization in the cortex. *Nature Reviews. Neuroscience, 3*, 606-616.

Paulus, M. P. (2015). Pragmatism instead of mechanism: A call for impactful biological psychiatry. *JAMA Psychiatry, 72*, 631-632.

Paulus, M. P., Feinstein, J. S., Castillo, G., Simmons, A. N., & Stein, M. B. (2005). Dose-dependent decrease of activation in bilateral amygdala and insula by lorazepam during emotion processing. *Archives of General Psychiatry, 62*, 282-288.

Pearson, J. M., Watson, K. K., & Platt, M. L. (2014). Decision making: the neuroethological turn. *Neuron, 82*, 950-965.

Pereira, A. C., Huddleston, D. E., Brickman, A. M., Sosunov, A. A., Hen, R., McKhann, G. M., . . . Small, S. A. (2007). An in vivo correlate of exercise-induced neurogenesis in the adult dentate gyrus. *Proceedings of the National Academy of Sciences of the United States of America, 104*, 5638-5643.

Perez-Edgar, K., McDermott, J. N., Korelitz, K., Degnan, K. A., Curby, T. W., Pine, D. S., & Fox, N. A. (2010). Patterns of sustained attention in infancy shape the developmental trajectory of social behavior from toddlerhood through adolescence. *Developmental Psychology, 46*, 1723-1730.

Pessoa, L. (2017). A network model of the emotional brain. *Trends in Cognitive Sciences, 21*, 357-371.

Pfeiffer, U. J., Vogeley, K., & Schilbach, L. (2013). From gaze cueing to dual eye-tracking: novel approaches to investigate the neural correlates of gaze in social interaction. *Neuroscience and Biobehavioral Reviews, 37*, 2516-2528.

Poldrack, R. A. (2016). Why preregistration no longer makes me nervous. http://www.russpoldrack.org/2016/09/why-preregistration-no-longer-makes-me.html

Poldrack, R. A., Baker, C. I., Durnez, J., Gorgolewski, K. J., Matthews, P. M., Munafo, M. R., . . . Yarkoni, T. (2017). Scanning the horizon: towards transparent and reproducible neuroimaging research. *Nature Reviews. Neuroscience, 18*, 115-126.

Poldrack, R. A., & Yarkoni, T. (2016). From brain maps to cognitive ontologies: Informatics and the search for mental structure. *Annual Review of Psychology, 67*, 587-612.

Reeck, C., Ames, D. R., & Ochsner, K. N. (2016). The social regulation of emotion: An integrative, cross-disciplinary model. *Trends Cogn Sci, 20*, 47-63.

Reynolds, S. M., & Berridge, K. C. (2008). Emotional environments retune the valence of appetitive versus fearful functions in nucleus accumbens. *Nature Neuroscience, 11*, 423-425.

Rime, B. (2009). Emotion elicits the social sharing of emotion: Theory and empirical review. *Emotion Review, 1*, 60-85.

Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: a quantitative review of longitudinal studies. *Psychological Bulletin, 126*, 3-25.

Roberts, B. W., Luo, J., Briley, D. A., Chow, P. I., Su, R., & Hill, P. L. (2017). A systematic review of personality trait change through intervention. *Psychological Bulletin, 143*, 117-141.

Roberts, B. W., & Mroczek, D. (2008). Personality trait change in adulthood. *Curr Dir Psychol Sci, 17*, 31-35.

Roberts, B. W., Walton, K. E., & Viechtbauer, W. (2006). Patterns of mean-level change in personality traits across the life course: a meta-analysis of longitudinal studies. *Psychological Bulletin, 132*, 1-25.

Romero, L. M., Platts, S. H., Schoech, S. J., Wada, H., Crespi, E., Martin, L. B., & Buck, C. L. (2015). Understanding stress in the healthy animal - potential paths for progress. *Stress, 18*, 491-497.

Rozin, P., Haidt, J., & McCauley, C. R. (2008). Disgust. In M. Lewis, J. M. Haviland-Jones & L. F. Barrett (Eds.), *Handbook of emotions* (pp. 757-776). New York: Guilford Press.

Saffran, J. R. (2003). Statistical language learning: Mechanisms and constraints. *Current Directions in Psychological Science, 12*, 110-114.

Sahay, A., & Hen, R. (2007). Adult hippocampal neurogenesis in depression. *Nature Neuroscience, 10*, 1110-1115.

Samson, A. C., Kreibig, S. D., Soderstrom, B., Wade, A. A., & Gross, J. J. (2015). Eliciting positive, negative and mixed emotional states: A film library for affective scientists. *Cognition and Emotion, 30*, 827-856.

Sano, A., Phillips, A. J., Yu, A. Z., McHill, A. W., Taylor, S., Jaques, N., . . . Picard, R. W. (2015). *Recognizing academic performance, sleep quality, stress level, and mental health using personality traits, wearable sensors and mobile phones.* Paper presented at the 12th International IEEE Conference on Wearable and Implantable Body Sensor Networks.

Santarelli, L., Saxe, M., Gross, C., Surget, A., Battaglia, F., Dulawa, S., . . . Hen, R. (2003). Requirement of hippocampal neurogenesis for the behavioral effects of antidepressants. *Science, 301*, 805-809.

Schaafsma, S. M., Pfaff, D. W., Spunt, R. P., & Adolphs, R. (2015). Deconstructing and reconstructing theory of mind. *Trends Cogn Sci, 19*, 65-72.

Scherer, K. R. (2009). The dynamic architecture of emotion: Evidence for the component process model. *Cognition and Emotion, 23*, 1307-1351.

Scherer, K. R., Schorr, A., & Johnstone, T. (Eds.). (2001). *Appraisal processes in emotion: Theory, methods, research*. New York: Oxford University Press.

Schmitter, A. M. (2014). 17th and 18th century theories of emotions. In E. N. Zalta (Ed.), *Stanford encyclopedia of philosophy*.

Schnyer, D. M., Beevers, C. G., deBettencourt, M. T., Sherman, S. M., Cohen, J. D., Norman, K. A., & Turk-Browne, N. B. (2015). Neurocognitive therapeutics: from concept to application in the treatment of negative attention bias. *Biol Mood Anxiety Disord, 5*, 1.

Schwartz, S. J., Lilienfeld, S. O., Meca, A., & Sauvigné, K. C. (2016). The role of neuroscience within psychology: A call for inclusiveness over exclusiveness. *American Psychologist, 71*, 52-70.

Senn, V., Wolff, S. B., Herry, C., Grenier, F., Ehrlich, I., Grundemann, J., . . . Luthi, A. (2014). Long-range connectivity defines behavioral specificity of amygdala neurons. *Neuron, 81*, 428-437.

Shackman, A. J., & Fox, A. S. (2016a). Contributions of the central extended amygdala to fear and anxiety. *Journal of Neuroscience, 36*, 8050-8063.

Shackman, A. J., & Fox, A. S. (2016b). Contributions of the central extended amygdala to fear and anxiety. *Journal of Neuroscience, 36*, 8050-8063.

Shackman, A. J., Fox, A. S., Oler, J. A., Shelton, S. E., Davidson, R. J., & Kalin, N. H. (2013). Neural mechanisms underlying heterogeneity in the presentation of anxious temperament. *Proceedings of the National Academy of Sciences of the United States of America, 110*, 6145-6150.

Shackman, A. J., Kaplan, C. M., Stockbridge, M. D., Tillman, R. M., Tromp, D. P. M., Fox, A. S., & Gamer, M. (2016). The neurobiology of anxiety and attentional biases to threat: Implications for understanding anxiety disorders in adults and youth. *Journal of Experimental Psychopathology, 7*, 311-342.

Shackman, A. J., Maxwell, J. S., McMenamin, B. W., Greischar, L. L., & Davidson, R. J. (2011). Stress potentiates early and attenuates late stages of visual processing. *Journal of Neuroscience, 31*, 1156-1161.

Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews. Neuroscience, 12*, 154-167.

Shackman, A. J., Sarinopoulos, I., Maxwell, J. S., Pizzagalli, D. A., Lavric, A., & Davidson, R. J. (2006). Anxiety selectively disrupts visuospatial working memory. *Emotion, 6*, 40-61.

Shackman, A. J., Tromp, D. P. M., Stockbridge, M. D., Kaplan, C. M., Tillman, R. M., & Fox, A. S. (2016). Dispositional negativity: An integrative psychological and neurobiological perspective. *Psychological Bulletin, 142*, 1275-1314.

Shackman, A. J., Weinstein, J. S., Hudja, S. N., Bloomer, C. D., Barstead, M. G., Fox, A. S., & Lemay, E. P. (*in press*). Dispositional negativity in the wild: Social environment governs momentary emotional experience. *Emotion*.

Sharma, L., Markon, K. E., & Clark, L. A. (2014). Toward a theory of distinct types of "impulsive" behaviors: A meta-analysis of self-report and behavioral measures. *Psychological Bulletin, 140*, 374-408.

Shrout, P. E., & Rodgers, J. L. (*in press*). Psychology, science, and knowledge construction: Broadening perspectives from the replication crisis. *Annual Review of Psychology*.

Sitaram, R., Ros, T., Stoeckel, L., Haller, S., Scharnowski, F., Lewis-Peacock, J., . . . Sulzer, J. (2017). Closed-loop brain training: the science of neurofeedback. *Nature Reviews. Neuroscience, 18*, 86-100.

Somerville, L. H., Whalen, P. J., & Kelley, W. M. (2010). Human bed nucleus of the stria terminalis indexes hypervigilant threat monitoring. *Biological Psychiatry, 68*, 416-424.

Soto, C. J., & John, O. P. (*in press*). The next Big Five Inventory (BFI-2): Developing and assessing a hierarchical model with 15 Facets to enhance bandwidth, fidelity, and predictive power. *Journal of Personality and Social Psychology*.

Spellman, B. A. (2015). A short (personal) future history of revolution 2.0 *Perspectives on Psychological Science, 10*, 886-899.

Spunt, R. P., Elison, J. T., Dufour, N., Hurlemann, R., Saxe, R., & Adolphs, R. (2015). Amygdala lesions do not compromise the cortical network for false-belief reasoning. *Proceedings of the National Academy of Sciences of the United States of America, 112*, 4827-4832.

Stellar, J. E., & Keltner, D. (2014). Compassion. In M. M. Tugade, M. N. Shiota & L. D. Kirby (Eds.), *Handbook of positive emotion*. New York: Guilford Press.

Swartz, J. R., Knodt, A. R., Radtke, S. R., & Hariri, A. R. (2015). A neural biomarker of psychological vulnerability to future life stress. *Neuron, 85*(3), 505-511.

Tackett, J. L., Lilienfeld, S. O., Patrick, C. J., Johnson, S. L., Krueger, R. F., Miller, J. D., . . . Shrout, P. E. (*in press*). It's time to broaden the replicability conversation: Thoughts for and from clinical psychological science. *Perspect Psychol Sci*.

Tambini, A., Rimmele, U., Phelps, E. A., & Davachi, L. (2017). Emotional brain states carry over and enhance future memory formation. *Nature Neuroscience, 20*, 271-278.

Tang, Y. Y., Holzel, B. K., & Posner, M. I. (2015). The neuroscience of mindfulness meditation. *Nature Reviews. Neuroscience, 16*, 213-225.

Thorndike, E. L. (1904). *An introduction to the theory of mental and social measurements*. New York: Teachers College, Columbia University.

Tomarken, A. J. (1995). A psychometric perspective on psychophysiological measures. *Psychological Assessment, 7*, 387-395.

Tovote, P., Esposito, M. S., Botta, P., Chaudun, F., Fadok, J. P., Markovic, M., . . . Luthi, A. (2016). Midbrain circuits for defensive behaviour. *Nature, 534*, 206-212.

Tovote, P., Fadok, J. P., & Luthi, A. (2015). Neuronal circuits for fear and anxiety. *Nature Reviews. Neuroscience, 16*, 317-331.

Tracy, J., Klonsky, E. D., & Proudfit, G. H. (2014). How affective science can inform clinical science: An introduction to the special series on emotions and psychopathology. *Clinical Psychological Science, 2*, 371-386.

Urban, D. J., & Roth, B. L. (2015). DREADDs (designer receptors exclusively activated by designer drugs): chemogenetic tools with therapeutic utility. *Annual Review of Pharmacology and Toxicology, 55*, 399-417.

van Praag, H., Christie, B. R., Sejnowski, T. J., & Gage, F. H. (1999). Running enhances neurogenesis, learning, and long-term potentiation in mice. *Proceedings of the National Academy of Sciences of the United States of America, 96*, 13427-13431.

Vyas, A., Kim, S. K., Giacomini, N., Boothroyd, J. C., & Sapolsky, R. M. (2007). Behavioral changes induced by Toxoplasma infection of rodents are highly specific to aversion of cat odors. *Proceedings of the National Academy of Sciences of the United States of America, 104*, 6442-6447.

Wager, T. D., & Atlas, L. Y. (2015). The neuroscience of placebo effects: connecting context, learning and health. *Nature Reviews. Neuroscience, 16*, 403-418.

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin, 134*, 163-206.

Watson, D. (1988). Intraindividual and interindividual analyses of positive and negative affect: their relation to health complaints, perceived stress, and daily activities. *Journal of Personality and Social Psychology, 54*, 1020-1030.

Watson, D., Clark, L. A., McIntyre, C. W., & Hamaker, S. (1992). Affect, personality, and social activity. *Journal of Personality and Social Psychology, 63*, 1011-1025.

Watson, D., Stanton, K., & Clark, L. A. (*in press*). Self-report indicators of negative valence constructs within the research domain criteria (RDoC): A critical review. *Journal of Affective Disorders*.

Weisz, E., & Zaki, J. (*in press*). Empathy building interventions: A review of exisiting work and suggestions for future directions. In J. Doty, E. Seppala, E. Simon-Thomas, D. Cameron, S. Brown & M. Worline (Eds.), *Oxford handbook of compassion science*. New York: Oxford University Press.

Weng, H. Y., Fox, A. S., Shackman, A. J., Stodola, D. E., Caldwell, J. Z., Olson, M. C., . . . Davidson, R. J. (2013). Compassion training alters altruism and neural responses to suffering. *Psychol Sci, 24*, 1171-1180.

Whiteford, H. A., Degenhardt, L., Rehm, J., Baxter, A. J., Ferrari, A. J., Erskine, H. E., . . . Vos, T. (2013). Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *Lancet, 382*, 1575-1586.

Wilson, S. J., Smyth, J. M., & MacLean, R. R. (2014). Integrating ecological momentary assessment and functional brain imaging methods: new avenues for studying and treating tobacco dependence. *Nicotine Tob Res, 16 Suppl 2*, S102-110.

Woo, C. W., Chang, L. J., Lindquist, M. A., & Wager, T. D. (2017). Building better biomarkers: brain models in translational neuroimaging. *Nature Neuroscience, 20*, 365-377.

Wrzus, C., & Mehl, M. R. (2015). Lab and/or field? Measuring personality processes and their social consequences. *European Journal of Personality, 29*, 250-271.

Xiu, J., Zhang, Q., Zhou, T., Zhou, T. T., Chen, Y., & Hu, H. (2014). Visualizing an emotional valence map in the limbic forebrain by TAI-FISH. *Nature Neuroscience, 17*, 1552-1559.

Yarkoni, T. (2009). Big correlations in little studies: Inflated fMRI correlations reflect low statistical power. Commentary on Vul et al. (2009). *Perspectives on Psychological Science, 4*, 294-298.

Yarkoni, T., & Westfall, J. A. (*in press*). Choosing prediction over explanation in psychology: Lessons from machine learning. *Perspectives on Psychological Science*.

Yong, E. (2016). Psychology's replication crisis can't be wished away. *The Atlantic*. https://www.theatlantic.com/science/archive/2016/03/psychologys-replication-crisis-cant-be-wished-away/472272/

Zaki, J., Wager, T. D., Singer, T., Keysers, C., & Gazzola, V. (2016). The anatomy of suffering: Understanding the relationship between nociceptive and empathic pain. *Trends Cogn Sci, 20*, 249-259.

Zaki, J., & Williams, W. C. (2013). Interpersonal emotion regulation. *Emotion, 13*, 803-810.

Zald, D. H., & Treadway, M. T. (2017). Reward processing, neuroeconomics, and psychopathology. *Annu Rev Clin Psychol, 13*, 471-495.

Zhang, T.-Y., & Meaney, M. J. (2010). Epigenetics and the environmental regulation of the genome and its function. *Annual Review of Psychology, 61*(439-66).