Afterword: *How are emotions physically embodied?*

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In response to Question 1, most of the contributors seemed to agree with Darwin that emotions are functional (Darwin, 1872/2009). Thus, it may not be surprising that emotions cannot be easily separated from their physical expressions. In fact, as Salomons notes here, early theorists suggested that emotions and their somatomotor expressions were virtually one and the same. Over time, theories of emotion-body interactions have become considerably more nuanced (Damasio & Carvalho, 2013; LeDoux, 2015; Rolls, 2005). Here, we asked the contributors to discuss how the physical embodiment of emotion shapes its experience and expression. The Editors' phrasing of this question was intentionally broad to encourage varied perspectives and the contributors did not disappoint. Although they all seem to agree that physical embodiment is part of what distinguishes emotion from other psychological states, there was a diversity of opinion on the exact nature of embodiment.

All of the authors seem to agree that emotions are fundamentally embodied. Wood, Martin, & Niedenthal begin by elaborating on what has become the classical take on embodied emotions. Marshaling a range of wide variety of evidence, they argue that each of the basic emotions—fear, anger, and so on (Ekman, 1994a)—mobilize the body to respond to specific environmental challenges (Ekman, 1994b; Frijda, 1994; Lazarus, 1994), leading to emotion-specific patterns of expressive behavior and autonomic physiology. Some of the most compelling evidence for this perspective comes from their description of work by Susskind and colleagues (2008; see also Lee & Anderson's response to Question 10). Building on Darwin's argument that facial expressions confer direct benefits to the actor (Darwin, 1872/2009), Susskind et al. demonstrated that the wide eyes and flared nostrils of fear enhance the intake of visual and olfactory information, whereas the expression of disgust is associated with the reverse effects. While they acknowledge weak evidence for emotion-specificity of autonomic physiology—a conclusion consistent with evidence described in the first edition of this volume (Davidson & Ekman, 1994)—Wood et al. suggest that "the important point for an embodied perspective is that emotions do in fact regulate the [autonomic nervous system], and that they do so in a way that prepares the body to act to meet

environmental challenges." They go on to suggest that the links connecting basic emotions to the body are so strong that emotion-specific expressions often occur even when their original function is no longer relevant, as with disgust elicited by moral transgressions (Rozin, Haidt, & McCauley, 2008).

Picard weighs in on the link between emotions and their physical embodiment from a very different perspective—that of affective computing. Her goal is to build machines that can accurately de-code feelings using sensors to detect facial expressions and psychophysiological signals. Picard highlights recent efforts by her group to develop an automated means of distinguishing between two kinds of spontaneous smiles, smiles of frustration and genuine smiles of delight (Hoque, McDuff, & Picard, 2012). Consistent with other work, most participants smiled during the frustration condition and human raters were at chance when attempting to discriminate the two kinds of smiles based on photographs of the frustrated participants. Interestingly, Picard's team was able to use machine learning tools to develop an automated classifier with 92% accuracy—but only when the classifier had access to the frame-by-frame trajectory of the expression, making it possible to compute onset, sustain, and decay parameters. This and other work, both behavioral and biological, highlights the importance of assessing the full temporal dynamics of emotional expression (Blackford, Avery, Shelton, & Zald, 2009; Davidson, 1998; Ekman, 2001; Frank, Ekman, & Friesen, 1993; Schuyler et al., 2012; Tracy, Klonsky, & Proudfit, 2014).

Bradley & Lang broaden our focus by focusing on the importance of context (see also Lang & Bradley's response to Question 1). They tell us that "the bodily responses…will be determined by the actions that most effectively serve the current motivational goal," given the current context. For example, the adaptive response to a distant predator and a distant prey are the same—*stop, look, and listen*—despite eliciting diametrically opposed emotional and motivational states (i.e., defensive/withdrawal vs. appetitive/approach). Given these observations, Bradley & Lang argue that a one-to-one correspondence between emotions, behavior, and peripheral physiology is untenable. This is contrary to a strong version

of Woods and colleagues' position, but consistent with many of the responses to Question 1 (e.g., Adophs, Clore, Rolls) and other recent work (Quigley & Barrett, 2014). Put simply, for Bradley & Lang, emotional responses stem from the interaction of goals (e.g., avoid harm) and contexts (e.g., escape impossible), not emotions *per se* (e.g., fear).

Eisenberger reflects on the distinction between emotional experience and other embodied feelings, including hunger, nausea, and pain (for related accounts, see Question 1 and Lazarus, 1991). She argues that emotions differ from these more elementary drives or 'homoeostatic emotions' (Craig, 2003a, 2003b) in two major ways. First, "emotions 'feel like' they are in the mind, whereas somatic states 'feel like' they are in the body;" that emotions and drives rely on distinct neural circuits (but cf. Kurth, Zilles, Fox, Laird, & Eickhoff, 2010; Shackman et al., 2011; Tataranni et al., 1999); and that interventions targeting the body (e.g., systemic epinephrine administration, spinal cord transection) often have minimal consequences for emotional experience (Chwalisz, Diener, & Gallagher, 1988; Cobos, Sanchez, Garcia, Nieves Vera, & Vila, 2002; Deady, North, Allan, Smith, & O'Carroll, 2010; LeDoux, 2015; Rolls, 2005). The latter data provide a counterpoint to evidence presented by Wood et al. suggesting that manipulations of the facial musculature are sufficient to change feelings (but cf. Engber, 2016). In particular, Wood et al. highlight work demonstrating that injections of botulinum toxin ('Botox') into the upper face, which block contraction of the brow-lowering muscles that contribute to expressions of fear and anger (Coan, Allen, & Harmon-Jones, 2001; Ekman & Friesen, 1978), can improve mood.

The second way in which true emotional feelings differ from physiological drives, according to Eisenberger, is that "we can consciously access the eliciting factors of emotion but not those of somatic states," a view that is consistent with the earlier speculations of Ekman (Ekman, 1994c). Feelings of hunger, for example, indicate the need to eat but do not directly tell us about the circulating levels of glucose or insulin that evoke hunger. As a consequence, she argues that it is much easier to re-live or

simulate emotional experiences (for a related perspective, see Ekman, Friesen, & Simons, 1985). Although, Eisenberger notes, "no matter how many times we imagine having low glucose levels, this will not elicit feelings of hunger." A skeptic might question whether we would have any better success imagining the activity of basolateral amygdala projection neurons. Nevertheless, Eisenberger's point that emotional feelings differ from other physically embodied states in the extent to which their causes are open to introspection and in the degree to which they can be deliberately cultivated remains interesting (see also Engen & Singer's response to Question 7).

How well Eisenberger's account generalizes across drives is unclear. The source of peripheral pain, for example, is often quite clear, a point highlighted by Salomons. Although theorists differ in whether they consider pain a *bona fide* emotion—with many saying *No* (Ekman, 1997) and a few *Yes* (Rolls, 2005)—there is ample evidence that pain elicits negative feelings and shares many of the other core features of emotion, including a consistent set of antecedents, automaticity, rapid onset, robust alterations in peripheral physiology, a distinctive facial expression, and conservation across species. Furthermore, the subjective experience of pain can be precisely and consistently manipulated in the laboratory, unlike most emotions (Shackman et al., 2006). With this in mind, Salomons tells us that pain is associated with a specific set of 'labeled lines' in the periphery. Activation of A-delta fibers, for example, is specific to the sensation of sharp, stabbing pain. Is this specificity maintained in the brain? For a number of years, it appeared that the answer was 'yes.' The results of hundreds of imaging studies indicate that acute nociceptive stimulation—electric shock to the fingers, noxious thermal stimulation of the forearm, and so on—consistently recruits the 'pain matrix,' a network of brain regions encompassing the cingulate, insula, periaqueductal gray, and several other regions (Shackman et al., 2011; Wager et al., 2016; http://neurosynth.org/analyses/terms/pain/). But recent work by Salomons and his colleagues indicates that engagement of the pain matrix is neither necessary nor sufficient for the experience of pain or pain-related negative affect. In particular, they have demonstrated that individuals who are genetically

insensitive to pain still show robust activation of the pain matrix during the delivery of painful stimulation (Salomons, 2016; Salomons, Iannetti, Liang, & Wood, 2016). In a complementary study, Salomons and his colleagues found evidence of intact pain perception and pain-related negative affect—indexed using a battery of subjective, behavioral, and psychophysiological assays—in a patient with extensive damage to the pain matrix (Feinstein et al., 2016). Taken together, these observations indicate that activity in the pain matrix is not specific to the experience of pain-related feelings, consistent with other recent work focused on both pain (Hu & Iannetti, 2016; Iannetti, Salomons, Moayedi, Mouraux, & Davis, 2013; Krishnan et al., 2016; Legrain, Iannetti, Plaghki, & Mouraux, 2011; Wager et al., 2013; Woo et al., 2014) and emotion (see Question 5). More broadly, these observations underscore the value of using a combination of brain imaging techniques and rigorous assessments of affect to study patients with specific neurobiological and emotional deficits. As Salomons notes, this multi-pronged approach can also be fruitfully applied in animal models, opening the door to even more specific mechanistic insights.

While most of the contributors adopted a classical perspective on physical embodiment, focusing 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, McEwen and Rosenkranz remind 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. McEwen emphasizes that the brain itself is a bodily organ and he argues that it is the single most important physical substrate for the embodiment of emotion. McEwen reminds us that this vast physical infrastructure is incredibly plastic—"malleable, structurally and functionally"—in the face of experience, and that this epigenetically governed remodeling can have profound consequences for emotion and the emotional disorders. For example, repeated stress leads to increases in dendritic length, number of branch points, and number of branch tips in the basolateral amygdala, whereas the opposite pattern is observed in hippocampus and medial prefrontal cortex (Henckens et al., 2015). Other work indicates that broadly similar architectural changes

occur in response to much more mundane emotional experiences. For example, simple auditory fear conditioning is associated with highly specific remodeling of amygdalocortical synapses, including the formation of new axon terminals ('boutons') and spines (Yang et al., 2016). In short, the emotional brain, indeed, the entire brain, is not immutable and is constantly revising itself throughout the lifespan. McEwen makes it clear that the degree of malleability differs across the lifespan: "These alterations are largely reversible in young animals, but are less resilient in aging brains and in mood and perhaps other behavioral disorders." As McEwen notes, this work highlights the potential value of pharmaceutical and behavioral interventions (e.g., aerobic exercise) that enhance neuronal plasticity, particularly when combined with more targeted emotional interventions, such as cognitive-behavioral therapy.

In the first half of her response, Rosenkranz focuses on the immune system, marshaling observational and experimental evidence that alterations in immune function and inflammation can have profound consequences for mood and mood disorders. For example, many agents that mobilize the immune system, such as pro-inflammatory cytokines (e.g., interferon-alpha) and vaccines (e.g., typhoid), have been shown to elicit persistent symptoms of depression, anhedonia, and anxiety in humans and other mammals. Likewise, alterations in immune function early in life (e.g., due to neonatal bacterial infection) can lead to dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and changes in stress reactivity that persist into adulthood. Rosenkranz highlights evidence suggesting that many of these effects are proximally governed by the same brain regions implicated in the development of mood and anxiety disorders, noting that experimental activation of the immune system can increase the sensitivity of these regions to aversive stimuli and decrease their sensitivity to reward. She also makes it clear that relations between mood and the body are bi-directional, with interventions targeting one (e.g., mindfulness mediation training) often influencing the other (e.g., antibody response).

In the second half of her response, Rosenkranz extends classical perspectives on embodiment to include the many trillion microorganisms that live within the human body. Among these microorganisms, there is a growing recognition that the bacteria that inhabit the gut form a crucial node in the microbiota-gut-brain (MGB) axis, a complex, bi-directional network by which these microorganisms can influence and be influenced by the central nervous system (Cryan & Dinan, 2012; Dinan, Stilling, Stanton, & Cryan, 2015; Mayer, Knight, Mazmanian, Cryan, & Tillisch, 2014). Although cautioning that much remains unknown, Rosenkranz highlights recent experimental work suggesting that the gut microbiota play a key role in regulating the expression of anxiety and depression. For example, animals immunized with *Mycobacterium vaccae*—a harmless, common bacterium that is thought to strengthen the immune system—show reduced reactivity to novelty and chronic psychosocial stressors (Reber et al., 2016). Among humans, initial studies suggest that administration of pre- and probiotic supplements may reduce symptoms of anxiety and depression, decrease daily cortisol, and dampen neural responses to threat-related cues. Collectively, these observations highlight the potential of developing microbiome- and immunoregulation-based strategies for the prevention and treatment of anxiety and mood disorders. This approach is likely to be particularly important in light of evidence that global trends in urbanization are reducing human exposure to immunoregulatory microorganisms, increasing the risk of inflammatory disease and emotional disorders (Lowry et al., 2016; Stamper et al., 2016). On a broader note, Rosenkranz emphasizes that these kinds of data teach us that emotion is "the product of a mutualistic balance between our own optimal fitness and that of [the microorganisms] with which we share our environment and on which we are mutually reliant."

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