Neuroscientific Evidence for Simulation and Shared Substrates in Emotion Recognition: Beyond Faces

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Abstract

According to simulation or shared-substrates models of emotion recognition, our ability to recognize the emotions expressed by other individuals relies, at least in part, on processes that internally simulate the same emotional state in ourselves. The term "emotional expressions" is nearly synonymous, in many people's minds, with *facial* expressions of emotion. However, vocal prosody and whole-body cues also convey emotional information. What is the relationship between these various channels of emotional communication? We first briefly review simulation models of emotion recognition, and then discuss neuroscientific evidence related to these models, including studies using facial expressions, whole-body cues, and vocal prosody. We conclude by discussing these data in the context of simulation and shared-substrates models of emotion recognition.

Keywords

amygdala, empathy, simulation, somatosensory cortex

According to simulation or shared-substrates models of emotion recognition, our ability to recognize the emotions expressed by other individuals relies, at least in part, on processes that internally simulate the same emotional state in ourselves. The term "emotional expressions" is nearly synonymous, in many people's minds, with *facial* expressions of emotion, and the preponderance of studies of emotion recognition has focused on faces. However, other nonverbal cues such as vocal prosody, body posture, gestures, and locomotor patterns all convey emotional information—a fact well known to actors, dancers, mimes, and animators. What is the relationship between these various channels of emotional communication? Do the same neural structures play the same roles in recognizing, for example, fear from a facial expression as from a trembling voice, a cowering posture, or a hesitant, halting gait? Models of emotion recognition that were developed largely based on data from studies of facial expression may also work for other channels or cues—but evidence from these other cues might also necessitate refinement of face-based models. In the present article, we will first briefly review simulation models of emotion recognition, and then will discuss neuroscientific evidence related to these models. In each section, we will first review evidence from studies using facial expressions, and then findings based on whole-body and, to a lesser extent, vocal prosody cues. We conclude by discussing these data in the context of simulation and shared-substrates models of emotion recognition.

Simulation or Shared-Substrates Models of Emotion Recognition

Simulation models of emotion recognition propose that at least part of the mechanism by which we recognize another individual's emotional state relies on internally simulating the same emotional state in ourselves. As such, these models are consonant with theories of embodied cognition, which emphasize the utilization of body representations in a variety of behaviors from memory to attitude formation (Barsalou, Kyle Simmons, Barbey, & Wilson, 2003; Crawford, 2009; Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005; Ping, Dhillon, & Beilock, 2009), as well as with perception-action models of movement perception and imitation (Prinz, 1997). Simulation models can be contrasted with their logical alternative: one might imagine that emotion recognition is possible by a learned

rule-based system whereby configurations of facial features, vocal prosody patterns, and body gestures, postures, or movements come to be associated with knowledge acquired about the associated experience through other, perhaps verbal, means. However, as we will review below, sufficient evidence exists for simulation processes that it is difficult or impossible to believe they do not play at least some role in emotion recognition.

Emotion recognition via simulation might take at least two different forms, which are not mutually incompatible: emotional contagion, in which perceiving another's emotional expression gives rise somehow directly to an emotional experience in the perceiver (Preston & de Waal, 2002); and simulating the viewed emotional state by referencing representations of the associated body state (Adolphs, 2002) or motor programs (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003; Gallese, Keysers, & Rizzolatti, 2004). Both models are presumed to be fairly automatic, and both rely at least to some extent on the idea of *shared substrates*, or overlapping neural circuitry subserving both experience and recognition. Another related term is *vicarious responding* (Morrison, 2007), which refers to the activation of body-related brain areas by another person's expressions or imagined situations. As such, they are conceptually related to two other ideas relying on shared substrates for perception and action: mirror neurons, and pain empathy.

Mirror neurons were originally reported based on neurophysiological studies in macaques, in which single cells in region F5 (homologous to premotor cortex in humans) fired both when the monkey performed a goal directed action (e.g., reaching and grasping) and when it viewed another individual performing the same action (e.g., di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992).[1] Presumably homologous and analogous regions in humans—in premotor cortex near the frontal operculum—are active in functional neuroimaging studies when humans view actions with the intent to imitate (e.g., Decety & Grèzes, 1999; Iacoboni et al., 1999). This pattern of responding, not surprisingly, has been interpreted to underlie empathy (Blakemore & Decety, 2001; Decety & Lamm, 2006) and even other social behaviors (Gallese et al., 2004). Notably, premotor regions in humans have also been implicated in emotion recognition (e.g., Carr et al., 2003; Winkielman, McIntosh, & Oberman, 2009). The link between motor behavior and perception is also critical in the perception of body movement more generally, a process in which posterior superior temporal sulcus (pSTS) is known to play a key role (reviewed in Blake & Shiffrar, 2007).

Evidence for self–other overlap for pain empathy initially came from a serendipitous finding: Hutchison, Davis, Lozano, Tasker, and Dostrovsky (1999) were testing anterior cingulate cortex (ACC) responses to painful stimuli such as cold and pinpricks in neurosurgical patients who had consented to experimental studies while awaiting surgery. When an experimenter accidentally pricked herself with a testing probe, the ACC neuron from which Hutchison et al. were recording responded as though the patient had been similarly pricked (Hutchison et al., 1999). Two contemporaneous studies examined this dual coding of self- and other-pain in healthy subjects using slightly different paradigms: the same region of dorsal ACC is active both when

subjects' fingers are pricked with a sharp probe and when they see videos of anonymous fingers being stabbed with a hypodermic needle (Morrison, Lloyd, di Pellegrino, & Roberts, 2004), and both dorsal ACC and anterior insula are commonly active when female participants receive painful (vs. mild) electric shocks to the hand and when they believe that their partners, seated outside but right next to the scanner, are receiving similar shocks (Singer et al., 2004). All of these findings (and several others not included here) imply a degree of overlap between representing one's own pain and representing the pain of a known or imagined other.

Evidence for some overlap between representations of self and other facial expressions is not unique to neuroscientific studies: several studies have demonstrated that people move their facial muscles to mirror observed facial expressions. For example, Dimberg (1982) used facial electromyography to record facial muscle contractions, some too subtle to be detected otherwise, when subjects viewed emotional facial expressions. Such mimicry has been correlated with recognition (Wallbott, 1991) and with empathy scores (Sonnby-Borgström, 2002), and its effects need not be conscious: EMG studies revealed subtle contractions even when emotional facial expressions were presented to the subject subliminally (Dimberg, Thunberg, & Elmehed, 2000). Amazingly, viewers even spontaneously contract or dilate their pupils to match those of viewed sad faces (Harrison, Singer, Rotshtein, Dolan, & Critchley, 2006). Inhibiting mimicry has also been shown to impair emotion recognition in at least one study: when subjects were asked to hold a pencil laterally between their teeth, a manipulation which forces a smile-like contraction of the zygomaticus muscles in the cheek (Strack, Martin, & Stepper, 1988), their emotion recognition performance was reduced (Niedenthal, Brauer, Halberstadt, & Innes-Ker, 2001).[2] That said, Hess and Blairy (2001) did not find any relation between emotion recognition performance, as measured by an emotion rating task, and subjects' tendency to mimic dynamic displays of happiness, sadness, anger, and disgust. Subjects did show evidence of mimicry, but mimicry was not correlated with self-reported emotional contagion. In addition, patients with facial paralysis have not shown impaired discrimination or recognition of emotional facial expressions, as measured by matching and naming tasks with standardized facial expressions (Calder, Keane, Cole, Campbell, & Young, 2000; Keillor, Barrett, Crucian, Kortenkamp, & Heilman, 2002).[3] In summary, though people spontaneously mimic observed facial expressions, and this mimicry may often play a role in recognition of others' emotions, the evidence regarding the necessity of mirroring movements for emotion recognition is still mixed.

A long, though sparser, history of research has examined how emotional information is conveyed by whole-body cues. Darwin famously included postural descriptions in his *The Expression of the Emotions in Man and Animals* (Darwin, 1872/1965), and William James examined emotion recognition from whole-body posture photographs in 1932. In that latter series of experiments, James noted that subjects viewing photographs of posed whole bodies frequently were seen to imitate the posture they were looking at, and noted further that this
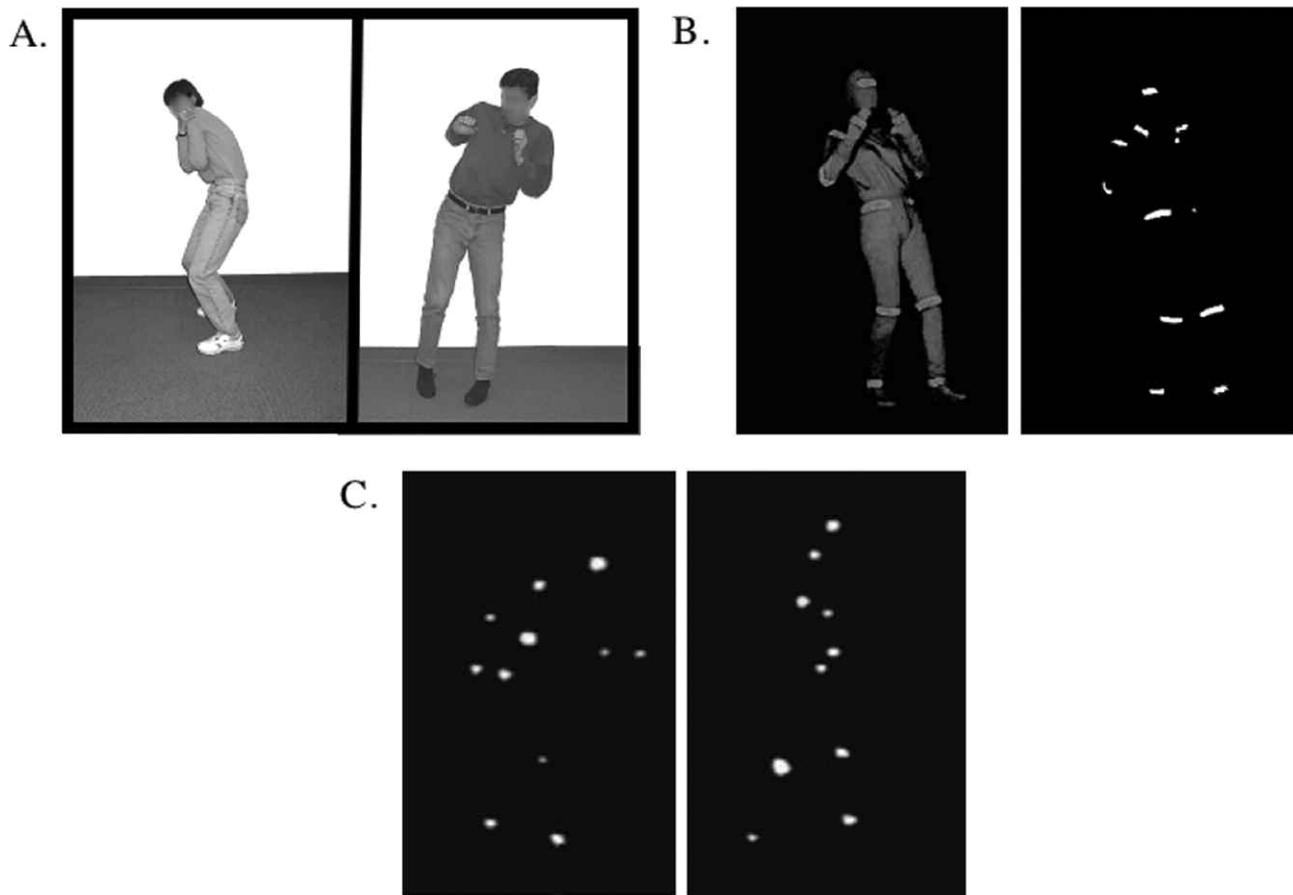
Figure 1. Examples of whole-body emotional expression stimuli.

(a) Examples of emotional posture photographs used in Atkinson, Heberlein, and Adolphs (2007) (created by A. S. Heberlein). Both of these are labeled "fearful" by the majority of normal participants.

(b) Still shots from full-light and corresponding patch-light emotional movement movies (first described in Atkinson et al., 2004). These patch-light displays are similar to the point-light displays originally developed by Johansson (1973) to study the perception of body and other biological motion. Small lights or reflective patches are placed on the major joints of a person, who is then filmed while making body movements. Video clips are then produced such that the only visible elements are these moving small light points or patches. The dynamic stimuli are instantly recognizable as human motion, even when a human form is not readily recognizable in stills from such stimuli. Point-light and patch-light stimuli can be ideal for studies of movement-based emotion perception because both face and morphological cues are absent or minimal, but kinematic cues are preserved. The movement of the figure shown in B is recognized as fearful by normal participants, whether viewed in full-light or patch-light; still shots, in contrast, are only recognizable as fearful in the full-light version, as is evident here.

(c) Still shots from two point-light emotional movement movies (first described in Heberlein, Adolphs, Tranel, & Damasio, 2004). Both movements are recognized as fearful by normal participants, but without kinematic information the figures are unlikely to even be recognizable as human forms, especially without the context of the other figures.

tendency rarely resulted in an outright assumption of the posture, but . . . was either directly reported or was revealed by reports of kinaesthesis in the arms, legs, back, neck or other part of the body significant for the assumption of the posture. (James, 1932, p. 419)

James also reported evidence for emotion contagion:

In some instances the observers also experienced the feeling or emotion which was attributed to the postural expression. The feeling . . . resulted from the tendency of the observer to put himself into the same situation and posture as that of the figure. (p. 419)

James was skeptical, however, about the possibility of getting high inter-subject agreement about the emotion portrayed by any given posture—or, for that matter, facial expression. In contrast, more recent studies with both static and dynamic whole-body stimuli have indeed found a high degree of agreement, using a combination of (a) stimuli which were specifically constructed to convey emotional expressions, often using trained actors (as opposed to James' more open-ended approach in stimulus construction and selection) and (b) more constrained methods than James' subjects' free responses (e.g., Atkinson, Dittrich, Gemmell, & Young, 2004; de Meijer, 1989; Dittrich, Troscianko, Lea, & Morgan, 1996; Wallbott, 1998). Much of this research has focused on identifying the visual cues used to identify bodily expressed emotions and, as we discuss below, the neural mechanisms underpinning bodily emotion recognition. For example, it is clear from studies that have isolated body or body-part motion using techniques such as the point-light display (see Figure 1) that such motion cues (especially kinematics) convey important information about others' emotional states.

Below, we review further evidence for simulation models of emotion recognition from neuroscientific studies. Such evidence falls into two general categories: support for the role of specific neural structures in both recognition and experience measures (such as for the amygdala in both fear experience and fear recognition) and support for the role of known motor or sensory structures in emotion recognition (such as for premotor and somatosensory cortices in recognizing emotional faces). Because many of the same regions are implicated in emotion recognition based on facial, prosodic and/or whole-body cues, we will organize this discussion by neural structure or region, focusing first on faces and then on prosody or whole-body emotion recognition. Along the way we briefly and somewhat selectively review functional imaging and neuropsychological studies of face emotion recognition and, more completely (because the relevant literature is smaller), whole-body emotion recognition. We also include discussions of data regarding emotional prosody recognition in sections where these enable a more complete understanding of the role of a particular region in emotion recognition processes. (For recent, more comprehensive reviews of emotion recognition from vocal cues, see Bachorowski & Owren, 2008 and Grandjean, Banziger, & Scherer, 2006.) We will focus particularly on three anatomical structures or regions: the amygdala and right somatosensory cortex, primarily because their roles in emotion recognition based on other nonverbal cues have been explored, and the face- and body-selective areas of occipitotemporal cortex. Both the amygdala and right somatosensory regions have played prominent roles in simulation models of emotion recognition, although as we will discuss below, the evidence for amygdala involvement in at least simple simulation models is quite mixed. We will also briefly discuss the roles of two frontal cortical regions in emotional face recognition: left inferior frontal cortex (about which more is said in the Winkielman et al. article in this issue) and ventromedial frontal cortex. Note that other regions, which we will not discuss here, are known to be critical for processing at least certain emotions in facial expressions, prominent among them the insula and basal ganglia (Calder, Lawrence, & Young, 2001; for more comprehensive reviews of the neuroscience of face emotion recognition please see Adolphs, 2002 and Atkinson, Heberlein, & Adolphs, in press).

The Amygdala in Emotion Perception and Experience

The Amygdala in Face Emotion Processing

The amygdala, a paired, almond-sized and -shaped nucleus buried within the medial temporal lobe, has long been known to be important for emotional behavior, and particularly for aversive or "fear" conditioning in both animal models (Fanselow & LeDoux, 1999) and in humans (e.g., Bechara et al., 1995; LaBar, LeDoux, Spencer, & Phelps, 1995). The importance of the amygdala in social behavior was established by studies of nonhuman primates with anterior temporal ablations or, in more recent studies, selective damage to the amygdala, who showed impairments in social interactions (Amaral, 2002; Klüver & Bucy, 1939/1997). However, a series of studies of rare patients with

bilateral amygdala damage, conducted and reported in the 1990s, highlighted an apparently specific deficit in fear recognition consequent to amygdala damage in humans (e.g., Adolphs, Tranel, Damasio, & Damasio, 1995; Calder, Young, Perrett, Hodges, & Etcoff, 1996; Sprengelmeyer et al., 1999). These and other patient studies used standardized emotional face stimuli such as those created by Ekman and Friesen (1976), and required participants either to label the faces (usually by choosing from a list of emotion words) or to rate them (usually rating the level of each of several "basic" emotions in each of a set of faces).

Functional neuroimaging studies of neurologically intact participants yielded convergent results regarding the role of the amygdala in fearful face processing, though notably most did not directly address emotion recognition: participants were generally passively viewing emotional faces, or performing a task such as gender categorization which required attention to the faces but not to their expressions. Amygdala activity was greater when participants viewed fearful versus neutral faces (e.g., Morris et al., 1996; Pessoa, McKenna, Gutierrez, & Ungerleider, 2002) or happy faces (e.g., Morris et al., 1996; Pessoa et al., 2002; see Phan, Wager, Taylor, & Liberzon, 2002 for a review). In some studies, amygdala activity was observed even during presentation of subliminally presented faces (e.g., Whalen, 1998—but see Phillips et al., 2004) or during faces that were viewed at the same time as, but incidental to, an attention-demanding task (e.g., Vuilleumier, Armony, Driver, & Dolan, 2001—but see Pessoa et al., 2002). This activation may be due to input from the subcortical pathway to the amygdala, that is, from the superior colliculus to the pulvinar and thus to the amygdala (Morris, Öhman, & Dolan, 1999).

This collection of lesion and imaging findings has been interpreted in light of amygdalar involvement in aversive conditioning and in other measures of fear-related experience. For example, in addition to the amygdala's established role in conditioning, intracranial stimulation of the amygdala has been associated with a feeling of anxiety (Halgren, Walter, Cherlow, & Crandall, 1978). This dual involvement in *recognition* and *experience* was thus interpreted as support for a model of the amygdala as a "convergence zone" for fear (Damasio, 1989): The amygdala was thought to coordinate representations of and responses to fear-related stimuli in cortical and subcortical regions (Adolphs et al., 1995). One fairly recent study cast doubt on the role of the amygdala specifically in fear experience: one subject with bilateral amygdala damage as well as several with unilateral damage reported normal affective states both in a retrospective consideration of the past month of experience, and in a day-by-day measure over the course of a month. This was true even for anxiety and fear states, implying that the amygdala is not critical for normal experience of a range of emotional feelings, even though it may be recruited during such experience in intact brains (Anderson & Phelps, 2002). However, the results of another study suggest that the emotional lives of individuals with amygdala lesions might not be entirely normal. Independent clinical interviews of another individual with complete bilateral amygdala damage indicated that she experienced a normal range of affect and emotion, and yet she recounted the considerable amount of adversity in her

life in a matter-of-fact way, without any sign of dysphoria, and she denied having felt strong emotions at the time (Tranel, Gullickson, Koch, & Adolphs, 2006). In contrast, when talking of positive life events, the same individual was much more animated and positive. She was also judged by clinicians naive to her neuropsychological condition to lack a normal sense of distrust and danger of others (Tranel et al., 2006), which is consistent with the finding that she rates as highly trustworthy and approachable those faces that are normally judged to look the least trustworthy and approachable (Adolphs, Tranel, & Damasio, 1998). Consider also the recent finding that individuals with amygdala lesions showed abnormally low levels of self-reported arousal in response to negative (but not positive) picture stimuli (Berntson, Bechara, Damasio, Tranel, & Cacioppo, 2007). Thus, Anderson and Phelps' (2002) finding of no emotional experience differences consequent to amygdala damage may have been due to the use of an insufficiently sensitive measure requiring self-report and introspection. The amygdala may in fact be important for the experience of at least certain types of negative affect (though perhaps not specifically fear).

To further muddy the "convergence zone for fear" picture, recent data suggest that the link between fearful face processing and the amygdala is not as straightforward as the initial lesion and imaging reports suggested. Amygdala activity is observed not just during viewing of fearful faces, but also when participants view happy faces (relative to neutral ones; e.g., Pessoa et al., 2002). Furthermore, patterns of face emotion recognition impairment after amygdala damage can be variable, depending both on the task used and on individual differences. For example, Adolphs and colleagues (1999) compared nine people with bilateral amygdala damage on the same sensitive emotion-rating task. They found that while fear was the most commonly and most severely impaired emotion, only four of the nine were impaired on fear recognition, and some were impaired on other emotions (three each impaired on sadness, disgust, and surprise, and four on anger). Functional imaging studies explicitly examining individual differences in amygdala activity have found that variance in personality traits and in gene polymorphisms has a large effect on amygdala response to emotional faces (e.g., Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002; Hariri et al., 2002).

Recent work focusing on the amygdala's role in facial expression recognition has focused on a role in directing attention to and within emotional faces. Whalen and colleagues (2004) found amygdala activity when healthy subjects viewed backward-masked "eye whites" isolated from fearful facial expressions (vs. eye whites isolated from happy expressions), but not inverted "eye blacks." Careful testing of a single bilateral-amygdala-damaged patient, SM, provided convergent evidence about the necessity of the amygdala for normal direction of attention to the eyes in emotional face processing (Adolphs et al., 2005): SM and a group of demographically matched control subjects were asked to make two-alternative (happy vs. fear) emotion judgments based on viewing only small, variably-placed and -sized regions of the face at once (the "bubbles" technique developed by Schyns, Bonnar, & Gosselin, 2002). Normal controls use information from the eye region to make

judgments of happiness, fear, sadness and anger, and especially the latter three of these (Smith, Gosselin, & Schyns, 2004). In contrast, Adolphs and colleagues showed that SM did not use information from the eyes when making two-alternative judgments. This finding parsimoniously explains SM's known impairment in fearful face recognition. In a direct test of this, her fear recognition was "rescued" when she was instructed to attend to the eyes in a free-viewing emotion recognition task (Adolphs et al., 2005). Thus, rather than merely responding to fearful faces in a sort of large-scale pattern recognition, the amygdala may participate in emotion recognition by responding to the presence of certain facial features, such as eyes, and directing attention for further processing to those features and the rest of the object—or even the environment—surrounding them. This interpretation is consistent with findings indicating that subjects with bilateral amygdala damage perform poorly on other social judgments requiring attention to the eyes, such as inferring intention or attention from eye gaze (Young et al., 1995) or making judgments about social emotions such as jealousy or guilt from the eye region of facial expressions (Adolphs, Baron-Cohen, & Tranel, 2002).

To summarize: the amygdala is reliably recruited during both fearful experience and fearful face processing, thus serving at least nominally as a "shared substrate" for both experience and recognition. However, its role relating these processes is not as clearcut as was once thought. This is due both to inconsistencies in data linking the amygdala specifically to fearful experience as opposed to threat-related emotional experience or negative emotional experience more generally, and to the increasing support for models positing a more general role for the amygdala in directing attention to environmental stimuli indicative of potential threat, including but not at all limited to (certain features of) fearful faces (Whalen et al., 2004). We return to this point below, in discussing links between the amygdala and visual face-processing regions in the service of attending to emotional faces. However, recent data linking emotional experience to trait negative affect and amygdala activity help to indicate what sort of role the amygdala might play in a shared substrates model of emotion experience and recognition: Barrett and colleagues (2007) specifically addressed this issue by correlating trait negative affect, measured by experience sampling over several weeks, with amygdala reactivity when subjects viewed briefly presented fearful faces months later inside the scanner. Their results indicate that amygdala reactivity might underlie both perceptual sensitivity to negative environmental stimuli—perhaps especially social stimuli—and the affective sequelae of perceiving negative information (Barrett, Bliss-Moreau, Duncan, Rauch, & Wright, 2007). The kind of attentional processes that the amygdala mediates in the service of emotion recognition may mediate experience as well, *because* awareness of negative environmental stimuli leads to negative emotion.

The Amygdala in Prosodic and Whole-Body Emotion Recognition

Given the role that the amygdala is now thought to play in facial emotion recognition, it may not be surprising that the evidence

for amygdala involvement in recognizing emotion from vocal cues is mixed. If at least part of the amygdala's role in emotion recognition consists of low-level cue recognition and directing attention to stimuli thus detected (e.g., wide eye whites), would one postulate a corresponding role in vocal emotion recognition? One bilaterally amygdala-damaged subject (DR) was found to be impaired at labeling fear and anger from both prosody and non-verbal vocal sounds in addition to faces (Scott et al., 1997), and another (NE) was impaired in recognizing fear (forced choice labeling) from faces, prosody, and body postures (Sprengelmeyer et al., 1999). However, three different bilaterally amygdala-damaged subjects (SP, SM and RH) tested in two separate studies showed no selective impairment recognizing fear when labeling prosodic stimuli (Adolphs & Tranel, 1999; Anderson & Phelps, 1998). While the exact regions of extra-amygdaloid damage differ between these subjects, the anatomical differences do not line up with the differences in their impairments (Calder et al., 2001), so it is hard to draw conclusions about the importance of the amygdala in recognition of fear, or any other emotions, from vocal prosody or non-verbal vocal sounds. Imaging studies have not resolved this question: hearing either fearful vocal expressions or laughter and crying activated amygdalar nuclei in two studies (Phillips et al., 1998; Sander & Scheich, 2001), but another study found a *decrease* in amygdala activation while participants listened to nonverbal expressions of fear (Morris et al., 1999). It is at present unclear if these discrepancies, like those found for faces (see above), are due to individual differences, to task differences, or, in the case of the imaging studies, to limitations in BOLD fMRI resolution. Nonetheless, it is not at present possible to conclude that the amygdala is critically involved in recognizing fear from vocal cues.

The evidence for amygdala involvement in whole-body emotional expression recognition is similarly mixed: many imaging studies find amygdala activity during viewing of emotional whole-body stimuli, but lesion studies have not provided converging evidence regarding the necessity of the amygdala for whole-body emotion recognition, and in fact have provided some evidence against such a necessity. Bonda and colleagues (1996) may have been the first to examine neural activations in response to viewing emotional body movements. Using PET imaging, they found greater activity in both amygdala and superior temporal sulcus when subjects looked at emotional whole-body movements (with the instruction to attend for future recognition memory testing), as compared with goal-directed hand-and-arm movements (Bonda, Petrides, Ostry, & Evans, 1996), but they did not specifically examine fearful body movements. Two recent functional imaging studies by de Gelder and colleagues that did focus on fear have also yielded amygdala activity in response to whole-body emotion cues. In one, passively viewing photos of fearful body postures with blurred faces, as compared to neutral postures, yielded right amygdala activity and fusiform activity (in the separately-localized fusiform face area; Hadjikhani & de Gelder, 2003), implying a role for the amygdala in the processing of fearful visual expressions other than faces. In another analysis, right amygdala was more active during passive viewing of fearful stimuli than

during viewing of neutral photographs, as were a suite of other regions including orbitofrontal cortex, body-representing regions including fusiform cortex, and motor planning regions (de Gelder, Snyder, Greve, Gerard, & Hadjikhani, 2004).

De Gelder and colleagues interpreted these findings as supporting a model of emotional body cue processing with two separate but connected circuits: the first is a more automatic system involving the subcortical route to the amygdala (i.e., via the superior colliculus and pulvinar nucleus of the thalamus) and the striatum, with a critical role in preparing appropriate motor responses to environmental threat, as communicated by another's expression; this system is conceptualized in reflexive terms. The second, in contrast, is a primarily cortical system involving amygdala; body-representing perceptual areas including fusiform and lateral occipitoparietal regions; and motor planning areas; with a role that encompasses detailed perceptual processing of another's emotional body expressions, connecting this perception to stored knowledge (i.e., recognition), and planning an appropriate motor response in light of this detailed information (de Gelder, 2006). Note that the descriptions of both of these circuits emphasize links between the amygdala and motor planning structures, highlighting these authors' focus on the connection between emotion perceived in others—particularly from the rich cues of whole-body stimuli—and behavioral responses. However, three recent studies have failed to replicate the specificity of the amygdala response to fearful whole-body stimuli: Pichon, de Gelder, and Grèzes (2007) found increased amygdala activity for passive viewing of *angry* relative to neutral static and dynamic bodies, and Grèzes, Pichon, and de Gelder (2007) found greater amygdala activity during viewing of *both* fearful and neutral whole-body stimuli (both static and dynamic) relative to scrambled versions of the same stimuli; regions with greater activity during viewing of fearful whole-body stimuli included bilateral temporal pole and posterior superior temporal sulcus, as well as right premotor area and a lateral occipital region which may be the extrastriate body area (EBA), a visual region selective for bodies and body parts over other objects (Downing, Jiang, Shuman, & Kanwisher, 2001). Pichon et al. and Grèzes et al. interpret the discrepancy between their findings and those reported in the previous studies with static stimuli as resulting from the more closely matched stimuli: in the first two experiments, emotional expressions included still shots from such actions as opening a door and expressing fear at an imagined robber, whereas neutral stimuli consisted of instrumental actions such as combing one's hair or pouring water. In contrast, Grèzes et al. focused on the door-opening action but varied only the emotion expressed by the actor upon opening the door; they suggest that this "instrumental action . . . invite[s] by itself . . . a social meaning," which by implication is sufficient to recruit the amygdala—and to do so comparably to the presence of fearful expression. The absence of increased amygdala activity during observation (for subsequent rating of intensity) of fearful compared to neutral whole-body movements has also been reported by Peelen, Atkinson, Andersson and Vuilleumier (2007), who nevertheless did find greater amygdala activation for both happy and angry (but not sad or disgusted) body movements.

Evidence from lesion studies that would confirm the necessity of the amygdala for whole-body emotion processing has generally *not* supported such a role. As noted above, Sprengelmeyer et al. (1999) found impaired fearful posture recognition in a subject with bilateral amygdala damage, consistent with this individual's impairments recognizing fear from faces and vocal expressions. However, two other studies have demonstrated normal emotion recognition from several differing whole-body stimulus sets in another subject with bilateral amygdala damage, SM, who has been previously demonstrated to have impairments recognizing fearful faces. In one of these studies, photographs of dramatic, emotionally-charged movie scenes were altered so that the facial expressions were not visible. Though this alteration significantly reduced normal subjects' ability to recognize all emotions, including fear, from the characters, SM recognized fear normally in a forced-choice paradigm—in fact, her performance was *better* for these stimuli than for the intact photographs (Adolphs & Tranel, 2003; note that three other participants with bilateral damage that included amygdala as well as further medial temporal lobe structures were similarly normal on both the masked and intact photographs). In the second study, SM was tested with four different whole-body stimulus sets (see Figure 1): a set of emotional body posture photographs with the faces blurred; dynamic stimuli in which the actors faced forward and expressed emotion with a full-body gesture; the same stimuli edited to be patch-light (similar to point-light; Atkinson et al., 2004); and a set of emotional point-light walkers in which the actors were filmed walking, creeping, dancing, or otherwise locomoting across the field of view (Heberlein et al., 2004; Heberlein & Saxe, 2005). Another bilaterally amygdala-damaged subject, AP, was tested on two of these (the posture photographs and the point-light walkers). Both participants recognized fear normally, in forced-choice tasks, in all the stimulus sets that they judged (Atkinson et al., 2007). These two findings make it impossible to claim that the amygdala is necessary for normal recognition of emotional body movements. Consistent with the data of de Gelder and colleagues, it may be the case that in intact brains the amygdala serves to associate perceived bodily expressions of fear with relevant motor plans. By this view, SM—though she is able to know that the perceived individual is afraid—would not prepare escape behavior in response to seeing another person's fearful body expression. Potential future experiments examining the role of the amygdala in responding to whole-body expressions might include an examination of evoked motor responses or motor-related activity in patients with bilateral amygdala damage as well as further studies of amygdala responses to dynamic and static whole-body emotional expressions, ideally with the inclusion of individual differences measures.

Right Somatosensory Cortex in Emotion Perception and Experience

Right Somatosensory Cortex in Face Emotion Processing

Damage to cortices in the right hemisphere has long been known to result in impaired recognition of emotional expressions (e.g.,

Bowers, Bauer, Coslett, & Heilman, 1985). Recent evidence from both functional neuroimaging studies (Winston, O'Doherty, & Dolan, 2003) and from lesion overlap studies (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000) suggests that the critical regions within the right hemisphere lie within somatosensory cortices, broadly defined to include not just primary but also more posterior secondary somatosensory regions. Adolphs and colleagues tested 108 brain-damaged individuals on a face emotion-rating task, then compared the overlap of brain lesion locations of impaired (i.e., abnormal) patients with the overlap of brain lesion locations of unimpaired patients. The region of maximal lesion overlap among impaired patients was in right posterior postcentral gyrus, bordering on supramarginal gyrus (Adolphs et al., 2000). Notably, single patients with lesions to right somatomotor cortices, but sparing somatosensory cortices, were unimpaired, whereas patients with lesions to somatosensory cortices sparing motor cortices were impaired. A nearly identical region was highlighted in a functional imaging study in which participants attended either to the emotional content or the gender of pairs of morphed faces (morphed from a neutral face of one gender to an emotional face of the other gender). When participants attended to the emotional content (answering "Which is more emotional?"), this region of right somatosensory cortex was significantly more active than when participants viewed the same faces but attended to gender ("Which is more male?"; Winston et al., 2003).

A recent study using transcranial magnetic stimulation (TMS) has confirmed the critical role of right somatosensory regions in emotion recognition. Single pulse TMS over right somatosensory cortex slows facial emotion discrimination (in a match-detection paradigm), relative to similar stimulation over superior lateral temporal cortex (the converse pattern was obtained for eye gaze discrimination; Pourtois et al., 2004). Interestingly, right somatosensory stimulation slowed judgments of fearful faces, but not happy faces; such a distinction has not been observed for this region in lesion or neuroimaging studies. However, it is unclear how to reconcile these TMS findings with the lesion and functional imaging findings implicating right somatosensory regions in emotion recognition across categories.

More generally, however, one might ask why somatosensory cortex—a region of the brain named for its role in representing bodily sensations such as touch and pressure—is important for recognizing emotion in others. Adolphs (e.g., 2002) and others have interpreted this involvement by appealing to simulation models of emotion recognition, specifically the idea that at least one component of emotion recognition processes entails a reliance on internal representations of the body state associated with an observed emotion—in other words, what it *feels like* to be experiencing the emotion that we view another person expressing. Right somatosensory cortex might thus be involved in the representation of a "somatosensory image" associated with a felt emotion (Adolphs, 2002); we return to this point below.[4]

Right Somatosensory Cortex in Prosodic and Whole-Body Emotion Recognition

Given such a model of right somatosensory involvement in emotion recognition, it is not surprising that other forms of

nonverbal emotional expressions also appear to rely at least partly on right somatosensory regions. In contrast to the amygdala's posited role—detecting certain features of environmental stimuli relevant to threat, or other emotional salience, and focusing further attention on these stimuli—right somatosensory regions are posited to represent either the feeling-state associated with an observed emotional expression or the actual sensations associated with producing an emotional expression. Right hemisphere cortices have consistently been implicated in studies of emotional vocal prosody recognition (e.g., Kucharska-Pietura, Phillips, Gernand, & David, 2003; Ross, Thompson, & Yenkosky, 1997), and a lesion overlap study like the one discussed above for facial expression similarly found a focus of maximal overlap in posterior regions of right somatosensory cortex, including portions of both postcentral and supramarginal gyri, for subjects impaired on an emotional prosody rating task (Adolphs et al., 2002). Convergent evidence comes from TMS, with a twist—a dissociation between approach and avoidance emotions. In a study similar to the one by Pourtois et al. described above for face recognition, van Rijn et al. (2005) delivered repetitive pulse TMS to participants' right frontoparietal operculum, encompassing the somatosensory representation of the mouth region. Immediately afterwards, the same participants detected "withdrawal emotions," that is, fear and sadness, significantly slower in heard sentences (compared to after sham stimulation). In contrast, no effect was observed for the "approach emotions," that is, happiness and anger. Van Rijn and colleagues interpret the laterality effects that they and Pourtois et al. (2004) observed in light of Davidson's (e.g., 1992) theories regarding hemispheric differences in approach versus withdrawal emotion experience, in which right frontal regions are more involved in withdrawal-related emotions, and left frontal regions are more involved in approach-related emotions.

The role of right somatosensory regions in emotion recognition also extends to recognizing whole-body emotional expressions. Benowitz and colleagues (1983) tested both right- and left-hemisphere-damaged patients using the Profile of Nonverbal Sensitivity (PONS; Rosenthal, Hall, DiMatteo, Rogers, & Archer, 1979), which requires emotion labeling of both faces and body movements/gestures in short dynamic stimuli. Interestingly, though they found face recognition impairments in five of six right-hemisphere damaged subjects, only one of these five was also impaired at the whole-body emotion recognition task—and notably, this patient had parietal damage, potentially corresponding to somatosensory cortex involvement. More recent work has attempted to localize regions critical for whole-body emotion recognition more precisely: a lesion overlap study examining impairments in emotion recognition (forced-choice labeling) based on whole-body point-light walker cues (Heberlein et al., 2004) identified similar right-somatosensory regions to those implicated in the earlier studies of facial (Adolphs et al., 2000) and vocal (Adolphs et al., 2002) expressions. As in the initial lesion overlap study on face emotion recognition, examinations of cases with precentral versus postcentral gyrus damage found impairments in individuals with damage to sensory but not motor cortex, and preserved performance in

individuals with the reverse pattern of damage, indicating that motor impairments could not explain this pattern of results.

The involvement of right somatosensory regions in whole-body emotion recognition was supported by an fMRI study using the same stimuli as the lesion overlap study (Heberlein & Saxe, 2005): a region at the border of right postcentral and supramarginal gyri was more active when subjects made emotion judgments about point-light walkers (given one of a known set of emotion words, rating how well it fits the stimulus) than when they made personality trait judgments (comparable task with trait words) based on the same stimuli. Further, a region of interest based on the maximal lesion overlap of the previous study—that is, the posterior right somatosensory cortex—was significantly more active for emotion, as compared to personality trait, judgments. A separate region, in left inferior frontal gyrus, was associated with personality trait judgments in both the lesion and fMRI articles (Heberlein & Saxe, 2005).

In summary, right somatosensory cortices appear to play a role in emotion recognition based not just on faces, but also on emotional prosody and whole-body emotional expressions. This evidence is consistent with two different simulation roles: modeling what a specific body part *feels like* when it produces an emotional expression, or representing the overall (or perhaps just visceral) body state associated with a felt emotion. In either case, we recognize the emotional expressions that we see or hear from others by relying (to some extent) on representations of what our own bodies feel like. While both lesion and functional imaging data implicate right, and not left, somatosensory cortex, at least two "functional lesion" TMS studies instead suggest that right and left regions both participate, depending on the specific emotion in question; additional work is necessary to explore this possible dissociation further.

Category-Selective Visual Regions in Emotion Perception

Visual Cortex Regions in Face Emotion Recognition: Fusiform, Occipital, and Superior Temporal Face Areas

Faces constitute a category of visual object for which there is both selectivity (a preference for faces over other visual objects) and functional specialization in higher-level visual cortices (for reviews, see e.g. Atkinson et al., in press; Kanwisher & Yovel, 2006). Prominent amongst these regions are the occipital face area (OFA) and the fusiform face area (FFA), whose functions are principally in the extraction and integration of structural information from faces (featural and configural cues). Face selectivity is also evident in posterior regions of superior temporal cortex, which, in contrast to the processing of relatively invariant aspects of faces in fusiform cortex, are more involved in processing facial movements and other, relatively changeable aspects of faces (Haxby, Hoffman, & Gobbini, 2000), or in the multimodal integration of changeable and invariant aspects of social information signaled by the face, body or voice (Calder & Young, 2005). What are the roles of these regions in emotion recognition, and in particular, in

simulation processes underlying the ability to infer emotional states from facial expressions?

In the influential face-processing model of Haxby et al. (2000), the OFA sends outputs to both the FFA and posterior superior temporal cortex (principally the superior temporal sulcus or STS). Facial expression perception is underpinned by the activity of posterior STS in association with other regions involved in emotion perception generally, including the amygdala and insula. The FFA, on the other hand, has little or no role in processing emotional expressions, in this model, being more involved in processing identity, sex, and other more invariant aspects of faces. However, a more recent survey of the evidence indicates that this bifurcation of identity and expression processing reflects more of a bias or relative segregation than a categorical dissociation (Calder & Young, 2005). Consistent with this proposal is evidence that the FFA's activity is modulated by the emotional expressions of viewed faces, which we discuss below. Consider also the very recent finding of early discrimination of emotional expression in ventral temporal cortex (including fusiform), as revealed by intracranial recordings in humans viewing emotional faces: cells in ventral temporal cortex responded differentially to emotions in dynamic morphing faces, and did so more quickly and accurately than did a region of lateral temporal cortex that included STS (Tsuchiya, Kawasaki, Howard, & Adolphs, 2008).

Consistent with the suggestion that the OFA encodes structural properties of faces at an early stage of visual processing, the first face-selective response over posterior sites occurs at around 100 ms after stimulus onset (e.g., Liu, Harris, & Kanwisher, 2002). Indeed, there is evidence that the OFA is critically involved in the processing of individual facial features but not configural cues at this very early stage: repetitive TMS applied to right (but not left) OFA within a window of 60–100 ms from stimulus onset disrupted accurate discrimination of individual facial features but not of the spacing between those features in a delayed match-to-sample task (Pitcher, Walsh, Yovel, & Duchaine, 2007). Furthermore, a recent study showed that TMS applied to right OFA impaired discrimination of emotional facial expressions in a similar task (Pitcher, Garrido, Walsh, & Duchaine, 2008). This study also confirmed previous findings of TMS to right somatosensory cortex disrupting facial emotion recognition. Importantly, for present purposes, Pitcher et al. (2008) found different critical periods for right OFA and right somatosensory cortex involvement in emotion expression processing: emotion discrimination accuracy was impaired only when TMS was delivered over right OFA at 60–100 ms or over right somatosensory cortex at 100–140 ms and 130–170 ms post-stimulus onset—notably early, but later than OFA. Thus, the OFA and somatosensory cortices contribute to emotion recognition at different times, consistent with hierarchical models of facial emotion processing, according to which information about structural properties of faces relevant to the perception of emotional expressions is fed forward to somatosensory regions implicated in the simulation of body states associated with the viewed emotional state. It remains to be seen what roles, if any, the FFA and posterior STS have in such a processing hierarchy.

A consistent finding from functional imaging studies of emotional face perception is an enhanced activation of occipital and temporal regions, including the FFA and OFA, in response to viewing faces expressing emotions, relative to emotionally neutral faces (for a review, see e.g., Vuilleumier & Driver, 2007). Emotional enhancement of FFA activation has been shown to correlate with activity in the amygdala (e.g., Morris et al., 1998), which is consistent with findings from animal studies demonstrating substantial bidirectional connections between the amygdala and much of ventral temporal cortex (e.g., Amaral, Behniea, & Kelly, 2003), and thus implicates the amygdala as the source of the emotional modulation of visual cortex. More direct evidence of the amygdala's role as the source of the emotional modulation comes from a combined lesion and fMRI study. Vuilleumier, Richardson, Armony, Driver, and Dolan (2004) found that individuals with hippocampal damage but no amygdala damage showed the normal modulation of fusiform cortex by viewing fearful compared to neutral facial expressions while making a same/different judgment, whereas individuals with amygdala and hippocampal damage did not. Furthermore, in an important control condition, fusiform cortex activity was modulated by attention to faces as opposed to houses in both groups of patients. Vuilleumier and colleagues have proposed that this emotional modulation of occipitotemporal face-processing regions serves an essentially attentional role, albeit one that is functionally and anatomically distinct from task-related or purely stimulus-related attentional modulation; specifically, the emotional significance of stimuli triggers, via feedback connections from the amygdala, an enhancement or prioritization of their visual processing (e.g., Vuilleumier, 2005; Vuilleumier & Driver, 2007).

Modulation of occipitotemporal activity during viewing of emotional relative to neutral faces occurs around 170 ms after stimulus onset, as indexed by a modulation of the N170 or M170 peak of the face-selective evoked-response potential (although some studies have failed to show such emotional modulation of the face-selective ERP; for reviews, see Vuilleumier & Pourtois, 2007). It has recently been reported that not only does the N170 show different latencies for viewing different facial emotions, but also that it reflects an integration of visual information specific to each expression, beginning 50 ms prior to, and ending at the peak of, this ERP (Schyns, Petro, & Smith, 2007). For all facial expressions tested, this integration begins at the eyes and moves down the face, stopping once the diagnostic information for the relevant expression has been resolved (e.g., at the eyes for fear expressions, the corners of the nose for disgust, and the mouth for happiness). It is the behavioral goal of emotion classification that determines when the integration of facial information ceases, which suggests top-down cognitive control of processing in occipitotemporal cortex, perhaps from prefrontal regions (Schyns et al., 2007).

Visual Cortex Regions in Whole-Body Emotion Recognition: Extrastriate Body Area and Fusiform Body Area

Like faces, the form of the human (or primate) body is a category of visual object for which there is both selectivity and functional specialization in higher-level visual cortices. Evidence for

body-selective visual mechanisms comes from studies of both humans and non-human primates (reviewed by Peelen & Downing, 2007). In humans, the evidence points to two distinct regions, dubbed the extrastriate body area (EBA), located in lateral occipitotemporal cortex (Downing et al., 2001), and the fusiform body area (FBA), located in fusiform gyrus (Peelen & Downing, 2005; Schwarzlose, Baker, & Kanwisher, 2005). The EBA and FBA respond selectively to viewing human bodies and body parts compared with objects, faces, and other control stimuli, despite considerable anatomical overlap between the FBA and the face-selective FFA (Peelen & Downing, 2005; Schwarzlose et al., 2005) and between the EBA, motion processing area V5/MT, and object-form-selective lateral occipital complex (Downing, Wiggett, & Peelen, 2007).

Paralleling the findings with facial expressions, research on emotional body perception has similarly reported enhanced activation in visual cortex when people perceive emotional bodies and body parts. Such modulation by emotional bodies was consistently reported in the fusiform gyrus (e.g., de Gelder et al., 2004), lateral occipitotemporal cortex (e.g., Grèzes et al., 2007) and STS (Grèzes et al., 2007; Pichon et al., 2007). For example, de Gelder and colleagues have shown that fusiform and occipital gyri, as well as the amygdala, are activated during passive viewing of static whole-body postures of fear, relative to emotionally neutral postures (de Gelder et al., 2004; Hadjikhani & de Gelder, 2003). Fearful body actions have also been shown to activate right middle temporal gyrus, in the region of the EBA and overlapping MT/V5, irrespective of whether these were presented as static or dynamic images (Grèzes et al., 2007). Taken together, these findings raise the intriguing possibility that perceiving emotion signals from the body might modulate precisely those populations of neurons that code for the viewed stimulus category (see Sugase, Yamane, Ueno, & Kawano, 1999), instead of reflecting "synergies" between the perception of facial and bodily expressions (de Gelder et al., 2004), or a global boost to all visual processing in extrastriate visual cortex. There is now evidence of just such category-specific emotional modulation. Peelen et al. (2007) found increased activation in the EBA when people viewed angry, disgusted, happy, and fearful (but not sad) body movements (for subsequent intensity ratings on emotion scales), compared to neutral controls, and increased activation in the FBA for angry, disgusted, and happy (but not fearful or sad) body movements. Importantly, multi-voxel pattern analysis showed that the strength of this emotional modulation was related, on a voxel-by-voxel basis, to the degree of body selectivity, while there was no relation with the degree of selectivity for faces, supporting the idea that emotional cues from body movements produce topographically selective influences on category-specific populations of neurons in the visual cortex. Furthermore, across subjects, amygdala responses to emotional bodies positively correlated with the modulation of the EBA and FBA but not the FFA. This result parallels the findings of correlations between amygdala and fusiform activity to facially expressed emotions, discussed above, and thus implicates feedback modulatory influences on visual cortex from the amygdala, but in addition suggests that this modulatory feedback is category-specific.

We suggest that the role of the EBA in emotion perception is similar to that of the OFA (with the caveat that their respective functions in body and face processing per se are not entirely analogous). In particular, this body-selective region is critically involved in processing structural cues related to the form of the human body, information which may then be fed to somatosensory cortices to aid emotion recognition via simulation (although there is currently no direct evidence to support this latter proposal). It might also be the case that the FBA and FFA play analogous roles in emotion perception from bodies and faces, respectively, acting in concert with the EBA (for bodily expressions) and OFA (for facial expressions) to extract expression-relevant structural cues. Presumably, later emotional modulation of the EBA and FBA specifically by bodily expressions reflects prioritized or enhanced visual processing of those bodily forms so as to allow more efficient or accurate discrimination of others' emotions.

Yet there is also evidence suggesting a possible additional role in emotion recognition for the EBA, at least, via simulation. This evidence shows that the EBA is sensitive to certain differences between self and other; in particular, right EBA activity is increased when (a) bodies (Chan, Peelen, & Downing, 2004) or body parts (Saxe, Jamal, & Powell, 2006) are presented in allocentric relative to egocentric views, regardless of whether the image shows the observer's own body or that of another person (Chan et al., 2004); and (b) when visual information (the movement of a cursor) is consistent with an assumed other person's joystick movements rather than with one's own (David et al., 2007). Indeed, this latter finding has recently been confirmed in a TMS study, suggesting that the EBA plays a critical role in identifying ourselves as agents of self-generated movements (David et al., 2007). Taken together, this evidence raises the intriguing possibility that the EBA could be part of an emotion simulation network, helping to distinguish the simulated body states or emotional actions of another from one's own body states or actions. Such a network could also involve posterior parietal cortex, which has been implicated in the detection of sensorimotor congruence and the sense of agency (e.g., Chaminade & Decety, 2002; Jeannerod, 2004), and which, in David et al.'s (2007) study, showed enhanced functional connectivity with the EBA when visual feedback was incongruent with the participants' own movements. Despite this evidence of EBA involvement in distinguishing self from others and the evidence of emotional modulation of EBA, a recent study indicates that it is not specifically involved in empathy for pain (Lamm & Decety, 2008), which raises doubts as to whether it has a direct role in simulating the emotional states of others.

Prefrontal Cortex in Emotion Perception and Experience

Prefrontal Cortex in Face Emotion Recognition: Ventromedial Frontal Cortex and Left Inferior Frontal/Premotor Cortex

The prefrontal cortex has repeatedly been implicated in emotional face processing, with two regions most frequently identified as

critical: ventromedial and (left or bilateral) inferior frontal or premotor regions. Functional imaging studies have yielded somewhat inconsistent results, due perhaps to differences in task requirements, stimuli used, specific emotions tested, or differential involvement of PFC regions in the processing of specific emotions. However, three regions are reported most consistently during viewing, categorizing, matching, or holding in memory emotional faces (with tasks varying between studies): orbitofrontal cortex or OFC (generally but not always ventral, and therefore included within the designation of ventromedial prefrontal cortex; Blair, Morris, Frith, Perrett, & Dolan, 1999; Dolan et al., 1996; Vuilleumier et al., 2001), anterior cingulate cortex (ACC; e.g., Blair et al., 1999; Dolan et al., 1996; Vuilleumier et al., 2001), and left or bilateral inferior frontal gyrus (e.g., Carr et al., 2003). Loss-of-function studies of subjects with prefrontal damage due to stroke, surgery, or fronto-temporal dementia have also implicated these regions: ventromedial and OFC (e.g., Heberlein, Padon, Gillihan, Farah, & Fellows, 2008; Hornak et al., 2003; Marinkovic, Trebon, Chauvel, & Halgren, 2000); ACC (Hornak et al., 2003) and inferior frontal gyrus or the frontal opercular region more generally (e.g., Adolphs et al., 2000; Marinkovic et al., 2000).

Two somewhat different explanations for the involvement of ventromedial and/or orbitofrontal regions in emotion recognition both relate to simulation, albeit of a somewhat different form than is played by the regions described above. For example, OFC and ACC regions are thought to play a role in representing arousal, and this role has been invoked to explain medial PFC involvement in recognizing high-arousal emotions (Adolphs, 2002; Öngur & Price, 2000). Two recent studies have examined the effects of PFC damage on both emotional experience and recognition (Heberlein et al., 2008; Hornak et al., 2003), and both of these studies found a relationship between measures of specifically sad mood experience and sadness recognition. Heberlein et al. (2008) found that in people with ventromedial frontal (but not other prefrontal) cortex damage, there was a relationship between ratings of sad faces and lab-induced sad emotion. They explained these findings by positing a model which bears some similarity to that proposed by Barrett et al. (2007), discussed above, for the amygdala: people with damage to VMF cortices may be less sensitive to others' emotional expressions, including of distress, and therefore may show less sad mood in situations including laboratory mood inductions. In support of this idea, there was a higher correlation between sad face recognition and induced sad mood for a film-based sadness induction, than between sad face recognition and induced mood for an autobiographical recall-based sadness induction (Heberlein et al., 2008). This fits theoretically with a general role for VMF cortex in the top-down direction of attention to others' emotions and other salient emotional stimuli, which we later incorporate in a model of emotion recognition (see the section, Summary and a Simulation Model of Emotion Recognition Incorporating Both Face and Whole-Body Data).

In contrast, the role of premotor or frontal opercular areas has generally been understood in terms of "mirror-neuron-like" activity (e.g., Carr et al., 2003; Winkielman et al., 2009). In support of this model, a posterior section of left inferior frontal gyrus at the frontal operculum is active bilaterally when people imitate emotional facial expressions (Lee, Josephs, Dolan, & Critchley, 2006). It is unclear, at present, how to integrate these findings with the model of emotional face processing that we described above, relating the roles of the amygdala, extrastriate cortex, and right somatosensory regions.

Prefrontal Cortex in Voice or Whole-Body Emotion Recognition: Ventromedial Frontal Cortex and Left Inferior Frontal/Premotor Cortex

Given its repeated, if inconsistent, implication in emotion recognition from faces, it is somewhat surprising how little evidence there is for prefrontal cortical involvement in emotion recognition from other nonverbal cues. A detailed lesion study (Hornak et al., 2003) compared emotion recognition from faces and vocal prosody in groups with damage to several different prefrontal regions. Though they found no group-wise impairment on facial emotion identification in a labeling task (some individuals with OFC or unilateral dorsomedial damage were impaired), both OFC and dorsomedial PFC were reliably associated with voice emotion recognition. The authors posit that vocal prosody is a more sensitive test of emotion recognition ability than facial expressions because it is less easily translated into verbalizable cues (Hornak et al., 2003).

Premotor and inferior frontal regions have been implicated in several studies of emotion recognition based on both prosodic and whole-body cues. Thus, premotor cortex is active, especially on the right side, when subjects process emotional prosody (George et al., 1996; Imaizumi et al., 1997). These data converge with evidence from Adolphs and colleagues' (2002) lesion overlap study of emotional prosody recognition, which found a focus of lesion overlap around the left frontal operculum (i.e., posterior left inferior frontal gyrus), in addition to the above-mentioned right somatosensory regions. It is unclear, however, why these findings do not agree with respect to laterality. Though Heberlein and colleagues' (2004) lesion overlap study did *not* identify inferior frontal regions as critical for whole-body emotion recognition (rather, these were found to be critical for personality trait judgments from dynamic whole-body stimuli), imaging studies have reported inferior frontal and premotor activity during viewing of dynamic whole-body emotion cues (e.g., Grèzes et al., 2007).

Summary and a Simulation Model of Emotion Recognition Incorporating Both Face and Whole-Body Data

The evidence we have reviewed here suggests considerable overlap between the neural systems involved in recognizing emotion from face cues and from other nonverbal cues, and suggests as well that not all instances of "shared substrates" between emotion recognition and emotional experience are evidence for simulation, in the sense of representing another's behavior and/or feelings in one's self. In addition, based on the evidence reviewed here, we might usefully distinguish between different

senses of simulation by focusing on the varied roles suggested for different emotion-recognition-relevant neural regions.

(1) *Right somatosensory cortex and representing body sensation.* Because of their defined role in representing self body sensations, it is difficult to conceive of a role for somatosensory cortices that is not somehow a simulation account: part of how one knows what another person feels like when that person acts a certain way is by representing what oneself feels like when one acts in a similar way. This presumed role is bolstered by the findings of somatosensory cortex involvement across multiple stimulus types and, indeed, multiple modalities of nonverbal cues. However, it is at present unclear whether the contribution of these regions is body-part specific (i.e., a more proprioceptive "what my arm feels like when my arm is expressing anger" role) or more global (i.e., a whole-body gestalt and/or somatovisceral representation). In either case, this role in emotion recognition appears to extend across at least multiple emotions; further evidence from studies that follow up the TMS laterality-specific results will be welcome.

(2) *The amygdala, fear detection and fear response: simulation or just shared substrates?* In contrast to somatosensory cortex, the amygdala's role in emotion recognition processes appears to be most extensive for, although not restricted to, negative or potentially threat-related information. Furthermore, the amygdala and related structures play many different roles related to both sensory processes (e.g., responding to specific threat-related environmental features—including facial features—and directing visual attention to these objects) and endocrine and motor processes (e.g., coordinating responses to detected threats). As may have emerged from the multiple instances of the phrase "the evidence . . . is mixed" in this section of our review, it is at present unclear how to extend what is known about the role of the amygdala in facial expression processing to other forms and modalities of nonverbal emotional communication; results are strikingly variable across studies. Thus, while the amygdala surely plays roles in both the experience of fear and the recognition of at least certain types of fearful expressions, these roles may depend on fairly unrelated processes in which the amygdala participates, making it fit less neatly into a "simulation" model, despite still fitting a "shared substrates" model.[5] Ventromedial prefrontal cortices may similarly play roles in both emotion recognition and emotional experience without an actual overlapping "simulation" process. That said, it remains to be confirmed whether the amygdala has a more direct role in simulation processes underlying emotion recognition via activating somatic states associated with the viewed emotional expression. This role would facilitate recognition via "as-if" circuits (Damasio, 1999) or even actual motor behavior, a form of simulation like that in which somatosensory cortices are posited to play a role.

(3) *Premotor areas and simulating expressions.* Premotor regions are thought to play a role somewhat similar to that of somatosensory cortices: while somatosensory cortices represent what one *feels like* in associating an observed expression with an emotional concept or label, premotor cortices may represent one's own *motor plans* toward the same end, in order to associate an observed expression with an emotional concept or label. However, unlike for somatosensory regions, there is less evidence that this role extends to whole-body emotional expressions; further work is needed to address this discrepancy.

(4) *Visual regions: participating via connections to regions involved in simulation.* The relevance to simulation of the visual regions we reviewed above lies in their interactions with other regions, rather than direct roles in simulation per se. Note, however, that the EBA results (e.g. activity differentiating self- vs. other-centered views of body parts) may prove an exception to this generalization.

To briefly summarize all of the above, current evidence suggests the following hierarchical model, involving both feedforward and feedback connections between visual and somatosensory cortices and regions involved in processing emotional information: Visual cues important for discriminating facial emotional expressions are extracted at early stages of visual processing, largely in cortices specialized for face and body processing, and are fed forward to somatosensory cortices. These regions are implicated in simulating certain aspects of the body states associated with the viewed emotional state—possibly including proprioceptive (and thus body part-specific) and/or somatovisceral sensations. (Note that we might postulate a similar model for auditory cues, but that very little evidence currently exists to support such a model.)

Overlapping in time are at least two other separate sets of processes, which may also interlink. In one, the amygdala responds to specific features signaling the emotional significance of the viewed expressions, enabling, via feedback connections, enhanced or prioritized visual processing of those expressions (a consequence of which may be to allow more efficient or accurate discrimination of others' emotions).[6] Thus, for example, when the presence of widened eye whites is detected, the facial features (and other context) surrounding this feature are processed in greater detail. In another set of processes, information about the current processing goals (e.g., that one's task is to identify the viewed expression) and other motivational information (e.g., the proclivity to attend to others' emotional states) are fed back from prefrontal to early visual cortices to influence the extent to which information specific to different emotional expressions is integrated. In theory, the results of these latter two sets of processes could in turn influence continuing or subsequent simulation processes in premotor and somatosensory cortices. Providing evidence for (and more specific models of) such feedback processes would be yet another welcome area of future research.

Notes

1 Please note that due to space constraints, we cited just one or two articles to support numerous findings which have been documented by several independent replications and extensions. These are generally denoted by an "e.g." before a reference or abbreviated list of references.

2 As reviewed in another article in this volume (Glenberg, Webster, Mouilso, Havas, & Lindeman, 2009), manipulating spontaneous facial expressions also affects judgments of sentence valence.

3 The term *perception* typically refers to processes that make explicit the distinct features of stimuli and their geometric configurations to allow discrimination among different stimuli on the basis of their appearance. By *discrimination* of emotional expressions we are referring to abilities such as deciding whether two (or more) stimuli (e.g., faces) have the same or different emotion, or matching a target face with one of several other faces based on their expression. In contrast, *recognition* refers to processes that require additional knowledge that could not be obtained solely from an inspection of the features of the stimulus, such as conceptual knowledge of a particular emotion, the retrieval of appropriate lexical labels, and the perception of the emotional response (or a central representation thereof) that the stimulus triggers in the observer. Tasks that tap recognition, rather than purely perceptual, processes include those in which participants are asked to use selected emotion words to identify emotional expressions, or to label or describe the viewed emotional state in a more open-ended way, or to rate how much of a given emotion is represented in a stimulus. As our main interest here is in the recognition of emotional expressions, we shall reserve the term perception for processes that occur relatively early in time subsequent to the onset of the stimulus that are presumed to rely largely on early sensory cortices and that do not rely on or necessarily invoke conceptual or lexical knowledge.

4 Note, however, that in none of these studies were participants with right somatosensory cortex damage at chance levels of performance; the role played by these regions in emotion recognition extends across multiple specific emotions, but humans must have multiple, at least partly independent, pathways for recognizing emotion.

5 As noted in the section "The Amygdala in Emotion Perception and Experience", there may well be correlations between these roles across individuals: for example, Barrett's recent finding of amygdala sensitivity to negative social cues correlating with month-long records of experienced mood, explained via a correlation between amygdala-mediated increased awareness of negative social cues and a corresponding increase in the normal, negative response to such awareness.

6 Note that the amygdala may be involved at an even earlier stage, somehow directing the visual system to seek out and attend to certain salient visual features; a key topic for ongoing research is in elucidating the nature of this interaction.

References

Adolphs, R. (2002). Recognizing emotion from facial expressions: Psychological and neurological mechanisms. *Behavioral and Cognitive Neuroscience Reviews*, *1*(1), 21–61.

Adolphs, R., Baron-Cohen, S., & Tranel, D. (2002). Impaired recognition of social emotions following amygdala damage. *Journal of Cognitive Neuroscience*, *14*(8), 1264–1274.

Adolphs, R., Damasio, H., Tranel, D., Cooper, G., & Damasio, A. R. (2000). A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. *The Journal of Neuroscience*, *20*(7), 2683–2690.

Adolphs, R., Gosselin, F., Buchanan, T. W., Tranel, D., Schyns, P., & Damasio, A. R. (2005). A mechanism for impaired fear recognition after amygdala damage. *Nature*, *433*(7021), 68–72.

Adolphs, R., & Tranel, D. (1999). Intact recognition of emotional prosody following amygdala damage. *Neuropsychologia*, *37*, 1285–1292.

Adolphs, R., & Tranel, D. (2003). Amygdala damage impairs emotion recognition from scenes only when they contain facial expressions. *Neuropsychologia*, *41*, 1281–1289.

Adolphs, R., Tranel, D., & Damasio, A. R. (1998). The human amygdala in social judgment. *Nature*, *393*, 470–474.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1995). Fear and the human amygdala. *The Journal of Neuroscience*, *15*(9), 5879–5891.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., et al. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, *37*, 1111–1117.

Amaral, D. G. (2002). The primate amygdala and the neurobiology of social behavior: Implications for understanding social anxiety. *Biological Psychiatry*, *51*, 11–17.

Amaral, D. G., Behniea, H., & Kelly, J. L. (2003). Topographic organization of projections from the amygdala to the visual cortex in the macaque monkey. *Neuroscience*, *118*(4), 1099–1120.

Anderson, A. K., & Phelps, E. A. (1998). Intact recognition of vocal expressions of fear following bilateral lesions of the amygdala. *Neuroreport*, *9*, 3607–3613.

Anderson, A. K., & Phelps, E. A. (2002). Is the human amygdala critical for the subjective experience of emotion? Evidence of intact dispositional affect in patients with amygdala lesions. *Journal of Cognitive Neuroscience*, *14*(5), 709–720.

Atkinson, A. P., Dittrich, W. H., Gemmell, A. J., & Young, A. W. (2004). Emotion perception from dynamic and static body expressions in point-light and full-light displays. *Perception*, *33*(6), 717–746.

Atkinson, A. P., Heberlein, A. S., & Adolphs, R. (2007). Spared ability to recognise fear from static and moving whole-body cues following bilateral amygdala damage. *Neuropsychologia*, *45*(12), 2772–2782.

Atkinson, A. P., Heberlein, A. S., & Adolphs, R. (in press). Are people special? A brain's eye view. In R. B. Adams, Jr., N. Ambady, K. Nakayama, & S. Shimojo (Eds.), *The science of social vision*. Oxford: Oxford University Press.

Bachorowski, J., & Owren, M. J. (2008). Vocal expressions of emotion. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett (Eds.), *Handbook of emotions* (3rd ed., pp. 196–210). New York: Guilford Press.

Barrett, L. F., Bliss-Moreau, E., Duncan, S. L., Rauch, S. L., & Wright, C. I. (2007). The amygdala and the experience of affect. *Social Cognitive and Affective Neuroscience*, *2*, 73–83.

Barsalou, L. W., Kyle Simmons, W., Barbey, A. K., & Wilson, C. D. (2003). Grounding conceptual knowledge in modality-specific systems. *Trends in Cognitive Sciences*, *7*(2), 84–91.

Bechara, A., Tranel, D., Damasio, H., Adolphs, R., Rockland, C., & Damasio, A. R. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science*, *269*, 1115–1118.

Benowitz, L. I., Bear, D. M., Rosenthal, R., Mesulam, M. M., Zaidel, E., & Sperry, R. W. (1983). Hemispheric specialization in nonverbal communication. *Cortex*, *19*, 5–11.

Berntson, G. G., Bechara, A., Damasio, H., Tranel, D., & Cacioppo, J. T. (2007). Amygdala contribution to selective dimensions of emotion. *Social Cognitive and Affective Neuroscience*, *2*, 123–129.

Blair, R. J., Morris, J. S., Frith, C. D., Perrett, D. I., & Dolan, R. J. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain*, *122*, 883–893.

Blake, R., & Shiffrar, M. (2007). Perception of human motion. *Annual Review of Psychology*, *58*, 47–73.

Blakemore, S.-J., & Decety, J. (2001). From the perception of action to the understanding of intention. *Nature Reviews Neuroscience*, *2*, 561–567.

Bonda, E., Petrides, M., Ostry, D., & Evans, A. (1996). Specific involvement of human parietal systems and the amygdala in the perception of biological motion. *The Journal of Neuroscience*, *16*(11), 3737–3744.

Bowers, D., Bauer, R. M., Coslett, H. B., & Heilman, K. M. (1985). Processing of faces by patients with unilateral hemisphere lesions: I. Dissociation between judgments of facial affect and facial identity. *Brain and Cognition*, *4*, 258–272.

Calder, A. J., Keane, J., Cole, J., Campbell, R., & Young, A. W. (2000). Facial expression recognition by people with Mobius syndrome. *Cognitive Neuropsychology*, *17*, 73–87.

Calder, A. J., Lawrence, A. D., & Young, A. W. (2001). Neuropsychology of fear and loathing. *Nature Reviews Neuroscience*, 2, 352–363.

Calder, A. J., & Young, A. W. (2005). Understanding the recognition of facial identity and facial expression. *Nature Reviews Neuroscience*, 6, 641–651.

Calder, A. J., Young, A. W., Perrett, D. I., Hodges, J. R., & Etcoff, N. L. (1996). Facial emotion recognition after bilateral amygdala damage: Differentially severe impairment of fear. *Cognitive Neuropsychology*, 13, 699–745.

Canli, T., Sivers, H., Whitfield, S. L., Gotlib, I. H., & Gabrieli, J. D. (2002). Amygdala response to happy faces as a function of extraversion. *Science*, 296, 2191.

Carr, L., Iacoboni, M., Dubeau, M. C., Mazziotta, J. C., & Lenzi, G. L. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of the National Academy of Sciences USA*, 100, 5497–5502.

Chaminade, T., & Decety, J. (2002). Leader or follower? Involvement of the inferior parietal lobule in agency. *Neuroreport*, 13, 1975–1978.

Chan, A. W., Peelen, M. V., & Downing, P. E. (2004). The effect of viewpoint on body representation in the extrastriate body area. *Neuroreport*, 15(15), 2407–2410.

Crawford, L. E. (2009). Conceptual metaphors of affect. *Emotion Review*, 1(2), 129–139.

Damasio, A. R. (1989). Time-locked multiregional retroactivation: A systems-level proposal for the neural substrates of recall and recognition. *Cognition*, 33(1–2), 25–62.

Damasio, A. R. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. New York: Harcourt Brace.

Darwin, C. (1872/1965). *The expression of the emotions in man and animals.* (Reprinted from the D. Appleton and Company authorized edition.) Chicago: The University of Chicago Press.

David, N., Cohen, M. X., Newen, A., Bewernick, B. H., Shah, N. J., Fink, G. R., et al. (2007). The extrastriate cortex distinguishes between the consequences of one's own and others' behavior. *Neuroimage*, 36(3), 1004–1014.

Davidson, R. J. (1992). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition*, 20, 125–151.

de Gelder, B. (2006). Towards the neurobiology of emotional body language. *Nature Reviews Neuroscience*, 7(3), 242–249.

de Gelder, B., Snyder, J., Greve, D., Gerard, G., & Hadjikhani, N. (2004). Fear fosters flight: A mechanism for fear contagion when perceiving emotion expressed by a whole body. *Proceedings of the National Academy of Sciences USA*, 101, 16701–16706.

Decety, J., & Grèzes, J. (1999). Neural mechanisms subserving the perception of human actions. *Trends in Cognitive Sciences*, 3(5), 172–178.

Decety, J., & Lamm, C. (2006). Human empathy through the lens of social neuroscience. *Scientific World Journal, 6*, 1146–1163.

de Meijer, M. (1989). The contribution of general features of body movement to the attribution of emotions. *Journal of Nonverbal Behavior*, 13(4), 247–268.

Dimberg, U. (1982). Facial reactions to facial expressions. *Psychophysiology*, 19(6), 643–647.

Dimberg, U., Thunberg, M., & Elmehed, K. (2000). Unconscious facial reactions to emotional facial expressions. *Psychological Science*, 11(1), 86–89.

di Pellegrino, G., Fadiga, L., Fogassi, L., Gallese, V., & Rizzolatti, G. (1992). Understanding motor events: A neurophysiological study. *Experimental Brain Research*, 91(1), 176–180.

Dittrich, W. H., Troscianko, T., Lea, S. E., & Morgan, D. (1996). Perception of emotion from dynamic point-light displays represented in dance. *Perception*, 25(6), 727–738.

Dolan, R. J., Fletcher, P. C., Morris, J. S., Kapur, N., Deakin, J. F. W., & Frith, C. D. (1996). Neural activation during covert processing of positive emotional facial expressions. *Neuroimage*, 4, 194–200.

Downing, P. E., Jiang, Y., Shuman, M., & Kanwisher, N. (2001). A cortical area selective for visual processing of the human body. *Science*, 293, 2470–2473.

Downing, P. E., Wiggett, A. J., & Peelen, M. V. (2007). Functional magnetic resonance imaging investigation of overlapping lateral occipitotemporal activations using multi-voxel pattern analysis. *Journal of Neuroscience*, 27(1), 226–233.

Ekman, P., & Friesen, W. (1976). *Pictures of facial affect*. Palo Alto, CA: Consulting Psychologists.

Fanselow, M. S., & LeDoux, J. E. (1999). Why we think plasticity underlying Pavlovian fear conditioning occurs in the basolateral amygdala. *Neuron*, 23(2), 229–232.

Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Science*, 8(9), 396–403.

George, M. S., Parekh, P. I., Rosinsky, N., Ketter, T. A., Kimbrell, T. A., Heilman, K. M., et al. (1996). Understanding emotional prosody activates right hemisphere regions. *Archives of Neurology*, 53, 665–670.

Glenberg, A., Webster, B., Mouilso, E., Havas, D., & Lindeman, L. (2009). Gender, emotion, and the embodiment of language comprehension. *Emotion Review*, 1(2), 151–161.

Grandjean, D., Banziger, T., & Scherer, K. R. (2006). Intonation as an interface between language and affect. *Progress in Brain Research*, 156, 235–268.

Grèzes, J., Pichon, S., & de Gelder, B. (2007). Perceiving fear in dynamic body expressions. *Neuroimage*, 35(2), 959–967.

Hadjikhani, N., & de Gelder, B. (2003). Seeing fearful body expressions activates the fusiform cortex and amygdala. *Current Biology*, 13, 2201–2205.

Halgren, E., Walter, R. D., Cherlow, D. G., & Crandall, P. H. (1978). Mental phenomena evoked by electrical stimulation of the human hippocampal formation and amygdala. *Brain*, 101(1), 83–117.

Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002). Serotonin transporter genetic variation and the response of the human amygdala. *Science*, 297, 400–403.

Harrison, N. A., Singer, T., Rotshtein, P., Dolan, R. J., & Critchley, H. D. (2006). Pupillary contagion: Central mechanisms engaged in sadness processing. *Social Cognitive and Affective Neuroscience*, 1(1), 5–17.

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Science*, 4(6), 223–233.

Heberlein, A. S., Adolphs, R., Tranel, D., & Damasio, H. (2004). Cortical regions for judgments of emotions and personality traits from point-light walkers. *Journal of Cognitive Neuroscience*, 16(7), 1143–1158.

Heberlein, A. S., Padon, A. A., Gillihan, S. J., Farah, M. J., & Fellows, L. K. (2008). Ventromedial frontal lobe plays a critical role in facial emotion recognition. *Journal of Cognitive Neuroscience*, 20(4), 721–733.

Heberlein, A. S., & Saxe, R. (2005). Dissociation between emotion and personality judgments: Convergent evidence from functional neuroimaging. *Neuroimage*, 28, 770–777.

Hess, U., & Blairy, S. (2001). Facial mimicry and emotional contagion to dynamic emotional facial expressions and their influence on decoding accuracy. *International Journal of Psychophysiology*, 40(2), 129–141.

Hornak, J., Bramham, J., Rolls, E. T., Morris, R. G., O'Doherty, J., Bullock, P. R., et al. (2003). Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. *Brain*, 126, 1691–1712.

Hutchison, W. D., Davis, K. D., Lozano, A. M., Tasker, R. R., & Dostrovsky, J. O. (1999). Pain-related neurons in the human cingulate cortex. *Nature Neuroscience*, 2(5), 403–405.

Iacoboni, M., Woods, R., Brass, M., Bekkering, H., Mazziotta, J., & Rizzolati, G. (1999). Cortical mechanisms of human imitation. *Science*, 286, 2526–2528.

Imaizumi, S., Mori, K., Kiritani, S., Kawashima, R., Sugiura, M., Fukuda, H., et al. (1997). Vocal identification of speaker and emotion activates different brain regions. *Neuroreport*, 8(12), 2809–2812.

James, W. (1932). A study of the expression of bodily posture. *Journal of General Psychology*, 7, 405–437.

Jeannerod, M. (2004). Visual and action cues contribute to the self–other distinction. *Nature Neuroscience*, 7(5), 422–423.

Johansson, G. (1973). Visual perception of biological motion and a model of its analysis. *Perception and Psychophysics*, 14, 202–211.

Kanwisher, N., & Yovel, G. (2006). The fusiform face area: A cortical region specialized for the perception of faces. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *361*, 2109–2128.

Keillor, J. M., Barrett, A. M., Crucian, G. P., Kortenkamp, S., & Heilman, K. M. (2002). Emotional experience and perception in the absence of facial feedback. *Journal of the International Neuropsychological Society*, *8*(1), 130–135.

Klüver, H., & Bucy, P. C. (1997). Preliminary analysis of functions of the temporal lobes in monkeys. 1939 [classical article]. *Journal of Neuropsychiatry and Clinical Neurosciences*, *9*, 606–620.

Kucharska-Pietura, K., Phillips, M. L., Gernand, W., & David, A. S. (2003). Perception of emotions from faces and voices following unilateral brain damage. *Neuropsychologia*, *41*, 1082–1090.

LaBar, K. S., LeDoux, J. E., Spencer, D. D., & Phelps, E. A. (1995). Impaired fear conditioning following unilateral temporal lobectomy in humans. *The Journal of Neuroscience*, *15*, 6846–6855.

Lamm, C., & Decety, J. (2008). Is the extrastriate body area (EBA) sensitive to the perception of pain in others? An fMRI investigation. *Cerebral Cortex*, *18*, 2369–2373.

Lee, T. W., Josephs, O., Dolan, R. J., & Critchley, H. D. (2006). Imitating expressions: Emotion-specific neural substrates in facial mimicry. *Social Cognitive and Affective Neuroscience*, *1*(2), 122–135.

Liu, J., Harris, A., & Kanwisher, N. (2002). Stages of processing in face perception: An MEG study. *Nature Neuroscience*, *5*(9), 910–916.

Marinkovic, K., Trebon, P., Chauvel, P., & Halgren, E. (2000). Localized face processing by the human prefrontal cortex: Face-selective intracerebral potentials and post-lesion deficits. *Cognitive Neuropsychology*, *17*, 187–199.

Morris, J. S., Friston, K. J., Buchel, C., Frith, C. D., Young, A. W., Calder, A. J., et al. (1998). A neuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain*, *121*, 47–57.

Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., et al. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature*, *383*, 812–815.

Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdala mediating "unseen" fear. *Proceedings of the National Academy of Sciences USA*, *96*, 1680–1685.

Morrison, I. (2007). A motivational perspective on the neural bases of empathy. In T. F. D. Farrow & P. W. R. Woodruff (Eds.), *Empathy in mental illness and health*. Cambridge: Cambridge University Press.

Morrison, I., Lloyd, D., di Pellegrino, G., & Roberts, N. (2004). Vicarious responses to pain in anterior cingulate cortex: Is empathy a multisensory issue? *Cognitive, Affective, and Behavioral Neuroscience*, *4*(2), 270–278.

Niedenthal, P. M., Barsalou, L. W., Winkielman, P., Krauth-Gruber, S., & Ric, F. (2005). Embodiment in attitudes, social perception, and emotion. *Personality and Social Psychology Review*, *9*(3), 184–211.

Niedenthal, P. M., Brauer, M., Halberstadt, J. B., & Innes-Ker, A. H. (2001). When did her smile drop? Facial mimicry and the influences of emotional state on the detection of change in emotional expression. *Cognition & Emotion*, *15*, 853–864.

Öngur, D., & Price, J. L. (2000). The organization of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. *Cerebral Cortex*, *10*(3), 206–219.

Peelen, M. V., Atkinson, A. P., Andersson, F., & Vuilleumier, P. (2007). Emotional modulation of body-selective visual areas. *Social Cognitive and Affective Neuroscience*, *2*(4), 274–283.

Peelen, M. V., & Downing, P. E. (2005). Selectivity for the human body in the fusiform gyrus. *Journal of Neurophysiology*, *93*(1), 603–608.

Peelen, M. V., & Downing, P. E. (2007). The neural basis of visual body perception. *Nature Reviews Neuroscience*, *8*(8), 636–648.

Pessoa, L., McKenna, M., Gutierrez, E., & Ungerleider, L. G. (2002). Neural processing of emotional faces requires attention. *Proceedings of the National Academy of Sciences USA*, *99*, 11458–11463.

Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage*, *16*(2), 331–348.

Phillips, M. L., Williams, L. M., Heining, M., Herba, C. M., Russell, T., Andrew, C., et al. (2004). Differential neural responses to overt and covert presentations of facial expressions of fear and disgust. *Neuroimage*, *21*(4), 1484–1496.

Phillips, M. L., Young, A. W., Scott, S. K., Calder, A. J., Andrew, C., Giampietro, V., et al. (1998). Neural responses to facial and vocal expressions of fear and disgust. *Proceedings of the Royal Society B: Biological Sciences*, *265*, 1809–1817.

Pichon, S., de Gelder, B., & Grèzes, J. (2007). Emotional modulation of visual and motor areas by dynamic body expressions of anger. *Social Neuroscience*, EPub 1–14.

Ping, R., Dhillon, S., & Beilock, S. (2009). Reach for what you like: The body's role in shaping preferences. *Emotion Review*, *1*(2), 140–150.

Pitcher, D., Garrido, L., Walsh, V., & Duchaine, B. (2008). TMS disrupts the perception and embodiment of facial expressions [Abstract]. *Journal of Vision*, *8*(6), 700.

Pitcher, D., Walsh, V., Yovel, G., & Duchaine, B. (2007). TMS evidence for the involvement of the right occipital face area in early face processing. *Current Biology*, *17*(18), 1568–1573.

Pourtois, G., Sander, D., Andres, M., Grandjean, D., Reveret, L., Olivier, E., et al. (2004). Dissociable roles of the human somatosensory and superior temporal cortices for processing social face signals. *European Journal of Neuroscience*, *20*(12), 3507–3515.

Preston, S. D., & de Waal, F. B. M. (2002). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences*, *25*, 1–72.

Prinz, W. (1997). Perception and action planning. *European Journal of Cognitive Psychology*, *9*, 129–154.

Rosenthal, R., Hall, J. A., DiMatteo, M. R., Rogers, P. L., & Archer, D. (1979). *Sensitivity to nonverbal communication: The PONS test*. Baltimore: John Hopkins University Press.

Ross, E. D., Thompson, R. D., & Yenkosky, J. (1997). Lateralization of affective prosody in brain and the callosal integration of hemispheric language functions. *Brain Lang*, *56*(1), 27–54.

Sander, K., & Scheich, H. (2001). Auditory perception of laughing and crying activates human amygdala regardless of attentional state. *Cognitive Brain Research*, *12*(2), 181–198.

Saxe, R., Jamal, N., & Powell, L. (2006). My body or yours? The effect of visual perspective on cortical body representations. *Cerebral Cortex*, *16*(2), 178–182.

Schwarzlose, R. F., Baker, C. I., & Kanwisher, N. (2005). Separate face and body selectivity on the fusiform gyrus. *Journal of Neuroscience*, *25*, 11055–11059.

Schyns, P. G., Bonnar, L., & Gosselin, F. (2002). Show me the features! Understanding recognition from the use of visual information. *Psychological Science*, *13*(5), 402–409.

Schyns, P. G., Petro, L. S., & Smith, M. L. (2007). Dynamics of visual information integration in the brain for categorizing facial expressions. *Current Biology*, *17*(18), 1580–1585.

Scott, S. K., Young, A. W., Calder, A. J., Hellawell, D. J., Aggleton, J. P., & Johnson, M. (1997). Impaired auditory recognition of fear and anger following bilateral amygdala lesions. *Nature*, *385*, 254–257.

Singer, T., Seymour, B., O'Doherty, J., Kaube, H., Dolan, R. J., & Frith, C. D. (2004). Empathy for pain involves the affective but not sensory components of pain. *Science*, *303*, 1157–1162.

Smith, M. L., Gosselin, F., & Schyns, P. G. (2004). Receptive fields for flexible face categorizations. *Psychological Science*, *15*(11), 753–761.

Sonnby-Borgström, M. (2002). Automatic mimicry reactions as related to differences in emotional empathy. *Scandinavian Journal of Psychology*, *43*(5), 433–443.

Sprengelmeyer, R., Young, A., Schroeder, U., Grossenbacher, P. G., Federlein, J., Buettner, T., et al. (1999). Knowing no fear. *Proceedings of the Royal Society B: Biological Science*, *266*, 2451–2456.

Strack, F., Martin, L. L., & Stepper, S. (1988). Inhibiting and facilitating conditions of the human smile: A nonobtrusive test of the facial feedback hypothesis. *Journal of Personality and Social Psychology*, *54*(5), 768–777.

Sugase, Y., Yamane, S., Ueno, S., & Kawano, K. (1999). Global and fine information coded by single neurons in the temporal visual cortex. *Nature*, *400*, 869–873.

Tranel, D., Gullickson, G., Koch, M., & Adolphs, R. (2006). Altered experience of emotion following bilateral amygdala damage. *Cognitive Neuropsychiatry*, *11*(3), 219–232.

Tsuchiya, N., Kawasaki, H., Howard, M. A., & Adolphs, R. (2008). Decoding frequency and timing of emotion perception from direct intracranial recordings in the human brain [Abstract]. *Journal of Vision*, *8*(6), 962.

van Rijn, S., Aleman, A., van Diessen, E., Berckmoes, C., Vingerhoets, G., & Kahn, R. S. (2005). What is said or how it is said makes a difference: Role of the right fronto-parietal operculum in emotional prosody as revealed by repetitive TMS. *European Journal of Neuroscience*, *21*(11), 3195–3200.

Vuilleumier, P. (2005). How brains beware: Neural mechanisms of emotional attention. *Trends in Neuroscience*, *21*(11), 585–594.

Vuilleumier, P., Armony, J. L., Driver, J., & Dolan, R. J. (2001). Effects of attention and emotion on face processing in the human brain: An event-related fMRI study. *Neuron*, *30*(3), 829–841.

Vuilleumier, P., & Driver, J. (2007). Modulation of visual processing by attention and emotion: Windows on causal interactions between human brain regions. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *362*, 837–855.

Vuilleumier, P., & Pourtois, G. (2007). Distributed and interactive brain mechanisms during emotion face perception: Evidence from functional neuroimaging. *Neuropsychologia*, *45*(1), 174–194.

Vuilleumier, P., Richardson, M. P., Armony, J. L., Driver, J., & Dolan, R. J. (2004). Distant influences of amygdala lesion on visual cortical activation during emotional face processing. *Nature Neuroscience*, *7*(11), 1271–1278.

Wallbott, H. G. (1991). Recognition of emotion from facial expression via imitation? Some indirect evidence for an old theory. *British Journal of Social Psychology*, *30*, 207–219.

Wallbott, H. G. (1998). Bodily expression of emotion. *European Journal of Social Psychology*, *28*, 879–896.

Whalen, P. J. (1998). Fear, vigilance, and ambiguity: Initial neuroimaging studies of the human amygdala. *Current Directions in Psychological Science*, *7*(6), 177–188.

Whalen, P. J., Kagan, J., Cook, R. G., Davis, F. C., Kim, H., Polis, S., et al. (2004). Human amygdala responsivity to masked fearful eye whites. *Science*, *306*, 2061.

Winkielman, P., McIntosh, D., & Oberman, L. (2009) Embodied and disembodied emotion processing: Learning from and about typical and autistic individuals. *Emotion Review*, *1*(2), 178–190.

Winston, J. S., O'Doherty, J., & Dolan, R. J. (2003). Common and distinct neural responses during direct and incidental processing of multiple facial emotions. *Neuroimage*, *20*(1), 84–97.

Young, A. W., Aggleton, J. P., Hellawell, D. J., Johnson, M., Broks, P., & Hanley, J. R. (1995). Face processing impairments after amygdalotomy. *Brain*, *118*, 15–24.