CHAPTER 21

Sensory and Motor Brain Areas Supporting Biological Motion Perception: Neuropsychological and Neuroimaging Studies

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Perceiving and interpreting another individual's movements and actions is one of the most fundamental processes for an organism's survival and well-being. Whether the process is one of tracking and hunting prey, detecting and avoiding predators, learning to solve a problem from observation, or inferring and acting in accordance with social cues, in many biologically relevant situations, organisms must be able to observe their conspecifics and understand what their movements and actions mean.

In primates, the perception of body movements is supported by a network of lateral superior temporal, inferior parietal, and inferior frontal brain areas (Rizzolatti & Craighero, 2004). At least two of these regions contain mirror neurons in the macaque monkey, which are neurons that fire not only during the execution of an action, but also during the visual perception of the same action (see below). Thus, this network has become known as the *mirror neuron system*. However, whether the macaque mirror neuron system and the brain areas involved in action perception in the human brain are analogous is currently a topic of debate (e.g., Dinstein, Thomas, Behrmann, & Heeger, 2008; Kilner, Neal, Weiskopf, Friston, & Frith, 2009). Thus, here, we will instead use the more neutral term *action perception system* (APS).

In this chapter, I discuss the APS with a focus on biological motion. The goal will be to link the visual perception literature on biological motion to the APS. I will start with a summary of the neuroanatomy and connectivity of the human brain, with particular focus on the APS. Then, I will summarize neuroimaging and neuropsychological experiments that have revealed brain regions that are involved in and necessary for biological motion perception. I'll conclude with a discussion of some active research questions that are of interest to biological motion and action perception research today.

SENSORY AND MOTOR AREAS INVOLVED IN ACTION AND BIOLOGICAL MOTION PERCEPTION

The process of understanding others' body movements is so ubiquitous that it seems deceptively simple. To illustrate, suppose that you are looking at another person raising her arm and waving her hand. You can effortlessly perceive the person's form, identify the body and its parts, and which body part is being moved in which manner. You also often recognize what this action means (e.g., a greeting) and can also sometimes infer something about the person's intention or goal (e.g., a friendly approach or a call for your attention, as opposed to ingestion of food or a threat of violence).

In this example, your experience of the other individual's action enters your system through the visual sensory modality. However, your experience of your own arm and what it is like to move and wave it around, and what

that action may mean about your internal states is rarely visually perceived. That first-person knowledge is largely motor and kinesthetic, linked also to your own internal motivational and emotional states. It is thus remarkable that you can so effortlessly and quickly perceive what this *other* person is doing and *know* what that is even though the representations you are working with are in different modalities. Such a mapping between a third-person action (which is most often visually perceived) and a first-person action (which is mostly kinesthetically experienced but rarely visually perceived) is not trivial (Barresi & Moore, 1996).

It is possible for an organism to sense and process the actions of its conspecifics in circuitry separate and independent from its own sensorimotor circuitry (e.g., eigenmannia, a weakly electric fish, Heiligenberg, 1991). However, the range of behaviors that can be subserved by such systems are limited and, indeed, in more complex organisms, we find more interconnected and interactive sensory and motor/executive systems. In primate cortex, sensory areas lie posterior to the central sulcus, whereas motor planning, actions, and executive processes are primarily controlled by areas anterior to the central sulcus. However, since perception and action are intimately linked and operate in concert, distinct parts of frontal cortex are connected with different posterior regions by several dense fiber pathways or fasciculi.

Of specific relevance to action and biological motion processing are the parietofrontal connections (Cipolloni & Pandya, 1999; Matelli & Luppino, 2004). The major association pathway between the parietal and frontal cortices is the superior longitudinal fasciculus (SLF), mediating the perception and processing of action and space. The dorsal component of the SLF connects the superior and medial parietal areas (PE, PEc, PGm) that code locations of body parts, in a body-centered coordinate system, to the dorsal premotor and supplementary motor regions in frontal areas F2 and F7 (Matelli & Luppino, 2001). The ventral portion of area F2 (F2vr) is a major target of areas MIP (in the caudal part of the medial bank of the intraparietal sulcus) and V6A (in the dorsal part of the anterior

bank of the parietooccipital sulcus). This MIP/V6A–F2vr circuit is thought to be involved with the transformation of somatosensory and visual information for the control of the transport of the hand toward a target (Gregoriou, Luppino, Matelli, & Savaki, 2005). F7 has a dorsal portion called the supplementary eye field (SEF), which is richly connected with the frontal eye field (FEF) and may be involved in coding object locations for attention and orienting (Luppino, Rozzi, Calzavara, & Matelli, 2003). The middle component of the SLF runs between the caudal inferior parietal lobule and dorsolateral and mid-dorsolateral prefrontal areas (BA 6, 8, 9, and 46, including the FEF). This pathway, especially the lateral intraparietal area (LIP)-FEF circuit, plays a big role in oculomotor aspects of spatial function, which uses eye position and retinotopic information for computing positions in space and programming eye movements (Stanton, Bruce, & Goldberg, 1995). The rostral component of the SLF connects the inferior parietal lobule (PF/PFG) to the ventral premotor cortex and the adjacent frontal opercular region (F4 and F5). This pathway is important for goal-directed action processing (Gregoriou, Borra, Matelli, & Luppino, 2006; Petrides & Pandya, 2009; Rozzi et al., 2006). In particular, F4 is connected with area VIP (ventral intraparietal area), and this circuit is thought to be involved with representing peripersonal space and planning actions toward objects within this space. F5 is connected with AIP (anterior intraparietal area) and PF, and is thought to be important for representing properties of objects (such as size and shape) and planning appropriate grasping and handling patterns in interacting with them (Matelli & Luppino, 2001).

While there are ample connections for perception and action to communicate effectively, a body of evidence now shows that the nervous system may sometimes code perceptual and executive/motor processes even more directly. Of particular relevance here is the discovery of mirror neurons. Mirror neurons are a particular class of visuo-motor neurons that were first found in the premotor cortex of the macaque monkey in area F5 (Caggiano, Fogassi, Rizzolatti, Thier, & Casile, 2009; Cattaneo &

Rizzolatti, 2009; Fadiga, Fogassi, Pavesi, & Rizzolatti, 1995; Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Rizzolatti, Fadiga, Gallese, & Fogassi, 1996). While some F5 neurons are purely motor neurons, some respond not only when the monkey executes a particular goal-directed action, but also when it observes another individual perform the same or a similar action. For instance, a mirror neuron that fires as the monkey itself cracks a peanut will also fire as the monkey observes an experimenter crack a peanut. Indeed, some mirror neurons are multisensory—the same neuron will fire when the monkey merely hears a peanut being cracked (Kohler et al., 2002). Later studies have revealed neurons with similar response patterns also in parietal cortex (Fogassi et al., 2005; Rizzolatti & Craighero, 2004; Rozzi, Ferrari, Bonini, Rizzolatti, & Fogassi, 2008).

The existence of a similar mirror system in humans has been suggested by a variety of magnetic stimulation and electrophysiological studies (Fadiga et al., 1995; Hari et al., 1998; Nishitani & Hari, 2000; Strafella & Paus, 2000), and human positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies have revealed activation in premotor areas (sometimes as part of a larger network involving superior temporal and parietal regions) during action observation and imitation (e.g., Decety & Grezes, 1999; Gallese et al., 1996; Grafton, Arbib, Fadiga, & Rizzolatti, 1996; Iacoboni et al., 1999; Rizzolatti et al., 1996). It also appears that such mirror-like neuronal responses exist in a variety of brain areas: Neuroimaging studies have reported that visual perception of pain can activate brain areas that are active when experiencing pain (Botvinick et al., 2005); visual observation of touch sensation evokes responses in somatosensory areas (e.g., Keysers et al., 2004); viewing another person who is disgusted can activate regions of the insula that are responsive during disgust (Wicker et al., 2003), and so on. Thus, in addition to subserving action processing, the current view suggests that the function of mirror and similar neurons may be more general and that this system or property may be a basis for forming a connection between self and other, and thus have implications in the emotional and social functioning of organisms (Gallese, Keysers, & Rizzolatti, 2004; Iacoboni, 2009; Keysers & Gazzola, 2006; Rizzolatti & Craighero, 2004).

The discovery of mirror neurons was very exciting for neuroscience and psychology because they constituted evidence for perceptual stimuli and motor responses sharing direct neural substrates at some level. This, of course, does not mean that premotor cortex "sees" or "hears" just like primary visual and auditory areas—but that perception involves regions of the nervous system that would not traditionally be considered sensory and is more dynamic and distributed than previously envisioned (see also Bar, 2009; Kveraga, Ghuman, & Bar, 2007). However, it is important to note that we still do not know the underlying neural computations and the roles of mirror and similar neurons in this process. Further work is needed to specify the functional properties of mirror neurons, as well as their role in larger sensorimotor networks.

BIOLOGICAL MOTION AND THE ACTION PERCEPTION SYSTEM: FUNCTIONAL MAGNETIC RESONANCE IMAGING

A specific question of interest in our research has been the relationship between the APS and the visual perception of biological motion. In vision science, point-light biological motion stimuli have been used for decades to study perceptual and neural processes underlying the processing of simplified representations of human body movement (Blake & Shiffrar, 2007). Image sequences constructed from point-lights attached to the limbs of a human actor can readily be identified as depicting actions, although they do not define a recognizable form when stationary (Johansson, 1973). These animations convey surprisingly detailed information about movements of the human body, despite using motion signals almost exclusively and lacking other visual cues, such as color, shading, and contours. Observers can infer characteristics such as gender, affect, or identity from

point-light animations (Cutting & Kozlowski, 1977). Children are able to recognize point-light figures from early ages (Bertenthal, Proffitt, & Kramer, 1987; Fox & McDaniel, 1982; Pavlova, Krageloh-Mann, Sokolov, & Birbaumer, 2001). Newborn humans (Simion, Regolin, & Bulf, 2008), as well as chicks, appear to have sensitivity to point-light biological motion (but not specifically to chicken biological motion; Vallortigara, Regolin, & Marconato, 2005). Pigeons can be trained to identify point-light pecking movements (Dittrich, Lea, Barrett, & Gurr, 1998).

Point-light animations have several qualities that make them useful stimuli: They are particularly compelling examples of the form-from-motion effect, evoking very specific percepts even with relatively few dots. They exemplify that, despite constituting impoverished visual input (e.g., lacking in contrast, texture, or color cues), motion signals alone can carry much information about the action represented. Furthermore, control stimuli for point-light biological motion are readily available since it is easy to temporally or spatially "scramble" the dots—thus, in the scrambled animations, local motion signals can be kept the same but without evoking the percept of a coherently moving animate form.

Although point-light motion stimuli have been used for many decades to study visual processing of motion, they had not typically been used in studies of action perception. A number of neuroimaging studies have examined point-light biological motion perception in the human brain (see also Pyles & Grossman, Chapter 17, this volume). Areas identified in these studies include the posterior superior temporal gyrus (pSTG) and sulcus (pSTS), motion sensitive area V5/MT+, ventral temporal cortex, and occasionally parietal cortex (Beauchamp, Lee, Haxby, & Martin, 2002; Bonda, Petrides, Ostry, & Evans, 1996; Grezes et al., 2001; Grossman et al., 2000; Peelen, Wiggett, & Downing, 2006; Peuskens, Vanrie, Verfaillie, & Orban, 2005; Saygin, Wilson, Hagler, Bates, & Sereno, 2004; Servos, Osu, Santi, & Kawato, 2002; Vaina, Solomon, Chowdhury, Sinha, & Belliveau, 2001). The involvement of the

pSTG/STS is perhaps the most robust finding (see Puce & Perrett, 2003), supported also by electrophysiological recordings in the macaque monkey (Oram & Perrett, 1996). Like parietal cortex, posterior and middle temporal cortex are also connected with frontal regions via white matter fibers. Posterior area Tpt is linked to area 8Ad via the arcuate fasciculus, and the middle region (areas PaAlt, TS3, and TPO) gives rise to a different fiber system that runs in the extreme capsule and connects mainly with Brodmann area 45, as well as parts of 9, 46, and 8Ad (Petrides & Pandya, 1988). These pathways transmit auditory spatial and auditory object information to frontal cortex. Whether these connections could also communicate information about biological motion is not known. The STS takes part in different functions ranging from auditory, visual, and multisensory perception to social cognition (Beauchamp, 2005; Calvert, 2001; Hein & Knight, 2008; Redcay, 2008; Rolls, 2007). Biological motion–sensitive portions of the STS and frontal cortex are likely not directly connected, but linked via the inferior parietal lobule.

But, given that point-light biological motion stimuli evoke vivid action percepts, does their perception also recruit the APS? Or, are motion signals alone insufficient to drive neural responses in these higher areas?

We explored these questions in an fMRI study (Saygin, Wilson, Hagler, Bates, & Sereno, 2004). Twelve adults with no known visual or neurological abnormalities were scanned as they viewed point-light biological motion animations, scrambled versions of the same animations, and stationary point-light figures. Point-light biological motion sequences were created by videotaping an actor performing various activities (e.g., jogging, jumping jacks, bowling), then encoding the joint positions in the digitized videos (Ahlstrom, Blake, & Ahlstrom, 1997) for presentation with Matlab and the Psychophysics Toolbox (Brainard, 1997). Scrambled animations were created by randomizing the starting positions of the point-lights while keeping the trajectories intact and thus contained the same local motions, but not the global form defined by biological motion

(Grossman et al., 2000). We used a high-field scanner (4 Tesla), distortion correction, and cortical surface-based methods (Dale, Fischl, & Sereno, 1999; Fischl, Sereno, & Dale, 1999; Hagler, Saygin, & Sereno, 2006). Data were averaged on the spherical surface-based coordinate system, which uses cortical surface curvature information to align the major sulci and gyri of the brain more precisely across subjects (Fischl, Sereno, Tootell, & Dale, 1999).

In many neuroimaging studies of visual perception, including those of biological motion perception, researchers use a one-back working memory task to keep subjects attending (Grossman et al., 2000). However, we found that performance in this task is significantly different for biological and scrambled motion (accuracy for biological motion = 91.9%; for scrambled motion = 87.0%; for static dots = 96.1%; all pairwise differences significant p <0.05). In order to not have an attention or task difficulty confound, we asked subjects to perform a simple but orthogonal task of judging whether or not the color of the point-lights in each trial were green. Behavioral performance in this task was well balanced across conditions (accuracy for biological motion = 98.2%; scrambled motion, 98.4%; static point-lights, 97.8%; pairwise differences not significant).

When biological motion observation was compared with the static point-light observation baseline, we found extensive activation in occipital, temporal, and parietal cortex,

extending along both the ventral and dorsal visual streams. Additionally, a robustly responsive region along the inferior frontal and precentral sulci was found bilaterally, indicating that point-light animations indeed recruit frontal areas known to be involved in action observation. This activation followed inferior frontal and precentral sulci in a fairly continuous manner in both the group results and in individual subjects. Scrambled biological motion activated many of the same regions, although the activation was noticeably less extensive. When biological motion and scrambled biological motion responses were compared directly (Figure 21-1), in line with previous work, we found lateral temporal regions that responded more strongly to biological motion than to scrambled motion. Additionally, a region in left ventrolateral inferotemporal cortex showed significant responses to biological motion compared with scrambled biological motion.

The lateral temporal activation for biological motion almost certainly overlaps with multiple visual areas (Nelissen, Vanduffel, & Orban, 2006). In examining individual subjects' data in relation to results from other scans performed in our laboratory, we observed that the lateral temporal region responsive to biological motion had considerable overlap with areas that responded to simple motion, object form, human faces, and, especially, human body form (see also Grossman & Blake, 2002; Peelen et al., 2006). In a later study, we also reported that this
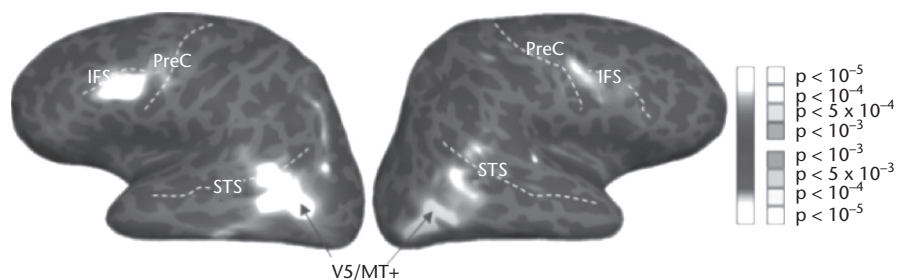

Figure 21-1. Brain activity for viewing biological motion compared with scrambled biological motion, shown on the inflated cortical surface. Brain areas that show a preferential response to biological motion were found in lateral temporal cortex, including MT+ and posterior superior temporal sulcus (pSTS), and in frontal cortex, in the vicinity of inferior frontal sulcus (IFS) and precentral sulcus (PreC). Adapted from Saygin, A. P., Wilson, S. M., Hagler, D. J., Jr., Bates, E., & Sereno, M. I. (2004). Point-light biological motion perception activates human premotor cortex. *Journal of Neuroscience, 24*(27), 6181–6188.

lateral temporal region has multiple retinotopic patches that respond to the presentation of biological motion and show attentional modulation (Saygin & Sereno, 2008).

Importantly, in frontal cortex, we found that the inferior frontal sulcus (IFS), at its junction with and partially extending into the precentral sulcus, responded significantly more to biological motion (Figure 21-1). In fact, the left hemisphere peak in the IFS was the most significantly responsive area for this contrast in the whole brain (peak Talairach coordinates [-41, 14, 18]). There were also significant subpeaks in the inferior precentral sulci bilaterally (peak Talairach coordinates [-37, 5, 25] and [34, 7, 27]). Thus, we found support for the hypothesis that motion information in body actions can drive neural activity in frontal cortical regions that are part of the APS. Indeed, a further region-of-interest (ROI) analysis revealed that inferior frontal and premotor cortex were just as selective for biological motion as was the pSTS, since the size of the response in the scrambled motion condition as a fraction of the response in the biological motion condition was very similar across the three ROIs (inferior frontal, 56.3%; premotor, 55.7%; and pSTS, 58.6%).

These results show that motion information in body actions is sufficient to drive activation in premotor areas that are part of the APS. Combined with studies that manipulated the observers' visual and motor experience with perceived movements (Calvo-Merino, Grezes, Glaser, Passingham, & Haggard, 2006; Casile & Giese, 2006; Jacobs & Shiffrar, 2005; Pinto & Shiffrar, 2009; Saunier, Papaxanthis, Vargas, & Pozzo, 2008), we hypothesize that this response reflects a partial internal simulation of visually perceived actions (Jeannerod, 2001).

Whereas many fMRI studies of biological motion have revealed frontal activations (e.g., De Lussanet et al., 2008; Jung et al., 2009; Michels, Kleiser, de Lussanet, Seitz, & Lappe, 2009; Michels, Lappe, & Vaina, 2005; Vaina et al., 2001), not all of them do. While it is possible that the variability between studies is caused by the different stimuli or tasks used, we suggest that the main reason for this is because the location of this response is variable between

individuals. We noticed that the strongest frontal activations were most consistently found in the IFS, close to its junction with the precentral sulcus. Since the angle of these sulci and the location of the junction varies among individuals, it is possible that some studies would not detect this activation following group averaging. Using a cortical surface-based coordinate system at the second level of analysis may have helped us reveal this response since it allows for a more precise intersubject alignment (Fischl, Sereno, Tootell, & Dale, 1999). Even using this method however, intersubject anatomical variability can be an issue: Note that we observed weaker activation in premotor cortex in the right hemisphere compared with the left (Figure 21–1). However, a ROI analysis, in which the regions were drawn on each subject's own anatomy, revealed that the right inferior frontal and precentral activation in the right hemisphere was not significantly different from the activation in the left hemisphere (see Figure 3 in Saygin, Wilson, Hagler, Bates, & Sereno, 2004). Therefore, it appears that in our sample of participants, there was more anatomical variability in the right hemisphere, rather than a true hemispheric bias in the data.

In the macaque brain, mirror neurons are reported to respond to actions performed in front of the monkey, but not to videotaped stimuli (Ferrari, Gallese, Rizzolatti, & Fogassi, 2003). In contrast, human premotor cortex responds even to point-light biological motion representing actions. Other kinds of sparse representations can also evoke activation in these regions in the human brain (Alaerts, Van Aggelpoel, Swinnen, & Wenderoth, 2009). It is therefore possible that the human mirror neuron system is capable of processing more abstract or varied representations for actions. It is also worth noting that while the STS region's involvement in biological motion processing has been known for a long time, recent studies have reported different kinds of cells in this region, including "snapshot neurons" (Jellema & Perrett, 2003; Oram & Perrett, 1996; Vangeneugden, Pollick, & Vogels, 2009). Future physiology and fMRI studies may be able to address whether a cross-species difference exists in biological motion

perception, as for other domains of motion perception (Sereno & Tootell, 2005).

LESION CORRELATES OF BIOLOGICAL MOTION PERCEPTION DEFICITS

While functional neuroimaging is an excellent tool for studying brain areas *involved* in a particular process or task, its power is limited when it comes to making inferences about brain areas that are *necessary* for the task. Lesion-symptom mapping is thus an excellent complement to these studies as this method enables us to infer more direct causal relationships between brain and behavior (Rorden & Karnath, 2004).

There is a small literature on biological motion processing following brain injury (Battelli, Cavanagh, & Thornton, 2003; Billino, Braun, Bohm, Bremmer, & Gegenfurtner, 2009; Pavlova & Sokolov, 2003; Saygin, 2007; Schenk & Zihl, 1997; Serino et al., 2010; Sokolov, Gharabaghi, Tatagiba, & Pavlova, 2010; Vaina & Gross, 2004). Individual case reports of patients with deficits in low-level motion analysis who have preserved biological motion processing have been reported (McLeod, Dittrich, Driver, Perrett, & Zihl, 1996; Vaina, Lemay, Bienfang, Choi, & Nakayama, 1990), as have patients with deficiencies in recognizing form-from-motion, including biological motion, in the absence of early visual deficits (Cowey & Vaina, 2000).

In terms of lesion sites, the results are not particularly consistent. Schenk and Zihl (1997) reported two patients considered deficient in perceiving biological motion, both with bilateral lesions in superior parietal cortex. Battelli et al. (2003) tested three patients with unilateral inferior parietal lesions (one left hemisphere and two right hemisphere lesioned) and found them impaired in point-light biological motion processing, but not in motion coherence judgments. Vaina and Gross (2004) reported on four patients who could not recognize point-light biological motion and whose lesions included temporal cortex, but with variability in location and extension into other areas (two patients had lesions primarily in the anterior temporal lobe, the other two had lesions including portions of

both the parietal and anterior temporal lobes). Outside of cortex, lesions in the cerebellum have been reported to be linked to impaired biological motion perception (Sokolov et al., 2010). In addition, a series of studies have reported deficits in biological motion processing in patients with early periventricular lesions, suggesting that disruption of cortical connectivity can lead to deficits (Pavlova, Sokolov, Birbaumer, & Krageloh-Mann, 2006; Pavlova, Staudt, Sokolov, Birbaumer, & Krageloh-Mann, 2003).

To explore brain areas that are "necessary" for biological motion perception, we tested a large group of stroke patients, unselected for lesion site, and performed lesion-symptom mapping using *voxel-based lesion-symptom mapping* or VLSM (Bates et al., 2003). The vast majority of lesion-symptom mapping work has employed one of two basic approaches: In the groups-defined-by-behavior method, a cutoff is stipulated on the behavioral measure(s). Patients who perform below the cutoff are categorized as impaired. An overlay of all the impaired patients' lesions can be constructed to determine whether there is an area that is consistently damaged in these patients. In the groups-defined-by-lesion method, patients are classified based on neuroanatomical criteria. For instance, patients might be divided into groups according to whether or not their lesions involve a particular brain area. These groups are then compared on the behavioral measures of interest. Voxel-based lesion-symptom has advantages compared to both of these approaches. It allows the researcher to avoid predefining lesion region(s) of interest, avoid specifying performance levels to be considered impaired, explore the independence of effects between different lesion foci, and use templates and methods that are commonly used in the functional neuroimaging literature, thus making the closest possible comparisons of lesion results to functional neuroimaging data. Matlab-based software to perform VLSM analyses is freely available online at http://www.neuroling.arizona.edu/resources.html. Voxel-based lesion-symptom and similar methods have attracted a lot of interest in recent years and have been applied to different domains in several laboratories (e.g.,

Borovsky, Saygin, Bates, & Dronkers, 2007; Bouvier & Engel, 2006; Dronkers, Wilkins, Van Valin, Redfern, & Jaeger, 2004; Mort et al., 2003; Saygin, Wilson, Dronkers, & Bates, 2004).

We tested 60 chronic stroke patients (mean age = 64.1 years) and 19 age-matched controls. Exclusionary criteria were dementia, tumors, multiple infarcts, and any visual, psychiatric, or neurologic abnormalities. Only patients with unilateral lesions due to a single cerebrovascular accident (CVA) participated. None of the patients presented with spatial neglect or other attentional disorders. Forty-seven patients had left-hemisphere damage (LHD), 13 had right hemisphere damage (RHD). Given that a subset of patients had computerized lesion reconstructions, constructing group lesion maps was possible only within the left hemisphere. However, note that our sample of RHD patients is still sizable in comparison with the existing literature on biological motion and is sufficient to explore any lateralization of behavioral deficits in this task.

Lesion reconstructions were based on structural scans acquired at least 5 weeks post onset of stroke. When possible, reconstructions were drawn directly onto three-dimensional MRI scans of the patients using MRICro software (Rorden & Brett, 2000). All reconstructions were morphed onto the publicly available Montreal Neurological Institute (MNI) single-subject template brain (often called the MNI brain or "colin27") that has been constructed by averaging 27 scans of a single individual (Collins, Neelin, Peters, & Evans, 1994).

We used an adaptive psychophysical paradigm to obtain a measure of biological motion perception. Two animations were presented, along with a variable number of moving noise dots. In each trial, participants were presented with the point-light motion and its scrambled equivalent on either side of the screen and were asked to point to the set of dots that "contains the man." The stimuli were the same as described above in the fMRI study. To yield a psychometric measure of performance, we varied the number of noise dots and used a Bayesian adaptive procedure that estimates the number of noise dots

at which a subject performs at a desired level of accuracy (Watson & Pelli, 1983).

As a group, patients could tolerate only about half as many noise dots as controls in order to perform at the same level of accuracy (mean for controls = 21.2; LHD = 11.0; RHD = 10.4). For both LHD and RHD patients, this performance level was significantly different from that for controls ($P <0.01$, two-tailed, corrected), but LHD and RHD groups did not differ from one another ($P = 0.7$). There does not seem to be a laterality effect for biological motion perception deficits, which is unlikely to be due to lack of power in this sample since other measures do significantly differ between these two groups (e.g., in the same patient set, WAB aphasia quotient is significantly lower for LHD (72.8/100) than for RHD (96.5/100) patients, $P <0.0001$). Conversely, note that several prior studies on biological motion perception have reported right lateralized activity (e.g., Pavlova, Bidet-Ildei, Sokolov, Braun, & Krageloh-Mann, 2009; Pelphrey, Morris, & McCarthy, 2004).

Patients' gender and age did not correlate with thresholds for biological motion perception (mean for males = 10.8, females = 10.7; $r = 0.03$ both Ps >0.05); lesion volume tended toward a relationship, but this did not reach significance ($r = 0.4$; $P = 0.08$ uncorrected for multiple comparisons). We explored correlations between patients' biological motion perception thresholds with behavioral scores from other visual tests (judgment of line orientation, face recognition, and motion coherence thresholds), as well as tests from other domains (language measures, performance IQ). None of these correlations was significant, with the exception of face recognition scores ($r = 0.52$, $P <0.05$ corrected). Future work is needed to interpret the correlation observed between biological motion perception and face recognition, and on individual differences in biological motion perception in general (Miller & Saygin, 2012).

For constructing a group lesion map, at each voxel, patients were divided into two groups according to whether they did or did not have a lesion involving that voxel (for a similar approach that additionally uses lesion information continuously, see Leff et al., 2009).

Behavioral scores were then compared for these two groups at each voxel, yielding a map that contains a statistical value at each voxel (e.g., a t-score, or a measure of effect size) that can then be plotted on a color scale. We made maps of the t-statistic, comparing lesioned and intact groups' perceptual thresholds for biological motion perception at each voxel. Axial slices from this map are shown in Figure 21-2: Two distinct regions emerged as especially important lesion correlates of compromised biological motion perception. An anterior focus in the inferior frontal and precentral gyri (corresponding to Brodmann areas 44 and 45, extending partly into area 6) and a larger, posterior region along the STG/STS, additionally including parts of the posterior middle temporal and supramarginal gyri (parts of Brodmann areas 21, 22, 37, 39, and 40).

In lesion studies, an area may be falsely identified as relevant due to a relationship between separate lesion sites, as opposed to having an actual causal role on behavior. Thus, we wondered, for example, whether the inferior frontal involvement we observed in our lesion analyses was an indirect consequence of lesions to another area (e.g., temporal cortex). We explored the independence of emerging lesion foci by making similar maps that used an ANCOVA instead of an ANOVA, covarying out the effect in the inferior frontal and the posterior temporal regions,

respectively, and verified that the effect in each region is not attributable to indirect effects of the lesion in the other area. In other words, the lesion effect in posterior temporoparietal region remained after factoring out the effect in inferior frontal cortex (Figure 21-3A), and factoring out the effect in superior temporal cortex still shows an involvement of frontal cortex (Figure 21–3B).

Capitalizing on the fact that the lesions have been morphed onto a common space, we also formally compared results from our lesion analyses to those from our previously published fMRI study reviewed above (Saygin, Wilson, Hagler, Bates, & Sereno, 2004). We used a volume-based group average of this fMRI data to assess the fMRI statistics in the same normalized space as the lesion reconstructions. A voxel-by-voxel correlation analysis of t-values across our lesion map and the biological motion versus scrambled biological motion comparison from the fMRI study of healthy subjects revealed a sizable overall relationship between the two images, at a correlation of $r = 0.55$.

We then used the lesion maps in Figure 21–3 to obtain ROI masks (shown in Figure 21-4) for the fMRI data by thresholding this image at a voxelwise $P < 0.05$. This yielded two ROI masks, one in posterior superior temporal (Figure 21–4A) and one in inferior frontal cortex (Figure 21-4B). In both regions, there
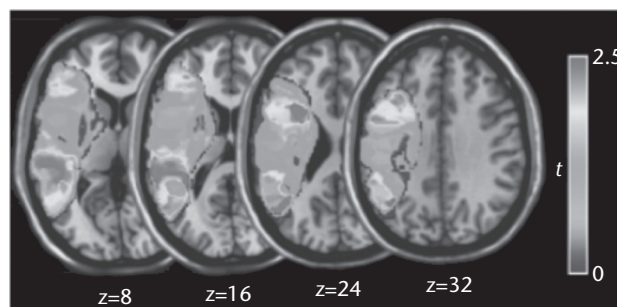

Figure 21-2. Axial slices showing the relationship between tissue damage in stroke patients and behavioral deficits in biological motion. In each voxel, biological motion perception thresholds were compared between patients with a lesion in that voxel and patients who do not have a lesion in that voxel. High *t* values (*red, orange*) indicate a highly significant effect of lesions on biological motion perception. Reprinted from Saygin, A.P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain, 130*(9), 2452–2461, with permission of the publisher, Oxford University Press.
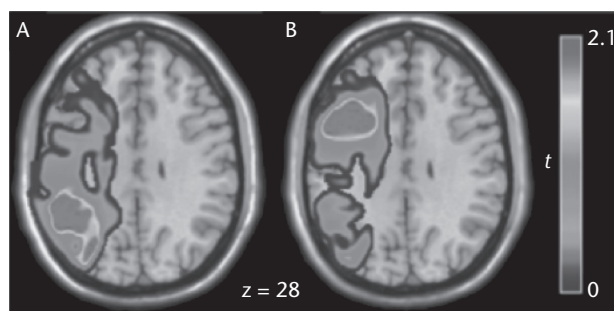
Figure 21-3. Axial slices from ANCOVA maps. Voxel-by-voxel ANCOVAs covarying out voxels of interest were carried out (A) factoring out the peak voxel in frontal cortex, (B) factoring out the peak voxel in posterior cortex. Both superior temporal and inferior frontal lesion foci remain implicated. Reprinted from Saygin, A.P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain, 130*(9), 2452–2461, with permission of the publisher, Oxford University Press.
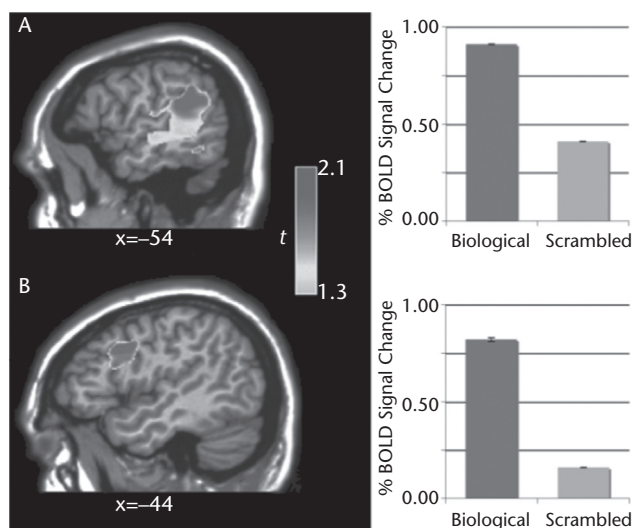

Figure 21-4. The relationship between lesion findings and functional magnetic resonance imaging (fMRI) data from healthy controls. The ANCOVA maps in Figure 21-3 were thresholded to yield two regions of interests (ROIs): one in temporoparietal cortex (A), one in frontal (B), shown in the left panel of the figure. These ROIs were then used as masks onto independently collected fMRI data (Figure 1 from Saygin, Wilson, Hagler, Bates, & Sereno, 2004), shown in the right panel of the figure. Both ROIs revealed clear selectivity for biological motion (significantly more activation for biological as compared with scrambled motion). Thus, the two brain-mapping methodologies yielded convergent results. Reprinted from Saygin, A.P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain, 130*(9), 2452–2461, with permission of the publisher, Oxford University Press.

was significantly more response to biological motion compared with scrambled motion in the healthy brain, with percentage blood oxygen level-dependent (BOLD) signal change values highly consistent with our time course ROI analysis of the fMRI data reported

earlier. We also ran the voxel-by-voxel correlation between the lesion data and the fMRI parameter estimates for biological motion versus scrambled biological motion within these ROIs. In the posterior temporal ROI, the correlation was $r = 0.58$, whereas there was

an even stronger correlation of $r = 0.83$ in the inferior frontal ROI.

It is important to note that these ROIs are based on the lesion maps and thus are completely independent from the fMRI data collected from healthy subjects. Nevertheless, the lesion foci obtained in the present study and fMRI activity specific to biological motion exhibit strong overlap, indicating crucial roles for posterior temporal and inferior frontal areas in this task.

Lesion maps and fMRI are complementary brain mapping methods that tap into different aspects of neural function. At the same time, each approach also brings with it the limitations inherent to that method. Lesion studies are limited in inferential power by the distribution of lesions in the patients studied, and our study is not an exception. If there are no lesions to a particular brain area in the sample (or only a very small number of patients), we cannot determine whether lesions to this region have any effect on the function under question. Accordingly, there are areas active during biological motion perception that are not present in the lesion maps. For example, in our sample of patients (of

which a large proportion had suffered middle cerebral artery strokes), we did not have any patients with lesions in primary visual cortex or inferotemporal cortex (Figure 21-5A). We have carried out additional studies to explore the necessity ventral temporal cortex for biological motion perception (Gilaie-Dotan, Bentin, Harel, Rees, & Saygin, 2011; Gilaie-Dotan, Bentin, Rees, Behrmann, & Saygin, 2012). Functional MRI, however, is limited when it comes to the involvement of white matter. The lesion foci tend to be medial when compared to activation foci for fMRI, often containing large extensions into the white matter (Figure 21-5B).

Our study showed, in an unselected group of patients, that lesions in premotor and superior temporal cortex were associated with deficits in biological motion perception. While it does not necessarily follow from this that intact motor representations of the perceiver's body per se are necessary for uncompromised perception of biological motion, a recent study on hemiplegic patients suggests this interpretation is likely (Serino et al., 2010). In this study, patients with and without hemiplegia were asked to process
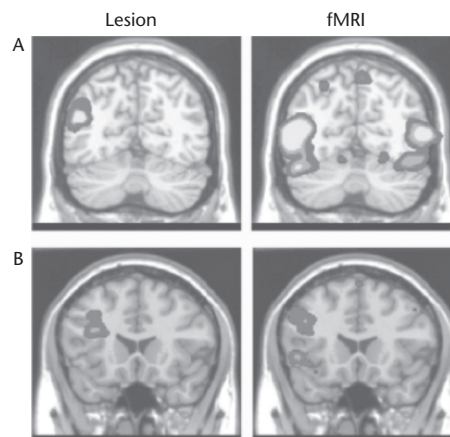

Figure 21-5. The thresholded lesion map in Figure 21-4A and in Saygin (2007), shown with the functional magnetic resonance imaging (fMRI) data shown in Figure 21-1 and in Saygin, Wilson, Hagler, Bates, and Sereno (2004) analyzed and displayed in three dimensions. Despite overall agreement between the two methods, there are also differences. In the coronal slices through posterior temporal cortex (A), we can see that the lesion map did not extend as far inferiorly as the fMRI activation. This is due to reduced power in these regions due to the distribution of lesions in our sample. In premotor cortex (B), the lesion focus lay more medially compared with the fMRI focus. This is because the signal in fMRI comes largely from the gray matter, but in the lesion map, we can also use lesion information extending into the white matter.

point-light arm movements. Perception of these stimuli were compromised in hemiplegic patients, but not in non-hemiplegic patients and controls. Furthermore, hemiplegic patients were more accurate when they processed actions that appeared to have been performed by their unaffected arm compared with those that appeared to have been performed by their hemiplegic arm or the corresponding arm of another person.

CONCLUSION

Current and Future Directions

I have summarized research on the brain areas subserving biological motion perception using functional neuroimaging on the one hand and neuropsychological lesion mapping on the other. These two methods, despite their inherent differences, showed very good agreement, verifying the importance of both posterior superior temporal and premotor brain areas for biological motion perception. Thus, these regions are not only involved in the perception of biological motion, but they are also necessary for the correct processing of biological motion.

It is interesting that premotor areas are activated during biological motion perception and that lesions in such high-level areas have effects on performance in visual perception of body movements (see also Saygin, Wilson, Dronkers, & Bates, 2004; Tranel, Kemmerer, Damasio, Adolphs, & Damasio, 2003), even when the task was not explicitly engaging processes related to social cognition or motor imagery. Thus, it appears that even during relatively passive perception, the brain processes stimuli in an embodied manner (Barsalou, 1999).

Having established the importance of superior temporal and premotor regions in biological motion, it will now be important to identify the precise functional roles played by each region in biological motion perception. How do these regions operate together in order to subserve action perception? What kinds of representations are used, and what computations are performed? How do they communicate with each other, and what signals do they send to each other?

In our group, we are beginning to explore the functional properties of the APS further, using additional methods and new stimuli. One direction we took is using transcranial magnetic stimulation (TMS). This method allows researchers to reversibly disrupt the function of selected brain areas to explore effects of virtual lesions on behaviors of interest (although this is almost certainly an oversimplification; see Silvanto & Muggleton, 2008; Walsh & Rushworth, 1999).

Grossman et al. had reported that repetitive TMS (rTMS) over pSTS led to a decrease in sensitivity for biological motion (Grossman, Battelli, & Pascual-Leone, 2005). Although there are no reports with biological motion stimuli, TMS over inferior frontal areas has been reported to show effects on action perception tasks (Pobric & Hamilton, 2006). It is, however, difficult to use rTMS over inferior frontal and premotor cortex since stimulation here can cause twitching and discomfort in facial muscles, as well as blinking, which can confound results, especially in visual tasks. In a recent study, we used theta-burst TMS to explore the effect of TMS over STS and premotor cortex on biological motion perception (van Kemenade, Muggleton, Walsh, & Saygin, 2012). We used the same stimuli as in the above experiments. In each trial, either a point-light action or its scrambled counterpart was presented with a variable number of similarly moving noise dots (Hiris, 2007; Saygin, 2007). The observers' task was to determine whether human motion was present. We first estimated a 75% noise dot threshold for each subject (Watson & Pelli, 1983). Subjects were then tested at this level before and after TMS. We found that TMS targeting premotor cortex (the junction of inferior frontal sulcus and precentral sulcus) decreased accuracy in the task. A signal detection analysis showed that TMS decreased sensitivity, but also changed response bias. More specifically, subjects were more likely to make false alarms (respond that biological motion was present, when it was not) following TMS of premotor cortex. This was not, however, a generalized response bias because a control experiment showed no effect of TMS at the same site on sensitivity or bias when subjects

were asked to detect nonbiological structure from motion stimuli (simple geometric shapes).

In another line of work, we are exploring biological motion perception in multisensory experiments, an area that has been attracting attention recently (Petrini et al., 2009; Petrini, Russell, & Pollick, 2009; Schutz & Lipscomb, 2007). In natural settings, the perception of biological movements is often accompanied by related inputs from other modalities, notably audition, as when footsteps are heard as well as seen. But little is known about multisensory perception for movements of other people. In a recent study, we added sound to point-light animations and found that observers were much better at judging whether a visual motion pattern shared the same temporal frequency as an auditory pattern when the visual stimuli depicted body movements, compared with inverted or scrambled versions of the same moving dots (Saygin, Driver, & de Sa, 2008). This effect was seen only when the auditory stimuli could be heard as the "footsteps" of the point-light stimuli and not when they were temporally offset from the visual footsteps, suggesting an "audiovisual Gestalt" for biological motion. Interestingly, the pSTS has been identified as a critical site for multisensory integration of information from different modalities (Beauchamp, 2005). It is unclear whether these are the same parts of pSTS that are sensitive to biological motion, but single-unit recordings in monkeys revealed that some biological motion-sensitive pSTS cells respond more when visual stimuli are presented with congruent sounds (Barraclough, Xiao, Baker, Oram, & Perrett, 2005). Currently, we are exploring modulations of those brain areas sensitive to biological motion by auditory information using fMRI. Also of interest is whether modulations are found in sensory-specific (visual and auditory) areas, as seen recently with simple, nonbiological stimuli (Driver & Noesselt, 2008).

Research on special clinical populations can also help us elucidate more on biological motion perception. Of specific interest is autism spectrum disorders (ASD) since abnormalities within the APS have been suggested to underlie

the problems with social interaction observed in individuals with this condition (Iacoboni & Dapretto, 2006; Oberman & Ramachandran, 2007). Although there have been reports of impaired biological motion perception in children and adults with autism, the results are inconsistent (Atkinson, 2009; Blake, Turner, Smoski, Pozdol, & Stone, 2003; Cook, Saygin, Swain, & Blakemore, 2009; Freitag et al., 2008; Murphy, Brady, Fitzgerald, & Troje, 2009; Parron et al., 2008; Saygin, Cook, & Blakemore, 2010). Some of these findings challenge the theory that the dysfunction of the APS underlies the problems in social functioning seen in individuals with ASD (Hamilton, Brindley, & Frith, 2007). At the very least, future work is needed to disentangle factors behind the variability in the experiments to date and, in turn, inform how the perception of biological motion relates to higher level social processes (Miller & Saygin, 2012).

Another interesting direction is exploring the specificity and plasticity of the action perception system (e.g., Calvo-Merino et al., 2006; Cross, Kraemer, Hamilton, Kelley, & Grafton, 2009; Jastorff, Kourtzi, & Giese, 2006). At present, our group is interested in using artificial agents, such as robots and animations, to manipulate the visual form and the motion dynamics of action stimuli in order to further specify functional properties of the APS (Chaminade & Hodgins, 2006; Chaminade, Hodgins, & Kawato, 2007; Saygin, Chaminade, & Ishiguro, 2010; Saygin, Chaminade, Ishiguro, Driver, & Frith, 2012; Saygin & Stadler, 2012). The results are beginning to show that both visual appearance and biological motion modulate neural responses in the APS. Furthermore, the interaction of these cues is also important, with an especially significant role for parietal cortex (area AIP), which, as we mentioned, provides the anatomical connection between the visual and the motor components of the APS. These experiments will hopefully help us elucidate the functional properties of the APS on one hand, and help us develop psychological and neural methods for evaluating future artificial agents on the other (Saygin, Cicekli, & Akman, 2000).

REFERENCES

Ahlstrom, V., Blake, R., & Ahlstrom, U. (1997). Perception of biological motion. *Perception, 26*(12), 1539–1548.

Alaerts, K., Van Aggelpoel, T., Swinnen, S. P., & Wenderoth, N. (2009). Observing shadow motions: Resonant activity within the observer's motor system? *Neuroscience Letters, 461*(3), 240–244.

Atkinson, A. P. (2009). Impaired recognition of emotions from body movements is associated with elevated motion coherence thresholds in autism spectrum disorders. *Neuropsychologia.*

Bar, M. (2009). Predictions: A universal principle in the operation of the human brain [Introduction]. *Philosophical Transactions of the Royal Society of London B: Biological Sciences, 364*(1521), 1181–1182.

Barraclough, N. E., Xiao, D., Baker, C. I., Oram, M. W., & Perrett, D. I. (2005). Integration of visual and auditory information by superior temporal sulcus neurons responsive to the sight of actions. *Journal of Cognitive Neuroscience, 17*(3), 377–391.

Barresi, J., & Moore, C. (1996). Intentional relations and social understanding. *Behavioral and Brain Sciences, 19*, 107–122.

Barsalou, L. W. (1999). Perceptual symbol systems. *Behavioral and Brain Sciences, 22*, 577–660.

Bates, E., Wilson, S. M., Saygin, A. P., Dick, F., Sereno, M. I., Knight, R. T., et al. (2003). Voxel-based lesion-symptom mapping. *Nature Reviews. Neuroscience, 6*(5), 448–450.

Battelli, L., Cavanagh, P., & Thornton, I. M. (2003). Perception of biological motion in parietal patients. *Neuropsychologia, 41*(13), 1808–1816.

Beauchamp, M. S. (2005). See me, hear me, touch me: Multisensory integration in lateral occipital-temporal cortex. *Current Opinion in Neurobiology, 15*(2), 145–153.

Beauchamp, M. S., Lee, K. E., Haxby, J. V., & Martin, A. (2002). Parallel visual motion processing streams for manipulable objects and human movements. *Neuron, 34*(1), 149–159.

Bertenthal, B. I., Proffitt, D. R., & Kramer, S. J. (1987). Perception of biomechanical motions by infants: implementation of various processing constraints. *Journal of Experimental Psychology, Human Perception and Performance, 13*(4), 577–585.

Billino, J., Braun, D. I., Bohm, K. D., Bremmer, F., & Gegenfurtner, K. R. (2009). Cortical networks for motion processing: Effects of focal brain lesions on perception of different motion types. *Neuropsychologia, 47*(10), 2133–2144.

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

Blake, R., Turner, L. M., Smoski, M. J., Pozdol, S. L., & Stone, W. L. (2003). Visual recognition of biological motion is impaired in children with autism. *Psychological Science, 14*(2), 151–157.

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

Borovsky, A., Saygin, A. P., Bates, E., & Dronkers, N. (2007). Lesion correlates of conversational speech production deficits. *Neuropsychologia.*

Botvinick, M., Jha, A. P., Bylsma, L. M., Fabian, S. A., Solomon, P. E., & Prkachin, K. M. (2005). Viewing facial expressions of pain engages cortical areas involved in the direct experience of pain. *NeuroImage, 25*(1), 312–319.

Bouvier, S. E., & Engel, S. A. (2006). Behavioral deficits and cortical damage loci in cerebral achromatopsia. *Cerebral Cortex, 16*(2), 183–191.

Brainard, D. H. (1997). The psychophysics toolbox. *Spatial Vision, 10*(4), 433–436.

Caggiano, V., Fogassi, L., Rizzolatti, G., Thier, P., & Casile, A. (2009). Mirror neurons differentially encode the peripersonal and extrapersonal space of monkeys. *Science, 324*(5925), 403–406.

Calvert, G. A. (2001). Crossmodal processing in the human brain: Insights from functional neuroimaging studies. *Cerebral Cortex, 11*(12), 1110–1123.

Calvo-Merino, B., Grezes, J., Glaser, D. E., Passingham, R. E., & Haggard, P. (2006). Seeing or doing? Influence of visual and motor familiarity in action observation. *Current Biology, 16*(19), 1905–1910.

Casile, A., & Giese, M. A. (2006). Nonvisual motor training influences biological motion perception. *Current Biology, 16*(1), 69–74.

Cattaneo, L., & Rizzolatti, G. (2009). The mirror neuron system. *Archives of Neurology, 66*(5), 557–560.

Chaminade, T., & Hodgins, J. K. (2006). Artificial agents in social cognitive sciences. *Interaction Studies, 7*(3), 347–353.

Chaminade, T., Hodgins, J., & Kawato, M. (2007). Anthropomorphism influences perception of

computer-animated characters' actions. *Social Cognitive and Affective Neuroscience, 2*(3), 206–216.

Cipolloni, P. B., & Pandya, D. N. (1999). Cortical connections of the frontoparietal opercular areas in the rhesus monkey. *Journal of Comparative Neurology, 403*(4), 431–458.

Collins, D. L., Neelin, P., Peters, T. M., & Evans, A. C. (1994). Automatic 3D intersubject registration of MR volumetric data in standardized Talairach space. *Journal of Computer Assisted Tomography, 18*(2), 192–205.

Cook, J., Saygin, A. P., Swain, R., & Blakemore, S. J. (2009). Reduced sensitivity to minimum-jerk biological motion in autism spectrum conditions. *Neuropsychologia*.

Cowey, A., & Vaina, L. M. (2000). Blindness to form from motion despite intact static form perception and motion detection. *Neuropsychologia, 38*(5), 566–578.

Cross, E. S., Kraemer, D. J., Hamilton, A. F., Kelley, W. M., & Grafton, S. T. (2009). Sensitivity of the action observation network to physical and observational learning. *Cerebral Cortex, 19*(2), 315–326.

Cutting, J. E., & Kozlowski, L. T. (1977). Recognizing friends by their walk: Gait perception without familiarity cues. *Bulletin of the Psychonomic Society, 9*, 353–356.

Dale, A. M., Fischl, B., & Sereno, M. I. (1999). Cortical surface-based analysis. I. Segmentation and surface reconstruction. *NeuroImage, 9*(2), 179–194.

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

De Lussanet, M. H. E., Fadiga, L., Michels, L., Seitz, R. J., Kleiser, R., & Lappe, M. (2008). Interaction of visual hemifield and body view in biological motion perception. *European Journal of Neuroscience, 27*(2), 514–522.

Dinstein, I., Thomas, C., Behrmann, M., & Heeger, D. J. (2008). A mirror up to nature. *Current Biology, 18*(1), R13–R18.

Dittrich, W., Lea, S., Barrett, J., & Gurr, P. (1998). Categorization of natural movements by pigeons: Visual concept discrimination and biological motion. *Journal of the Experimental Analysis of Behavior, 70*(3), 281–299.

Driver, J., & Noesselt, T. (2008). Multisensory interplay reveals crossmodal influences on 'sensory-specific' brain regions, neural responses, and judgments. *Neuron, 57*(1), 11–23.

Dronkers, N. F., Wilkins, D. P., Van Valin, R. D., Jr., Redfern, B. B., & Jaeger, J. J. (2004). Lesion analysis of the brain areas involved in language comprehension. *Cognition, 92*(1–2), 145–177.

Fadiga, L., Fogassi, L., Pavesi, G., & Rizzolatti, G. (1995). Motor facilitation during action observation: A magnetic stimulation study. *Journal of Neurophysiology, 73*(6), 2608–2611.

Ferrari, P. F., Gallese, V., Rizzolatti, G., & Fogassi, L. (2003). Mirror neurons responding to the observation of ingestive and communicative mouth actions in the monkey ventral premotor cortex. *European Journal of Neuroscience, 17*(8), 1703–1714.

Fischl, B., Sereno, M. I., & Dale, A. M. (1999). Cortical surface-based analysis. II: Inflation, flattening, and a surface-based coordinate system. *NeuroImage, 9*(2), 195–207.

Fischl, B., Sereno, M. I., Tootell, R. B., & Dale, A. M. (1999). High-resolution intersubject averaging and a coordinate system for the cortical surface. *Human Brain Mapping, 8*(4), 272–284.

Fogassi, L., Ferrari, P. F., Gesierich, B., Rozzi, S., Chersi, F., & Rizzolatti, G. (2005). Parietal lobe: From action organization to intention understanding. *Science, 308*(5722), 662–667.

Fox, R., & McDaniel, C. (1982). The perception of biological motion by human infants. *Science, 218*(4571), 486–487.

Freitag, C. M., Konrad, C., Haberlen, M., Kleser, C., von Gontard, A., Reith, W., et al. (2008). Perception of biological motion in autism spectrum disorders. *Neuropsychologia, 46*(5), 1480–1494.

Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain, 119*(Pt. 2), 593–609.

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

Gilaie-Dotan, S., Bentin, S., Harel, A., Rees, G., & Saygin, A. P. (2011). Normal form from biological motion despite impaired ventral stream function. *Neuropsychologia, 49*(5), 1033–1043.

Gilaie-Dotan, S., Bentin, S., Rees, G., Behrmann, M., & Saygin, A. P. (2012). Biological motion perception in patients with form processing deficits. *Society for Neuroscience*, Oct 2012, New Orleans, LA.

Grafton, S. T., Arbib, M. A., Fadiga, L., & Rizzolatti, G. (1996). Localization of grasp representations

in humans by positron emission tomography. 2. Observation compared with imagination. *Experimental Brain Research, 112*(1), 103–111.

Gregoriou, G. G., Borra, E., Matelli, M., & Luppino, G. (2006). Architectonic organization of the inferior parietal convexity of the macaque monkey. *Journal of Comparative Neurology, 496*(3), 422–451.

Gregoriou, G. G., Luppino, G., Matelli, M., & Savaki, H. E. (2005). Frontal cortical areas of the monkey brain engaged in reaching behavior: A (14)C-deoxyglucose imaging study. *NeuroImage, 27*(2), 442–464.

Grezes, J., Fonlupt, P., Bertenthal, B., Delon-Martin, C., Segebarth, C., & Decety, J. (2001). Does perception of biological motion rely on specific brain regions? *NeuroImage, 13*(5), 775–785.

Grossman, E. D., Battelli, L., & Pascual-Leone, A. (2005). Repetitive TMS over posterior STS disrupts perception of biological motion. *Vision Research, 45*(22), 2847–2853.

Grossman, E. D., & Blake, R. (2002). Brain areas active during visual perception of biological motion. *Neuron, 35*(6), 1167–1175.

Grossman, E. D., Donnelly, M., Price, R., Pickens, D., Morgan, V., Neighbor, G., et al. (2000). Brain areas involved in perception of biological motion. *Journal of Cognitive Neuroscience, 12*(5), 711–720.

Hagler, D. J., Jr., Saygin, A. P., & Sereno, M. I. (2006). Smoothing and cluster thresholding for cortical surface-based group analysis of fMRI data. *NeuroImage, 33*(4), 1093–1103.

Hamilton, A. F., Brindley, R. M., & Frith, U. (2007). Imitation and action understanding in autistic spectrum disorders: How valid is the hypothesis of a deficit in the mirror neuron system? *Neuropsychologia, 45*(8), 1859–1868.

Hari, R., Forss, N., Avikainen, S., Kirveskari, E., Salenius, S., & Rizzolatti, G. (1998). Activation of human primary motor cortex during action observation: A neuromagnetic study. *Proceedings of the National Academy of Science USA, 95*(25), 15061–15065.

Heiligenberg, W. (1991). Sensory control of behavior in electric fish. *Current Opinion in Neurobiology, 1*(4), 633–637.

Hein, G., & Knight, R. T. (2008). Superior temporal sulcus—It's my area: Or is it? *Journal of Cognitive Neuroscience, 20*(12), 2125–2136.

Hiris, E. (2007). Detection of biological and nonbiological motion. *Journal of Vision, 7*(12), 4 1–16.

Iacoboni, M. (2009). Imitation, empathy, and mirror neurons. *Annual Review of Psychology, 60*, 653–670.

Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews. Neuroscience, 7*(12), 942–951.

Iacoboni, M., Woods, R. P., Brass, M., Bekkering, H., Mazziotta, J. C., & Rizzolatti, G. (1999). Cortical mechanisms of human imitation. *Science, 286*(5449), 2526–2528.

Jacobs, A., & Shiffrar, M. (2005). Walking perception by walking observers. *Journal of Experimental Psychology: Human Perception and Performance, 31*(1), 157–169.

Jastorff, J., Kourtzi, Z., & Giese, M. A. (2006). Learning to discriminate complex movements: Biological versus artificial trajectories. *Journal of Vision, 6*(8), 791–804.

Jeannerod, M. (2001). Neural simulation of action: A unifying mechanism for motor cognition. *NeuroImage, 14*(1).

Jellema, T., & Perrett, D. I. (2003). Cells in monkey STS responsive to articulated body motions and consequent static posture: A case of implied motion? *Neuropsychologia, 41*(13), 1728–1737.

Johansson, G. (1973). Visual perception of biological motion and a model for its analysis. *Perception and Psychophysics, 14*(2), 201–211.

Jung, W. H., Gu, B. M., Kang, D. H., Park, J. Y., Yoo, S. Y., Choi, C. H., et al. (2009). BOLD response during visual perception of biological motion in obsessive-compulsive disorder. *European Archives of Psychiatry and Clinical Neuroscience, 259*(1), 46–54.

Keysers, C., & Gazzola, V. (2006). Towards a unifying neural theory of social cognition. *Progress in Brain Research, 156*, 379–401.

Keysers, C., Wicker, B., Gazzola, V., Anton, J. L., Fogassi, L., & Gallese, V. (2004). A touching sight: SII/PV activation during the observation and experience of touch. *Neuron, 42*(2), 335–346.

Kilner, J. M., Neal, A., Weiskopf, N., Friston, K. J., & Frith, C. D. (2009). Evidence of mirror neurons in human inferior frontal gyrus. *Journal of Neuroscience, 29*(32), 10153–10159.

Kohler, E., Keysers, C., Umilta, M. A., Fogassi, L., Gallese, V., & Rizzolatti, G. (2002). Hearing sounds, understanding actions: action representation in mirror neurons. *Science, 297*(5582), 846–848.

Kveraga, K., Ghuman, A. S., & Bar, M. (2007). Top-down predictions in the cognitive brain. *Brain Cognition, 65*(2), 145–168.

Leff, A. P., Schofield, T. M., Crinion, J. T., Seghier, M. L., Grogan, A., Green, D. W., et al. (2009). The left superior temporal gyrus is a shared substrate for auditory short-term memory and speech comprehension: Evidence from 210 patients with stroke. *Brain, 132*(Pt. 12), 3401–3410.

Luppino, G., Rozzi, S., Calzavara, R., & Matelli, M. (2003). Prefrontal and agranular cingulate projections to the dorsal premotor areas F2 and F7 in the macaque monkey. *European Journal of Neuroscience, 17*(3), 559–578.

Matelli, M., & Luppino, G. (2001). Parietofrontal circuits for action and space perception in the macaque monkey. *NeuroImage, 14*(1 Pt. 2), S27–S32.

Matelli, M., & Luppino, G. (2004). Architectonics of the primate's cortex: Usefulness and limits. *Cortex, 40*(1), 209–210.

McLeod, P., Dittrich, W., Driver, J., Perrett, D., & Zihl, J. (1996). Preserved and impaired detection of structure from motion by a "motion blind" patient. *Visual Cognition, 3*, 363–391.

Michels, L., Kleiser, R., de Lussanet, M. H. E., Seitz, R. J., & Lappe, M. (2009). Brain activity for peripheral biological motion in the posterior superior temporal gyrus and the fusiform gyrus: Dependence on visual hemifield and view orientation. *NeuroImage, 45*(1), 151–159.

Michels, L., Lappe, M., & Vaina, L. M. (2005). Visual areas involved in the perception of human movement from dynamic form analysis. *Neuroreport, 16*(10), 1037–1041.

Miller, L., & Saygin, A. P. (2012) Intersubject variability in the use of form and motion cues during biological motion perception. *Vision Sciences Society*, May 2012, Naples, Florida, USA.

Mort, D. J., Malhotra, P., Mannan, S. K., Rorden, C., Pambakian, A., Kennard, C., et al. (2003). The anatomy of visual neglect. *Brain, 126*(Pt. 9), 1986–1997.

Murphy, P., Brady, N., Fitzgerald, M., & Troje, N. F. (2009). No evidence for impaired perception of biological motion in adults with autistic spectrum disorders. *Neuropsychologia*.

Nelissen, K., Vanduffel, W., & Orban, G. A. (2006). Charting the lower superior temporal region, a new motion-sensitive region in monkey superior temporal sulcus. *Journal of Neuroscience, 26*(22), 5929–5947.

Nishitani, N., & Hari, R. (2000). Temporal dynamics of cortical representation for action. *Proceedings of the National Academy of Science USA, 97*(2), 913–918.

Oberman, L. M., & Ramachandran, V. S. (2007). The simulating social mind: The role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychological Bulletin, 133*(2), 310–327.

Oram, M. W., & Perrett, D. I. (1996). Integration of form and motion in the anterior superior temporal polysensory area (STPa) of the macaque monkey. *Journal of Neurophysiology, 76*(1), 109–129.

Parron, C., Da Fonseca, D., Santos, A., Moore, D. G., Monfardini, E., & Deruelle, C. (2008). Recognition of biological motion in children with autistic spectrum disorders. *Autism, 12*(3), 261–274.

Pavlova, M., Bidet-Ildei, C., Sokolov, A. N., Braun, C., & Krageloh-Mann, I. (2009). Neuromagnetic response to body motion and brain connectivity. *Journal of Cognitive Neuroscience, 21*(5), 837–846.

Pavlova, M., Krageloh-Mann, I., Sokolov, A., & Birbaumer, N. (2001). Recognition of point-light biological motion displays by young children. *Perception, 30*(8), 925–933.

Pavlova, M., & Sokolov, A. (2003). Prior knowledge about display inversion in biological motion perception. *Perception, 32*(8), 937–946.

Pavlova, M., Sokolov, A., Birbaumer, N., & Krageloh-Mann, I. (2006). Biological motion processing in adolescents with early periventricular brain damage. *Neuropsychologia, 44*(4), 586–593.

Pavlova, M., Staudt, M., Sokolov, A., Birbaumer, N., & Krageloh-Mann, I. (2003). Perception and production of biological movement in patients with early periventricular brain lesions. *Brain, 126*(Pt. 3), 692–701.

Peelen, M. V., Wiggett, A. J., & Downing, P. E. (2006). Patterns of fMRI activity dissociate overlapping functional brain areas that respond to biological motion. *Neuron, 49*(6), 815–822.

Pelphrey, K. A., Morris, J. P., & McCarthy, G. (2004). Grasping the intentions of others: The perceived intentionality of an action influences activity in the superior temporal sulcus

during social perception. *Journal of Cognitive Neuroscience, 16*(10), 1706–1716.

Petrides, M., & Pandya, D. N. (1988). Association fiber pathways to the frontal cortex from the superior temporal region in the rhesus monkey. *Journal of Comparative Neurology, 273*(1), 52–66.

Petrides, M., & Pandya, D. N. (2009). Distinct parietal and temporal pathways to the homologues of Broca's area in the monkey. *PLoS Biology, 7*(8), e1000170.

Petrini, K., Dahl, S., Rocchesso, D., Waadeland, C. H., Avanzini, F., Puce, A., et al. (2009). Multisensory integration of drumming actions: Musical expertise affects perceived audiovisual asynchrony. *Experimental Brain Research, 198*(2–3), 339–352.

Petrini, K., Russell, M., & Pollick, F. (2009). When knowing can replace seeing in audiovisual integration of actions. *Cognition, 110*(3), 432–439.

Peuskens, H., Vanrie, J., Verfaillie, K., & Orban, G. A. (2005). Specificity of regions processing biological motion. *European Journal of Neuroscience, 21*(10), 2864–2875.

Pinto, J., & Shiffrar, M. (2009). The visual perception of human and animal motion in point-light displays. *Social Neuroscience, 4*(4), 332–346.

Pobric, G., & Hamilton, A. F. (2006). Action understanding requires the left inferior frontal cortex. *Current Biology, 16*(5), 524–529.

Puce, A., & Perrett, D. (2003). Electrophysiology and brain imaging of biological motion. *Philosophical Transactions of the Royal Society of London B: Biological Sciences, 358*(1431), 435–445.

Pyles, J. A., & Grossman, E. D. (2012). Neural mechanisms for biological motion and animacy. In K. Johnson & M. Shiffrar (Eds.), People watching: Social, perceptual, and neurophysiological studies of body perception (Chapter 17). New York: Oxford University Press.

Redcay, E. (2008). The superior temporal sulcus performs a common function for social and speech perception: Implications for the emergence of autism. *Neuroscience and Biobehavioral Reviews, 32*(1), 123–142.

Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Review of Neuroscience, 27*, 169–192.

Rizzolatti, G., Fadiga, L., Gallese, V., & Fogassi, L. (1996). Premotor cortex and the recognition of motor actions. *Brain Research. Cognitive Brain Research, 3*(2), 131–141.

Rolls, E. T. (2007). The representation of information about faces in the temporal and frontal lobes. *Neuropsychologia, 45*(1), 124–143.

Rorden, C., & Brett, M. (2000). Stereotaxic display of brain lesions. *Behavioral Neurology, 12*(4), 191–200.

Rorden, C., & Karnath, H. O. (2004). Using human brain lesions to infer function: A relic from a past era in the fMRI age? *Nature Reviews. Neuroscience, 5*(10), 813–819.

Rozzi, S., Calzavara, R., Belmalih, A., Borra, E., Gregoriou, G. G., Matelli, M., et al. (2006). Cortical connections of the inferior parietal cortical convexity of the macaque monkey. *Cerebral Cortex, 16*(10), 1389–1417.

Rozzi, S., Ferrari, P. F., Bonini, L., Rizzolatti, G., & Fogassi, L. (2008). Functional organization of inferior parietal lobule convexity in the macaque monkey: Electrophysiological characterization of motor, sensory and mirror responses and their correlation with cytoarchitectonic areas. *European Journal of Neuroscience, 28*(8), 1569–1588.

Saunier, G., Papaxanthis, C., Vargas, C. D., & Pozzo, T. (2008). Inference of complex human motion requires internal models of action: Behavioral evidence. *Experimental Brain Research, 185*(3), 399–409.

Saygin, A. P. (2007). Superior temporal and premotor brain areas necessary for biological motion perception. *Brain, 130*(Pt. 9), 2452–2461.

Saygin, A. P., Chaminade, T., & Ishiguro, H. (2010). *The perception of humans and robots: Uncanny hills in parietal cortex.* Paper presented at the Cognitive Science Society, Portland, Oregon.

Saygin, A. P., Chaminade, T., Ishiguro, H., Driver, J., & Frith, C. (2012). The thing that should not be: Predictive coding and the uncanny valley in perceiving human and humanoid robot actions. *Social Cognitive Affective Neuroscience*, *7*(4), 413–422.

Saygin, A. P., Cicekli, I., & Akman, V. (2000). Turing test: 50 years later. *Minds and Machines, 10*(4), 463–518.

Saygin, A. P., Cook, J., Blakemore, S.-J. (2010). Unaffected perceptual thresholds for biological and non-biological form-from-motion perception in Autism Spectrum Conditions. *PLoS ONE, 5*(10), e13491.

Saygin, A. P., Driver, J., & de Sa, V. R. (2008). In the footsteps of biological motion and multisensory perception: Judgments of audiovisual

temporal relations are enhanced for upright walkers. *Psychological Science, 19*(5), 469–475

Saygin, A. P., & Sereno, M. I. (2008). Retinotopy and attention in human occipital, temporal, parietal, and frontal cortex. *Cerebral Cortex, 18*(9), 2158–2168.

Saygin, A. P., & Stadler, W. (2012). The role of visual appearance in action prediction. *Psychological Research*. In Press.

Saygin, A. P., Wilson, S. M., Dronkers, N. F., & Bates, E. (2004). Action comprehension in aphasia: Linguistic and non-linguistic deficits and their lesion correlates. *Neuropsychologia, 42*(13), 1788–1804.

Saygin, A. P., Wilson, S. M., Hagler, D. J., Jr., Bates, E., & Sereno, M. I. (2004). Point-light biological motion perception activates human premotor cortex. *Journal of Neuroscience, 24*(27), 6181–6188.

Schenk, T., & Zihl, J. (1997). Visual motion perception after brain damage: II. Deficits in form-from-motion perception. *Neuropsychologia, 35*(9), 1299–1310.

Schutz, M., & Lipscomb, S. (2007). Hearing gestures, seeing music: Vision influences perceived tone duration. *Perception, 36*(6), 888–897.

Sereno, M. I., & Tootell, R. B. (2005). From monkeys to humans: What do we now know about brain homologies? *Current Opinion in Neurobiology, 15*(2), 135–144.

Serino, A., De Filippo, L., Casavecchia, C., Coccia, M., Shiffrar, M., & Ladavas, E. (2010). Lesions to the motor system affect action perception. *Journal of Cognitive Neuroscience, 22*(3), 413–426.

Servos, P., Osu, R., Santi, A., & Kawato, M. (2002). The neural substrates of biological motion perception: An fMRI study. *Cerebral Cortex, 12*(7), 772–782.

Silvanto, J., & Muggleton, N. G. (2008). New light through old windows: Moving beyond the "virtual lesion" approach to transcranial magnetic stimulation. *NeuroImage, 39*(2), 549–552.

Simion, F., Regolin, L., & Bulf, H. (2008). A predisposition for biological motion in the newborn baby. *Proceedings of the National Academy of Science USA, 105*(2), 809–813.

Sokolov, A. A., Gharabaghi, A., Tatagiba, M. S., & Pavlova, M. (2010). Cerebellar engagement in an action observation network. *Cerebral Cortex, 20*(2), 486–491.

Stanton, G. B., Bruce, C. J., & Goldberg, M. E. (1995). Topography of projections to posterior cortical areas from the macaque frontal eye fields. *Journal of Comparative Neurology, 353*(2), 291–305.

Strafella, A. P., & Paus, T. (2000). Modulation of cortical excitability during action observation: A transcranial magnetic stimulation study. *Neuroreport, 11*(10), 2289–2292.

Tranel, D., Kemmerer, D., Damasio, H., Adolphs, R., & Damasio, A. R. (2003). Neural correlates of conceptual knowledge for actions. *Cognitive Neuropsychology, 20*, 409–432.

Vaina, L. M., & Gross, C. G. (2004). Perceptual deficits in patients with impaired recognition of biological motion after temporal lobe lesions. *Proceedings of the National Academy of Science USA, 101*(48), 16947–16951.

Vaina, L. M., Lemay, M., Bienfang, D. C., Choi, A. Y., & Nakayama, K. (1990). Intact "biological motion" and "structure from motion" perception in a patient with impaired motion mechanisms: A case study. *Visual Neuroscience, 5*(4), 353–369.

Vaina, L. M., Solomon, J., Chowdhury, S., Sinha, P., & Belliveau, J. W. (2001). Functional neuroanatomy of biological motion perception in humans. *Proceedings of the National Academy of Science USA, 98*(20), 11656–11661.

Vallortigara, G., Regolin, L., & Marconato, F. (2005). Visually inexperienced chicks exhibit spontaneous preference for biological motion patterns. *PLoS Biology, 3*(7), e208.

Vangeneugden, J., Pollick, F., & Vogels, R. (2009). Functional differentiation of macaque visual temporal cortical neurons using a parametric action space. *Cerebral Cortex, 19*(3), 593–611.

van Kemenade, B. M., Muggleton, N., Walsh, V., & Saygin, A. P. (2012). The effects of TMS over STS and premotor cortices on the perception of biological motion. *Journal of Cognitive Neuroscience*, 24(4), 896–904.

Walsh, V., & Rushworth, M. (1999). A primer of magnetic stimulation as a tool for neuropsychology. *Neuropsychologia, 37*(2), 125–135.

Watson, A. B., & Pelli, D. G. (1983). QUEST: A Bayesian adaptive psychometric method. *Perception & Psychophysiology, 33*(2), 113–120.

Wicker, B., Keysers, C., Plailly, J., Royet, J. P., Gallese, V., & Rizzolatti, G. (2003). Both of us disgusted in My insula: The common neural basis of seeing and feeling disgust. *Neuron, 40*(3), 655–664.