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Embodied and Disembodied Emotion Processing: Learning From and About Typical and Autistic Individuals

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Abstract

Successful social functioning requires quick and accurate processing of emotion and generation of appropriate reactions. In typical individuals, these skills are supported by embodied processing, recruiting central and peripheral mechanisms. However, emotional processing is atypical in individuals with autism spectrum disorder (ASD). Individuals with ASD show deficits in recognition of briefly presented emotional expressions. They tend to recognize expressions using rule-based, rather than template, strategies. Individuals with ASD also do not spontaneously and quickly mimic emotional expressions, unless the task encourages engagement. When processing emotional scenes, ASD individuals show atypical basic motivational responses, despite intact ability to verbally determine stimulus valence. We discuss how these findings highlight the contribution of both embodied and disembodied mechanisms to typical and atypical emotional functioning.

Keywords
autism, embodiment, emotion

Social life is filled with emotional information. Friends smile and embrace. Enemies frown and shrug. Lovers flirt with eyes and bodies. Sales people may grin and rattle with excitement or give us that "don't bother me" look, just as students may look at us with admiration or boredom. Cats and dogs, too, wiggle with joy or tremble with anxiety. Even computers tease us with emoticons or flash alluring images on Web pages, and fast food restaurants and big box stores greet us with smiley faces. We are also often exposed to emotional scenes in the movies and television, ranging from uplifting, cute and delightful to painful, horrifying and disgusting. Much emotional information is perceived and understood within a blink of an eye, prompting us to like or dislike, approach or avoid, engage or disengage.

Clearly, social interaction frequently requires us to recognize emotion from facial, vocal, and postural information. This recognition may then influence our physiology, motivation, behavior, thought, and judgment. But how does this process work? And how does it go awry? In this article, we argue that new insights into how humans perceive, learn, understand, represent, and use emotionally significant information can be offered by looking at individuals with atypical social-emotional functioning, such as individuals with autism spectrum disorders (ASD). Further, we argue that understanding of both autism and emotion can be advanced by theories of embodied cognition. That is, we aim to show how our understanding of autism has benefited from what we have learned about emotion embodiment, just as our understanding of emotion embodiment has benefited from what we have learned about autism.

To accomplish this, we first introduce embodiment theories of emotion, sketch out some of the underlying neural mechanisms, and provide background information on autism. Then,

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we outline the stages of the emotion process, including emotion perception, reaction, and understanding. After that, we review evidence for the role of embodied and disembodied processing in typical and atypical individuals at different stages of emotion processing. We close by discussing limitations of current theories and findings and outlining some questions for future research.

Embodied and Disembodied Theories of Emotional Processing

It helps to start with brief background on *embodied cognition theories* and their relation to more traditional models. Essentially, the embodiment theories arose as an alterative to symbolic accounts of information processing (Fodor, 1975). Under the symbolic models, information is initially encoded in the sensory modalities (e.g., vision, olfaction, audition, interoception). But, to interact with higher cognitive processes such as thought and language, this modal information must be "transduced" into arbitrary amodal "language-like" symbols that bear no analogical relationship to the experienced event. In contrast, the modern embodied cognition theories propose that low-order as well as high-order processing relies on modalities—perceptual, somatosensory, introceptive, and motor resources (Barsalou, 1999, 2008; Glenberg & Robinson, 2000; Prinz, 2002; Wilson, 2002). In this account, modalities are a critical part of "on-line" cognition (perceiving and understanding the present stimulus) as well as "off-line" cognition (thinking about the absent stimulus). A notion shared by many embodiment theories is that recruitment of somatosensory resources often involves "embodied simulation" (Gallese, 2003). "Simulation" can be thought of as the off-line projection of a perceived stimulus back onto the observer's own motor, cognitive, and emotional representations. Thus, the mechanisms by which we understand the emotional states of others overlap with mechanisms by which we experience those states ourselves.[1]

An important assumption of many (though not all) modern embodiment theories is that specific task characteristics determine the extent to which embodied simulation, rather than "disembodied" processing, is required for perceptual or conceptual operation. Further, task characteristics determine what aspects need to be simulated (e.g., what specific modality). In other words, modern embodiment theorists tend to see embodied simulation as a goal-driven, situated and flexible process that works collaboratively with more "cold" mechanisms for associative and rule-based processes (Barsalou, 2008).

Recently, embodiment framework has been more specifically applied to understanding the processing of emotional information (Niedenthal, 2007; Niedenthal, Barsalou, Winkielman, Krauth-Gruber, Ric 2005; Winkielman, Niedenthal, & Oberman, 2008). The embodied emotional processing account challenges the traditional amodal view, which assumes that individuals perceive and understand emotion equivalent to how they perceive and understand most other things (e.g., Ortony, Clore, & Collins, 1988). For example, according to the traditional symbolic amodal theories, the process by which perception of a face results in the belief "this person is smiling" is not fundamentally different from the process by which perception of a watch face results in a belief that "this watch is showing a time of 2:50." Similarly, just as people conceptually understand that the "car" possesses the features "engine" and "tires," they understand that "anger" possesses the features of "thwarted goal" and a "desire to strike out."

In contrast, the embodied framework assumes that perceptual and conceptual emotion processing recruits somatosensory resources (which can be peripheral and central, as discussed shortly). For example, when recognizing a smiling face, the perceiver may partially generate her own happy expression, using either peripheral feedback or the central face representation. In another example, when analyzing a stressful situation, such as her partner's pain, the perceiver may partially generate her own introceptive experience of pain (Singer et al., 2004). Similarly, thinking about an absent emotional stimulus (e.g., recalling a lying politician) recruits states triggered during the original encounter with an emotion-eliciting stimulus (e.g., internal cringe felt when first hearing his speech) and is informed by new somatosensory reactions generated during the thought process (e.g., clenching of her own fist when thinking about the liar).

As we will review shortly, there is growing evidence for the role of embodiment across different stages of emotion processing. Admittedly, much of it is still in the form of simply showing a cross-talk between emotional and sensorimotor processes. This raises the potential issue of the extent to which embodiments are truly constitutive or reflect causally inefficacious byproducts of higher-order processes. Fortunately, as we discuss later, there are now several intervention studies using various blocking and facilitation methods which show that peripheral and central sensorimotor processes play a causal role in emotional perception and understanding.

Neural Basis of Emotional Embodiment: Peripheral and Central Mechanisms

The modality-based view of emotion processing is informed by the growing knowledge about the biological mechanisms of emotion processing. A detailed review of neural substrates is outside the scope of this article and is offered in the article by Heberlein and Atkinson (2009). However, it is useful to touch on some general issues and highlight some distinctions and issues (we will also discuss neural instantiation when reviewing specific findings).

At the outset, it is worth emphasizing that modern embodiment theories view emotion processing as a closed loop in which perceptual and conceptual operations "run on" intrinsically intertwined peripheral and central representations (Barsalou, 1999, 2008). Modern embodiment theories do not assume a particular priority of peripheral and central processes, as different tasks and contexts may require more peripheral or central simulation.

It is also worth addressing the criticism of the idea that peripheral feedback, which informs the CNS about the state of the muscles and the viscera via the afferent neurons to the cortex, can be an important part of the emotion process. The critics often recall Cannon's (1927) rebuttal of James-Lange

emotion theory (James, 1896/1994) and argue that peripheral feedback is too undifferentiated and too slow to meaning fully support emotion processing. However, many of these arguments are misplaced (see Zajonc & Markus, 1984).[2] Take the example of peripheral feedback from facial muscles. It certainly can be quite differentiated and fast. After all, the human face has at least 24 muscles relevant for facial expressions—a number allowing an impressive number of combinations (Tassinary & Cacioppo, 2000). Further, facial expressions can emerge as fast as 300 ms after the stimulus onset (Dimberg, Thunberg, & Elmehed, 2000). Most importantly, a review of the evidence indicates that facial actions can both initiate and modulate emotional experience (McIntosh, 1996). As we will discuss, there are limits to the size of these feedback effects, and there are other pathways to emotional recognition and emotional experience, but it is incorrect to principally dismiss the role of peripheral feedback on anatomical and physiological grounds.

As mentioned, modern embodiment theories assume that peripheral input (muscles, viscera) works together with the central systems which represent the body and can engage in modal simulations (Damasio, 1994; Gallese, 2003). Those quick and high-resolution central systems include perceptual modalities (i.e., vision, audition, touch, etc.); somatosensory and motor cortices (i.e., internal representation of the body state); the putative "mirror neuron system" (i.e., mapping of action goals and linking self representation to others); and value-representing regions in the limbic system and orbitofrontal cortex (i.e., representation of emotional information within "as-if loops").

Finally, it is worth highlighting that modern embodiment theories do not wish to revive the Jamesian notion of bodily feedback, with their emphasis on the priority of the peripheral nervous system, as contrasted with the central nervous system. Or to move the Jamesian position up the processing chain and simply place the peripheral bodily feedback in "as-if" loops of the brain (Damasio, 1994). The essence of modern embodiment theories is the emphasis on *modal*, analogue, perception-like representations (thus, the Perceptual Symbol Systems approach). Those representations can be generated peripherally, but they can also be generated entirely centrally, across a variety of sensory systems, including the central components of the affective system (Barsalou, 1999, 2008).[3]

Autism

Our main argument here is that, in addition to the more standard "disembodied" mechanisms, emotion perception and understanding are supported by embodied simulation mechanisms that draw on peripheral and central resources. Thus, the idea naturally arises that there could be important consequences if these mechanisms dysfunction. This idea underlies the current "embodied simulation" theories of the etiology of autism spectrum disorders (ASD). Before we go any further, it is useful to provide some basic information about these disorders.

ASD includes Autism, Asperger syndrome (AS), and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS) and represents a complex and multifaceted condition characterized by a mosaic of deficits in three general areas: (a) social interaction, such as lack of social interest, social skills, or theory-of-mind, (b) communicative skills, including pragmatic language, and (c) the presence of restricted, repetitive, and stereotyped patterns of behaviors, interests and activities (American Psychiatric Association, 1994).

One of the most striking aspects of ASD are emotional deficits, which some researchers view as primary to other social and cognitive dysfunctions (e.g. Hobson, 1999; Rogers & Pennington, 1991). Notably, not all areas of emotional functioning are impaired. Individuals with ASD, at least on the high-functioning end of the spectrum, express full range of emotion, show attachment behaviors, and comprehend a variety of emotional situations. But, these individuals seem atypical on affective processes involved in creating emotional reciprocity (Hobson, 1999; Rogers & Pennington, 1991). Though we focus on emotion in this review, it is important to note that simulation theories of ASD are not restricted to emotional functioning but cover a wide spectrum of processes ranging from action perception, imitation, and understanding of other minds (Iacoboni & Dapretto, 2006; Oberman & Ramachandran, 2007; Williams, Whiten, & Singh, 2004).

A few notes are in order about the general challenges of research in ASD. Elucidating the underlying neural bases of ASD is difficult because the behavioral manifestations of these disorders vary both in severity (low and high-functioning) as well as in expression (Autism, AS, PDD-NOS). Currently, different ASDs are often lumped together when reporting findings, sometimes obscuring the information about the level of functioning for which the findings are relevant. It is fair to say, however, that though high-functioning individuals with autism and individuals with AS do not make up the majority of the ASD population (around 25%), they do make up the majority of the participants in published studies. This makes it difficult to know whether the findings are generalizable to the whole spectrum. However, there are many ethical and practical reasons why it is difficult to include lower functioning individuals in research studies. Frequently research requires the person to pay attention to a task, stay engaged for an extended period of time, require a certain degree of cognitive skill to follow instruction, and be tolerant of experimental procedures (e.g., electrode attachment). These requirements often make it practically difficult to include lower-functioning children in these studies. Still, the reader may want to keep these limitations in mind when we later discuss specific studies on individuals with ASD.

Finally, because our review focuses on spontaneous, largely automatic, low-level emotional processes, we are unable to cover a full range of what is known about emotion in ASD. We also do not discuss any sensory and cognitive atypicalities. For a more comprehensive perspective on this complex condition the reader should consult Frith (2003) or Schreibman (2005).

Emotion Processes

In the previous sections, we articulated the basic ideas behind the embodiment approach to emotion, sketched some of the

neural mechanisms, and provided some basic background information about autism. In the sections that follow, we review some work that investigated emotion processing in typical individuals and individuals with ASD. This review is organized roughly in terms of different emotion tasks that an individual faces in social life. That is, we start with emotion perception—how does the individual figure out what stimulus is presented? Then we move to emotional reaction—how does the individual mobilize internal resources in order to properly cope with an emotional situation? After that, we will turn to emotional understanding—how does the individual represent abstract emotion concepts? Importantly, this division—into perception, reaction and understanding—is primarily expositional because, as mentioned earlier, the essence of the embodiment approach is that perception, reaction and understanding are intrinsically intertwined and support each other.

Emotional Perception

How do people perceive another's emotion? For example, how do they figure out whether someone's face is happy or angry? As mentioned earlier, the symbolic models treat perception of emotion, including from the face, the same as perception of other complex stimuli. In contrast, the embodiment approach points out that people do not only see faces, they also *have* faces. This raises the possibility that mechanisms underlying moving and sensing one's own face are involved in perceiving other's emotional expressions. The supportive evidence for the role of embodied simulation in emotion perception comes from a variety of behavioral and neuroscience studies (for a review, see Adolphs, 2006; Goldman & Sripada, 2005). Those studies examined the involvement of somatosensory resources by manipulating and measuring both peripheral and central mechanisms in typical individuals and individuals with autism.

Peripheral Mechanisms in Typical Individuals. Much work on peripheral contributions to emotion perception comes from the paradigm in which participants passively watch emotional expressions, while their facial reactions are monitored using facial electromyography (EMG). Thus, only 300–400 ms after seeing an expression, typical individuals react to a smile with increased activity of the zygomaticus major (cheek muscle that pulls the corners of the mouth up and back), and to a frown with increased activity of the corrugator supercilii (muscle that knits the brow) (Lundqvist & Dimberg, 1995). Interestingly, such reactions can occur with minimal stimulus input, such as subthreshold presentations of expressions of happiness or anger (Dimberg et al., 2000).

Of course, activation of facial muscles while viewing facial expressions does not offer causal evidence for the role of these reactions in emotion perception. Answering these questions requires manipulating these muscular reactions and assessing its effect on face recognition. To do so, Niedenthal, Brauer, Halberstadt, and Innes-Ker (2001) asked participants to identify the point at which a morphed face changed from happy to sad and vice versa. During this task, some participants were free to move their faces naturally, whereas other participants were holding a pen sideways in their mouths, between their teeth and lips. This manipulation prevents facial mimicry and thus reduces somatic feedback that supports the detection of change in the observed expressions. Participants whose facial movements were blocked by the pen detected the change in expression later in both directions (happy to sad and sad to happy) than those who were able to move their face freely, supporting the role of facial mimicry in the recognition of facial expressions.

Oberman, Winkielman, and Ramachandran (2007) extended this study by adding several controls and, more importantly, examining the specificity of the mimicry-blocking effect. Note that the embodiment account predicts that recognition of a specific type of facial expressions should be impaired by blocking mimicry in the group of facial muscles used in the production of this type of expression. The authors tested this hypothesis using four expressions (happy, disgust, fear, and sad) and four manipulations of facial mimicry: holding a pen sideways between the teeth, chewing gum, holding the pen just with the lips, and no task. Experiment 1 employed EMG and found that holding a pen sideways between the teeth strongly activated muscles involved in producing expressions of happiness, and to some extent disgust and fear. In contrast, the gum manipulation broadly activated several facial muscles, but only intermittently (the lip manipulation had no effect on EMG). Testing for the accuracy of emotion discrimination, Experiment 2 found that the pen-biting manipulation significantly impaired the recognition of happiness (by about 12%), but had a smaller effect on the recognition accuracy for disgust (6%) and fear (4%), and no effect at all on recognition of sad expressions. This finding suggests that recognition of a specific type of facial expression involves the selective recruitment of muscles used to produce that expression, as predicted by embodiment accounts.

It is interesting to relate these findings to reports showing little relation between the perceivers' mimicry and their ability to recognize facial expressions. For example, Hess, Philippot & Blairy (1999) critically examined the relation between facial EMG to static and dynamic emotional faces, observer's decoding accuracy, and emotional contagion. For example, while in some of their studies observers did mimic the faces, and showed some contagion, the mimicry was not correlated with decoding accuracy (Blairy, Herrera, & Hess, 1999). Similarly, Calder, Keane, Cole, Campbell, & Young (2000a) found that three patients with Mobius Syndrome—a congenital condition that causes facial paralysis and thus prevents mimicry—were generally able to appropriately categorize standard emotional faces, with impairments noticeable only at high levels of recognition difficulty. Thus, it is important to keep in mind that peripheral feedback represents only one source of information that can be used for emotion recognition, and that it may play a causal role in recognition only under fairly specific task conditions, such as when the recognition task is novel, difficult, and cannot be solved using fast central embodied mechanisms or using simple "disembodied," feature-based mechanisms (e.g., detection of simple happiness-related features, such as lips turned up). We will return to this issue shortly.

Central Mechanisms in Typical Individuals. The central mechanisms of emotion recognition are complex and rely on a distributed cortical network (Adolphs, 2006; Heberlein & Atkinson, 2009). Thus, processing of facial expressions will draw on "core areas," such as the inferior occipital gyrus (IOG), which performs basic feature extraction and structural encoding; the fusiform gyrus (FG), which codes higher-order invariant features of the face; and superior temporal sulcus (STS), which codes variant and movement-related features (Haxby, Hoffman, & Gobbini, 2000). But, in addition to these core areas, recognizing facial expressions also recruits areas involved in (a) detecting emotional significance (amygdala), (b) sensing and moving the perceivers' own faces, such as the somatosensory and motor cortex, (c) interoception, such as the insula, and (d) linking bodily feedback to abstract cortical representation, such as the ventromedial prefrontal cortex (vmPFC). There are now several studies that point to the importance of these areas for emotional perception.

Winston, O'Doherty, and Dolan (2003) asked participants to view faces displaying disgust, fear, happiness, or sadness while their brain activity was measured with fMRI. Participants either made gender judgments (incidental task) or explicitly judged the presented emotion (direct task). The incidental task activated the core face perception network and the amygdala. However, during explicit emotion judgments, there was activation of somatosensory cortex and ventromedial prefrontal cortex, suggesting that participants engaged in embodied simulation.

Wicker and colleagues (2003) examined the connection between perception and experience of a specific emotion—disgust. They asked participants to first inhale disgusting odors, and then watch videos displaying other individuals expressing disgust. Results showed that the areas of the anterior insula and, to some extent, the anterior cingulate cortex were activated both when individuals experienced disgust themselves and when individuals observed disgust in others, presumably reflecting embodied simulation. Importantly, the interest value of these findings does not derive from specifically tying disgust to the insula, which as part of the introceptive cortex is involved in many hedonic and motivational states (e.g., Naqvi, Rudrauf, Damasio, & Bechara, 2007). These findings are interesting because they again highlight that perception of other people's emotional expressions involves "extended" cortical areas which represent the perceiver's own bodily state.

In discussing these studies we have not yet mentioned the classic mirror neuron area (inferior frontal cortex).[4] This is because this region is not always activated in imitation and emotion recognition tasks (Adolphs, 2006; Decety & Jackson, 2004). However, some studies do report such activation. Carr, Iacoboni, Dubeau, Mazziotta, and Lenzi (2003) asked participants to just observe or to observe and imitate emotional facial expressions. Compared to rest, both observation and imitation tasks activated a similar group of regions, including the inferior frontal cortex as well as the superior temporal cortex, insula, and amygdala. This inconsistency of findings in the fMRI literature raises some interesting methodological issues with potential theoretical implications. First, there is the standard issue of region of interest (ROI) analysis, with many studies not focusing on, and thus not reporting about, activations in the admittedly very large, and somewhat unspecified "mirror-neuron area" (Poldrack, 2006). Second, the emotion perception tasks used by different studies vary in the extent to which participants are engaged in the task, identify with the presented stimulus, and are encouraged to process it emotionally. For example, as mentioned earlier, the type of task can play a critical role, with only the explicit emotion-recognition tasks eliciting reliable somatosensory activations (Winston et al., 2003). Third, the studies vary in the degree to which motor activity is encouraged and permitted in the study. In fact, the study by Carr and colleagues (2003) mixed observation (perception) and imitation (action) blocks, which perhaps contributed to the observation of pre-motor activity on observation-only trials. As we discuss shortly, these variables (engagement, motor demands) also play a critical role in whether mirroring phenomena are observed in ASD. So we expect that future research, on typical and autistic individuals, will carefully examine how task parameters determine the engagement of a particular area.

Lesion and Inactivation Studies. The just-discussed fMRI studies leave unclear whether these "embodiment" areas causally contribute to emotion recognition or are merely a byproduct of perhaps a frequent pairing of emotional perception and emotion action. Fortunately, this question has been addressed by lesion and inactivation studies. Adolphs, Damasio, Tranel, Cooper, and Damasio (2000) asked 108 patients with a variety of focal brain lesions and 30 normal control participants to perform three visual emotion recognition tasks. In the first task, participants rated the intensity of basic emotional facial expressions. In the second task, participants matched a facial expression to its name. In the third task, participants sorted facial expressions into emotional categories. Though each task identified a slightly different group of regions, damage to primary and secondary somatosensory cortices impaired performance in all three tasks. This finding is consistent with the embodiment view in which emotion perception involves simulating the relevant state in the perceiver using somatosensory resources. Similarly, there is some evidence that insula damage results in a paired impairment in the experience and recognition of disgust (Calder, Keane, Cole, Campbell, & Young, 2000b). Though interesting for the causality issue, again these findings do not imply specificity of insula for disgust, as damage to this region affects a variety of positive and negative affective processes (e.g., Naqvi et al., 2007).

Focal brain lesion studies, while critical for establishing necessity, are subject to complex interpretations because of the inevitably messy nature of lesions, in terms of their extent, location, and non-specific effects on processing. Further, such studies cannot answer questions about the timing of area contribution. To address these issues, Pitcher, Garrido, Walsh, and Duchaine (2008) temporarily blocked relevant brain activity by applying targeted transcranial magnetic stimulation (TMS) at the occipital face area (rOFA) and right somatosensory cortex while participants discriminated facial expressions. TMS impaired discrimination of facial expressions at both sites, but had no effect on a matched face identity task. Further, recognition accuracy dropped most when pulses were delivered at

100–140 ms and 130–170 ms to the right somatosensory cortex. Pulses at earlier times (20–100 ms) and later times (170–290 ms) were ineffective. These findings suggest not only that the right somatosensory cortex supports expression recognition, but also that it contributes relatively early, though not immediately, to the recognition process.

Individuals with Autism Spectrum Disorder. The just-reviewed literature on typical individuals highlights that, in addition to the more standard "disembodied" mechanisms, emotion perception is supported by embodied mechanisms that draw on peripheral and central resources. But, what is the role of these mechanisms in atypical functioning? As mentioned earlier, several accounts postulate that embodied stimulation mechanisms might be dysfunctional in ASD. As such, examination of these atypical individuals offers insights not only into possible etiology of ASD, but also into typical emotion functioning. Below, we review behavioral and physiological findings that point towards impaired or atypical recruitment of embodied processes in ASD. We first show evidence for impairments of rapid emotion processing and for the use of "cold" (rule-based) rather than "hot" (embodied) strategies for emotion perception. We next discuss involvement of peripheral processes in emotional perception, specifically rapid mimicry. The data suggest that in ASD embodiment processes are less spontaneous, and their engagement requires additional time and motivation. Next, we review data indicating atypical patterns of activation of the central mechanisms of embodiment in ASD. Finally, we present a study suggesting that people with ASD show a dissociation of subjective and motivational responding to emotional stimuli, which is consistent with findings showing that embodiment is not an automatic process in this population.

Behavioral Findings in Individuals with Autism Spectrum Disorder. There is a very rich literature on how individuals with ASD perceive emotional stimuli, especially emotional facial expressions (Humphreys, Minshew, Lee Leonard, & Behrmann, 2007). Many researchers have reported atypicalities and impairments. Thus, individuals with ASD are less likely than controls to categorize faces on the basis of emotional expression, instead preferring to sort them according to characteristics such as articles of clothing (Weeks & Hobson, 1987). Individuals with ASD are also less accurate on unimodal matching (faces to faces) and cross modal matching (voices to faces) of emotional stimuli (Hobson, Ousten, & Lee, 1988). Further, ASD individuals are less likely, and less able, than controls to match faces on the basis of emotion, even though they are equally able to match faces on the basis of identity (Celani, Battacchi, & Arcidiacono, 1999). However, in many tasks requiring recognition of clearly presented facial expression, individuals with ASD do well (Humphreys et al., 2007). This raises the question of what exactly are the conditions and the nature of the impairment. One clue to this comes from recent studies in our labs, which suggest that (a) ASD impairments are limited to challenging recognition situations, and (b) when ASD participants are successful at recognition, they achieve it using alternative, rule-based emotion recognition strategies.

Clark, Winkielman, & McIntosh (2008) compared ASD and control individuals on extraction of emotional and non-emotional information from stimuli presented briefly or for a long time. In the brief condition, participants were shown images for durations in the range of microexpressions (15 and 30 ms), thus reducing the reliance on higher-level cognitive skills. Participants detected if (a) emotional faces were happy or angry, (b) neutral faces were male or female, and (c) neutral images were animals or objects. ASD individuals performed selectively worse on emotion extraction from faces (60% vs. about 75% for control groups). There were no group differences on gender or animal-object tasks, with groups all performing around 65%–70%. Importantly, there were no group differences in accuracy, which was perfect (100%) on any type of stimuli when pictures were presented at long stimulus duration (3 seconds). This finding emphasizes again that participants with ASD can successfully perform emotion detection under favorable presentation conditions.

But how do individuals with ASD succeed under long conditions? To answer this question, Rutherford and McIntosh (2007) examined strategies used for perception of emotion from faces. The prediction was that individuals with ASD would rely on specific facial features in a rule-based strategy, rather than a holistic, template-based strategy. Participants were tested in a forced-choice paradigm on six caricatured emotion expressions with a goal of always choosing "which of the two images looks like a real person would look if they were feeling this way." The expressions varied in the level of the caricature, with the extreme expressions being greatly exaggerated (e.g., sadness with lips curled down to a biologically unrealistic degree). Still, for five of six emotions, individuals with ASD were more likely than controls to accept the most exaggerated images as most realistic, presumably because those expressions represented "best fits" to the rule. In short, these results suggest that people with ASD relied more heavily on a "cold" rule-based strategy than a more typical template-based strategy in perceiving emotional facial expressions. Applying rules is more consistent with a symbolic, disembodied approach to emotion perception than one that posits internal simulation of real-life, biologically-constrained expressions.[5]

In sum, the behavioral studies on emotion perception suggest that individuals with ASD are impaired on tasks requiring fast emotional recognition. They can do well on emotional recognition tasks with longer presentations, but appear to perform these tasks using more descriptive, rule-based mechanisms. Both these results raise the possibility that ASD individuals do not embody emotion to the same extent as typical individuals. This issue was addressed by examining both peripheral and central mechanisms.

Peripheral Mechanisms in Individuals with Autism Spectrum Disorder. Do ASD individuals engage peripheral resources during emotion perception? This question was addressed in several studies on facial mimicry of emotional expression. One of the first studies compared spontaneous (unprompted) and voluntary (instructed) imitation of emotional facial expressions in ASD and matched controls (McIntosh,

Reichmann-Decker, Winkielman, & Wilbarger, 2006). In this study, participants viewed large pictures of happy and angry facial expressions presented for several seconds. In one condition, participants were simply asked to "watch the pictures as they appear on the screen." In another condition, participants were asked to "make an expression just like this one." Mimicry was measured by EMG, with electrodes placed over the cheek (smiling) and brow (frowning) regions. In the voluntary condition, there were no group differences, with ASD participants showing a normal pattern of fast voluntary mimicry (smile to a smile, frown to a frown). However, in the spontaneous condition, only typical participants mimicked, with ASD individuals showing no differential responses.

To determine whether there are ASD deficits in mimicry earlier in development, Beall, Moody, McIntosh, Hepburn, and Reed (2008) used a similar psychophysiological paradigm with children (aged 8–13 years) with ASD. The children with ASD did not show facial EMG responses to happy or angry faces, and showed undifferentiated responses to fear faces. However, typically developing children of the same age showed rapid mimicry of happy faces and fear responses to angry faces.

A similar result was obtained in a recent behavioral study by Stel, van den Heuvel, and Smeets (2008) using dynamic social stimuli. Here participants (adolescents) were asked to simply watch a five minute video in which a male student talked about his adventures in an amusement park, displaying happy expressions and gestures. Afterwards, the experimenter coded participants' facial expressions and gestures for a match between the model and a participant (mimicry was defined as a similar expression occurring within ten seconds). Data analysis revealed that adolescents with autism showed less spontaneous mimicry, as compared to PDD-NOS and control participants. Additionally, there was a significant correlation for controls between mimicked facial expressions and experienced positive emotions while the same comparison was not significant for the ASD group. Finally, as in McIntosh and colleagues' (2006) study which used static pictures and EMG, the group differences in Stel et al. (2008) were observed despite no differences in the amount of time spent looking at the screen and no differences in voluntary mimicry. This reduces that likelihood that spontaneous mimicry impairments are due to purely perceptual or attentional factors.

The studies discussed above show that individuals with ASD are impaired on spontaneous mimicry during emotion perception, suggesting a deficit in the automatic engagement of embodiment mechanisms. However, this does not mean that ASD individuals principally lack such mechanisms. Note that Beall et al., (2008), McIntosh et al. (2006), and Stel et al. (2008) examined a situation in which recognition of emotion is not the primary task—participants are simply asked to watch faces on the screen, without the experimenter putting any emphasis on recognizing the expressions. Thus, these studies say more about the conditions of engagement, rather than the absence of mechanisms. Consistent with this possibility, some recent studies suggest that people with ASD do show spontaneous mimicry when processing of emotion-relevant aspects of the stimuli is explicitly encouraged by the task. Oberman, Winkielman, & Ramachandran (in press) investigated timing and magnitude of spontaneous and voluntary mimicry in ASD children and matched controls using facial EMG. However, instead of "just watching" faces in the spontaneous phase, participants were asked to classify them into happy, sad, fear, anger, disgust, and neutral expressions. Later, just as in McIntosh et al. (2006) participants were specifically instructed to mimic the expressions. There were no group differences on emotion recognition and amplitude of expression-appropriate EMG activity. However, ASD participants' spontaneous, but not voluntary, mimicry activity was delayed about 160 ms. This delay occurred across different expressions, suggesting a non-specific impairment in the timing of spontaneous mimicry.

A similar finding that requiring emotion processing facilitates spontaneous mimicry in ASD was reported by Magnee, de Gelder, van Engeland, and Kemner (2007). In this study, ASD and control participants performed a task that required integration of visual and auditory cues to a person's emotional state. Specifically, participants saw happy or fearful faces, presented for 900 ms before the onset of emotion auditory cues (happy or fearful voice). These faces, which participants judged on gender, were paired with either congruent or incongruent voice, thus encouraging attention to and processing of the emotional dimension of both the face and voice stimuli. Under these task conditions, the results showed comparable amplitude of emotion-congruent facial EMG responses between typical and ASD participants. Unfortunately, because these authors did not look at timing of facial mimicry, it is difficult to compare this study with reports of mimicry delays by Oberman et al. (in press).

In sum, the literature on the role of peripheral involvement in emotion perception suggests that under conditions of spontaneous observations ASD individuals do not embody the expression, unless the task directly calls for emotion discrimination. Further, even when they do embody, the peripheral reactions are delayed. The functional importance of this delay is speculatively suggested by the ASD impairments in fast emotion recognition reported by Clark and colleagues (2008). The reason for the delay is not known, and there are several interesting possibilities. It may be that the voluntary engagement of embodiment takes longer than the typical spontaneous use of embodiment. Alternatively, it may be that the delay is related to the use of atypical cognitive or affective processes in emotion perception. Without the benefit of fast, spontaneous embodiment, participants may first need to engage a slower compensatory rule-based strategy to initially recognize the expression (Rutherford & McIntosh, 2007). This then results in delayed motor matching of the expression as the recognition process is being completed. Or, the delayed matching expression may be the result of a slower affective contagion process, reflecting an induction of a similar emotional experience, rather than an automatic aid in the initial perception of the emotion (see Moody, McIntosh, Mann, & Weisser, 2007).

Because no studies have yet directly linked differences in mimicry with emotion recognition or strategy, we cannot know which of these or other possibilities are more plausible. The EMG mimicry studies we described went to great pains to

ensure that emotional expressions themselves were clear and easily distinguishable, ensuring 100% recognition performance (McIntosh et al., 2006, Oberman, in press). This was done to ensure that any impairment in EMG mimicking response was due to the output processes, rather than basic perception. Further, as mentioned, Clark and colleagues (2008) found that autistic impairments in emotions recognition are noticeable under quite short (30 ms) conditions, but not under typically long presentation conditions—a finding consistent with much research in autism literature (Humphreys et al., 2007). So, we need studies that simultaneously measure emotion recognition and embodiment with EMG under difficult processing conditions and relate the recognition accuracy to the degree of embodiment. As far as we know, such studies do not yet exist, though they are being planned in our labs.

Central Mechanisms in Individuals with Autism Spectrum Disorder. Only recently have researchers begun to investigate the role of central mechanisms of embodiment in ASD individuals. Further, these investigations have so far focused on only some elements of relevant circuitry, especially the "mirror neuron system," as we discuss next. For discussion of the role of somatosensory cortices and the amygdala in autism, see Adolphs (2006).

Several authors have proposed that ASD individuals have dysfunctional mirror neuron system (Iacoboni & Dapretto, 2006; Oberman & Ramachandran, 2007; Williams et al., 2004). This impairment might affect their ability to spontaneously map the mental representation of the self to the representation of the other, spontaneously grasp the meaning of actions, and generally impair the ability to perform "embodied simulation." Behavioral and physiological evidence consistent with this proposal has been obtained by several research groups using a variety of techniques.[6] Much of this evidence came from studies of simple, non-emotional motor tasks, but, as we review shortly, recently this work has also been extended to emotion.

Nishitani, Avikainen and Hari (2004) showed ASD and control participants pictures of a woman performing orofacial gestures and asked them to imitate these gestures. Cortical activations were recorded using magnetoencephalography (MEG), an electrophysiological technique that offers good temporal resolution. Compared to controls, the ASD group showed weaker activations in the inferior frontal lobe and primary motor cortex, suggesting reduced mirror neuron activity. Focusing on spontaneous imitation, Oberman, Hubbard, McCleery, Ramachandran, and Pineda (2005) asked typical and ASD individuals to simply view videos of a person executing simple actions, or to perform the same actions. During these tasks, the experimenters recorded mu wave suppression, an electroencephalogram (EEG) index of activity in the primary motor cortex, and proposed it to be indicative of activity in the premotor "mirror neuron area" during the observation of action. The typically developing individuals showed mu wave suppression to both the execution and observation of action. However, individuals with ASD showed mu wave suppression when performing their own actual movement but not when observing movement (i.e., reduced mirror neuron activity).

Consistent with social psychological literature on the role of self–other overlap in contagion-like phenomena, there is evidence that ASD impairments in spontaneous mirroring might relate to a deficit in mapping the representation of the observed action to the self. Theoret and colleagues (2005) asked typical and ASD groups to view videos of index finger and thumb movements that were directed either toward or away from the participants. During these tasks, the experimenters recorded motor evoked potentials (MEP) induced by TMS. In the typical group, both participant-directed and other-directed actions increased MEPs recorded from the participant's muscles, suggesting spontaneous mirroring. However, the ASD group showed increased MEPs (spontaneous mirroring) when viewing actions directed toward the participant, but not when viewing actions directed away from the participant. This suggests that ASD participants' mirroring failures might be due to a reduction in self–other mapping. Consistent with these results, Oberman, Ramachandran, and Pineda (2008) found that children with ASD showed a typical degree of mu suppression (an EEG index of mirroring activity) in response to an action performed by a family member, or the participant himself, but not to the same action performed by a stranger.

In this context, it is useful to mention some behavioral studies which suggest no ASD impairments in representation of another's action. For example, Sebanz, Knoblich, Stumpf, and Prinz (2005) used a spatial compatibility reaction time (RT) task, which is performed by the participant alone, or with a co-actor sitting nearby. The co-actor is instructed to respond to a different stimulus dimension. The results showed similar RT interference effects from learning about the co-actor's incompatible actions for the typical and ASD group. This suggests that both groups mentally represented the co-actor's task. Sebanz and colleagues interpret this finding as evidence for an intact system matching observed actions onto representations of one's own actions in ASD. However, an alternative interpretation is that both groups were simply able to remember the instructions for the co-actor's task, and this memory is what created the interference. Further, because the Sebanz et al. (2005) study did not include process measures (e.g., neural measures of MNS activity) it is impossible to determine whether the similar behavioral performance in typical and ASD groups was caused by the same processes (as discussed earlier, compensatory processes can produce similar performance). To take a broader perspective, we think it is generally more useful to treat the question of self–other mapping in ASD not as categorical "presence or absence," but as a question of task conditions under which ASD individuals engage in such mapping. Thus, it is possible that a set-up used by Sebanz and colleagues, which requires joint task performance with a familiar confederate sitting right next to the participant, may prompt ASD individuals sufficiently to represent others' goals, something they may not do spontaneously with a distant, passive stranger. This perspective is consistent with research which found "mirror system" activation to familiar people, but not strangers (Oberman et al., 2008). It is also consistent with research which found spontaneous facial mimicry under tasks conditions requiring emotion discrimination (Oberman et al., in press), but not under "just watch" condition (McIntosh et al., 2006).

The majority of studies on central "mirroring circuits" focused on understanding of non-emotional actions. Interestingly, despite the abundance of behavioral and peripheral work on emotional imitation, there is not much parallel neural evidence concerning the involvement of the central mirroring circuits. However, one fMRI study investigated the role of mirror neurons in the imitation of facial expressions in ASD and controls (Dapretto et al., 2005). Participants were asked to both actively imitate and passively observe emotional facial expressions. During active imitation, ASD participants showed activation differences in a wide variety of regions, including visual cortices, primary motor, limbic, and cerebellum. However, during both the active imitation, and the passive observation task, there was lower activation in the presumed "mirror neuron region" (inferior frontal gyrus). In addition, the authors report an impressive negative correlation of the activity in a selective region of the mirror neuron area with the severity of autism symptoms, measured by the Autism Diagnostic Interview ($r = -.85$) and Autism Diagnostic Observation Schedule ($r = -.70$). Of course, there are general methodological reasons to worry about reports of extremely high brain-behavior correlations, especially between noisy behavioral measures and small selected neural regions. Still, the findings of Dapretto and colleagues at least hint that ASD emotional processing deficits could be partly due to atypicalities in central mechanisms of simulation. Unfortunately, there are no studies that examine the engagement of the central mechanisms, such as the MNS, in ASD participants during an active task requiring emotion recognition, rather than just passive perception of emotional faces (e.g., Oberman et al., in press). Parallel to the peripheral studies using EMG, and the central studies with non-emotional tasks, we would expect to see an enhancement of MNS activation even in ASD participants, though perhaps this activation would be delayed and have lower intensity, especially if the tasks concerned the emotions of strangers.

In sum, just as with peripheral mechanisms, participants with ASD show atypical pattern of activation of the central mechanisms of embodiment, such as the MNS. Importantly, the exact conditions of recruitment of this mechanism are again complex, with task variables—such as engagement, familiarity, and interest—playing an important role. Further, it is not yet clear which central "embodied" resources (MNS or body representations) are most atypical, or if the engagement of these resources reflects some other variables. Finally, as with peripheral studies, we know little about the relation of the central embodiment to emotion recognition accuracy. Thus, there is much room for investigation by future studies.

Emotional Reactions and Understanding

Clearly, the use of embodiment in emotion processing goes far beyond the perception of specific, simple, and common emotional stimuli, such as facial expressions. It is possible that the notion of embodied simulation is most useful when applied to more advanced processes of emotional responsiveness and understanding. Indeed, the utility of embodied social perception may be most evident in complex social situations, during which active on-line evaluation of another's emotions and intensions is required (see Reed & McIntosh, 2008). Social psychologists have long argued that "putting oneself in someone else's shoes" can facilitate empathy and understanding (for review, see Batson, 1991). There is now evidence that this process might be supported by embodied simulation, at least in typical individuals (Decety & Jackson, 2004).

Typical Individuals. One interesting case where embodiment can facilitate understanding is the case of "feeling someone's pain." One study assessed activity in areas related to the experience of pain with single cell recording and found the activation of pain-related neurons when a painful stimulus was applied to the participant's own hand, and also when the patient watched the painful stimulus applied to the experimenter's hand (Hutchison, Davis, Lozano, Tasker, & Dostrovsky, 1999). This finding was extended by a fMRI study that revealed overlapping changes in pain-related brain regions (anterior cingulate and insula) of female participants while painful stimulation was applied to their own hand and to their partners' hand (Singer et al., 2004). Further, the study showed that the change in relevant brain activations depended on the participants' level of empathy, suggesting the role of motivation to simulate. Indeed, this interpretation is consistent with recent studies from the same laboratory, which found an increase in activation of pain-related regions to the observation of a confederate receiving a painful stimulus, but only if the confederate had played fairly in a previous economic game (Singer et al., 2004). This finding is important as it highlights the goal- and context-dependent nature of simulation—a notion emphasized by modern, more process-oriented embodiment theories. That is, the embodied responses to another person are not simply automatic but are situated in a particular context that reflect the relationship with the person, or shared group memberships, and require active engagement of the perceiver in the process of constructing a simulation (see also Markman & Brendl, 2005).

The idea that social perceivers can use embodiment conditionally, depending on their needs and motivation, has also garnered support in a recent study by Niedenthal, Winkielman, Modillon, and Vermeulen (in press). This study evaluated the role of embodied simulation in higher-order emotion understanding by manipulating the possible strategies that participants can use to perform the very same task—the listing of emotional concept properties (e.g., features of "anger"). Specifically, some participants were implicitly encouraged to use an embodied simulation strategy whereas others were encouraged to use a more lexical strategy. Participants in the "embodied" condition were told to think about the potential audience of their concept listings as being interested in "hot" features of emotional concepts (e.g., an artistic friend). In contrast, participants in the "lexical" condition were told to think about the potential audience of their concept lists as being interested in more "cold" features (e.g., a technical friend). While participants performed the task, we assessed the emergence of embodied reactions using EMG.[7] The behavioral results of this study showed that participants successfully performed the concept listing task in both conditions, as evidenced by the number

and valence of properties generated for each emotion concept. However, those participants who considered the "hot" audience engaged in more facial activity in general and embodied positive emotions (i.e., smiled) when generating positively-valenced properties of concepts, as compared to individuals who considered the "cold" audience. Taken together, this demonstration supports a situated cognition view in which the current (social or other) context influences the way in which a concept is represented in a conceptual task, and the extent that people recruit embodied information to solve it.

Finally, some more ecologically suitable evidence for how social context influences the engagement of embodiment comes from McIntosh (2006). Among participants watching a live model's responses to emotion-eliciting videos, those who liked the participant showed stronger mimicry of smiles. This was true both when liking was experimentally manipulated, and when mimicry of pairs of friends was compared to pairs of strangers.

Individuals with ASD. The literature on ASD has looked at emotion responsivity and understanding in many different ways, starting with basic reactions to emotional scenes to complex reactions to emotional situations. The work described earlier suggests atypical engagement of embodiment among people with ASD. To summarize, in the studies described earlier, the mimicry shown by typically developing children and adults was not evident in those with ASD, unless the task explicitly requested emotion discrimination—and even then the matching was delayed. If ASD involves a disruption of the embodied components of the emotional processes, then one consequence may be that "hot" low-level representations supporting bodily reactions would be decoupled from the "cold" verbally reported emotional reaction. One study from our lab examined a possible decoupling using the phenomenon of affective startle modulation (Wilbarger, McIntosh, & Winkielman, in press). In humans and animals, affectively negative stimuli have been shown to prime an aversive motivational response, increasing the startle response to a sudden loud noise, whereas positive stimuli prime an appetitive motivational response, decreasing the startle response (Lang, 1995). Physiologically, this modulation involves potentiation or suppression of the connections between basic sensory and motor process by the amygdala (Davis, 1997). Thus, monitoring startle can provide information about the nature of basic motivational response to valenced stimuli. In our study, individuals with ASD and controls viewed pictures of positive and negative emotional scenes (from the IAPS set) while their eye blink startle responses were assessed. In addition, we collected self-reports of picture valence. As predicted, ASD individuals did not differ from controls in self-reports of valence. However, they differed in their physiological responses. Specifically, ASD individuals showed startle potentiation to both negatively and *positively* valenced stimuli. This finding suggests that the low-level automatic physiological response in ASD individuals can differ from the more explicit subjective response, with individuals verbally reporting positive stimuli as positive, but physiologically showing "avoidant" motivational responding. Overall, these findings are consistent with the idea that ASD participants have multiple pathways to emotion responding and their impairments might be particularly pronounced in the basic "embodied," rather than "disembodied" components.

Summary, Conclusions and Open Issues

In this article, we argued that theories of embodied cognition offer a fruitful approach to the processing of emotional information in both typical and atypical individuals, such as individuals with ASD. We started by highlighting the limitations of symbolic models that focus solely on amodal operations. As an alternative account, we proposed that emotion processing is grounded in modality-specific systems, in which perceptual and conceptual operations involve the partial reactivations and recreations (simulation) of the actual emotional response. We then reviewed evidence supporting the role of peripheral and central "embodiments" in typical and atypical emotion perception and emotion understanding and responsiveness. Thus, we pointed out the role of peripheral facial reactions and central engagement of somatosensory and premotor cortices in emotion recognition. We also pointed out the involvement of embodied responses in understanding the emotional state of others, as well as generating proper motivational responses. Throughout we highlighted a causal, rather than correlational, role of embodiment in emotion processing, as evidenced by the effects of peripheral and central activation and deactivation manipulations and physical lesions on embodied mechanisms.

The main message of our review is that scientific understanding of emotion in ASD can benefit from embodiment theories, just as embodiment theories of emotion can benefit from research on ASD. Let us highlight again some of those benefits, though the relative scarcity of the relevant data, as well as the newness of the theoretical approach, make the conclusions necessarily speculative.

Regarding the benefits of embodiment theories for understanding ASD, it is worth highlighting the value of the framework for generating predictions about conditions in which ASD individuals should demonstrate strengths and weaknesses in emotion processing. Specifically, we predicted and found that ASD-related atypicalities of emotion processing manifest themselves in situations where the task (a) spontaneously recruits basic bodily responses, (b) requires embodied simulation, and (c) cannot be performed with the same efficacy using alternative disembodied strategies or pathways. Importantly, the reviewed studies also suggest that the ability to engage embodied processes (e.g., peripheral and central bodily feedback, mimicry, action mirroring) *strategically* or under conditions of high task relevance (which ASD individuals clearly have) may be distinguishable from the ability to *spontaneously* engage these processes in fleeting, everyday situations.

Our understanding of the role of embodiment in emotion processing has also benefited from research on ASD. Most generally, the strengths that ASD individuals show in many emotion tasks make evident that people have multiple strategies

available, including "cold" rule-based strategies that allow for successful processing. Further, the conditional engagement of embodied resources by ASD individuals highlights that embodied simulation is a dynamic, goal-dependent process.

Of course, open issues remain. As we have indicated throughout the article, we need more research that directly ties embodiment to emotion recognition and emotional understanding in ASD. This is especially true in research on more complex forms of emotional understanding, rather than simple emotional perception. Accordingly, some of the proposed relations remain necessarily speculative. This links to a more general theoretical challenge for the embodiment account of emotion. It is still not clear what role embodiment plays in the representation and processing of abstract emotional information. For example, do typical and atypical individuals differ in their understanding of complex emotional concepts and states, like resentment, schadenfreude, or gratitude? How do they understand the subtle differences between similar emotions, such as shame, embarrassment, and guilt? Note that such understanding certainly involves the ability to simulate a relevant experience. But it also requires the ability to connect the simulation to a more abstract knowledge about respective eliciting conditions and implication. For example, proper understanding of these emotions includes recognition that shame and guilt, but not necessarily embarrassment, involve some form of norm violation, and that guilt, but not necessarily shame or embarrassment, implies recognition of responsibility. And, what are the consequences of this different understanding? After all, a more "embodied" understanding of shame may invite different inferences and lead to different judgments and feelings, including a different sense of one's self.

Another important issue is the role of specific processes underlying embodiment. For example, in spontaneous mimicry, are the deficits related to processes of mapping between self and the other? If so, at what level? Or, do deficits occur even when such self–other overlap is successfully created and reflect the failure of mechanisms responsible for generating matching responses? Related to these questions is the respective role of specific modalities. Note that research with typical individuals demonstrated embodiment effects in a variety of modalities, including the motor, visual, auditory, gustatory, and affective systems. Thus, one wonders whether all kinds of embodiments are equally affected in ASD. Perhaps the deficits are quite general and involve a variety of simulations across modalities? But perhaps they are more restricted to the core motor control circuits, as suggested by findings that ASD individual have atypical cerebellum (Palmen, van Engeland, Hof, & Schmitz, 2004)? This could be interestingly related to the proposals that treat the cerebellum as an "emulator"—a device that mimics and predicts the outcomes of motor actions (Grush, 2004). For emotion researchers, the crucial question concerns modalities supporting basic affective processes, such as the limbic system, including the amygdale; the introceptive system, including the insula; and central and peripheral circuits representing the body. Impairments in any of these systems could have consequences for attention, reactions and processing of emotional stimuli.

Though challenges remain, it is clear that the embodiment approach has inspired, and is continuing to generate, research that advances the understanding of emotional perception, reaction, and understanding in typical and atypical individuals. We hope this review has captured some of this excitement and pointed out some useful directions for future research.

Notes

1 When the simulation process results in an actual response in the individual that closely matches that of the observed stimulus, we will refer to this response as "mirroring."

2 Cannon was certainly correct that cutting feedback from the viscera, via the spinal cord and the vagus nerve, does not eliminate many forms of emotion, such as rage or fear. However, he did not consider the role of other peripheral pathways, such as the endocrine system, and their critical effects on central neurotransmitters, such as dopamine, norepinephrine, or serotonin (Craig, 2002; Damasio, 1994).

3 The assumption here is that core limbic circuits generate analogue, perception-like modal inputs. For example, ventral striatum can generate "raw" feelings of excitement or pleasure via natural rewards (e.g., sex), symbolic rewards (e.g., money), or via dopaminergic drugs (e.g., Knutson, Wimmer, Kuhnen, & Winkielman, 2008).

4 The original work in monkeys emphasized a unique role of neurons located in the inferior parietal and inferior frontal cortex, which discharge both when a monkey performs an action and when it observes another individual's action. Some scientists argue that humans have a dedicated "mirror neuron area" in the premotor cortex (Broadmann area 44—human homologue of the monkey F5 region). This mirror area may compute complex operations such as mapping the correspondence between self and other, or differentiating between goal-oriented versus non-intentional action (Gallese, 2003).

5 It is also possible that the exaggerated expressions made it easier for the individuals with ASD to process and integrate the relevant pieces of information. This interpretation would fit the central coherence theory of autism (Frith, 2003).

6 There are some reports of anatomical differences in the mirror neuron system. For example, Hadjikhani, Joseph, Snyder, and Tager-Flusberg (2006) reported local decreases of gray matter in the MNS area among ASD individuals, and a correlation of this cortical thinning with severity of ASD symptoms. Similarly, Villalobos, Mizuno, Dahl, Kemmotsu, and Muller (2005) reported reduced functional connectivity between the primary visual cortex and area 44, the prefrontal mirror neuron area in individuals with ASD.

7 Similar logic for testing the conditional nature of simulation has been used by other researchers. For example, in studies by Barsalou and his colleagues, some participants perform a feature generation task via listing of words associated to the concept label (lexical condition), whereas other participants were asked to perform this task via generation of typical features (simulation condition). The results show that fewer modality-specific properties are generated in the lexical, rather than simulation condition (for review see Barsalou, 2008).

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