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From emotion resonance to empathic understanding: A social developmental neuroscience account

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Abstract

The psychological construct of empathy refers to an intersubjective induction process by which positive and negative emotions are shared, without losing sight of whose feelings belong to whom. Empathy can lead to personal distress or to empathic concern (sympathy). The goal of this paper is to address the underlying cognitive processes and their neural underpinnings that constitute empathy within a developmental neuroscience perspective. In addition, we focus on how these processes go awry in developmental disorders marked by impairments in social cognition, such as autism spectrum disorder, and conduct disorder. We argue that empathy involves both bottom-up and top-down information processing, underpinned by specific and interacting neural systems. We discuss data from developmental psychology as well as cognitive neuroscience in support of such a model, and highlight the impact of neural dysfunctions on social cognitive developmental behavior. Altogether, bridging developmental science and cognitive neuroscience helps approach a more complete understanding of social cognition. Synthesizing these two domains also contributes to a better characterization of developmental psychopathologies that impacts the development of effective treatment strategies.

While enjoying a walk in the park with your son, you suddenly notice a young woman with a sad expression on her face who is sitting on a bench reading a letter. A wave of melancholy consumes you, and your son and you both express a wish to console the woman. This natural tendency to share and understand the emotions and feelings of others in relation to oneself, whether one actually witnesses another person's expression, perceived it from a photograph, read about it in a fictive novel, or imagined it, refers to the phenomenological experience of empathy.

Various domains of psychology suggest that one function of empathy is to promote social interaction. For example, social psychologists regard empathy as a proximate factor motivating prosocial behavior (Batson, 1991; Davis, 1994). Similarly, a large tradition in developmental science has been to study the onset and development of empathy, as some theorists suggest that empathy plays a crucial role in moral development, motivating prosocial behavior and inhibiting aggression toward others (e.g., Hoffman, 2001; Miller & Eisenberg, 1988). Indeed, empathy develops from infancy, and by 2 years of age most children manifest prosocial helping responses' to others' distress (Zahn-Waxler & Radke-Yarrow, 1990). In contrast, certain developmental disorders, such as autism spectrum disorder (ASD) and conduct disorder (CD) are marked by empathy deficits that likely influence their antisocial responses to other's distress, albeit with aloof apathy or active aggression, respectively.

The link between empathy and social interaction likely derives from the relationship between empathy and intersubjectivity. It has been

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postulated that empathy is a primary source of intersubjectivity, as the sense of shared experience is a prerequisite for understanding what drives other people's intentions, emotions, and motivations (Gallagher, 2001; Meltzoff & Decety, 2003; Trevarthen & Aitken, 2001). That is, intersubjectivity, the ability to share the subjective states of others and resonate with their perspective, strongly relies on the ability to read (in the sense of reacting and understanding) others' emotions to determine their psychological state. Indeed, the absence of intersubjectivity deprives individuals of the opportunity to develop prosocial behaviors, empathy, and moral judgments that are important byproducts of developing social cognition (Rochat & Striano, 1999).

In addition to intersubjectivity, empathy is also phenomenologically tied to psychological constructs that may be partly innate in humans, offering further evidence of the evolutionary basis of forming social bonds, and the role of empathy in this process. Indeed, some of the basic building blocks of empathy, such as emotion sharing and an ecological sense of self, seem to be present in the first days of life, suggesting a neurobiologically based predisposition for humans to be connected to others (Rochat, 2002). These processes prepare the individual for later empathic connections through affective interaction with others. Humans are indeed social animals, and virtually all of their actions are directed toward or are produced in response to others (Batson, 1990). Humans rely on others for survival and are endowed with a motivation to form and maintain strong interpersonal relationships, what Baumeister and Leary (1995) have termed "the need to belong."

Recent data from cognitive neuroscience also offer new insights regarding the neural mechanisms and brain areas that underpin empathy (Decety & Jackson, 2004, 2006; Decety & Lamm, 2006; Leiberg & Anders, 2006). The goal of this paper is to address the underlying cognitive–neural architecture that instantiates empathy and to highlight the dysfunction of these processes in developmental disorders marked by social–cognitive impairments. Based on empirical findings from cognitive neuroscience and developmental science, we argue that a number of components contribute to the experience of empathy: (a) affective sharing, a bottom-up process grounded in perception–action coupling and potentially underpinned by mirror neuron systems; (b) the ability to differentiate oneself from a perceived target, which relies on a sense of agency, self-, and other awareness, and likely involves frontoparietal and prefrontal circuits; and (c) executive functions instantiated in the prefrontal cortex (PFC), which operate as a top-down mediator, helping to regulate emotions and yield mental flexibility (Figure 1). Taken together, drawing from these multiple sources of data help paint a more complete picture of the phenomenological experience of empathy, as well as the respective mechanisms driving the phenomenon.

A Clarification of Terms

Empathy is a loaded term, with various definitions roaming the literature. Broadly construed, empathy has been defined as an affective response stemming from the understanding of another's emotional state or condition similar to what the other person is feeling or would be expected to feel in the given situation (Eisenberg, Shea, Carlo, & Knight, 1991). In line with this conception, empathy can concede an interaction between two individuals, with one experiencing and sharing the feeling of the other (Feshbach, 1997). Other theorists more narrowly define empathy as one specific set of congruent emotions, those feelings that are more other focused than self-focused (Batson, Fultz, & Schoenrade, 1987). Similarly, according to Hoffman (2000), empathy refers to the psychological processes that allow a person to experience feelings more congruent with another's situation than with his own situation.

Many developmental theories highlight the role of empathy in moral development, suggesting that when humans experience others' emotions of distress they are motivated to respond with prosocial help (Eisenberg, Spinrad, & Sadovsky, 2006). However, whether experiencing others' emotional states entails prosocial responding is unclear. During perspective-taking tasks (using cognitive means to adopt another person's point of view), social psychological research demonstrates that when individuals imagine how the other person would feel in a given situation versus how they would feel in the same situation, different emotions arise: the individuals are prone to feel sympathy for the other in

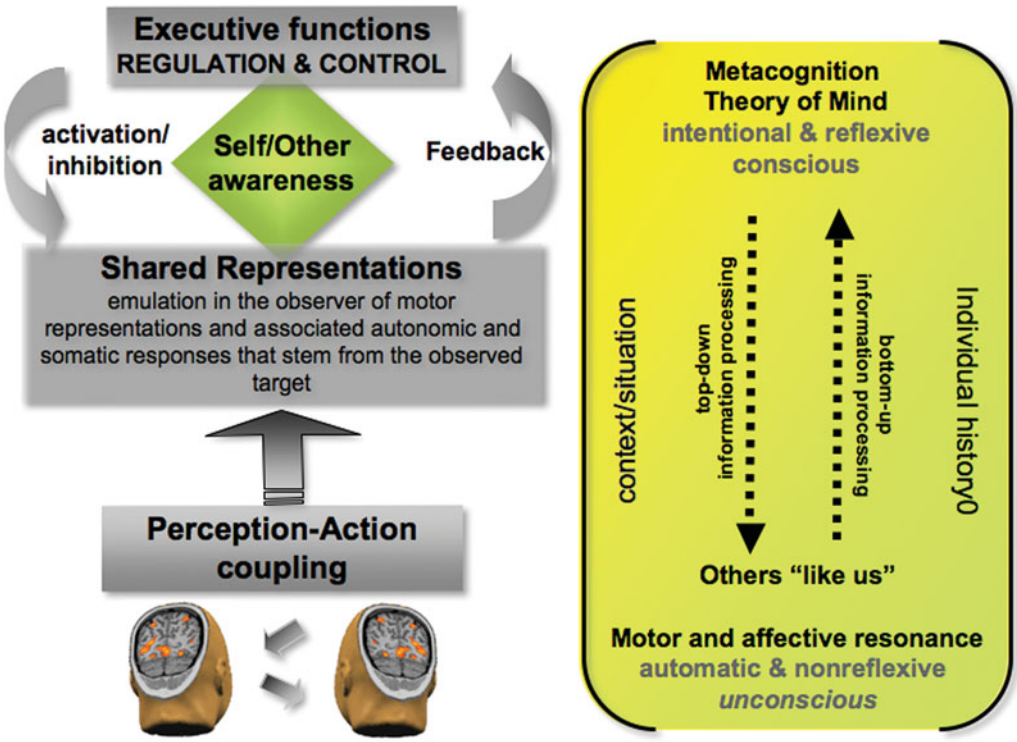

Figure 1. Schematic representation of bottom-up (i.e., direct matching between perception and action) and top-down (i.e., regulation and control) information processes involved in empathy. These two levels of processing are interrelated. The low level, which is automatically activated (unless inhibited) by perceptual input, accounts for emotion sharing. Executive functions, implemented in the prefrontal cortex, serve to regulate both cognition and emotion, notably through selective attention and self-regulation. This metalevel is continuously updated by bottom-up information, and in return controls the lower level by providing top-down input. Thus, the top-down regulation, through executive functions modulates low levels and adds flexibility, making the individual less dependent on external cues. The metacognitive feedback plays a crucial role in taking into account one's own mental competence in order to react (or not) to the affective states of others.

the former, whereas the latter can lead to personal distress, that is, a self-oriented aversive emotional response such as anxiety or discomfort (Batson, Early, & Salvarani, 1997). Personal distress may lead an observer to relieve her own stress, and not necessarily help the other. Thus, it seems that the cognitive means used to assess an automatic shared affective state with another's distress influences the likeliness to respond prosocially. In the following sections, we review the affective and cognitive components that give way to empathy, reviewing first the automatic proclivity to share emotions with others, and the cognitive process of perspective taking and executive control, which allow individuals to be aware of their intentions and feelings and keep separate self and other perspectives.

Here we will consider empathy as a kind of induction process by which emotions, both positive and negative, are automatically shared. Empathy can be the source of an emotional response emanating from the self and directed to the other, a conceptualization congruent with many scholars (see Table 1). It is important that such a definition stresses the distinction between empathic concern and personal distress, both of which spring from empathy but have different goals and consequences. Furthermore, we will consider the construct of empathy within an overarching conceptual framework. This framework suggests that empathy involves parallel and distributed processing in a number of dissociable neurocomputational mechanisms (Figure 1). Shared neural circuits, self-awareness, mental flexibility, and

Table 1. *Definitions of empathy*

- The ability to put oneself into the mental shoes of another person to understand her emotions and feelings (a form of simulation or inner imitation; Goldman, 1993)
- A complex form of psychological inference in which observation, memory, knowledge, and reasoning are combined to yield insights into the thoughts and feelings of others (Ickes, 1997)
- An affective response more appropriate to someone else's situation than to one's own (Hoffman, 1975)
- An other-oriented emotional response congruent with the other's perceived welfare (Batson, Sager, et al., 1997)
- An affective response that stems from the apprehension or comprehension of another's emotional state or condition and that is similar to what the other person is feeling or would be expected to feel in the given situation (Eisenberg, 2000)

Note: These definitions point to an emotional experience that is more congruent with another's situation than with one's own. Another important aspect of the construct of empathy is that it must involve some sort of self–other differentiation, which makes it distinct from related reactions such as emotional contagion.

emotion regulation constitute the basic macrocomponents of empathy, which are mediated by specific and interacting neural circuits, including aspects of the PFC, insula, limbic system, and frontoparietal networks. Consequently, this model assumes and predicts that dysfunction in each of these macrocomponents may lead to an alteration of the experience of empathy, and correspond with selective social cognitive disorders depending on which aspect is disrupted (Decety & Moriguchi, 2007).

In the following sections, we marshal support for this model by discussing first the behavioral evidence and then the putative neural mechanisms that underpin them. We also suggest that inadequacies in these mechanisms may help account for various social–cognitive disorders. These sections are organized according to each major aspect that contributes to empathy: emotion sharing, self- or other awareness, and executive control and emotion regulation. We conclude by speculating on the consequences of the experience of empathy in moral reasoning. We believe that combining developmental science with cognitive neuroscience can provide a more comprehensive understanding of empathy and related emotions, which also has the potential for generating new hypotheses regarding social–cognitive disorders and thus contributes to better treatment and intervention in developmental psychopathology.

Emotion Sharing

The automaticity of emotion sharing

Bodily expressions help humans and other animals communicate various types of information to members of their species. Specifically, emotional expression and perception play pivotal roles in human social interaction (Schulkin, 2005). Emotions are short-lived psychological–physiological phenomena that represent efficient modes of adaptation to changing environmental demands. It has long been suggested that emotion expression is an evolutionary adaptation that facilitates survival (Darwin, 1872). Such a claim is supported by the observation that rules govern emotional expressions, which can be elicited by simple stimuli, as in the example of disgust in the presence of bitter taste, as well as the speculation that detection of emotional expression offers clear adaptive advantages, particularly in the formation and maintenance of social relationships.

Emotional expression not only informs an individual of another's subjective (and physiological) experience but also serves as a sort of social glue maintaining emotional reciprocity among dyads and groups. Emotional contagion, defined as the tendency to automatically mimic and synchronize facial expressions, vocalizations, postures, and movements with those of another person and, consequently, converge emotionally with the other (Hatfield, Cacioppo, & Rapson, 1994) is a social phenomenon of shared emotional expression that given its automaticity occurs at a basic level outside of conscious awareness.

From infancy, complex facial motor patterns permit infants to match facial emotion expressions with others (e.g., Field, Woodson, Greenberg, & Cohen, 1982; Haviland & Lelwica, 1987). Very young infants are able to send emotional signals and to receive and detect the emotional signals sent by others. Shortly after birth, healthy infants convey facial expressions of

interest, sadness, and disgust (Field, 1989). Likewise, discrete facial expressions of emotion have been identified in newborns, including joy, interest, disgust, and distress (Izard, 1982). These findings suggest that subcomponents of full emotional expressions are present at birth (Table 2), supporting the possibility that these processes are hard wired in the brain. It has been suggested that infant arousal in response to feelings, affects, and emotions signaled by others serves as an instrument for social learning, reinforcing the significance of the social exchange, which then become associated with the infant's own emotional experience (Nielsen, 2002). Consequently, infants would come to experience emotions as shared states and learn to differentiate their own states, in part, by witnessing the resonant responses they elicit in others. This automatic emotional resonance between other and self provides the basic mechanism on which social cognition in general and empathy in particular later develops.

Infant affective resonance manifests in infant cry reactions to peer crying. One-day-old infants selectively cry in response to the vocal characteristics of another infant's cry, a finding that led to the speculation that from birth infants are endowed with an innate precursor of empathic distress (Hoffman, 1975). Moreover, infants exposed to newborn cries cry significantly more often than those exposed to silence and those exposed to a synthetic newborn cry of the same intensity (Sagi & Hoffman, 1976). The finding demonstrates that infants' auditory perception of another's aversive affective state elicits the same distressful emotional state in the self. Importantly, this reaction exists before infants develop a sense of others as physical entities distinct from the self (Hastings, Zahn-Waxler, & McShane, 2006). This convergence between the self and other's aversive affective experience reflects the instantiation of the initial building block that precedes the experience of empathy: a behavior matching response to other's emotional states.

Infants experience emotion contagion through interaction with their caretakers. Such a behavior scaffolds what Bowlby (1958) termed attachment, that is, the inclination to seek proximity to another person and feel secure in the presence of that person. Attachment theory fits neatly with evolutionary theory, which contends that kin-related altruism and reciprocal altruism (a potential result of empathy) may reflect activation of brain processes that mediate social attachments (Panksepp, 1986). By the first months of infanthood, infants recognize and mimic the distinct emotions of their mothers, a behavior that ultimately facilitates attachment (Haviland & Lelivica, 1987). This resonance or echoing of affect, feelings, and emotions that takes place in the reciprocal interaction between infants and their caretakers is a necessary element for the development of empathy and advanced social cognition (Rochat & Striano, 1999).

Table 2. *Classification of three basic categories of subjective experiences*

- Feelings correspond to the perception of private experiences such as pain, hunger, or frustration. This category of subjective experiences in general terminates following particular actions such as feeding for hunger, comfort for pain, or fulfilling a goal for frustration.
- Affects qualify the perception of a general mood or perceived private tone that exists as a background to both feelings and emotions. Affects are diffused and protracted in comparison to feelings. They fluctuate along a continuum from low to high tone.
- Emotions are the actual observable expressions of feelings and affections by invariant movement dynamics, postures, and facial display as in the expressions of pain, joy, disgust, sadness, surprise, and anger.

Note: Rochat and Striano (1999) introduced a useful classification of three basic categories of subjective experiences which are often confused in the literature, and which include feelings, affects and emotions.

The primary source of intersubjectivity impacts the quality of the infant's affective state. For example, 3-month-old infants of depressed mothers are less inclined to match emotional states with their mother than 3-month-old infants of nondepressed mothers. For instance, depressed mothers and their infants synchronize negative expression states more frequently than nondepressed mothers and their infants (Field, Healy, Goldstein, & Guthertz, 1990). This seems to indicate that under normal conditions, mothers stimulate and arouse their infant while modulating their infant's behavior. Through this process, mother and infant's emotional expression synchronize. Emotional unavailability and affective unresponsiveness may lead to the lack of such emotional

modulation among the depressed mother–infant dyad. The greater matching of negative emotion among depressed mother–infant dyad likely reflects experienced emotion contagion of negative affect. This interpretation is in line with findings related to atypical autonomic arousal in anxiously attached children, a likely aftermath attachment style of infants raised by depressed mothers. Autonomic arousal is associated with degree of facial mimicry, with anxiously depressed children showing increased arousal in response to negative facial expressions (Rogeness, Cepeda, Macedo, Fischer, & Harris, 1990).

Infants of healthy, nondepressed mothers show similar behaviors to the infants of depressed mothers in experiments using perturbation tests in which researchers disrupted the flow of contingent expression modulation between healthy mothers and infants. In the still or blank face test (Tronick & Weinberg, 1997) a mother who previously established a protoconversational flow with her infant arrests her expression, and looks to the infant without a response to the infant's behavior. Infants show several petitions for communication via smiling, vocalizing, and gesturing. When the mother continues to adopt a "still face," the infants show eye contact avoidance and distress, much like the infant's reactions to depressed mother's despondent affective state.

Results from the still face perturbation test have been replicated, however, using a double-video DTV link so that infants who are a few weeks old and mothers could communicate live (Weinberg & Tronick, 1996). After rhythmic communication stabilized, a 1-min recording of the mother's behavior was rewound and replayed. In this case, the mother's behavior was no longer contingent with the infant's movements. The infants intermittently tried to interact with the taped behavior, and showed confusion when the mother failed to respond with similar timing or appropriate expression, and eventually showed prolonged distress and avoidance. It is of interest that replay of the infant's behavior to the mother caused the mother to feel uncomfortable, and verbal reports demonstrate that she worried that the infant was unable "to connect." It should be noted that many scholars remain skeptical of such findings related to early infant intersubjectivity as measured by video dialogue between infant and mother. Attempt to replicate these results show mixed success, and furthermore, researchers of the reproduced studies interpret the infants' interactive behaviors as nothing more than social contingency outside of intersubjectivity (for a review, see Rochat, 1999).

In sum, the developmental data suggest that the mechanism subserving emotion (as the observable expressions of feelings and affects) sharing between infant and caretaker is immediately present from birth. Newborns are innately and highly attuned to other people and motivated to socially interact with others. From the earliest months of their lives, infants engage with other people and with the actions and feelings expressed through other people's bodies (Hobson, 2002; Rochat & Striano, 2002). Such a mechanism is grounded in the automatic perception–action coupling of sensorimotor information, which seems present at birth in some form. This mimicry between self and other is critical for many facets of social functioning. For instance, it facilitates attachment and provides information about the other's emotional state. Mimicry also constitutes a primary source of interpersonal engagement with others, what has been termed primary intersubjectivity (Gallagher & Meltzoff, 1996; Gallagher, 2004). This mechanism provides the foundation for understanding that others are "like me," and underlie the development of theory of mind and empathy for others (Meltzoff & Decety, 2003). In the following section, we discuss in detail the mechanism that drives emotional sharing and mimicry, the direct link between perception and action.

Perception–action coupling mechanism and the mirror neuron system

The automatic mapping between self and other is supported by considerable empirical literature in the domain of perception and action, which has been marshaled under the prominent common-coding theory. This theory claims that somewhere in the chain of operation that leads from perception to action, the system generates certain derivatives of stimulation and certain antecedents of action that are commensurate in the sense that they share the same system of representational dimensions (Prinz, 1997). The core assumption of the common coding theory is that actions are coded in terms of the perceivable effects (i.e.,

the distal perceptual events) they should generate. Performing a movement leaves behind a bidirectional association between the motor pattern it was generated by and the sensory effects that it produces. Such an association can then be used backward to retrieve a movement by anticipating its effects (Hommel, Musseler, Aschersleben, & Prinz, 2001). These perception–action codes are also accessible during action observation, and perception activates action representations to the degree that the perceived and the represented actions are similar. Such a mechanism has also been proposed to account for emotion sharing and its contribution to the experience of empathy (Decety, 2002; Decety & Jackson, 2004; Preston & de Waal, 2002). In the context of emotion processing, it is posited that perception of emotion activates in the observer the neural mechanisms that are responsible for the generation of similar emotion. It should be noted that a similar mechanism was previously proposed to account for emotion contagion. Indeed, Hatfield et al. (1994) argued that people catch the emotions of others as a result of afferent feedback generated by elementary motor mimicry of others' expressive behavior, which produces a simultaneous matching emotional experience.

Neurophysiological evidence for this perception–action coupling comes from electrophysiological recordings in monkeys in which a unique class of visuomotor neurons have been found in the ventral premotor and posterior parietal cortices. These neurons, called mirror neurons, are active during a specific motor action and the perception of the same action made by another individual (Rizzolatti, Fogassi, & Gallese, 2001). Evidence for the existence of mirror neurons in humans is more indirect, and principally relies on functional neuroimaging studies that indicate that the neural circuits involved in action execution overlap with those activated when actions are observed (Blakemore & Decety, 2001; Decety & Grèzes, 2006), as well as transcranial magnetic stimulation (TMS) and motor-evoked potentials (MEP) studies that show changes in the excitability of the observer's brain regions that encode the execution of observed actions (Fadiga & Craighero, 2004). This shared neural network for action production and observation includes the premotor cortex, the inferior frontal gyrus, the parietal lobule, the supplementary motor area, and the cerebellum. Recent neuroimaging experiments demonstrate that the mirror–neuron system is flexible, and that experience and motivation modulate its functioning. For instance, regions that belong to the mirror–neuron system showed greater hemodynamic response when hungry participants were presented with videos of people grasping food. In contrast, decreased activity was detected in these regions when participants were in a satiated state (Cheng, Meltzoff, & Decety, 2007). In addition, a number of neuroimaging studies have shown that similar brain areas, pertaining to the same network are reliably activated during imagining one's own action, imagining another's action, and imitating actions performed by a model (Decety & Chaminade, 2003; Decety & Grèzes, 2006). For instance, a similar neural network is engaged when individuals observe or imitate emotional facial expressions (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). Within this network, there is greater activity during imitation, compared with observation of emotions, in premotor areas including the inferior frontal cortex, as well as in the superior temporal cortex, insula, and amygdala. Such shared neural circuits reflect an automatic transformation of other people's behavior (actions or emotions) into the neural representation of one's own behavior, and provides a functional bridge between first and third person perspectives, culminating in empathic experience (Decety & Sommerville, 2003; Sommerville & Decety, 2006).

The perception of other people in pain has revealed to be of particular importance for the investigation of the neural mechanisms underlying empathy. Pain is a window through which one can obtain a detailed view of the cognitive and neurophysiological mechanism underlying the experiences of empathy and sympathy. The perception of pain in others thus constitutes an ecologically valid way to investigate the mechanisms underpinning the experience of empathy for two main reasons: first, most humans understand what is "pain"; it is a common and universal experience; and understands what are its physical and psychological manifestations; second, we have good knowledge about the neurophysiological pathways that are involved in processing nociceptive information that include the somatosensory cortex, the supplementary motor area

(SMA), the anterior midcingulate cortex (aMCC), the insula, the periaqueductal gray (PAG), and thalamus. Numerous functional magnetic resonance imaging (fMRI) studies have shown that when we perceive other people in pain, the neural circuits underpinning the processing of first-hand experience of pain are activated in the observer (for a meta-analysis, see Jackson, Rainville, & Decety, 2006). In one recent study, typically developing middle school-aged children were scanned while observing dynamic visual stimuli depicting other people in pain (Decety, Michalska, & Akitsuki, 2008). Results show that neural circuits subserving the processing of nociceptive information are recruited by the sight of other people in pain (Figure 2). Such a pattern of activation in children should not be surprising given the behavioral and physiological data that document that affective sharing and vicarious emotional arousal, especially in response to others distress, is hard wired and functional very early in life (e.g., Eisenberg & Eggum, 2008; Hoffman, 2000). This rudimentary capacity for resonating with the pain of others may trigger empathic distress in the observer, and provides the affective and motivational base for moral development (Hoffman, 1982).

It can be speculated that the perception–action coupling physiological mechanism is already present at birth and develops gradually through experience and exposure to actions performed by self and others (Lepage & Theoret, 2007). This will account for neonate imitation as demonstrated by the work of Meltzoff and Moore (1997). Recent empirical findings offer evidence for a mirror neuron system encoding perceived and executed human action in the child's developing brain. For instance, one study recorded electroencephalographic signals via intracranial electrodes from a 36-month-old child with epilepsy while the infant observed an experimenter either drew with his right hand or kept his right hand still (Fecteau et al., 2004). Cortical areas responding to the observation of biological movements partially overlapped with those that were active during the execution of the same movements. An electrophysiological study demonstrated that the neural response to the processing of biological motion was in place by 8 months (Hirai & Hiraki, 2005).

Mirror neuron dysfunction in ASD

Burgeoning research efforts suggest that an aberrant mirror neuron system may contribute to motor and social problems experienced in ASD. Research with humans using TMS demonstrates selective changes in the amplitude of the MEPs (M1) during action observation (e.g., Fadiga, Fogassi, Pavesi, & Rizzolati, 1995). To build on this finding, TMS was applied over the motor cortex of adults with ASD and matched healthy controls. Compared to the controls, individuals with ASD showed significantly less M1 amplitude change during the observation of transitive, meaningless finger movements (Theoret et al., 2005). In contrast, observation of the finger movements in control subjects selectively modulated the excitability of the motor cortex in areas delivering signals to the muscles concerned with the observed action. The weaker M1 modulation in individuals with ASD suggests that the less mirror neuron activation in the motor cortex may be partly responsible for a cascade of deficits in social cognition.

The fMRI experiments are in line with these TMS findings, and indicate abnormal activation of mirror neuron systems during imitation in adults with Asperger syndrome (Nishitani, Avikainen, & Hari, 2003) and reduced functional connectivity in mirror neuron system areas (Villalobos, Mizuno, Dahl, Kemmostsu, & Muller, 2004). In attempt to examine a potential link between mirror neuron dysfunction and developmental delay of social cognitive skills, one fMRI study found a lack of activation in the inferior frontal gyrus (a key mirror neuron area) in children with ASD compared to controls during the observation and imitation of basic facial emotion expression (Dapretto et al., 2006). However, this finding was recently challenged by Bastiaanen, Thioux, and Keysers (2008, April), who scanned a group of 17 adults with ASD during the observation of dynamic facial expressions, including disgust. The authors found that ASD participants activate their mirror system not less, but more strongly than controls when observing dynamic facial expressions.

Structural neuroanatomical evidence also implicates aberrations in mirror neuron systems in ASD. One morphometric study reported locally diminished gray matter in adults with high-functioning ASD in areas incorporated in the mirror

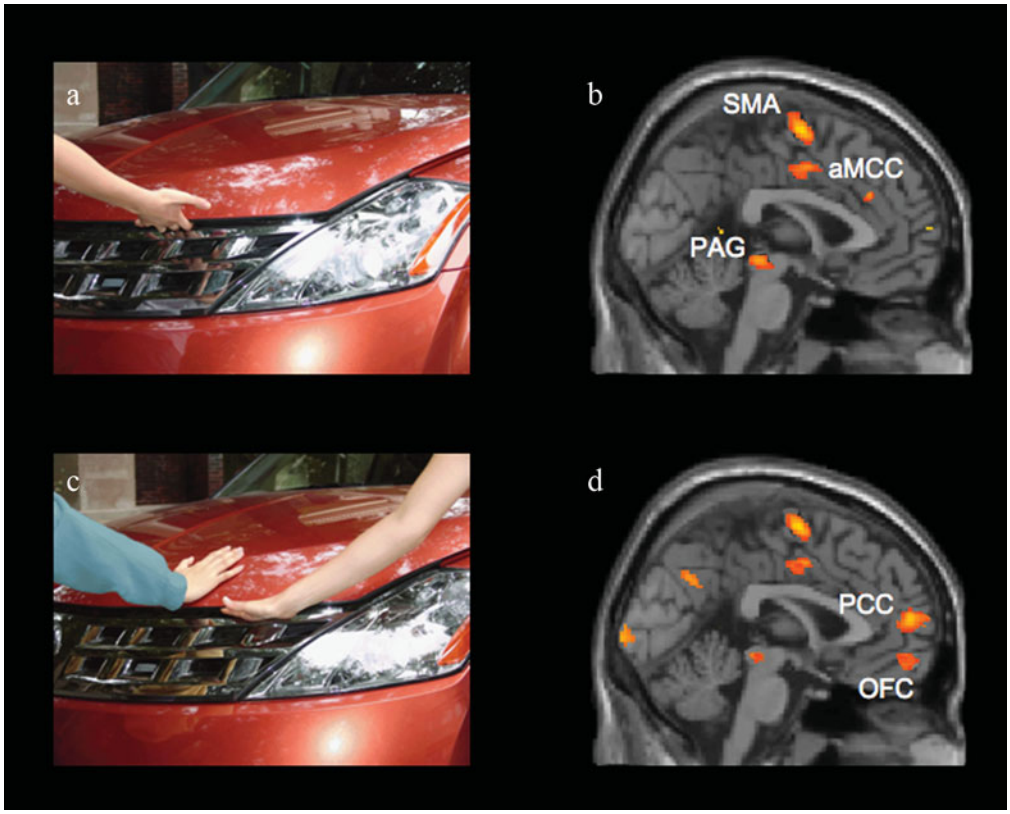

Figure 2. In this fMRI study, 17 typically developing children (9 ± 1 years) were scanned while presented with short dynamic (2.2-s duration) visual stimuli depicting painful and nonpainful situations (Decety et al., 2008). These situations involved either (a) a person whose pain was caused by accident or (c) a person whose pain was intentionally inflicted by another individual, as well as situations without any pain with one or two agents for baseline. Consistent with previous fMRI studies of pain empathy with adults, the perception of other people in pain in children was associated with increased neurohemodynamic activity in the neural circuits involved in the processing of first-hand experience of pain, including the insula, somatosensory cortex (not shown), the aMCC, PAG, and SMA. (b) It was important that when children observed an individual harming another, regions of the prefrontal cortex that are consistently involved in representing social interaction and moral behavior such as the temporoparietal junction (not shown), (d) the paracingulate cortex (PCC), and orbitofrontal cortex (OFC) were also recruited. Of interest, children indicated in the postscan debriefing that they thought that the situations in which pain was caused by another person were unfair, and they asked about the reason that could explain this behavior.

neuron system compared to controls matched for gender, age, intelligence quotient, and handedness (Hadjikhani, Joseph, Snyder, & Gager-Flushber, 2005). Cortical thinning of the mirror system was correlated with the severity of ASD symptoms (as measured scores on the Autism Diagnostic Interview—Revised), and cortical thinning was also seen in areas engaged in emotion recognition and social cognition. Thus, irregular thinning of cortical areas that implement mirror neurons and the broader network of cortical areas subserving social cognition may contribute to the emotional deficits characteristic of autism, such as problems engaging in intersubjective transactions and displays of empathic responding. It should be noted, however, that in the tables included in the paper, not only mirror neuron cortical areas, but all areas of the brain, show a significant reduction of gray matter. Thus, one needs to be cautious with these new findings, as cortical thinning may not be specific to areas where mirror neurons are located. In fact, an earlier study using magnetoecephalography failed to find any difference in motor cortex activation between individuals with ASD and healthy controls while observing action (Avikainen, Kulomaki, & Hari, 1999), a result

that is in contradiction with the study by Theoret and colleagues (2005).

Perhaps more convincing evidence of anomalies in the mirror neuron system in ASD derives from EEG studies that examine mu rhythm in sensorimotor areas. Robust evidence suggests that the magnitude of mu rhythm in sensorimotor areas is strongly suppressed during the execution and observation of an action in adults (e.g., Muthukumaraswamy, Johnson, & McNair, 2003) and typically developing children (Lepage & Theoret, 2006). In children and adults with high-functioning ASD, however, mu rhythm suppression occurs when individuals observe their own action, however it fails to suppress during the observation of other persons' action (Oberman et al., 2005), suggesting dysfunction in mirror neuron systems. Moreover, this result did not significantly correlate with age, implicating that this deficit manifests early and shows little improvement with age. Bernier, Dawson, Webb, and Murias (2007) extended these results to show that in individuals with high-functioning ASD, the degree of mu wave suppression during action observation is associated with behavioral assessments of imitation ability. That is, less mu suppression correlates with poorer imitation abilities, and is most robust for facial imitation skills.

Together, these recent findings, which need to be replicated, seem to suggest that dysfunctions of the mirror neuron system may hamper the normal development of self–other connectedness, creating a cascade of deficient processes that lead to social deficits, including empathy. However, it is worth noting that this account is still debated, and that the results from a recent fMRI study do not support a global failure of the mirror neuron system in children with autism (Hamilton, Brindley, & Frith, 2007). In the following section we consider the implications of perception–action coupling deficits in a behavior that is considered a manifestation of emotion contagion, which is facial mimicry.

Facial mimicry of emotions in children with ASD and developmental coordination disorder (DCD)

An effective means to measure the role of perception–action coupling in emotion sharing is via electromyography (EMG) recording of the activation of specific facial muscles in response to viewing other people's facial expressions. Facial mimicry has been defined narrowly as the congruent facial reactions to the emotional facial displays of others, and is thus an expressive component (Hess & Blairy, 2001). More broadly construed, emotion contagion is an affective state that matches the other's emotional display. Thus, facial mimicry can be conceived of as a physical manifestation of emotion contagion, and it occurs at an automatic level in response to viewing others' emotions (Bush, Barr, McHugo, & Lanzetta, 1989).

Individuals with ASD are often reported to lack automatic and spontaneous mimicry of facial expressions. A recent study measured adolescents and adults with ASD and controls' automatic and voluntary mimicry of emotional facial expressions via EMG recordings of the cheek and brow muscle regions while participants viewed still photographs of happy, angry, and neutral facial expressions (McIntosh, Reichmann-Decker, Winkelman, & Wilbarger, 2006). The cheek and brow muscles of individuals with ASD failed to activate in response to the videos, indicating that they did not automatically mimic the facial expressions, whereas the muscles of the normally developing controls showed activation. It is important that both groups showed evidence of successful voluntary mimicry. Difficulties in mimicking other people's emotional expression may thus prevent individuals with ASD from the afferent feedback that informs them of what others are feeling (Rogers, 1999). Indeed, in real-life situations, individuals with ASD are likely drawing on distinct cognitive processing when gauging others' emotional states (Baron-Cohen, 2002).

Other developmental disorders known for their motor deficits are less obviously tied to problems with emotion sharing and empathy. For example, DCD is characterized by delayed motor development and weak motor skills, as well as poor social skills, including deficient empathy (Gillberg, 1992). It is plausible that poor motor skills are the primary problem, because a child with motor deficits may be shunned from social inclusion. Alternatively, the child may purposefully engage him/herself in activities beyond the realm of physical activity, for example, indulge in mathematics. Thus, weak social skills may develop by default as much of social interaction relies on motor

skills, especially in childhood. However, children with DCD show information processing deficits, specifically in visual–spatial processing (Wilson & McKenzi, 1998). Such a weakness would influence perception–action coupling of emotional expression. Therefore, the mechanism underlying their difficulty in coordinating movements may also contribute to their inability empathize.

One study tried to tease out the nature of the relationship between motor skills and social skills in children with DCD (Cummins, Piek, & Dyck, 2005). In a sample of 39 children with DCD and 39 normally developing children, children with motor problems performed worse on scales that measured the capacity to recognize static and dynamic facial expressions of emotion. What is more, when visuospatial processing was controlled, this difference remained; thus, the child's motor ability was a significant predictor of social behavior. Children with DCD's motor impairments may negatively influence their ability to calibrate sensorimotor information about their own body, and thus hinder activation of shared motor responses between self and other during interactions of emotion expression.

The shared neural representations account suggests that problems with one's own motor or body schematic system may undermine capacities for understanding others. Consequently, it is possible that developmental problems involving sensory–motor processes may have an effect on the capabilities that make up "primary intersubjectivity," or the ability to react contingently to others' emotional expressions (Trevarthen & Aitken, 2001), and therefore the child's ability to resonate emotionally with others. It thus seems plausible that the defects in social and sensory–motor problems in ASD and DCD may, in part, reflect a disturbed motor representation matching system at the neuronal level. This speculation not only helps explain problems in primary intersubjectivity, but also the other sensory–motor symptoms of autism: oversensitivity to stimuli, repetitious and odd movements, and possibly, echolalia (Gallagher, 2004).

Self- and Other Awareness

Although emotion contagion provides the observer with direct information of the other's emotional state, this process only accounts for what has been termed "motor empathy," or "empathic mimicry." However, that the observation of an emotion elicits the activation of analogous motor representation in healthy observers, begs the question why there is not complete overlap between internally generated and externally engendered motor representations.

In a complete empathic experience, observers must be able to separate themselves from others and have some minimal mentalizing ability. This aspect is a landmark of mature empathic experience (Eisenberg et al., 2006; Zahn-Waxler & Radke-Yarrow, 1990). Affective sharing must be modulated and monitored by the sense of whose feelings belong to whom (Decety & Jackson, 2004). Thus, self-awareness generally and agency in particular are crucial aspects in promoting a prosocial regard for the other rather than a desire to escape aversive arousal. Phenomenologically speaking, self-awareness pertains to the embodied, and contextually embedded first-person point of view in subjective experience. In a similar vein, research in the neurosciences and developmental science use the term agency to describe the ability to recognize oneself as the agent of an action, thought, or desire, which is crucial for attributing a behavior to its proper agent.

Developmental work demonstrates that infants come into the world with an ecological sense of self, that is, the self as perceived in relation to the physical environment (Neisser, 1991). The ecologic sense of self is analogous to what phenomenologists term, prereflective self-awareness, or the subjective, qualitative "feel" of entertaining experiences (Gallagher, 2000). An implicit, ecologic sense of self develops from birth, prior to an explicit (conceptual) manifestation of self-knowledge by the second year, and this sense of self is discriminated from the sense of others (Rochat & Striano, 2000). By 2 months, infants become incrementally systematic and deliberate in the exploration of their own body and the perceptual consequences of self-produced action. For example, infants delineate between perceptual events that are self-generated or not self-generated. In one study, Rochat and Hespos (1997a) tested whether newborn infants within 24 hr of birth discriminate between double touch stimulation specifying themselves and external (one way) tactile stimulation indicating nonself

objects via the robust rooting response manifest by healthy infants from birth. Recording the frequency of the rooting in response to external or self tactile stimulation indicated that newborns are inclined to manifest rooting responses almost three times more often in response to external compared to self-stimulation. The finding suggests that from birth, infants discriminate between intermodal invariants that specify self- compared to external stimulation. Thus, infants develop an understanding of their own body as a differentiated entity, situate, and agent in the environment.

The study by Martin and Clark (1982) is also of special interest; they tested 1-day-old babies reactions to audiotapes of neonatal crying, the crying of an 11-month-old, and the newborn's own crying. They not only replicated Simmer's results that infants cry in response to other infant cries but also showed the more interesting trait that newborns did not respond to the sound of their own cries. Another investigation, conducted by Dondi, Simion, and Caltran (1999), also demonstrated that newborns are able to discriminate their own and other infants' cries. These results suggest that there is some self-other distinction already functioning from birth.

By 3 months of age infants become aware of their own body as a dynamic and organized entity with specific featural characteristics. In another series of studies, infants faced two on-line video images presented on a split screen. Infants viewed a split videotape screen showing contingent movements of the body from the waist down (Morgan & Rochat, 1997). One view showed infant's their own legs as they would be specified via direct visual proprioceptive feedback, whereas the other showed experimentally modified on-line view of their own legs. From 3 months of age, infants look significantly longer at the unfamiliar view of the legs that violates visual proprioceptive feedback. Thus, by this age infants experience an intermodal calibration of the body, developing an intermodal body schema that serves as a perceptual based "protorepresentation" of the body.

Although the above data highlights that infant representations of self and other actions are distinct, research also suggests that infants form shared representations of their own and others' actions. Neonates imitate the actions of others in a flexible and goal-directed way, suggesting that infants represent the other as "like me" (e.g., similar to the self in some respect; Meltzoff & Brooks, 2001). Further evidence suggests that infants may productively use information from their own action capacities to understand the actions of others (Woodward, Sommerville, & Guajardo, 2001). Affective sharing among infants (reviewed above) also highlights the automatic overlap between other and self in infancy, which provides the basis for the development of intersubjectivity and social cognition.

A sense of agency can also be traced to infancy. From birth, infants learn to be effective in relation to objects and events. Within hours following birth, neonates can learn to suck in certain ways and apply specific pressures on a dummy pacifier to hear their mother's voice or see their mother's face (Decasper & Fifer, 1980; Walton, Bower, & Bower, 1992). The finding suggests that infants manifest a sense of themselves as agentive in the environment. Furthermore, by 2 months of age, infants also show positive affect, such as smiling and pleasure expression, when they accomplish causing an auditory and visual event (by activating a music box by pulling a cord attached to a limb). When the cord is then furtively disconnected from the box, hindering infants' effectiveness, they switch expressions from pleasure to anger (Rochat & Striano, 2000).

In sum, the studies reviewed indicate that in addition to the early roots of perception–action coupling leading to emotional expression, a sense of self, agency, and other distinction emerge early in infancy. An ecologic sense of self develops immediately via proprioceptive calibration of sensory–motor experiences. Both this resonance mechanism and an ecological sense of self situate the individual in the social environment and account for the duality of human beings who are strongly motivated to be connected to others as well as to retain independence and autonomy. In the following section, we will highlight that primacy of the self-experience permeates throughout development, and can be seen in findings from cognitive neuroscience studies showing immediate activation of self-produced actions prior to other-produced actions. In addition, we provide neurophysiological evidence for a cerebral mechanism specifically devoted to self–other distinction.

Cognitive neuroscience of self–other awareness and agency

One role that cognitive neuroscience can contribute to the study of the self and other is to ground in physiological mechanisms the distinct dimensions, aspects, and characteristics of the self and other to help address the potential separability or relatedness of each component part of self-processing. It has been proposed that nonoverlapping parts of the neural circuit mediating shared representations (i.e., the areas that are activated for self-processing and not for other processing) generates a specific signal for each form of representation (Jeannerod, 1999). This set of signals involved in the comparison between self-generated actions and actions observed from others ultimately allow the attribution of agency (comparison between efferent motor signals and afferent sensory signals). It has also been suggested that the dynamics of neural activation with the shared cortical network is an important aspect to distinguish one's own actions from the actions of others, and that the latency difference between the changes in activity elicited by the perception of self versus others' actions reflects the calibration process of shared representations (Decety & Jackson, 2004; Jackson & Decety, 2004). Furthermore, the fact that the onset of the hemodynamic signal is earlier for the self than for the others (Jackson, Brunet, Meltzoff, & Decety, 2006; Grèzes, Frith, & Passingham, 2004) may be considered as a neural signature of the privileged and readily accessible self-perspective.

Accumulating evidence from neuroimaging studies in both healthy people and psychiatric populations, as well as lesion studies in neurological patients, indicates that the right inferior parietal cortex, at the temporoparietal junction (TPJ) with the posterior temporal cortex, plays a critical role in the distinction between self-produced actions and actions generated by others (Blakemore & Frith, 2003; Jackson & Decety, 2004). In addition, some recent data suggest that this region is specifically involved in theory of mind (Apperly, Samson, Chiavarino, & Humphreys, 2004; Saxe & Wexler, 2005). The TPJ is a heteromodal association cortex, which integrates input from the lateral and posterior thalamus, as well as visual, auditory, somesthetic, and limbic areas. It has reciprocal connections to the PFC and to the temporal lobes. Because of these anatomical characteristics, this region is a key neural locus for self-processing that is involved in multisensory body-related information processing, as well as in the processing of phenomenological and cognitive aspects of the self (Blanke & Arzy, 2005). Its lesion can produce a variety of disorders associated with body knowledge and self-awareness such as anosognosia, asomatognosia, or somatoparaphrenia (Berluchi & Aglioti, 1997). For instance, Blanke, Ortigue, Landis, and Seeck (2002) demonstrated that out-of-body experiences (i.e., the experience of dissociation of self from body) can be induced by electrical stimulation of the right TPJ. Of interest, one study found aberrant white matter adjacent to the TPJ, as well as in the ventromedial prefrontal cortices and anterior cingulate gyri (Barnea-Goraly et al., 2003) in children with ASD. Thus, deficits in self-other processing in individuals with HFA may be due to, in part, structural differences in pertinent brain areas or in abnormal connectivity between these areas.

In addition, a number of functional imaging studies point out the involvement of the right TPJ in the process of agency (i.e., the awareness of oneself as an agent who is the initiator of actions, desires, thoughts, and feelings). In one fMRI study, participants were instructed to open and close their hand slowly and continuously (0.5 Hz), whereas this movement was filmed and projected to them online onto a screen (Leube et al., 2003). The authors reported a positive correlation between the extent of temporal delay between hand movement and its visual feedback and the hemodynamic increase in the right TPJ. In another fMRI study, Farrer and Frith (2002) instructed participants to use a joystick to drive a circle along a T-shaped path. They were told that the circle would be driven either by themselves or by the experimenter. In the former case, subjects were requested to drive the circle, to be aware that they drove the circle, and thus to mentally attribute the action seen on the screen to themselves. In the latter case, they were also requested to perform the task, but they were aware that the experimenter drove action seen on the screen. The results showed that being aware of causing an action was associated with activation in the anterior insula, whereas being aware of not causing the action and attributing it to another person was

associated with activation in the right TPJ. It is interesting that individuals experiencing incorrect agency judgments, as it can be the case in schizophrenia, feel that some outside force is creating their own actions. One neuroimaging study found hyperactivity in the right TPJ when patients with schizophrenia experienced alien control during a movement selection task compared with healthy controls (Spence et al., 1997). Such delusions of control may arise due to a disconnection between frontal brain regions, where actions are initiated, and parietal regions where the current and predicted states of limbs are represented.

Another study used a device that allowed modifying the participant's degree of control of the movements of a virtual hand presented on a screen (Farrer, Franck, Georgieff, Frith, Decety, & Jeannerod, 2003). Experimental conditions varied to the degree of distortion of the visual feedback provided to the participants about their own movements. Results demonstrated a graded hemodynamic activity of the right TPJ that parallels the degree of mismatch between the executed movements and the visual reafference. Strikingly, such a pattern of neural response was not detected in schizophrenic patients who were scanned under the same procedure (Farrer et al., 2004). Instead, an aberrant relationship between the subject's degree of control of the movements and the hemodymamic activity was found in the right TPJ and no modulation in the insular cortex. Additional evidence for the contribution of the right TPJ in self-awareness and the sense of agency derives from studies on imitation that document the selective involvement of this region during reciprocal imitation, in which it may be difficult to keep track of agency, that is, who is imitating whom (Chaminade & Decety, 2002; Decety, Chaminade, Grèzes, & Meltzoff, 2002). Results from these studies provide strong support for the implication of the right TPJ in the process of self-agency by demonstrating a clear dissociation between the left and the right TPJ. When participants imitated the other, the left TPJ was strongly engaged, whereas greater activation was detected in the right TPJ when they were being imitated. Only this later condition involved discrepancies between predicted outcomes of the action performed by the participants and those perceived.

The right TPJ is also selectively activated when participants are asked to mentally simulate actions from someone else's perspective but not from their own (Ruby & Decety, 2001). Similarly, this region was specifically involved when participants imagined how another person would feel in everyday life situations that elicit social emotions (Ruby & Decety, 2004) or painful experiences (Jackson et al., 2006; Lamm, Batson, & Decety, 2007) but not when they imagined these situations for themselves. Such findings point to the similarity of the neural mechanisms that account for the correct attribution of actions, emotions, pain, and thoughts to their respective agents when one mentally simulates actions for oneself or for another individual. Further, they support a crucial role for the right TPJ, not only in mental state processing, but also in lower level processing, including reorienting attention to salient stimuli (Decety & Lamm, 2007).

Other areas implicated in self-processing, such as the medial PFC, posterior cingulate cortex, and precuneus, have been shown to be active when individuals are at rest and deactivate during cognitively demanding tasks (Raichle et al., 2001). It has been hypothesized that this "resting state" network contribtes to self-reflective thought, social perceptions, and theory of mind (Gusnard, Akbudak, Shulman, & Raichle, 2001). It is of interest that in line with the social deficits observed in ASD, individuals with ASD demonstrate atypical resting state activation in these networks (Kennedy, Redcay, & Courchesne, 2006). However, further research is needed to understand the nature of the relationship between self-experience, the resting state networks, and social behavior in typically developing children, and those with ASD.

Self, other, agency, and intersubjective exchange in ASD

A mature sense of self-, agency, and other awareness are crucial for full-blown empathy, as they allow the observer to move beyond shared representations and accurately gauge the other's emotional state in relation to oneself, in other words engage in intersubjective exchanges. According to clinical descriptions, autistic children show problems in thinking, relating, and communicating to the world as well as moving through alternative perspectives that others entertain (Hobson & Meyer, 2006), all of which we suggest draw from more than basic

level motor resonance, as they include an agentive stance over one's actions and an ability to interpret others as agentive actors as well.

Empirical evidence supports the suggestion that children with ASD fail to effectively discriminate the actions of the self and other. A recent study examined self–other orientation (identification) among 16 older children with autism, and 16 comparison, developmentally delayed children matched for age and IQ (Meyer & Hobson, 2004). Participants performed four object-oriented tasks such as rolling a wheel and stacking objects. Lines on the floor segregated the child's and experimenter's "personal space," and tasks were either directed toward the child's "personal space" or toward the adult's "personal space." After the model, children were encouraged to imitate actions with a toy. In comparison to control participants, children with autism performed significantly fewer responses that modeled the self–other orientation to the object. Instead, a significant portion of the children with ASD showed "geometric repetition." That is, they accurately imitated the experimenter's actions (i.e., rolling the wheel); however, they did so without either a "self"-orientation or an "other" norientation. For example, children with ASD would roll a wheel horizontally in the center of the testing space, disregarding the experimenters' wheel rolling either toward themselves or the child. These findings suggest that it is not motor mimicry that is problematic in autism per se, but instead, specific impairments in intersubjective imitation. Children in the control group did not show this pattern of responding, which was interesting. Instead, they fell into one of two categories of responding: either consistently adopting appropriate self- or other orientation, or showing a mix a strategies, including not imitating at all. The authors speculated that the findings reflect the failure of identification in autism, highlighting correspondence between performance on this task and the capacity to comprehend others' perspectives and to mentally shift from one perspective to another. The finding is in line with other data suggesting an autism-specific difficulty in accurately imitating the orientation of an action in relation to the model's body (e.g., Ohta, 1987; Smith & Bryson, 1998). However, future research should replicate these findings with an additional control group of typically developing children, as it is unclear whether observed differences reflect unique responding in children with ASD or unique responding in children with developmental, cognitive delay.

Further evidence of weak self–other differentiation among children with ASD derives from a study utilizing a visual perspective taking task (Lee, Hobson, & Chiat, 1994). During such a task autistic children were significantly less prone to use the pronoun "me" to answer whether they were the subject in a photograph. Moreover, autistic subjects were less likely than controls to employ the pronoun "you" to refer to the experimenter. The experimenters suggest that these results do not reflect vocabulary or semantic problems, as the children with ASD showed high verbal ability, which was measured by the British Picture Vocabulary Scale. Instead, it was suggested that failures to use pronouns reflect deficits in perspective taking. Person pronoun use is among the few trademarks signifying self-consciousness, others including self-recognition in mirrors and demonstration of self-conscious emotions such as shame (Zelazo, 2004). By age 2, executive function allows individuals to control aspects of conscious awareness, including conscious control of emotion, thought, and action. Thus, executive functions may allow individuals to regulate their egocentric bias, and engage in accurate perspective taking. In the case of ASD, problems seem to reflect not a bias in one's own perspective per se, but a weakness in adopting a conscious perspective albeit personal or other oriented. Thus, it is likely that empathy deficits in autism reflect problems at each or several of the components integral to empathy. In the following section, we discuss the crucial role of executive function in mature empathy.

Mental Flexibility and Self-Regulation

Given the sharedness of the representations of one's own emotional states and others, as well as similarities in brain circuits involved during first- and third-person perspective taking, it would seem difficult not to experience emotional distress while viewing another's distressed state and personal distress does not contribute to the empathic concern and prosocial behavior (Batson et al., 2003; Decety & Lamm, 2008). Indeed, distress in the self can hinder one's inclination to

soothe the other's distress. However, it would not be adaptive if this automatic sharing mechanism between self and other was not modulated by cognitive control and metacognition. It is necessary that executive functions work in a top-down fashion to regulate our proclivity to be biased in our self-perspective while gauging another persons' emotional state, and promoting a sympathetic regard for the other rather than a desire to escape aversive arousal (Decety, 2005). Ventral and dorsal regions of the PFC have been associated with response inhibition and self-control, which are key components of emotion regulation (i.e., adjustments in type, magnitude, and duration of emotional responses that are made to meet personal, situational and interpersonal demands; Ochsner & Gross, 2005). Support for this hypothesis in the domain of pain empathy comes from a recent fMRI study in which physicians who practice acupuncture were compared to naïve participants while observing animated visual stimuli depicting needles being inserted into different body parts including the mouth region, hands, and feet. Results indicate that the anterior insula, periaqueducal gray, and anterior cingulate cortex (ACC; i.e., neural regions that belong to the pain matrix) were significantly activated in the control group, but not in the physician group, who instead showed activation of the dorsal and ventral medial regions of the PFC, as well as the right TPJ, involved in emotion regulation and metacognition (Cheng et al., 2007).

It is of interest that the development of self and other mental state understanding is functionally linked to that of executive functions, that is, the processes that serve to monitor and control thought and actions, including self-regulation, planning, cognitive flexibility, response inhibition, and resistance to interference (Russell, 1996). There is increasingly clear evidence of a specific developmental link between the development of mentalizing (i.e., the process of making sense of mental states in oneself and other persons) and improved self-control at around the age of four (e.g., Carlson & Moses, 2001). Improvement in inhibitory control corresponds with increasing metacognitive abilities (Zelazo, Craik, & Booth, 2004), as well as with maturation of brain regions that underlie working memory and inhibitory control (Tamm, Menon, & Reiss, 2002). A series of studies by Posner and Rothbart (2000) strongly suggest that executive regulation undergoes dramatic change during the third year of life.

The PFC develops slowly compared to other regions during ontogeny, and reaches its maturation only late in adolescence (Bunge, Dudukovic, Thomason, Validya, & Gabrieli, 2002). Evidence for this delayed maturation is provided by measures of myelination, gray matter reduction, synaptogenesis, and resting metabolism (Huttenlocher & Dabholkar, 1997). Imaging studies indicate that prefrontal areas do not attain full maturations prior to adolescence (Paus et al., 1999; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). Childhood cognitive development relates to maturation of the middorsolateral frontal cortex as well as the ACC, which are critical for the development of executive functions, especially the dramatic increase in children' ability to suppress external influences (Paus et al., 1999). Specifically, changes in cerebral blood flow, which reflects synaptic activity, in the PFC, almost doubles from age 0 to 2 years (Chiron et al., 1992). Further, the frontal association cortex is the last area to increase blood flow from infancy to childhood, and reaches adult values only by adolescence (Takahashi, Shirane, Sato, & Yoshimoto, 1999). Direct support for age-related changes in brain activity associated with metacognition is provided by a neuroimaging investigation of theory of mind in participants whose age ranged between 9 and 16 years (Moriguchi, Ohnishi, Mori, Matsuda, & Komaki, 2007). Both children and adolescents demonstrated significant activation in the neural circuits associated with mentalizing tasks, including the TPJ, the temporal poles, and the medial PFC. Furthermore, the authors found a positive correlation between age and the degree of activation in the dorsal part of the medial PFC. Impairment of the medial/cingulate PFC is commonly associated with deficits in social interaction and self-conscious emotions (Sturm, Rosen, Allison, Miller, & Levenson, 2006). Such patients may become apathetic, disinterested in the environment, and unable to concentrate their attention on behavioral and cognitive tasks. It has also been suggested that frontal damage hinders perspective taking ability (Price, Daffner, Stowe, & Mesulam, 1990). The current understanding of the role of the PFC in executive functioning fits well with developmental research, which

indicates that empathic concern is strongly related to effortful control and self-regulation, with children high in effortful control expressing greater sympathy and less personal distress (Rothbart, Ahadi, & Hershey, 1994). Effortful control may support empathy and prosocial behavior by allowing the child to attend to the thoughts and feelings of another without becoming overwhelmed by their own distress (Posner & Rothbart, 2000).

Adopting another's perspective is integral to human empathy and is linked to the development of moral reasoning (Kohlberg, 1976), altruism (Batson, 1991) and a decreased likelihood of interpersonal aggression (Eisenberg et al., 2006). Of special interest are findings from social psychology that document the distinction between imagine the other and imagine oneself (Batson, Sager, et al., 1997). These studies show that the former may evoke empathic concern (defined as an other-oriented response congruent with the perceived distress of the person in need), whereas the latter induces both empathic concern and personal distress. This observation may help explain why empathy, or sharing someone else's emotion, need not yield prosocial behavior. If perceiving another person in an emotionally or physically painful circumstance elicits personal distress, then the observer may tend not to fully attend to the other's experience and as a result lack sympathetic behaviors.

The effect of perspective taking to generate empathic concern was documented in a study conducted by Stotland (1969). In his experiment, participants viewed an individual whose hand was strapped in a machine that participants were told generated painful heat. One group of subjects were instructed to watch the target person carefully, another group of participants were instructed to imagine the way the target felt, and the third group was instructed to image themselves in the target's situation. Physiological (palm sweating and vasoconstriction) and verbal assessments of empathy demonstrated that the deliberate acts of imagination yielded a greater response than passive viewing. Empathy specifically seems to be sensitive to perspective taking, as demonstrated by a series of studies demonstrate the effectiveness of perspective-taking instructions in inducing empathy (Batson, Sager, et al., 1997) and that empathy-inducing conditions do not compromise the distinction between the self and other (Batson, Sager, et al., 1997, but see Cialdini, Brown, Lewis, Luce, & Neuberg, 1997, for a different account of empathy and self-other merging).

A recent study by Lamm et al. (2007) investigated the distinction between empathic concern and personal distress combining a number of behavioral measures and event-related fMRI. Participants were asked to watch a series of videoclips featuring patients (their face only) undergoing painful medical treatment either with the instruction to put themselves explicitly in the shoes of the patient ("imagine self"), or, in another condition, to focus their attention on the feelings and reactions of the patient ("imagine other"). Behavioral measures confirmed previous social psychology findings that projecting oneself into an aversive situation leads to higher personal distress and lower empathic concern while focusing on the emotional and behavioral reactions of another's plight is accompanied by higher empathic concern and lower personal distress (e.g., Batson et al., 2003). Neuroimaging data were consistent with such findings. Both the self and other' perspectives were associated with hemodynamic signal increase in the neural regions that belong to the pain matrix including the insula and ACC. However, the self-perspective evoked stronger hemodynamic responses in brain regions involved in coding the motivational– affective dimensions of pain, including bilateral insular cortices and aMCC. In addition, the self-perspective led to stronger activation in the amygdala, a limbic structure that plays a critical role in fear-related behaviors, such as the evaluation of actual or potential threats. It is of interest that the amygdala receives nociceptive information from the spinoparabrachial pain system and the insula, and its activity appears closely tied to the context and level of aversiveness of the perceived stimuli (Zald, 2003). Imagining oneself to be in a painful and potentially dangerous situation thus triggers a stronger fearful and/or aversive response than imagining someone else to be in the same situation. Alternatively and less specifically, the stronger involvement of the amygdala might also reflect a general increase of arousal evoked by imagining oneself to be in a painful situation. Regarding the insular activation, it is worth noting that it was located in the middorsal section of this area. This part of the insula plays a role in coding the

sensory–motor aspects of painful stimulation, and it has strong connections with the basal ganglia, in which activity was also higher when adopting the self-perspective. Taken together, activity in this aspect of the insula possibly reflects the simulation of the sensory aspects of the painful experience. Such a simulation might both lead to the mobilization of motor areas (including the SMA) to prepare defensive or withdrawal behaviors, and to interoceptive monitoring associated with autonomic changes evoked by this simulation process.

Various domains of research suggest that mental flexibility to adopt another person's point of view is an effortful and controlled process. Moreover, the capacity to take the conceptual perspective of the other is thought to be a necessary component in the fully developed, mature theory of mind. Developmental research indicates that perspective taking develops progressively. In the affective domain, children demonstrate emerging awareness of the subjectivity of other people's emotions around 18 months. By this age, infants understand that they should provide an experimenter with a piece of food that the experimenter reacts to with apparent happiness (e.g., broccoli) rather than one that the experimenter previously reacted to with disgust (fish crackers), even if the infant prefers the latter food (Repacholi & Gopnik, 1997). In contrast, 14-month-olds fail to demonstrate this understanding. This is the first empirical evidence that infants of this age have at least a limited ability to reason nonegocentrically about people's desires (Flavell, 1999).

Executive functions not only facilitate perspective taking, but also control attention and metacognitive capacities, both of which facilitate prosocial responding in reaction to another's distress. Attention to others and the environment occurs when individuals are able to attend to external stimuli and disregard to some extent their self-experience. Metacognitive capacities allow for recursive thinking about the self's actions, and are thus linked to emotions such as shame or guilt which emerge as a result of causing another's distress. Children first demonstrate responses to the distress of others with other-focused behaviors like concern, attention to the distress of the other, cognitive exploration of the event, and prosocial interventions around the second year of life. At this age children manifest a self-concept and self-conscious emotions, and children's reparative behaviors after they cause distress in the other emerge (Zahn-Waxler & Radke-Yarrow, 1990). A longitudinal study of young children's development of concern for others' distress showed that prosocial behaviors, such as hugs and pats, emerge around the beginning of the second year of life, increasing in intensity throughout this year and sometimes provide self-comfort. However, by the end of the second year, prosocial behaviors appear to be more appropriate to the victims needs, are not necessarily self-serving, and children's emotions appear to be better regulated (Radke-Yarrow & Zahn-Waxler, 1984).

The ability to regulate emotions may be subject to individual differences, and may interact with the degree to which individuals experience emotions. Eisenberg and her colleagues (1994) proposed a model suggesting an interaction between the intensity at which emotions are experienced and the extent to which individuals can regulate their emotions. In line with her model, multimethod regression analysis of empathy-related responses combining self-report measures and facial muscle activity in response to empathy-inducing videos (of impoverished children), suggest that increased emotional intensity and decreased regulation on standard self-report measures predict personal distress in response to viewing the video vignettes. These interactions are first seen in infancy, as findings from infant development demonstrate that 4-month-olds low in self-regulation are prone to personal distress at 12 months of age (Ungerer et al., 1990). In childhood, individuals with increased levels of emotional intensity (based on self-report, teacher–parent report, and autonomic measurements) and weak regulation are prone to personal distress in response to another's predicament, as they become overwhelmed due to their vicariously induced negative emotions (Miller & Eisenberg, 1988).

Mental flexibility deficits in developmental disorders of empathy

Children with empathy deficits likewise show deficits in executive function and children with ASD specifically show deficits in mental flexibility

(Bennetto, Pennington, & Rogers, 1996; Minshew, Meyer, & Goldstein, 2002; Ozonoff & McEvoy, 1994; Ozonoff, Pennington, & Rogers, 1991). A series of studies found that when an experimenter feigns distress in a room where children were playing, children with ASD looked to the experimenter much less than healthy and mentally retarded children (Corona, Dissanayake, Arbelle, Wellington, & Sigman, 1998; Dissanayake & Sigma, & Kasari, 1996; Sigman, Kasari, Kwon, & Yirmiya, 1992). However, when Blair (1999) replicated such studies, but controlled for executive function demands of attention, children with ASD performed similar to healthy children. That is, when experimenters' feigned distress was unambiguous and took place under conditions of low distractibility, children with ASD showed autonomic responses similar to controls. In studies measuring facial mimicry, when given ample time, individuals with ASD do show affective compensatory tactics to accomplish emotion reading; and in emotion recognition tasks, they show activation in brain areas related to intentional attentional provision and categorization (Hall, Szechtman, & Nahmias, 2003). These data indicate that alongside bottom-up information processing deficits (e.g., affective mimicry), top-down executive control is also impaired in individuals with ASD.

Empathy deficits in Asperger syndrome also seem to reflect poor executive function. A case study of two adolescents with Asperger syndrome with severe inabilities for emotional and cognitive aspects of empathy suggests that these weaknesses do not reflect significantly worse emotion recognition or cognitive perspective taking per se, but instead, reveal poor integration of the cognitive and affective components of empathy (Shamay-Tsoory, Tomer, Yaniv, & Aharon-Peretz, 2002).

Indeed, these executive function deficits in ASD have been documented by fMRI experiments using cognitive tasks. Aberrant activity in prefrontal, frontal, as well as atypical fronto-parietal interactions, have all been documented in fMRI experiments probing executive function in ASD (Kana, Keller, Minshew, & Just, 2007; Silk et al., 2006). It is likely that deficits in mental flexibility are grounded in atypical structure and function in prefrontal, frontal, and parietal lobes and contribute to perspective-taking difficulties observed in individuals with ASD.

It is noteworthy that violent offenders, and children with aggressive behavior problems, experience deficits in empathy and empathic concern, although the result of the lack of empathy manifests in behavior differently than that seen in ASD or DCD. The former responds aggressively to others' distress (Arsenio & Lemerise, 2001), whereas the later simply lack prosocial behavior. The distinction can be understood as the difference between apathy and hostility, both of which are categorized as unempathetic in the traditional sense, although one is "passive," and the other is "active." Individuals with ASD seem to lack either an interest or capacity to resonate emotionally with others, or engage in intersubjective transactions (Gallagher, 2001). In contrast, children with developmental aggression disorders react aggressively to the observation of others' distress.

CD is a mental disorder of childhood and adolescence that is characterized by a longstanding pattern of violations of rules and laws. Symptoms of CD include aggression, frequent lying, running away from home overnight, and destruction of property. CD is important partly because it is the major childhood precursor to antisocial personality disorder in adulthood (Lahey, Loeber, Burke, & Applegate, 2005). Children with aggressive behavior problems show deficits in regulating emotions, which may result in harmful patterns of interpersonal behavior. Lewis, Granic, and Lamm (2006) reviewed several of their recent studies investigating individual and developmental differences in cortical mechanisms of emotion regulation, corresponding with different patterns of interpersonal behavior. Their methods include event-related potentials and cortical source modeling, using dense-array EEG, as well as videotaped observations of parent–child interactions, with both normal and aggressive children. By relating patterns of brain activation to observed behavioral differences, the authors found (a) a steady decrease in cortical activation subserving self-regulation across childhood and adolescence, (b) different cortical activation patterns as well as behavioral constellations distinguishing subtypes of aggressive children, and (c) robust correlations between the activation of cortical mediators of emotion regulation and flexibility in parent–child emotional communication in children referred for aggressive behavior problems.

Emotion is normally regulated in the human brain by a complex circuit that includes several regions of the PFC (dorsal and ventral), the amygdala, hypothalamus, ACC, insula, and ventral striatum. Descending pathways from orbitofrontal and medial prefrontal cortices, which are also linked with the amygdala, provide the means for speedy influence of the PFC on the autonomic system, in processes underlying appreciation and expression of emotions (Barbas, Saha, Rempel-Clover, & Ghashghael, 2003). It is of interest that key areas found to be functionally or structurally impaired in antisocial populations include dorsal and ventral regions of the PFC, amygdala, hippocampus, TPJ, and ACC (Raine & Yang, 2006). Raine has hypothesized that the rule-breaking behavior common to antisocial, violent, and psychopathic individuals may in part be attributable to impairments in some of the structures (dorsal and ventral PFC, amygdala, and TPJ) or dysfunction in amygdala–orbitofrontal cortex (OFC) structural connectivity, normally subserving moral cognition and emotion.

In contrast, impulsive aggression may be the product of failed emotion regulation. Impulsive aggression is associated with a low threshold for activating negative affect and with failure to respond appropriately to the anticipated negative consequences of behaving aggressively. Davidson, Jackson, and Kalin (2000) have proposed that the mechanism underlying suppression of negative emotion is via an inhibitory connection from regions of the PFC to the amygdala.

In addition to functional brain abnormalities corresponding with emotion dysregulation and empathy deficits in CD, impulsive aggression in CD is associated with decreased noradrenergic (NA) function, which also correlates with poor empathic ability in this population (Raine, 1996). In fact, a low resting heart rate, a partly heritable trait reflecting fearlessness and stimulation seeking, at 3 years of age predicted aggressive behavior at 11 years of age (Raine, Venables, Mednick, & Sarnoff, 1997). Children with clinical levels of behavior problems, often a precursor to the development of CD, show increased disregard for others, for example, anger, avoidance, and/or amusement by another's distress, a negatively toned response pattern that differs significantly from normal children's responses. It is likely that decreased NA function, which is associated with aggressive behavior, contributes to these antisocial reactions.

One way to determine whether CD is associated with low arousal or poor regulation is to investigate patterns and changes in autonomic nervous system. Sympathetic activation or parasympathetic inhibition leads to changes in cardiac functioning, which can be interpreted as bodily cues of discomfort or distress and a need for action. Porges (1996) has found that parasympathetic nervous system functioning, as reflected in heart rate variability (heart rate [HR] variability measures the variability in HR associated with breathing and indexes an individual's competency to physiologically and behaviorally reacts to external stimuli) influenced by the vagal system, is related to the control of attention, emotion and behavior. Porges suggests that the tonus of the vagus nerve provides a theoretical basis for the child's ability to focus attentional processes, inhibit irrelevant activity, regulate emotion, and appropriately engage with the environment (Porges, 1995).

In the domain of empathy, the work of Eisenberg and Fabes (1994) suggests that deceleration of HR may be associated with attention to others that characterizes empathic concern, and HR deceleration is also associated with an increase in desire to help and comforting. A number of studies indicate that antisocial behavior, and CD are associated with, and predictable from, low resting HR in children, adolescents, and adults (e.g., Lahey, Hart, Pliszka, Applegate, & McBurrett, 1993; Raine, Venables, & Sarnoff, 1997). Because low resting HR is associated with greater aggression, and aggression and concern for others are inversely related, it has been predicted that high HR should predict greater concern for others. One study found that HR was positively correlated with concerned responses toward adults who were simulating injuries (Zahn-Waxler, Cole, Welsh, & Fox, 1995). However, Calkins and Dedmond (2000) reported that aggressive children displayed no physiological indicators of underarousal, as indexed by resting HR. The authors did find, however, that these children displayed poor behavioral and physiological regulation, as indexed by a lack of HRV during challenging situations. This latter finding supports the idea of a failure of self-regulation in relation to empathy and aggression. Cardiac vagal regulation was

found to differentiate among children at risk for behavior problems (Calkins, Graziano & Keane, 2007). In that study (a sample of 335 children), there was a trend for the children at risk for externalizing problems to display less vagal withdrawal than the control group. Future research should ultimately clarify the relationship between structural/functional differences in patterns of brain activation with NA function in individuals with CD.

The maturation of executive functions, including emotion regulation (subserved by the dorsal and ventral PFC and their connections with the amygdala) by 2 years of age contributes to the development of prosocial behaviors. Conversely, if executive functioning is not intact, self and other perspectives may not be regulated and individuals may over- or underidentify with an observed target. In the case of childhood aggression and CD, it is likely that poor executive control and dysfunction of emotion regulation contributes to empathy deficits, although other factors (NA function) also contribute to reactionary behaviors.

Conclusions

We have argued that empathy depends upon both bottom-up processes, which are driven by emotion expressions, and top-down processes, including self-regulation and executive control. These different aspects are underpinned by distinct neural systems that develop at different stages. Notably, emotion sharing relies on the perception–action coupling mechanism, which seems functional very early in development, and allows the newborn to implicitly share subjective bodily experiences with others. Controlled processes, subserved by the PFC, develop later and play a major role in metacognition, including taking into account a cognitive representation of one's own mind and other's mind, aspects that are necessary for social emotions which require self-monitoring (see Figure 1). We have also shown that developmental disorders related to empathy reflect dysfunction of these different aspects. It is noteworthy that the current knowledge in social cognitive neuroscience is at a preliminary stage, and many findings need to be reproduced. In relation to the goal of this paper, ties drawn between developmental and neuroscientific evidence in emotion sharing and empathy can only be loosely strung together, as many neuroscience findings pertain to adult participants, not children. Future studies should aim to incorporate children in the investigation of the neural basis of empathy, especially because contradictions surround the few neuroscience studies that examine emotion sharing in children.

Finally, the model reviewed here offers interesting insights for debates surrounding the naturalization of normative ethics. Empathy has been associated with the propensity to respond to another's predicament in a prosocial, or "moral" way. Yet, an understanding of what motivates us to feel empathy in the sense of caring for the other (Batson et al., 2003) and then help them is unclear. In fact, humans fail to consistently respond to others' negative situations prosocially across contexts. Further research in child development and cognitive neuroscience may help elucidate these interrelationships and hopefully offer a better understanding of emotional resonance, empathy, and their relationship with prosocial, moral reasoning (and the lack thereof). The results of a recent fMRI study on empathy and theory of mind with children seem to indicate that there is little overlap in the brain circuits associated with empathy for pain and moral reasoning, although this does not mean that these circuits do not communicate (Decety et al., 2008). In this study, children watched situations involving either a person whose pain was caused accidentally or a person whose pain was intentionally inflicted by another individual, as well as situations without any pain with one or two agents. It is important that when children observed an individual intentionally harming another, regions of the PFC that are consistently involved in representing social interaction and moral behavior such as the temporoparietal junction, the paracingulate cortex (PCC), and OFC were recruited (see Figure 2). It is of interest that children indicated in the postscan debriefing that they thought that the situations in which pain was caused by another person were unfair, and were asking about the reason that could explain this behavior. Evidence from moral neuroscience suggests a critical role of a corticolimbic network subserving moral judgment. This network includes the medial OFC, the TPJ, the amygdala, and anterior PCC (Moll, de Oliveria-Souze, & Eslinger, 2003). Furthermore, the monitoring of outcomes that relate to punishments and rewards is linked to activity in the OFC

(Kringelbach & Rolls, 2004). It is worth emphasizing that the regions selectively associated with the perception of an agent harming the other belong to the neural systems underlying moral thinking.

We have argued that genuine empathy goes beyond emotion sharing and a simple resonance of affect between the self and other. It depends crucially on self–other awareness and on the ability to regulate one's own emotional state, allowing proper identification of the other's condition and freeing up resources for coping with another's distress in prosocial ways. Successful emotion regulation in infancy is essential for the development of the ability to control one's own arousal and, with the sense of agency, be able to tag the aroused state as indicative of the state of the other. Children as well as adults who become overaroused by another's distress, due to lack of emotion regulation and/or self–other distinction, may use up too many cognitive resources dealing with their own emotion and fail to act in a prosocial fashion (Nielsen, 2002).

Limitations of the scope

It is important to note several limitations of our review. The first limitation pertains to the extent to which conclusions can be drawn from the relationship between the development of empathy in healthy children and the manifestation of empathy deficits that occur in developmental disorders. We covered findings from infant development to address the innateness of the interacting components of empathy; however, most of the findings reviewed from developmental disorders pertain to observations from early childhood. This limitation in part reflects the difficulty to obtain infant data across developmental disorders as they are often not diagnosed until childhood. Some analysis of infant behavior of children diagnosed with developmental disorders comes from home video analysis (see Baranek, 1999), although such studies are sparse, and findings are ambiguous due to weak scientific control.

Second, the relationship between neural evidence and behavioral data is indirect, and thus speculations may be far reaching. Neuroimaging data help to answer fundamental questions about the mechanisms subserving imitation. However, their interpretation in relating structure to function should be done with caution. It is difficult to derive the computational function of an area without taking into account its extrinsic and intrinsic connectivity, the distribution of receptor types, and the information processing of the intrinsic neurons. Such information is generally lacking. In addition, a set of cortical areas may be active in a wide range of functions from action perception to empathy and theory of mind, but across those functions the networks in which they participate may be quite different (see Cacioppo et al., 2003). More empirical data from both developmental science and cognitive neuroscience in the future will contribute to a fuller understanding of the mechanisms involved in empathy and their developmental time course. Furthermore, brain imaging investigations of emotional processes often draw from limited methodologies that may overlook flaws in the operational definitions of the emotional phenomena studied. For example, many fMRI experiments require participants to identify the emotion in static faces or short animations, implicitly considering that emotion recognition can be equated with emotional experience. Clinical investigations with neurological patients, however, indicate that these processes (emotion recognition and experience) involve quite different neural substrates (Levenson, 2007). Future neuroimaging studies of emotion in general, and empathy in particular, should base stimuli on the discrete emotional experience examined. Meeting this goal would be facilitated by the further collaboration between cognitive neuroscientists and developmental psychologists.

Third, and finally, empathy is a complex construct and the above model does not account for all that empathy entails. The phenomenological experience of empathy and its role in initiating prosocial, empathic reactions likely draws on several interacting factors (and complicated distributed brain networks) not mentioned in our review. For example, motivation likely influences empathic accuracy: people who are motivated to produce empathically accurate responses to another's predicament are less susceptible to social inference biases such as the fundamental attribution error (Fletcher, Reeder, & Bull, 1990; Tetlock, 1985). In addition, the type of rapport between observer and target influences the intensity of emotion contagion between members of a dyad, with patients and therapists, mothers and infants, and spouses, and even individuals who

perceive others as similar to themselves ranking high in autonomic synchrony (Levenson & Ruef, 1997).

Another important area of interest regarding the contribution of empathy to the development of intersubjectivity is its relation to the so-called attachment system described by Bowlby (1958), that is, an innate psychobiological system that motivates infants to seek proximity to people who will protect them. One can argue that the mechanisms that underpin the development of empathy, especially through interaction with caregivers who are responsive, partly overlaps, and are functionally linked with promoting optimal functioning of the attachment system. The consequences of this link between empathy and attachment are paramount. Indeed, Mikulineer and Shaver (2005) have documented the idea that people who have the benefits of secure social attachments find it easier to perceive and respond to other people suffering, compared to those who have insecure attachments.

Personality traits, temperaments, and cultural norms of emotional display also contribute to the degree to which empathy may be experienced in the observer (Eisenberg & Fabes, 1994; Posner & Rothbart, 2000). Likewise, children from cultures that promote reciprocal relations and cooperation tend to be better at perspective-taking tasks than children living in individualistic cultures (Eisenberg, Bridget, & Shepard, 1997).

Social psychologists emphasize the role of situational context as opposed to personality in the experience of empathy (or the absence of it), although many recognize the combination of situation and personality as the best predictor of social behavior (Fiske, 2004). Although situational context is important, creating ecologically valid situations in a laboratory setting or in an MRI scanner poses a challenge. Theoretically, assessing personality traits and correlating them with task performance and biological markers should pose no great challenge. These constructs, however, are rarely stable and they are dependent on many variables. Thus, designing ecologically valid experiments remains a challenging process, especially with children.

A current aim in cognitive neuroscience is to study the interaction between affect and cognition. Empathy, a valuable social phenomenon, exemplifies this complex relationship because it draws on aspects such as emotion sharing and self-regulation. In addition, affective and social cognitive developmental neuroscience offers promising insights for both our understanding of typical and psychopathological social behavior.

References

Apperly, I., Samson, D., Chiavarino, C., & Humphreys, G. (2004). Frontal and temporo-parietal lobe contributions to theory of mind: Neuropsychological evidences from a false-belief task with reduced language and executive function. *Journal of Cognitive Neuroscience*, *16*, 1773–1784.

Arsenio, W. F., & Lemerise, E. A. (2001). Varieties of childhood bullying: Values, emotion processes, and social competence. *Review of Social Development*, *10*, 59–73.

Avikainen, S., Kulomaki, T., & Hari, R. (1999). Normal movement reading in Asperger subjects. *NeuroReport*, *10*, 3467–3470.

Baranek, G. T. (1999). Autism during infancy: A retrospective video analysis of sensory-motor and social behaviors at 9–12 months of age. *Journal of Autism and Developmental Disorders*, *199*, 213–224.

Barbas, H., Saha, S., Rempel-Clover, N., & Ghashghaei, T. (2003). Serial pathways from primate prefrontal cortex to autonomic areas may influence emotion expression. *BMC Neuroscience*, *4*, 1–12.

Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences*, *6*, 248–254.

Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A. L. (2004). White matter structure in autism: Preliminary evidence from diffusion tensor imaging. *Biological Psychiatry*, *55*, 323–326.

Bastiaanen, J., Thioux, M., & Keysers (2008, April). *Mirror-neuron system not broken in adults with ASD for viewing emotions of others*. Paper presented at the Fifteen Cognitive Neuroscience Society Meeting, San Francisco.

Batson, C. D. (1990). How social an animal? The human capacity for caring. *American Psychologist*, *45*, 336–346.

Batson, C. D. (1991). *The altruism question: Toward a social–psychological answer*. Hove: Erlbaum.

Batson, C. D., Fultz, J., & Schoenrade, P. (1987). Distress and empathy: Two qualitatively distinct vicarious emotions with different motivational consequences. *Journal of Personality*, *55*, 19–39.

Batson, D. C., Early, S., & Salvarani, G. (1997). Perspective taking: Imagining how another feels versus imagining how you would feel. *Personality & Social Psychology Bulletin*, *23*, 175–178.

Batson, C. D., Lishner, D. A., Carpenter, A., Dulin, L., Harjusola-Webb, S., Stocks, E. L., et al. (2003). As you would have them do unto you: Does imagining yourself in the other's place stimulate moral action? *Personality & Social Psychology Bulletin*, *29*, 1190–1201

Batson, C. D., Sager, K., Garst, E., Kang, M., Rubchinsky, K., & Dawson, K. (1997). Is empathy-induced helping because of self-other merging? *Journal of Personality & Social Psychology*, *73*, 495–509.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, *117*, 497–529.

Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development*, *67*, 1816–1835.

Berluchi, G., & Aglioti, S. (1997). The body in the brain: Neural bases of corporeal awareness. *Trends in Neuroscience*, *20*, 560–564.

Bernier, R., Dawson, G., Webb, S., & Murias, M. (2007). EEG mu rhythm and imitation impairments in individuals with autism spectrum disorder. *Brain and Cognition*, *64*, 228–237.

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

Blakemore, S.-J., & Frith, C. D. (2003). Self-awareness and action. *Current Opinion in Neurobiology*, *13*, 219–224.

Blair, R. J. R. (1999). Psycho-physiological responsiveness to the distress of others in children with autism. *Personality and Individual Differences*, *26*, 477–485.

Blanke, O., & Arzy, S. (2005). The out-of-body experience: Distributed self-pricessing at the temporo-parietal junction. *The Neuroscientist*, *11*, 16–24.

Blanke, O., Ortigue, S., Landis, T., & Seeck, M. (2002). Stimulating illusory own-body perceptions: The part of the brain that can induce out-of-body experiences has been located. *Nature*, *419*, 269–270.

Bowlby, J. (1958). The nature of the child's tie to his mother. *International Journal of Psychoanalysis*, *39*, 350–373.

Bunge, S. A., Dudukovic, N. N., Thomason, M. E., Vaidya, C. J., & Gabrieli, J. D. E. (2002). Immature frontal lobe contributions to cognitive control in children: Evidence from fMRI. *Neuron*, *33*, 301–311.

Bush, L. K., Barr, C. L., McHugo, G. J., & Lanzetta, J. T. (1989). The effects of facial control and facial mimicry on subjective reactions to comedy routines. *Motivation and Emotion*, *13*, 31–52.

Cacioppo, J. T., Berntson, G. G., Lorig, T. S., Norris, C. J., Rickett, E., & Nusbaum, H. (2003). Just because you're imaging the brain doesn't mean you can stop using your head: A primer set of first principles. *Journal of Personality and Social Psychology*, *85*, 650–661.

Calkins, S. D., & Dedmon, S. (2000). Physiological and behavioral regulation in two-year-old children with aggressive/destructive behavior problems. *Journal of Abnormal Child Psychology*, *28*, 103–118.

Calkins, S. D., Graziano, P. A., & Keane, S. P. (2007). Cardiac vagal regulation differentiates among children at risk for behavioral problems. *Biological Psychology*, *74*, 144–153.

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

Carlson, S. M., & Moses, L. J. (2001). Individual differences in inhibitory control and children's theory of mind. *Child Development*, *72*, 1032–1053.

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

Cheng, Y., Lin, C., Liu, H. L., Hsu, Y., Lim, K., Hung, D., et al. (2007). Expertise modulates the perception of pain in others. *Current Biology*, *17*, 1708–1713.

Cheng, Y., Meltzoff, A. N., & Decety, J. (2007). Motivation modulates the activity of the human mirror system: An fMRI study. *Cerebral Cortex*, *17*, 1979–1986.

Chiron, C., Raynaud, C, Maziere, B., Zilbovicius, M., Laflamme, L., Masure, M. C., et al. (1992). Changes in regional cerebral blood flow during brain maturation in children and adolescents. *The Journal of Nuclear Medicine 33*, 696–703.

Cialdini, R., Brown, S., Lewis, B., Luce, C., & Neuberg, S. (1997). Reinterpreting the empathy-altruism relationship: When one into one equals oneness. *Journal of Personality and Social Psychology*, *73*, 481–494.

Corona, C., Dissanayake, C., Arbelle, A., Wellington, P., & Sigman, M. (1998). Is affect aversive to young children with autism? Behavioral and cardiac response to experimenter distress. *Child Development*, *69*, 1494–1502.

Cummins, A., Piek, J. P., & Murray, J. D. (2005). Motor coordination, empathy, and social behavior in school-aged children. *Developmental Medicine & Child Neurology*, *47*, 437–432.

Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., et al. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, *9*, 28–31.

Darwin, C. (1872). *The expression of the emotions in man and animals*. London: John Murray Editions.

Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspective from affective neuroscience. *Psychological Bulletin*, *126*, 890–909.

Davis, M. H. (1994). *Empathy: A social psychological approach*. Boulder, CO: Westview Press.

DeCasper, A. J., & Fifer, W. P. (1980). Of human bonding: Newborns prefer their mothers' voices. *Science*, *208*, 1174–1176.

Decety, J. (2002). Naturaliser l'empathie [Naturalized empathy]. *L'Encephale*, *28*, 9–20.

Decety, J. (2005). Perspective taking as the royal avenue to empathy. In B. F. Malle & S. D. Hodges (Eds.), *Other minds: Other minds: How humans bridge the divide between self and others* (pp. 135–149). New York: Guilford Press.

Decety, J., & Chaminade, T. (2003). When the self represents the other: A new cognitive neuroscience view of psychological identification. *Consciousness and Cognition*, *12*, 577–596.

Decety, J., Chaminade, T., Grèzes, J., & Meltzoff, A. N. (2002). A PET exploration of the neural mechanisms involved in reciprocal imitation. *NeuroImage*, *15*, 265–272.

Decety, J., & Grèzes, J. (2006). The power of simulation: Imagining one's own and other's behavior. *Brain Research*, *1079*, 4–14.

Decety, J., & Lamm, C. (2008). Empathy versus personal distress—Recent evidence from social neuroscience. In J. Decety & W. Ickes (Eds.), *The social neuroscience of empathy*. Cambridge, MA: MIT Press.

Decety, J., Michalska, K., & Akitsuki, Y. (2008). Who caused the pain? A functional MRI investigation of empathy and intentionality in children. *Neuropsychologia*, *46*, 2607–2614.

Decety, J., & Jackson, P. (2004). The functional architecture of human empathy. *Behavioral and Cognitive Neuroscience Reviews*, *3*, 71–100.

Decety, J., & Jackson, P. (2006). A social neuroscience perspective on empathy. *Current Directions in Psychological Science*, *12*, 406–411.

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

Decety, J., & Lamm, C. (2007). The role of the right temporoparietal junction in social interaction: How low-level computational processes contribute to meta-cognition. *The Neuroscientist*, *13*, 580–593.

Decety, J., & Moriguchi, Y. (2007). The empathic brain and its dysfunction in psychiatric populations: Implications for intervention across different clinical conditions. *BioPsychoSocial Medicine*, *1*, 22–65.

Decety, J., & Sommerville, J.A. (2003). Shared representations between self and others: A social cognitive neuroscience view. *Trends in Cognitive Sciences*, *7*, 527–533.

Dissanayake, C., Sigman, M., & Kasari, C. (1996). Long-term stability of individual differences in the emotional responsiveness of children with autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *37*, 461–467.

Dondi, M., Simion, F., & Caltran, G. (1999). Can newborns discriminate between their own cry and the cry of another newborn infant? *Developmental Psychology*, *35*, 418–426.

Eisenberg, N. (2000). Emotion, regulation, and moral development. *Annual Review of Psychology*, *51*, 665–697.

Eisenberg, N., Bridget, C. M., & Shepard, S. (1997). The development of empathic accuracy. In W. Ickes (Ed.), *Empathic accuracy* (pp. 194–215). New York: Guilford Press.

Eisenberg, N., & Eggum, N. D. (in press). Empathic responding: Sympathy and personal distress. In J. Decety and W. Ickes, *The Social Neuroscience of Empathy*. Cambridge, MA: MIT Press.

Eisenberg, N., & Fabes, R.A. (1990). Empathy: Conceptualization, measurement, and relation to prosocial behavior. *Motivation and Emotion*, *14*, 131–149.

Eisenberg, N., Fabes, R. A., Murphy, B., Karbon, M, Maszk, P., Smith, M., et al. (1994). The relations of emotionality and regulation to dispositional and situational empathy-related responding. *Journal of Personality and Social Psychology*, *66*, 776–797.

Eisenberg, N., Shea, C. L., Carlo, G., & Knight, G. P. (1991). Empathy-related responding and cognition: A "chicken and the egg" dilemma. In W. M. Kurtines & J. L. Gewirtz (Eds.), *Handbook of moral behavior and development research* (Vol. 2., pp. 63–88). New York: Erlbaum.

Eisenberg, N., Spinrad, T. L., & Sadovsky, A. (2006). Empathy-related responding in children. In M. Killen & J. Smetana (Eds.), *Handbook of moral development* (pp. 517–549). Mahwah, NJ: Erlbaum.

Fadiga, L., & Craighero, L. (2004). Electrophysiology of action representation. *Journal of Clinical Neurophysiology*, *21*, 157–169.

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

Farrer, C., & Frith, C. D. (2002). Experiencing oneself vs. another person as being the cause of an action: The neural correlates of the experience of agency. *NeuroImage*, *15*, 596–603.

Farrer, C., Franck, N., Frith, C. D., Decety, J., Damato, T., & Jeannerod, M. (2004). Neural correlates of action attribution in schizophrenia. *Psychiatry Research: Neuroimaging*, *131*, 31–44.

Farrer, C., Franck, N., Georgieff, N., Frith, C. D., Decety, J., & Jeannerod, M. (2003). Modulating the experience of agency: A positron emission tomography study. *NeuroImage*, *18*, 324–333.

Fecteau, S., Carmant, L., Tremblay, C., Robert, M. Bouthillier, A., & Theoret, H. (2004). A motor resonance mechanism in children? Evidence from subdural electrodes in a 6-month-old child. *NeuroReport*, *15*, 22625–22627.

Feshbach, N. D. (1997). Empathy: The formative years—Implications for clinical practice. In A. C. Bohart & L. S. Greenberg (Eds.), *Empathy reconsidered: New directions in psychotherapy* (pp. 33–59). Washington, DC: American Psychological Association.

Field, T. (1989). Individual and maturational differences in infant expressivity. In N. Eisenberg (Ed.), *Empathy and related emotional responses*, (pp. 9–23). San Francisco, CA: Jossey–Bass.

Field, T., Healy, B., Goldstein, S., & Guthertz, M. (1990). Behavior-state matching and synchrony in mother–infant interactions of nondepressed versus depressed dyads. *Developmental Psychology*, *26*, 7–14.

Field, T. M., Woodson, R., Greenberg, R., & Cohen, D. (1982). Discrimination and imitation of facial expression by neonates. *Science*, *219*, 179–181.

Fiske, S. (2004). *Social beings: A core motives approach to social psychology*. New York: Wiley.

Flavell, J. H. (1999). Cognitive development: Children's knowledge about the mind. *Annual Review of Psychology*, *50*, 21–45.

Fletcher, G. J. O., Reeder, G., & Bull, V. (1990). Bias and accuracy in attitude attribution: The role of attributional complexity. *Journal of Experimental Psychology*, *28*, 320–388.

Gallagher, S. (2000). Philosophical conceptions of the self: Implications for cognitive science. *Trends in Cognitive Sciences*, *4*, 14–21.

Gallagher, S. (2004). Understanding interpersonal problems in autism: Interaction theory as an alternative to theory of mind. *Philosophy, Psychiatry, & Psychology*, *11*, 199–217.

Gallagher, S. (2001). The practice of mind: Theory, simulation, or interaction? *Journal of Consciousness Studies*, *8*, 83–107.

Gallagher, S., & Meltzoff, A. N. (1996). The earliest sense of self and others: Merleau-Ponty and recent developmental studies. *Philosophical Psychology*, *9*, 211–233.

Gillberg, C. (1992). Autism and autistic like conditions: Subclasses among disorders of empathy. *Journal of Child Psychology and Psychiatry*, *33*, 813–842.

Goldman, A. (1993). Ethics and cognitive science. *Ethics*, *103*, 337–360.

Grèzes, J., Frith, C. D., & Passingham, R. E. (2004). Inferring false beliefs from the actions of oneself and others: An fMRI study. *NeuroImage*, *21*, 744–750.

Gusnard, D. A., Akbudak, E., Shulman, G. L., & Raichle, M. E. (2001). Medial prefrontal cortex and self-referential mental activity: Relation to a default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America*, *98*, 4259–4264.

Hadjikhani, N., Joseph, R., Snyder, J., & Gager-Flushberg, H. (2005). Anatomical differences in the mirror neuron system and social cognition network in autism. *Cerebral Cortex*, *10*, 1093.

Hall, G. B. C., Scezchtman, H., & Nahmias, C. (2003). Enhanced salience and emotion recognition in autism: A PET study. *American Journal of Psychiatry*, *160*, 1439–1441.

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*, 1859–1868.

Hastings, P. D., Zahn-Waxler., & McShane, K. (2006). We are, by nature, moral creatures: Biological bases of concern for others. In M. Killen & J. Smetana (Eds.), *Handbook of moral development* (pp. 483–516). Mahwah, NJ: Erlbaum.

Hatfield, E., Cacioppo, J., & Rapson, R. (1994). *Emotional contagion*. New York: Cambridge Press.

Haviland, J. M., & Lelwica, M. (1987). The induced affect response: 10-week-old infants' responses to three emotion expressions. *Developmental Psychology*, *23*, 97–104.

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

Hirai, M., & Hiraki, K. (2005). An event-related potentials study of biological motion perception in human infants. *Brain Research*, *22*, 301–304.

Hobson, R. P. (2002). *The cradle of thought*. London: Macmillan.

Hobson, P., & Meyer, J. (2006). Imitation, identification, and the shaping of mind: Insights from autism. In S. J. Rogers & S. H. G. Williams (Eds.), *Imitation and the social mind: Autism and typical development* (pp. 198–224). New York: Guilford Press.

Hoffman, M. L. (1975). Developmental synthesis of affect and cognition and its implications of altruistic motivation. *Developmental Psychology*, *23*, 97–104.

Hoffman, M. L. (1982). Development of prosocial motivation: Empathy and guilt. In N. Eisenberg (Ed.), *The development of prosocial behavior* (pp. 281–313). New York: Academic Press.

Hoffman, M. L. (2000). *Empathy and moral development: implications for caring and justice*. New York: Cambridge University Press.

Hoffman, M. L. (2001). A comprehensive theory of prosocial moral development. D. Stipek & A. Bohart (Eds.), *Constructive and Destructive Behavior* (pp. 61–86). Washington, DC: American Psychological Association.

Hommel, B., Musseler, J., Aschersleben, G., & Prinz, W. (2001). The theory of event coding: A framework for perception and action. *Behavioral and Brain Sciences*, *24*, 849–878.

Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology*, *387*, 167–178.

Ickes, W. (1997). *Empathic accuracy*. New York: Guilford Press.

Izard, C. E. (1982). *Measuring emotions in infants and young children*. New York: Cambridge University Press.

Jackson, P. L., Brunet, E., Meltzoff, A. N., & Decety, J. (2006). Empathy and the neural mechanisms involved in imagining how I feel versus how you would feel pain: An event-related fMRI study. *Neuropsychologia*, *44*, 752–761.

Jackson, P. L., & Decety, J. (2004). Motor cognition: A new paradigm to study self other interactions. *Current Opinion in Neurobiology*, *14*, 259–263.

Jackson, P. L., Rainville, P., & Decety, J. (2006). To what extent do we share the pain of others? Insight from the neural bases of pain empathy. *Pain*, *125*, 5–9.

Jeannerod, M. (1999). To act or not to act: Perspective on the representation of actions. *Quarterly Journal of Experimental Psychology*, *42*, 1–29.

Kana, R. K., Keller, T. A., Minshew, N. J., & Just, M. A. (2007). Inhibitory control in high-functioning autism: Decreased activation and underconnectivity in inhibition networks. *Biological Psychiatry*, *62*, 198–206.

Kennedy, D. P., Redcay, E., & Courchesne, E. (2006). Failing to deactivate: Resting functional abnormalities in autism. *Proceedings of the National Academy of Sciences of the United States of America*, *103*, 8275–8280.

Kohlberg, L. (1976). Moral stages and moralization: The cognitive–developmental approach. In T. Lickona (Ed.), *Moral development and behavior* (pp. 31–53). New York: Holt, Rinehart & Winston.

Kringelbach, M. L., & Rolls, E. T. (2004). The functional neuroanatomy of the human orbitofrontal cortex: Evidence from neuroimaging and neuropsychology. *Progress in Neurobiology*, *72*, 341–372.

Lahey, B. B., Loeber, R., Burke, J. D., & Applegate, B. (2005). Predicting future antisocial personality disorder in males from a clinical assessment in childhood. *Journal of Consulting and Clinical Psychology*, *73*, 389–399.

Lahey, B. B., Hart, E. L., Pliszka, S., Applegate, B., & McBurnett, K. (1993). Neurophysiological correlates of conduct disorder: A rational and review of research. *Journal of Clinical Child Psychology*, *22*, 141–153.

Lamm, C., Batson, C. D., & Decety, J. (2007). The neural basis of human empathy—Effects of perspective-taking and cognitive appraisal: An event-related fMRI study. *Journal of Cognitive Neuroscience*, *19*, 42–58.

Lee, A., Hobson, P. R., & Chiat, S. (1994). I, you, me, and autism: An experimental study. *Journal of Autism and Developmental Disorders*, *24*, 155–176.

Leiberg, S., & Anders, S. (2006). The multiple facets of empathy: A survey of theory and evidence. *Progress in Brain Research*, *156*, 419–440.

Lepage, J. F., & Theoret, H. (2006). EEG evidence for the presence of an action observation-execution matching system in children. *European Journal of Neuroscience*, *23*, 2505–2510.

Lepage, J. F., & Theoret, H. (2007). The mirror neuron system: Grasping others' action from birth? *Developmental Science*, *10*, 513–529.

Leube, D. T., Knoblich, G., Erb, M., Grodd, W., Bartels, M., & Kircher, T. T. J. (2003). The neural correlates of perceiving one's own movements. *NeuroImage*, *20*, 2084–2090.

Levenson, R. W. (2007). Emotion elicitation with neurological patients. In J. A. Coan & J. J. B. Allen (Eds.), *The handbook of emotion elicitation and assessment* (pp. 158–168). New York: Oxford University Press.

Levenson, R. W., & Ruef, A. M. Physiological aspects of emotional knowledge and rapport. In W. J. Ickes (Ed.), *Empathic accuracy* (pp. 194–215). New York: Guilford Press.

Lewis, M. D., Granic, I., & Lamm, C. (2006). Behavioral differences in aggressive children linked with neural mechanisms of emotion regulation. *Annals of the New York Academy of Science*, *1094*, 164–177.

Martin, G. B., & Clark, R. D. (1982). Distress crying in neonates: Species and peer specificity. *Developmental Psychology*, *18*, 3–9.

McIntosh, D. N., & Reichmann-Decker, A., Winkelman, P., & Wilbarger, J. L. (2006). When the social mirror breaks: Deficits in automatic, but not voluntary mimicry of emotional facial expressions in autism. *Developmental Science*, *9*, 295–302.

Meltzoff, A. N., & Brooks, R. (2001). "Like Me" as a building block for understanding other minds: Bodily acts, attention, and intention. In B. F. Malle, L. J. Moses & D. A. Baldwin (Eds.), *Intentions and intentionality:*

Foundations of social cognition (pp. 171–191). Cambridge, MA: MIT Press.

Meltzoff, A. N., & Decety, J. (2003). What imitation tells us about social cognition: A rapprochement between developmental psychology and cognitive neuroscience. *The Philosophical Transactions of the Royal Society, London*, *358*, 491–500.

Meltzoff, A. N., & Moore, M. K. (1997). Explaining facial imitation: A theoretical model. *Early Development and Parenting*, *17*, 83–99.

Meyer, J., & Hobson, R. P. (2004). Orientation in relation to self and other; The case of autism. *Interaction Studies*, *5*, 221–244.

Mikulineer, M., & Shaver, P. R. (2005). Attachment security, compassion, and altruism. *Current Directions in Psychological Science*, *14*, 34–38.

Miller, P. A., & Eisenberg, N. (1988). The relation of empathy to aggressive and externalizing/antisocial behavior. *Psychological Bulletin*, *103*, 324–344.

Minshew, N. J., Meyer, J., & Goldstein, G. (2002). Abstract reasoning in autism: A dissociation between concept formation and concept identification. *Neuropsychology*, *16*, 327–334.

Moll, J., de Oliviera-Souza, R., & Eslinger, P. (2003). Morals and the human brain. *NeuroReport*, *14*, 299–305.

Morgan, R., & Rochat, P. (1997). Intermodal calibration of the body in early infancy. *Ecological Psychology*, *9*, 1–23.

Moriguchi, Y., Ohnishi, T., Mori, T., Matsuda, H., & Komaki, G. (2007). Changes of brain activity in the neural substrates for theory of mind in childhood and adolescence. *Psychiatry and Clinical Neuroscience*, *61*, 355–363.

Muthukumaraswamy, S. D., Johnson, B. W., & McNair, N. A. (2004). Mu rhythm modulation during observation of an object-directed grasp. *Cognitive Brain Research*, *19*, 195–201.

Nielsen, L. (2002). The simulation of emotion experience: On the emotional foundations of theory of mind. *Phenomenology and the Cognitive Sciences*, *1*, 255–286.

Neisser, U. (1991). Two perceptually given aspects of the self and their development. *Developmental Review*, *11*, 197–209.

Nishitani, N., Avikainen, S., & Hari, R. (2004). Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Annals of Neurology*, *55*, 558–562.

Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Cognitive Brain Research*, *24*, 190–198.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Science*, *9*, 242–249.

Ohta, M. (1987). Cognitive disorders of infantile autism: A study employing the WISC, spatial relationships, conceptualization, and gestural imitation. *Journal of Autism and Developmental Disorders*, *17*, 45–62.

Ozonoff, S., & McEvoy, R. E. (1994). A longitudinal study of executive function and theory of mind development in autism. *Development and Psychopathology*, *6*, 415–431.

Ozonoff, S., Pennington, B. F., & Rogers, S. J. (1991). Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry*, *32*, 1081–1105.

Panksepp, J. (1986). The neurochemistry of behavior. *Annual Review of Psychology*, *37*, 77–107.

Paus, T., Zijdenbos, A., Worsley, K., Collins, D. L., Blumenthal, J., Giedd, J. N., et al. (1999). Structural maturation of neural pathways in children and adolescents: In vivo study. *Science 283*, 1908–1911.

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage—A polyvagal theory. *Psychophysiology 32*, 301–318.

Porges, S. W. (1996). Physiological regulation in high-risk infants: A model for assessment and potential intervention. *Development and Psychopathology*, *8*, 43–58.

Posner, M. I., & Rothbart, M. K. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, *12*, 427–441.

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

Price, B. H., Daffner, K. R., Stowe, R. M., & Mesulam, M. M. (1990). The comportmental learning-disabilities of early frontal lobe damage. *Brain*, *113*, 1383–1393.

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

Radke-Yarrow, M., & Zahn-Waxler, C. (1984). Roots, motives, and patterning in children's prosocial behavior. In E. Staub, K. D. Bartal, J. Karylowski & J. Raykowski (Eds.), *The development and maintenance of prosocial behavior: International perspectives on positive morality* (pp. 81–99). New York: Plenum Press.

Raichle, M. E., MacLeod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2000). A default mode of brain function. *Proceedings of the National Academy of Sciences of the United States of America*, *98*, 676–682.

Raine, A. (1996). Autonomic nervous system factors underlying disinhibited, antisocial, and violent behavior: Biosocial perspectives and treatment implications. *Annals of the New York Academy of Sciences*, *794*, 46–59.

Raine, A., Venables, P., Mednick, S., & Sarnoff, A. (1997). Low resting heart rate at age 3 years predisposes to aggression at age 11 years: Evidence form the Mauritius child health project. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*, 1457–1464.

Raine, A., & Yang, Y. (2006). The anatomical bases of psychopathy: A review of brain imaging findings: In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 278–295). New York: Guilford Press.

Repacholi, B. M., & Gopnik, A. (1997). Early reasoning about desires: Evidence from 14–18-months-olds. *Developmental Psychology*, *33*, 12–21.

Rizzolatti, G., Fogassi, L., & Gallese, V. (2001). Neurophysiological mechanisms underlying the understanding and the imitation of action. *Nature Review Neuroscience*, *2*, 661–670.

Rochat, P. (1999). *Early social cognition*. Mahwah, NJ: Erlbaum.

Rochat, P. (2002). *The infant's world*. Cambridge, MA: Harvard University Press.

Rochat, P., & Hespos, S. J. (1997a). Differential rooting response by neonates: Evidence for an early sense of self. *Early Development & Parenting*, *6*, 105–112.

Rochat, P., & Hespos, S. J. (1997b). Social–cognitive development in the first year. In P. Rochat (Ed.), *Early social cognition* (pp. 3–34). Mahwah, NJ: Erlbaum.

Rochat, P., & Striano, T. (1999). Social cognitive development in the first year. In P. Rochat (Ed.), *Early social cognition* (pp. 3–34). Mahwah, NJ: Erlbaum.

Rochat, P., & Striano, T. (2000). Perceived self in infancy. *Infant Behavior & Development*, *23*, 513–530.

Rochat, P., & Striano, T. (2002). Who's in the mirror? Self-other discrimination in specular images by four- and nine-month-old infants. *Child Development*, *73*, 35–46.

Rogeness, G. A., Cepeda, C., Macedo, C., Fischer, C., & Harris, W. (1990). Differences in heart rate and blood

pressure in children with conduct disorder, major depression, and separation anxiety. *Psychiatry Research*, *33*, 199–206.

Rogers, S. J. (1999). An examination of the imitation deficit in autism: The roles of imitation and executive function. In J. Nadel & G. Butterworth (Eds.), *Imitation in infancy* (pp. 254–283). New York: Cambridge University Press.

Rothbart, M. K., Ahadi, S. A., & Hershey, K. L. (1994). Temperament and social behavior in childhood. *Merrill–Palmer Quarterly*, *40*, 21–39.

Ruby, P., & Decety, J. (2001). Effect of subjective perspective taking during simulation of action: A PET investigation of agency. *Nature Neuroscience*, *4*, 546–550.

Ruby, P., & Decety, J. (2004). How would you feel versus how do you think she would feel? A neuroimaging study of perspective-taking with social emotions. *Journal of Cognitive Neuroscience*, *16*, 988–999.

Russell, J. (1996). *Agency and its role in mental development*. Hove: Psychology Press.

Sagi, A., & Hoffman, M. L. (1976). Empathic distress in the newborn. *Developmental Psychology*, *12*, 175–176.

Saxe, R., & Wexler, A. (2005). Making sense of another mind: The role of the right temporo-parietal junction. *Neuropsychologia*, *43*, 1391–1399.

Schulkin, J. (2005). Moral sensibility, visceral representations, and social cohesion: A behavioral neuroscience perspective. *Mind and Matter*, *26*, 31–56.

Shamay-Tsoory, S. G., Tomer, R., Yaniv, S., & Aharon-Peretz, J. (2002). Empathy deficits in Asperger syndrome: A cognitive profile. *Neurocase*, *8*, 245–252.

Sigman, M. D., Kasri, C., Kwon, J., & Yirmiya, N. (1992). Responses to the negative emotions of others by autistic, mentally retarded and normal children. *Child Development*, *63*, 797–807.

Silk, T. J., Rinehart, N., Bradshaw, J. L., Tonge, B., Egan, G., et al. (2006). Visuospatial processing and the function of prefrontal-parietal networks in autism spectrum disorders: A functional MRI study. *The American Journal of Psychiatry*, *163*, 1440–1443.

Smith, I. M., & Bryson, S. E. (1998). Gesture Imitation in autism I: Non symbolic postures and sequences. *Cognitive Neuropsychology*, *15*, 747–770.

Sommerville, J. A., & Decety, J. (2006). Weaving the fabric of social interaction: Articulating developmental psychology and cognitive neuroscience in the domain of motor cognition. *Psychonomic Bulletin & Review*, *13*, 179–200.

Sowell, R. E., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, *2*, 859–861.

Spence, S. A., Brooks, D. J., Hirsch, S. R., Liddle, P. F., Meehan, J., & Grasby, P. M. (1997). A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain*, *120*, 1997–2011.

Stotland, E. (1969). Exploratory investigations of empathy. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 4, pp. 271–314). New York: Academic Press.

Sturm, V. E., Rosen, H. J., Allison, S., Miller, B. L., & Levenson, R. W. (2006). Self-conscious emotions deficits in frontotemporal lobar degeneration. *Brain*, *129*, 2508–2516.

Takahashi, T., Shirane, R., Sato, S., & Yoshimoto, T. (1999). Developmental changes of cerebral blood flow and oxygen metabolism in children. *American Journal of Neuroradiology*, *20*, 917–922.

Tamm, L., Menon, V., & Reiss, A.L. (2002). Maturation of brain function associated with response inhibition. *Journal of American Children and Adolescent Psychiatry*, *41*, 1231–1238.

Tetlock, P. E. (1985). Accountability: A social check on the fundamental attribution error. *Social Psychology Quarterly*, *48*, 227–236.

Theoret, H., Halligan, E., Kobayashi, M., Fregni, F., Tager-Flusberg, H., & Pascual-Leone, A. (2005). Impaired motor facilitation during action observation in individuals with autism spectrum disorder. *Current Biology*, *15*, 84–85.

Trevarthen, C., & Aitken, K. J. (2001). Infant intersubjectivity: Research, theory, and clinical applications. *Journal of Child Psychiatry*, *42*, 3–48.

Tronick, E. Z., & Weinberg, M. K. (1997). Depressed mothers and infants: Failure to form dyadic states of consciousness. In L. Murray & P. J. Cooper (Eds.), *Postpartum Depression and Child Development* (pp. 54–81). New York: Guilford Press.

Ungerer, J. A., Dolby, R., Waters, B., Barnett, B., Kelk, N., & Lewin, V. (1990). The early development of empathy: Self-regulation and individual differences in the first year. *Motivation and Emotion*, *14*, 93–106.

Villalobos, M. E., Mizuno, A., Dahl, B. C., Kemmotsu, N., & Muller, R. A. (2004). Reduced functional connectivity between V1 and inferior frontal cortex associated with visuomotor performance in autism. *NeuroImage*, *25*, 916–925.

Walton, G. E., Bower, N. J. A., & Bower, T. G. R. (1992). Recognition of familiar faces by newborns. *Infant Behavior and Development*, *15*, 265–269.

Weinberg, M. K., & Tronick, E. Z. (1996). Infant affective reactions to the resumption of maternal interaction after the still-face. *Child Development*, *67*, 905–914.

Wilson, P. H., & McKenzie, B. E. (1998). Information processing deficits associated with developmental coordination disorder: A meta-analysis of research findings. *Journal of Child Psychology and Psychiatry*, *39*, 829–840.

Woodward, A. L., Sommerville, J. A., & Guajardo, J. J. (2001). How infants make sense of intentional action. In B. Malle, L. Moses, & D. Baldwin (Eds.), *Intentions and intentionality: Foundations of social cognition* (pp. 149–169). Cambridge, MA: MIT Press.

Zahn-Waxler, C., Cole, P. M., Welsh, J. D., & Fox, N. A. (1995). Psychophysiological correlates of empathy and prosocial behaviors in preschool children with behavior problems. *Development and Psychopathology*, *7*, 27–48.

Zahn-Waxler, C., & Radke-Yarrow, M. (1990). The origins of empathic concern. *Motivation and Emotion*, *14*, 107–130.

Zald, D. H. (2003). The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research Reviews*, *41*, 88–123.

Zelazo, P. D. (2004). The development of conscious control in childhood. *Trends in Cognitive Sciences*, *8*, 12–17.

Zelazo, P. D., Craik, F. I., & Booth, L. (2004). Executive function across the life span. *Acta Psychologica*, *115*, 167–183.