

available at www.sciencedirect.comwww.elsevier.com/locate/brainres**BRAIN
RESEARCH**

Research Report

Intentional attunement: A neurophysiological perspective on social cognition and its disruption in autism

Vittorio Gallese

Dipartimento di Neuroscienze, Università di Parma, Via Volturmo 39, 43100 Parma, Italy

ARTICLE INFO

Article history:

Accepted 11 January 2006

Keywords:

Autism
Embodied simulation
Intentional attunement
Mirror neuron
Shared manifold
Social cognition

ABSTRACT

A direct form of experiential understanding of others, “intentional attunement”, is achieved by modeling their behavior as intentional experiences on the basis of the activation of shared neural systems underpinning what the others do and feel and what we do and feel. This modeling mechanism is embodied simulation. In parallel with the detached sensory description of the observed social stimuli, internal representations of the body states associated with actions, emotions, and sensations are evoked in the observer, as if he/she would be doing a similar action or experiencing a similar emotion or sensation. Mirror neuron systems are likely the neural correlate of this mechanism. By means of a shared neural state realized in two different bodies, the “objectual other” becomes “another self”. A defective intentional attunement caused by a lack of embodied simulation might cause some of the social impairments of autistic individuals.

© 2006 Elsevier B.V. All rights reserved.

1. Introduction

In primates species, the relationship between social complexity and cognition is well established. [Humphrey \(1976\)](#) originally suggested that the intelligence of primates primarily evolved to solve social problems. This view is supported by empirical data. Several studies revealed the unique capacity of non-human primates of understanding the quality of the relationships within their social group, not only in terms of kin, but also in terms of coalitions, friendship, and alliances. The capacity to understand conspecifics' behaviors as goal-related provides considerable benefits to individuals, as they can predict others' actions. The advantage of such a cognitive skill would allow individuals also to influence and manipulate the behavior of conspecifics (see the Machiavellian Intelligence hypothesis [Whiten and Byrne, 1997](#)) or to achieve better social cooperation within a group.

As pointed out by [Tomasello and Call \(1997\)](#), primates can categorize and understand third-party social relationships.

The evolution of this cognitive trait seems to be related to the necessity to deal with social complexities that arose when group-living individuals had to compete for scarce and patchily distributed resources. [Dunbar \(1992\)](#) posited a relationship between primates' group size and the degree of expansion of the neocortex. The increase of social group complexity exerted a powerful pressure for the development of more sophisticated cognitive skills.

The problem of intentionality in primates was almost simultaneously and independently raised by [Humphrey \(1978, 1980\)](#) and [Premack and Woodruff \(1978\)](#). The traditional view in the cognitive sciences holds that human beings are able to understand the behavior of others in terms of their mental states by exploiting what is commonly designated as “Folk Psychology”. The capacity for attributing mental states—intentions, beliefs, and desires—to others has been defined Theory of Mind (ToM, [Premack and Woodruff, 1978](#)). The attributes of “Folk Psychology” have been thus basically identified with the notion of Theory of Mind ([Carruthers and](#)

E-mail address: vittorio.gallese@unipr.it.

Smith, 1996). A common trend on this issue has been to emphasize that non-human primates, apes included, do not rely on mentally based accounts for others' behavior (Hayes, 1998; Povinelli et al., 2000). According to this perspective, social cognition becomes almost synonymous of mind reading abilities.

The dichotomous account of primate social cognition based on a sharp evolutionary discontinuity between species of behavior readers (non-human primates) and one species of mind readers (humans) appears though over simplistic.

As recently pointed out by Barrett and Henzi (2005, p. 1866), this traditional approach is "...heavily oriented toward a particular model of cognition that focuses solely on internal mental representations, whereas recent work in cognitive science and neurobiology argues for a more distributed and embodied approach".

In the present paper, I propose a different approach. I submit that social cognition is not only "social metacognition", that is, explicitly thinking about the contents of someone else's mind by means of abstract representations. There is also an experiential dimension of interpersonal relationships, which enables a direct grasping of the *sense* of the actions performed by others, and of the emotions, and sensations they experience. This dimension of social cognition is embodied in that it mediates between the multimodal experiential knowledge we hold of our lived body and the experience we make of others.

I proposed that our capacity to share experiences with others rests on the constitution of a shared meaningful interpersonal space. This "shared manifold" (Gallese, 2001, 2003, 2005a) can be characterized at the functional level as embodied simulation (Gallese, 2005a), a specific mechanism by means of which our brain/body system models its interactions with the world (for a similar account of the mechanisms at the basis of empathy, see Preston and De Waal, 2002). I submit that embodied simulation constitutes a crucial functional mechanism in social cognition.

The self/other distinction in my opinion is not the most difficult problem in social cognition, neither from a theoretical, nor from an empirical point of view. The "hard problem" in social cognition is to understand how the epistemic gulf separating single individuals can be overcome. The solipsistic attitude, inspired by Folk Psychology and purported by the approach of classic cognitive science, leaves this hard problem unsolved. Recent neuroscientific evidence suggests alternative answers. Here, I discuss this evidence and provide a theoretical framework for its interpretation. Before doing so, I want to briefly discuss the problem of social cognition from an ontogenetic point of view.

2. The ontogenesis of social cognition

The notion of "social cognition" sounds almost as a pleonasm, since from an ontogenetic point of view, the relationship between cognition and the social dimension is intrinsically tight.

At the very onset of our life, interpersonal relations are established when a full-blown self-conscious subject of

experience is not yet constituted. However, the absence of a subject does not preclude the presence of a primitive "we-centric space". The infant shares this space with others. Few hours after birth, neonates display facial imitation (Meltzoff and Brooks, 2001). Furthermore, empirical research has shown that mothers and infants systematically engage in mutually coordinated activities during which their movements, facial expressions, and voice intonation synchronize in time (Reddy et al., 1997). From 4 months of age onwards, infants and mothers show protodialogic behaviors in which they time their behavior in a bidirectional coordinated way (Trevarthen, 1979). Approximately at the same age, infants become sensitive to social contingencies (Striano et al., 2005). According to Daniel Stern (1985/2000), this evidence suggests that such protodialogic behaviors enable mother and infant to establish an affective attunement by means of which inner feeling states can be shared.

According to my hypothesis, the shared we-centric space enables the social bootstrapping of cognitive and affective development because it provides a powerful tool to detect and incorporate coherence, regularity, and predictability in the course of the interactions of the individual with the environment. The we-centric space is likely paralleled by the development of perspectival spaces defined by the establishment of the capacity to distinguish self from other, as long as self-control develops. Within each of these newly acquired perspectival spaces, information can be better segregated in discrete channels (visual, somatosensory, etc.) making the perception of the world more finely grained. The concurrent development of language possibly contributes to further segregate single characters or modalities of experience from the original multimodal perceptive world. Yet, the more mature capacity to segregate the modes of interaction, together with the capacity of carving out the subject and the object of the interaction, does not annihilate the shared we-centric space.

In fact, the establishment of a self-centered perspective is paralleled by the creation of an epistemic gap between self and others. The gulf separating self from non-self poses a challenge to any account of intersubjectivity and social cognition. I posit that the intersubjective we-centric space may provide the individual with a powerful tool to help overcome such epistemic gap. If my hypothesis is correct, social identity, the "selfness" we readily attribute to others, the inner feeling of "being-like-you" triggered by our encounter with others, are the result of a preserved shared we-centric space.

The proposition that self-other physical and epistemic interactions are shaped and conditioned by the same type of body and environmental constraints sounds almost as a truism. Less trivial in my opinion is the fact that this common relational character of intersubjectivity is underpinned, at the level of the brain, by shared neural networks, the mirror neuron systems, compressing the "who-done-it", "who-is-it" specifications into a narrower content state. This content specifies what kinds of interaction or state are at stake. A concise overview of the evidence supporting the existence of such shared neural mechanisms will be the focus of the next sections.

3. The mirror neuron system for actions in monkeys and humans: empirical evidence

About 10 years ago, a new class of premotor neurons was discovered in the ventral premotor cortex of the macaque monkey brain. These neurons discharge not only when the monkey executes goal-related hand actions like grasping objects but also when observing other individuals (monkeys or humans) executing similar actions. They were called “mirror neurons” (Gallese et al., 1996; Rizzolatti et al., 1996). Neurons with similar properties were later discovered in a sector of the posterior parietal cortex reciprocally connected with area F5 (Gallese et al., 2002; Rizzolatti and Craighero, 2004; Fogassi et al., 2005).

Action observation causes in the observer the automatic activation of the same neural mechanism triggered by action execution. It has been proposed that this mechanism could be at the basis of a direct form of action understanding (Gallese et al., 1996; Rizzolatti and Craighero, 2004; Rizzolatti et al., 2001).

Further studies carried out by our research group at the Department of Neuroscience of the University of Parma corroborated and extended the original hypothesis. In a paper by Umiltà et al. (2001), it was shown that F5 mirror neurons are also activated when the final critical part of the observed action, that is, the hand–object interaction, is hidden. In a second study, Kohler et al. (2002) showed that a particular class of F5 mirror neurons, “audio-visual mirror neurons”, could be driven not only by action execution and observation but also by the sound produced by the same action.

More recently, the most lateral part of area F5 was explored where a population of mirror neurons related to the execution/observation of mouth actions was described (Ferrari et al., 2003). The majority of these neurons discharge when the monkey executes and observes transitive, object-related ingestive actions, such as grasping object with the mouth, biting, or licking. However, a small percentage of mouth-related mirror neurons discharge during the observation of intransitive, communicative facial actions performed by the experimenter in front of the monkey (“communicative mirror neurons Ferrari et al., 2003). Thus, mirror neurons seem also to underpin aspects of monkeys’ social facial communication.

Several studies using different experimental methodologies and techniques have demonstrated also in the human brain the existence of a mirror neuron system matching action perception and execution. During action observation, there is a strong activation of premotor and parietal areas, the likely human homologue of the monkey areas in which mirror neurons were originally described (Rizzolatti and Craighero, 2004; Rizzolatti et al., 2001; Gallese et al., 2004). Furthermore, the mirror neuron matching system for actions in humans is coarsely organized in a somatotopic fashion, with distinct cortical regions within the premotor and posterior parietal cortices being activated by the observation/execution of mouth-, hand-, and foot-related actions (Buccino et al., 2001).

The involvement of the motor system during observation of communicative mouth actions in humans is testified by the results of recent fMRI and TMS studies (Buccino et al., 2004; Watkins et al., 2003). The observation of communicative, or

speech-related mouth actions, facilitate the excitability of the motor system involved in the production of the same actions.

A recent study carried out by Buxbaum et al. (2005) on posterior parietal patients with Ideomotor Apraxia has shown that they were not only disproportionately impaired in the imitation of transitive as compared to intransitive gestures, but they also showed a strong correlation between imitation deficits and the incapacity of recognizing observed goal-related meaningful hand actions. As argued by the authors of this study, these results further corroborate the notion that the same action representations underpin both action production and action understanding. In the next section, I will introduce new empirical results, suggesting that embodied simulation may play a role even in more complex social cognitive abilities.

4. Mirror neurons and the understanding of intentions

Monkeys may exploit the mirror neuron system to optimize their social interactions. As we have seen in the previous section, audio-visual mirror neurons can be driven not only by action execution and observation but also by the sound produced by the same action (Kohler et al., 2002). This mirroring mechanism can support social facilitation in monkeys. It has been recently shown that the observation and hearing of noisy eating actions facilitates eating behavior in pigtailed macaque monkeys (*Macaca nemestrina*) (Ferrari et al., 2005).

Another recently published study showed that pigtailed macaque monkeys recognize when they are imitated by a human experimenter (Paukner et al., 2005). Pigtailed macaques preferentially look at an experimenter imitating the monkeys’ object-directed actions compared with an experimenter manipulating an identical object but not imitating their actions. Since both experimenters acted in synchrony with the monkeys, the monkeys based this preference not on temporal contingency but took into account the structural components of the experimenters’ actions.

It may well be the case, as repeatedly argued, that macaque monkeys are not able of motor imitation—though recent evidence show that they are capable of cognitive imitation (Subiaul et al., 2004). The study by Paukner et al. (2005) nevertheless shows that macaque monkeys do entertain the capacity to discriminate between very similar observed goal-related actions on the basis of their degree of similarity with the goal-related actions the monkeys themselves have just executed. This capacity seems to be rather cognitively sophisticated in that it implies a certain degree of metacognition relative to the domain of goal-related action.

I posit that macaque monkeys might entertain a rudimentary form of “teleological stance”, a likely precursor of a full-blown intentional stance. This hypothesis extends to the phylogenetic domain the ontogenetic scenario proposed for human infants (Gergely and Csibra, 2003). New experiments are being designed in my laboratory to test this hypothesis. Monkeys certainly do not entertain full-blown explicit mentalization. Thus, what makes humans different? Language

certainly plays a key role. Though, at present we can only make hypotheses about the relevant neural mechanisms underpinning the mentalizing abilities of humans, still poorly understood from a functional point of view. In particular, we do not have a clear neuroscientific model of how humans can understand the intentions promoting the actions of others they observe.

When an individual starts a movement aimed to attain a goal, such as picking up a pen, he/she has clear in mind what he/she is going to do, for example, writing a note on a piece of paper. In this simple sequence of motor acts, the final goal of the whole action is present in the agent's mind and is somehow reflected in each motor act of the sequence. The action intention, therefore, is set before the beginning of the movements. This also means that when we are going to execute a given action, we can also predict its consequences. But a given action can be originated by very different intentions. Suppose one sees someone else grasping a cup. Mirror neurons for grasping will most likely be activated in the observer's brain. The direct matching between the observed action and its motor representation in the observer's brain, however, can only tell us what the action is (it is a grasp) and not why the action occurred. This has led to argue against the relevance of mirror neurons for social cognition and, in particular, for determining the intentions of others (Jacob and Jeannerod, 2005).

But what is an action intention? Determining why action A (grasping the cup) was executed, that is, determining its intention, can be equivalent to detecting the goal of the still not executed and impending subsequent action (say, drink from the cup). In a recently published fMRI study (Iacoboni et al., 2005), we tried to experimentally address these issues. Subjects watched three kinds of stimuli: grasping hand actions without a context, context only (a scene containing objects), and grasping hand actions embedded in contexts. In the latter condition, the context suggested the intention associated with the grasping action (either drinking or cleaning up). Actions embedded in contexts, compared with the other two conditions, yielded a significant signal increase in the posterior part of the inferior frontal gyrus and the adjacent sector of the ventral premotor cortex where hand actions are represented. Thus, premotor mirror areas—areas active during the execution and the observation of an action—previously thought to be involved only in action recognition—are actually also involved in understanding the “why” of action, that is, the intention promoting it.

Another interesting result of this study is that being or not being explicitly instructed to determine the intention of the observed actions of others makes no difference in terms of the activation of the premotor mirror areas. This means that—at least for simple actions as those employed in this study—the ascription of intentions occurs by default, and it is underpinned by the mandatory activation of an embodied simulation mechanism.

The neurophysiological mechanism at the basis of the relationship between intention detection and action prediction was recently unveiled. Fogassi et al. (2005) described a class of parietal mirror neurons whose discharge during the observation of an act (e.g., grasping an object) is conditioned by the type of not yet observed subsequent act (e.g., bringing the object to the mouth) specifying the overall action

intention. This study shows that the inferior parietal lobe of the monkey contains mirror neurons discharging in association with monkey motor acts (grasping) only when they are embedded in a specific action aimed at different goals. For example, a neuron discharges when the monkey grasps an object only if the grasping act is aimed at bringing the object into the mouth and not if it is aimed to place it into a cup. It appears therefore that these neurons code the same motor act differently depending on the distal, overarching action goal. Single motor acts are dependent to each other as they participate to the overarching distal goal of an action, thus forming pre-wired intentional chains, in which each next motor act is facilitated by the previously executed one.

The visual response of many of these parietal mirror neurons is similar to their motor response. In fact, they discharge differentially depending on whether the observed grasping is followed by bringing the grasped object to the mouth or by placing it into a cup. It must be emphasized that the neurons discharge before the monkey observes the experimenter starting the second motor act (bringing the object to the mouth or placing it into the cup). This new property of parietal mirror neurons suggests that in addition to recognizing the goal of the observed motor act, they discriminate identical motor acts according to the action in which these acts are embedded. Thus, these neurons not only code the observed motor act but also seem to allow the observing monkey to predict the agent's next action, henceforth, his/her overall intention. It is possible to interpret this mechanism as the neural correlate of the dawning of the sophisticated mentalizing abilities characterizing our species.

The mechanism of intention understanding just described appears to be rather simple: depending on which motor chain is activated, the observer is going to activate the motor schema of what, most likely, the agent is going to do. How can such a mechanism be formed? At present, we can only make speculations. It can be hypothesized that the statistical detection of what actions most frequently follows other actions, as they are habitually performed or observed in the social environment, can constrain preferential paths chaining together different motor schemata. At the neural level, this can be accomplished by the chaining of different populations of mirror neurons coding not only the observed motor act but also those that in a given context would normally follow.

Ascribing simple intentions would therefore consist in predicting a forthcoming new goal. According to this perspective, action prediction and the ascription of intentions are related phenomena, underpinned by the same functional mechanism, embodied simulation. In contrast with what mainstream cognitive science would maintain, action prediction and the ascription of intentions—at least of simple intentions—do not appear to belong to different cognitive realms, but both pertain to embodied simulation mechanisms underpinned by the activation of chains of logically related mirror neurons (Fogassi et al., 2005; Iacoboni et al., 2005).

If this is true, it follows that one important difference between humans and monkeys could be the higher level of recursivity attained by the mirror neuron system for actions in our species. A similar proposal has been recently put forward in relation to the faculty of language by contrasting our species, capable of mastering hierarchically complex “phrase

structure grammars”, with other non-human primate species, confined to the use of much simpler “finite state grammars” (Hauser and Fitch, 2004; Hauser et al., 2002). A quantitative difference in computational power and degree of recursivity could produce a qualitative leap in social cognition.

5. Mirroring emotions and sensations

As already suggested by Charles Darwin (1872), the coordinated activity of sensory-motor and affective neural systems results in the simplification and automatization of the behavioral responses that living organisms are supposed to produce in order to survive. Emotions constitute one of the earliest ways available to the individual to acquire knowledge about its situation, thus enabling a reorganization of this knowledge on the basis of the outcome of the relations entertained with others. The integrity of the sensory-motor system indeed appears to be critical for the recognition of emotions displayed by others (Adolphs, 2003) because the sensory-motor system appears to support the reconstruction of what it would feel like to be in a particular emotion, by means of simulation of the related body state. The implication of this process for empathy should be obvious.

In a recently published fMRI study, we showed that experiencing disgust and witnessing the same emotion expressed by the facial mimicry of someone else both activate the same neural structure—the anterior insula—at the same overlapping location (Wicker et al., 2003). When we see the facial expression of someone else, and this perception leads us to experience a particular affective state, the other’s emotion is constituted, experienced and therefore directly understood by means of an embodied simulation producing a shared body state. It is the activation of a neural mechanism shared by the observer and the observed to enable direct experiential understanding. A similar simulation-based mechanism has been proposed by Goldman and Sripada (2005) as “unmediated resonance”.

Let us now examine somatic sensations as the target of our social perception. As repeatedly emphasized by phenomenology, touch has a privileged status in making possible the social attribution of lived personhood to others. “Let’s be in touch” is a common clause in everyday language, which metaphorically describes the wish of being related, being in contact with someone else. Such examples show how the tactile dimension can be intimately related to the interpersonal dimension.

As predicted by the shared manifold hypothesis (Gallese, 2001, 2003, 2005a), empirical evidence suggests that the first-person experience of being touched on one’s body activates the same neural networks activated by observing the body of someone else being touched (Keysers et al., 2004; Blakemore et al., 2005). This double pattern of activation of the same somatosensory-related brain regions suggests that our capacity to experience and directly understand the tactile experience of others could be mediated by embodied simulation, that is, by the externally triggered activation of some of the same neural networks underpinning our own tactile sensations. The study by Blakemore et al. (2005) actually shows that the *degree of activation* of the same somatosensory areas activated during both the subjective tactile experience and

its observation in others could be an important mechanism enabling the subject to disentangling *who* is being touched. In fact, what this paper shows is that the difference between empathizing with someone else’s tactile sensation, and actually feeling on one’s body the same sensation (as in the case of synesthesia) is only a matter of degrees of activation of the same shared brain areas. These data support the notion that disentangling *who* is *who* (patient vs. observer) does not pose a problem to my hypothesis.

I posit that a similar embodied simulation mechanism underpins our experience of the painful sensations of others. Single neuron recording experiments carried out in awake neurosurgical patients (Hutchison et al., 1999) and fMRI (Singer et al., 2004; Morrison et al., 2004; Jackson et al., 2005; Botvinick et al., 2005) and TMS experiments (Avenanti et al., 2005) carried out in healthy subjects, all show that the same neural structures are activated both during the subjective experience of pain and the direct observation or symbolically mediated knowledge that someone else is likely experiencing the same painful sensation.

It should be noted that the results of the fMRI (Singer et al., 2004; Morrison et al., 2004; Jackson et al., 2005; Botvinick et al., 2005) and TMS (Avenanti et al., 2005) studies show that the overlap of activation in the self/other experience conditions can be modulated in terms of the brain areas involved by the cognitive demands imposed by the type of tasks. When subjects are required to simply watch the painful stimulation experienced by some stranger’s body part (Avenanti et al., 2005), the observer extracts the basic sensory qualities of the pain experienced by others mapping it somatotopically onto his/her own sensory-motor system. However, when subjects are required to imagine the pain suffered by their partner out of their sight (Singer et al., 2004), only brain areas mediating the affective quality of pain (the anterior cingulate cortex and the anterior insula) are activated. In particular, the anterior cingulate cortex (ACC) appears to mediate the affective dimension of pain processing and the motivational aspects of response selection. Thus, in the context of the perception of pain, ACC could play a role in linking events with outcomes, allowing the prediction and avoidance of noxious stimuli (see Singer et al., 2004). As it has been convincingly argued (Singer and Frith, 2005), one can conclude that the particular mental attitude of individuals could be the key variable determining the degree and quality of the activation of shared neural circuits when experiencing the sensations of others, like in the case of pain.

6. Intentional attunement, embodied simulation, and empathy

Some functional mechanism must mediate between the multimodal experiential knowledge we hold of our lived body and the experience we make of others. Such body-related experiential knowledge enables a direct grasping of the sense of the actions performed by others and of the emotions and sensations they experience.

When confronting the intentional behavior of others, we experience a specific phenomenal state of “intentional attunement”. This phenomenal state generates a peculiar

quality of familiarity with other individuals, produced by the collapse of the others' intentions into the observer's ones. This seems to be one important component of what being empathic is about (for a recent similar account of empathy, see Preston and De Waal, 2002).

Of course, self-other identity is not all there is in empathy. Empathy, at difference with emotional contagion, entails the capacity to experience what others do experience while being able to attribute these shared experiences to *others* and not to the self. The quality of our lived experience of the external world and its content are constrained by the presence of other subjects that are intelligible while preserving their alterity character. This alterity is in fact also evident at the sub-personal level, instantiated by the different neural networks coming into play and/or by their different degree of activation, when *I* act with respect to when others act, or when *I* experience an emotion or a sensation with respect when others do the same.

When we confront the intentional behavior of others, according to my hypothesis, embodied simulation, a specific mechanism by means of which our brain/body system models its interactions with the world, generates a specific phenomenal state of "intentional attunement". This phenomenal state in turn generates a peculiar quality of familiarity with other individuals, produced by the collapse of the others' intentions into the observer's ones. Embodied simulation constitutes a crucial functional mechanism in social cognition, and it can be neurobiologically characterized. The different mirror neuron systems represent its sub-personal instantiations. By means of embodied simulation, we do not just "see" an action, an emotion, or a sensation. Side by side with the sensory description of the observed social stimuli, internal representations of the body states associated with these actions, emotions, and sensations are evoked in the observer, "as if" he/she would be doing a similar action or experiencing a similar emotion or sensation.

Any intentional relation can be mapped as a relation between a subject and an object. The mirror neuron matching systems described in this paper map the different intentional relations in a fashion that is neutral about the specific quality or identity of the agentive/subjective parameter. By means of a shared functional state realized in two different bodies that nevertheless obey to same functional rules, the "objectual other" becomes "another self".

Embodied simulation is probably not the only functional mechanism underpinning social cognition. The meaning of social stimuli can also be decoded on the basis of the explicit cognitive elaboration of their contextual perceptual features, by exploiting previously acquired knowledge about relevant aspects of the situation to be analyzed. Our capacity of attributing false beliefs to others, our most sophisticated metacognitive mind reading abilities, likely involves the activation of large regions of our brain, certainly larger than a putative and domain-specific Theory of Mind Module. These brain sectors certainly encompass the sensory-motor system. In fact, it has been shown that brain areas reportedly active during mind reading tasks, such as the Superior Temporal Sulcus (STS) region and the paracingulate cortex, also activate in a simple action prediction task (Ramnani and Miall, 2004).

It must be stressed that the use in social transactions of the belief-desire propositional attitudes of Folk Psychology is probably overstated (Hutto, 2004). As emphasized by Bruner (1990, p. 40), "When things are as they should be, the narratives of Folk Psychology are unnecessary". Furthermore, recent evidence shows that 15-month-old infants understand false beliefs (Onishi and Baillargeon, 2005). These results seem to suggest that typical aspects of mind reading, like the attribution of false beliefs to others, can be explained on the basis of low-level mechanisms, which develop well before full-blown linguistic competence.

The idea put forward by Gergely and Csibra (2003) that the intentional stance can be anticipated by a much earlier developed "teleological stance" is consistent with this view. It should be added, though, that non-mentalistic accounts of the so-called "teleological stance" displayed by young infants are possible (e.g., by relying on mechanisms such as attention, physical causality, stimulus relevance, and the like), thus corroborating the notion that the role of propositional attitudes in social cognition is exaggerated, and that they are much less frequently employed than what assumed by classic cognitive science. A target for future research will be to determine how embodied simulation, which is experience-based, and probably the most ancient mechanism from an evolutionary point of view, may scaffold more sophisticated, language-mediated forms of mind reading.

A possibility is that embodied simulation mechanisms might be crucial in the course of the long learning process required to become fully competent in how to use propositional attitudes, like during the repetitive exposure of children to the narration of stories (for a putative role of narrative practices in the development of a competent use of Folk Psychology, see Hutto, 2004). In fact, embodied simulation is certainly at play during language processing (Gallese, 2005a; Rizzolatti and Craighero, 2004; Gallese and Lakoff, 2005).

If intentional attunement does indeed play a crucial role in intersubjectivity, one should expect its deficit to cause problems at various levels in social cognition. In the next section, I explore the possibility of applying the intentional attunement hypothesis to interpret pathological aspects of social cognition, as those exemplified by the Autistic Spectrum Disorder (ASD).

7. Psychopathological implications of intentional attunement: the autistic spectrum disorder as a deficit of embodied simulation

The Autistic Spectrum Disorder (ASD) is a severe and chronic developmental disorder, characterized by social and communicative deficits and by a reduced interest for the environment, towards which restricted and often stereotyped initiatives are taken (Dawson et al., 2002). To be an autistic child means, with variable degrees of severity, to be incapable to establish meaningful social communications and bonds, to establish visual contact with the world of others, to share attention with the others, to be incapable to imitate others' behavior or to understand others' intentions, emotions, and sensations.

Let us briefly focus on some of the early onset symptoms. Towards the end of the first year of life, autistic children

experience difficulties or even the impossibility to orient on the basis of cues provided by others. They are incapable to share attention with others and are incapable to react in a congruent fashion to others' emotions. They are also highly impaired in recognizing human faces or in displaying imitative behaviors. All of these early manifestations of autism share a common root: the cognitive skills required to establish meaningful bonds with others are missing or seriously impaired.

My hypothesis is that these deficits, like those observed in the related Asperger Syndrome, are to be ascribed to a deficit or malfunctioning of "intentional attunement" because of a malfunctioning of embodied simulation mechanisms, in turn produced by a dysfunction of the mirror neuron systems. If it is true—as held throughout the paper—that at the basis of our social competence is in primis the capacity to constitute an implicit and directly shared we-centric space, enabling us to establish a link with the multiple intentional relations instantiated by others, then it follows that a disruption of this shared manifold and the consequent incapacity to develop a full and comprehensive intentional attunement with others could be the core problems of the autistic mind.

The lack of a full-blown intentional attunement will produce various and diversified cognitive and executive deficits, all sharing the same functional origin: a lack or malfunctioning of embodied simulation routines, likely underpinned by impairments in connectivity and/or functioning of the mirror neuron system. If my hypothesis is correct, the posited intentional attunement deficit should become manifest at the various levels of social cognition it normally underpins. A series of experimental data seem to suggest this to be the case.

A recent study investigating postural adjustments in autistic children has shown that at difference with healthy individuals, they use motor strategies basically relying on feed-back information, rather than on feed-forward modes of control. Such disturbance of executive control strategies prevents autistic children to adopt anticipatory postural adjustments (Schmitz et al., 2003). Given the functional characterization of embodied simulation as an anticipatory mechanism, it is difficult not to interpret these data as evidence of a simulation deficit. Such postural deficits are not intrinsically social, however, they could stem from a disruption within the executive control domain of a functional mechanism—embodied simulation—which I proposed to be at the root of the constitution of the we-centric experiential interpersonal space.

Two recent studies employing different techniques such as EEG (Oberman et al., 2005) and Transcranial magnetic Stimulation (Theoret et al., 2005) show that individuals with ASD might be suffering an action simulation deficit induced by a dysfunction of their mirror system for action. The study by Oberman et al. (2005) showed that ASD individuals, at difference with healthy controls, did not show mu frequency suppression over the sensory-motor cortex during action observation. The study by Theoret et al. (2005) showed that, again at difference with healthy controls, ASD individuals did not show TMS-induced hand muscle facilitation during hand action observation.

A further indication of embodied simulation deficits in ASD is exemplified by imitation deficits. Autistic children have problems in both symbolic and non-symbolic imitative behaviors, in imitating the use of objects, in imitating facial gestures, and in vocal imitation (Rogers, 1999). These deficits characterize both high- and low-functioning forms of autism. Furthermore, imitation deficits are apparent not only in comparison with the performances of healthy subjects but also with those of mentally retarded non-autistic subjects. According to my hypothesis, imitation deficits in autism are determined by the incapacity to establish a motor equivalence between demonstrator and imitator, most likely due to a malfunctioning of the mirror neuron system, and/or because of a disrupted emotional/affective regulation of the same system. Imitation deficits thus can be hypothesized to be further examples of a disrupted shared manifold.

Let me now briefly turn to emotional-affective deficits. Several studies reported the severe problems autistic children experience in the facial expression of emotions and its understanding in others (Snow et al., 1988; Yirmiya et al., 1989; Hobson et al., 1988, 1989). In a recent fMRI study, Dapretto et al. (2006) specifically investigated the neural correlates of the capacity of imitating the facial expressions of basic emotions in high-functioning ASD individuals. The results of this study showed that during observation and imitation autistic children did not show activation of the mirror neuron system in the pars opercularis of the inferior frontal gyrus. It should be emphasized that activity in this area was inversely related with symptom severity in the social domain. The authors of this study concluded, "...a dysfunctional mirror neuron system may underlie the social deficits observed in autism". Furthermore, McIntosh et al. (in press) recently showed that individuals with ASD, at difference with healthy controls, do not show automatic mimicry of the facial expression of basic emotions, as revealed by EMG recordings. Hobson and Lee (1999) reported that autistic children score much worse than healthy controls in reproducing the affective qualities of observed actions.

I posit that all these deficits can be explained as instantiations of intentional attunement deficits produced by defective embodied simulation, likely underpinned by a malfunctioning of the mirror neuron systems. This hypothesis is further corroborated by the recent finding that the brains of ASD individuals show abnormal thinning of the grey matter in cortical areas known as being part of the mirror neuron system (Hadjikhani et al., 2005), such as ventral premotor, posterior parietal, and superior temporal sulcus cortices. Interestingly, cortical thinning of the mirror neuron system correlated with ASD symptoms severity.

My hypothesis to interpret the ASD as an intentional attunement deficit is quite divergent from many of the mainstream ideas concerning the origin of this developmental disorder. One of the most credited theories, in spite of its different—not always congruent—articulations, posits that ASD and the related Asperger syndrome are caused by a deficit of a specific mind module, the Theory of Mind module, selected in the course of evolution to build theories about the mind of others (Baron-Cohen, 1988; Baron-Cohen et al., 1985a). This theory is at present totally untenable, even more so in the light of recent empirical results. A recent study carried out on

a patient who suffered a focal bilateral lesion of the Anterior cingulate cortex (ACC), previously identified as the candidate site for the Theory of Mind Module, showed no evidence of mind reading deficits (Bird et al., 2004).

A further problem of the mainstream theory of ASD as a deficit Theory of Mind is that it can be hardly reconciled with what we learn from the reports of high-functioning autistics or Asperger individuals like Temple Grandin (1995). They claim that in order to understand how they supposedly should feel in given social contexts, and what others supposedly feel and think in those same contexts, they must rely on theorizing. What these reports seem to suggest is that, as I argued elsewhere (Gallese, 2001), theorizing about the others' mind is not quite the basic deficit of ASD or of Asperger syndrome, but the only compensating strategy available in the absence of more elementary and basic cognitive skills enabling a direct experiential take on the world of others.

8. Conclusions

The shared manifold of intersubjectivity and the intentional attunement it generates constitute a general hypothesis on many aspects of social cognition (Gallese, 2005a,b) that as we have seen has already empirical grounding at multiple levels, both in healthy and sociopath individuals. Furthermore, this proposal and the approaches it generates have the merit of disclosing the possibility to establish more insightful therapeutic bonds with patients suffering impairments in social cognition and, more generally, in any psychotherapeutic setting.

The appeal of such a scenario consists not only in its parsimony, but also in providing for the first time a neurophysiological mechanism at the basis of the activation of many of the cortical regions involved in social cognition. We cannot be satisfied of merely knowing that a given cortical area is active during social cognitive tasks. In doing so, we face the risk of resurrecting Phrenology. Social cognitive neuroscience must seek to know *why*, *how*, and because of *which functional mechanism* a particular brain area or cortical network happens to be activated in a particular social cognitive task.

Of course, embodied simulation and its neural underpinnings fall short of providing a thorough account of what is implied in our sophisticated mentalizing skills. What the results presented here indicate though is that embodied mechanisms involving the activation of the sensory-motor system seem to play a major role in social cognition. This should persuade us that the automatic translation of the tenets of Folk Psychology into brain modules or circuitries specifically dedicated to mind reading abilities should be carefully scrutinized as it might be a very poor epistemic strategy to disclose what social cognition really is.

Acknowledgments

This work was supported by MIUR and, as part of the European Science Foundation EUROCORES Programme OMLL, was supported by funds to V.G. from the Italian C.N.R.

REFERENCES

- Adolphs, R., 2003. Cognitive neuroscience of human social behaviour. *Nat. Rev., Neurosci.* 4 (3), 165–178.
- Avenanti, A., Buetti, D., Galati, G., Aglioti, S.M., 2005. Transcranial magnetic stimulation highlights the sensorimotor side of empathy for pain. *Nat. Neurosci.* 8, 955–960.
- Baron-Cohen, S., 1988. Social and pragmatic deficits in autism: cognitive or affective? *J. Autism Dev. Disord.* 18, 379–402.
- Baron-Cohen, S., Leslie, A.M., Frith, U., 1985a. Does the autistic child have a “Theory of Mind”? *Cognition* 21, 37–46.
- Barrett, L., Henzi, P., 2005. The social nature of primate cognition. *Proc. R. Soc. Biol.* 272, 1865–1875.
- Bird, C.M., Castelli, C., Malik, O., Frith, U., Husain, M., 2004. The impact of extensive medial frontal lobe damage on ‘Theory of Mind’ and cognition. *Brain* 127, 914–928.
- Blakemore, S.-J., Bristow, D., Bird, G., Frith, C., Ward, J., 2005. Somatosensory activations during the observation of touch and a case of vision–touch synaesthesia. *Brain* 128, 1571–1583.
- Botvinick, M., Jha, A.P., Bylsma, L.M., Fabian, S.A., Solomon, P.E., Prkachin, K.M., 2005. Viewing facial expressions of pain engages cortical areas involved in the direct experience of pain. *NeuroImage* 25, 315–319.
- Bruner, J., 1990. *Acts of Meaning*. Harvard University Press, Cambridge, MA.
- Buccino, G., Binkofski, F., Fink, G.R., Fadiga, L., Fogassi, L., Gallese, V., Seitz, R.J., Zilles, K., Rizzolatti, G., Freund, H.-J., 2001. Action observation activates premotor and parietal areas in a somatotopic manner: an fMRI study. *Eur. J. Neurosci.* 13, 400–404.
- Buccino, G., Lui, F., Canessa, N., Patteri, I., Lagravinese, G., Benuzzi, F., Porro, C.A., Rizzolatti, G., 2004. Neural circuits involved in the recognition of actions performed by nonconspecifics: an fMRI study. *J. Cogn. Neurosci.* 16, 114–126.
- Buxbaum, L.J., Kyle, K.M., Menon, R., 2005. On beyond mirror neurons: Internal representations subserving imitation and recognition of skilled object-related actions in humans. *Brain Res. Cogn. Brain Res.* 25 (1), 226–239.
- Carruthers, O., Smith, P.K. (Eds.), 1996. *Theories of Theories of Mind*. Cambridge Univ. Press, Cambridge.
- Dapretto, L., Davies, M.S., Pfeifer, J.H., Scott, A.A., Sigman, M., Bookheimer, S.Y., Iacoboni, M., 2006. Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. *Nat. Neurosci.* 9, 28–30.
- Darwin, C., 1872. *The Expressions of the Emotions in Man and Animals*. Albemarle, London.
- Dawson, G., Webb, S., Schellenberg, G.D., Dager, S., Friedman, S., Aylward, E., Richards, T., 2002. Defining the broader phenotype of autism: genetic, brain, and behavioral perspectives. *Dev. Psychopathol.* 14, 581–611.
- Dunbar, R.I.M., 1992. Neocortex size as a constraint on group size in primates. *J. Hum. Evol.* 20, 469–493.
- Ferrari, P.F., Gallese, V., Rizzolatti, G., Fogassi, L., 2003. Mirror neurons responding to the observation of ingestive and communicative mouth actions in the monkey ventral premotor cortex. *Eur. J. Neurosci.* 17, 1703–1714.
- Ferrari, P.F., Maiolini, C., Addessi, E., Fogassi, L., Visalberghi, E., 2005. The observation and hearing of eating actions activates motor programs related to eating in macaque monkeys. *Behav. Brain Res.* 161, 95–101.
- Fogassi, L., Ferrari, P.F., Gesierich, B., Rozzi, S., Chersi, F., Rizzolatti, G., 2005. Parietal lobe: from action organization to intention understanding. *Science* 302, 662–667.
- Gallese, V., 2001. The “shared manifold” hypothesis: from mirror neurons to empathy. *J. Conscious. Stud.* 8 (5–7), 33–50.
- Gallese, V., 2003. The manifold nature of interpersonal relations:

- the quest for a common mechanism. *Philos. Trans. R. Soc. London, Ser. B Biol. Sci.* 358, 517–528.
- Gallese, V., 2005a. Embodied simulation: from neurons to phenomenal experience. *Phenom. Cogn. Sci.* 4, 23–48.
- Gallese, V., 2005b. “Being like me”: self–other identity, mirror neurons and empathy. In: Hurley, S., Chater, N. (Eds.), *Perspectives on Imitation: From Cognitive Neuroscience to Social Science*, vol. 1. MIT Press, Cambridge, MA, pp. 101–118.
- Gallese, V., Lakoff, G., 2005. The brain’s concepts: the role of the sensory-motor system in reason and language. *Cogn. Neuropsychol.* 22, 455–479.
- Gallese, V., Fadiga, L., Fogassi, L., Rizzolatti, G., 1996. Action recognition in the premotor cortex. *Brain* 119, 593–609.
- Gallese, V., Fogassi, L., Fadiga, L., Rizzolatti, G., 2002. Action Representation and the inferior parietal lobule. In: Prinz, W., Hommel, B. (Eds.), *Atten. Perform.*, vol. XIX. Oxford Univ. Press, Oxford, pp. 247–266.
- Gallese, V., Keysers, C., Rizzolatti, G., 2004. A unifying view of the basis of social cognition. *Trends Cogn. Sci.* 8, 396–403.
- Gergely, G., Csibra, G., 2003. Teleological reasoning in infancy: the naive theory of rational action. *TICS* 7, 287–292.
- Goldman, A., Sripada, C.S., 2005. Simulationist models of face-based emotion recognition. *Cognition* 94, 193–213.
- Grandin, T., 1995. *Thinking in Pictures*. Doubleday, New York.
- Hadjikhani, N., Joseph, R.M., Snyder, J., Tager-Flusberg, H., 2005. Anatomical differences in the mirror neuron system and social cognition network in Autism. *Cereb. Cortex (Advanced On-line access)*, Nov. 23 2005).
- Hauser, M.D., Fitch, W.T., 2004. Computational constraints on syntactic processing in a non human primate. *Science* 303, 377–380.
- Hauser, M.D., Chomsky, N., Fitch, W.T., 2002. The faculty of language: what is it, who has it, and how did it evolve? *Science* 298, 1569–1579.
- Hayes, C.M., 1998. Theory of Mind in nonhuman primates. *Behav. Brain Sci.* 21, 101–148.
- Hobson, R.P., Lee, A., 1999. Imitation and identification in autism. *J. Child Psychol. Psychiatry* 40, 649–659.
- Hobson, R.P., Ouston, J., Lee, A., 1988. Emotion recognition in autism: coordinating faces and voices. *Psychol. Med.* 18, 911–923.
- Hobson, R.P., Ouston, J., Lee, A., 1989. Naming emotion in faces and voices: abilities and disabilities in autism and mental retardation. *Br. J. Dev. Psychol.* 7, 237–250.
- Humphrey, N.K., 1976. The social function of intellect. In: Bateson, P., Hinde, R.A. (Eds.), *Growing Points in Ethology*. Cambridge Univ. Press, Cambridge, pp. 303–321.
- Humphrey, N.K., 1978. Nature’s Psychologists. *The New Scientist* (29 June).
- Humphrey, N.K., 1980. Nature’s psychologists. In: Josephson, B.D., Ramachandran, V.S. (Eds.), *Consciousness and the Physical World*. Pergamon, Oxford, pp. 57–75.
- Hutchison, W.D., Davis, K.D., Lozano, A.M., Tasker, R.R., Dostrovsky, J.O., 1999. Pain related neurons in the human cingulate cortex. *Nat. Neurosci.* 2, 403–405.
- Hutto, D.H., 2004. The limits of spectatorial Folk Psychology. *Mind Lang.* 19, 548–573.
- Iacoboni, M., Molnar-Szakacs, I., Gallese, V., Buccino, G., Mazziotta, J., Rizzolatti, G., 2005. Grasping the intentions of others with one’s own mirror neuron system. *PLOS Biol.* 3, 529–535.
- Jackson, P.L., Meltzoff, A.N., Decety, J., 2005. How do we perceive the pain of others: a window into the neural processes involved in empathy. *NeuroImage* 24, 771–779.
- Jacob, P., Jeannerod, M., 2005. The motor theory of social cognition: a critique. *Trends Cogn. Neurosci.* 9, 21–25.
- Keysers, C., Wickers, B., Gazzola, V., Anton, J.-L., Fogassi, L., Gallese, V., 2004. A touching sight: SII/PV activation during the observation and experience of touch. *Neuron* 42, 1–20 (April 22).
- Kohler, E., Keysers, C., Umiltà, M.A., Fogassi, L., Gallese, V., Rizzolatti, G., 2002. Hearing sounds, understanding actions: action representation in mirror neurons. *Science* 297, 846–848.
- McIntosh, D.N., Reichman-Decker, A., Winkielman, P., Wilbarger, J., in press. When the social mirror breaks: deficits in automatic, but not voluntary mimicry of emotional facial expressions in autism. *Dev. Sci.*
- Meltzoff, A.N., Brooks, R., 2001. “Like Me” as a building block for understanding other minds: bodily acts, attention, and intention. In: Malle, B.F., Moses, L.J., Baldwin, D.A. (Eds.), *Intentions and Intentionality: Foundations of Social Cognition*. MIT Press, Cambridge, MA, pp. 171–191.
- Morrison, I., Lloyd, D., DiPellegrino, G., Roberts, N., 2004. Vicarious responses to pain in anterior cingulate cortex: is empathy a multisensory issue? *Cogn. Affect. Behav. Neurosci.* 4, 270–278.
- Oberman, L.M., Hubbard, E.H., McCleery, J.P., Altschuler, E., Ramachandran, V.S., Pineda, J.A., 2005. EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Cogn. Brain Res.* 24, 190–198.
- Onishi, K.H., Baillargeon, R., 2005. Do 15 months-old understand false beliefs? *Science* 308, 255–258.
- Paukner, A., Anderson, J.R., Borelli, E., Visalberghi, E., Ferrari, P.F., 2005. Macaques (*Macaca nemestrina*) recognize when they are being imitated. *Biol. Lett.* 1, 219–222.
- Povinelli, D.J., Bering, J.M., Giambrone, S., 2000. Toward a science of other minds: escaping the argument by analogy. *Cogn. Sci.* 24 (3), 509–541.
- Premack, D., Woodruff, G., 1978. Does the chimpanzee have a Theory of Mind? *Behav. Brain Sci.* 1, 515–526.
- Preston, S.D., De Waal, F.B.M., 2002. Empathy: its ultimate and proximate bases. *Behav. Brain Sci.* 25, 1–72.
- Ramnani, N., Miall, C.R., 2004. A system in the human brain for predicting the actions of others. *Nat. Neurosci.* 7, 85–90.
- Reddy, V., Hay, D., Murray, L., Trevarthen, C., 1997. Communication in infancy: mutual regulation of affect and attention. In: Bremner, G., Slater, A., Butterworth, G. (Eds.), *Infant Development: Recent Advances*. Psychology Press, East Sussex, pp. 247–273.
- Rizzolatti, G., Craighero, L., 2004. The mirror neuron system. *Annu. Rev. Neurosci.* 27, 169–192.
- Rizzolatti, G., Fadiga, L., Gallese, V., Fogassi, L., 1996. Premotor cortex and the recognition of motor actions. *Cogn. Brain Res.* 3, 131–141.
- Rizzolatti, G., Fogassi, L., Gallese, V., 2001. Neurophysiological mechanisms underlying the understanding and imitation of action. *Nat. Neurosci. Rev.* 2, 661–670.
- Rogers, S., 1999. An examination of the imitation deficit in autism. In: Nadel, J., Butterworth, G. (Eds.), *Imitation in Infancy*. Cambridge Univ. Press, Cambridge, pp. 254–279.
- Schmitz, C., Martineau, J., Barthélemy, C., Assaiante, C., 2003. Motor control and children with autism: deficit of anticipatory function? *Neurosci. Lett.* 348, 17–20.
- Singer, T., Frith, C., 2005. The painful side of empathy. *Nat. Neurosci.* 8, 845–846.
- Singer, T., Seymour, B., O’Doherty, J., Kaube, H., Dolan, R.J., Frith, C.F., 2004. Empathy for pain involves the affective but not the sensory components of pain. *Science* 303, 1157–1162.
- Snow, M.E., Hertzog, M.E., Shapiro, T., 1988. Expression of emotion in young autistic children. *Annu. Prog. Child Psych. Child Dev.* 514–522.
- Stern, D.N., 1985/2000. *The Interpersonal World of the Infant*. Basic Books, New York.
- Striano, T., Henning, A., Stahl, D., 2005. Sensitivity to social contingencies between 1 and 3 months of age. *Dev. Sci.* 8, 509–518.
- Subiaul, F., Cantlon, J.F., Holloway, R.L., Terrace, H.S., 2004. Cognitive imitation in Rhesus macaque. *Science* 305, 407–410.
- 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. *Curr. Biol.* 15, 84–85.
- Tomasello, M., Call, J., 1997. *Primate Cognition*. Oxford Univ. Press, Oxford.
- Trevarthen, C., 1979. Communication and cooperation in early infancy: a description of primary intersubjectivity. In: Bullowa, M. (Ed.), *Before Speech: The Beginning of Interpersonal Communication*. Cambridge Univ. Press, New York, pp. 321–347.
- Umiltà, M.A., Kohler, E., Gallese, V., Fogassi, L., Fadiga, L., Keysers, C., Rizzolatti, G., 2001. "I know what you are doing": a neurophysiological study. *Neuron* 32, 91–101.
- Watkins, K.E., Strafella, A.P., Paus, T., 2003. Seeing and hearing speech excites the motor system involved in speech production. *Neuropsychologia* 41 (8), 989–994.
- Whiten, A., Byrne, R.W., 1997. *Machiavellian Intelligence 2 Evaluations and Extensions*. Cambridge Univ. Press.
- Wicker, B., Keysers, C., Plailly, J., Royet, J.-P., Gallese, V., Rizzolatti, G., 2003. Both of us disgusted in my insula: the common neural basis of seeing and feeling disgust. *Neuron* 40, 655–664.
- Yirmiya, N., Kasari, C., Sigman, M., Mundy, P., 1989. Facial expressions of affect in autistic, mentally retarded and normal children. *J. Child Psychol. Psychiatry Allied Discipl.* 30, 725–735.