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### Abstract

The mirror mechanism allows the direct translation of a perceived (seen, felt, heard) action into the same motor representation of its related goal. This mechanism allows a direct comprehension of others' goals and motor intentions, enabling an embodied link between individuals. Because the mirror mechanism is a functional expression of the motor system, these findings suggest the relevance of the motor system to social cognition. It has been hypothesized that the impaired understanding of others' intentions, sensations, and emotions reported in autism spectrum disorder (ASD) could be linked to an alteration of the mirror mechanism in all of these domains. In this review, we address the theoretical issues underlying the social impairments in ASD and discuss them in relation to the cognitive role of the mirror mechanism.

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### Reference

GALLESE, Vittorio, ROCHAT, Magali J, BERCHIO, Cristina. The mirror mechanism and its potential role in autism spectrum disorder. *Developmental Medicine and Child Neurology*, 2013, vol. 55, no. 1, p. 15-22

DOI : 10.1111/j.1469-8749.2012.04398.x

PMID : 22924341

Available at:

<http://archive-ouverte.unige.ch/unige:129091>

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# The mirror mechanism and its potential role in autism spectrum disorder

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## PUBLICATION DATA

Accepted for publication 14th May 2012.

Published online 28th August 2012.

## ABBREVIATION

ASD Autism spectrum disorder

The mirror mechanism allows the direct translation of a perceived (seen, felt, heard) action into the same motor representation of its related goal. This mechanism allows a direct comprehension of others' goals and motor intentions, enabling an embodied link between individuals. Because the mirror mechanism is a functional expression of the motor system, these findings suggest the relevance of the motor system to social cognition. It has been hypothesized that the impaired understanding of others' intentions, sensations, and emotions reported in autism spectrum disorder (ASD) could be linked to an alteration of the mirror mechanism in all of these domains. In this review, we address the theoretical issues underlying the social impairments in ASD and discuss them in relation to the cognitive role of the mirror mechanism.

Autistic spectrum disorder (ASD) is a neurodevelopmental disorder occurring with a frequency of one in 150 children. Albeit with consistent differences in their clinical profiles, people with autism share an impairment in three specific domains: social interactions, verbal communication, and non-verbal communication, and a narrow repertoire of behaviours and fields of interest. In other words, in people with autism all behaviours necessary to establish and regulate social interactions successfully seem to be impaired.

Although research is now mostly focused on finding a plausible genetic cause for ASD, the origins of the disorder are still controversial. For a long time, parents' unresponsiveness to their child has been suggested as the underlying cause of autistic behaviour.<sup>1,2</sup> However, without minimizing the contribution of developmental, environmental, and relational factors, the hypothesis that links ASD exclusively to an early failure in intersubjectivity has gradually been abandoned. In parallel with diagnostic advances that have radically changed the understanding of autism, arguments for an organic deficit have come from studies that demonstrated a distinct cognitive profile in children with autism. For example, a fragmented perceptual–cognitive style endows people with autism with an accurate perception of details at the expense of the general picture that emerges when all the details are put together.<sup>3</sup>

The theory of mind deficit account<sup>4,5</sup> proposed that persons with autism are unable to represent their own and others' mental states (such as desires and beliefs), and are thus unable to understand and predict behaviours in terms of these states. According to this view, the essence of social cognition is to be found in the ability to reflect consciously upon different states of mind, thus equalling social cognition to social meta-cognition. Moreover, meta-representational abilities were defined as

intrinsically linked to the acquisition of a full-blown linguistic competence, thus pertaining uniquely to the humans.

The assertion that there is an evolutionary discontinuity in social cognition<sup>6</sup> needs, however, to be revised in the light of recent findings from several interdisciplinary studies. These studies, investigating the pre-verbal and non-meta-representational aspects of social cognition, revealed that crucial aspects of social cognition appear both in phylogenetically and ontogenetically much earlier than previously thought. It has been demonstrated that sophisticated aspects of mentalizing develop well before the maturation of linguistic competences<sup>7</sup> and that theory of mind precursors, such as sensibility to deictic pointing<sup>8</sup> and intention understanding,<sup>9–11</sup> are shared among different species.

Most importantly, recent findings in cognitive neuroscience have provided a solid neurophysiological substrate to the hypothesis of a cognitive continuity in phylogeny. Evidence showed the existence of a mirror mechanism, a neural mechanism common to both human and non-human primates, which matches action perception to action execution. It has been proposed that this mechanism could account for direct understanding of actions and motor intentions. It could be an automatic, pre-reflexive mechanism that would provide essential building blocks of social cognition through phylogeny.<sup>12,13</sup>

We therefore propose that important aspects of social cognition basically rely on the capacity to predict and understand the motor goals and motor intentions of the actions of others, an ability that is intrinsically embodied in the organization of the cortical motor system. We termed this ability as motor cognition (see references 14–16).

A consequence of our hypothesis is that an understanding of action is closely related to the functional development and organization of the motor system, a domain that might be

compromised in persons with autism for whom theorizing about the others' minds would be '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'.<sup>17</sup> This is contrary to what was maintained by supporters of classic cognitivism.

In this review we will first consider the neuroscientific evidence relating the existence and functions of mirror mechanism with action-understanding abilities in humans. The neural substrate and development of motor cognition will be discussed in the light of its relevance for understanding important aspects of ASD.

### **Evidence of a mirror mechanism in humans and hypothesis about its development**

Twenty years ago, the discovery of mirror neurons in the macaque monkey premotor cortex urged the scientific community to revise the role of the motor system in social cognition. Mirror neurons fire both during action perception (seeing or hearing) and execution. It has been proposed that their discharge reflects the translation of the perceived motor act into the same motor programme in the observer's brain. As in the premotor cortex, motor acts are represented in terms of their motor purpose, the activation of the related motor programme would allow the individual to grasp directly, through a motor simulation, the meaning (i.e. the motor goal) of others' action.

The mirror mechanism reveals the existence of a simpler form of social cognition, motor cognition, that is implemented by the functional organization of the cortical motor system. Motor cognition would, therefore, rely on the automatic detection of 'proximal' and (to some extent) 'distal' action goals, to the extent that they match the cortical representation of similar actions in the observer's motor system. As the mirror mechanism was found to be shared among primates, it could be proposed as a phylogenetic precursor in social understanding that precedes and then coexists with the development of more complex meta-cognitive abilities in humans.

Indirect investigating techniques have demonstrated the existence of the mirror mechanism in humans involving the activation of the same specific brain areas during action execution and observation (see references 18 and 19 for a review). According to brain imaging studies, the likely human homologues of macaque monkey mirror mechanism-related areas include the ventral part of the precentral gyrus, the posterior part of the inferior frontal gyrus, the rostral part of the inferior parietal lobe, and regions within the intraparietal sulcus and the superior temporal sulcus.<sup>20</sup> Moreover, a recent single-cell recording experiment has provided direct evidence for the existence of mirror neurons in the human brain.<sup>21</sup> Contrary to macaque monkeys, human mirror neurons have also been found in non-canonical mirror areas: in the supplementary motor area, an area mainly dedicated to movement initiation and sequencing and the medial temporal lobe, principally involved in memory tasks.

The mirror mechanism in humans and in nonhuman primates presents several functional similarities, although numer-

### **What this paper adds**

- A review of the most recent literature on mirror mechanism theory.
- A review of the impaired aspects of social cognition in ASD and their possible links with impaired motor cognition.
- A critical analysis of the most controversial aspects of mirror mechanism theory.

ous studies have suggested a higher level of abstraction in humans. For example, the mirror mechanism presents a rough somatotropic organization both in monkey and humans,<sup>22,23</sup> but unlike macaque monkeys, the observation of intransitive meaningless movements<sup>24-27</sup> and pantomimes<sup>22,28,29</sup> seem to activate mirror mechanism-related areas fully in humans. Moreover, motor goals can be extracted from observed motor acts even when they are executed by a robotic arm,<sup>30</sup> or when hand motor movements are observed by congenitally limb deficient individuals.<sup>31</sup> Thus, also in human mirror mechanism, motor goals appear to be coded at an abstract level, independent of the physical variants of both effectors and movements.<sup>32,33</sup> Neuroimaging studies have further demonstrated the implication of the human mirror mechanism in coding motor intentions of basic actions.<sup>34,35</sup> In fact, during the observation of the same motor acts, the activity of human mirror mechanism-related areas is modulated by contextual information, suggesting different motor intentions.<sup>34</sup> Finally, the data reported by Mukamel et al.<sup>21</sup> suggest 'the existence of multiple systems in the human brain endowed with neural mirroring mechanism for flexible integration and differentiation of the perceptual and motor aspects of actions performed by self and others'.

The early existence of a motor simulation mechanism in the premotor and posterior parietal cortices has been confirmed by several experiments conducted in infants.<sup>36-38</sup> Since several studies have demonstrated that the degree of motor simulation of an observed action correlates with the observer's previous motor experience both in adults<sup>39-43</sup> and children,<sup>44</sup> some authors suggested a role for motor experience in modelling the ontogenic development of mirror mechanism.<sup>15,45,46</sup> This proposal also finds resonance in a series of experiments in developmental psychology showing the crucial effect of action experience on infants' understanding of the goal-relatedness of an action.<sup>47-49</sup>

The cortical representation of motor goals appears to be the organizing element upon which the whole architecture of the cortical motor system is functionally structured. Recent studies have demonstrated that the motor system is organized around specific motor goals even *before* birth. Zoia et al.<sup>50</sup> demonstrated that at around 22 weeks' gestation, the level of motor planning in fetuses is already compatible with the execution of 'intentional actions'. In another intrauterine study on twin fetuses,<sup>51</sup> it was demonstrated that even by the 14th week of gestation the movements directed to the self or specifically aimed at the co-twin display a kinematic profile (e.g. longer movement duration and deceleration time) that significantly differs from other accidental movements.

Although there is actually no direct evidence for the existence of a rudimentary mirror mechanism at birth, its presence could be speculatively linked to the newborn infant's innate ability to imitate simple facial movements.<sup>52-56</sup> In fact,

imitative ability entails the presence of a neural mechanism that translates the incoming sensory description of an action in terms of its motor programme. Neuroimaging evidence gathered in adult participants indicates that the mirror mechanism is a plausible candidate for the neural underpinning of imitation.<sup>29,57–61</sup> Indeed it has been proposed that ‘neonates and infants, by means of specific connectivity developed during the late phase of gestation between motor and ‘to-become-visual’ regions of the brain, would be ready to imitate the gestures they see performed by adult caregivers, and would be endowed with the neural resources enabling the rich interpersonal behaviours like turn-taking, proto-conversations, affective attunement, and the like, characterizing our post-natal life since its very beginning’.<sup>62</sup> However, while imitating a simple motor act would automatically activate the mirror neural network, the reproduction of a new complex behaviour requires an additional orchestration of its composing elements that go beyond the standard functions of the mirror mechanism-related areas.<sup>63</sup> Lepage and Théoret<sup>46</sup> proposed that mirror mechanism develops along with the maturation of inhibiting processes in prefrontal cortices, by which the child would gradually learn to refrain from acting out the automatic matching mechanism linking action perception and execution.

### The motor cognition hypothesis

The preservation of a mirror mechanism in phylogeny has raised the question of its functional role for survival in social species. Against the classic conception of primate social cognition based on an evolutionary discontinuity between behaviour-reading and mind-reading,<sup>64,65</sup> we propose the mirror mechanism as a functional neural mechanism for action understanding, common throughout phylogeny. Both human and nonhuman primates are endowed with a mirror mechanism that matches others’ actions to their own action repertoire; several studies further demonstrated the presence of a behavioural mirroring in disparate species (see reference 66 for a review). It is noteworthy that in its primitive aspect, which is shared among species, the mirror mechanism is nothing more than a mere action-perception matching mechanism. This neural mechanism allows the translation of the different perceptual aspects of an action into the same motor programme interlaced with a specific goal. By matching a perceived motor act with those contained in one’s own motor repertoire, it allows an inner, non-cognitively mediated, recognition of the others’ action goal. Thus, motor cues can be detected and used to understand and predict the goals of others through a basic mechanism that precedes in ontogeny the maturation of the uniquely human ability to reflect upon intentionality by relying upon a propositional format. Abilities like goal detection, action anticipation, and hierarchical representation of action with respect to a distal goal, can be considered as the direct consequence of the particular functional architecture of the cortical motor system, organized in terms of goal-directed motor action.<sup>67–69</sup>

Motor cognition finds its neural substrate in the cortical areas of the brain involved in matching action perception to

action execution. A consequence of our hypothesis is that action understanding is closely related to the correct development and functional organization of the cortical motor system. Following the discovery of mirror neurons, several authors have proposed that abnormalities in the mirror mechanism functioning could be critical in ASD.<sup>17,70–78</sup> The ‘motor cognition hypothesis’ provides a new approach to the study of the developmental breakdown of social cognition in ASD.<sup>62</sup>

### The pervasive presence of motor deficits in ASD

Although recurrently signalled, the presence of motor deficits in children with autism has been commonly regarded as peripheral to the autistic syndrome. By analysing home videos of infants later diagnosed as children with autism, Teitelbaum et al.<sup>79</sup> showed that early disturbances of movement could be clearly detected in infants aged from 4 to 6 months. Muscle tone and reflex abnormalities are very common in children with autism, in particular, the persistence of newborn reflexes and increased or decreased muscle tone. Motor problems assessed in preschool age children with ASD seem to endure over time.<sup>80</sup> Since their early appearance and their continuance in development, it has been proposed that motor impairments serve a crucial role in defining the behavioural phenotype of the autistic disorder.<sup>81,82</sup>

Motor symptoms observed in autism include awkwardness and clumsiness, hyperactivity, and stereotypical movements.<sup>83</sup> Moreover, children with autism frequently exhibit postural instabilities<sup>84</sup> and atypical gait patterns displaying shortened steps, ‘toe walking’, and generally poor coordination of limb movements.<sup>85,86</sup> They often present with delays in the initiation, change, or arrest of a motor sequence and expressionless faces with little spontaneous movements, all symptoms dramatically resembling those depicted in extrapyramidal motor disorders. Children with ASD regularly show motor coordination problems that might be associated with cerebellar dysfunctions.<sup>87</sup> Moreover, while typically developing children control their action using feed-forward information, children with ASD preferentially rely on feedback information. Such motor disturbance prevents children with autism from adopting anticipatory postural adjustments.<sup>88</sup> A primary causal link between an abnormal development of the cerebellum in ASD and impairments in motor adaptation has, nevertheless, been challenged recently by a study demonstrating a preserved ability to learn from sensory prediction errors and a normal acquisition of internal models of action in children with autism.<sup>89</sup> Several authors thus suggest that the deficits in motor function and skill acquisition may be due to other regions critical for motor learning or to abnormalities in connections between these regions.<sup>90,91</sup>

The reported motor disorders assume a clear theoretical relevance in the light of recent findings documenting an atypical functional organization of the motor system in children with ASD. One experiment<sup>92</sup> revealed that children with autism fail to anticipate the motor consequences of the action’s final goal, both when the action is executed and observed. It has been proposed that, contrary to typically developing children, in children with ASD the observed or to-be-executed

action is not represented as a whole in the function of the overall motor intention. A deficit in fluidly orchestrating the motor acts in an intentional format could account for their difficulty in directly understanding the intention of the observed action when executed by others. Moreover, the presence of a deficit in chaining motor acts into a global action could further explain the reported difficulties on action planning.<sup>93–96</sup>

The observation of a specific motor act and the context in which it is executed allows the extraction of information about the action goal and the motor intention underlying it. Boria et al.<sup>97</sup> demonstrated that children with autism have severe difficulties in understanding others' intentions when they have to rely exclusively on motor cues.

Further aspects of the motor sphere, such as action simulation, mimicry and imitation (see below), have been recently explored by a number of studies, all confirming an impairment of the core mechanisms of motor cognition in children with autism.

### **Atypical brain development: new evidence**

Several investigators are now trying to define the neurological causes underpinning social impairments in ASD. Some have suggested the presence of abnormal neural organization and connectivity during cerebral growth. An increase of white matter seems to be at the origin of the anomalous brain dimensions most frequently observed in infants with ASD.<sup>98–100</sup> Cerebral peak overgrowth appears to involve regions mediating the high-order social, emotional, cognitive, and language functions. Courchesne and Pierce<sup>101</sup> proposed that a surplus of neurons within the frontal and temporal cortices would produce an imbalance between the increased local short-distance connectivity within brain areas and the decreased long-range distant connectivity among brain areas. An imbalance between short-distance and long-distance connectivity would disrupt the formation of large scale brain cell assemblies, thus altering integrative information processing between brain regions.<sup>102–104</sup> According to the theory of neural maldevelopment, early brain development in autism is characterized by two phases of brain growth pathology:<sup>98,101</sup> an early brain overgrowth at the beginning of life and a slowing or arrest of growth during early childhood. In some individuals, a third phase, degeneration, may be present in some brain regions by preadolescence. These data were further confirmed by the data of Hadjikhani et al.<sup>73</sup> reporting that the cerebral thickness of the superior parietal, temporal, and frontal cortices is particularly reduced in adolescents with autism. Interestingly, these regions include areas involved in social cognition, facial expression and facial recognition, and those characterized by the mirror mechanism.<sup>18,20</sup>

### **Social impairments and implications of the mirror mechanism**

#### **Gaze**

Individuals with autism tend to avoid direct gaze, and struggle to decode facial expressions, postures, or gestures. Children with ASD have difficulties in selecting faces among other

stimuli,<sup>105</sup> a finding that has been recently corroborated by the fact that in contrast to typically developing children, faces have a lower capacity to engage the attention of toddlers with ASD.<sup>106</sup> Several studies confirmed impairments in face processing<sup>107</sup> and recognition,<sup>108,109</sup> indicating the presence of atypical scanning patterns.<sup>110–114</sup> When processing faces, individuals with autism generally prefer a 'piecemeal' encoding strategy to a holistic processing,<sup>115,116</sup> and a specific avoidance of the eyes region (especially when gaze is direct) has frequently been reported.<sup>108,117,118</sup> Zwaigenbaum et al.<sup>119</sup> have demonstrated that by 12 months of age, children who are later diagnosed with autism already show atypical eye contact, eye tracking, and disengagement of attention. In addition, Becchio et al.<sup>120</sup> showed that, while the kinematics of grasping an object in typically developing children is automatically influenced by the gaze of others, this is not true for children with autism. It should be added that Shepherd et al.<sup>121</sup> recently discovered a class of neurons in the lateral intraparietal area of macaque monkeys involved in oculomotor control. Those neurons' activity was triggered both when the monkey looked in a given direction and when it observed conspecific gazing in the same direction. The authors proposed that these lateral intraparietal area mirror neurons might be involved in the ability to share attention, thus playing a role in imitative behaviour.

Taken together, these results confirm that atypical visual processing and encoding of social stimuli are a characteristic feature in ASD and suggest the existence of abnormalities at a neurophysiological level.

#### **Emotion**

Probably linked with atypical face processing are the difficulties in recognizing the facial expression of emotions reported in some studies;<sup>122–125</sup> however, other studies have shown that the ability to recognize basic emotions remains intact in individuals with autism.<sup>126–128</sup> Adolphs et al.<sup>129</sup> showed that adults with autism are able to recognize simple emotions, but are severely impaired when asked to retrieve social knowledge on the basis of facial cues, for example, judging the trustworthiness and approachability of a person by watching their faces. Wang et al.<sup>130</sup> confirmed that cognitive assessment of basic emotions is relatively unimpaired in children and adolescents with autism, while showing that different neural networks seem to be recruited during the automatic processing of socio-emotional information. All together, these studies suggest that the emotion recognition deficit in autism may involve difficulties with more complex emotions rather than basic ones (see also Golan et al.<sup>131</sup>). The hypothesis that links deficits in socio-emotional processing in autism to a dysfunctional neural network has been supported by several functional imaging studies reporting atypical activation of the fusiform gyrus and lower amygdala activity in participants with autism involved in social evaluation tasks.<sup>132–134</sup>

Individuals with autism commonly find it difficult to express, understand, regulate, and share emotions. Ramachandran and Oberman<sup>74</sup> have proposed that impaired abilities in the emotional domain are due to alterations in the neural net-

works involved in emotional processing, such as abnormal connections between sensory areas and the amygdala. In a recent diffusion tensor imaging study, Jou et al.<sup>135</sup> found impaired neural connections between the key processing nodes of the 'social brain' in individuals with autism. They showed an aberrant neural connectivity between the amygdala, fusiform face area, and superior temporal sulcus, all cerebral structures that are critical for normal social perception and cognition.

According to Ebisch et al.,<sup>136</sup> abnormal gazing, expressing emotions, and emotional awareness in patients with autism could be linked to alterations in the functional connectivity of insular cortices. The insular cortex is considered 'a principal node of a neural mechanism integrating bodily arousal, autonomic and valence information from sensory, limbic, memory and motor regions' (see Craig<sup>137</sup>). The authors found the connections between the right anterior insula and amygdala to be altered in individuals with autism, which could lead to a distorted attribution of emotional valence to external events. Moreover, a compromised functional connectivity between the posterior insula and somatosensory cortices could alter interoceptive awareness and thus the subjective feelings. The altered ability to resonate with and to understand emotions in other individuals might be attributed to an impaired shared mechanism for emotional awareness.<sup>64,70</sup>

### **Communication skills**

Impairments in the domain of language and communication are part of the core deficits observed in individuals with autism. Linguistic deficits are largely variable among individuals with ASD, although pragmatic deficits seem to be the most pervasive.<sup>138,139</sup> In fact, despite the severity of autism, what remains lifelong are the semantic and pragmatic deficits such as unusual word choice, unusual prosody, echolalia, difficulties in sustaining conversation, turn-taking, and allowing the conversational partner to introduce his or her topic. Bishop<sup>140</sup> highlights that children with autism have communicative impairments not seen in other developmental language disorders; these difficulties pertain to appropriate use of language (i.e. using more oddities, minor use of gestures, etc.).

It has been suggested that a set of neural structures subserving a coupling mechanism between action observation and execution, together with an appropriate learning mechanism, could support the cultural evolution of human languages in all their richness.<sup>141</sup> Gallese and Lakoff<sup>142</sup> (see also Glenberg and Gallese<sup>143</sup>) proposed that 'the same circuitry that can move the body and structure perceptions, also structures abstract thought'. According to that model, a sensory motor system is necessary to understand at least concrete concepts.

In an exhaustive review of the literature, Bates and Dick<sup>144</sup> discussed the linking between the development of language and gesture. The authors highlighted some 'milestones' that have supported the idea of a connection between the evolution of these two domains. For example, it has been widely documented that from 6 to 8 months the emergence of canonical babbling matches that of hand rhythmic movements, from 8 to 10 months word comprehension occurs together with deic-

tic gesture, and from 11 to 13 months word production occurs with gesture. The linking between gesture development and language development has also been supported by the wide body of research in atypical populations, which has shown how the appearance of linguistic ability (i.e. word comprehension) is connected with the appearance of gesture behaviour (i.e. naming gesture). Furthermore, in a recent study<sup>145</sup> it has been shown that from 8 to 18 months the production of gestures and actions (with and without object interaction) is strongly correlated with word comprehension.

In adults, several electrophysiological studies provided evidence of a common neural substrate for language, perception and action. For example, an event related potentials study has demonstrated that around 400ms after stimulus onset, unanticipated semantic processes in both spoken and written language are reflected by a centroparietal negative potential, called N400.<sup>146</sup>

N400 has also been investigated and found during observation of hand postures,<sup>147</sup> and recent work demonstrated the existence of a neural system enabling rapid discrimination of semantic information in actions.<sup>148</sup> N400 activity increased when viewing the unanticipated action consequences as compared with the anticipated ones (i.e. answering a telephone by placing the phone on the top of the head). Interestingly, the N400 pattern seems to be different in participants with autism, being unaffected both when participants listen to sentences ending with anomalous words and reading sentences ending with a semantically incongruous word.<sup>149,150</sup>

In addition, Boria et al.<sup>97</sup> showed that children with ASD have difficulties in understanding others' intentions when the motor information is incongruent with the functional one (i.e. a hand replacing a telephone). These results seem to highlight a possible common root to the basis of action and language/communication semantic and pragmatic difficulties.

What is known today is that at a very early age, children with autism have a peculiar profile of communication with a poor repertoire of gestures (lower proportion of deictic pointing with major use of more primitive ones) and, in general, a lower proportion of communicative acts.<sup>151</sup> Moreover, it has been demonstrated that there is a significant co-occurrence of verbal and neuromotor deficits in children with autism and in children with a specific developmental language disorder.<sup>152</sup>

Altogether, on the basis of the available empirical evidence (for a review, see references 153 and 154) we suggest that, in individuals with ASD, a dysfunction in the same circuit involved in action understanding could simultaneously determine impairments in the appropriate use of language/communication intentionality. However, further empirical studies are needed to shed more light on these aspects.

### **Imitation**

Imitation constitutes one of the core neuropsychological deficits in autism. Present very early in development,<sup>155</sup> its dramatic effect on social interaction and learning ability can be further observed in adulthood.<sup>77,156,157</sup>

In patients with ASD motor dyspraxia and basic perceptual and attention impairments have been proposed as the main cause of the generally observed lack of imitative abilities and of the failure to use gestures for communicative purposes.<sup>87,158,159</sup> Dewey et al.<sup>160</sup> found that deficits in motor coordination abilities significantly co-occurred in children with ASD, developmental coordination disorder, or attention-deficit-hyperactivity disorder; however, severe difficulties during voluntary execution and imitation of gestures were observed only in children with autism.

Several studies have further highlighted a severe impairment of imitation abilities in individuals with ASD (for a review, see references 158 and 159). Particularly compromised in individuals with autism is the capacity to imitate symbolic elements like pantomimes,<sup>158,159,161</sup> meaningless gestures,<sup>162</sup> or unconventional action with a common object.<sup>158</sup> Imitation involves the ability to translate the action plan of the demonstrator into one's own perspective.<sup>78</sup> The consistent presence of 'reversal errors' when imitating a sequence of actions confirms the presence of a general incapacity to translate others' perspective into the imitator's one.<sup>163</sup>

Several authors have suggested that the imitation impairment in autism results from impaired self-other representation or difficulties in detecting similarities between self and others' body movements.<sup>156,164-166</sup> In fact, Helt et al.<sup>167</sup> found that children with ASD are impaired even in involuntary motor mechanisms evolved to warrant social and emotional attunement, such as susceptibility to contagious yawning (see also reference 168). Pierno et al.<sup>169</sup> showed that in a reach-to-grasp imitation task, children with autism were facilitated when primed with the observation of a robotic but not with a human arm movement. As control children showed the exact reverse pattern, the authors suggested that children with ASD are more tuned to process stimuli that are simpler both in terms of movement variabilities and socio-emotional content. Hobson and Hobson<sup>170</sup> explained the failure of children with ASD in incorporating aspects of the style displayed by the demonstrator into their own repertoire as resulting from their specifically weak propensity to identify with others.

By automatically mapping observed movements onto a motor programme, the mirror mechanism has been proposed as a crucial neural mechanism scaffolding imitation.<sup>77</sup> Although, it is important to distinguish the different types of imitation on the basis of the level of consciousness implied in the process (from contagion and mimicry to voluntary imitation of complex behaviours), several authors have assimilated the automatic inner simulation of an observed action with an implicit process of imitation.

As such, studies using different investigation techniques have suggested the existence of an action simulation deficit in individuals with autism. For example, Théoret et al.<sup>71</sup> demonstrated that by using transcranial magnetic stimulation (TMS), a TMS-induced hand muscle facilitation during hand action observation was present in healthy controls, while completely lacking in participants with autism. EEG studies on individuals with ASD showed that the mu frequency over the sensory-

motor cortex was not suppressed during action observation.<sup>75,76,171-173</sup> Dapretto et al.<sup>72</sup> showed that activity in a region of the inferior frontal cortex pertaining to the mirror mechanism (BA 44, pars opercularis) was diminished or absent in participants with autism during observation and imitation of the facial expression of basic emotions, while Williams et al.<sup>78</sup> found diminished activation of mirror mechanism during the imitation of finger movements. Finally, using the magneto encephalography technique, Nishitani et al.<sup>174</sup> revealed the presence of an abnormal imitation-related cortical activation sequence in patients with Asperger syndrome. The authors suggested that the delayed activation of the mirror mechanism-related areas could be ascribed to a lack of connections between these areas.

The reviewed evidence of an altered simulation process in autism has been challenged by different experiments showing a relatively spared mirror mechanism when the observed action is executed by a familiar agent,<sup>76</sup> when the action is goal-directed instead of intransitive and meaningless,<sup>173</sup> and during tasks triggering involuntary mimicry.<sup>175-177</sup>

Fan et al.<sup>178</sup> have demonstrated that mu suppression during the observation of a goal-directed action (hand grasping a chess piece) did not significantly differ between those with ASD and a control group. Moreover, a correlation has been drawn between the degree of mu suppression in ASD with the assessed level of communication skills of participants with ASD. The authors sustained the hypothesis of the coexistence in ASD of an altered motor representation of intransitive movements along with an intact representation of goal-directed action.

Contrary to what was observed in macaque monkeys, the peculiarity of the human mirror mechanism is to respond both to goal-directed actions and to meaningless movements.<sup>22,23,27</sup> It has been proposed that this peculiarity could be the basis of human imitation skills.<sup>179</sup> Several studies have shown reduced activation of the cortical motor system in individuals with ASD during the observation of movements without purpose;<sup>76,172,173</sup> the question of whether and how this deficit is actually spread to the entire ASD population needs further investigations. It is, therefore, important to focus future studies on this point in order to understand whether the conflicting results could be the expression of the heterogeneity of the ASD experimental sample,<sup>172</sup> of the participants' level of autism severity,<sup>178,180,181</sup> or of their age. In order to select the experimental sample across studies homogeneously, the use of a well-defined battery of diagnostic tests for autism should be recommended. It might then be possible to verify the hypothesis that only individuals with a severe deficit show a profound impairment of motor simulation at a basic level, while in individuals with high functioning ASD it might be more appropriate to investigate the finer aspects of the mirror mechanism involved in action understanding.

## CONCLUSION

The preservation of a mirror mechanism in phylogeny has raised the question of its functional role for survival in social species. It has been proposed that in non-human primates the

primary function of the mirror mechanism is to allow a direct comprehension of the motor acts of others, while further cognitive functions present only in humans, like imitation and language,<sup>141</sup> would have evolved on top of this.<sup>182</sup> The perspective of a low-level explanation for action understanding has, however, triggered several criticisms and attempts to downplay the role of the mirror mechanism in social cognition.

Hickok<sup>183</sup> argued against the claim that monkeys have mirror neurons that support action understanding and objected to an improper attribution of semantic properties to visuomotor neurons. It would be helpful to bear in mind that mirror neurons are simple neural cells with a binary response to sensory-motor stimuli. The mirror mechanism that they constitute is a coupling mechanism of executed and observed actions whose function is basically to integrate, within the motor system, inputs from other brain areas.<sup>14,66,184,185</sup> This process enables a direct social connection.

For decades the prevalent opinion has been that in humans, action understanding predominantly – or even exclusively – relies upon reading the minds of others<sup>4,5</sup> by relying on representations in propositional format. This view is based on the assumption that the observable behaviour of others is intrinsically intentionally opaque as it only consists of biological motion. According to the same view, only mind reading can translate a ‘moving hand’ into a ‘grasping hand’. The discovery of the mirror mechanism in the macaque monkey brain and subsequent evidence for mirroring mechanisms in the human brain suggested a more straightforward mechanism enabling the understanding of others’ behaviour.

Other authors proposed that a direct and pre-reflexive simulation mechanism is necessary though not sufficient to raise self-consciousness and meta-cognition and thus should necessarily be coupled with prefrontal control of the executive functions in order to play a role in social cognition.<sup>186</sup> Using a similar assumption, Saxe<sup>187</sup> disputed the possibility that a mechanism of inner simulation could help to infer others’ states of mind, especially in the case of false beliefs attribution. A recent study<sup>188</sup> showed that the intensity of the discharge of F5 mirror neurons is significantly stronger during action execution than during action observation. Moreover, the discovery that a subset of mirror neurons inhibited their discharge during action observation, while the opposite was true during action execution, suggests the existence of a ‘simple neuronal mechanism for maintaining self-other differentiation’<sup>21</sup> (see also references 183 and 189). This means that the mirror mechanism also probably contributes to individuals’ implicit sense of being the owners of their actions. In other words, there is a primitive bodily self-awareness that is before and below any reflective self-awareness and before any explicit sense of agency and sense of ownership.<sup>184,185</sup> Thus, the motor system provides both the common ground for understanding others and the criteria for distinguishing between bodily awareness of self and others.

Mirror activation has even been reinterpreted as based on a solely visual reconstruction of action rather than on a visuomotor matching mechanism, thus depriving mirror neurons of their fundamental motor simulation property, by reducing

their role to a mere emulation of the observed action<sup>190</sup> (but see references 19 and 185 for counterinterpretation). The genesis of mirror neurons has raised further controversy. The idea that mirror neurons are the result of learning processes has been opposed by the hypothesis of a pre-wired neural network inherited throughout phylogeny. Some authors have therefore proposed mirror neurons activity as the by-product of a Hebbian associative learning between the vision of one’s own action and the contiguous proprioceptive feedback of its execution.<sup>183,191,192</sup> However, the assumption that associative learning is exclusively based on the information’s contiguity has been disputed by scholars who have highlighted instead the importance of the information’s contingency allowing the experience of ‘a predictive relationship between observation and execution’.<sup>193</sup> Catmur et al.<sup>193</sup> employed selective motor training to manipulate the responses of the motor system during finger movement observation. It should be added, however, that this experiment did not reveal a proper mirror mechanism, but simply showed a mere associative motor inhibition – and this explains why the supposed counter-mirror activation only occurred with a much later delivery of a TMS impulse with respect to that normally employed to study the mirror mechanism (see a recent empirical confutation of the conclusions of Catmur et al.<sup>193</sup> in Barchiesi and Cattaneo<sup>194</sup>). Finally, those findings<sup>193</sup> do not tell us anything about the role of the mirror mechanism in social cognition, because such experiments dealt with mere movements only and not with motor goals and intention understanding.

Altogether, the associative learning hypotheses are able to explain the plasticity of the mirror mechanism, but are highly problematic with respect to its ontogenesis. First, these hypotheses do not account for mirroring mechanisms pertaining to motor acts performed with body parts like the mouth and the face, to which neither monkeys nor humans have direct visual access. Second, these hypotheses are forced to downplay, or even to deny, the plausibility of evidence for neonatal imitation both in non-human primates<sup>195,196</sup> and humans.<sup>52</sup> Third, these hypotheses cannot explain why motor experience obtained without any visual feedback can affect perception of human biological motion related to that experience. However, efforts in integrating both views have recently been made.<sup>197–199</sup> Attempting to solve the paradox of neonatal facial imitative abilities, Casile et al.<sup>199</sup> proposed that two different mirror systems underlie facial and hand-action understanding. While the latter might develop through the experiential couplings of the individual’s own movement execution and observation, the mirror mechanism for facial movements was proposed to be pre-wired and further shaped by the visual feedbacks conveyed by the caregivers’ facial movements. The idea that the caregivers’ automatic mimicry of the infant’s facial expression would provide the visual counterpart of the infant’s motor output, however, remains incompatible with the idea of a Hebbian learning, considering the lack of perfect contingency between motor and visual information. Thus, we prefer to stick to our hypothesis<sup>62</sup> of a rudimentary form of innate mirroring for both facial and hand actions, to be shaped later on by means of visuomotor learning.



Beyond the unsolved question of the mirror neurons' development, the motor cognition hypothesis provides a new approach to the study of the developmental breakdown of social cognition in ASD. Being aware that the clinical complexity of this multilayered disorder cannot be reduced to a simple deficit in motor cognition or to a malfunctioning of the mirror mechanism only, we propose that many of the social cognitive impairments manifested in autism can be ascribed to a general impairment in the intrinsic goal-related organization of motor behaviour.<sup>62,185</sup>

#### ACKNOWLEDGEMENTS

This work was supported by the EU grants TESIS and Rossi to VG, the European Research Council Grant CogSystem to MJR, and Fondazione Monte to CB.

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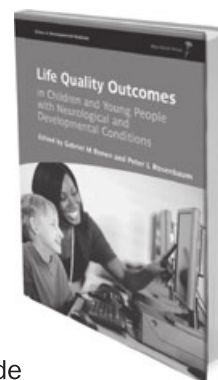
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