Review

So close yet so far: Motor anomalies impacting on social functioning in autism spectrum disorder

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A B S T R A C T

Difficulties in the social domain and motor anomalies have been widely investigated in Autism Spectrum Disorder (ASD). However, they have been generally considered as independent, and therefore tackled separately. Recent advances in neuroscience have hypothesized that the cortical motor system can play a role not only as a controller of elementary physical features of movement, but also in a complex domain as social cognition. Here, going beyond previous studies on ASD that described difficulties in the motor and in the social domain separately, we focus on the impact of motor mechanisms anomalies on social functioning. We consider behavioral, electrophysiological and neuroimaging findings supporting the idea that motor cognition is a critical “intermediate phenotype” for ASD. Motor cognition anomalies in ASD affect the processes of extraction, codification and subsequent translation of “external” social information into the motor system. Intriguingly, this alternative “motor” approach to the social domain difficulties in ASD may be promising to bridge the gap between recent experimental findings and clinical practice, potentially leading to refined preventive approaches and successful treatments.

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1. Introduction

Autism Spectrum Disorder (ASD) is a set of heterogeneous neurodevelopmental conditions affecting about 1% of the population (Lai et al., 2014). Actually, clinical diagnoses of ASD are based on behavioral symptoms that include early-onset difficulties in the social domain and restricted, repetitive conducts and interests (APA, 2013; Dawson and Bernier, 2013). Being ASD a multifactorial disorder with both complex genetic architecture (Rudie et al., 2012; Chaste et al., 2015) and neurobiological underpinnings (Courchesne et al., 2011; Di Martino et al., 2014; Stoner et al., 2014; Haar et al., 2014), it has been claimed that ASD behavioral symptoms emerge from a multi-level interaction between pre-existing neuropsychological vulnerabilities and the individual’s environment (for a review, Kim and Leventhal, 2015). In the last decade, the concept of “intermediate phenotype” (IP) has been extensively used in genetics for unravelling the aetiology of complex psychiatric disorders (Gottesman and Gould, 2003; Cannon, 2005; Cannon and Keller, 2006; Kendler and Neale, 2010). This construct fits well with ASD and it can constitute a more solid, alternative basis in the etiological investigation of complex neurodevelopmental disorder
(Pennington, 2006). Recent behavioral, neurophysiological and neuroimaging studies (mostly in children and adolescents) have explored putative neurocognitive IPS of ASD in order to disentangle its pathogenesis, to deeply characterize the neurobiological features of such disorder and to bridge the gap between clinical and preclinical (animal models) studies. Among the targets of utmost importance for future research in neuropsychiatric disorders there is the need to identify well-characterized neurocognitive IPS of complex functions. Indeed, this methodology, together with the data of clinical and preclinical studies, could inform the healthcare system, leading to: (i) better preventive approaches, by identifying children who are vulnerable at the neurocognitive level; and (ii) more refined and successful treatments by a better understanding of fundamental ASD neurocognitive etiological mechanisms that subsequently provides useful hints to foster the identification of new targets for treatments. Consistently with this view, recent approaches have also explored the possibility to detect ASD even in infancy, that is before the onset of unequivocal behavioral symptoms (Iversen and Wozniak, 2007; Leonard et al., 2014).

Considering that ASD is a lifelong condition that usually requires permanent assistance, early rehabilitative protocols to improve social functioning are urgent (Zwaigenbaum et al., 2013; Rogers et al., 2014). The most frequent approach in the identification of very early ASD markers is the prospective study of infant siblings of older children with ASD (but see Ronconi et al., 2014 for a potential novel approach based on parental autistic traits), which are at high risk of developing this disorder (for reviews see Keenin et al., 2013; Jones et al., 2014; Klin et al., 2015). Although it goes beyond the aims of this work, the potential role of motor cognition as a neurocognitive IP for at-risk infants for ASD should be carefully considered. However, before proceeding on this purpose, it is necessary to deeply characterize the role of motor cognition anomalies in the manifestation of core ASD symptomatology. To this aim, we consider behavioral, electrophysiological and neuroimaging findings supporting the idea that motor cognition is a critical neurocognitive IP for ASD.

2. The social-communicative and motor domains in autism

In the literature on ASD, difficulties in the social domain and motor anomalies are widely investigated. However, in the past decades they have been generally considered as independent, and therefore tackled separately.

On one side, most studies investigating impaired social skills in ASD focused on high-level “theory of mind” (ToM) abilities (i.e., the capability to impute mental states to oneself and to others, usually referred to as “mindreading” or “mentalizing”) (Premack and Woodruff, 1978; Koster-Hale and Saxe, 2013; Heyes and Frith, 2014). However, both the presence of heterogeneous experimental protocols (for a critical overview describing the “false-belief”, “reading in the eyes”, “strange stories”, “faux pas” test, see Boucher, 2012) and controversial neuroimaging findings (Von dem Hagen et al., 2014; Mitchell, 2008; for a meta-analysis, Schurz et al., 2014) call the construct of ToM into question (Bloom and German, 2000; Schaafsmna et al., 2015). On the other side, studies on the motor system in ASD reported—according to a widespread clinical tradition—gross and fine motor coordination anomalies (Mostofsky et al., 2006; Crippa et al., 2013, for a meta-analysis, Fournier et al., 2010), difficulties in controlling the force/direction of throwing gestures (Staples and Reid, 2010; Cook et al., 2013) and locomotor disturbances (Nobile et al., 2011; for a review, Gowen and Hamilton, 2013). In addition, anomalies in motor skills learning (that can be included in the broader construct of procedural learning, see Dziuk et al., 2007; Gidley Larson and Mostofsky, 2008) and the possibility that children with ASD rely upon different motor strategies (Sparaci et al., 2015) have been also explored.

Recent advances in cognitive and behavioral neuroscience have opened new promising perspectives, hypothesizing that the cortical motor system can play a role not only as a controller of elementary physical features of movement (e.g., force, direction, amplitude) but also in a complex domain as social cognition (Jeanerod, 2006; Gallesse et al., 2009; Rizzolatti and Sinigaglia, 2010; Casarotti and Chiamulera, 2015). Therefore, the notion of “motor cognition” has been introduced for characterizing these aspects and for supporting the idea that anomalies in these specific motor mechanisms may be a marker of—and, in some extent, contribute to—social interaction difficulties in ASD (Gallesse et al., 2013). Obviously, such an alternative approach does not necessarily reject that other motor difficulties—without any “social” implication in the ability to understand others’ action (Torres et al., 2013; Gowen and Hamilton, 2013)—may be present in ASD. Similarly, individuals with ASD may manifest different impairments in other functions supporting social cognition (e.g., mentalizing/mindreading, see Boucher, 2012).

Here, we go beyond previous studies on ASD that described difficulties in the motor and in the social domain separately, and we focus on the impact of motor mechanisms anomalies on social functioning. In a sense, studies on motor imitation in ASD (i.e., the capacity to translate some aspects of observed behavior into motor programs that can reproduce them) might be considered an important antecedent given that motor imitation can rely—among others reasons—also on the desire to conform to social norms or affiliate oneself to the demonstrator (Rizzolatti et al., 2001; Byrne, 2003, 2009; Iacoboni, 2009). However, in this paper we leave aside this widely explored point to focus on novel potential aspects because the notion of imitation, its putative neural correlates and clinical implications in ASD, remains controversial in the literature. Among the major reasons of such a controversy there is the ambiguity in defining the term and—consequently—experimental protocols to test it (Brass et al., 2009; Catmur et al., 2009; Ferrari et al., 2009; for a review, Casarotti and Moltieni, 2014). We describe two different phenomena characterizing motor cognition: the “motor resonance” and the “motor interference”. Referring to ASD, the first one can impact the ability to directly understand (i.e., motor-based understanding) others’ behavior, whereas the second one may be considered a more general and pervasive motor marker of social anomalies. Crucially, the study of social cognition that starts from motor functions may contribute to deeply characterize the early derailment from the typical developmental course in ASD, in turn, results in well-characterized behavioral autistic symptoms (Karmiloff-Smith, 1998, 2013).

3. Motor cognition in autism: motor resonance mechanisms

Motor resonance is a well-established phenomenon that provides the (motor) matching between the observed and the executed
action, transforming the “external” sensory (e.g., visual or auditory) information into the correspondent “internal” motor representation (Ferrari et al., 2013; Sinigaglia, 2013). This motor matching supports the idea that others’ action can be understood at different and non-mutually exclusive levels (Gallese, 2007; Casartelli and Molteni, 2014). We can access others’ mind not only by meta-cognitive and inferential processes (i.e., mentalizing/mindreading), but also in a more direct (motor) way without any meta-cognitive mediation (i.e., motor-based action understanding or action understanding “from the inside”, see Rizzolatti and Sinigaglia, 2010). The neural substrates of such mechanism has been widely investigated with single-cell recording techniques in non-human primates (Kohler et al., 2002; Fogassi et al., 2005; Bonini et al., 2014). Moreover, behavioral, neurophysiological and neuroimaging studies in adults (Gazzola and Keysers, 2009; Cattaneo et al., 2010; Senna et al., 2014; for a meta-analysis see Molenberghs et al., 2012), children (Lepage and Theoret, 2007; Bello et al., 2014; Berchio et al., 2014) and even infants (Falk–Ytter et al., 2006; Van Elk et al., 2008; Turati et al., 2013), support the existence of a similar mechanism also in humans (for reviews see Kilner and Lemon, 2013; Hunnius and Bekkering, 2014; Rizzolatti et al., 2014). Interestingly, such direct/motor action understanding seems to be compromised in ASD. In an electromyography (EMG) study, Cattaneo et al. (2007) asked to typically developing (TD) and ASD children to grasp a piece of food in order to eat it or to place a piece of paper in a container located near their own shoulder (execution condition), and to observe the same tasks performed by an experimenter (observation condition). From a purely kinematic perspective, these actions were very similar whereas their meanings (i.e., the intentionality, to eat vs. to place) were different. Recording the activity of the mouth-opening mylohyoideus (MH) muscle with EMG, the authors found that TD children activated the MH muscle during the reaching phase in the reaching-for-eating task, but not in the reaching-for-placeing task. This occurred both in the execution and observation conditions. In contrast, in both tasks children with ASD showed no MH muscle activation in the observation condition whereas in the execution condition they showed only a late activation in the reaching-for-eating task (i.e. in the bringing-to-the-mouth phase, that finally corresponds to the “real” eating phase). These data indicated that—in contrast with the TD group—ASD children have difficulties in encoding the goal of an action (i.e., to eat vs. to place) in a motor way (Cattaneo et al., 2007). Interestingly, the muscle activation observed in TD children has been hypothesized to be the behavioral manifestation of the mirror neurons activity (chained neural organization of motor acts, see Fogassi et al., 2005; Bonini et al., 2010). In turn, this supposition has led to hypothesize an implication of the mirror mechanism activity in explaining the children with ASD impairment to encode the intentions (e.g., grasp-to-eat) in their motor organization.

Other behavioral studies demonstrated that children with ASD, as TD children, were able to understand simple intentions (e.g., grasping a phone to call, see Boria et al., 2009) by means of inferences on the objects functional use and context (see also, Sparaci et al., 2014). In contrast, ASD children had difficulties to recognize intentions when they can rely exclusively on motor cues, without the assistance of functional and/or contextual information (e.g., grasping a phone to move it, Boria et al., 2009). In addition, motor resonance has been tackled in behavioral studies reporting anomalies in motor planning (Fabbri-Destro et al., 2009; Forti et al., 2011; Von Hofsten and Rosander, 2012), difficulties in imitating (Hobson and Hobson, 2008) and recognizing (Rochat et al., 2013) the motor “style” of an action (i.e., “motor prosody”, see the notion of “vitality forms”; for fMRI studies in TD adults, see Di Cesare et al., 2014, 2015). Fabbri-Destro et al. (2009) started from previous kinematics studies demonstrating that TD individuals plan an action—from several motor acts—from the very early phases according to the final goal (Marteniuk et al., 1987; Johnson-Frey et al., 2004; Ansuini et al., 2006). If two actions are composed by many acts, so that the first ones are identical whereas the last ones vary, motor planning should impact also on the execution of the first acts. This hypothesis has been elegantly explored by Fabbri-Destro et al. (2009) that compared TD and ASD children motor planning ability. Children were asked to perform a simple task consisting of two motor acts (i.e., pick up a metal object from a touch sensitive plate and drop it into a container) (Fabbri-Destro et al., 2009). In TD children modification of the size of container modulated both motor acts, indicating that they had encoded the goal of the motor action globally (i.e., to drop the object in the small container vs. in the big container) and that they had modulated the first motor acts in accordance with the final goal (i.e., small container—slower first motor act vs. big container—faster first motor act). In contrast, ASD children programmed the first motor acts independently, as if their motor organization would be immune to the influence of the final goal. Motor planning ability has been interpreted in the light of the motor intention understanding ability (for single-cell recording studies in macaques, see Fogassi et al., 2005; Bonini et al., 2010; for studies with ASD children, see Cattaneo et al., 2007; Fabbri-Destro et al., 2009; for a theoretical perspective, see Sinigaglia, 2013; for a review, Rizzolatti and Sinigaglia, 2010) (Fig. 1a).

Overall, these findings are consistent with the idea that impaired social skills in ASD may be—at least partially—explained by anomalies in the motor resonance mechanisms supporting the direct/motor action and intention understanding. In other words, anomalies in the neural circuits that sustain motor-based understanding may have cascade effects on the capabilities to access to others’ mind, contributing to explain social difficulties in ASD (Casartelli and Molteni, 2014). Consequently, anomalies/impaired in motor resonance mechanisms can be considered a neurocognitive IP for ASD.

4. Motor cognition in autism: motor interference mechanisms

Let’s return to the more general and pervasive motor marker defined as “motor interference”. The idea that the observation of a movement may interfere with the simultaneous execution of a (congruent or incongruent) movement has been extensively investigated in the last decade (Brass et al., 2001; Kilner et al., 2003, 2007; Stanley et al., 2007). For instance, a study by Kilner et al. (2003) found a significant “interference effect” when TD adult volunteers execute an horizontal sinusoidal arm movement whilst simultaneously observe an incongruent movement (i.e., vertical sinusoidal arm movement) performed by another individual. Interestingly, they did not find this significant effect when TD adults observed incongruent movement executed by a robotic arm (see also, Kilner et al., 2007; for a different view, see Oberman et al., 2007). In this study, the “interference effect” was intended as the measure of how individual’s movements are more variable if executed simultaneously to the observation of incongruent rather than congruent movements (Cook et al., 2014). The hypothesis that in TD population the modulation of the interference effect may be tuned—preferentially—to biological motion (biological/minimum-jerk velocity profile vs. non-biological/constant velocity profile, see Flash and Hogan, 1985) is fascinating but—by now—not completely clarified in the literature (Kilner et al., 2007; Oberman et al., 2007; Gazzola et al., 2007; Pierno et al., 2008; Cook et al., 2014). However, recent data seem to suggest that individuals with ASD have a reduced sensitivity in detecting the difference between biological (minimum-jerk velocity profile) and non-biological (constant velocity profile) motion (Cook et al., 2009). This hypothesis might be intriguing in the light of a further study by Cook et al. (2014) that
hypothesizes an atypical interference effects in individuals with ASD. Authors found that control participants exhibited an interference effect during simultaneous observation of real human and virtual human agent’s movements (observation of incongruent vs. congruent movements), whereas they did not show such difference when participants observed virtual robot agent’s movements. In contrast, in ASD individuals the interference effect after the observation of incongruent vs. congruent movements was not significant neither in observing real human, nor virtual human/robot agent’s movements (Cook et al., 2014, but see also Gowen et al., 2008). Even if further studies are necessary to definitively shed light on this important point, a fascinating and promising idea is that in individuals with ASD the execution of simple movements during simultaneous observation may be differently interfered compared with TD individuals.

These studies focused on motor interference effect during meaningless movement observation. However, in the context of motor cognition, a more intriguing aspect concerns goal-directed action involving the interaction with an object (here, for the sake of simplicity, consider the term “action” in the text as “act(ion)”).

Behavioural (Castaello, 2003; Edwards et al., 2003) and neuroimaging (Pierno et al., 2006a) studies are consistent with the idea that kinematics of an action can be influenced not only by the observation of other’s action, but also by the observation of another person simply looking at the target. Pierno et al. (2006b) found that TD children show facilitation effects in a reach-to-grasp task not only following the observation of a model grasping the object, but also when the model simply gazes at the object. On the contrary, ASD children did not show any evidence of facilitation neither in the model-grasping nor in the model-gazing condition (Pierno et al., 2006b). Consistent findings were found by Becchio et al. (2007) using a “transfer of interference” paradigm. This paradigm predicts that the perturbation of participants’ action kinematics after the observation of a model gazing at a distractor object is similar to the perturbation produced by the observation of a model executing an interfered action. Becchio et al. (2007) showed that ASD children—in contrast to TD children—did not show any interference effect neither from action nor from gaze observation. Taken together these findings seem to suggest that, in contrast to typical individuals, ASD children are not able to efficiently use both model’s action and gaze to set their subsequent action (Pierno et al., 2006b).

Studies in non-human primates showed that specific motor acts are encoded at the single-neuron level (Umlita et al., 2008; Rochat et al., 2010). Motor action has been defined as a sequence of motor acts organized in a “chain” and directed towards a (distal) goal (e.g., to reach a pen, grasping it, and placing it into the box). This “chain mechanism” is supposed to play a critical role in the (motor) action/intention understanding, as shown by studies in monkeys (Fogassi et al., 2005; Bonini et al., 2010) and humans (Cattaneo et al., 2007; Turati et al., 2013).

Fig. 1. An alternative “motor” approach to the social domain difficulties in ASD that focuses on motor resonance (a) and motor interference (b) mechanisms.
These findings may lead to an intriguing hypothesis: in children with ASD, the way in which social cues (e.g., other’s action, other’s gaze) are encoded and processed by the motor system is atypical, as if the motor system had difficulties in being influenced by any social cue. In other words, children with ASD seem unable to extract, codify and therefore translate this “external” social information into their own motor system (i.e., in their “internal” motor representation). However, an interesting alternative interpretation may be that in ASD the motor system is not immune to any social interference, but it is influenced by different social cues in respect to typical individuals. In such a case, ASD individuals’ motor system should not be considered as impenetrable by social cues, but it should be conceived as influenceable in an anomalous way.

To date, this point has not been yet consistently investigated in the literature. However, a thought-provoking support to this hypothesis can be found in a recent study focusing on automatic imitation, that can be considered a prosocial behavior promoting cooperative actions. Automatic imitation in typical subjects is stimulated simply by the presence of a model, whereas individuals with ASD seems to be unperturbed by his/her presence (Casartelli and Molteni, 2014). Parma et al. (2013) showed that maternal body odor promotes—in children with ASD—the same motor facilitation effect in automatic imitation observed in TD children with the mere model’s presence. As described in the literature, effects on attention and emotional regulation by body odors appear to have a clear evolutionary prosocial significance. Since early infancy, humans show a preferential attitude towards their own mother’s odor (Doucet et al., 2007; Ferdhenzi et al., 2008). This evidence is supported also by recent findings describing different neural pathways for processing body and common non-body odors (Lundström et al., 2009). Parma et al. (2013) tested the claim that such an odor can interfere also on motor kinematics in a group of TD and ASD children (see also Castiello et al., 2006; Tubaldi et al., 2008 for previous studies in TD population). They manipulated the role of the model (action vs. no-action), the familiarity of the model (familiar vs. non-familiar), and the presence of odors (familiar odor vs. non-familiar odor vs. no-odor). Results indicated that TD children have a motor facilitation effect (i.e., reduction in “movement time”, a particularly sensitive measure to visuo-motor priming effects previously reported—among others—by Edwards et al., 2003; Pierno et al., 2008) in all conditions where the model performed the requested reach-to-grasp action before the execution of their action (action condition), regardless of the presence of different type of odors (familiar, non-familiar, no-odor) or models (participant’s own mother, stranger mother). In contrast, ASD children showed a reduction in movement time needed to perform the reach-to-grasp action in the action condition only when their own mother’s odor was present (regardless of the presence of their own mother or a stranger one as model) (Parma et al., 2013). These data demonstrate that the exposure to the maternal odor in ASD children may compensate for the impairments in the motor facilitation effect previously described. Therefore, insensitivity to facilitation following model’s action observation in ASD children seems to disappear in presence of a familiar odor, which may be considered a prosocial cue. These findings are consistent with the claim that motor facilitation effect in ASD is regulated by different strategies given that it is not affected by the social cues relevant for typical subjects (e.g., other’s action, other’s gaze), but by other prosocial cues (e.g., maternal odor). Alternatively, it may be that maternal odor is so salient for ASD children that even the “immunity” of their motor system to typical social cues (i.e., other’s action, other’s gaze) is overcome. At this point, it would be crucial to verify whether in individuals with ASD maternal odor is a prosocial modulator only for the motor system or it represents a broader factor modulating also different (non-motor) functions (e.g., basic sensory/perceptual processes, higher social abilities). Regardless of this interpretation, we can state that not only the anomalies/impairments in motor resonance mechanisms, but also in motor interference mechanisms, can be considered as valuable neurocognitive IPs for ASD. Interestingly, such anomalies/impairments appear to be rather pervasive being present at different levels (i.e., movement observation, action observation, other’s gaze observation) (Fig. 1b).

5. Conclusion

Social cognition is the area of research dealing with how we make sense of the behavior of other human beings (Gallagher and Varga, 2015). Tackling ASD referring to motor cognition anomalies can help to explain ASD difficulties in social interactions (see, motor resonance in ASD and its impact on motor-based action understanding). It is important to shed light on motor markers of social anomalies in ASD individuals (e.g., see motor interference) in order to ascertain their more general and pervasive difficulty in extracting, codifying and subsequently translating “external” social information into their own motor system (Fig. 1). Given that these motor markers develop very early in infancy (Van Elk et al., 2008; Turati et al., 2013; Ambrosini et al., 2013), they could represent—as previously outlined—an additional intriguing instrument for early ASD detection. Further researches should disentangle whether motor cognition difficulties in ASD may be better considered as an impairment (Pierno et al., 2006b; Becchio et al., 2007; Rochat et al., 2013; Sparaci et al., 2014) or an anomaly (Fabbri-Destro et al., 2009; Parma et al., 2013, 2014; Cook et al., 2014). Such a point is pivotal for setting rehabilitative protocols. Although the growing body of experimental studies on putative IPs of ASD has considerably improved our knowledge on the pathophysiology of the disorder, it seems to persist a gap between the restricted circle of researchers working on ASD and a part of clinicians that may perceive the gradual accumulation of data as a sign of uncertainty and confusion (Maj, 2011). Sometimes clinicians seem at least skeptical on the practical significance and utility of the amount of experimental data published in the literature. This surely represents a critical aspect that needs to be addressed in the next years (Constantino and Charman, 2015), also in the light of the changes in the nosography of ASD and in the new DSM 5 strategy more prone to consider quantitative marker studies (APA, 2013; Lai et al., 2014). As already described, being the burden of ASD often extremely demanding for families and caregivers, rehabilitative and therapeutic protocols to improve ASD symptomatology are urgent. Today drugs have only a minor role in the treatment of ASD. Although educational and behavioral interventions for ASD children/toddlers have been shown to be—to some extent—efficient (Zwaigenbaum et al., 2015), future researches should surely focus on putative brain anomalies/impairments in ASD that may—in turn—be useful to establish more direct treatment. To achieve this aim, the only way is the characterization of reliable ASD IPs. A successful characterization of IP of ASD has been carried out in the domain of visual attention (for reviews see Sacrey et al., 2014; Klin et al., 2015), and has led to hypothesize the utility of using eye-tracking based attentional training in infant at risk (Wass et al., 2011). Other authors have hypothesized the use of neurofeedback training in children with ASD (Pineda et al., 2014; Friederich et al., 2015). To be set, all these behavioral and brain-based strategies demand a preliminary and rigorous characterization of IPs. Far to be the only IP for ASD, motor cognition can be surely a promising and innovative way to address this crucial aim. Obviously, autism is not caused by abnormalities in motor cognition, whereas they may be considered a critical marker of a more generalized failure in social adaptation at the level of the brain and epigenesis.
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