

## Review



**Cite this article:** Vivanti G, Rogers SJ. 2014 Autism and the mirror neuron system: insights from learning and teaching. *Phil. Trans. R. Soc. B* **369**: 20130184.  
<http://dx.doi.org/10.1098/rstb.2013.0184>

One contribution of 19 to a Theme Issue 'Mirror neurons: fundamental discoveries, theoretical perspectives and clinical implications'.

### Subject Areas:

health and disease and epidemiology,  
developmental biology, cognition

### Keywords:

autism, mirror neuron system, social learning,  
imitation

### Author for correspondence:

Sally J. Rogers  
e-mail: [sally.rogers@ucdmc.ucdavis.edu](mailto:sally.rogers@ucdmc.ucdavis.edu)

# Autism and the mirror neuron system: insights from learning and teaching

Giacomo Vivanti<sup>1,2</sup> and Sally J. Rogers<sup>3</sup>

<sup>1</sup>Olga Tennison Autism Research Centre, School of Psychological Science, and <sup>2</sup>Victorian Autism Specific Early Learning and Care Centre, La Trobe University, Melbourne, Victoria, Australia

<sup>3</sup>The MIND Institute, University of California, Davis Medical Center, Sacramento, CA 95817, USA

Individuals with autism have difficulties in social learning domains which typically involve mirror neuron system (MNS) activation. However, the precise role of the MNS in the development of autism and its relevance to treatment remain unclear. In this paper, we argue that three distinct aspects of social learning are critical for advancing knowledge in this area: (i) the mechanisms that allow for the implicit mapping of and learning from others' behaviour, (ii) the motivation to attend to and model conspecifics and (iii) the flexible and selective use of social learning. These factors are key targets of the Early Start Denver Model, an autism treatment approach which emphasizes social imitation, dyadic engagement, verbal and non-verbal communication and affect sharing. Analysis of the developmental processes and treatment-related changes in these different aspects of social learning in autism can shed light on the nature of the neuropsychological mechanisms underlying social learning and positive treatment outcomes in autism. This knowledge in turn may assist in developing more successful pedagogic approaches to autism spectrum disorder. Thus, intervention research can inform the debate on relations among neuropsychology of social learning, the role of the MNS, and educational practice in autism.

## 1. Introduction

Several decades after Vygotsky first suggested that participation in cultural practices is the foundation of human psychological development [1], this notion is receiving increasing empirical support. To illustrate, recent literature documents that early social interactions are critical for language learning [2], and engagement in pedagogical practices influences the development of a number of neuropsychological functions, including visuo-spatial skills, language processing and short-term memory [3]. According to Vygotsky, imitation is one of the key mechanisms through which participation in social practices fosters cognitive development. This idea has been the focus of much theoretical and empirical investigation in the past decade, in part driven by interest in the role of the mirror neuron system (MNS) in imitation and learning in typical development and autism ([4–6], see also [7]). Importantly, the debate on the possible link between the MNS and autism has shifted the focus from higher level cognitive processes that are affected in autism (e.g. understanding of false beliefs) to foundational processes related to infant understanding and infant social learning.

In this paper, we argue that three aspects of imitation and social learning are particularly relevant for our understanding of autism and potential roles of MNS: (i) the mechanisms that allow for the implicit mapping of and learning from others' behaviour, (ii) the motivation to attend to and model conspecifics and (iii) the flexible and selective use of social learning. These factors are also critical for further development of a science of autism pedagogy. We also argue that crucial insights on the nature of social learning difficulties in autism and possible roles of the MNS can be gained through the investigation of the effects of targeted educational treatments.

## 2. Three critical aspects of social learning: the how, why and when of social learning

Social learning, i.e. the goal-directed transmission of behaviour or knowledge from one person to another through play, pedagogical and other social exchanges built into cultural practices, has its roots in early infancy. Infants make sense of, imitate and learn from the actions of others using implicit learning processes long before they have the cognitive and linguistic capacities for declarative learning involving explicit reasoning, verbal mediation and inferential understanding of others' behaviour [8,9]. Cognitive development is therefore built up from a pre-linguistic, implicit understanding of others' behaviour, and early engagement with others, and the social learning opportunities that occur during typical social exchanges between infants and others, are the starting point, rather than the outcome, of the explicit representation/understanding of others' minds [1,10,11].

Moreover, from the beginnings of infancy onwards, social interaction and the participation in socio-cultural practices are intrinsically rewarding. The motivation to learn from others and do what others do is not driven only by the goal-related outcomes of social experiences (e.g. learning how to use a tool or a toy is rewarding because it allows to accomplish personal goals with the toy), but also by the pleasure in social interaction and being like others that accompanies the process of social learning [12,13]. Most children experience participation in activities and cultural practices with social partners as more rewarding than solitary activities, owing to both intrinsic social reward systems and to extrinsic rewards for participation provided by others. This intrinsic reward value of being like others is a distinctively human characteristic [4,14–16].

Finally, social learning is flexible and adaptive. Participation in social exchanges does not result in the mere acquisition and reproduction of others' actions; rather, children are selective in the way they use knowledge gained through engagement with and observation of others. For example, a recent study showed that sighted infants raised by blind mothers are not different from their peers in using eye contact and gaze following when interacting with sighted adults, but use less eye contact and more sounds and words when interacting with their blind mothers [17]. This demonstrates a flexible, adaptive use of non-verbal communication that reflects an early emerging sensitivity to immediate cues from others, and the selection of the communication strategies most successful under different circumstances. It also demonstrates that eye contact and other non-verbal strategies are under operant control, and not hard-wired. Other studies indicate that children, from infancy onwards, are very selective in terms of what to learn, who to learn from and when to learn. For example, infants are more likely to imitate actions when they experience social connectedness with the model and are more likely to follow the example of a prosocial versus antisocial model [18,19]. In this latter study, it can be assumed that infants learned the observed behavioural patterns via observation in both circumstances, but they chose what to incorporate in their behavioural repertoire based on the nature of their relationships, a specific feature of the environment. Moreover, children select 'what to imitate' in the stream of adults' behaviour by parsing the stream of behaviour and assigning salience of various acts

via relevant social-communicative cues provided by the adult, including gaze direction, posture and facial expression, as well as more subtle signals indicating social status [20–23].

These three aspects of social learning, i.e. its pre-inferential, implicit nature, its intrinsic rewards and its flexible/selective nature, are critical for our understanding of the social differences seen in autism, and the role of the MNS. Moreover, they provide crucial insights for a science of autism pedagogy.

## 3. Autism spectrum disorder as a social learning impairment

Autism spectrum disorders (ASDs) are a heterogeneous group of neurodevelopmental conditions that manifest in infancy and early childhood and are characterized by impairments in social communication and behavioural flexibility [24]. Despite the heterogeneity in clinical presentation, one unifying feature of these conditions is the disruption of the processes that are crucial for participation in cultural practices and social learning experiences, including imitation, joint attention, affect sharing, goal understanding, communicative use of language and play [6,25–27]. Difficulties in learning from others have dramatic effects on outcomes, with the majority of individuals affected by ASD needing considerable support to participate in activities involving school, work, recreation and personal independence, both in childhood and in adulthood. The fact that only a small proportion of adults with autism with average intellectual and language abilities live independently or in committed relationships reflects the severity and specificity of the social learning deficits in ASD [28–30].

Clinical observations and empirical studies suggest that the three distinctive aspects of social learning discussed above (implicit understanding of others' actions, intrinsic reward value associated with the experience of social learning, and flexible use of social learning) are all impaired in ASD. For example, a number of studies documented that individuals with ASD are often successful in solving social-emotional tasks involving declarative processes that are based on explicit knowledge, verbal mediation and inferential reasoning (e.g. labelling or imitating emotional expressions on demand) but show abnormalities in performance of social-emotional tasks that require the implicit processing of others' actions and emotions (e.g. physiological and motor response to others' actions and emotions [31–36]).

Furthermore, there are data suggesting that individuals with ASD do not find the participation to social learning experiences as rewarding or salient as do others. A number of studies document fewer behaviours aimed at creating and maintaining affiliative bonds (e.g. spontaneous imitation, social orienting, shared attention, helping), as well as decreased expressions of pleasure in social situations in individuals with ASD compared to those without [37,38]. Moreover, the amount of interest shown in non-social versus social stimuli has been found to be a predictor of ASD in infancy [39–41].

Finally, individuals with ASD have difficulties with 'strategic' aspects of social learning; e.g. understanding what to learn, who to learn from and in which contexts/circumstances to apply what they have learned from social experiences. For example, they might have difficulties with

selecting the relevant, to-be-imitated information during imitation tasks [42,43], with generalizing what they have learned in settings other than the one where they originally acquired the skill [44], and with applying knowledge they have gained from others in flexible, adaptive ways according to circumstances (e.g. making personal comments, telling jokes or sharing anecdotes in situations in which a more formal social interaction is appropriate [45–47]).

#### 4. Autism and the mirror neuron system

A number of abnormalities documented in ASD have been linked to the MNS [27,48–50]. Some of these are relevant to the social learning processes described above. The MNS activates in response to both self-generated and observed actions, gestures and emotional responses [51]. This process has been described as a ‘mirror mechanism’, enabling experience of others’ actions, emotions and sensations using the same systems through which we experience these actions emotions and sensations ourselves [51,53]. This mechanism provides a foundation for the idea that knowledge of the others’ behaviours is essentially grounded in our own self-experiences, and, in parallel, that many self-experiences are grounded in social behaviours of others directed towards us, rather than being based on explicit, language-mediated, inferential reasoning (i.e. an explicit theory of other minds). Some scholars have suggested that this mirror mechanism is disrupted in ASD, leaving individuals with ASD without this automatic flow of shared felt experiences of self and other behaviours and with, instead, ‘disembodied’ and declarative social knowledge (based on explicit inferential reasoning) as the primary foundation for social understanding and social learning [54]. This notion is supported by a number of studies that documented atypical/reduced activation of the sensorimotor system in response to the observation of another person’s motor behaviour in ASD [31,55–58]. Other studies however have challenged the notion of a global MNS impairment in ASD [59–61], and more empirical research is needed to clarify the role of the MNS in the neuropathophysiology and symptomatology of ASD, as well as the interplay between the MNS and other systems and processes involved in social learning and its disruption in ASD. Furthermore, available data on MNS abnormalities in ASD leave open the question of whether these findings indicate (i) an initial MNS disruption as a starting state in ASD [62], (ii) the effects of atypical social attention to social stimuli [61,63] or (iii) downstream neuropsychological effects of an altered history of social engagement and social learning experiences.

#### 5. From social learning to social teaching: the Early Start Denver Model

Further insights on the nature of the social learning impairment in ASD and the possible roles of the MNS can be gained from the study of pedagogical approaches to ASD that emphasize social learning processes, one of which includes the Early Start Denver Model (ESDM). The ESDM is a comprehensive early intervention approach developed specifically for toddlers and preschoolers with ASD, representing a fusion of behavioural, relational and developmental approaches [64]. One aspect of the developmental

model underlying the principles and strategies of the ESDM was first detailed by Rogers & Pennington [7]. According to this theoretical framework, autism sets in motion atypicalities in the processes that facilitate bodily and affective synchrony during infant–parent dyadic interactions, such as imitation, reciprocal vocalization and sharing of affect, and the underdeveloped foundations in dyadic reciprocity do not allow for the infant and young toddler to construct a working model of the partner’s ‘mind’—their attentional focus, the sources of their emotional responses, their goal-directedness, the ‘aboutness’ of their behaviours necessary for social learning and the development of social cognition. Through decreased experiences in synchronous dyadic exchanges during early development, infants with ASD lack learning opportunities needed to provide the appropriate input that supports the organization and specialization of the social brain circuitry in neurotypical development. While based on different theoretical tenets (in particular, [65]), these principles underlying the ESDM are consistent with the notions of motor resonance and intentional attunement that have emerged from the MNS literature. In both models, cognition is grounded in bodily actions, and participation in social exchanges is based on a pre-inferential, pre-linguistic grasp of the meaning of others’ behaviour. Similarly, both models posit a relationship between imitation, social understanding (theory of mind) and empathy. Moreover, the ESDM targets many of the processes that are supposedly implemented by the MNS, and therefore research on the effects of the ESDM has the potential to shed new light on the role of the MNS in ASD.

Additionally, the ESDM is informed by the neurodevelopmental model developed by Dawson [66] and Mundy [26,67], grounded on the notion that a biologically based decrease in the salience attributed to social stimuli during early developmental stages might virtually ‘isolate’ children with ASD from the social world, with downstream effects on social-cognitive development. ESDM techniques focus on increasing the salience and reward value of social attention and engagement via decreasing competition for the child’s attention, adding salience via inducing positive emotion in children during social engagement through movement, touch songs and other types of positive sensory social input. Teaching episodes are embedded within the framework of intrinsically rewarding joint activities built on the child’s spontaneous interest and motivation. The role of the therapist is to build up these joint activity routines from the initial interest of the child (e.g. spinning a toy) so that attention and excitement for the activity becomes shared excitement with a partner for doing the activity. Increasingly complex learning tasks are systematically incorporated into the joint activity framework, and flexible, adaptive use of acquired skills and knowledge is targeted through the systematic elaboration of established routines and by creating natural opportunities for the occurrence of the target skills across a variety of contexts.

Thus, pedagogical practices of the ESDM address the three foundational aspects of social learning described above (implicit ‘grasping’ of others’ actions and emotions, motivation for social learning and flexibility) by targeting multiple social-cognitive domains that are affected in ASD: verbal and non-verbal communication, imitation, emotion sharing, joint attention, play, social orienting and attention. Children learn the procedures and outcomes of the social activities that they are sharing with others while learning

about the process of doing things together, which involves the appreciation of the partner's social-communicative and emotional facial and bodily cues (e.g. smiling expectantly when stacking the last block of a tower to communicate a feeling of 'suspense') and the active role of self and partner in creating the activity by initiating, responding to, and continuing social exchanges through bodily actions, facial expressions, sounds and words. The process of learning *from* others is therefore parallel to learning *about* others, and it is based on the participation in shared sensorimotor and social-affective reciprocal exchanges, rather than on explicit knowledge/inferential reasoning.

Evidence for the effectiveness of the ESDM is documented in a number of studies [68], including a randomized control trial [69], showing significant gains across developmental domains. A critical question concerns the mechanisms underlying the changes that result from the intervention, or, in other words the 'active ingredients' that cause the positive effects of the therapy. If the MNS is involved in the social learning impairments in ASD, then changes in the MNS activity might be the active ingredient of the ESDM. Below, we illustrate a possible model supporting this notion.

## 6. Early Start Denver Model and mirror neuron system

According to some scholars, sensorimotor associations between the experience of self-generated behaviour and the observation of similar behaviour in others provide the foundation or contribute to the development of the mirror properties of the MNS via Hebbian learning (i.e. neurons that fire in response to action execution will also fire in response to action observation if execution and observation co-occur systematically [70–72]). The co-occurrence of observed and executed behaviour is particularly frequent in the context of early dyadic interactions, in which parent and child engage in temporally coordinated and mutually reinforcing matching of actions, emotions and vocalizations (i.e. dyadic synchrony [73]), as well as in the context of pedagogical and shared cultural practices (e.g. synchronous behaviours during sports, music, dance training, play and religious activities). Participation in such social exchanges is limited in children with ASD, possibly as a consequence of early lack of social orienting and attention towards others' behaviour [58,74]. One possibility is that the positive effects of the ESDM reflect changes at the MNS level resulting from associative learning mechanisms. As the emphasis in the ESDM is on play and care joint routines in which the adult and the child are engaged in the same activity in a coordinated way, involving reciprocal body movements and vocal imitation, sharing of affect and turn taking, one active ingredient of this therapy might be the formation of the sensorimotor associations that contribute to the emergence or the refinement of mirror properties in the developing brain. This model can provide some insights into the interplay between treatment-related changes in implicit learning, reward processing and flexible use of learning. The enhanced reward value of joint experiences promoted by the ESDM and the consequent increase in spontaneous engagement in joint activity routines might result in increased opportunities for registering/appreciating correspondences between own and others' actions. At the neural level, this process might provide

the foundation for the mirroring mechanisms that are critical for advanced social learning processes (imitation, empathy, action understanding). This process might promote the flexible/adaptive use of learning, as self-generated behavioural responses will be linked to others' behaviours and grounded on social exchanges that are inherently flexible and subjected to variations and elaborations. This model is consistent with the notion that (i) abnormalities at the MNS level might be the outcome, rather than the origin, of early abnormalities in social orientation/attention in ASD [63], (ii) abnormalities at the MNS level might result in further disruptions at the social and cognitive level [75] and (iii) this cascade of events might be avoided/mitigated by educational practices focused on intrinsically rewarding social exchanges [66].

The model outlined here could be tested empirically by examining whether treatment-related changes following the ESDM therapy are modulated by changes in MNS activity. Unfortunately, however, research in this area is in its infancy. In a recent study by Dawson *et al.* [76], electroencephalography was used to measure brain activity patterns in response to social (faces) versus non-social (objects) stimuli in children with ASD receiving the ESDM versus a control group of children with ASD receiving other interventions routinely available in their communities at similar levels of intensity. Following 2 years of intervention, greater brain responses to faces over objects were found in the ESDM group compared with the children with ASD in the community group who showed the opposite pattern (greater brain activity when viewing objects than faces). The brain activity pattern in the ESDM group was the same as found in an age matched typically developing (TD) group of children. Unfortunately, however, the evoked response potential (ERP) methodology used in the study does not allow inference on which brain regions were involved.

Another recent study employed functional magnetic resonance imaging to measure brain activity in response to social stimuli in two children with ASD receiving an early intensive intervention program called Pivotal Response Training, which shares many principles and strategies with the ESDM [77]. Their results showed increased brain activity compared with baseline in areas associated with social processing during biological motion tasks in the same areas that are activated by these tasks in children with typical development: including the fusiform gyrus, the prefrontal cortex and the posterior superior temporal sulcus, a region that provides input to the MNS.

While these studies are preliminary, their findings converge in suggesting that behavioural changes promoted by this type of intervention are accompanied by changes in brain response to social stimuli and in social reward/salience. However, existing data are insufficient to support or reject the notion that treatment-related changes reflect changes specifically in the MNS activity.

Future research should address this issue, developing fine-grained experimental tasks that capture the social learning processes that are disrupted in ASD, targeted by therapy and implemented by the MNS, as well as ASD-friendly brain imaging techniques, so that younger, and more affected children in the spectrum can be involved. These designs should also include the use of eye-tracking techniques to gain further insight on whether differences in MNS activities are modulated by differences in attention towards relevant social stimuli.

This line of research could help to inform both the debate on the nature of ASD difficulties and the possible link to the MNS, as well as clinical/educational practice.



## 7. Conclusion

According to Vygotsky, adults are able to do so many things by themselves because they grow up doing things with others. Autism affects the ability to live an adult independent life, perhaps more than any other neurodevelopmental condition. Difficulties in 'doing things by themselves' in the ASD population might be related to difficulties in doing things with others during infancy, childhood and throughout the lifespan. Following this line of thought, ASD can be seen as a social learning disorder, characterized by reduced/atypical engagement in social activities that provide social learning opportunities.

Three elements that are crucial for successful social learning all appear to be disrupted in ASD: the ability for implicit, non-inferential understanding of others' actions, the rewarding value of social participation and learning, and the strategic use of learning. The notion of disrupted mirror mechanisms in ASD provides a plausible account for the first of the three elements. Other brain networks and processes outside of the MNS are likely to be involved in other aspects of the social learning impairment in ASD. Future research should focus on the interplay of these different processes and the implications for typical and atypical mechanisms of social learning.

Consideration of these three key issues is also crucial in developing targeted education strategies. The ESDM addresses

these areas by focusing on shared 'bodily' construction of meaning, on the reward value associated with social learning experiences, and on the flexible use of knowledge across social contexts. Investigation of brain changes that are induced by these types of educational treatments can be used to illuminate the mechanisms of typical and atypical social learning.

Finally, the analysis of how children with ASD learn from others, and under what circumstances they learn from others, should continue to focus also on the behavioural level, as current knowledge in this area is still meagre. For example, a number of studies have documented normative processes of social behaviour, social understanding and social learning in ASD under particular circumstances (e.g. when processing information related to familiar versus unfamiliar people [78] or when processing emotionally meaningful versus emotionally neutral social signals [79]). Recent studies also suggest that social reward is not diminished in ASD under all circumstances [80].

Consideration of the multifaceted nature of social learning, and the interplay of mirror mechanisms, reward processes and top-down strategic use of knowledge is crucial for advancing understanding of the neuropsychology of typical and atypical social learning, and for the development of a science of autism pedagogy.

## References

- Vygotsky L. 1978 *Mind in society: the development of higher psychological processes*. Cambridge, MA: Harvard University Press.
- Kuhl PK. 2007 Is speech learning 'gated' by the social brain? *Dev. Sci.* **10**, 110–120. (doi:10.1111/j.1467-7687.2007.00572.x)
- Petersson KM, Reis A. 2006 Characteristics of illiterate and literate cognitive processing: implications for brain-behavior co-constructivism. In *Lifespan development and the brain: the perspective of biocultural co-constructivism* (eds PB Baltes, F Rosler, PA Reuter Lorenz), pp. 279–305. New York, NY: Cambridge University Press.
- Heyes C. 2012 Grist and mills: on the cultural origins of cultural learning. *Phil. Trans. R. Soc. B* **367**, 2181–2191. (doi:10.1098/rstb.2012.0120)
- Nielsen M, Subiaul F, Galef B, Zentall T, Whiten A. 2012 Social learning in humans and nonhuman animals: theoretical and empirical dissections. *J. Comp. Psychol.* **126**, 109–113. (doi:10.1037/a0027758)
- Rogers SJ, Williams JHG. 2006 *Imitation and the social mind: autism and typical development*. New York, NY: Guilford Press.
- Rogers S, Pennington B. 1991 A theoretical approach to the deficits in infantile autism. *Dev. Psychopathol.* **3**, 137–162. (doi:10.1017/S0954579400000043)
- Paulus M, Hunnius S, Bekkering H. 2012 Examining functional mechanisms of imitative learning in infancy: does teleological reasoning affect infants' imitation beyond motor resonance? *J. Exp. Child Psychol.* **116**, 487–498. (doi:10.1016/j.jecp.2012.10.009)
- Sommerville JA, Woodward AL. 2005 Pulling out the intentional structure of action: the relation between action processing and action production in infancy. *Cognition* **95**, 1–30. (doi:10.1016/j.cognition.2003.12.004)
- Piaget J. 1953 *The origin of intelligence in the child*. London, UK: Routledge.
- Shook J. 2013 Social cognition and the problem of other minds. In *Handbook of neurosociology* (eds D Franks, J Turner), pp. 33–46. New York, NY: Springer.
- Hobson RP. 2004 *The cradle of thought*. London, UK: Pan Macmillan.
- Meltzoff AN, Decety J. 2003 What imitation tells us about social cognition: a rapprochement between developmental psychology and cognitive neuroscience. *Phil. Trans. R. Soc. Lond. B* **358**, 491–500. (doi:10.1098/rstb.2002.1261)
- Syal S, Finlay BL. 2011 Thinking outside the cortex: social motivation in the evolution and development of language. *Dev. Sci.* **14**, 417–430. (doi:10.1111/j.1467-7687.2010.00997.x)
- Tomasello M, Vaish A. 2013 Origins of human cooperation and morality. *Annu. Rev. Psychol.* **64**, 231–255. (doi:10.1146/annurev-psych-113011-143812)
- Nielsen M. 2008 The social motivation for social learning. *Behav. Brain Sci.* **31**, 33. (doi:10.1017/S0140525X0700324X)
- Senju A, Tucker L, Pasco G, Hudry K, Elsabbagh M, Charman T, Johnson MH. 2013 The importance of the eyes: communication skills in infants of blind parents. *Proc. R. Soc. B* **280**, 20130436. (doi:10.1098/rspb.2013.0436)
- Hamlin JK, Wynn K. 2012 Who knows what's good to eat? Infants fail to match the food preferences of antisocial others. *Cogn. Dev.* **33**, 227–239. (doi:10.1016/j.cogdev.2012.05.005)
- Nielsen M, Simcock G, Jenkins L. 2008 The effect of social engagement on 24-month-olds' imitation from live and televised models. *Dev. Sci.* **11**, 722–731. (doi:10.1111/j.1467-7687.2008.00722.x)
- Frith CD. 2008 Social cognition. *Phil. Trans. R. Soc. B* **363**, 2033–2039. (doi:10.1098/rstb.2008.0005)
- Over H, Carpenter M. 2012 Putting the social into social learning: explaining both selectivity and fidelity in children's copying behavior. *J. Comp. Psychol.* **126**, 182–192. (doi:10.1037/a0024555)
- Csibra G, Gergely G. 2009 Natural pedagogy. *Trends Cogn. Sci.* **13**, 148–153. (doi:10.1016/j.tics.2009.01.005)
- Wang Y, de C. Hamilton AF. 2012 Social top-down response modulation (STORM): a model of the control of mimicry in social interaction. *Front. Hum. Neurosci.* **6**, 153. (doi:10.3389/fnhum.2012.00153)
- American Psychiatric Association. 2013 *Diagnostic and statistical manual of mental disorders*, 5th edn. Arlington, VA: American Psychiatric Publishing.
- Dawson G, Bernier R. 2007 Social brain circuitry in autism. In *Human behavior and the developing brain* (eds D Coch, G Dawson, K Fischer), pp. 28–55. New York, NY: Guilford Press.
- Mundy P. 2011 The social behavior of autism: a parallel and distributed information processing perspective. In *Autism spectrum disorders* (eds

- DG Amaral, Dawson, DH Geschwind), pp. 149–171. New York, NY: Oxford University Press.
27. Vivanti G, Hamilton A. 2014 Imitation in autism spectrum disorders. In *Handbook of autism and pervasive developmental disorders*, 4th edn (eds FR Volkmar, R Paul, SJ Rogers, K Pelphrey), pp. 278–301. Hoboken, NJ: Wiley.
  28. Vivanti G, Barbaro J, Hudry K, Dissanayake C, Prior M. 2013 Intellectual development in autism spectrum disorders: new insights from longitudinal studies. *Front. Hum. Neurosci.* **7**, 354. (doi:10.3389/fnhum.2013.00354)
  29. Howlin P, Goode S, Hutton J, Rutter M. 2004 Adult outcome for children with autism. *J. Child Psychol. Psychiatry* **45**, 212–229. (doi:10.1111/j.1469-7610.2004.00215.x)
  30. Howlin P, Moss P, Savage S, Rutter M. 2013 Social outcomes in mid- to later adulthood among individuals diagnosed with autism and average nonverbal IQ as children. *J. Am. Acad. Child Adolesc. Psychiatry* **52**, 572–581. (doi:10.1016/j.jaac.2013.02.017)
  31. Cattaneo L, Fabbri-Destro M, Boria S, Pieraccini C, Monti A, Cossu G, Rizzolatti G. 2007 Impairment of actions chains in autism and its possible role in intention understanding. *Proc. Natl Acad. Sci. USA* **104**, 17 825–17 830. (doi:10.1073/pnas.0706273104)
  32. Klin A, Jones W, Schultz R, Volkmar F. 2003 The enactive mind, or from actions to cognition: lessons from autism. *Phil. Trans. R. Soc. Lond. B* **358**, 345–360. (doi:10.1098/rstb.2002.1202)
  33. Nuske H, Vivanti G, Dissanayake C. 2013 Normative reactivity to the emotions of familiar people in young children with autism spectrum disorder. In *Paper presented at the International Meeting for Autism Research (IMFAR), 2–4 May 2013, San Sebastián, Spain*. Hartford, CT: INSAR.
  34. Nuske HJ, Vivanti G, Dissanayake C. 2013 Are emotion impairments unique to, universal, or specific in autism spectrum disorder? A comprehensive review. *Cogn. Emot.* **27**, 1042–1061. (doi:10.1080/02699931.2012.762900)
  35. Vivanti G, Trembath D, Dissanayake C. 2014 Atypical monitoring and responsiveness to others' goal-directed behaviour in Autism Spectrum Disorder. *Exp. Brain Res.* **232**, 695–701. (doi:10.1007/s00221-013-3777-9)
  36. Oberman LM, Winkielman P, Ramachandran VS. 2009 Slow echo: facial EMG evidence for the delay of spontaneous, but not voluntary, emotional mimicry in children with autism spectrum disorders. *Dev. Sci.* **12**, 510–520. (doi:10.1111/j.1467-7687.2008.00796.x)
  37. Chevallier C, Kohls G, Troiani V, Brodwin ES, Schultz RT. 2011 The social motivation theory of autism. *Trends Cogn. Sci.* **16**, 231–239. (doi:10.1016/j.tics.2012.02.007)
  38. Liebal K, Colomby C, Rogers SJ, Warneken F, Tomasello M. 2008 Helping and cooperation in children with autism. *J. Autism Dev. Disord.* **38**, 224–238. (doi:10.1007/s10803-007-0381-5)
  39. Maestro S, Muratori F, Cavallaro MC, Pei F, Stern D, Golse B, Palacio-Espasa F. 2002 Attentional skills during the first 6 months of age in autism spectrum disorder. *J. Am. Acad. Child Adolesc. Psychiat.* **41**, 1239–1245. (doi:10.1097/00004583-200210000-00014)
  40. Maestro S, Muratori F, Cavallaro MC, Pecini C, Cesari A, Paziente A, Stern D, Golse B, Palacio-Espasa F. 2005 How young children treat objects and people: an empirical study of the first year of life in autism. *Child Psychiatry Hum. Dev.* **35**, 383–396. (doi:10.1007/s10578-005-2695-x)
  41. Pierce K, Conant D, Hazin R, Stoner R, Desmond J. 2011 Preference for geometric patterns early in life as a risk factor for autism. *Arch. Gen. Psychiatry* **68**, 101–109. (doi:10.1001/archgenpsychiatry.2010.113)
  42. D'Entremont B, Yazbek A. 2007 Imitation of intentional and accidental actions by children with autism. *J. Autism Dev. Disord.* **37**, 1665–1678. (doi:10.1007/s10803-006-0291-y)
  43. Spengler S, Bird G, Brass M. 2010 Hyperimitation of actions is related to reduced understanding of others' minds in autism spectrum conditions. *Biol. Psychiatry* **68**, 1148–1155. (doi:10.1016/j.biopsych.2010.09.017)
  44. Hume K, Plavnick J, Odom SL. 2012 Promoting task accuracy and independence in students with autism across educational setting through the use of individual work systems. *J. Autism Dev. Disord.* **42**, 2084–2099. (doi:10.1007/s10803-012-1457-4)
  45. Barbaro J, Dissanayake C. 2007 A comparative study of the use and understanding of self-presentational display rules in children with high functioning autism and Asperger's disorder. *J. Autism Dev. Disord.* **37**, 1235–1246. (doi:10.1007/s10803-006-0267-y)
  46. Grossi D, Marcone R, Cinquegrana T, Gallucci M. 2012 On the differential nature of induced and incidental echolalia in autism. *J. Intellect. Disabil. Res.* **57**, 903–912. (doi:10.1111/j.1365-2788.2012.01579.x)
  47. Jones EJ, Webb SJ, Estes A, Dawson G. 2013 Rule learning in autism: the role of reward type and social context. *Dev. Neuropsychol.* **38**, 58–77. (doi:10.1080/87565641.2012.727049)
  48. Williams JH, Whiten A, Suddendorf T, Perrett DI. 2001 Imitation, mirror neurons and autism. *Neurosci. Biobehav. Rev.* **25**, 287–295. (doi:10.1016/S0149-7634(01)00014-8)
  49. Iacoboni M, Dapretto M. 2006 The mirror neuron system and the consequences of its dysfunction. *Nat. Rev. Neurosci.* **7**, 942–951. (doi:10.1038/nrn2024)
  50. von Hofsten C, Rosander K. 2012 Perception-action in children with ASD. *Front Int. Neurosci.* **6**, 1–6.
  51. Rizzolatti G, Craighero L. 2004 The mirror-neuron system. *Annu. Rev. Neurosci.* **27**, 169–192. (doi:10.1146/annurev.neuro.27.070203.144230)
  52. Iacoboni M. 2008 *Mirroring people: the new science of how we connect with others*, 1st edn. New York, NY: Farrar, Straus and Giroux.
  53. Rizzolatti G, Sinigaglia C. 2008 *Mirrors in the brain: how our minds share actions and emotions*. Oxford, UK: Oxford University Press.
  54. Winkielman P, McIntosh DN, Oberman L. 2009 Embodied and disembodied emotion processing: learning from and about typical and autistic individuals. *Emotion Rev.* **1**, 178–190. (doi:10.1177/1754073908100442)
  55. Bernier R, Dawson G, Webb S, Murias M. 2007 EEG mu rhythm and imitation impairments in individuals with autism spectrum disorder. *Brain Cogn.* **64**, 228–237. (doi:10.1016/j.bandc.2007.03.004)
  56. Dapretto M, Davies MS, Pfeifer JH, Scott AA, Sigman M, Bookheimer SY, Iacoboni M. 2006 Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. *Nat. Neurosci.* **9**, 28–30. (doi:10.1038/nn1611)
  57. Hadjikhani N, Joseph RM, Snyder J, Tager-Flusberg H. 2006 Anatomical differences in the mirror neuron system and social cognition network in autism. *Cereb. Cortex* **16**, 1276–1282. (doi:10.1093/cercor/bhj069)
  58. Enticott PG, Kennedy HA, Rinehart NJ, Tonge BJ, Bradshaw JL, Taffe JR, Daskalakis ZJ, Fitzgerald PB. 2012 Mirror neuron activity associated with social impairments but not age in autism spectrum disorder. *Biol. Psychiatry* **71**, 427–433. (doi:10.1016/j.biopsych.2011.09.001)
  59. Dinstein I, Thomas C, Humphreys K, Minshew N, Behrmann M, Heeger DJ. 2010 Normal movement selectivity in autism. *Neuron* **66**, 461–469. (doi:10.1016/j.neuron.2010.03.034)
  60. Fan YT, Decety J, Yang CY, Liu JL, Cheng YW. 2010 Unbroken mirror neurons in autism spectrum disorders. *J. Child Psych. Psychiatry* **51**, 981–988. (doi:10.1111/j.1469-7610.2010.02269.x)
  61. Enticott PG, Kennedy HA, Rinehart NJ, Bradshaw JL, Tonge BJ, Daskalakis ZJ, Fitzgerald PB. 2013 Interpersonal motor resonance in autism spectrum disorder: evidence against a global mirror system deficit. *Front. Hum. Neurosci.* **7**, 218. (doi:10.3389/fnhum.2013.00218)
  62. Oberman LM, Ramachandran VS. 2007 The simulating social mind: the role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychol. Bull.* **133**, 310–327. (doi:10.1037/0033-2909.133.2.310)
  63. Colomby C, Vivanti G, Rogers S. 2011 The neuropsychology of imitation deficit in autism. In *The neuropsychology of autism* (ed. D Fein), pp. 243–266. Oxford, UK: Oxford University Press.
  64. Rogers SJ, Dawson G. 2010 *Early Start Denver Model for young children with autism: promoting language, learning, and engagement*. New York, NY: Guilford Press.
  65. Stern DN. 1985 *The interpersonal world of the infant: a view from psychoanalysis and developmental psychology*. New York, NY: Basic Books.
  66. Dawson G. 2008 Early behavioral intervention, brain plasticity, and the prevention of autism spectrum

- disorder. *Dev. Psychopathol.* **20**, 775–803. (doi:10.1017/S0954579408000370)
67. Mundy P, Burnette C. 2005 Joint attention and neuro developmental models of autism. In *Handbook of autism and pervasive developmental disorders*, vol. 1 (eds F Volkmar, R Paul, A Klin, D Cohen), pp. 650–681. Hoboken, NJ: John Wiley & Sons.
  68. Vismara LA, Rogers SJ. 2010 Behavioral treatments in autism spectrum disorder: what do we know? *Annu. Rev. Clin. Psychol.* **6**, 447–468. (doi:10.1146/annurev.clinpsy.121208.131151)
  69. Dawson G, Rogers S, Munson J, Smith M, Winter J, Greenson J, Donaldson A, Varley J. 2010 Randomized, controlled trial of an intervention for toddlers with autism: the Early Start Denver Model. *Pediatrics* **125**, e17–e23. (doi:10.1542/peds.2009-0958)
  70. Keysers C, Perrett DI. 2004 Demystifying social cognition: a Hebbian perspective. *Trends Cogn. Sci.* **8**, 501–507. (doi:10.1016/j.tics.2004.09.005)
  71. Heyes C. 2010 Where do mirror neurons come from? *Neurosci. Biobehav. Rev.* **34**, 575–583. (doi:10.1016/j.neubiorev.2009.11.007)
  72. Casile A, Caggiano V, Ferrari PF. 2011 The mirror neuron system: a fresh view. *Neuroscientist* **17**, 524–538. (doi:10.1177/1073858410392239)
  73. Trevarthen C. 1979 Communication and cooperation in early infancy: a description of primary intersubjectivity. In *Before speech: the beginning of interpersonal communication* (ed. M Bullowa), pp. 321–348. New York, NY: Cambridge University Press.
  74. Jones W, Klin A. 2013 Attention to eyes in present but in decline in 2–6 month-old infants later diagnosed with autism. *Nature* **504**, 427–431. (doi:10.1038/nature12715)
  75. Williams JH. 2008 Self-other relations in social development and autism: multiple roles for mirror neurons and other brain bases. *Autism Res.* **1**, 73–90. (doi:10.1002/aur.15)
  76. Dawson G *et al.* 2012 Early behavioral intervention is associated with normalized brain activity in young children with autism. *J. Am. Acad. Child Adolesc. Psychiatry* **51**, 1150–1159. (doi:10.1016/j.jaac.2012.08.018)
  77. Voos AC, Pelphrey KA, Tirrell J, Bolling DZ, Vander Wyk B, Kaiser MD, McPartland JC, Volkmar FR, Ventola P. 2012 Neural mechanisms of improvements in social motivation after pivotal response treatment: two case studies. *J. Autism Dev. Disord.* **43**, 1–10. (doi:10.1007/s10803-012-1683-9)
  78. Oberman LM, Ramachandran VS, Pineda JA. 2008 Modulation of mu suppression in children with autism spectrum disorders in response to familiar or unfamiliar stimuli: the mirror neuron hypothesis. *Neuropsychologia* **46**, 1558–1565. (doi:10.1016/j.neuropsychologia.2008.01.010)
  79. Vivanti G, McCormick C, Young GS, Abucayan F, Hatt N, Nadiq A, Ozonoff S, Rogers SJ. 2011 Intact and impaired mechanisms of action understanding in autism. *Dev. Psychol.* **47**, 841–856. (doi:10.1037/a0023105)
  80. McCormick C, Young GS, Rogers SJ. 2013 Assessing the effect of reward on learning: a novel eye-tracking marker of treatment outcome. In *Paper presented at the International Meeting for Autism Research (IMFAR), 2–4 May 2013, San Sebastián, Spain*. Hartford, CT: INSAR.