

Understanding the mechanisms behind deficits in imitation:

Do individuals with autism know 'what' to imitate and do they know 'how' to imitate?

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Running head Selection or correspondence problem?

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Abstract

Although imitation problems have been associated with autism for many years, the underlying mechanisms of these problems remain subject to debate. In this article, the question whether imitation problems are caused by selection or correspondence problems is explored and discussed. This review revealed that hypotheses on the nature of imitation problems in autism are complicated and inconclusive at the present time. There is some evidence for impaired selection, especially implicating poor preferential attention to biological motion and poor ascription of intention to action. There is also some evidence that both transformations of perspectives and mapping of visual to motor information are impaired, characterized as correspondence problems. However, it is not yet clear how poor selection processes contribute to correspondence problems and vice versa. Insight in this interaction may provide a valuable contribution to our understanding of imitation problems in autism. For further research we recommend that tasks should be constrained to target as few mechanisms as possible in given experiments.

Key words: Imitation; Underlying mechanisms; Selection processes; Correspondence processes

1. Introduction

Motor imitation is defined as the capacity of an individual to replicate an observed motor act. It requires the ability to transform visual-perceptual information into a motor copy of it (Prinz, 2002). It is a neurocognitive process that powers cognitive and social development in infancy and childhood; that promotes empathy, cooperation and well-being in our relationships with others; and provides a channel of evolutionary, cultural inheritance that makes us distinctively human (Heyes, 2009). The neurological circuit of motor imitation contrasts with that of vocal imitation at least at the stimulus input level (visual versus auditory input) and feedback level (kinesthetic and occasionally visual feedback versus auditory feedback) (Masur, 2006). This rejects the frequently made remark that the excessive vocal imitation or echolalia, described by McEvoy (1988) is not compatible with the impaired motor imitation in individuals with autism. In the present review the term motor imitation refers to the imitation of actions with and without objects. These actions can be goal-directed and non-goal-directed, respectively meaningful and non-meaningful. The paper reviews recent research relevant to problems of motor imitation in individuals with autism and considers these problems from two main perspectives, termed the "selection process" and the "correspondence process." In general, the former refers to stimulus input ("what" to imitate) and the latter to motor output ("how" to imitate).

Core theories of motor imitation (hereafter "imitation") can be divided into two main frameworks: the framework of separate and of common representational coding. The first framework assumes that perception and action have independent representational formats. The most prominent model according to this framework is the Active Intermodal Mapping (AIM) model of imitation (Meltzoff & Moore, 1997). The AIM-model proposes that visually perceived acts are actively mapped onto motor output via a supramodal representation system.

The second framework assumes that codes related to perception and action share a common representational domain. This common-coding or direct mapping approach, states that the motor system is directly activated by the perception of an action. The motor system of the imitator receives direct input from observing the demonstrator's movement. This framework has generated several theories of imitation. In the Ideomotor Theory of Imitation, the observer acts what he sees, i.e., perceptual induction (Prinz, 1997; Prinz, 2002) or what he would like to see, i.e., intentional induction (Prinz, 2002). The latter is related to the Goal Directed Theory of Imitation, which claims that imitation is guided by goals and that goals are hierarchically organized (Bekkering, Wohlschläger, & Gattis, 2000). Another issue with the ideomotor approach of imitation is whether and how well a person imitates depend on the past experiences of the imitator. This issue is central in the Associative Sequence Learning model of imitation. A person will be able to imitate an observed action, only if he has had the opportunity to form a link between visual and motor representations of this particular action by sensorimotor experiences (Heyes, 2001; Heyes & Ray, 2004). To resolve seemingly contradictory ideas of previous models, the Dual Route Theory of Imitation was forwarded. This theory assumes that the pattern of imitation depends on the type of the extrinsic properties of an action presented. For that reason, the Dual Route Theory distinguishes two distinctive routes for imitation: the direct and indirect route (Rumiati & Tessari, 2002). The direct non-linguistic mediated route is used for the imitation of novel non-meaningful and non-goal directed actions. The indirect linguistically mediated route is used for the imitation of well-trained familiar meaningful or goal directed actions (Rumiati & Tessari, 2002).

Although imitation problems have been associated with autism for many years, the issue of whether these problems are a core deficit in autism is yet to be determined (see recent reviews of Williams, Whiten, & Singh, 2004; Seveler & Gillis, 2010; Vanvuchelen, Roeyers, & De Weerd, 2011a). In their meta-analysis Williams and colleagues (2004) pooled the

findings from twelve well-controlled case-control studies, involving 196 individuals with autism. They calculated the combined p -value of group differences with respect to imitation problems to an appropriate control group, resulting in a p -value of .00002 (Williams, Whiten, & Singh, 2004). Sevillever and Gillis (2010) discussed imitation problems in autism from a methodological perspective. The authors recommended a comparative taxonomy of imitation, a standardized methodology across researchers, and a standardized imitation battery for children with autism to improve imitation research in this population (Sevillever & Gillis, 2010). Vanvuchelen and colleagues addressed the question whether autism problems fulfil the criteria of uniqueness, specificity, universality, persistency, precedence and broadness. The findings of this review suggest that there is only partial evidence for the idea that imitation problems are unique, specific and broad to autism, and that these problems are long-lasting and persistent. In addition, imitation problems seem not to be universal in autism at an early age. Mental and motor impairment may affect imitation performance but they do not seem to explain imitation problems in a sufficient way (Vanvuchelen, Roeyers, & De Weerd, 2011a). The findings of the same research group suggest that delay in imitation of actions with objects that go beyond the nonverbal mental delay may predict the diagnosis of autism at preschool age (Vanvuchelen, Roeyers, & De Weerd, 2011b).

The present review lays out several current and some older theories related to the processes behind the imitation problems seen in many individuals with autism. It provides an overview of important advances in autism imitation research summarizing the state of play with respect to two key questions: Do individuals with autism know 'what' to imitate? And do they know 'how' to imitate? Imitation appears to result from the interaction of two distinct cognitive processes: the selection and the correspondence process (Breazeal & Scassellati, 2002; Lopes & Santos-Victor, 2005). Solving the selection problem is based on non-specific mechanisms which are involved in both imitative and non-imitative tasks, including social

attention and motivation, visual attention, biological motion preference, action and intention recognition. On the contrary, the correspondence problem is solved by specific mechanisms which are exclusively involved in imitative tasks. Viewpoint transformation and visuomotor mapping are typical to converse observed actions into executed actions. There is relatively more research in the field of these specific mechanisms.

2. Are imitation problems in autism the result of difficulties in the selection process?

The question “how does an individual know ‘what’ to imitate?” may be coined as the selection problem. When imitating another person, how does the individual determine which visual-perceptual aspects are relevant to the task? The individual needs to detect the demonstrator, observe his actions, and determine which elements are part of the instructional process and which are circumstantial. This is a challenge for the visual-perceptual system. It involves not only the ability to perceive human movements, but also the capability to direct attention and to determine the salient points. The question “Do individuals with autism know ‘what’ to imitate?” leads to five hypotheses to explain the underlying mechanisms of imitation problems in autism.

2.1. The social hypothesis

According to the social hypothesis, imitation problems in autism may be caused by poor social attention and poor social motivation. Compared to developmentally matched non-autistic controls, children with autism more frequently fail to orient to social and non-social stimuli. This failure is most pronounced for social stimuli (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998). This may result in a lack of attention to social partners, in particular

the demonstrator, and thus precludes the development of imitation. However, some experimental autism imitation studies have controlled for social attentiveness to the demonstrator. They found that the autism group showed a similar attentiveness to the demonstrator as the control group (Meyer & Hobson, 2004; Rogers, Hepburn, Stackhouse, & Wehner, 2003). Meyer and Hobson (2004) compared an autism group and a learning disabled control group matched on both chronological and verbal mental age, for amount of copying behavior before analyzing the quality of imitation. The group difference in quality of imitation was not determined by global inattentiveness on the part of children with autism. Rogers and colleagues (2003) compared imitation abilities of pre-schoolers with autism, fragile X syndrome, other developmental disorders, with those of typically-developing children. The scores on the imitation battery were first examined for level of subject participation in the test procedure to make sure that the findings were not influenced by a lack of response from any particular group, particularly the children with autism. The diagnostic groups did not differ in their participation in the imitation battery (Rogers et al., 2003).

Dawson and Adams (1984) ruled out a motivational explanation underlying autistic children's general failure to engage with other people. They demonstrated that autistic children's attention to and their interactions with people could be enhanced by manipulating the partner's responses. They found that children with autism were more socially responsive, showed more eye contact, and played with toys in a less perseverative manner, when the experimenter imitated their behavior (Dawson & Adams, 1984). Some experimental autism imitation studies have controlled for motivation. These studies demonstrated consistently that children with autism engage well with the imitation tasks (Libby, Powell, Messer, & Jordan, 1997; Meyer & Hobson, 2004; Rogers et al., 2003; Vanvuchelen, Roeyers, & De Weerd, 2007a). Libby and colleagues found that children with autism were less negative than younger mental age matched children with Down syndrome and typically developing children (Libby

et al., 1997). In Vanvuchelen et al. (2007a) school-aged children with autism made more attempts to imitate than non-autistic controls. This finding suggests that the children with autism made an effort to comply. It has to be mentioned that the results of these social attentiveness and motivational studies were obtained in an elicited imitation condition. Therefore, it is not possible to extrapolate these findings to spontaneous imitation conditions.

2.2. The visual attentiveness hypothesis

Direct evidence of primary visual search and oculomotor problems in autism is lacking (Brenner, Turner, & Muller, 2007). Vivanti and colleagues (2008) investigated whether differences in visual attention when observing an action to be imitated may contribute to imitative difficulties in autism in both non-meaningful gestures and goal directed actions on objects. Results of eye-tracking analyses indicated that a group of high-functioning 8- to 15-year-olds with autistic disorder, in comparison with a chronological, nonverbal mental and language age matched group of typically developing children, showed reduced attention to the demonstrator's face, but similar patterns of visual attention when observing a demonstrator perform an action. Different action types triggered distinct visual attention patterns that did not differ between groups. The autism group demonstrated reduced imitative precision for both action types. The duration of visual attention to the demonstrator's action was related to imitation precision for non-meaningful gestures, but not for goal directed actions on objects in the autism group. This relationship was not found in the typically developing comparison group, and this may have been due to the near-ceiling level of imitation precision in this group (Vivanti, Nadig, Ozonoff, & Rogers, 2008). Overall there is no clear evidence for an impaired visual attention deficit underlying autism imitation problems.

2.3. *The biological motion preference hypothesis*

Human vision not only detects motion directions in human beings, but also distinguishes different standard types of limb motion patterns, such as walking, running and dancing. Such motion patterns are termed as biological motion (Johansson, 1973; Pelphrey & Carter, 2008). Preferential attention to biological motion is a fundamental mechanism facilitating adaptive interaction with other living beings (Klin, Lin, Gorrindo, Ramsay, & Jones, 2009). The study of biological motion has traditionally used the Johansson's paradigm of human motion display. The motion of the body is represented by a few bright spots describing the motions of the main joints. The motion pattern is dissociated from the form of people's bodies (Johansson, 1973). The moving point lights evoke a compelling impression of basic human movements (e.g., walking, running, dancing) as well as of emotional movements (e.g., approaching, fighting, embracing).

A number of studies have explored whether individuals with autism have compromised perception of biological motion, and the results are not entirely consistent. Klin and colleagues used Johansson point-light displays to depict a series of social approaches that are part of the typical experience of young children (e.g., 'pat-a-cake', 'peek-a-boo'). The authors found that two-years-olds with autism, compared to typically developing peers, failed to orient towards biological motions (Klin & Jones, 2008; Klin, Jones, Schultz, & Volkmar, 2003; Klin et al., 2009). Blake and colleagues (2003) employed Johansson-type stimuli for non-emotional human movements. They reported impaired biological motion recognition in school-aged mentally impaired children with autism compared to younger typically developing controls. The correlation between severity of autism, as indicated by both the ADOS-G and CARS total scores, and the score on the biological motion test, indicates a significant relationship between severity of autistic symptoms and performance on the biological motion test (Blake, Turner, Smoski, Pozdol, & Stone, 2003). In the above

mentioned studies (Blake et al., 2003; Klin & Jones, 2008; Klin et al., 2003; Klin et al., 2009) individuals with autism did show an increased error rate and not a complete lack of biological motion preference and recognition.

In other studies which have included child, adolescent and adult populations, individuals with autism differed from controls in their ability to recognize emotions, but not in their ability to describe actions. These studies have suggested that individuals with autism are unaffected in the perceptual processing of form-from-motion, but may exhibit impairments in higher order judgments such as emotion processing (Atkinson, 2009; Hubert et al., 2007; Moore, Hobson, & Lee, 1997; Parron, Da, Santos, Moore, Monfardini, & Deruelle, 2008). Overall, although studies have yielded inconsistent findings, there is some evidence of an impairment to perceive biological motion in individuals with autism.

2.4. Action recognition hypothesis

The group of Bartak (1975) postulated that children with autism show poorer understanding and expression of meaningful gestures than children with receptive language disorders (Bartak, Rutter, & Cox, 1975). Baron-Cohen (1988) also concluded that individuals with autism were more impaired than mental age matched controls only on imitation of symbolic meaningful actions (Baron-Cohen, 1988).

By contrast, Rogers and Pennington (1991) found autism-specific deficits in imitation of non-meaningful gestures. Subsequently, well-controlled experiments have confirmed the latter view. Moreover, meaningful contents improved the imitation performances of children with autism (Charman, Swettenham, Baron-Cohen, Cox, Baird, & Drew, 1997; Hobson & Lee, 1999; Rogers, Bennetto, McEvoy, & Pennington, 1996; Smith & Bryson, 2007; Stone, Ousley, & Littleford, 1997; Vanvuchelen, et al., 2007a, Vanvuchelen, Roeyers, & De Weerd, 2007b).

2007b). Another argument of an intact action recognition system is the finding of body-part-as-an-object errors in transitive gestural imitations of school-aged children with autism. The children proved that they recognized an imaginary tool and comprehended the meaning of the action with this tool. They performed the gesture in a simpler manner without losing the meaning (Vanvuchelen et al., 2007a). Other studies have explicitly examined memory for the observed acts (Rogers et al., 1996; Smith & Bryson, 1998). No study reported any group difference in the ability of individuals with autism to remember the tasks correctly over time. Overall, these findings suggest that the action representation and memory system, commonly termed as the action recognition system, are not the source of the autism imitation problems.

2.5. *Intention recognition hypothesis*

In contrast to the similar patterns of visual attention to the demonstrator's actions, Vivanti and colleagues (2008) results of eye-tracking analyses indicated that children with autism showed decreased attention to the demonstrator's face when observing a model to be imitated. Attention to the face is essential to pick up ostensive cues from the demonstrator's intentions.

To determine whether children with autism would selectively imitate intentional, as opposed to accidental actions, an experimenter demonstrated either an "intentional" or an "accidental" action or two "intentional" actions on the same toy. Intentional actions were marked by the experimenter saying "There". Accidental actions were marked by saying "Whoops" (Carpenter, Akhtar, & Tomasello, 1998). Three to five year old children with autism tended not to differentiate between intentional and accidental actions. The demonstrator's actions drew attention to the affordances of the objects and the children with autism then performed the actions that the objects afforded. This mechanism is called

stimulus enhancement. In contrast, younger typically developing children mostly produced only the intentional action. It was concluded that, contrary to comparison groups, the children with autism did not show an appreciation of the demonstrator's intentions (D'Entremont & Yazbek, 2007). So, there is some evidence of an impairment to perceive and recognize intentional actions in children with autism.

3. Are imitation problems in autism the result of difficulties in the correspondence process?

The question “how does an individual know ‘how’ to imitate?” may be coined as the correspondence problem. How are the perceived movements of another agent translated into similar performances by the observer? How are the movements organized into a temporal and spatial pattern that replicates what is shown by the demonstrator? How are associations formed between the visual-perceptual and motor representations of an action?

Among the wide diversity of actions that human beings can imitate, non-meaningful gestures pose a particular challenge for our brain. Similarity between the actions of the demonstrator and one’s own actions can be derived neither from their common meaning nor from the similarity of their impact on external objects. It must result from direct matching between the own body configuration and that of the demonstrator (Heyes, 2009). Once a relevant action has been selected and perceived, the person with autism must convert his perception into its own motor response. The key question “Do individuals with autism know ‘how’ to imitate?” leads to two hypotheses to explain the underlying mechanisms of autism imitation problems.

3.1. The viewpoint transformation hypothesis

For the imitation of non-meaningful hand postures the imitator has not only to detect visually the demonstrator's hand, but also to conceive a "mental rotation". That will place the demonstrator's hand (allo-image) in correspondence with the learner's own body (ego-image) (Lopes & Santos-Victor, 2005). Viewpoint transformation may be considered as a kind of visual-perceptual perspective taking ability and a developmental foundation for the later emergence of higher-order perspective taking skills in human social cognition (Meltzoff, 2007).

Without an appropriate viewpoint transformation, one would expect reversal errors during imitation. Such errors imply that a person holds up the palm of his hand facing towards himself when he has observed the demonstrator facing his palm of the hand towards the other person. A number of studies reported reversal errors in imitation of actions without objects (Beadle-Brown, 2004; Dewey, Cantell, & Crawford, 2007; Ohta, 1987, Smith & Bryson, 1998; Whiten & Brown, 1998) and actions with objects (Avikainen, Wohlschlager, Liuhanen, Hanninen, & Hari, 2003; Hobson & Lee, 1999; Meyer & Hobson, 2004) in children and adolescents with autism in contrast to non-autistic controls. Compared to appropriate controls, they were significantly less likely to imitate the orientation of an action in relation to the demonstrator's body. This phenomenon did not take place in the age-and IQ-matched control groups, but became apparent in younger typically developing children. In contrast, Vanvuchelen and colleagues (2007a) did not find evidence for reversal errors in school-aged children with autism. It has to be mentioned that in their approach errors that did not occur in more than 10% of the observations were considered as less relevant and not further analyzed regarding group differences. Their finding that reversal errors are exceptional and that an impaired viewpoint transformation cannot explain imitation problems in autism is consistent with the results of the research group of Green (Green, Baird, Barnett, Henderson, Huber, & Henderson, 2002) and Mostofsky (Mostofsky, Dubey, Jerath, Jansiewicz, Goldberg, &

Denckla, 2006). In addition, the finding in the study of Mostofsky et al. (2006), that children with autism showed a better gesture performance in the imitation condition than on verbal command, supports this view. It is likely due to abnormalities in processes common to both conditions, such as mapping the precise kinesthetic spatial aspects of movements or the planning of goal directed actions (Mostofsky et al., 2006). Overall, there is some but no clear evidence for an impaired viewpoint transformation underlying autism imitation problems.

3.2. *The visuomotor map hypothesis*

The visuomotor map converts the visual features directly to motor data (Lopes & Santos-Victor, 2005). Rogers and Pennington (1991) were the first to postulate a primary self-other mapping problem in autism. They suggested that a biological impairment in autism restricted the capacity of the infant to self-other correspondence. Williams and colleagues reviewed the self-other correspondence hypothesis of Rogers and Pennington (1991) alongside recent findings in the field of neuroscience. They were the first to propose the idea that a defect in the human mirror neuron system (hMNS) may cause the self-other correspondence problem. They suggest that early developmental failures of the hMNS are likely to result in a cascade of developmental impairments (Williams, Whiten, Suddendorf, & Perrett, 2001).

Studies of hMNS in autism have postulated two different hypotheses. First, researchers hypothesized that the primary autism deficit is a lack of responsiveness of the hMNS to the observation of others' actions (Gallese, 2006; Williams et al., 2001). In typically developing individuals, internal representations of the body states associated with actions, emotions, and sensations are evoked in the observer, as if he would be performing a similar action or experiencing a similar emotion or sensation. Based on over 200 fMRI studies, the

meta-analysis of Van Overwalle & Baetens (2009) demonstrates that the mirror system - consisting of the anterior intraparietal sulcus and the premotor cortex – is engaged when one perceives articulated motions of body parts as well as when the perceiver executes them. This confirms the matching role of the mirror system in understanding biological action (Van Overwalle & Baetens, 2009). By means of a shared neural state of the hMNS the other becomes “another self”. Individuals with autism lack this direct form of experiential understanding of others, or this intentional attunement (Gallese, 2006). Indirect evidence of McIntosh et al.’s study (2006) using the automatic imitation paradigm supports this view. In tests of automatic imitation, participants are not asked to imitate modelled movements. Instead, they observe actions. The experimenter measures mimicry and automatic imitation of simple movements. The research group of McIntosh (2006) examined automatic mimicry and voluntary imitation of simple emotional facial expressions among adolescents and adults with autism and a typical sample matched on chronological and verbal mental age. Participants viewed pictures of happy and angry expressions while the activity over their cheek and brow muscle region was monitored with electromyography. In contrast to typically developing controls, the participants with autism did not automatically mimic facial expressions. However, both groups showed evidence of successful voluntary facial imitation (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006). The research group of Bird (2007) used non-meaningful hand movements for their automatic imitation tasks. Participants were required to perform a pre-specified hand movement (opening or closing) as soon as they saw a hand stimulus. Hand movements of both a human being and a human-like robot were used. The movement of the hand stimulus was either the same (compatible trials) or the opposite (incompatible trials) of the pre-specified response. Although voluntary actions were performed, any effect of imitation on these actions was automatic in the sense that the participants were neither instructed nor intended to imitate, and in half of the trials

(incompatible trials) imitation leads to poor task performance. Compared to chronological and mental age matched healthy adults, adults who met the criteria for autism regarding clinical judgement and ADOS-G classification, showed an equivalent automatic imitation effect. Responses on compatible trials were faster than those on incompatible trials in all participants. This effect was greater when responses were made to human than to robotic actions (Bird, Leighton, Press, & Heyes, 2007). Anatomical investigations of the brains of adults with autism showed abnormal thinning of the gray matter in cortical areas known as being part of the hMNS, such as ventral premotor, posterior parietal, and superior temporal sulcus cortices. Cortical thinning of these areas correlated with autism symptom severity (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006). In addition, brain imaging studies on action observation in children and adults employing different techniques such as EEG (e.g. Oberman, Hubbard, McCleery, Altschuler, Ramachandran, & Pineda, 2005), TMS (e.g. Theoret, Halligan, Kobayashi, Fregni, Tager-Flusberg, & Pascual-Leone, 2005), fMRI (e.g. Dapretto et al., 2006) and MEG (e.g. Nishitani, Avikainen, & Hari, 2004) showed that individuals with autism might be suffering an action-observation deficit induced by a dysfunction of their hMNS. In contrast with previous studies, other research groups did not find hMNS problems when observing or executing simple hand movements in persons with autism (Avikainen, Kulomaki, & Hari, 1999; Dinstein, Thomas, Humphreys, Minshew, Behrmann, & Heeger, 2010; Fan, Decety, Yang, Liu, & Cheng, 2010; Raymaekers, Wiersema, & Roeyers, 2009; Southgate & Hamilton, 2008).

Second, the research group of Rizzolatti has postulated that the primary deficit is not the responsiveness of the hMNS to the observation of others' actions, but the impaired organization of motor chains underlying the action representations (Cattaneo et al., 2007; Fabbri-Destro, Cattaneo, Boria, & Rizzolatti, 2009). They draw attention to the recent finding of action-constrained parietal mirror neurons in monkeys. These mirror neurons become

maximally activated when the coded motor act is embedded into a specific motor action. For example, action-constrained grasping neurons strongly discharge when grasping a piece of food is followed by bringing it to the mouth, but not when it is followed by placing it into a container. Their activation provides not only information about the fact that an individual is grasping, but also gives clues why the individual is doing this. Through this mechanism of intention understanding the observer is able to predict what will be the final goal of the action. By activating a specific action chain from its very outset, this mechanism allows the observers to have an internal copy of the whole action before its execution, thus enabling them to understand directly the agent's intention. Cattaneo and colleagues have shown that this chained organization exists in typically developing school-aged children, whereas it is impaired in children with autism. When typically developing children moved the hand to reach for food by bringing it to the mouth, there was an increase of the EMG activity of the muscles involved in the mouth opening (reaching-for-eating). This activation was lacking when the child grasped an object to place it into a container (reaching-for-placing). A similar pattern was found during the observation of these actions done by others. Both during the execution and the observation of actions, a behavior radically different from that of typically developing children was found in children with autism. There was no activation of the muscles for mouth-opening during the reaching and grasping phase. Its activation was only found during bringing the food to the mouth. This experiment supports the idea that children with autism are able to understand the “what”, but not the “why” of observed actions. Children with autism systematically attributed to the demonstrator the intention that could be derived by the semantics of the object per se (e.g., an intention to eat when food was shown) regardless of how the object was grasped (Cattaneo et al., 2007). Although studies have yielded inconsistent findings, there is some evidence of visuomotor mapping problems involving the hMNS underlying imitation problems in autism.

4. Conclusion

The primary aim of this paper was to characterize different cognitive processes that may underlie imitation impairment in autism as studied in behavioral studies. An association between autism and imitative impairment might result from dysfunction in the selection and/or the correspondence process. Despite decades of research the underlying mechanisms of imitation problems in autism are unclear. Some evidence is found for an impaired selection mechanism due to a poor preferential attention to biological motion and an impairment to recognize intentional actions. Some evidence is found for an impaired correspondence mechanism due to a poor viewpoint transformation and visuomotor mapping.

Given that there is more than one contender for generating imitation difficulties in autism, it does seem unlikely that this impairment is driven by deficits within a single cognitive mechanism. Further research is necessary to single out possible interactions between selection and correspondence processes. Given this multitude of impaired mechanisms that may contribute to an individual performing poorly on a simple imitation task, the tasks should be constrained to target as few mechanisms as possible in a given experiment. It is our intention to study these interactions by using simple imitation tasks of actions with objects and by close monitoring of both processes. Our action-observation-execution model predicts that when children look at an adult's action upon an object they detect both the adult and the object (detection) and identify critical motor referential cues which characterize the adult's intentionality regarding the object (intention identification). As a result of this style of action observation, similar action patterns are provoked in the children (simulation) and these action patterns provoke spontaneous copying behavior (imitation). We will combine several non-invasive methods, in particular eye-tracking techniques for

assessing the observation style; EMG registrations with surface electrodes for assessing simulation; and comparison of the children's spontaneous actions with the objects before and after the observation of the adults' actions for assessing imitation. Our results may lead to increased insight in altered interactions between selection and correspondence processes in individuals with autism, with an emphasis on young children. In addition, brain imaging studies should investigate the functional (dis)-connectivity between various brain areas involved in these processes.

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