

Running Head: Using Imitation to Engage Mirror Neurons to Treat Autism

An Approach to Treatment for Autism Spectrum Disorders
Using the Sensory and Motor System to Simultaneously Target
the Mirror Neuron System via Imitation
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Abstract

Imitation of others is thought to be a prerequisite to advanced social competencies in humans. Language acquisition, empathy, and Theory of Mind (ToM) all develop as children build a repertoire of motor movements via imitating others. The neurological substrates implicated in this behavior include the mirror neuron system (MNS). Research on children with autism (CWA) has demonstrated significant differences in imitative behavior and brain structure when compared to typically developing children. Given this information, it may prove beneficial to incorporate socially salient models and activities into treatments for autism and focus on imitation of intransitive motor tasks using peers as models. Specifically, imitation of full body actions, with simultaneous sensory input, of peers in a typical environment is discussed.

24 The ability to observe the actions of another and predict the outcome, as well
25 as the intention, and emotion of the observed is a skill most humans acquire
26 naturally. This ability is adaptive for survival in a social species – such as humans –
27 because it allows us to work together toward a shared goal, thus increasing our
28 chance of accomplishing the goal (Rizzolatti & Craighero, 2004). Additionally,
29 learning is facilitated through imitation as we develop a sense of self versus others
30 and procure the tools needed to survive in human culture (Meltzoff, 2007; Rizzolatti
31 & Craighero, 2004). As we observe others we come to recognize how and why we
32 might perform the same action, eventually giving us the ability to predict the
33 intention and actions of those around us based on our own experience (Meltzoff,
34 2007). Presently, it is believed that the neural substrates most readily involved in
35 this behavior are a network of neurons known as mirror neurons. However, there is
36 much debate as to the degree of involvement, which will be discussed later.

37 Impaired imitation in children has been linked to deficits in many areas, including
38 the development of language, theory of mind, empathy and social cognition, which
39 are also many of the manifestations of autism. Research examining the neurological
40 and behavioral distinctions between children with autism and typically developing
41 children reveals stark differences in imitative behavior as well as brain structure
42 (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006; Williams, Waiter, Gilchrist,
43 Perrett, Murray, & Whiten, 2006; Vivanti, Nadig, Ozonoff, & Rogers, 2008). Given
44 this, I intend to argue that the deficits in imitation, in conjunction with the motor
45 and sensory components of the mirror neuron system (MNS) in children with
46 autism (CWA), crystallizes the need for a new approach to treat the core deficits of

47 this disorder using motor imitation of a familiar model while simultaneously
48 providing sensory input.

49 Mirror neurons, originally discovered by Gallese, Fadiga, Fogasse, &
50 Rizzolatti (1996), in the ventral premotor cortex of macaque monkeys, fire when
51 observing another monkey engaged in an action as if the observer were performing
52 the action themselves. Since then, human mirror neuron equivalents have been
53 found in several areas of the brain, including the posterior part of the frontal lobe
54 and the anterior part of the inferior parietal lobule (IPL) (Iacoboni & Dapretto,
55 2006). Together these areas create a network that integrates motor movement with
56 visual and somatosensory input (Iacoboni & Dapretto, 2006). More accurately, the
57 superior temporal sulcus (STS) relays visual input to the IPL, but does not contain
58 neurons that function as mirror neurons (Iacoboni & Dapretto, 2006). While
59 patterns of motor neurons are indistinguishable whether a person is watching or
60 performing an action, sensory mirror neurons, in the IPL, only fire if exposed to a
61 goal-directed action, further implicating imitation as a learning device involving the
62 MNS (Rizzolatti, Fabbri-Destro, & Cattaneo, 2009; Iacoboni & Dapretto, 2006). The
63 involvement of the IPL adds the function of comprehension of the goal of an
64 observed action, implicating the importance of salience in the imitative act
65 (Rizzolatti, Fabbri-Destro, & Cattaneo, 2009).

66 Moreover, although not considered part of the MNS at this time, many
67 scientists are exploring similar brain mechanisms that facilitate emotional
68 mirroring between observed and observer (Gallese, Keysers, & Rizzolatti, 2004).
69 This is the construct that binds us to each other as well as to our larger social group

70 (Singer, 2006). Research is expanding the known boundaries of what is directly
71 mapped to our own brains when observing the experiences of another to include
72 sensory stimuli as well as emotion (Gallese, Keysers, & Rizzolatti, 2004).

73 Interestingly, neurons in the insula and cingulate cortex are
74 cytoarchitecturally similar to mirror neurons in the motor cortex and have
75 indeed been implicated in mirroring the emotions of at least pain and disgust
76 (Rizzolatti, Fabbri-Destro, & Cattaneo, 2009; Singer, 2006). The mirroring of
77 emotional and sensory states in typically developing children is subsequent to the
78 ability to mentalize intention as described earlier because of the later maturation of
79 the limbic and sensory systems (Singer, 2006). Whether defined as part of the MNS
80 or not, dysfunction in these areas impedes the motivation to imitate, thus
81 compromising learning. Further research may provide important information
82 regarding the reasons that children naturally imitate.

83 Other functions of the MNS that have been posited include: (a) facilitation of
84 empathy with another when past experience allows for understanding of motor
85 movement, (b) determination of how the actions of another person relate to the self
86 and modulation of attentional systems toward another persons behavior, (c) the
87 fostering of joint-attention, (d) development of a repertoire of motor responses
88 through imitative behavior, and (e) aid in the development of theory of mind (ToM)
89 by drawing on knowledge gained through comparisons of self to others during
90 imitative learning (Williams, et al., 2006).

91 Anatomical differences between neurotypical children and children with
92 autism highlight common sense assumptions about imitative behaviors seen in

93 CWA. More specifically, studies have shown that CWA have thinner cortical matter
94 in the MNS and related areas, such as the IPL and STS when compared to typically
95 developing children (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006).
96 Furthermore, a correlation has been established between thinner cortical material
97 and symptom severity in CWA (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006).
98 Indeed, if the foundations of social functioning, including language, ToM, and
99 empathy are facilitated by the MNS through imitation, symptoms of autism could in
100 part be viewed as being a result of a dysfunctional MNS (Hadjikhani, Joseph, Snyder,
101 & Tager-Flusberg, 2006; Williams, 2008). While this finding is important, it should
102 be noted that the technology used is incapable of measuring sub-cortical brain
103 structures. Therefore, it is imprudent to assume that cortical thinning is solely
104 responsible for symptom severity in CWA.

105 Other findings include diminished activity in the right parietal lobe in CWA
106 when compared to controls during an imitation task (Williams, et al., 2006). The
107 most significant difference in the Williams, et al., (2006) study was under activity in
108 the parietal-temporal junction, an area associated with ToM, another skill believed
109 to be built on the foundation of imitation.

110 In putting this information together one might be quick to develop an
111 assumption that CWA do not imitate at all; such is not the case. Generally speaking,
112 CWA do not perform as well as neurotypical children on imitation tasks (Ingersoll &
113 Schreibman, 2006). However, upon closer examination, there are certain types of
114 imitation with which CWA particularly struggle. One way to begin to tease this apart
115 is to find out what CWA are attending to during observation and imitation tasks.

116 During tasks with a specific goal, children with high-functioning autism attend to the
117 same visual areas as typical children. This pattern consists of a minimal amount of
118 time looking at the face of a model and more time spent examining the action of the
119 model (Vivanti, Nadig, Ozonoff, & Rogers, 2008). However, when the goal of the
120 action is ambiguous, typical children spend more time looking at the face of the
121 observed, presumably to infer the intention of the action. The result is that typical
122 children significantly outperform their autistic counterparts in accuracy of imitating
123 the model during ambiguous goal directed tasks (Vivanti, Nadig, Ozonoff, & Rogers,
124 2008).

125 When imitation tasks are divided into goal directed (transitive) and non-goal
126 directed (intransitive), CWA show increased impairment with intransitive imitation
127 tasks when compared to matched controls, while imitation of transitive tasks is
128 impacted, but to a lesser extent (Gowen, Stanley, & Miall, 2008). This has led to the
129 hypothesis that transitive and intransitive actions are processed by different
130 neuronal pathways (Gowen, Stanley, & Miall, 2008). There is some discrepancy in
131 the field regarding the neurological substrates responsible for differentiating and
132 processing transitive and intransitive imitative tasks, but there is consensus that
133 they should be differentiated. (For detailed discussion see Rumiati, Weiss, Tessari,
134 Assmus, Zilles, Herzog, et al., 2005; Tessari, Canessa, Ukmar, & Rumiati, 2007;
135 Molnar-Szakacs, Iacoboni, Koske, & Mazziotta, 2005; & Hamilton, 2008). The more
136 accurate processing of transitive imitation tasks further illuminates the role of
137 salience of the task to the observer during imitation tasks.

138 If the mirror neuron systems of CWA were indeed impaired then interference
139 between neurons that occurs in typically developing children would not be expected
140 to occur in CWA. A theory explaining increased reaction times when observing and
141 producing incompatible motor actions posits an interference effect between groups
142 of mirror neurons (Gowen, Stanley, & Miall, 2008). Previous research suggests that
143 CWA perform goal-directed imitation closer to typical levels, but if there were
144 indeed multiple pathways then interference during intransitive imitation tasks
145 would be expected to produce less interference between mirror neurons. Gowen,
146 Stanley, and Miall (2008) explored this issue and the results contradicted the
147 expectation. CWA performed similarly to typical participants during transitive
148 imitation, but struggled significantly more during intransitive imitation. The authors
149 posit that CWA may rely exclusively on pathways used for transitive processing,
150 thus accounting for the increased delay in intransitive imitation tasks. Another
151 element included in this study was that of interpersonal interaction. Participants
152 interacted with three sets of stimuli including a computerized dot, a dot controlled
153 by a human, and a videotape of a human model that was the same sex as the
154 participant. Both groups performed better when imitating stimuli involving human
155 interaction over the computerized dot stimulus.

156 Functional magnetic imaging has revealed that when CWA are observing
157 intransitive motor activities the visual processing areas of the brain are active;
158 however, the premotor cortex remains inactive (Jackson, Meltzoff, & Decety, 2006).
159 Usually during imitation tasks, other areas of the brain – including the contralateral
160 somatosensory and motor areas as well as the IPL – are active as well (Jackson,

161 Meltzoff, & Decety, 2006). Therefore, it may be that if CWA do not comprehend that
162 the observed activity pertains to them in some way, they may process it as a passive
163 observer, thus missing the opportunity to add skills to their motor and social
164 repertoire. This could also shed light on the dual pathway processing of motor acts
165 hypothesis as having more to do with not recognizing the “like me” qualities of the
166 observed (Meltzoff, 2007, p. 126).

167 At first glance it would seem that using peers of CWA in lieu of adults as
168 models may facilitate attention in CWA by engaging the “like me” qualities between
169 observer and observed (Meltzoff, 2007, p. 126). Studies that examine using peers to
170 model motor actions for CWA yield exciting results. Nikopoulos and Keenan (2004)
171 used peers to model behaviors in an effort to increase initiation and reciprocal play
172 behaviors in CWA. All participants in the study showed significant increases in the
173 target behaviors after observing peers. Also, at one- and three-month follow up
174 measurements, all participants had retained the new skills. While these results are
175 exciting the size of the sample consisted of only three participants.

176 In school settings, the use of peer modeling has been shown to increase pro-
177 social and imitative behavior in CWA (Garfinkle & Schwartz, 2002). Children were
178 broken into small groups that were facilitated by an assistant to the teacher. The
179 entire group was given an instruction to perform a motor act and then a group
180 “leader” was selected to model the new behavior. All children imitated the leader
181 and then were reinforced with social praise for the imitative behavior (Garfinkle &
182 Schwartz, 2002, p. 30). Each child alternated being the leader for the group and then
183 were allotted time to explore imitative objects in their own way. Later, children

184 were exposed to free play time. When compared to baseline levels, CWA showed
185 significant increases in imitative and pro-social behaviors after imitating their peers
186 (Garfinkle & Schwartz, 2002). Similar to other peer modeling studies, the sample
187 size is quite small, including three children diagnosed with autism and one child
188 with documented developmental delays. However, the use of a multiple baseline
189 design bolsters the validity of the study and should be a precursor to further
190 research in this area. It should also be noted that this study is one of the few using *in*
191 *vivo* modeling of a peer as opposed to a videotaped model.

192 Most research in this area incorporates the use of videotaping models,
193 presumably in an effort to hold the independent variables constant, which is proper
194 research protocol. However, the use of video in contrast to live models introduces a
195 confound that may be affecting the results in these studies. I believe that more
196 research is needed using live models in lieu of videotaped models to more
197 accurately assess imitative behavior as it occurs naturally.

198 Concordantly, variation in mu rhythm in the MNS that occurs when
199 observing the actions of another is understood as being correlated to the degree
200 that the observer is relating to the observed (Oberman, Ramachandran, & Pineda,
201 2008). Simply put, the more “like me” the observed is to the observer, the higher the
202 change in MNS brain waves in the observer (Oberman, Ramachandran, & Pineda,
203 2008; Meltzoff, 2007, p. 126). Evidence shows that when CWA are exposed to
204 actions performed by strangers they have lower levels of mu rhythm changes than
205 when observing family members or themselves (Oberman, Ramachandran, &
206 Pineda, 2008). The impaired MNS of CWA is increasingly engaged when mirroring

207 someone who is familiar. This supports the idea that social salience is an important
208 factor when considering treatments for autism that focus on imitation (Oberman,
209 Ramachandran, & Pineda, 2008).

210 This finding further suggests that dysfunction in the MNS and other systems
211 may contribute to a lack of identification to others as being “like me” underscoring
212 the impaired imitation skills seen in CWA (Oberman, Ramachandran, & Pineda,
213 2008; Meltzoff, 2007, p. 126). To overcome the deficits in the MNS of CWA,
214 increased familiarity with the model of the motor movement may provide a useful
215 tool to increase responsiveness and strengthen neural pathways used in imitation
216 (Oberman, Ramachandran, & Pineda, 2008).

217 In contrast, research has shown that CWA demonstrate increased imitative
218 behavior with adults who imitate them than with their non-imitative parents (Field,
219 Nadel, Diego, Hernandez-Reif, Russo, Vchulek, et al., 2008). Additionally, current
220 research reexamining data from a series of experiments in which an unfamiliar
221 adult alternately imitated or sat motionless during interactions with a CWA found
222 that more pro-social behaviors were exhibited towards the adult subsequent to
223 having been imitated by the adult (Nadel, Martin, Field, Escalona, & Lundy, 2008).

224 Given what we know, this line of research may seem contradictory to the
225 finding that familiarity increases MNS activity; however, it may not be. If the
226 increase in mu rhythm change were a response to the salience of the activity rather
227 than the agency of familiarity to the model, we would expect increased reaction
228 when the child relates to either the actor or the activity. Moreover, we would expect
229 to see the highest increases in brain activity and imitative behavior if both the actor

230 and the activity are salient to the CWA. Previous research has established that CWA
231 display increased amounts of social behavior during repeated exposures to imitative
232 and playful adults; also naturalistic imitation interventions increase imitative
233 behaviors (Field, Field, Sanders, & Nadel, 2001; Ingersoll & Schreibman, 2006). I
234 believe that future research should differentiate variance due to familiarity from
235 that due to imitating the CWA. This is an area where further study may yield
236 important insights to treatments for autism.

237 We have established that the MNS is involved in the development of many
238 needed social skills via imitation. However, we have not determined if the MNS is
239 amenable to alteration through behavioral experience. Evidence supports the claim
240 that the properties of the MNS are not solely innate or incapable of change, but
241 rather alter through exposure to sensorimotor activities (Catmur, Walsh, & Heyes,
242 2007.) Thus, interventions targeting imitation of sensorimotor movements stand a
243 chance of increasing functionality of the MNS as well as building the foundation of
244 social interaction in those who are impaired in these areas.

245 Although it is well established that mirror neurons are involved, arguments
246 against a MNS theory of autism elucidate the involvement and integration of many
247 brain systems responsible for different aspects of human behavior. They articulate
248 concern over focusing too narrowly on the MNS and in turn missing other important
249 neural substrates that may be involved (Hamilton, Brindley, & Firth, 2007). This is a
250 valid concern; however, it is possible that MNS dysfunction may be the underlying
251 trigger to the abnormal development of the other brain systems involved in
252 imitative, language, empathic, and ToM abilities (Hadjikhani, Joseph, Snyder, &

253 Tager-Flusberg, 2006). Other scientists have argued that the neurological studies on
254 the MNS thus far fail to correlate the behavioral aspects of imitation, ToM, language
255 acquisition, and empathy (Dinstein, Thomas, Behrmann, & Heeger, 2008). While it is
256 true that no one study incorporated all of these constructs simultaneously, it is also
257 true that these different constructs are being correlated as more information
258 becomes available. This is a newer line of research and it is wise to temper
259 excitement to avoid making assumptions while continuing to explore this area.

260 Hamilton, Brindley, & Firth (2007) argue that the deficits in CWA stem not
261 from a generalized imitation deficit due to impairment in the MNS, but rather a lack
262 of ToM that accounts for autistic symptoms. As described above, ToM is a skill built
263 through acquisition of motor movements via imitation, which involves the MNS
264 (Williams, 2006). The results found by Hamilton, Brindly, and Firth (2007) are not
265 mutually exclusive with MNS research. Rather they add to the base of knowledge
266 being created by the field. As a separate line of research, they do not as readily
267 explain the complex observed phenomena.

268 Interventions currently exist that are designed to teach imitative behaviors
269 to CWA and some of them have proven efficacious to some extent (Stahmer,
270 Ingersoll, & Carter, 2003; Ingersoll & Schreibman, 2006). However, all of them use
271 an adult instructor or therapist to directly interact with the child to teach new
272 behaviors. I posit CWA should be put into small compatible groups in order to model
273 behaviors of peers as opposed to adult therapists. This would capitalize on the
274 knowledge that CWA demonstrate increased MNS activity when exposed to a
275 stimulus that is somehow salient (Oberman, Ramachandran, & Pineda, 2008).

276 Ideally, these groups would function in a typical environment where exposure to the
277 motor activities of typically developing children can be modeled as well. Within
278 these groups, adult facilitators would work toward having the children imitate
279 learned skills from each other. For example, a facilitator would teach one child a
280 skill and have the others imitate the behavior. This can be done with both transitive
281 and intransitive motor movement imitation, providing an opportunity for
282 acquisition and mastery of both.

283 Using imitation of sensorimotor movement taps into the MNS, effectively
284 strengthening neural pathways and bolstering the foundation of the MNS (Catmur,
285 Walsh, & Heyes, 2007). Given the evidence that pathways in the MNS can change
286 based on environmental input, exposure to an environment that will provide intense
287 sensorimotor input, such as a gymnastics facility or a playground, may be shown to
288 increase imitative behavior owing to the intensity and full body imitation. Research
289 in this area may lead to important insights regarding innovative interventions for
290 autism.

291 It is important to consider that if a task to be imitated consists of neither an
292 understandable goal nor a model that can be related too in some way, CWA flounder
293 and fundamental social learning opportunities are compromised. Providing an
294 opportunity for CWA to learn appropriate behaviors from peers via imitation in a
295 setting conducive to full-body motor movement and intense sensory input may be
296 shown to ameliorate MNS dysfunction and promote fundamental social skills.

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299 References

- 300 Catmur, C., Walsh, V., & Heyes, C. (2007). Sensorimotor learning configures the
301 human mirror system. *Current Biology, 17*, 1527-1531.
- 302 Dinstein, I., Thomas, C., Behrmann, M., & Heeger, D. J. (2008). A mirror up to nature.
303 *Current Biology, 18*, 13-18.
- 304 Field, T., Nadel, J., Diego, M., Hernandez-Reif, M., Russo, K., Vchulek, D., Lendi, K., &
305 Siddalingappa, V. (2008). Children with autism are more imitative with an
306 imitative adult than with their parents. *Early Child Development and Care*,
307 99999, 1.
- 308 Field, T., Field, T., Sanders, C., & Nadel, J. (2001). Children with autism display more
309 social behaviors after repeated imitation sessions. *Autism, 5*, 317-323.
- 310 Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social
311 cognition. *Trends in Cognitive Sciences, 8*, 396-403.
- 312 Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the
313 premotor cortex. *Brain, 119*, 593-609.
- 314 Garfinkle, A. N., & Schwartz, I. S. (2002). Peer imitation: Increasing social interactions
315 in children with autism and other developmental disabilities in inclusive
316 preschool classrooms. *Topics in Early Childhood Special Education, 22*, 26-38.
- 317 Gowen, E., Stanley, J., & Miall, R. C. (2008). Movement interference in autism-
318 spectrum disorder. *Neuropsychologia, 46*, 1060-1068.
- 319 Hamilton, A. F. (2008). Emulation and mimicry for social interaction: A theoretical
320 approach to imitation in autism. *Quarterly Journal of Experimental*
321 *Psychology, 61(1)*, 101-115.

- 322 Hamilton, A. F., Brindley, R. M., & Firth, U. (2007). Imitation and action
323 understanding in autistic spectrum disorders: How valid is the hypothesis of
324 a deficit in the mirror neuron system? *Neuropsychologia*, *45*, 1859-1868.
- 325 Hadjikhani, N., Joseph, R. M., Snyder, J., & Tager-Flusberg, H. (2006). Anatomical
326 differences in the mirror neuron system and social cognition network in
327 autism. *Cerebral Cortex*, *16*, 1276-1282.
- 328 Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the
329 consequences of its dysfunction. *Nature Reviews Neuroscience*, *7*, 942-951.
- 330 Ingersoll, B., & Schreibman, L. (2006). Teaching reciprocal imitation skills to young
331 children with autism using a naturalistic behavioral approach: Effects on
332 language pretend play, and joint attention. *Journal of Autism & Developmental*
333 *Disorders*, *36*, 487-505.
- 334 Jackson, P. L., Meltzoff, A. N., & Decety, J. (2006). Neural circuits involved in imitation
335 and perspective-taking. *Neuroimage*, *31*, 429-439.
- 336 Meltzoff, A. N. (2007). "Like me": A foundation for social cognition. *Developmental*
337 *Science*, *10(1)*, 126-134.
- 338 Molnar-Szakacs, I., Iacoboni, M., Koski, L., & Mazziotta, J. C. (2005). Functional
339 segregation within pars opercularis of the inferior frontal gyrus: Evidence
340 from fMRI studies of imitation and action observation. *Cerebral Cortex*, *15*,
341 986-994.
- 342 Nadel, J., Martin, M., Field, T., Escalona, A., & Lundy, B. (2008). Children with autism
343 approach more imitative and playful adults. *Early Child Development and*
344 *Care*, *178*, 461-465.

- 345 Nikopoulos, C. K., & Keenan, M. (2004). Effects of video modeling on social
346 initiations by children with autism. *Journal of Applied Behavior Analysis, 37*,
347 93-96.
- 348 Oberman, L. M., Ramachandran, V. S., & Pineda, J. A. (2008). Modulation of mu
349 suppression in children with autism spectrum disorders in response to
350 familiar or unfamiliar stimuli: The mirror neuron hypothesis.
351 *Neuropsychologia, 46*, 1558-1565.
- 352 Rizzolatti, G, Fabbri-Destro, M., & Cattaneo, L. (2009). Mirror neurons and their
353 clinical relevance. *Nature Clinical Practice Neurology, 5*, 24-34.
- 354 Rizzolatti, G., & Craighero, L. (2004). The mirror-neuron system. *Annual Reviews*
355 *Neuroscience, 27*, 169-192.
- 356 Rumiati, R. I., Weiss, P. H., Tessari, A., Assmus, A., Zilles, K., Herzog, H., et al. (2005).
357 Common and differential neural mechanisms supporting imitation of
358 meaningful and meaningless actions. *Journal of Cognitive Neuroscience, 17*,
359 1420-1431.
- 360 Singer, T. (2006). The neuronal basis of ontogeny of empathy and mind reading:
361 Review of literature and implications of future research. *Neuroscience and*
362 *Biobehavioral Reviews, 30*, 855-863.
- 363 Stahmer, A. C., Ingersoll, B., & Carter, C. (2003). Behavioral approaches to
364 promoting play. *Autism, 7(4)*, 401-413.
- 365 Tessari, A., Canessa, N., Ukmar, M., & Rumiati, R. I., (2007). Neuropsychological
366 evidence for a strategic control of multiple routes in imitation. *Brain, 130*,
367 1111-1126.

- 368 Vivanti, G., Nadig, A., Ozonoff, S., & Rogers, S. J. (2008). What do children with autism
369 attend to during imitation tasks? *Journal of Experimental Child Psychology*,
370 *101*, 186-205.
- 371 Williams, J. H. G. (2008). Self-other relations in social development and autism:
372 Multiple roles for mirror neurons and other brain bases. *Autism Research*, *1*,
373 73-90.
- 374 Williams, J. H. G., Waiter, G. D., Gilchrist, A., Perrett, D. I., Murray, A. D., & Whiten, A.
375 (2006). Neural mechanisms of imitation and 'mirror neuron' functioning in
376 autistic spectrum disorder. *Neuropsychologia*, *44*, 610-621.