

Body Odors Promote Automatic Imitation in Autism

Valentina Parma, Maria Bulgheroni, Roberto Tirindelli, and Umberto Castiello

Background: Autism spectrum disorders comprise a range of neurodevelopmental pathologies characterized, among other symptoms, by impaired social interactions. Individuals with this diagnosis are reported to often identify people by repetitively sniffing pieces of clothing or the body odor of family members. Since body odors are known to initiate and mediate many different social behaviors, smelling the body odor of a family member might constitute a sensory-based action promoting social contact. In light of this, we hypothesized that the body odor of a family member would facilitate the appearance of automatic imitation, an essential social skill known to be impaired in autism.

Methods: We recruited 20 autistic and 20 typically developing children. Body odors were collected from the children's mothers' axillae. A child observed a model (their mother or a stranger mother) execute (or not) a reach-to-grasp action toward an object. Subsequently, she performed the same action. The object was imbued with the child's mother's odor, a stranger mother's odor, or no odor. The actions were videotaped, and movement time was calculated post hoc via a digitalization technique.

Results: Automatic imitation effects—expressed in terms of total movement time reduction—appear in autistic children only when exposed to objects paired with their own mother's odor.

Conclusions: The maternal odor, which conveys a social message otherwise neglected, helps autistic children to covertly imitate the actions of others. Our results represent a starting point holding theoretical and practical relevance for the development of new strategies to enhance communication and social behavior among autistic individuals.

Key Words: Autism, automatic imitation, body odor, reach-to-grasp, social behavior, visuomotor priming

Most recent reviews estimate the prevalence of autism spectrum disorders close to 6 per 1000 individuals (1). These neurodevelopmental pathologies affect social behavior and result in a severely invalidating condition (2). Therefore, exploring new approaches to overcome social deficits in autism is a timely and appropriate issue. Given the uncommunicative nature of this population, strategies promoting adaptive social contacts should preferably consider nonverbal stimuli. In this respect, it is well established that odors play an important role in regulating social interactions (3). They are fundamental cues for mother-offspring bonding in animals, as well as in human beings (4). Studies indicate that human newborns and infants are able to discriminate their own mother's odor, show preference for it, and are more easily comforted when they are exposed to it (4–10). Moreover, body odors seem to function as special stimuli for autistic children, who have been reported to be spontaneously attracted by familiar body odors (11). In the light of their limited social experience, this behavior might be viewed as a nonconventional attempt to shape social interactions. Therefore, body odors might be relevant, nonverbal stimuli pulling the strings in prosociality.

Among the several responses possibly grouped under the common label of social behavior, we focused our attention on imitation abilities. This skill merits priority for many different reasons. First, theoreticians have proposed that imitative deficits constitute a precursor of other kinds of social impairments in

autism, including scant emotion sharing, joint attention, pretend play, and theory of mind (12–15). In this respect, evidence suggests that, in autistic children, the lack of prompt imitation of the actions of others is not only a consistent observation (15) but also an effect relevant in magnitude (13). Second, researchers have demonstrated that motor imitation deficits are the best predictors for the acquisition of social behaviors, such as language skills (16,17), and are supposed to prevent the development of joint attention in the autistic population (18). As a consequence, clinicians are making increasingly stronger claims that imitation skills should be targeted early and heavily in intervention programs (18–22). Third, a focus on imitation skills allows researchers to extend the conclusions inferred from a sample of Western autistic children to children of different cultures. As importantly stated by DeWeerd (23), whereas non-Western countries present a different model of social interaction, which requires a diverse use of joint attention (e.g., eye contact behavior with adults is discouraged), they show more similar early-developing signs, such as imitation impairment.

Therefore, to test the link between olfactory cues and social behavior, we assessed how a specific type of imitation (i.e., automatic imitation), an essential part of communicative social abilities, was modulated by exposure to different body odors. Automatic imitation, as elegantly described by Heyes (24), is a type of stimulus-response compatibility effect. In motor terms, this effect explains how human motor performance is not only affected by features of the object relevant for the action but also by features that are apparently task-irrelevant. The time it takes to successfully grasp an object is dependent, for example, on accurate localization of that object with respect to the agent's position and on appropriate estimation of the object's size. In contrast, observing a model performing a reach-to-grasp action toward that object is task-irrelevant—or unessential—to the agent's execution of a successful grasp. However, while it may not be essential for a successful grasp, observation of a model performing the action before the agent executes a similar movement has a facilitation effect, which is evident in terms of a reduction in the time needed to accomplish the reach-to-grasp movement (25). Conversely, observing the model performing an

From the Department of General Psychology (VP, UC), University of Padova, Padova; Ab.Acus S.r.l. (MB), Milano; and Department of Neurosciences (RT), University of Parma, Parma, Italy.

Address correspondence to Umberto Castiello, Ph.D., Università di Padova, Dipartimento di Psicologia Generale, Via Venezia, 8, Padova 35131, Italy; E-mail: umberto.castiello@unipd.it.

Received Aug 17, 2012; revised and accepted Jan 8, 2013.

action in dissimilar conditions (e.g., object dislocated from the original position or object of a different size) produces an interference effect, resulting in a prolonged movement time of the agent's action (25).

Effects of this kind have been tested with different methodologies (24), such as the visuomotor priming (26–30) or motor facilitation by gaze observation paradigms (31,32). In detail, the visuomotor priming paradigms allow for the measurement of how an agent's motor response is facilitated by observation of a movement carried out by someone else. Likewise, the motor facilitation by gaze observation paradigms allow for examination of how the motor response of an agent is facilitated by observation of a model solely gazing at the to-be-grasped object. In both cases, kinematic analysis allows the assessment of facilitation effects defined—as an example—as the reduction in the amount of time needed by the agent to execute an action after viewing a model performing that same action. Such paradigms have been successfully used to assess automatic imitation skills in autistic children (30–32).

Here, we used a visuomotor priming paradigm and we added an olfactory component that consisted of body odors sampled from the axillary area, an essential source of these olfactory stimuli (33). A group of autistic children diagnosed with high-functioning autism, as well as a group of age- and gender-matched typically developing children (control group) were tested to evaluate whether a familiar (i.e., participant's own mother), a stranger (i.e., another participant's mother), or a nonexistent body odor influenced the ability to automatically imitate movements following the observation of an action (present or absent) performed by either a familiar (i.e., participant's own mother) or a stranger (i.e., another participant's mother) model. If the hypothesis of olfactory-driven automatic imitation in autism is correct, then we should be able to reveal visuomotor priming effects in the autistic group when participants are asked to act upon a stimulus imbued by their own mother's body odor, independent of whether their own mother or a stranger mother acted as a model.

Methods and Materials

Participants

Twenty children diagnosed with autism and 20 control subjects took part in the experiment. The autistic children were matched to the control group on age, gender, Wechsler Intelligence Scale for Children-Third Edition full IQ score (34), socioeconomic status (35), and handedness (36) (Table 1). All of the participants were recruited from the greater Padova area. Diagnosis of autism was based on DSM-IV Text Revision criteria (2); the Autism Diagnostic Observation Schedule (37), an instrument involving social interaction between the examiner and the

Table 1. Characteristics of the Autistic and Control Groups

	ASD Mean (SD)	Control Mean (SD)	F or χ^2	p
n	20	20	–	–
Age	13.2 (1.82)	13.4 (1.76)	.05	.58
Full Scale IQ	103.5 (10.38)	109 (8.52)	1.13	.22
Socioeconomic Status	51.23 (6.18)	52.18 (6.55)	.22	.35
Handedness (R:L)	20:0	20:0	.26	.31
Gender (M:F)	10:10	10:10	.22	.37

ASD, autistic spectrum disorder; F, female; IQ, intelligence quotient; L, left; M, male; R, right; SD, standard deviation.

subject; and the Autism Diagnostic Interview-Revised (38), a companion instrument involving an interview with the parents of the referred individual. Only the children who met the diagnostic criteria on all of these standardized measures and who were considered high functioning by a specialist were included in the experimental sample. None of the children were found to have any neurological or genetic disorders causing autism or smell dysfunctions. A standardized 40-item, four-alternative, cued olfactory identification test (University of Pennsylvania Smell Identification Test [UPSIT]) (39) revealed that, on average, the autistic children's performance could be considered microsmic (UPSIT mean [SD] scores: 23.55 [5.75]). The test requires a participant to scratch and sniff the 40 odorant patches constituting the test sequentially and to select from the four verbal descriptors provided for each patch the one descriptor that best matches that odor. The sum of the correct identifications define the UPSIT score (39). For the age range of interest, scores lower than 30 have been used as an indication of microsmia, namely a decrement in the ability to smell (39). The control children had no history of autism and scored normally on the Autism Diagnostic Observation Schedule and Autism Diagnostic Interview-Revised. Their mean UPSIT score fell in the normal range (mean [SD] scores: 33.67 [4.31]) and was significantly higher than the score obtained by the autistic group ($F_{1,39} = 24.62, p < .0001$). No concerns about autism were acknowledged within the control children's first- or second-degree relatives. All the children were right-handed, reported normal or corrected-to-normal vision, and had no hearing impairments. None used prescription drugs, and none of the children or their mothers had any motor impairment to the upper limbs, which could have interfered with the execution of reach-to-grasp movements. Participants' imitation abilities were assessed by an expert clinical psychologist observing structured situations.

Stimuli

The body odors were obtained from the mothers of both groups and classified as familiar when collected from the participant's own mother and as stranger when collected from the mother of another child. Starting from a month before the experiment commenced, all the mothers were instructed to bathe themselves and to launder their clothes with the provided scent-free body and laundry detergents (40,41). The same procedure was followed by the experimenter allowed to enter the experimental room. To avoid socioexperimental context effects (42), the same female experimenter tested all the participants. The day before the experimental session, the mothers were asked to wear the provided cotton pads under their armpits so as to permeate them with their body odor and to shield them from external odor sources (43). During the body odor collection time, the mothers were instructed to avoid as much as possible activities that could generate moderate to high anxiety or excessive sweating (e.g., a doctor's appointment, a gym session, an exam, etc.) and were debriefed at the end of the experiment (44–46). The mothers were instructed on how to remove and store the pads to prevent the odor from decomposing (40,41). The day of the experiment, each pad was defrosted and cut into four sections. Each section was treated with 200 μ L of 70% isopropyl alcohol and then refrozen immediately at -80°C in a glass vial (43). The mothers were asked to bring freshly laundered clothing stored in sealed plastic bags and to change into them in the experimental room, just before the session commenced. The experimenter allowed to enter the testing room followed the same procedure. All individuals entering the experimental room were forbidden to

wear any exogenous odor (e.g., perfume, cologne, deodorant). This minimized the effect of odors coming from the external environment.

Procedure

The paradigm required that one child at a time was seated at a table facing either a familiar (i.e., her own mother) or a stranger model (i.e., the mother of another child). A glass covered by a pad was located on the table and constituted the object to act upon. During a third of the trials, the pad was imbued with the familiar body odor; in another third of the trials, it was imbued with the odor of a stranger mother; and in the last third of the trials, the pad was odorless. Before each trial started, both the child and the model smelled the glass presented by the experimenter who, subsequently, positioned the glass on the table. The experiment was run under a double-blind procedure: neither the children and the models nor the experimenter presenting the glass knew the object code of the odor administered during trials. The objects were coded by a second experimenter who did not interact with either the children or the models. An informal test at the end of the experimental session revealed that among the three odors administered during the task, only 5 participants (3 autistic vs. 2 control children) out of 40 could correctly discriminate their own mother's odor. No significant difference was found in their motor performances when compared with the children who did not recognize their mother's odor ($p > .05$); therefore, their data were included in the final sample. During half the trials, at the sound of a signal, the model was instructed to reach for and grasp the glass, to pick it up, to put it down, and then to bring her hand back to the starting position. A few seconds later, the same signal sounded again, inviting the participant to carry out the same action (Figure 1). The experimenter made sure that both the child and the model did not contaminate the pad while grasping the glass. The trials in which some sort of contamination was suspected (e.g., partial touching of the pad) resulted in the substitution of the object with a second, identical glass carrying another portion of the same pad. Data from those trials (autistic group: 7%; control group: 2%) were not included within the final analyses. During the other half of the trials, the model was instructed to remain immobile when the signal was presented. And, again, a few seconds later, another signal was delivered (Figure 1 and Table 2). There were 12 experimental conditions, and each child took part in a total of 120 trials (10 for each experimental condition; Table 2) divided into four blocks of 30 and presented in randomized order. The model-child-object interactions were videotaped and subsequently analyzed using digitalization techniques to extract the children's movements toward the object and calculate the movement time of the action, as described below. The experimental session lasted from a minimum of 60 minutes to a maximum of 90 minutes, depending on the participant's compliance to the task and the setting. The experimenter was present throughout the session and ensured maintenance of attention (i.e., each child had to position her head toward the model and the working surface in front of them for a trial to initiate). A verbal instruction was used when a child of either group did not spontaneously position herself in the requested manner. This type of intervention was required in a limited number of the trials (less than 10%) for both the autistic and the typically developing children ($p > .05$). Apart from these rare circumstances, all participants were cooperative, attentive, and able to follow the given directions. The experimental procedures were in accordance with the Declaration of Helsinki

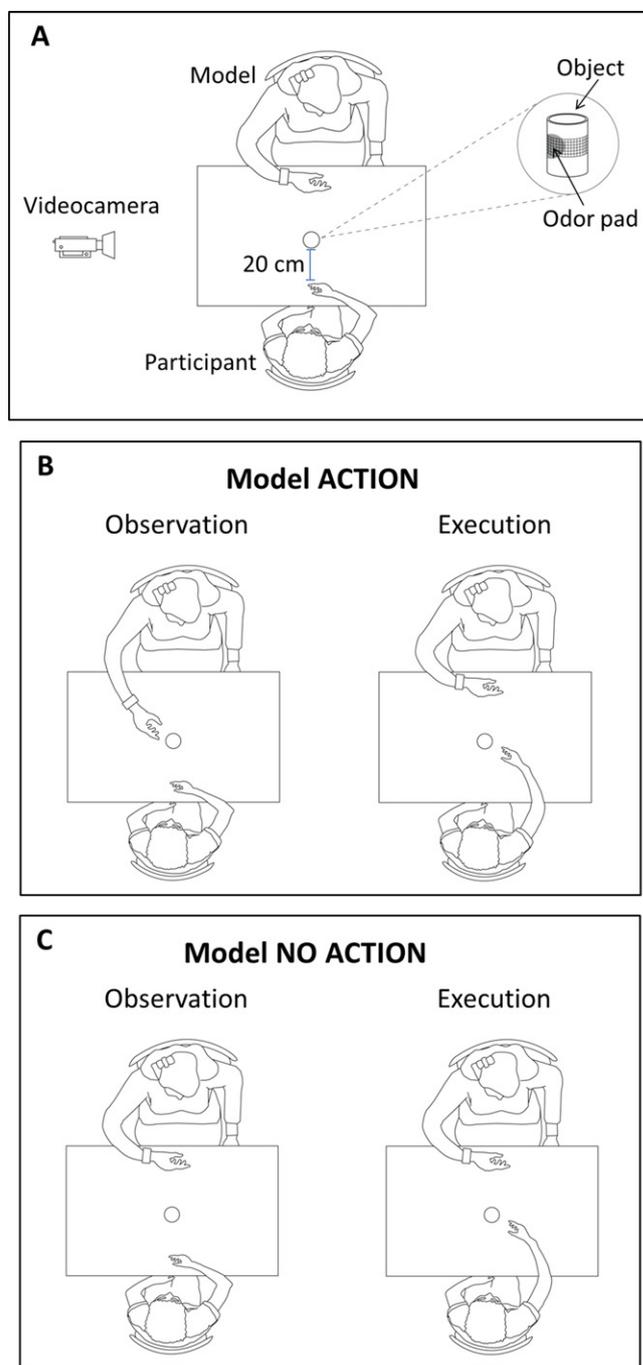


Figure 1. Experimental setup. (A) A child and a model were seated at a table facing one another. The object (e.g., a glass covered with a pad imbued [or not] with a body odor) was located in the middle of the table equidistant from both hand-starting positions. (B) At the sound of a signal, the model reached for and grasped the object. Then, the same signal sounded again, this time inviting the child to execute the same action. (C) At the sound of a signal, the model remained immobile. Then, the same signal sounded again, this time inviting the child to perform a reach-to-grasp action.

and were approved by the local Institutional Review Board. Written informed consent was obtained from all of the mothers, who were informed that they and their child could withdraw from the study at any time.

Table 2. Factor Combinations for Each of the 12 Experimental Conditions to Which the Participants Were Exposed

Odor	Model	Observed Behavior
O	M	A
O	M	nA
O	m	A
O	m	nA
o	M	A
o	M	nA
o	m	A
o	m	nA
nO	M	A
nO	M	nA
nO	m	A
nO	m	nA

A, action performed by the model; m, another participant’s mother; M, participant’s mother; nA, no action performed by the model; nO, no odor; o, stranger mother’s odor; O, participant mother’s odor.

Dependent Measures and Statistical Analyses

Movement time—a particularly sensitive measure to visuo-motor priming effects (25,30)—was calculated as the time between the beginning of the reaching phase (the first forward displacement of the moving wrist toward the object) and its conclusion upon grasping the object (the index finger and the thumb remained stationary around the object for at least two consecutive frames, 80 msec). Exploratory data analysis was performed before inferential statistics were analyzed. Only the trials in which a successful automatic imitation occurred were considered in the final 3 × 2 × 2 × 2 mixed analysis of variance (autistic group: 70%; control group: 100%). The number of trials excluded from the analysis was evenly distributed among conditions (Table 3). Odor (familiar, stranger, no odor), model (familiar/participant’s own mother vs. stranger/another participant’s mother), and observed behavior (action vs. no action) were included as within-subject factors, and group (autistic vs. control children) represented the between-subject factor. Mauchly’s test was applied to assess sphericity, which was not confirmed ($p > .05$). The violation of sphericity was addressed using the Greenhouse-Geisser correction. Effect sizes were calculated and reported as partial eta squared (η_p^2). Bonferroni’s corrections were applied when required (alpha level: $p < .05$).

Results

All main effects, two-way interactions (except for the model by group interaction, $p > .05$), and three-way interactions were significant ($ps < .05$). Please refer to Table 4 for the statistical tests of each effect. Given that the fourth-order interaction is

Table 4. F , p , and η_p^2 Values for Main Effects and Two-Way, Three-Way, and Four-Way Interactions Included in the Model

Effect	F	p	η_p^2
Model	$F_{1,38} = 23.21$	$<.0001$.38
Odor	$F_{2,76} = 36.90$	$<.0001$.49
Observed Behavior	$F_{1,38} = 679.54$	$<.0001$.95
Model × Odor	$F_{2,76} = 4.482$	$<.05$.11
Model × Observed Behavior	$F_{1,38} = 21.25$	$<.0001$.36
Model × Group	$F_{1,38} = 1.40$	ns	.04
Odor × Observed Behavior	$F_{2,76} = 14.87$	$<.0001$.28
Odor × Group	$F_{2,76} = 38.52$	$<.0001$.50
Observed Behavior × Group	$F_{1,38} = 245.64$	$<.0001$.87
Model × Odor × Group	$F_{2,76} = 3.71$	$<.05$.09
Model × Odor × Observed Behavior	$F_{2,76} = 4.39$	$<.05$.10
Model × Observed Behavior × Group	$F_{1,38} = 4.29$	$<.05$.10
Odor × Observed Behavior × Group	$F_{2,76} = 69.12$	$<.0001$.65
Model × Odor × Observed Behavior × Group	$F_{2,76} = 4.87$	$<.05$.11

significant, we are able to explore the various simple effects or interaction contrasts by using post hoc comparisons (47). Therefore, for the sake of clarity, here we extensively report the significant four-way interaction odor by model by observed behavior by group ($F_{2,76} = 4.87$, $p < .05$, $\eta_p^2 = .11$). When the autistic children interacted with either the familiar or the stranger model in the context of being exposed to their own mother’s body odor, they showed automatic imitation effects. This means that the movement time of their reach-to-grasp action toward the object was significantly reduced following the observation of any model performing that action. Post hoc contrasts revealed that control children were faster in performing their movements when they observed that action executed by a model, regardless whether the model was familiar or not ($ps < .01$; Figure 2A,B). Conversely, autistic children were impermeable to automatic imitation effects ($ps > .05$; Figure 2A,B). With reference to the odor variable, the control children did not reveal any automatic imitation effects when exposed to either the familiar or stranger body odor ($ps > .05$; Figure 2C–F). Instead, and this is the salient finding, exposure to the familiar body odor modified the behavior of the autistic children, who then showed an automatic imitation effect comparable with the one that emerged for the control group ($ps < .05$; Figure 2E,F). The automatic imitation effect was not confined to the familiar model; it was also extended to the stranger model (Figure 2E,F). In addition, it is worth noting that there was a tendency for the control group to move slower than the autistic group in all no-action-observation conditions. This trend reaches the significance threshold only when the children smelled no odor and were interacting with the unfamiliar model ($p < .05$; Figure 2B).

Table 3. Number (n) and Percentage (%) of Trials Excluded from the Final Analyses per Experimental Condition in the Whole Group of Autistic Children

		Familiar Odor		Unfamiliar Odor		No Odor		Total	
		n	%	n	%	n	%	n	%
Familiar Model	Action Observed	55/200	27.50	56/200	28.00	57/200	28.50	172/600	28.67
	No Action Observed	59/200	29.50	58/200	29.00	58/200	29.00	172/600	28.67
Unfamiliar Model	Action Observed	56/200	28.00	57/200	28.50	57/200	28.50	172/600	28.67
	No Action Observed	58/200	29.00	58/200	29.00	59/200	29.50	175/600	29.17
Total		228/800	28.50	229/800	28.63	231/800	28.88	688/2400	28.67

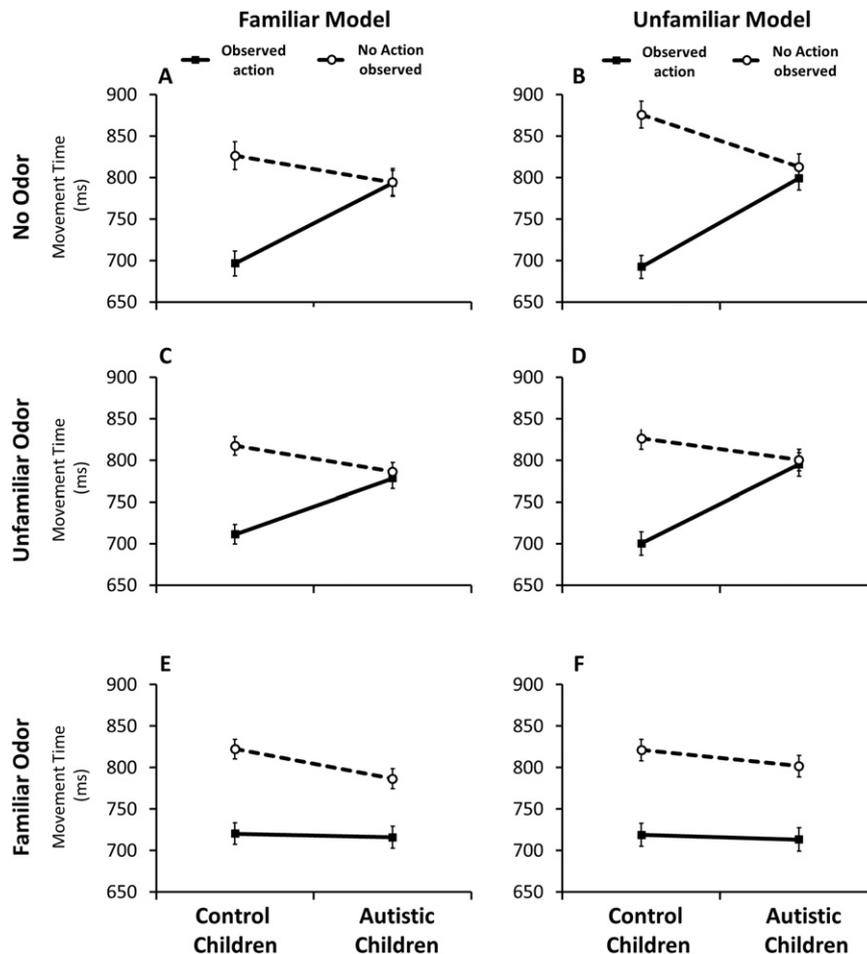


Figure 2. Movement time. Top panels represent the mean movement time of the action executed by control and autistic participants when exposed to no odor and when interacting with either their own (A) or a stranger’s mother (B) following the observation of an action or no action performed by the model. Central panels show the mean movement time of the action performed by control and autistic children when interacting with either the familiar (C) or a stranger (D) model either performing or not performing the action under the exposure of the stranger’s odor. Bottom panels report the mean movement time of the action performed by control and autistic participants interacting with either the familiar (E) or a stranger (F) model following the observation of an action or no action and when the familiar body odor was presented.

Discussion

The findings of the present study take what has been reported in the literature a step forward by demonstrating, for the first time, that a familiar body odor—the participant’s own mother’s odor—might selectively elicit in autistic children the appearance of automatic imitation effects. The exposure to the familiar body odor—but not to a stranger’s body odor or to no odor—elicited a reduction in the movement time needed to perform the reach-to-grasp action toward the object. This represents a dramatic change in the performance of autistic children, whose insensitivity to the facilitation following action observation has been previously well documented (30–32). If the appearance of automatic imitation effects had not been specifically driven by the familiar body odor, then we would have expected a movement time reduction following the exposure to the stranger’s body odor also. But this was not the case. In fact, the results for the stranger’s body odor condition, as well as those for the no odor condition, demonstrated the absence of any automatic imitation effects when considering the autistic group (31,32). However, consistent with previous evidence, children from the control group were

facilitated following the observation of an action performed by a model, either familiar or stranger (31,32), independent of the odor they were exposed to.

Interestingly, the control children tended to perform the action slower than the autistic group when no action was observed. This difference becomes significant only when the children interacted with an unfamiliar model and were exposed to no odor. Although it is difficult to fully explain this finding, we suggest that the control children—who are more easily involved in social interactions—might feel the effects of linking their action performance to the previously observed model’s action. On the contrary, the autistic children who performed their movement in a reliable amount of time across conditions when they did not observe the model performing any action (715–815 msec) seem not to be affected by the social aspect of this dyadic interaction. Therefore, this might be considered another example of the poor social interaction abilities showed by the autistic population (48).

The automatic imitation effects revealed by the present paradigm seem to be compatible with the activation of neural areas involved in the mirror system (24). Mirror neurons respond to both the observation (49) and the execution of actions and

can also be activated by olfactory cues (50). To show some kind of mirror activity, autistic individuals have to rely on familiarity, which allows them to identify in some personal way with the stimuli, the action, or the model (51,52). It would almost seem that the effects described here rely on access to the mirror neuron system via olfactory processing. If this is true, the results of the present study imply that maternal body odor constitutes a way to overcome mirror neuron system activation impairment in autistic children (53,54). Maternal body odor—similar to facial expressions (55)—might rapidly alert the limbic centers (via the nonthalamic direct route to the cortex) eliciting emotional empathy that, unlike rational empathy or theory of mind, is supposed to be preserved in autistic individuals (56). The implication of the direct route would also be consistent with the findings of reduced thalamic volume and functional connectivity in autistic individuals (57,58). Findings that are also compatible with the impaired olfactory identification skills were reported for autistic children when tested by means of standardized measures utilizing common odors and requiring explicit verbal stimulus recognition (59), which is known to be an ability lacking in this population (2). In addition, the direct route model would also be plausible when considering that the familiarity of the child's mother's odor could have increased the pleasantness of the maternal odor (60). In fact, it is known that, at the neural level, the amygdala is involved in the processing of pleasant (but not unpleasant) odors (61).

It is important to remember that body odors are processed via different neural pathways with respect to perceptually similar common odors (3,40,41). They do not activate the primary (entorhinal) or the secondary (orbitofrontal) olfactory cortices but recruit cortical and subcortical areas outside the circuit involved in conscious odor processing (40). The existence of this dedicated neural pathway for body odors may have evolutionary significance since the exposure to such stimuli is associated with heightened attention and increased emotional reactions (45), as well as faster motor responses, as demonstrated by the present study (Figure 2C–F).

In the effort of considering possible alternative explanations that might account for (or at least contribute to) the present results, we considered several factors. First, the lack in odor identification might be confined to common odors and not extend to body odors. In these circumstances, autistic children would be able to clearly recognize their own mother's odor and react to it differently. Although we cannot fully rule out this eventuality, it is unlikely that the automatic imitation effects revealed here are fully mediated by overt processes. Even if an explicit identification of the maternal odor occurs, the voluntary control of the time spent to execute natural reach-to-grasp movements is highly improbable.

Second, uneven odor detection abilities in the two groups might have influenced the noticeability of odors, possibly favoring the detection and the subsequent faster reaction to the child's own mother's odor when contrasted to the exposure to the body odor of another mother or no odor. However, the recent evidence obtained by testing the detection threshold to common odors that suggests autistic children show inferior (62) to normal (59) odor detection skills in comparison with their matched control subjects hints that this might not be the case.

Altogether, these findings suggest that body odors might be considered a form of social communication mediated by fast and nonvoluntary processes, which appear to be preserved and function adaptively in autistic people. We anticipate our study to be a promise for future research aiming at the development of

novel clinical strategies for autistic individuals. Future research will reveal whether automatic imitation effects in autistic children are selectively triggered by odors to which they were exposed early in their development, like their own mother's body odor (63,64), or whether postnatal or genetic familiarity with an odor plays a similar role (e.g., body odors of father and siblings). Moreover, the investigation of the pleasantness and the detectability of the body odors could shed light on the perceptual mechanisms that allow this special olfactory cue to assist imitation skills in autistic children. The inclusion of body odors in experimental paradigms measuring other aspects of social behavior—such as joint attention, eye contact, and social smiling—will define to what extent body odors breach social inability in autism. For the sake of tailored-to-age-appropriate treatment, it would also be of interest to evaluate whether the effect of body odors is stable throughout development or is confined to a critical period.

In conclusion, even though this topic deserves further exploration, the present data demonstrate that body odors are chemosignals apt to favor social interactions in autistic children.

This work was supported by a grant to UC from the Ministero dell'Istruzione, dell'Università e della Ricerca.

We thank the students who helped with data collection and the colleagues who proofread and commented on the previous versions of the manuscript.

The authors report no biomedical financial interests or potential conflicts of interest.

1. Newschaffer CJ, Croen LA, Daniels J, Giarelli E, Grether JK, Levy SE, *et al.* (2007): The epidemiology of autism spectrum disorders. *Annu Rev Public Health* 28:235–258.
2. American Psychiatric Association (2000): *Diagnostic and Statistical Manual Of Mental Disorders (DSM-IV-TR), 4th ed., Text Revision.* Washington, DC: American Psychiatric Press.
3. Pause B (2011): Processing of body odor signals by the human brain. *Chemosens Percept* 5:55–63.
4. Schaal B, Montagner H, Hertling E, Bolzoni D, Moysse A, Quichon R (1980): Olfactory stimulations in mother-child relations. *Reprod Nutr Dev* 20:843–858.
5. Montagner H (1974): Communication non verbale et discrimination olfactive chez le jeune enfant: Approche éthologique. In: Morin E, Piatell-Palmarini M, editors. *L'Unité de l'Homme.* Paris: Seuil, 246–270.
6. Macfarlane A (1975): Olfaction in the development of social preferences in the human neonate. In: Porter RH, O'Connell M, editors. *Ciba Foundation Symposium* 33. Amsterdam: Associated Scientific Publishers, 103–117.
7. Cernoch JM, Porter RH (1985): Recognition of maternal axillary odors by infants. *Child Dev* 56:1593–1598.
8. Schleidt M, Genzel C (1990): The significance of mother's perfume for infants in the first weeks of their life. *Ethol Sociobiol* 11:145–154.
9. Doucet S, Soussignan R, Sagot P, Schaal B (2007): The "smellscape" of mother's breast: Effects of odor masking and selective unmasking on neonatal arousal, oral, and visual responses. *Dev Psychobiol* 49: 129–138.
10. Ferdenzi C, Coureaud G, Camos V, Schaal B (2008): Human awareness and uses of odor cues in everyday life: Results from a questionnaire study in children. *Int J Behav Dev* 32:422–431.
11. Bogdashina O (2003): *Sensory Perceptual Issues in Autism and Asperger Syndrome: Different Sensory Experiences-Different Perceptual Worlds.* London: Jessica Kingsley Publishers.
12. Meltzoff AN, Gopnik A (1994): The role of imitation in understanding persons and developing a theory of mind. In: Baron-Cohen S, Tager-Flusberg H, Cohen D, editors. *Understanding Other Minds; Perspectives from Autism.* Oxford, UK: Oxford University Press, 335–366.
13. Rogers S (1999): An examination of the imitation deficit in autism. In: Nadel J, Butterworth G, editors. *Imitation in Infancy.* Cambridge, UK: Cambridge University Press, 254–279.

14. Rogers SJ, Pennington BF (1991): A theoretical approach to the deficits in infantile autism. *Dev Psychopathol* 3:137–162.
15. Smith IM, Bryson SE (1994): Imitation and action in autism: A critical review. *Psychol Bull* 116:259–273.
16. Stone W, Ousley O, Littleford C (1997): Motor imitation in young children with autism: What's the object? *J Abnorm Child Psychol* 25:475–485.
17. Stone WL, Yoder PJ (2001): Predicting spoken language level in children with autism spectrum disorders. *Autism* 5:341–361.
18. Carpenter M, Nagell K, Tomasello M (1998): Social cognition, joint attention, and communicative competence from 9 to 15 months of age. *Monogr Soc Res Child Dev* 63:1–174.
19. Tryon AS, Keane SP (1986): Promoting imitative play through generalized observational learning in autistic-like children. *J Abnorm Child Psychol* 14:537–549.
20. Klinger LG, Dawson G (1992): Facilitating early social and communicative development in children with autism. In: Warren SF, Reichle JE, editors. *Causes and Effects in Communication and Language Intervention*. Baltimore: Paul H. Brookes Publishing, 157–186.
21. Ingersoll B, Schreibman L (2006): Teaching reciprocal imitation skills to young children with autism using a naturalistic behavioral approach: Effects on language, pretend play, and joint attention. *J Autism Dev Disord* 36:487–505.
22. Ingersoll B (2012): Brief report: Effect of a focused imitation intervention on social functioning in children with autism. *J Autism Dev Disord* 42:1768–1773.
23. DeWeerd S (2012): Culture: Diverse diagnostics. *Nature* 491:518–519.
24. Heyes C (2011): Automatic imitation. *Psychol Bull* 137:463–483.
25. Edwards MG, Humphreys GW, Castiello U (2003): Motor facilitation following action observation: A behavioural study in prehensile action. *Brain Cogn* 53:495–502.
26. Craighero L, Fadiga L, Umiltà CA, Rizzolatti G (1996): Evidence of visuomotor priming effect. *Neuroreport* 7:347–349.
27. Craighero L, Fadiga L, Rizzolatti G, Umiltà CA (1998): Visuomotor priming. *Vis Cogn* 5:109–125.
28. Castiello U, Lusher D, Mari M, Edwards M, Humphreys GW (2002): Observing a human or a robotic hand grasping an object: Differential motor priming effects. In: Prinz W, Hommel B, editors. *Common Mechanisms in Perception and Action: Attention and Performance, Vol. XIX*. Oxford, UK: Oxford University Press, 315–333.
29. Castiello U (2003): Understanding other people's actions: Intention and attention. *J Exp Psychol Hum Percept Perform* 29:416–430.
30. Pierno AC, Mari M, Lusher D, Castiello U (2008): Robotic movement elicits visuomotor priming in children with autism. *Neuropsychologia* 46:448–454.
31. Pierno AC, Mari M, Glover S, Georgiou I, Castiello U (2006): Failure to read motor intentions from gaze in children with autism. *Neuropsychologia* 44:1483–1488.
32. Becchio C, Pierno A, Mari M, Lusher D, Castiello U (2007): Motor contagion from gaze: The case of autism. *Brain* 130:2401–2411.
33. Natsch A, Derrer S, Flachsmann F, Schmid J (2006): A broad diversity of volatile carboxylic acids, released by a bacterial aminoacylase from axilla secretions, as candidate molecules for the determination of human body odor type. *Chem Biodivers* 3:1–20.
34. Wechsler D (1991): *WISC-III: Wechsler Intelligence Scale for Children III Edition: Manual*. San Antonio, TX: Psychological Corporation.
35. Hollingshead AB (1975): Four Factor Index Social Status. Unpublished working paper. New Haven, CT: Yale University, Department of Sociology. Available at: <http://www.yale.edu/sociology/faculty/>. Accessed November 5, 2009.
36. Oldfield RC (1971): The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia* 9:97–113.
37. Lord C, Rutter M, DiLavore P, Risi S (2002): *Autism Diagnostic Observation Schedule: ADOS*. Torrance, CA: Western Psychological Services.
38. Le Couteur A, Lord C, Rutter M (2003): *Autism Diagnostic Interview-Revised (ADI-R)*. Torrance, CA: Western Psychological Services.
39. Doty RL, Shaman P, Kimmelman CP, Dann MS (1984): University of Pennsylvania Smell Identification Test: A rapid quantitative olfactory function test for the clinic. *Laryngoscope* 94:176–178.
40. Lundström JN, Boyle JA, Zatorre RJ, Jones-Gotman M (2008): Functional neuronal processing of body odors differ from that of similar common odors. *Cereb Cortex* 18:1466–1474.
41. Lundström JN, Boyle JA, Zatorre RJ, Jones-Gotman M (2009): The neuronal substrates of human olfactory based kin recognition. *Hum Brain Mapp* 30:2571–2580.
42. Jacob S, Hayreh DJS, McClintock MK (2001): Context-dependent effects of steroid chemosignals on human physiology and mood. *Physiol Behav* 74:15–27.
43. Stern K, McClintock MK (1998): Regulation of ovulation by human pheromones. *Nature* 392:177–178.
44. Chen D, Haviland-Jones J (2000): Human olfactory communication of emotion. *Percept Mot Skills* 91:771–781.
45. Mujica-Parodi LR, Strey HH, Frederick B, Savoy R, Cox D, Botanov Y, *et al.* (2009): Chemosensory cues to conspecific emotional stress activate amygdala in humans. *PLoS One* 4:e6415.
46. Prehn-Kristensen A, Wiesner C, Bergmann TO, Wolff S, Jansen O, Mehdorn HM, *et al.* (2009): Induction of empathy by the smell of anxiety. *PLoS One* 4:e5987.
47. Cohen BH (2007): Chapter 14: Two-way ANOVA. In: Cohen BH, editor. *Explaining Psychological Statistics*. Hoboken, NJ: John Wiley & Sons, 425–484.
48. Loftin RL, Odum SL, Lantz JF (2008): Social interaction and repetitive motor behaviors. *J Autism Dev Disord* 38:1124–1135.
49. Gallese V, Fadiga L, Fogassi L, Rizzolatti G (1996): Action recognition in the premotor cortex. *Brain* 119:593–609.
50. Rossi S, De Capua A, Pasqualetti P, Olivelli M, Fadiga L, Falzarano V, *et al.* (2008): Distinct olfactory cross-modal effects on the human motor system. *PLoS One* 3:e1702.
51. Tubaldi F, Turella L, Pierno AC, Grodd W, Tirindelli R, Castiello U (2008): Smelling odors, understanding actions. *Soc Neurosci* 6:31–47.
52. 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.
53. Williams JH, Whiten A, Suddendorf T, Perrett DI (2001): Imitation, mirror neurons and autism. *Neurosci Biobehav Rev* 25:287–295.
54. Williams JHG, Waite GD, Gilchrist A, Perrett DI, Murray AD, Whiten A (2006): Neural mechanisms of imitation and “mirror neuron” functioning in autistic spectrum disorder. *Neuropsychologia* 44:610–621.
55. Holmes A, Vuilleumier P, Eimer M (2003): The processing of emotional facial expression is gated by spatial attention: Evidence from event-related brain potentials. *Brain Res Cogn Brain Res* 16:174–184.
56. Blair RJR (2008): Fine cuts of empathy and the amygdala: Dissociable deficits in psychopathy and autism. *Q J Exp Psychol* 61:157–170.
57. Tamura R, Kitamura H, Endo T, Hasegawa N, Someya T (2010): Reduced thalamic volume observed across different subgroups of autism spectrum disorders. *Psychiatry Res* 184:186–188.
58. Cheon KA, Kim YS, Oh SH, Park SY, Yoon HW, Herrington J, *et al.* (2011): Involvement of the anterior thalamic radiation in boys with high functioning autism spectrum disorders: A Diffusion Tensor Imaging study. *Brain Res* 1417:77–86.
59. Bennetto L, Kuschner ES, Hyman SL (2007): Olfaction and taste processing in autism. *Biol Psychiatry* 62:1015–1021.
60. Sulmont C, Issanchou S, Köster EP (2002): Selection of odorants for memory tests on the basis of familiarity, perceived complexity, pleasantness, similarity and identification. *Chem Senses* 27:307–317.
61. Djoedjevic J, Boyle JA, Jones-Gotman M (2012): Pleasant or unpleasant: Attentional modulation of odor perception. *Chemosens Percept* 1: 11–21.
62. Dudova I, Vodicka J, Havlovicova M, Sedlacek Z, Urbánek T, Hrdlicka M (2011): Odor detection threshold, but not odor identification, is impaired in children with autism. *Eur Child Adolesc Psychiatry* 20: 333–340.
63. Davis LB, Porter RH (1991): Persistent effects of early odor exposure on human neonates. *Chem Senses* 16:169–174.
64. Ferdenzi C, Schaal B, Roberts SC (2010): Family scents: Developmental changes in the perception of kin body odor? *J Chem Ecol* 36:847–854.