

REPORT

When the social mirror breaks: deficits in automatic, but not voluntary, mimicry of emotional facial expressions in autism

Daniel N. McIntosh,¹ Aimee Reichmann-Decker,¹ Piotr Winkielman² and Julia L. Wilbarger³

1. Department of Psychology, University of Denver, USA

2. Department of Psychology, University of California, San Diego, USA

3. Department of Kinesiology, University of Wisconsin, Madison, USA

Abstract

Humans, infants and adults alike, automatically mimic a variety of behaviors. Such mimicry facilitates social functioning, including establishment of interpersonal rapport and understanding of other minds. This fundamental social process may thus be impaired in disorders such as autism characterized by socio-emotional and communicative deficits. We examined automatic and voluntary mimicry of emotional facial expression among adolescents and adults with autistic spectrum disorders (ASD) and a typical sample matched on age, gender and verbal intelligence. Participants viewed pictures of happy and angry expressions while the activity over their cheek and brow muscle region was monitored with electromyography (EMG). ASD participants did not automatically mimic facial expressions whereas the typically developing participants did. However, both groups showed evidence of successful voluntary mimicry. The data suggest that autism is associated with an impairment of a basic automatic social-emotion process. Results have implications for understanding typical and atypical social cognition.

Introduction

Psychologists and the general public alike are fascinated by the phenomenon of automatic mimicry, or how merely observing another person's behavior can elicit a corresponding behavior in the observer (Hatfield, Cacioppo & Rapon, 1992). Rudimentary mimicry, such as tongue protrusion or mouth opening, can be observed in newborns (Meltzoff & Moore, 1989). Adults match postures, gestures, prosody and syntactic constructions (Chartrand & Bargh, 1999; Niedenthal, Barsalou, Winkielman, Krauth-Gruber & Ric, 2005). Several authors have argued that automatic mimicry facilitates social functioning, including interpersonal rapport, fast learning and understanding of other minds (Decety & Chaminade, 2003; Iacoboni, in press; Lakin & Chartrand, 2003). In development, a mimicry deficit could impair a child's ability to grasp others' emotions, and if such a deficit occurred early, it could impair the child's ability to form self–other correspondences, perhaps contributing to autism (Rogers, 1999). In the current work we examined automatic and voluntary mimicry with two techniques inspired by the social neuroscience approach:

we use a population with the neurodevelopmental disorder of autism and the psychophysiological method of facial electromyography (Cacioppo & Berntson, 1992).

Automatic and voluntary mimicry of facial expressions

One of the most robust cases of mimicry is mirroring of emotional facial expressions. When viewing emotional expressions, adults spontaneously and quickly activate congruent facial muscles (i.e. they smile to a smile and scowl to a scowl). Automatic mirroring occurs even when expressions are presented without instructions to mimic (Dimberg, 1982; McIntosh, in press) or when they are presented subliminally (Dimberg, Thunberg & Elmehed, 2000). Automatic facial mimicry facilitates social interaction, including interpersonal rapport, emotional contagion and emotion recognition (Lundquist & Dimberg, 1995; McIntosh, 1996; McIntosh, in press; McIntosh, Druckman & Zajonc, 1994; Niedenthal *et al.*, 2005).

In contrast to automatic mimicry, voluntarily matching of observed facial expressions is effortful and slow (Dimberg, Thunberg & Grunedel, 2002). Voluntary

Address for correspondence: Daniel N. McIntosh, Department of Psychology, University of Denver, 2155 S. Race Street, Denver, CO 80208, USA; e-mail: dmcintos@du.edu

mimicry is more sensitive to situational demands and cultural influences (Ekman, 1992) and involves different neuronal pathways (Matsumoto & Lee, 1993; Tassinari & Cacioppo, 2000).

Despite much research on mimicry, little is known about its role in adaptive social functioning. In this article, we address this question by comparing automatic and voluntary mimicry of emotional facial expression in typically developing individuals and individuals with autism – a developmental disorder characterized by impairments in social functioning. We predict that autism will be associated with impairment of automatic, but not voluntary, mimicry of emotional expression.

Autism, mimicry and emotion

Autism is a developmental disorder, with a spectrum of clinical severity, characterized by impairments in social and emotional abilities, deficits in communication and language skills, and restricted interests and repetitive behaviors (Kanner, 1943; Rogers & Pennington, 1991). Emotional deficits are so apparent in Autistic Spectrum Disorders (ASD) that they are often the focus of clinical descriptions (Kanner, 1943). Some propose that emotion deficits are primary to ASD, producing other social and cognitive dysfunctions (e.g. Hobson, 1993; Mundy & Sigman, 1989; Rogers & Pennington, 1991). Interestingly, not all areas of emotional functioning are impaired. ASD individuals express a full range of emotion, show attachment behaviors, and comprehend a variety of emotional situations (Braverman, Fein, Lucci & Waterhouse, 1989; Rogers & Pennington, 1991; Sigman, Kasari, Kwon & Yirmiya, 1992). Accordingly, several authors have speculated that autism might especially influence automatic affective processes, such as those involved in creating emotional reciprocity (Hobson, 1993; Kasari, Sigman, Yirmiya & Mundy, 1993; Rogers & Pennington, 1991).

Despite the theorized importance of rapid, automatic emotional mimicry to social functioning of typical and atypical individuals, it is unknown whether such mimicry is affected by autism (Moody & McIntosh, *in press*). Some research shows impairments of imitation in autism (Rogers, Hepburn, Stackhouse & Wehner, 2003; Williams, Whiten, Suddendorf & Perrett, 2001). However, this research primarily examined instructed matching of goal-oriented actions with little emotional component (Rogers, 1999). Automatic mimicry occurs without external prompting, and involves mere replication of a model's actions, without any insight into why those actions are effective (Tomasello, 1996; Want & Harris, 2002). Recent reviews suggest that imitation in autism may involve two different processes. One involves 'an affective mechanism

modulating social exchanges', whereas the second involves 'a more executively constructed, cognitively mediated, intentional imitation system', with autistic individuals relying on the second, but not the first process (Rogers *et al.*, 2003, p. 777). Thus, imitation performance on complex, goal-oriented tasks tells us little about automatic processes that might contribute to rapid sharing of affective states (Moody & McIntosh, *in press*). Accordingly, we investigated a process involved in rapid emotional communication: automatic facial mimicry. In addition, examining facial mimicry minimizes the role of high-level verbal or visual skills, which may contribute to the superior performance of typical participants on more complex imitation tasks.

The present study

We compared automatic and voluntary mimicry of emotional facial expressions in ASD individuals and a matched comparison sample of typical individuals. Two hypotheses were tested.

First, we hypothesized that ASD individuals would be impaired on automatic mimicry of emotional expressions. Our theoretical perspective does not specify whether ASD individuals will show no automatic responses at all, or will respond automatically, but in a way that is non-specific to expression valence (happy vs. angry). However, previous research suggests that ASD individuals do in fact respond to emotional stimuli, but with less sensitivity to valence (Hobson, 1993; Kasari *et al.*, 1993). Accordingly, we expected the ASD group to show non-discriminative automatic responding.

Second, we hypothesized that both ASD individuals and typically developing individuals would be successful in voluntarily matching emotional facial expressions. This prediction was grounded in research that high-functioning ASD individuals can perform a range of emotion-related tasks when instructed to do so (Hobson, 1993).

We assessed mimicry using facial electromyography (EMG), which monitors electrical changes in muscle activity over the cheek and brow region. This measure has multiple advantages in investigating mimicry (Dimberg, 1982). First, EMG's temporal resolution and sensitivity can capture fast and subtle changes during automatic mimicry. Second, as compared to self-report methods, EMG is less dependent on factors such as verbal skills, praxis and motivation (Tassinari & Cacioppo, 2000). This is important for studying an atypical population in which self-report methods may not discriminate between performance deficits caused by differences in actual response and motivation to express that response.

Method

Participants

Participants were three female and 11 male high-functioning adolescents and adults with ASD and 14 typically developing individuals matched on gender, chronological age and verbal ability. ASD participants under age 18 were matched within one year of chronological age, those from ages 18 to 30 were matched within two years, and participants over age 30 were matched to others over 30 (ASD sample, $M = 27$, $SD = 13.8$, range = 13–64; Typical sample, $M = 24$ years, $SD = 8.6$, range = 14–43). Matched participants were within one standard deviation on the standard scores of the Peabody Picture Vocabulary Test (PPVT; ASD $M = 101.1$, $SD = 19.4$; typical $M = 110.1$, $SD = 12.3$, $t(26) = 1.47$, ns). Further details on participants' selection are provided in note 1.¹

Procedure

As in earlier mimicry research, the experiment had two phases (Dimberg, 1982). The first evaluated automatic mimicry, and participants were simply asked to 'Watch the pictures as they appear on the screen.' The second phase evaluated voluntary mimicry, and participants were asked to 'Make an expression just like this one.' (Two typical and two ASD participants did not perform the voluntary task due to equipment problems or fatigue.) The stimuli were eight angry and eight happy facial expressions (Ekman & Friesen, 1975a), sized 20 cm by 25 cm and presented on a 21-inch screen placed about 60 cm away from the participant. All faces of one valence were presented in one randomized block which was

¹ The individuals with ASD had received a diagnosis of either Infantile Autism or Asperger's Syndrome from a licensed clinical psychologist or developmental pediatrician and met DSM-IV (American Psychiatric Association, 1994) criteria for autism or Asperger's Syndrome (deficits in communications skills, social functioning and stereotypical or repetitive behaviors with an onset before 3 years of age). All ASD participants also previously met the criteria for autism or Asperger's Syndrome based on scores on either the Autism Diagnostic Inventory-Revised (ADIR; a semi-standardized parent interview, Lord, Rutter & Le Couteur, 1994) or the Autistic Diagnostic Observation-Generic (ADOS-G; a semi-structured standardized observational assessment Lord, Risi, Lambrecht, Cook, Leventhal, DiLavore, Pickles & Rutter, 2000) within three years of date of participation. The ADI-R and the ADOS-G are considered gold standard assessments for identifying the presence and severity of autism spectrum disorders. Typical participants were recruited from the community and university and screened to ensure no history of ASD or other developmental disorders. Individuals were excluded if they had a history of brain injury, seizures or premature birth (> 4 weeks before due date). In addition, individuals with ASD were excluded if they had a medical condition associated with autism (e.g. fragile X syndrome, tuberous sclerosis).

followed by a block of faces of opposite valence. Block order (happy vs. angry first) was randomized across participants (Dimberg, 1982). In the automatic phase, each trial started with a 50-ms soft orienting tone and the face 500 ms later. The voluntary phase was similar, except that each trial began with visual instructions to make the expression. In both phases, a picture of facial expression then appeared for eight seconds, followed by a 15- or 20-second interstimulus interval with a blank screen.

EMG data processing

Data collection and scoring

EMG processing followed psychophysiological standards (Fridlund & Cacioppo, 1986; Tassinari & Cacioppo, 2000). EMG was measured by pairs of 4-mm electrodes over the regions of zygomaticus major (cheek) and corrugator supercilii (brow) with inter-electrode impedances reduced to below 15 KOhms. Acquisition was controlled by Neuroscan SynAmps amplifier synchronized with E-Prime experimental software. The amplified EMG signals were filtered on-line with a low-pass of 500 Hz and a high-pass of 10 Hz, sampled 2048 times per second, and then integrated and rectified.

Data cleaning

EMG signals were screened for artifacts in two ways. First, a blind coder deleted trials with artifacts such as electrical noise (no participant had more than 5% of trials deleted). Second, a blind coder used session videotapes and deleted trials when participants were not looking at the screen or performed extraneous movements (e.g. yawning). This removed 2% of automatic trials and 1% of voluntary trials in the typical group, and 9% of the automatic trials and 2% of the voluntary trials in the ASD group.

Data reduction

EMG data reduction involved several steps. First, data were logarithmically transformed to reduce the impact of extreme values. Second, data were standardized (i.e. expressed as z -scores) within each participant and within each muscle group (cheek, brow) across both sessions. This allows for meaningful comparison of values between sessions and muscle groups as well as reducing the impact of individual differences in reactivity on the group mean. Third, we established baseline values for each trial by calculating average EMG activity in the time window from 1000 to 500 ms before the presentation of the face. Fourth, we calculated baseline-corrected

activity in each individual 100-ms post-stimulus window from 200 ms to 1500 ms after the presentation of each face. The 500-ms window period after the orienting tone and the 200-ms period after stimulus onset were excluded to avoid confounds from orienting reactions. Finally, to reduce variability of responses across trials, we averaged each participant's EMG activity in 100-ms windows separately for each stimulus type (eight angry, eight happy faces), for each muscle group (cheek, brow) and for each phase (automatic, voluntary).

Analysis

A facial expression can be described by (i) onset latency: how long it takes to appear, (ii) apex duration: how long it remains at maximum and (iii) magnitude: the strength of response (Ekman & Friesen, 1975b). In the current study the reduced EMG data were analyzed using a peak detection algorithm that tests whether EMG activity across windows of interest first rises over a specific threshold, sustains activity for a specific time and then falls by the same threshold (Tassinari & Cacioppo, 2000). The parameters for our detection algorithm were based on previous research. Specifically, automatic mimicry is characterized by (i) a fast rise in activity, with the apex around 400 ms after stimulus onset, (ii) short duration and (iii) a relatively small magnitude (Dimberg, 1982; Dimberg *et al.*, 2000). Accordingly, we analyzed EMG activity in the automatic phase in the window from 200 to 600 ms. To count as a response, the activity in this window had to rise and then fall by at least .1 Z, and be sustained for at least 100 ms. In contrast, voluntary mimicry is characterized by (i) a slow rise in activity, with the apex occurring around 1000 ms, (ii) long duration and (iii) large magnitude, with a maximum value at least five times higher than the value of the automatic activity (cf. Dimberg *et al.*, 2000, 2002). Accordingly, voluntary activity was analyzed in the window from 500 to 1500 ms post-stimulus onset. To count as a response, the activity had to rise and then fall by at least .5 Z, and be sustained for at least 300 ms.²

Our peak detection algorithm was applied to EMG data reduced, as described earlier, to averages over each stimulus type (happy, angry), each muscle group (cheek, brow) and each phase (automatic, voluntary). As a result, in each session (automatic and voluntary) there are four total opportunities per participant to show a

response (2 stimulus expressions \times 2 muscle sites). Of those four opportunities per session, two possible responses are congruent, as when the participant responds with smiles (zygomaticus activation) to happy expressions, and with scowls (corrugator activation) to angry expressions. The two other possible responses are incongruent, as when the participant responds with corrugator to happy expressions and with zygomaticus to angry expressions. A person who responds with both cheek and brow activity upon the presentation of a smiling face is showing both a congruent (cheek to smile) and incongruent (brow to smile) response.

Because each participant has two opportunities for congruent responses (one for each stimulus type), each participant could show congruent responses 0% of the time (no congruent responses), 50% of the time (one congruent response) or 100% (two congruent responses) of the time. The same opportunities exist for incongruent responses. Because proportions are derived independently for congruent responses (out of total opportunities for congruent responses) and for incongruent responses (out of total opportunities for incongruent responses), they may sum to over 100%. Mimicry is demonstrated when there is a higher proportion of congruent than incongruent responses. Indiscriminant facial reactions are indicated by a high proportion of both congruent and incongruent responses (e.g. smiling to both happy and angry faces). An absence of facial reactions is indicated by a low proportion of both congruent and incongruent responses (e.g. no smiling to happy or angry faces). Thus, our primary data analysis tested in each group and each session the proportion of congruent responses (out of total opportunities for congruent responses) versus proportion of incongruent responses (out of total opportunities for incongruent responses). Further, we tested whether groups differed in discrimination between expressions (proportion of congruent vs. proportion of incongruent responses) or overall responsiveness to faces (overall proportion of responses). Importantly, note that our data analysis strategy, which tested the proportion of responses across two groups, limits the possibility that the group effects are driven by a few highly reactive individuals – a particular concern when comparing typical and atypical populations.

Results

Our first hypothesis was that typically developing participants would show more congruent than incongruent muscle responses (demonstrating automatic mimicry) whereas ASD participants would not show this pattern. The proportions of congruent and incongruent

² Although EMG is able to pick up muscle activation too quick and too small for human observation, a review of videotapes of the expressions of our participants, especially in the voluntary phase, indicates that as expected zygomatic activation was associated with smiles, and corrugator activation was associated with scowls.

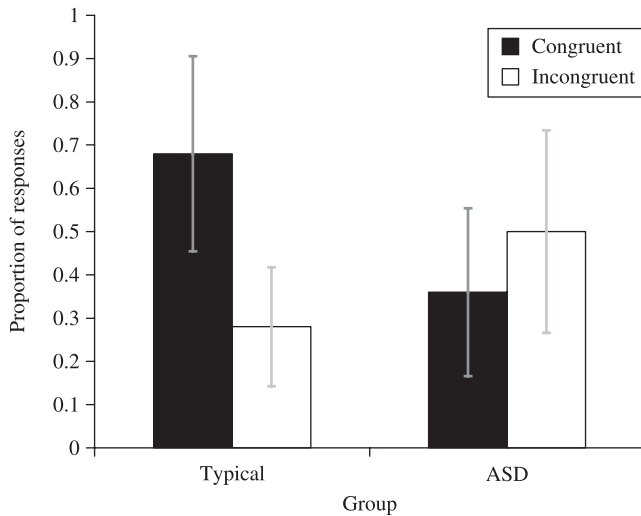


Figure 1 Proportion of spontaneous congruent and incongruent responses to emotion faces in typical participants and participants with ASD. Bars represent ± 2 standard errors.

responses in each group are displayed in Figure 1.³ As predicted, we found a significant Group by Congruence interaction, $F(1, 26) = 7.68$, $p = .01$, with t -tests showing that the typical group had a higher proportion of congruent (68%) than incongruent responses (29%), $t(13) = 4.20$, $p = .001$ (all t -tests are two-tailed). This demonstrates mimicry. The ASD group had fewer congruent (36%) than incongruent (50%) responses; this is the opposite of a mimetic pattern, but was not significant, $t(13) = 0.84$, $p = .41$. Moreover, the ASD group had a significantly lower rate (36%) of congruent responses than did the typical group (68%), $t(26) = 2.16$, $p = .04$.

Additional analyses help understand the pattern of facial responses in the ASD group. We also expected that ASD participants would demonstrate responses that are non-specific to valence (happy vs. angry faces), rather than a general absence of automatic responding. Combining congruent and incongruent responses, the rate of automatic responding in the ASD group was 43% (SE = 7%), which was significantly greater than 0%, $t(13) = 6.45$, $p < .0001$. The response rate in the typical group was 48% (SE = 8%), which also was significantly greater than 0%, $t(13) = 5.98$, $p < .0001$. Accordingly, the rates of the ASD (43%) and typical (48%) groups did not differ in terms of general responsiveness during the automatic phase, $t(26) = 0.51$, $p = .61$.

³ Recall that proportions are derived independently for congruent responses (out of total possibilities for congruent responses) and for incongruent responses (out of total possibilities for incongruent responses) and thus may sum to more than 100%.

In follow-up analyses, verbal ability, gender and age were unrelated to the degree to which ASD participants responded ($ps > .25$). We also explored whether the absence of mimicry in the ASD group was driven by incongruent responses to one particular type of face (e.g. did they simply scowl in response to happy *and* angry faces?). There was neither a significant effect of stimulus emotion, nor an interaction between stimulus emotion and congruence, indicating that responses did not differ based on whether the stimulus face was smiling or scowling ($ps > .59$). We also tested the possibility that the ASD group showed less automatic mimicry due to a slower response (delayed onset) or atypically sustained muscle activity (delayed offset) by examining windows from 400 ms to 800 ms post-stimulus onset. As with the standard window, there was no difference between congruent (43%, SE = 12%) and incongruent (50%, SE = 44%) response rates, $t(13) = 0.41$, $p = .69$.

Our second hypothesis was that both groups could voluntarily mimic emotional faces. The proportions of congruent and incongruent responses for each group displayed in Figure 2 suggest successful mimicry in both groups. The typical group showed a significantly higher proportion of congruent (100%) than incongruent responses (17%), $t(11) = 11.73$, $p < .0001$. Similarly, the ASD group showed a significantly higher proportion of congruent (96%) than incongruent (21%) responses, $t(11) = 9.95$, $p < .0001$. The ASD group's 96% congruent response rate was not different than the 100% rate of the typical group, $t(11) = 1.0$, $p = .34$. Again, there was no difference in overall responsiveness of the groups, when combining congruent and incongruent responses (58% for both groups).

Discussion

ASD participants did not automatically mimic facial expressions, whereas typical participants matched on age, gender and verbal intelligence did. In contrast, ASD and typical participants were equally successful on voluntary mimicry. Importantly, the superior performance of ASD participants on voluntary mimicry suggests that their absence of automatic mimicry was not due to deficits in perception, praxis, motivation or task understanding. Further, we found no evidence that the absence of automatic mimicry was due to differences in attention, latency or temporal profile of responding. Interestingly, in the automatic phase of the study, the ASD group showed the same overall rate of responding to faces, without, however, discriminating between happy or angry expressions. This finding additionally argues against inattention to the task as an explanation

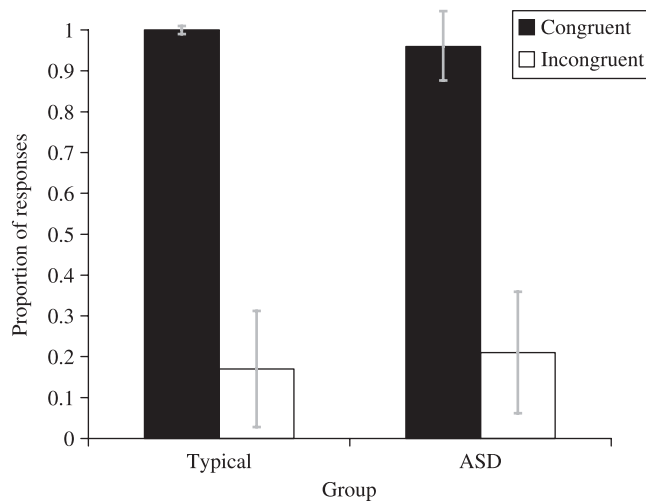


Figure 2 Proportion of voluntary congruent and incongruent responses to emotion faces in typical participants and participants with ASD. Bars represent ± 2 standard errors.

for the absence of mimicry. Finally, through the use of EMG, we were able to minimize the role of motivation to communicate a response. These results have implications for understanding mimicry and autism.

Mimicry and social functioning

As mentioned earlier, research documents a variety of automatic mimicry phenomena (Chartrand & Bargh, 1999; Dimberg, 1982; Lakin & Chartrand, 2003). Several authors have proposed that such mimicry is important for sociality (Bandura, 1977; Decety & Chaminade, 2003; Iacoboni, in press). Our data support this view by highlighting that a disorder of social functioning – autism – is associated with impairment of automatic mimicry.

Mimicry may involve a prefrontal ‘mirror circuit’ where neurons discharge when a similar action is executed and observed (Rizzolatti, Fadiga, Fogassi & Gallese, 2002). This circuit activates during facial mimicry among typical individuals (Carr, Iacoboni, Dubeau, Mazziotta & Lenzi, 2003). Mirror-circuit dysfunction in autism has been proposed (Williams *et al.*, 2001), and is supported by ASD individuals showing mirror-neuron abnormalities to observed hand actions (Obermann, Hubbard, McCleery, Ramachandran & Pineda, 2005). Future research could examine involvement of such abnormalities in automatic and voluntary facial mimicry. Interestingly, production of automatic (but not voluntary) facial expressions involves projections from both the prefrontal and premotor areas to the basal ganglia, limbic areas and brainstem via extrapyramidal pathways (Tassinari & Cacioppo, 2000). Given limbic involvement

in processing facial expressions, further research could explore contributions of amygdala abnormalities in autism (Baron-Cohen, Ring, Bullmore, Wheelwright, Ashwin & Williams, 2000).

Mimicry and imitation in autism

Our study found no autism deficit in voluntary mimicry. However, an earlier study found a deficit in voluntary imitation of certain facial actions (Rogers *et al.*, 2003). There are several important differences between these studies. We tested adolescents and adults and used EMG to assess simple muscle responses to static expressions. In contrast, Rogers *et al.* (2003) tested children and used observers to assess the quality of matching novel and complex facial actions performed by a live model (e.g. making a noisy kiss). These differences in procedures raise several possibilities for superior performance of our sample. First, our participants were older. Thus, they were more experienced with voluntary mimicry, and perhaps developed compensatory strategies. Second, our participants’ muscular responses detected by EMG could be typical, but their fully developed expressions could be different (although a review of their videotaped expressions did not show any grossly apparent differences). Third, our participants could be typical in mimicry of basic emotional expressions, but have a deficit in voluntary imitation of novel facial actions. Fourth, our participants could still be impaired in mimicry of live, dynamic, three-dimensional rather than static, two-dimensional expressions. Future research should address these possibilities.

Autism and affect

Our results support the idea of an emotion deficit in autism (Hobson, 1993; Rogers & Pennington, 1991). As mentioned above, the literature on emotion in autism is somewhat inconsistent, with some studies showing impairments and others not. Our work sheds light on this puzzle. The impairments may be present on tasks tapping automatic processes that involve quick generation of an appropriate, valence-specific response. However, impairments might be absent on emotion tasks tapping more voluntary processes. In fact, for many emotion tasks used in previous research, individuals with ASD, given sufficient time, might be able to use non-affective compensatory strategies to accomplish the task. Supporting this idea, in emotion recognition tasks, individuals with autism show activation of brain regions associated with intentional attentional allocation and categorization, rather than automatic processing (Hall, Szechtman & Nahmias, 2003).

Downstream consequences of mimicry deficits

The automatic mimicry deficit in our sample of adolescents and adults with ASD raises the questions of how early this deficit occurs and what are its downstream consequences. Typically developing newborns show rudimentary forms of facial mimicry (Meltzoff & Moore, 1989). If young autistic children have a mimicry deficit, the processes of social-emotional development that rely on co-experiencing others' affective states may be impaired. For example, a deficit in emotional contagion may prevent autistic children from developing the sense of intersubjectivity and emotional correspondence that are important for understanding of other minds and social learning (Bandura, 1977; Kasari *et al.*, 1993; Meltzoff & Gopnik, 1993).

More generally, examining rudimentary processes underlying mimicry and affective sharing can offer a more precise map of the psychological phenotype of autism, and insight into possible core deficits. Although there is evidence for cognitive deficits in autism (Baron-Cohen, 1995; Pennington, Rogers, Bennetto, Friffith, Reed & Shyu, 1997), these impairments do not fully characterize the autistic phenotype (Griffith, Pennington, Wehner & Rogers, 1999). Without denying the importance of cognitive deficits, we suggest that examining the role of automatic affective processes can advance theorizing about the mechanisms underlying autism (Dawson & Zanolli, 2003).

In conclusion, the current findings demonstrate that automatic mimicry, a basic feature of social interaction, is impaired in autism, a disorder of social cognition. As such, this research represents a step towards understanding what psychological processes shape both the typical and atypical social mind.

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