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PAPER

Slow echo: facial EMG evidence for the delay of spontaneous, but not voluntary, emotional mimicry in children with autism spectrum disorders

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Abstract

Spontaneous mimicry, including that of emotional facial expressions, is important for socio-emotional skills such as empathy and communication. Those skills are often impacted in autism spectrum disorders (ASD). Successful mimicry requires not only the activation of the response, but also its appropriate speed. Yet, previous studies examined ASD differences in only response magnitude. The current study investigated timing and magnitude of spontaneous and voluntary mimicry in ASD children and matched controls using facial electromyography (EMG). First, participants viewed and recognized happy, sad, fear, anger, disgust and neutral expressions presented at different durations. Later, participants voluntarily mimicked the expressions. There were no group differences on emotion recognition and amplitude of expression-appropriate EMG activity. However, ASD participants' spontaneous, but not voluntary, mimicry activity was delayed by about 160 ms. This delay occurred across different expressions and presentation durations. We relate these findings to the literature on mirroring and temporal dynamics of social interaction.

> One may find by one's own observation that the imitation of the bodily expression of a mental condition makes us understand it much better than the merely looking on. (W. Fechner, as quoted in James, 1890)

Introduction

Neurotypical individuals overtly and covertly mimic behavior of those around them, including gestures, postures, tone of voice, pronunciation patterns, and even breathing rates (Chartrand & Bargh, 1999; Condon & Ogston, 1967; Kendon, 1970; McFarland, 2001; Neumann & Strack, 2000; Paccalin & Jeannerod, 2000). One robust case of mimicry occurs when the observation of another person's emotional facial expression elicits a corresponding expression in the observer (Bush, Barr, McHugo & Lanzetta, 1989; Wallbott, 1991). In typical individuals, facial mimicry has three characteristics. First, it occurs spontaneously during mere observation of the face, without external prompting or a goal to mimic (Dimberg, 1982; Dimberg & Lundqvist, 1988).[1] Second, it requires little stimulus input, and occurs even when faces are presented subliminally (Dimberg, Thunberg & Elmehed, 2000). Third, it is fast, with the response typically emerging within one second after the stimulus onset (Dimberg, 1982; Dimberg *et al.*, 2000).

Spontaneous mirroring, including facial mimicry, has been proposed to facilitate social skills such as empathy and emotional reciprocity via a process of contagion and an internal simulation of the observed emotion (Decety & Chaminade, 2003; Iacoboni, 2005; Lipps, 1907; Niedenthal, 2007; Oberman, Winkielman & Ramachandran, 2007). This process is supported by several brain systems. In typical individuals, fMRI studies suggest that production and, in some reports, mere observation of facial expressions is associated with enhanced activation in a region of premotor cortex (Brodmann's Area 44), a putative component of the human mirror neuron system (Carr, Iacoboni, Dubeau, Mazziotta & Lenzi, 2003; Dapretto, Davies, Pfeifer, Scott, Sigman, Bookheimer & Iacoboni, 2005; Leslie, Johnson-Frey & Grafton, 2004). There is also evidence for the role of regions involved in emotional processes, such as the amygdala and anterior insula (Carr *et al.*, 2003) and regions involved in somatosensory representation of the face (Adolphs, 2002).

[1] Some mimicry components might be present shortly after birth (Meltzoff & Moore, 1997).

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Mimicry and autism spectrum disorder

There is much interest in how mimicry contributes to the development of typical and atypical social skills, especially empathy and emotional reciprocity (Rogers & Williams, 2006). Deficits in these domains are central to autism spectrum disorders (Kanner, 1943; Oberman & Ramachandran, 2007). Though several studies suggest deficits in spontaneous mimicry in ASD, the exact nature of the impairment remains mysterious.

McIntosh, Reichmann-Decker, Winkielman and Wilbarger (2006) investigated facial mimicry in ASD and typical individuals using electromyography (EMG). Their study had two phases. In the first 'spontaneous' phase, participants were asked to 'just watch' large pictures of happy and angry expressions presented for 8 seconds. In the second 'voluntary' phase, participants were explicitly asked to 'make the faces on the screen'. The results showed that in the spontaneous phase, typical individuals activated facial muscles corresponding to the observed expression (zygomaticus major, which lifts the cheeks, in response to happiness, and corrugator supercilii, which furrows the brows, in response to anger). In contrast, ASD participants showed a non-specific pattern of spontaneous EMG activity, with similar level of zygomaticus and corrugator responses to happiness and anger. In contrast, during the voluntary phase, both groups showed similar, expression-specific patterns of muscle activation. A related result was reported in an fMRI study by Dapretto and colleagues (2005). During passive observations of 2-second presentations of angry, fearful, happy, sad, and neutral expressions, ASD participants, compared to typical controls, showed a reduced level of activity in the premotor mirror neuron area. Again, there were no group differences during a voluntary imitation task. Finally, in a recent study by Stel, van den Heuvel and Smeets (2008), participants were asked to simply watch a 5-min video in which a male student talked about his adventures in an amusement park, displaying happy expressions. Analysis of experimenter-coded facial expressions and gestures revealed that ASD participants showed less spontaneous mimicry, as compared to PDD-NOS and control participants. Importantly, as in McIntosh *et al.* (2006), these effects were observed despite no differences in the amount of time spent looking at the screen and no differences in voluntary mimicry.

However, some recent studies suggest that ASD participants do show spontaneous mimicry when processing of mimicry-relevant aspects of the stimuli is encouraged by the task. For example, Magnee and colleagues (2007) gave typical and ASD participants a task that required integration of visual and auditory cues to a person's emotional state. In this task, participants saw happy or fearful faces, presented for 900 ms before the onset of emotion auditory cues (happy or fearful voice). These faces, which participants judged on gender, were paired with either congruent or incongruent voice, thus encouraging attention to and processing of the emotional dimension of both the face and voice stimuli. Under these task conditions, the results showed comparable amplitude of emotion-congruent facial EMG responses between typical and ASD participants.

Studies of non-emotional mimicry also suggest that when ASD participants are encouraged to process relevant stimulus dimensions, they show similar levels of spontaneous mirroring as neurotypical participants. For example, there are many reports of reduced motor mimicry in ASD participants (Nishitani, Avikainen & Hari, 2004; Oberman *et al.*, 2005; Theoret, Halligan, Kobayashi, Fregni, Tager-Flusberg, & Pascual-Leone, 2005; Villalobos, Mizuno, Dahl, Kemmotsu & Muller, 2005). However, Theoret and colleagues (2005) found that this reduction was present only when the action was displayed from an egocentric view (away from the observer). When the same stimulus was displayed from an allocentric perspective (toward the observer), a 'typical' response was found. Additionally, Oberman, Ramachandran and Pineda (2008) showed a typical degree of mu suppression (an EEG index of mirroring activity) in response to an action performed by a family member, or the participant himself, but not to the same action performed by a stranger.

In sum, it appears that the mimicry system is able to come on-line in ASD participants when they are encouraged to process the mimicry-relevant aspects of the stimulus, which can result from attentional, motivational, and other task factors (we will return to these mechanisms in the Discussion). However, even when mimicry occurs, is the timing typical? This question is imporant given the growing appreciation of the temporal dynamics of mimicry for emotion understanding and interpersonal coordination.

Temporal dynamics

Timely processing and responding to socially relevant information is 'the bedrock of all social interaction' (Crown, Feldstein, Jasnow, Beebe & Jaffe, 2002). Appropriate temporal resolution is important for interpersonal perception (Crown, 1982; Feldstein, 1982), communication of mood (Natale, 1978), empathy (Welkowitz & Feldstein, 1970), understanding of intentions (Baldwin, 1993; Tomasello, 1999), and theory of mind abilities (Blakemore, Boyer, Pachot-Clouard, Meltzoff, Segebarth & Decety, 2003). The ability to coordinate timing in social situations emerges as early as 4 months of age (Jasnow, Crown, Feldstein, Taylor, Beebe & Jaffe, 1988), suggesting its importance for development of social skills.

Several studies of temporal properties of perceptual and response systems suggest that ASD participants show delayed responses to social stimuli. ERP studies (McPartland, Dawson, Webb, Panagiotides & Carver, 2004; Webb, Dawson, Bernier & Panagiotides, 2006) found delayed latency of the face-processing n170 component during passive observation of faces. ASD children also show delays in the n300 component, which responds differentially to fearful faces, and this delay is predictive

of joint attention skills, social orienting, and time spent looking at an experimenter expressing distress (Dawson, Toth, Abbott, Osterling, Munson, Estes & Liaw, 2004). There is also evidence for atypical development of cerebellar systems, governing integration of sensory and motor signals, and their connections with the rest of the cortex (Courchesne, 1997; Ivry, Spencer, Zelaznik & Diedrichsen, 2002).

Given the reports of temporal abnormalities in analysis and response to social information in ASD individuals, it is likely that, even when ASD participants process relevant emotion information and show typical overall levels of responding, their spontaneous mimicry is delayed. This important possibility has not yet been addressed in previous studies, which have focused only on measures of amplitude, but not timing (Dapretto *et al.*, 2005; Magnee *et al.*, 2007; McIntosh *et al.*, 2006; Stel *et al.*, 2008).

Current study

The current study investigated the timing of spontaneous mimicry during viewing of emotional expressions in high functioning ASD individuals and controls using facial electromyography (EMG). This technique relies on changes in the electrical activity of the facial muscles and offers high temporal resolution and good spatial specificity. Thus, EMG is ideal for the current goal of capturing fast and subtle changes occurring during spontaneous facial mimicry (Dimberg, 1982). Furthermore, EMG is not dependent on verbal skills, praxis, or motivation (Tassinary & Cacioppo, 2000) which is a concern when studying children with ASD in whom it is difficult to discriminate between performance deficits caused by differences in actual response versus motivation or ability to express that response.

To engage participants in the task and to control for level of emotion recognition, we used different emotional expressions and asked participants to report on each trial what emotion they thought the person was experiencing. Because our task required participants to process facial emotion, we expected that both groups would exhibit spontaneous mimicry (Magnee *et al.*, 2007). However, given previous research indicating ASD deficits in temporal processing, we expected this group to show slower spontaneous mimicry. To control for potential impairments in perception of emotional faces, we included two additional manipulations. First, we presented stimuli with very fast (25 ms), fast (75 ms) and slow (1000 ms) speeds. We also used six different expressions that vary in the ease of recognition (happy, angry, disgust, fear, sad, and neutral). If the processing impairment is primarily perceptual, fast speeds and difficult to recognize emotions (e.g. sad and disgust) should be associated with greater mimicry delays. If the impairment lies primarily in spontaneous engagement of sensory-motor response mechanism, the delays of mimicry should occur even for long presentation durations, and even for easily recognizable expressions (such as happy and angry).

Following the test of spontaneous mimicry, participants performed a voluntary mimicry task, which controls for non-specific task impairments and any non-specific muscular delays. As in earlier studies, we did not expect any impairment in voluntary mimicry. In addition to standard autism diagnostics, we also measured participants' level of empathy.

Method

Participants

Participants were 13 male children with ASD and 13 age-matched male typically developing children (age range 8–12 years, ASD: $M = 10.2$, $SD = 1.4$; TD: $M = 10.2$, $SD = 1.4$). All participants had normal hearing and normal, or corrected to normal, vision. ASD participants were recruited through Valerie's List, a listserv of families and professionals in the autism community, and were diagnosed by a licensed clinical psychologist or medical doctor. This diagnosis was verified in our laboratory through administration of the Autism Diagnostic Observation Schedule – Generic (ADOS-G; Lord, Risi, Lambrecht, Cook, Leventhal & DiLavore, 2000) by a trained individual. Based on the results of these assessments and clinical judgment, seven of the 13 children met criteria for *Autistic Disorder* and six met criteria for *Autism Spectrum Disorder* (scores ranging from 7 to 20, $M = 13.6$, $SD = 5.3$). All participants were considered high-functioning, defined as having age-appropriate verbal comprehension abilities and an IQ greater than 80 as assessed by the Wechsler Abbreviated Scale of Intelligence (WASI) (ASD: scores ranging from 85 to 125, $M = 102.8$, $SD = 15.8$; TD: scores ranging from 91 to 147, $M = 112.5$, $SD = 17.3$). Neurotypical controls were from the San Diego area, had no neurological or psychological disorder, and were matched on chronological age and gender with a participant in the ASD group. Participants were given age-appropriate assents and the parent/guardian provided written consent for his/her child's participation. This project was approved by the university IRB board.

Empathy and theory of mind tests

Empathy was assessed with standard measures modified for the population of the study (Empathetic Concern Subscale of the Interpersonal Reactivity Index (IRI); Davis, 1980; and Basic Emotional Empathy Scale (BEES); Mehrabian, 1996). As these scales were originally designed for adults, to use them in the current study with children the statements were modified from a first-person viewpoint to a third-person viewpoint (e.g. 'Other people's misfortunes do not usually disturb me a great deal' was modified to read 'Other people's misfortunes do not usually disturb my child a great deal') so that the parent/guardian could evaluate how well the scale items described their child.

Table 1 *Results from behavioral testing. Means, standard deviations (in parentheses) and statistical comparison for the two groups*

Behavioral test	ASD	Neurotypical	Significance (one-tailed)
Basic Emotional Empathy Scale (BEES)	−10.04 (33.62)	51.00 (28.62)	$p < .0001$
Interpersonal Reactivity Index (IRI)-Empathetic Concern Scale	10.85 (7.12)	20.62 (3.71)	$p < .0002$
Interpersonal Reactivity Index (IRI)-Perspective Taking Scale	3.54 (4.07)	13.26 (4.27)	$p < .0001$

Theory of mind skills were assessed with the Perspective-Taking subscale of the IRI which is designed to measure the degree that the participant is able to cognitively take the perspective of another. The mean standardized scores and standard deviations for each group are presented in Table 1. As expected, the neurotypical group scored higher on empathy and theory of mind than the ASD group (BEES: $t(24) = 24.84$, $p < .0001$, IRI-EC: $t(24) = 19.27$, $p < .0002$, IRI-PT: $t(24) = 36.71$, $p < .0001$).

Stimuli

Stimuli were 192 photos of facial expressions (10×10 cm) from the Mac Brain Stimulus Set presented on a 15-in monitor approximately 80 cm away from the participant. Stimuli were presented at three exposure levels (25 ms, 75 ms, and 1000 ms), with the order of presentation of stimuli and exposure level randomized. Face presentation was preceded by a 1000 ms central fixation cross.

Procedure

As in earlier research, this study had two blocks. In the first 'spontaneous' block, participants were asked to watch facial expressions presented on a computer screen and classify them as expressing happy, sad, angry, fear, disgust, or neutral. Performance on this task was collected and analyzed for accuracy. In the second block, participants were explicitly told to make the same expression as they saw on the screen then classify it as they had done in block one. During this block the stimulus always remained on the screen for 1000 ms to ensure sufficient input. As in earlier research, the voluntary block was always second to ensure that the participant did not have any predisposition to voluntarily mimic during the spontaneous block.

EMG data processing

EMG signals were measured with pairs of 4-mm silver/silver-chloride electrodes. Two adjacent electrodes, referenced to one another, were each placed over five groups of facial muscles associated with different emotional expressions. Specifically, we expected happy expressions to activate zygomaticus major, which pulls up the cheek, and angry expressions to activate the corrugator supercilii, which furrows the brow (Dimberg, 1982; Tassinary & Cacioppo, 2000). Fear expressions were expected to activate the medial frontalis, which raises the inner eyebrow (Ekman & Friesen, 1978). Disgust expressions were expected to activate the levator, which crinkles the nose (Vrana, 1993), whereas sad expressions were expected to activate depressor anguli oris, which pulls the lips downward (Ekman & Friesen, 1978). An additional ground electrode was placed in the upper portion of the forehead. The impedances of all electrodes were reduced to less than 15 kΩ. The location of the electrodes and recording technique conformed to EMG standards (Tassinary & Cacioppo, 2000).

EMG signals were acquired with a Biopac hardware and Acknowledge software. The signals were sampled at 2000 Hz, amplified by 2000, and filtered with a 10–500 Hz bandpass. The signals were then integrated and rectified. Average values were obtained for each 100 ms interval from 1000 ms pre-stimulus onset to 2000 ms post-stimulus, creating 30 distinct 100 ms interval values per trial.

EMG data cleaning and reduction

The signals were screened for movement and electrical artifacts by a blind coder. Trials containing artifacts (less than 5%) were removed prior to analysis. Next, the data were logarithmically transformed, reducing the impact of extreme values, and standardized (i.e. expressed as Z scores) within participants and muscle sites, attenuating the impact of highly reactive individuals on group scores and allowing for meaningful comparisons across sites (Tassinary & Cacioppo, 2000). Next, baseline values for each trial were calculated as the average EMG activity from 500 ms to zero ms prior to stimulus presentation. Then, baseline-corrected activity was calculated for each 100 ms interval from 100 ms post-stimulus to 2000 ms post-stimulus (i.e. values greater than 0 represent an increase over stimulus baseline). Finally, to obtain one value for every 100 ms interval of each trial type, trials of the same emotion and stimulus presentation length were averaged.

Analysis

Several measures were obtained from the EMG data. First, we calculated the peak amplitude, defined as the highest value occurring between 300 and 2000 ms followed by a reduction in activity of a minimum of 0.1 Z. Second, we calculated a latency of the peak response, defined as the 100 ms interval in which the peak occurred. Third, in order to quantify the amplitude and

Table 2 *Results from emotional recognition task. Percent of correct recognitions for the two groups for the six emotional facial expressions and three duration conditions*

	Neurotypical				ASD			
	Very fast	Fast	Slow	Voluntary	Very fast	Fast	Slow	Voluntary
Angry	71.4	74.1	79.4	80.7	74.1	42.3	66.8	67.3
Disgust	41.9	51.3	62.9	75.3	38.9	57.2	69.1	75.1
Fear	53.9	70.4	66.3	66.8	50.0	56.7	61.1	60.7
Happy	76.1	82.7	97.5	93.3	79.3	87.0	97.6	94.5
Neutral	67.9	68.7	88.0	91.8	64.4	71.2	81.1	80.5
Sad	58.7	56.1	83.4	73.4	49.6	46.8	70.2	57.4

timing of overall response, we calculated an average activity within the window of 300 ms to 1000 ms (early time window) as well as 1000 ms to 2000 ms (late time window). These windows were chosen based on previous findings suggesting that in typical individuals, spontaneous mimicry typically begins around 300–500 ms and peaks around 1000 ms post-stimulus onset, with the exact timing of the peak depending on stimulus duration (Dimberg, Thunberg & Grunedal, 2002; McIntosh *et al.*, 2006).

Proper mimicry involves emotion-selective responding, which involves activation of the muscle matching the observed expression (zygomaticus to happy, corrugator to angry), but also the lack of activation of inconsistent muscles. Thus, as in earlier studies, in addition to activity for expression-appropriate muscles, we examined activity of expression-inappropriate muscles (corrugator to happy, and zygomaticus to angry).

Results

Behavioral performance checks

As shown in Table 2, participants in both groups did well on the emotional recognition task, with accuracy for every emotion well above chance (17%; 1 out of 6). In fact, even at the fastest speeds, the lowest recognition rate (for the ASD group for the disgust expression) was 39%. The behavioral performance was analyzed with the 6 (Emotion: Angry, Disgust, Fear, Happy, Neutral, Sad) by 3 (Duration: Very Fast, Fast, Slow) by 2 (Group: ASD, Neurotypical) mixed-model factorial ANOVA on accuracy of emotion recognition. This analysis revealed no main effects or interactions involving group (all *p*s > .23). This finding is consistent with reports that high-functioning ASD individuals show impairments only in difficult tasks of emotion recognition (see Humphreys, Minshew, Leonard & Behrmann, 2007).

The ANOVA did reveal, however, a main effect of Emotion, $F(5, 220) = 8.3$, $p < .001$, $\eta^2 = 0.27$, with happy (87%) and angry (68%) expressions recognized at higher rates than the other emotions (a standard effect in emotion literature; Ekman, 2004). There was also the expected main effect of duration, with longer presentations resulting in more accurate recognition, $F(2, 220) = 15.58$, $p < .001$, $\eta^2 = 0.41$.[2]

EMG response

EMG analyses employed two complementary ways of assessing effects on response timing and magnitude (see Tassinary & Cacioppo, 2000). For timing, we analyzed peak latency – the time point of the strongest response followed by a decline. We also compared relative levels of activity in early and late time windows, as defined earlier. For magnitude, we analyzed peak level (amplitude of the strongest response) and the average level of activity in early and late time windows. Note that the peak and time window analyses are not identical, as they use different temporal widths, but are complementary (e.g. slower peaks should correspond to more activity in a later window).

Preliminary mixed-model 3 × 5 × 2 ANOVA with Duration (Very Fast, Fast, Slow), Emotion (Happy, Anger, Sad, Disgust, and Fear), and Group (ASD, Neurotypical) showed that Duration did not interact with Group on any measure, showing only a main effect such that longer presentations yielded later peak latency response, $F(2, 160) = 5.26$, $p < .01$, $\eta^2 = 0.21$ (see Figure 1). Similarly, longer duration was associated with greater average activity in the later, as opposed to earlier time window ($p < .05$). Accordingly, subsequent EMG analyses collapsed across duration and used a simpler 2 (Group) × 5 (Emotion) mixed-model ANOVA.

Timing effects

As shown in Figure 2, there was a significant main effect of group on peak latency, with the ASD group responding 159 ms slower than the neurotypical group, $F(1, 24) = 8.70$, $p < .01$, $\eta^2 = 0.27$. This effect is also detectable in median latency (ASD group = 1133 ms, neurotypical group = 1033 ms). To further ensure that the results were not driven by outliers, we compared the percentage of participants in each group who were below the overall

[2] There was also a theoretically uninteresting Emotion by Speed interaction with all recognition of all emotion, but anger benefiting from longer presentations, $F(10, 220) = 2.3$, $p < .05$.

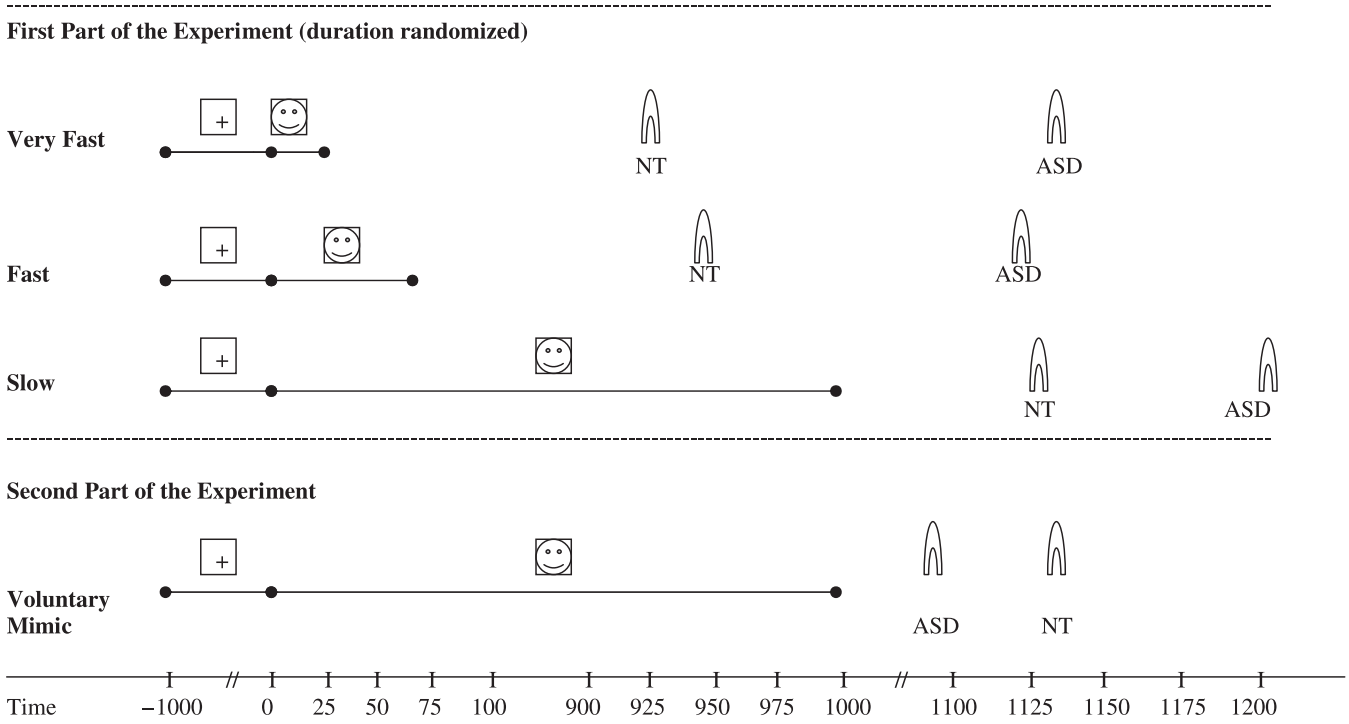

Figure 1 *Procedure and overall latency results. Cross symbol represents fixation cross presented to participant. Face represents presentation of facial expression. Arch represents peak facial mimicry response in the appropriate facial muscle for both the Neurotypical (NT) and Autism Spectrum Disorder (ASD) group for the very fast, fast, slow, and voluntary mimicry conditions.*

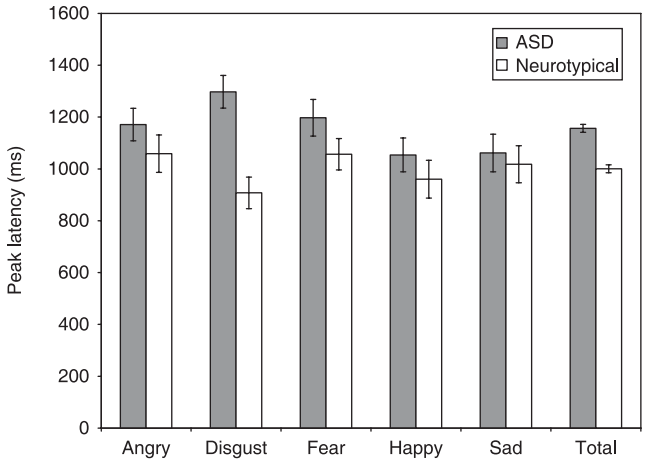

Figure 2 *Peak latency for each group. Bars represent the average latency of EMG peak in milliseconds for each group. Error bars represent the standard error of the mean.*

sample median of 1070 ms – 69% of typical participants (9 out of 13) were below the overall median, as compared to 31% (4 out of 13) of the ASD participants (binomial test, $p < .05$).

The 2 (Group) by 5 (Emotion) ANOVA revealed no interaction with Emotion, indicating that mimicry was delayed across all expressions ($p > .15$). A similar conclusion is suggested by simple comparison of the average level of activity of emotion-appropriate facial muscles in the early (300 ms–1000 ms) and late (1000 ms–2000 ms) time windows, shown in Table 3. In this analysis, the ASD group, but not the typical group, showed overall more activity in the late, as compared to the early window, $t(12) = 3.15$, $p < .01$. Finally, the group difference in the timing of spontaneous mimicry can be illustrated by aggregated waveforms shown in Figure 3. For every emotion, the highest value, followed by a decline, occurs earlier for the neurotypical than for the ASD group.[3] In short, it appears that on a variety of indicators, ASD participants show a delay in spontaneous mimicry.

Table 3 *EMG activity during the early and late time windows. Means and standard deviations (in parentheses) of EMG activity for the two groups for five emotional facial expressions*

	Neurotypical		ASD	
	Early	Late	Early	Late
Angry	0.23 (0.60)	0.28 (0.45)	0.28 (0.64)	0.64 (0.61)
Disgust	–0.08 (0.86)	–0.06 (0.63)	–0.21 (0.49)	0.33 (0.92)
Fear	0.24 (0.61)	0.27 (0.74)	0.12 (0.58)	0.30 (0.73)
Happy	0.54 (0.69)	0.50 (0.82)	0.50 (0.52)	0.73 (0.70)
Sad	0.01 (0.46)	0.44 (0.69)	–0.12 (0.72)	0.08 (0.67)
Total	0.19 (0.41)	0.29 (0.37)	0.11 (0.40)	0.42 (0.50)

[3] Note that our peak finding procedure was conducted on individual-level data using criteria discussed earlier, separately for each stimulus duration. For this, and other reasons, interpreting peaks in aggregated data should be done with caution, but we provide them for illustrative purposes.

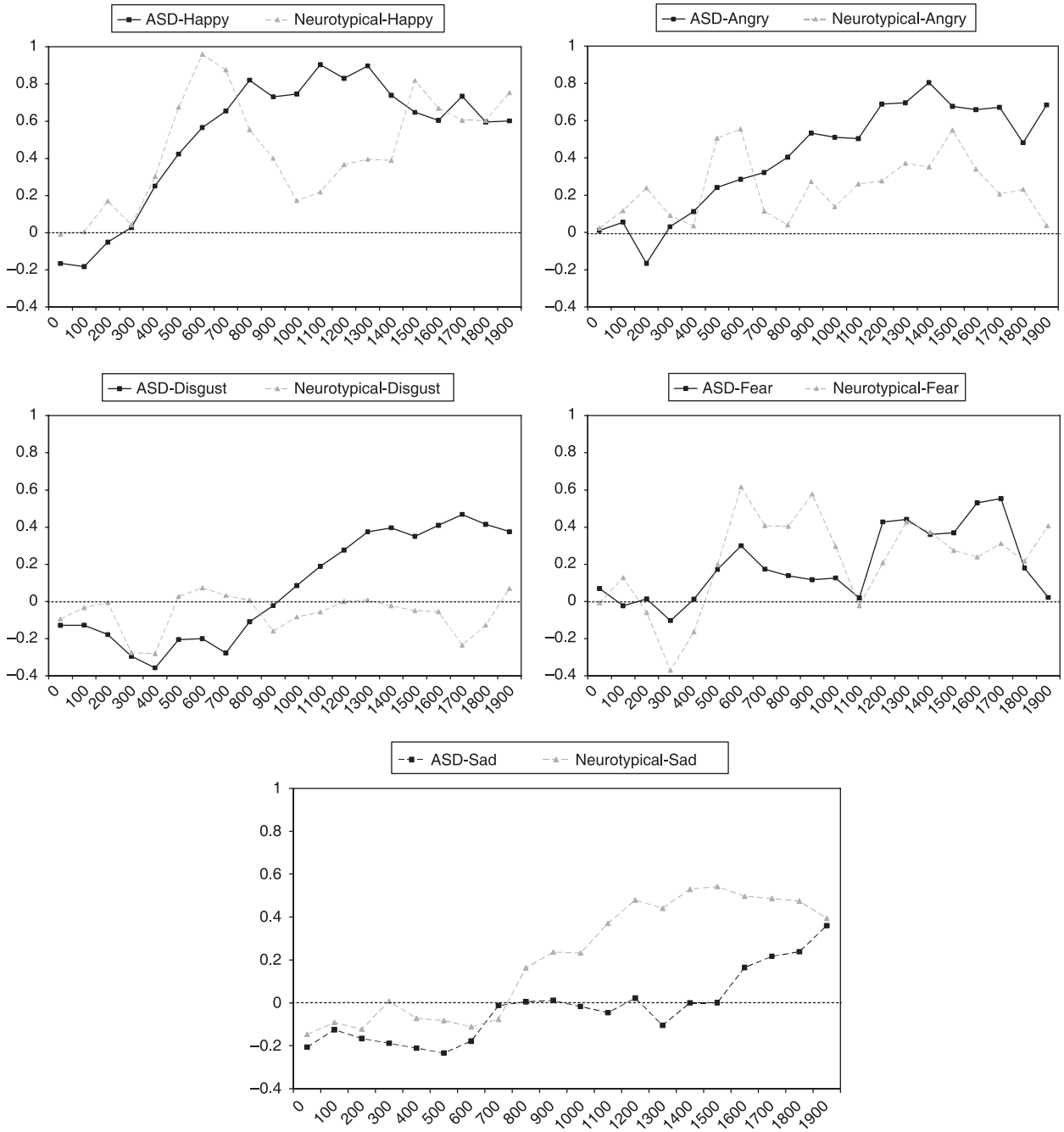

Figure 3 *Aggregated waveforms for each emotion for both groups. Graphs represent the time course of the normalized EMG response across the time window from stimulus onset to 2000 ms post-stimulus onset for the five emotional facial expressions. The dark black lines represent the ASD group while the lighter gray lines represent the neurotypical group. The dotted line represents the value at baseline (average of the EMG response for the 500 ms prior to stimulus onset).*

Response level

The aforementioned Group × Emotion ANOVA revealed no main effects or interactions involving Group on peak amplitude. For all emotions, peak amplitude was greater than zero (pre-stimulus baseline). There was, however, a main effect of emotion, suggesting that some emotions produced stronger responses than others, $F(4, 96) = 2.8$, $p < .05$, $\eta^2 = 0.10$. Subsequent *t*-tests revealed that the observation of anger resulted in significantly greater peak amplitude than fear, disgust, or sadness (all *p*s < .01). The observation of happiness resulted in significantly greater peak amplitude than disgust or sadness (both *p*s < .01). The observation of fear resulted in a significantly greater peak amplitude than disgust or sadness (both *p*s < .05).

Similar findings were obtained in analysis of average activity level in early and late time windows (see Table 3). There was a main effect of window, with overall more activity in the late than early window, $F(1, 96) = 13.00$,

$p < .001$, $\eta^2 = .35$, and a main effect of emotion, $F(4, 96) = 4.79$, $p < .001$, $\eta^2 = 0.17$. Overall response to happiness was greater than anger, fear, disgust, or sadness (all ps $< .05$), whereas a response to anger was greater than to disgust or sadness (both ps $< .05$). The observation of fear resulted in a significantly greater average activity than disgust ($p < .05$). In sum, the analysis of the response level showed that both groups spontaneously mimicked, with peak and average response to happy and angry expressions stronger than other expressions (Lundqvist & Dimberg, 1995).

Response selectivity

It was important to confirm that EMG responses in both groups were selective to the appropriate muscles, rather than reflecting non-specific activation across all muscles. In the facial mimicry literature, selectivity is typically tested by examining the responses of muscles that are most robustly activated by stimuli of different valence and that are physically distant as to minimize signal cross-talk. Typically, the two expressions eliciting responses matching these criteria are happiness and anger. These expressions are opposite in valence, elicit strong responses, and selectively engage distant muscles (zygomaticus in a lower part of the face versus corrugator in the upper part of the face). As described earlier, in our study, as in previous research, anger and happiness elicited the strongest EMG responses. Accordingly, in the current study we tested for selectivity defined as presence of congruent activation (zygomaticus to a smile and corrugator to a frown), but not incongruent activation (zygomaticus to a frown and corrugator to a smile). Both groups showed selective responses on peak amplitude, with congruent peak greater than incongruent peak, Typical, $t(12) = 7.34$, $p < .001$, ASD, $t(12) = 9.98$, $p < .001$. Similarly, analysis of responses across both early and late time windows revealed that overall activity of congruent muscles was greater than incongruent muscles, Typical, $t(12) = 8.16$ $p < .001$, ASD, $t(12) = 5.13$, $p < .001$. Further, consistent with the aforementioned timing effects, the ASD group responded more selectively in the late, as compared to earlier, time window, $t(12) = 2.45$, $p < .05$, whereas the typical groups responded similarly across both windows ($p > .85$).

Voluntary movement block

Similar to McIntosh and colleagues (2006), during the voluntary mimicry block we found no significant main effects or interaction with group on all relevant measures: peak latency, peak amplitude, or average activity in early and late time window. In fact, on peak latency, ASD participants were slightly, though non-significantly, faster (1073 ms) than neurotypical participants (1175 ms). Further, for each emotion, participants in both groups showed a peak amplitude and average activity in both time windows significantly above the baseline. Again, this activity was expression specific, with both groups showing smiling to smiles and frowning to frowns (all ps $< .01$).[4]

Discussion

Our primary finding is that ASD individuals show delayed spontaneous mimicry across a variety of expressions and stimulus durations. This delay occurred despite no group differences in amplitude or selectivity of emotion-relevant EMG activity or differences in behavioral performance on emotion recognition. Finally, as in earlier studies, we found no ASD impairments on any measure during a voluntary mimicry task.

Our results are consistent with recent research that ASD individuals show spontaneous mimicry when the task engages them, even indirectly, in emotion processing (Magnee *et al.*, 2007) and extend earlier research that passive observation of expressions fails to bring the mimicry system online (Dapretto *et al.*, 2005; McIntosh *et al.*, 2006; Stel *et al.*, 2008). Our results are also consistent with recent literature on motor mimicry. Thus, ASD children who fail to show spontaneous EEG mu suppression response (indicating motor mirroring) to a stranger, show typical response to a familiar person (Oberman *et al.*, 2008). They also show motor facilitation when the observed gesture is directed towards them (Theoret *et al.*, 2005). Thus, perhaps typically developing children activate their mirroring system automatically, while ASD children engage it only under specific conditions. Future studies are necessary to elucidate the exact factors that facilitate spontaneous mimicry in ASD, including attention to mimicry-relevant aspects of the stimulus, motivation to engage, ability to map self to others, and other task requirements.

Although spontaneous mirroring occurs in ASD, it is delayed. This delay is unlikely to reflect purely perceptual slowdown. After all, it was found across the range of presentation durations, across differently recognizable expressions, and was larger than typical perceptual delays observed in ERP studies (about 160 ms vs. 20 ms). The delay is also unlikely to reflect general motoric slowdown. After all, ASD participants were as fast as neurotypicals on voluntary mimicry (though spontaneous and voluntary mimicry are partially controlled by different circuits; Morecraft, Stilwell-Morecraft & Rossing, 2004). Our pattern of results, along with other studies, suggests a potential issue with automatic engagement of sensory-motor mechanisms involved in timing of social interactions. Such a proposal is consistent with recent neuroscientific

[4] Because the voluntary block always followed the spontaneous block, direct comparisons of timing in different mimicry blocks must be made with caution. However, it is interesting that the ASD group was slightly, though nonsignificantly, faster on voluntary mimicry than on spontaneous mimicry on comparable duration trials (1000 ms), whereas the neurotypical group had the opposite pattern. It is also interesting that we found no significant correlations between the latency of responses during the voluntary mimicry block and the spontaneous mimicry block for either group.

evidence. First, there are reports of abnormal functional connectivity in ASD. Specifically, studies using Diffusion Tensor Imaging (DTI) found a reduction in long-range axons in ASD (Barnea-Goraly, Kwon, Menon, Eliez, Lotspeich & Reiss, 2004) and an increase in local cortico-cortical connections as measured by volumetric studies indicating white matter hyperplasia (Carper, Moses, Tigue & Courchesne, 2002). These conditions might reduce or delay communication between visual cortices and frontal cortices necessary for initiating fast spontaneous responses to stimuli. Mimicry delays might also be related to ASD abnormalities in the cerebellum, which is involved in efficient and timely coordination of sensory input with motor output (Courchesne, 1997; Ito, 2002; Ivry & Keele, 1989). In fact, some suggest that the Purkinje cells, which are atypical in ASD, play a critical role in tasks requiring temporal resolution on the order of a few hundred milliseconds (Ivry, 1996; Ivry *et al.*, 2002).

The findings just discussed call for further research linking anatomical differences and functional differences, as observed in the current study, in timing of social responses. Research should also address how experience and training can improve not only the presence, but also the timing of spontaneous mimicry in ASD (Rogers & Williams, 2006). All these efforts will hopefully elucidate the role of timing and magnitude of mimicry in social interactions of typical and atypical individuals, including their capacities for emotional contagion, empathy, and accurate social and emotional understanding (Winkielman, Niedenthal & Oberman, 2008).

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References

Adolphs, R. (2002). Recognizing emotion from facial expressions: psychological and neurological mechanisms. *Behavioral and Cognitive Neuroscience Reviews*, **1**, 21–62.

Baldwin, D.A. (1993). Early referential understanding: infants' ability to recognize referential acts for what they are. *Developmental Psychology*, **29**, 832–843.

Barnea-Goraly, N., Kwon, H., Menon, V., Eliez, S., Lotspeich, L., & Reiss, A.L. (2004). White matter structure in autism: preliminary evidence from diffusion tensor imaging. *Biological Psychiatry*, **55**, 323–326.

Blakemore, S.J., Boyer, P., Pachot-Clouard, M., Meltzoff, A., Segebarth, C., & Decety, J. (2003). The detection of contingency and animacy from simple animations in the human brain. *Cerebral Cortex*, **13**, 837–844.

Bush, L.K., Barr, C.L., McHugo, G.J., & Lanzetta, J.T. (1989). The effects of facial control and facial mimicry on subjective reactions to comedy routines. *Motivation and Emotion*, **13**, 31–52.

Carper, R.A., Moses, P., Tigue, Z.D., & Courchesne, E. (2002). Cerebral lobes in autism: early hyperplasia and abnormal age effects. *NeuroImage*, **16**, 1038–1051.

Carr, L., Iacoboni, M., Dubeau, M.C., Mazziotta, J.C., & Lenzi, G.L. (2003). Neural mechanisms of empathy in humans: a relay from neural systems for imitation to limbic areas. *Proceedings of the National Academy of Sciences*, **100**, 5497–5502.

Chartrand, T.L., & Bargh, J.A. (1999). The chameleon effect: the perception–behavior link and social interaction. *Journal of Personality and Social Psychology*, **76**, 893–910.

Condon, W.S., & Ogston, W.D. (1967). A segmentation of behavior. *Journal of Psychiatric Research*, **5**, 221–235.

Courchesne, E. (1997). Brainstem, cerebellar and limbic neuroanatomical abnormalities in autism. *Current Opinions in Neurobiology*, **7**, 269–278.

Crown, C.L. (1982). Impression formation and the chronography of dyadic interactions. In M. Davis (Ed.), *Interaction rhythms: Periodicity in communicative behavior* (pp. 225–248). New York: Human Sciences Press.

Crown, C.L., Feldstein, S., Jasnow, M.D., Beebe, B., & Jaffe, J. (2002). The cross-modal coordination of interpersonal timing: neonatal gaze with adult vocal behavior. *Journal of Psycholinguistic Research*, **31** (1), 1–23.

Dapretto, M., Davies, M.S., Pfeifer, J.H., Scott, A.A., Sigman, M., Bookheimer, S.Y., & Iacoboni, M. (2005). Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, **9**, 28–30.

Davis, M.H. (1980). A multidimensional approach to individual differences in empathy. *JSAS Catalog of Selected Documents in Psychology*, **10**, 85.

Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: social orienting, joint attention, and attention to distress. *Developmental Psychology*, **40**, 271–283.

Decety, J., & Chaminade, T. (2003). When the self represents the other: a new cognitive neuroscience view on psychological identification. *Consciousness and Cognition*, **12**, 577–596.

Dimberg, U. (1982). Facial reactions to facial expressions. *Psychophysiology*, **19**, 643–647.

Dimberg, U., & Lundquist, O. (1988). Facial reactions to facial expressions: sex differences. *Psychophysiology*, **25**, 442–443.

Dimberg, U., Thunberg, M., & Elmehed, K. (2000). Unconscious facial reactions to emotional facial expressions. *Psychological Science*, **11**, 86–89.

Dimberg, U., Thunberg, M., & Grunedal, S. (2002). Facial reactions to emotional stimuli: automatically controlled emotional responses. *Cognition and Emotion*, **16**, 449–471.

Ekman, P. (2004). *Emotions revealed: Recognizing faces and feelings to improve communication and emotional life*. New York: Owl Books.

Ekman, P., & Friesen, W. (1978). *Facial action coding system: A technique for the measurement of facial movement*. Palo Alto, CA: Consulting Psychologists Press.

Feldstein, S. (1982). Impression formation in dyads: the temporal dimension. In M. Davis (Ed.), *Interaction rhythms: Periodicity in communicative behavior* (pp. 207–224). New York: Human Sciences Press.

Humphreys K., Minshew, N., Leonard, G.L., & Behrmann, M. (2007). A fine-grained analysis of facial expression processing in high-functioning adults with autism. *Neuropsychologia*, **45**, 685–695.

Iacoboni, M. (2005). Understanding others: imitation, language, empathy. In S. Hurley & N. Chater (Eds.), *Perspectives on imitation: From cognitive neuroscience to social science* (pp. 77–99). Cambridge, MA: MIT Press.

Iacoboni, M., Molnar-Szakacs, I., Gallese, V., Buccino, G., Mazziotta, J.C., & Rizzolatti, G. (2005). Grasping the intentions of others with one's own mirror neuron system. *Public Library of Science: Biology*, **3**, 1–7.

Ito, M. (2002). Historical review of the significance of the cerebellum and the role of Purkinje cells in motor learning. *Annals of the New York Academy of Sciences*, **978**, 273–288.

Ivry, R.B. (1996). The representation of temporal information in perception and motor control. *Current Opinions in Neurobiology*, **6**, 851–857.

Ivry, R.B., & Keele, S.W. (1989). Timing functions of the cerebellum. *Journal of Cognitive Neuroscience*, **1**, 136–152.

Ivry, R.B., Spencer, R.M.C., Zelaznik, H.N., & Diedrichsen, J. (2002). The cerebellum and event timing. *Annals of the New York Academy of Sciences*, **978**, 302–317.

James, W. (1890). *The principles of psychology*. New York: Holt.

Jasnow, M.D., Crown, C.L., Feldstein, S., Taylor, L., Beebe, B., & Jaffe, J. (1988). Coordinated interpersonal timing of Down syndrome and nondelayed infants with their mothers: evidence for a buffered mechanism of social interaction. *Biological Bulletin*, **175**, 355–360.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, **2**, 217–250.

Kendon, A. (1970). Movement coordination in social interaction: some examples described. *Acta Psychologica*, **32**, 101–125.

Leslie, K., Johnson-Frey, S.H., & Grafton, S.T. (2004). Functional imaging of face and hand imitation: toward a motor theory of empathy. *NeuroImage*, **21**, 601–607.

Lipps, T. (1907). Das wissen von fremden Ichen. In T. Lipps (Ed.), *Psychologische untersuchungen*, **1**, 694–722. Leipzig, Germany: Engelmann.

Lord, C., Risi, S., Lambrecht, L., Cook, E.H., Leventhal, B.L., & DiLavore, P.C. (2000). Autism Diagnostic Observation Schedule-Generic (ADOS-G). *Journal of Autism and Developmental Disorders*, **30**, 205–223.

Lundqvist, L., & Dimberg, U. (1995). Facial expressions are contagious. *Journal of Psychophysiology*, **9**, 203–211.

McFarland, D.H. (2001). Respiratory markers of conversational interaction. *Journal of Speech, Language, and Hearing Research*, **44**, 128–143.

McIntosh, D.N., Reichmann-Decker, A., Winkielman, P., & Wilbarger, J.L. (2006). When the social mirror breaks: deficits in automatic, but not voluntary, mimicry of emotional facial expressions in autism. *Developmental Science*, **9**, 295–302.

McPartland, J., Dawson, G., Webb, S.J., Panagiotides, H., & Carver, L.J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, **45**, 1235–1245.

Magnee, M.J.C.M., de Gelder, B., van Engeland, H., & Kemner C. (2007). Facial electromyographic responses to emotional information from faces and voices in individuals with pervasive developmental disorder. *Journal of Child Psychology and Psychiatry*, **48**, 1122–1130.

Mehrabian, A. (1996). Manual for the Balanced Emotional Empathy Scale (BEES). (Available from Albert Mehrabian, 1130 Alta Mesa Road, Monterey, CA 93940.)

Meltzoff, A.N., & Moore, M.K. (1997). Explaining facial imitation: a theoretical model. *Early Development and Parenting*, **6**, 179–192.

Morecraft, R.J., Stilwell-Morecraft, K.S., & Rossing, W.R. (2004). The motor cortex and facial expression: new insights from neuroscience. *The Neurologist*, **10**, 235–249.

Natale, M. (1978). Perceived empathy, warmth, and genuineness, as effected by interviewer timing of speech in a telephone interview. *Psychotherapy: Theory, Research and Practice*, **12**, 145–152.

Neumann, R., & Strack, F. (2000). 'Mood contagion': the automatic transfer of mood between persons. *Journal of Personality and Social Psychology*, **79**, 211–223.

Niedenthal, P.M. (2007). Embodying emotion. *Science*, **316**, 1002–1005.

Nishitani, N., Avikainen, S., & Hari, R. (2004). Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Annals of Neurology*, **55**, 558–562.

Oberman, L.M., Hubbard, E.M., McCleery, J.P., Altschuler, E.L., Ramachandran, V.S., & Pineda, J.A. (2005). EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Cognitive Brain Research*, **24**, 190–198.

Oberman, L.M., & Ramachandran, V.S. (2007). The simulating social mind: the role of simulation in the social and communicative deficits of autism spectrum disorders. *Psychological Bulletin*, **133**, 310–327.

Oberman, L.M., Ramachandran, V.S., & Pineda, J.A. (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.

Oberman, L., Winkielman, P., & Ramachandran, V.S. (2007). Face to face: blocking facial mimicry can selectively impair recognition of emotional expressions. *Social Neuroscience*, **2**, 167–178.

Paccalin, C., & Jeannerod, M. (2000). Changes in breathing during observation of effortful actions. *Brain Research*, **862**, 194–200.

Rogers, S.J., & Williams, J.H.G. (2006). *Imitation and the social mind: Autism and typical development*. New York: Guilford Press.

Stel, M., van den Heuvel, C., & Smeets, R. (2008). Facial feedback mechanisms in autistic spectrum disorders. *Journal of Autism and Developmental Disorders*. Doi: 10.1007/s10803-007-0505-y. (published 22 February 2008)

Tassinary, L.G., & Cacioppo, J.T. (2000). The skeletomotor system: surface electromyography. In J.T. Cacioppo, L.G. Tassinary, & G.G. Berntson (Eds.), *Handbook of psychophysiology* (2nd edn., pp. 163–199). New York: Cambridge University Press.

Theoret, H., Halligan, E., Kobayashi, M., Fregni, F., Tager-Flusberg, H., & Pascual-Leone, A. (2005). Impaired motor facilitation during action observation in individuals with autism spectrum disorder. *Current Biology*, **15**, R84–R85.

Tomasello, M. (1999). *The cultural origins of human cognition*. Cambridge, MA: Harvard University Press.

Villalobos, M.E., Mizuno, A., Dahl, B.C., Kemmotsu, N., & Muller, R.A. (2005). Reduced functional connectivity

between V1 and inferior frontal cortex associated with visuomotor performance in autism. *NeuroImage*, **25**, 916–925.

Vrana, S.R. (1993). The psychophysiology of disgust: differentiating negative emotional context with facial EMG. *Psychophysiology*, **30**, 279–286.

Wallbott, H.G. (1991). Recognition of emotion from facial expression via imitation? Some indirect evidence for an old theory. *British Journal of Social Psychology*, **30**, 207–219.

Webb, S.J., Dawson, G., Bernier, R., & Panagiotides, H. (2006). ERP evidence of atypical face processing in young children with autism. *Journal of Autism and Developmental Disorders*, **36**, 881–890.

Welkowitz, J., & Feldstein, S. (1970). Relation of experimentally manipulated interpersonal perception and psychological differentiation to be the temporal patterning of conversation. *Proceedings of the 78th Annual Convention of the American Psychological Association*, **5**, 387–388.

Winkielman, P., Niedenthal, P., & Oberman, L. (2008). The embodied emotional mind. In G.R. Semin & E.R. Smith (Eds.), *Embodied grounding: Social, cognitive, affective, and neuroscientific approaches* (pp. 263–288). New York: Cambridge University Press.

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