

Unbroken mirror neurons in autism spectrum disorders

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Background: The ‘broken mirror’ theory of autism, which proposes that a dysfunction of the human mirror neuron system (MNS) is responsible for the core social and cognitive deficits in individuals with autism spectrum disorders (ASD), has received considerable attention despite weak empirical evidence. **Methods:** In this electroencephalographic study, we examined *mu* suppression, as an indicator of sensorimotor resonance, concurrent with oculomotor performance while individuals ($n = 20$) with ASD and control participants ($n = 20$) either executed hand actions or observed hand actions or a moving dot. No difference in visual attention between groups was found as indicated by fixation duration and normalized fixation number on the presented stimuli. **Results:** The *mu* suppression over the sensorimotor cortex was significantly affected by experimental conditions, but not by group membership, nor by the interaction between groups and conditions. Individuals with ASD, similar to the controls, exhibited stronger *mu* suppression when watching hand actions relative to a moving dot. Notably, participants with ASD failed to imitate the observed actions while their *mu* suppression indicating the MNS activity was intact. In addition, the *mu* suppression during the observation of hand actions was positively associated with the communication competence of individuals with ASD. **Conclusion:** Our study clearly challenges the broken mirror theory of autism. The functioning of the mirror neuron system might be preserved in individuals with ASD to a certain degree. Less *mu* suppression to action observation coupled with more communicational severity can reflect the symptom heterogeneity of ASD. Additional research needs to be done, and more caution should be used when reaching out to the media. **Keywords:** Mirror neurons, autism spectrum disorders, *mu* suppression. **Abbreviations:** ASD: autism spectrum disorders.

Individuals with autism spectrum disorders (ASD) have been characterized by difficulties in social interaction, whose cause remains unknown. In recent years, the broken mirror theory of autism has received considerable attention far beyond the scientific community, and this despite little empirical evidence (Southgate & Hamilton, 2008). The discovery of mirror neurons in the monkey brain (ventral premotor area F5 and inferior parietal lobule) (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992) and the so-called mirror neuron system (MNS) in the human brain, a circuit consisting of ventral premotor and posterior parietal cortices (Fadiga, Fogassi, Pavesi, & Rizzolatti, 1995; Rizzolatti et al., 1996) has led to an increased interest in the biological mechanisms that underlie cognitive and social processes. The primary function of mirror neurons was proposed in relation to action understanding (Rizzolatti & Craighero, 2004), but it also has been implicated in various social-cognitive processes, including imitation (Iacoboni et al., 1999), theory of mind (Gallese & Goldman, 1998), language (Rizzolatti & Arbib, 1998), and empathy (Decety & Jackson, 2004; Decety & Meyer, 2008). The physiological

mechanisms of mirroring demonstrated by neuroscience investigations at single-cell and neural-system levels suggest that the MNS might provide an important neural substrate for humans’ ability to imitate (Iacoboni, 2009). Early developmental failure of the MNS has been postulated to contribute to imitation deficits followed by varied social-cognitive difficulties characteristic of ASD (Oberman & Ramachandran, 2007; Williams, Whiten, Suddendorf, & Perrett, 2001). However, the findings from neuroimaging and neurophysiological studies investigating MNS functioning in individuals are inconsistent, particularly in young children (e.g., Katagiri, Inada, & Kamio, 2010). In addition, empirical evidence in support of the implication of the MNS in imitation in humans seems controversial (e.g., Jackson, Meltzoff, & Decety, 2006; Williams et al., 2006). Importantly, one recent quantitative meta-analysis study, using activation likelihood estimation, revealed that the superior and inferior parietal cortex and dorsal premotor cortex, but not the inferior frontal gyrus (considered to be the homologue region of area F5), were the most commonly involved in imitation of hand and finger movements (Molenberghs, Cunnington, & Mattingley, 2009).

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Some studies have reported that activity in the MNS of individuals with ASD differed from those of control participants through indirect measures of neural activation of the MNS, such as reduced muscle activation (Cattaneo et al., 2007), corticospinal excitability (Théoret et al., 2005), resting-state (*mu*) suppression over sensorimotor cortex (Oberman et al., 2005; Oberman, Pineda, & Ramachandran, 2007; Oberman, Ramachandran, & Pineda, 2008), or task-dependent blood-oxygen-level-dependent (BOLD) response (Dapretto et al., 2006). However, reduced *mu* suppression over the sensorimotor cortex in individuals with ASD could be caused by differential processing in the MNS (Oberman et al., 2005), or by differences in early visual processing or attention (Hari & Salmelin, 1997). Reduced attention to social cues (Klin, Jones, Schultz, Volkmar, & Cohen, 2002), reduced processing of biological motion (Blake, Turner, Smoski, Pozdol, & Stone, 2003), atypical sensorimotor resonance in response to the observation of the biological versus non-biological model (Becchio, Pierno, Mari, Lusher, & Castiello, 2007; Pierno, Mari, Lusher, & Castiello, 2008), and differences in understanding complex visual information (Behrmann, Thomas, & Humphreys, 2006) have been documented in individuals with ASD. In contrast, some studies detected a normal MNS response in ASD. For instance, the observation of action without requesting imitation elicited significant *mu* suppression (Bernier, Dawson, Webb, & Murias, 2007), particularly when the action was attributed to a familiar individual (Oberman et al., 2008). Psychological interference from observed actions, suggesting that simulation of hand actions occurs automatically (Bird, Leighton, Press, & Heyes, 2007), and from action imitation (Hamilton, Brindley, & Frith, 2007) seems to be normal in ASD. Altogether, more research is needed to understand whether or to what extent the activity in the putative MNS is indeed atypical in individuals with ASD and the impact of aberrant neurodevelopment in frontal white matter (Cheng et al., 2010).

Previously, our group developed an electroencephalographic (EEG) paradigm to investigate gender differences in the human MNS (Cheng et al., 2008; Cheng, Tzeng, Decety, Imada, & Hsieh, 2006), which showed that female participants appear to have a stronger action/perception coupling than male participants. The *mu* suppression is considered to be a reliable indicator for the action/perception coupling activity (Muthukumaraswamy, Johnson, & McNair, 2004; Pineda, 2005), which has been considered as a neural-specific index of the MNS in adults, teens (Bernier et al., 2007; Cheng et al., 2008; Oberman et al., 2005, 2007, 2008), and children (Lepage & Théoret, 2006). The present study investigates changes in the *mu* rhythm with EEG recordings in 20 male participants with ASD and 20 age-, gender-, and full-scale IQ-matched typically developing control males while they executed hand

actions, or watched either dynamic visual stimuli depicting hand actions or a moving dot. The comparison of hand actions vs. a moving dot may help clarify if there is differential *mu* suppression in response to a biological vs. non-biological model in ASD. Eye-tracking recording was used to ensure that participants attended to the visual stimuli. Driven by the so-called broken mirror theory of autism (Oberman & Ramachandran, 2007; Williams et al., 2001), we predicted that participants with ASD as compared to the typically developing control participants would fail to modulate *mu* suppression when watching hand actions if there is no bias due to attention. On the contrary, if the attention bias is present, the failure of *mu* suppression in ASD cannot be exclusively explained by an MNS dysfunction. Further, if participants with ASD fail to imitate the observed hand actions while their *mu* suppression indicates a normal MNS activity, it will provide an argument against the broken mirror hypothesis of autism.

Methods

Participants

Twenty male individuals with ASD and 20 male controls were recruited. Participant characteristics are listed in Table 1. Individuals with a co-morbid psychiatric or medical condition (e.g., epilepsy), history of head injury or any genetic disorders associated with autism (e.g., fragile X syndrome) were excluded. Participants with ASD were aged between 11 and 26 years, non-medicated, and were recruited from a community autism program. At the time of EEG recording, these ASD individuals had the following diagnoses: 7 autism, 8 Asperger's disorder, and 5 pervasive developmental disorders not otherwise specified, confirmed by experienced clinicians' evaluations using DSM-IV criteria as well as the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & Le Couteur, 1994) translated into Chinese in-house. The administrators of the ADI-R were appropriately trained for research reliability.

Table 1 Demographic and clinical variables of study participants

Variable and group	Mean (SD)	Statistic	P Value
Age (years)			
ASD (<i>n</i> = 20)	17.73 (4.48)	<i>t</i> (38) = .67	.51
Control (<i>n</i> = 20)	17.50 (4.67)		
Full scale IQ			
ASD (<i>n</i> = 20)	110.62 (18.72)	<i>t</i> (38) = .47	.90
Control (<i>n</i> = 20)	107.25 (14.91)		
Autism Diagnostic Interview-Revised (ADI-R)			
Communication	15.5 (2.3)		
Social	19.5 (7.2)		
Repetitive behaviors	4.9 (2.3)		

Typically developing control individuals were aged between 10 and 26 years, and were recruited from local schools and screened for major psychiatric illness using a structured parental interview. All subjects had their IQ assessed with the Wechsler Intelligence Scale (WISC-III). Every participant and his parents gave informed consent for the study, whose protocol was approved by local Ethics Committee (National Yang-Ming University, Taipei, Taiwan).

Dynamic visual stimuli

Participants were shown a series of black and white video clips at a viewing distance of 96 cm with visual angle ($2^\circ\sim 5^\circ$). One of the clips depicted a right hand manipulating a white chessman from the palm to the fingertips at a rate of around 1 Hz. The displayed hand's sex was androgynous, and was of median gray color (8.6 cd/m^2) against a black background (3.7 cd/m^2). The other clip depicted a light gray dot (33.0 cd/m^2) moving randomly on a black background (1.0 cd/m^2) with the same visual angle, median grayness, and moving rate as the hand actions. The duration of each video was 80 seconds.

General procedures

EEG recordings were conducted under four conditions: 1) watching a static cross on a full screen with visual angle ($2^\circ\sim 5^\circ$) and mean luminance 3.7 cd/m^2 , which was presented as a baseline condition (*Baseline*); 2) watching a video of a manipulating hand (*Hand*); 3) watching a video of a moving white dot (*Dot*); and 4) manipulating a white chessman from right hand palm to fingertips at a rate of approximately 1 Hz (*Execution*) while subjects watched their hand at a comfortable viewing distance and held the hand at eye level. In the *Execution* condition, participants were verbally instructed to manipulate the chessman in the same manner as shown in the *Hand* condition. All conditions were presented once. The order of the conditions was counterbalanced across subjects. In addition, participants' performance during the *Execution* condition was videotaped for the later scoring on their imitation competence, in which the number of errors, i.e., additional or incorrect movements, were coded by trained graduate students who were blind to the diagnostic status of the participants (Bernier et al., 2007).

Oculomotor measures

In order to make sure that participants attended to the stimuli presentation, a Tobii X120 binocular eye tracker was used to obtain oculomotor simultaneously with EEG recordings. The tracking rate was 120 Hz. The Tobii eye-tracker is a bright-pupil eye tracker that uses a camera with a high resolution and large field of view to capture images of the sub-

ject's eyes. The tracker illuminates the participant's eyes with two near infrared diodes to generate reflection patterns on the corneas of the user. A video camera gathers the reflection patterns as well as the position of the subject relative to the screen. Digital image processing is carried out for extracting the pupils from the video signal. The pupil locations can be mapped to gaze locations on the screen by a 5-point calibrating system. We recorded fixation duration and number with fixation defined as a gaze of at least 100 ms duration.

EEG data acquisition

EEG data were collected from 40 electrodes embedded in a whole-head cap using the international 10–20 method of electrode placement. Disc electrodes used as bipolar horizontal and vertical electro-oculograms (EOG) were applied to the face area right above and below the eye, and behind each ear (mastoids, A1+ A2 as reference electrodes). Following placements of the cap, electrolytic gel was applied at each electrode site to reduce the impedance of the electrode–skin contact. The impedance on all electrodes was measured and confirmed to be less than $5\text{ K}\Omega$ both before and after testing. Once the electrodes were in place, the position of the electrodes was identified with a three-dimensional digitizer with respect to three predetermined landmarks (nasion and bilateral preauricular points) for the source localization processing. Subjects were seated inside an acoustically and electromagnetically shielded testing chamber.

EEG was recorded and analyzed using a Neuroscan Synamps system (Nu amplifier; Neuroscan, Compumedics Ltd, Melbourne, Australia) with bandpass .1–30 Hz. Data were collected for approximately 80 s per condition at a sampling rate of 500 Hz. Since the *mu* (8–13) rhythm overlapping with the posterior alpha band may have been affected by states of expectancy and awareness, the first and last 10 s of each block of data were removed from all subjects to eliminate the possibility of attention transients due to onset and termination of the stimulus. A 1-min segment of data following the initial 10-s was obtained. Eye blink and eye movements were manually identified by the EOG recordings. Segments with movement or eye blink artifacts were identified and removed prior to analysis through visual inspection as well as through an automated artifact detection algorithm, in which EOG amplitudes deviating more than $100\text{ }\mu\text{V}$ were rejected. The trial rejection rate was 8.92% on average across both groups. There were no differences between groups in rate of trial rejection (ASD: 8.95%; control: 8.89%).

For control purposes, the surface electromyograms (EMG) were recorded from the right first interosseus and thenar muscles. EMGs were high-pass filtered at 3 Hz and rectified. The background

EMG levels were compared across conditions. The root-mean-square levels of surface EMG were computed by taking the medians of the ten 2-sec segments for each of the observed conditions across groups (ASD vs. control). The *Baseline* EMG level did not differ from those during each observed condition.

EEG data analysis

Data were analyzed after removing any movement or eye blink artifacts. Using a Fast Fourier Transform (FFT), the integrated power in the 8–13 Hz range was computed for each clean segment. Data were segmented into epochs of 2 s beginning at the start of the segment. FFT were performed on the epoched data, which constituted a total of 512 points. A cosine window was used to control for artifacts resulting from data splicing.

The *mu* rhythm was measured as the ratio of the power during each condition (*Hand* vs. *Dot* vs. *Execution*) relative to the power during the *Baseline*. The *Baseline* correction was used to control for variability in absolute *mu* power as a result of individual differences, e.g., scalp thickness and electrode impedance, as opposed to mirror neuron activity. A log transformation was also calculated for each ratio to correct for the inherent non-normality of ratio data as a result of lower bounding. The log transformation (the logarithm to the base 2 of *mu* power data) results in a negative value representing *mu* rhythm suppression whereas a positive value indicates *mu* rhythm augmentation. Although data were obtained from all electrodes across the scalp, *mu* rhythm is defined as the mean *mu* power measured over the sensorimotor cortex (C3, Cz, and C4).

A one-way analysis of variance (ANOVA) was first conducted for the comparison between the ASD vs. control groups on the *Baseline*. For the comparison of the conditions, a two-way factorial mixed ANOVA was used [group (ASD vs. control) \times condition (*Execution* vs. *Hand* vs. *Dot*)] followed with *Bonferroni post hoc* tests, using the log ratio of the *mu* rhythm as a dependent variable. To elucidate if the *mu* suppression had developmental change, correlation analysis was performed for each participant's age with his *mu* suppression at each condition within each group, separately. In order to test if the *mu* suppressions could be related to ASD symptoms, correlation analysis was computed within the ASD group for each *mu* suppression value at each condition with each participant's communication, social, and repetitive behavior subscale ratings of ADI-R as dependent variables.

For source estimation of *mu* rhythm, the electrodes in the vicinity of left and right sensorimotor cortex were first selected for the regions of interest (ROIs). Then left and right ROIs were separately estimated with the use of equivalent current dipole (ECD, Curry V5.0, Compumedics Ltd., Melbourne, Australia). A

single dipole model was applied to explain the recorded EEG *mu* rhythm signals on the basis of a realistic head model (boundary element model, BEM). Finally, the electric dipoles estimated from left and right sensorimotor ROIs were localized and centered along the band of the central sulcus.

Results

Neither the electrode effect (C3 vs. Cz vs. C4: $F_{2, 76} = 3.10$, $P = .072$) nor the interaction of electrode by group ($F_{2, 76} = 1.09$, $P = .340$) reached significance. We thus calculated the mean *mu* power measured over central area (C3, Cz, and C4) to represent *mu* suppression. Results indicate that the *mu* suppression was significantly modulated by experimental conditions ($F_{2,76} = 10.812$, $P < .001$), but not by group membership ($F_{1,38} = .044$, $P = .835$). No significant interaction between groups and conditions ($F_{2,76} = 2.437$, $P = .094$) was found (Figure 1 and Supplementary Materials). *Bonferroni post hoc* tests further demonstrated that the significant effect of the condition was mainly driven by the differential *mu* suppression between the *Dot* and the other conditions (*Dot* vs. *Execution*, $P = .001$; *Dot* vs. *Hand*, $P = .009$; *Hand* vs. *Execution*, $P = .154$). Further, separate analysis for each group showed that the action observation condition was associated with significant *mu* suppression that was no more than the action execution, as compared to the observation of a moving dot (ASD: *Dot* vs. *Execution*, $P = .042$; *Dot* vs. *Hand*, $P = .035$; *Hand* vs. *Execution*, $P = .951$; Control: *Dot* vs. *Execution*: $P < .001$; *Dot* vs. *Hand*, $P = .037$; *Hand* vs. *Execution*, $P = .007$). Analysis at each condition also showed no differences between the two groups (*Execution*: $P = .455$; *Hand*: $P = .503$;

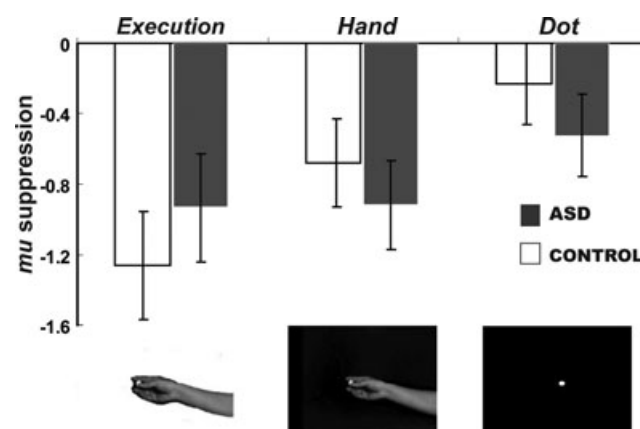


Figure 1 *mu* suppression during action execution (*Execution*), action observation (*Hand*) and dot observation (*Dot*) in individuals with ASD and controls. The *mu* suppression was significantly affected by experimental conditions (*Execution* vs. *Hand* vs. *Dot*) ($P < .001$), but not by group membership (ASD vs. control) ($P = .835$), nor by the interaction between groups and conditions ($P = .094$)

Dot: $P = .378$). This suggests that the paradigm is reliable enough to demonstrate activity of the human MNS.

To ensure that the effects of suppression were specific to the μ rhythm and not resulting from any other activity, we calculated μ power over electrodes from other scalp regions (frontal: Fp1, Fp2, and Fz; occipital: O1, O2, and Oz). Across the conditions, no consistent pattern of suppression was detected in the frequency band under investigation of these electrodes. This indicates that the observed suppression was specific to the central electrodes (C3, Cz, and C4), and did not result from other activities, such as alpha desynchronization.

The correlation analysis between age and μ suppression showed that, in participants with ASD, the μ suppression was not related to age in aspects of *Execution*, *Hand*, and *Dot* ($r = .40$, $P = .092$; $r = .14$, $P = .582$; $r = .06$, $P = .819$). In control participants, there was no correlation between μ suppression and age, respectively ($r = .40$, $P = .079$; $r = -.23$, $P = .323$; $r = .16$, $P = .500$). This indicates that neither the ASD nor the control participants have developmental change in μ suppression with age, ranging from adolescence to adulthood here (10–26 years old).

Importantly, the activity of the MNS in individuals with ASD was preserved, as shown by the presence of μ suppression during action observation. Specifically, in individuals with ASD, there was a significant positive correlation between the μ suppression during action observation and the communication subscale of the ADI-R ($r = .49$, $P = .034$) after an outlier was removed (Figure 2). No such correlation existed in the social ($r = .24$, $P = .331$) and repetitive behavior ($r = -.045$, $P = .854$). The preservation of the MNS activity was associated with the symptom severity of ASD. More communicational impairments were tightly coupled with less μ suppression during action observation. It is suggested that the dysfunction of the MNS could play a role in the heterogeneity observed in ASD.

Moreover, according to the video recording, the number of errors during the *Execution* condition was significantly greater in the ASD than in the control group (22.1 ± 11.3 vs. 12.8 ± 4.8). For individuals with ASD, the μ suppression in each condition was not correlated with the number of errors (*Execution* vs. *Hand* vs. *Dot*: $r = .15$, $P = .633$; $r = .11$, $P = .559$; $r = .18$, $P = .618$). This suggests that participants with ASD failed to imitate the observed actions while their μ suppression indicates a normal MNS activity.

Analyses of oculomotor performance during the three experimental conditions, in aspects of the fixation duration (ms) and the normalized fixation duration (%), showed that neither the groups (ASD vs. control: $F_{1, 38} = .518$, $P = .476$; $F_{1, 38} = 3.082$, $P = .088$), the conditions (*Baseline* vs. *Hand* vs. *Dot*: $F_{2, 76} = 2.913$, $P = .067$; *Hand* vs. *Dot*: $F_{1, 38} = .865$, $P = .359$), nor their interactions ($F_{2, 56} = 1.468$, $P = .243$; $F_{1, 38} = .045$, $P = .833$) reached significant differences. Besides, in the normalized fixation number, the ASD group appeared similar to the controls ($F_{1, 38} = .290$, $P = .594$) (Supplementary Materials). Further, neither the normalized fixation duration nor the fixation number was correlated with the μ suppression elicited by action observation ($r = .14$, $P = .430$; $r = .061$, $P = .725$). Thus, we can infer that the μ suppression between the groups was not biased by differential attention processing to the visual stimuli.

Discussion

Here, we demonstrated that the EEG μ suppression associated with action imitation (*Execution*) and action observation (*Hand*) did not significantly differ between individuals with ASD and control participants while controlling for visual attention by using eye-tracking recording to ensure similar oculomotor performance between groups. Importantly, the finding that participants with ASD failed to imitate the

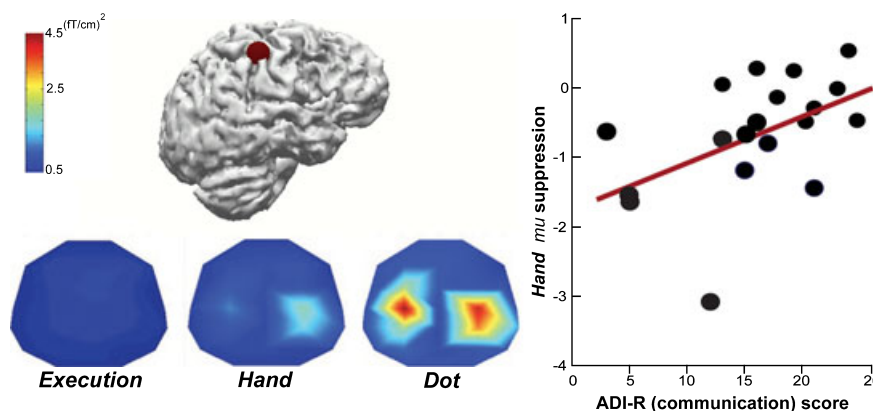


Figure 2 Correlation between μ suppression and symptom severity in individuals with ASD. The μ suppression, originating from the sensorimotor cortex, during the observation of hand actions (*Hand*) is associated with the symptom severity in the communication subscale of ADI-R ($r = .49$, $P = .034$)

observed actions (*Execution*) while their *mu* suppression was intact is a strong argument against the broken mirror theory of autism. Further, no age-related change between childhood and adulthood was found. Less *mu* suppression to action observation coupled with more communication severity may suggest that the MNS activity can reflect the symptom heterogeneity of ASD.

The discrepancy between these results and previous reports (Oberman et al., 2005, 2007) in aspect of action observation may reflect imitation performance in ASD. Children with ASD often fail to imitate actions (Rogers, Bennetto, McEvoy, & Pennington, 1996; Stone, Ousley, & Littleford, 1997; Williams, Whiten, & Singh, 2004), including those that involve an element of perspective taking (Ohta, 1987; Smith & Bryson, 2007). However, there are several recent studies that challenged these findings and demonstrated intact imitation in ASD, which are not easily accommodated by the MNS account. For example, individuals with ASD show an enhanced automatic imitation effect (Bird et al., 2007) and normal interference effects when observing an incompatible action (Gowen, Stanley, & Miall, 2008). Children with ASD can perform a variety of imitation tasks correctly when they are explicitly instructed to imitate (Dapretto et al., 2006; Hamilton et al., 2007; Southgate & Hamilton, 2008). Neural responses of the MNS to the observation of hand movements (closing and opening the right hand, intransitive movement) were reduced in children with autism (Oberman et al., 2005, 2007) whereas responses to the observation of hand actions (object-directed, transitive action) were normal in adults with Asperger's disorder (Avikainen, Kulomaki, & Hari, 1999) and high-functioning ASD (Bernier et al., 2007). Here, we found that individuals with ASD had preserved *mu* suppression when watching visual stimuli depicting a right hand manipulating an object (transitive action). Together, results from these previous studies and our new findings provide supportive evidence for the distinction between abnormal representations of intransitive movements and normal representations of transitive actions in ASD (Avikainen et al., 1999; Bernier et al., 2007; Oberman et al., 2005, 2007).

Furthermore, while requested to imitate the observed hand actions (*Execution*), participants with ASD showed worse performance than the controls, as indicated by more errors, in spite of normal *mu* suppression. The study of Bernier and his co-workers (2007) also reported that adults with ASD are poorer in a mature imitation task even though *mu* suppression in response to the observation and the imitation of transitive actions (by log ratio) did not differ between groups (ASD vs. control). They demonstrated that *mu* attenuation (by subtraction from the baseline) was correlated with imitation competence. However, we found no correlation between *mu* suppression (by log ratio) and imitation perfor-

mance. To our knowledge, the present findings may lead to strong arguments against the broken mirror theory of autism.

The degree to which activity in the MNS is preserved in individuals with ASD may depend upon symptom severity. For instance, the BOLD response in the right inferior frontal gyrus was inversely correlated with the social reciprocal impairments (Dapretto et al., 2006). Cortical thinning of Broca's area, a core region of the human MNS, was associated with more severity of the combined social and communication diagnostic algorithm (Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006). Larger gray matter volume in the right inferior frontal gyrus was related to less symptom severity in children with ASD (Parks et al., 2009). In addition, the MNS activity as shown by suppression of *mu* rhythm appears normal in adults with Asperger's disorder (Avikainen et al., 1999) and high-functioning ASD (Bernier et al., 2007). Here, while enrolling more ASD participants with a larger sample size compared to previous *mu* rhythm studies, we demonstrated that less *mu* suppression to action observation was associated with more communication severity. Besides, *mu* suppression seems to be sensitive to degree of familiarity. In addition, the MNS in people with ASD can respond to the observed actions when individuals can identify in some personal way with the stimuli (Oberman et al., 2008). Our results demonstrate that MNS functioning is relatively well preserved in individuals with ASD without any identification with the stimuli. Importantly, stronger *mu* suppression to action observation coupled with less communication deficit may implicate the symptom diversity of ASD.

Conclusion

Overall, our study clearly indicates that MNS functioning is preserved to a certain degree in individuals with ASD. When participants with ASD watched a right hand manipulating objects, significant suppression of the *mu* rhythm was detected, and this response was similar to that of control participants. In addition, an inverse relationship between the amount of *mu* suppression and the severity of communication deficits to ASD was found. Our results add to the recent controversy as to whether individuals with ASD have an abnormal *mu* suppression to the observation of hand movement (Oberman et al., 2005), or retain their imitation ability to hand-object interaction (Hamilton et al., 2007). Better neurocognitive models of social behavior within and beyond the broken mirror account are required to understand the causes of social communication deficits in ASD. Finally, our study shows that eye-tracking data are critical when conducting empirical research with people with ASD.

Supplementary material

The following supplementary material is available for this article:

Table S1 *mu* rhythm;

Table S2 Oculomotor performance;

Table S3 *Mu* suppression of each clinical subgroup in ASD;

Table S4 *Mu* suppression among clinical subgroups (Autism vs. Asperger's disorder vs. PDD NOS) and across conditions (*Execution* vs. *Hand* vs. *Dot*) (Word document)

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Key points

- The EEG *mu* (8–13 Hz) suppression during action observation is considered to reflect downstream modulation of primary sensorimotor areas by mirror neuron activity.
- Some studies have proposed that such a modulation is impaired in individuals with ASD.
- However, eye-tracking is rarely simultaneously recorded during observation tasks to ensure that differences in *mu* suppression are not due to attention deficits.
- Our results challenge the broken mirror theory of autism of some previous studies with limited numbers of participants, and clearly demonstrate that the functioning of the human mirror neuron system is preserved to a certain degree in people with ASD.
- EEG *mu* suppression cannot be a diagnostic marker, but rather may reflect symptom severity of ASD.

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