

Developmental changes in mu suppression to observed and executed actions in autism spectrum disorders

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There has been debate over whether disruptions in the mirror neuron system (MNS) play a key role in the core social deficits observed in autism spectrum disorders (ASD). EEG mu suppression during the observation of biological actions is believed to reflect MNS functioning, but understanding of the developmental progression of the MNS and EEG mu rhythm in both typical and atypical development is lacking. To provide a more thorough and direct exploration of the development of mu suppression in individuals with ASD, a sample of 66 individuals with ASD and 51 typically developing individuals of 6–17 years old were pooled from four previously published studies employing similar EEG methodology. We found a significant correlation between age and mu suppression in response to the observation of actions, both for individuals with ASD and typical individuals. This relationship was not seen during the execution of actions. Additionally, the strength of the correlation during the observation of actions did not significantly differ between groups. The results provide evidence against the argument that mirror neuron dysfunction improves with age in individuals with ASD and suggest, instead, that a diagnosis-independent developmental change may be at the root of the correlation of age and mu suppression.

Keywords: autism; mu suppression; mirror neuron system; development

INTRODUCTION

Autism Spectrum Disorders (ASDs) are behaviorally defined disorders affecting an estimated 1 in 110 individuals (Baird *et al.*, 2006; Baron-Cohen *et al.*, 2009), and characterized by qualitative impairments in language and social skills as well as the presence of restricted, repetitive and stereotyped patterns of behaviors, interests and activities. Converging methodologies have suggested that a specific neural system, the mirror neuron system, may play a key proximal role in these core behavioral deficits (Williams *et al.*, 2001; Oberman and Ramachandran, 2007; Perkins *et al.*, 2010).

Mirror neurons, first discovered in the macaque, are unique in that they respond to both the observation and execution of actions (di Pellegrino *et al.*, 1992). The existence of an analogous system in humans has been supported by population-level measures, including transcranial magnetic stimulation (TMS; Fadiga *et al.*, 1995), positron emission tomography (PET; Parsons *et al.*, 1995), electroencephalography (EEG; Altschuler *et al.*, 1997) and functional magnetic resonance imaging (fMRI; Iacoboni *et al.*, 1999).

Even prior to the discovery of mirror neurons, Gastaut and Bert (1954) reported that the so-called rolandic *en arceau rhythm* (now more commonly referred to as the mu rhythm) was reduced when stationary subjects identified themselves with an active person represented on a screen. Despite having relatively poor spatial resolution, the functional similarities between EEG mu rhythm and the mirror neuron system has led several researchers to suggest that suppression of the mu rhythm can be considered an index of MNS functioning (Cochin *et al.*, 1998, 1999, 2001; Martineau and Cochin, 2003; Muthukumaraswamy and Johnson, 2004; Muthukumaraswamy *et al.*,

2004; Pineda, 2005; Perry and Bentin, 2009). Consistent with this, Keuken *et al.* (2011) recently showed that using TMS to disrupt activity in the inferior frontal gyrus (the primary locus of mirror neurons in the frontal cortex) directly affects the modulation of mu rhythms over sensorimotor cortex.

In addition to action observation and production, the MNS has been implicated in higher level cognitive processes that are known to be impacted in ASD, including imitation, language, theory of mind and empathy leading many to suggest that individuals with ASD may have abnormalities in the functioning of the MNS (Williams *et al.*, 2001; Oberman and Ramachandran, 2007; Perkins *et al.*, 2010). Consistent with this proposal, evidence from magnetoencephalography (MEG) (Nishitani *et al.*, 2004), TMS (Théoret *et al.*, 2005), fMRI (Dapretto *et al.*, 2006; Hadjikhani *et al.*, 2006; Williams *et al.*, 2006; Martineau *et al.*, 2010) and EEG (Altschuler *et al.*, 2000; Oberman *et al.*, 2005; Bernier *et al.*, 2007; Martineau *et al.*, 2008) have supported abnormalities in the MNS in individuals with ASD.

Despite the aforementioned support, there is much disagreement over the direction of the abnormality in the MNS in ASD with some groups reporting a reduction in activity (Altschuler *et al.*, 2000; Nishitani *et al.*, 2004; Oberman *et al.*, 2005; Dapretto *et al.*, 2006; Hadjikhani *et al.*, 2006; Williams *et al.*, 2006; Bernier *et al.*, 2007; Martineau *et al.*, 2008), while others report an increase in activity (Martineau *et al.*, 2010) and others show a difference in selectivity (Théoret *et al.*, 2005). Still others have reported no apparent abnormality in the system (Avikainen *et al.*, 1999; Oberman *et al.*, 2008; Raymaekers *et al.*, 2009; Dinstein *et al.*, 2010; Fan *et al.*, 2010). Though it is tempting to interpret the absence of significant differences as evidence for normal functioning, it is plausible that abnormalities in this system were not detected in these studies for a number of reasons (population heterogeneity, differences in age, lack of power, particular stimuli used, etc.).

Recently, Raymaekers *et al.* (2009) reported no significant difference in mu suppression between 20 high-functioning children aged 8–13

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years with ASD and 20 matched control participants during observation of actions. Specifically, they highlight that they find a ‘nearly significant’ [$r(20) = -0.44$, $P = 0.05$] correlation with age in the ASD sample, that was not present in the control group. Based on this, these authors suggest that age has an influence on MNS functioning in ASD, with more suppression being linked to increasing age.

A recent fMRI study (Bastiaansen *et al.*, in press) also found evidence for an age effect with reduced activation in the inferior frontal gyrus in an ASD group during early adulthood (ages 18–35 years) followed by a normalization and then enhancement of activation in late adulthood (ages 35–54 years). Results of this study, therefore, suggest that mirror neuron system activity increases with age in ASD, but decreases with age in neurotypical development. Additionally, the increased activity in inferior frontal gyrus in the ASD group correlated with changes in gaze behavior and improved social functioning. Together with the Raymaekers *et al.* (2009) study finding a marginally significant correlation with age in childhood, these studies provide a potential mechanism for the observed amelioration in social functioning documented in adolescence and adulthood.

Like most findings, however, there have been conflicting reports finding no correlation between age and mirror neuron functioning in ASD. Of note, Oberman *et al.* (2005) who used almost identical methodology to the Raymaekers *et al.* (2009) study found no correlation between age and mu suppression in either the ASD or control group (control group: $r = -0.08$ and ASD group: $r = -0.05$). However, the sample in the Oberman *et al.* (2005) study included adults and a smaller sample than the Raymaekers *et al.* (2009) study. Additionally, a recent study (Enticott *et al.*, in press) using transcranial magnetic stimulation (TMS) also found no correlation between putative measures of mirror neuron functioning and ASD in their relatively large ($n = 34$) sample of adults with ASD.

Given the discrepant findings in the literature and the importance both clinically and theoretically to understand factors mediating the functioning or dysfunction of the mirror neuron system in ASD, we conducted a direct exploration of the development of mu rhythm suppression with age in individuals with ASD in ages 6–17 years by pooling data across four previously published studies (Oberman *et al.*, 2005, 2008; Pineda *et al.*, 2008; Raymaekers *et al.*, 2009). Each of the studies included in our analysis used the same methodology. Specifically, each study included a biological action observation and a non-biological control or resting baseline condition. Each study (i) defined mu power as power in the 8–13 Hz band, (ii) recorded EEG power over central electrodes (C3, Cz and C4), (iii) recorded EEG data across the scalp to ensure that the results are specific to rolandic mu rhythm and not generalized to alpha power fluctuations from other scalp locations and (iv) included both typically developing control participants and those with ASD. Finally, (v) the authors of each study analyzed their data using highly similar methodologies to compute power and suppression in the mu band.

EXPERIMENTAL PROCEDURE

Participants

Data from a total of 66 individuals with ASD and 51 typically developing individuals were included in the current analysis. The participants in the ASD group ranged in age from 6 to 17 years ($M = 10.28$, $s.d. = 2.44$). The typically developing participants were age matched and ranged in age from 6 to 17 years ($M = 10.54$, $s.d. = 2.22$). All participants with ASD were considered high functioning and had a diagnosis of ASD ($n = 20$), Autistic Disorder ($n = 17$), Aspergers Disorder ($n = 26$) or Pervasive Developmental Disorder–Not Otherwise Specified ($n = 3$) based on DSM-IV-TR criteria and

confirmed with an independent administration of either the Autism Diagnostic Observation Schedule (ADOS; Lord *et al.*, 2000) or the Autism Diagnostic Interview-Revised (ADI-R; Lord *et al.*, 1994) in 58 participants and by independent clinical evaluation in 8 participants (Oberman *et al.*, 2005).

Procedure

Data were obtained with permission from the corresponding authors of four published data sets (Oberman *et al.*, 2005, 2008; Pineda *et al.*, 2008; Raymaekers *et al.*, 2009) (see Table 1 for demographic information of participants in the original studies). Although the exact stimuli and display parameters differed across studies, there were sufficient methodological similarities that justified merging the data sets. For detailed methods for each of the studies, see the original published papers.

All of the studies in this analysis collected power in the 8–13 Hz (‘mu’) frequency band sampled over central electrodes (i.e. C3 and C4) during a baseline condition of either rest or the observation of a non-biological motion stimulus that did not itself show any mu suppression compared to rest, and during an observed hand action condition with a stranger. All studies included also examined other frequency bands, across the whole scalp, in order to ensure that the effect was specific to both this frequency and this scalp location. In all studies, the hand action condition was a continuous opening and closing of a stranger’s hand for a period of over 1 min. The EEG response to this continuous movement was then broken into 2-s epochs for analysis. All four studies used similar analysis techniques to derive power in the mu frequency for these conditions. All studies involved required participants in both groups to engage in a continuous performance task, which required the participant to maintain attention to the experimental stimuli. All studies reported nearly perfect performance on this task in both ASD and control groups.

Three of the studies also included an action execution condition, which was also analyzed for this subgroup of studies (ASD: $n = 41$, $M = 10.63$ years, $s.d. = 2.07$ years, range 6–16 years; controls: $n = 40$, $M = 10.47$ years, $s.d. = 1.58$ years, range 6–16 years). Although one of the data sets utilized was collected as part of an intervention study (Pineda *et al.*, 2008), the data used in the current analysis were collected prior to the intervention. Additionally, one of the data sets (Oberman *et al.*, 2008) included other conditions where the hand stimulus was that of a familiar individual. This condition was not included in the analysis; only the condition which included a stranger (stimulus identical to Oberman *et al.*, 2005) was included. Oberman *et al.* (2005) also included two adult participants. However, these data were not included in the analysis as no other study included adult participants.

Mu suppression, as indexed by the ratio of mu power over central electrodes (C3 and C4) during action observation or execution divided by mu power during baseline, was calculated for each individual participant. Values greater than one indicate greater power during the experimental condition compared to baseline and values less than one indicate ‘mu suppression’, that is, less power in the experimental

Table 1 Sample sizes and Age ranges for studies included in this analysis

Study	Number of ASD participants	Number of control participants	Ages (years); M (s.d.)
Oberman <i>et al.</i> (2005)	8	8	6–16; 10.00 (3.70)
Oberman <i>et al.</i> (2008)	13	13	8–12; 10.23 (1.37)
Pineda <i>et al.</i> (2008)	25	11	6–17; 10.04 (3.21)
Raymaekers <i>et al.</i> (2009)	20	19	8–13; 10.94 (1.36)
Total	66	51	6–16; 10.40 (2.34)

condition compared to the baseline condition. Normalizing power in the observation and execution conditions to a baseline condition was done in the original studies to control for any potential differences in overall power across participants due to factors such as differences in skull thickness and electrode impedance. However, this also allowed us to control for differences across studies based on differences in acquisition systems, or other across-study differences. The resulting ratios for the two hemispheres (C3 and C4) were then averaged to create a single mu suppression value for each participant for each condition (observation of actions and execution of actions). A Cook's distance analysis was then performed to identify outliers and one ASD individual's data in the execute condition was identified as an outlier based on having a Cook's Distance (D) value of greater than $4/n$ and removed from analysis.

We pooled the data across these four studies and calculated Pearson correlation coefficients in order to quantify the relationship between mu suppression and age across all subjects as well as for each group individually. Correlation coefficients were also directly compared between the two groups using the calculation for the test of the difference between two independent correlation coefficients (Preacher, 2002) in order to evaluate whether or not the relationship between age and mu suppression differed between the ASD and control groups. This calculation involves converting the two correlation coefficients into a z -scores using Fisher's r -to- z transformation. Then, making use of the sample size employed to obtain each coefficient, these z -scores are compared using formula 2.8.5 from Cohen and Cohen (1983, p. 54).

RESULTS

As ratio data sometimes deviate from normality, the goodness of fit for the normal curve was estimated based on the Kolmogorov–Smirnov test and was deemed to be not significantly different from normal for both the observation values ($P=0.36$) and execution values ($P=0.93$). Thus, parametric tests were used for analysis. There was a significant difference in mu suppression between the ASD ($M=0.94$, $s.d.=0.27$) and the control group ($M=0.84$, $s.d.=0.24$) ($t=2.09$, $P<0.05$) for the observation condition, but no significant difference between groups for the execution condition ($t=0.203$, $P=0.84$). The regression analysis resulted in a significant negative correlation between mu suppression and age during action observation ($R=-0.281$, $P<0.05$), indicating a greater degree of suppression (lower ratio of power during observation or execution with respect to the baseline condition) with increasing age (Figure 1). This relationship was not present during action execution where no significant correlation existed ($R=0.054$, $P=0.63$). When the correlation coefficients for the observation condition were calculated separately for the two groups, both samples reached significance individually (ASD: $R=-0.269$, $P<0.05$ and control: $R=-0.287$, $P<0.05$), while neither reached significance in the execution condition (ASD: $R=0.173$, $P=0.286$ and control: $R=-0.113$, $P=0.49$). Additionally, there was no significant difference in the degree of correlation between the two groups during action observation ($z=0.102$, $P=0.92$). Error variance and the covariance matrices across both groups during observation and execution were examined and no differences were observed (observe condition: Levene's $F=0.62$, $P=0.43$; execute condition: Levene's $F=0.553$, $P=0.46$; Box's $M=6.7$, $P=0.09$), suggesting that this lack of difference between groups is not due to differences in error variance in one group compared with the other.

DISCUSSION

The current study reflects the first large-scale examination of the development of the neural mechanisms that underlie body action

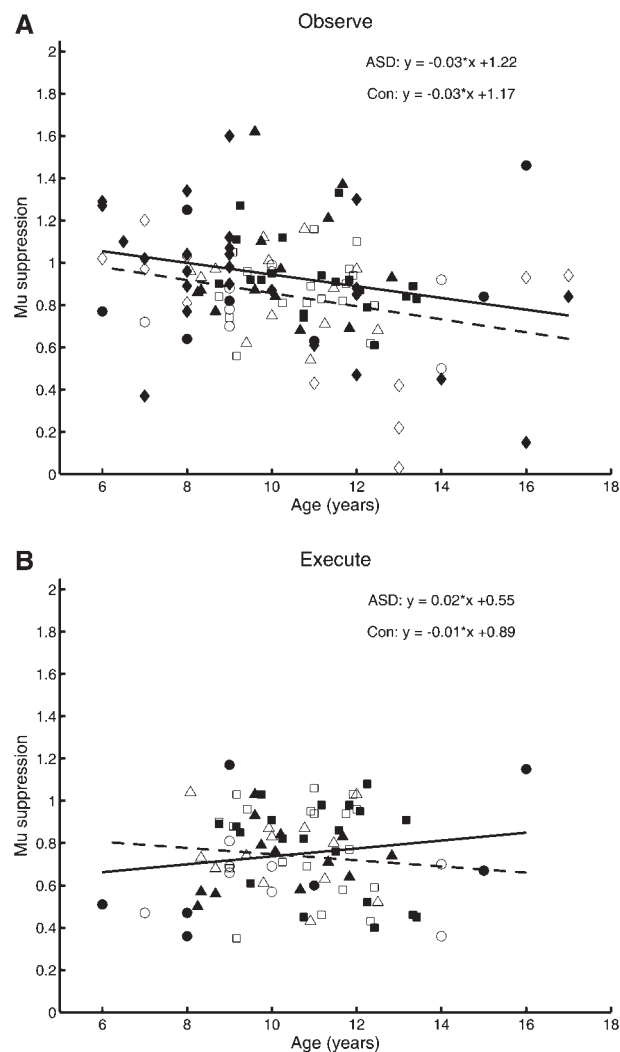


Fig. 1 Correlation between age and mu-suppression for the Observe (A) and Execute (B) conditions. In both graphs, solid symbols represent the ASD participants while open symbols represent control participants. The data from the four studies are differentiated by the shape of the symbols. Data from Raymaekers *et al.* (2009) are represented by squares, Oberman *et al.* (2005) are represented by circles, Oberman *et al.* (2008) are represented by triangles and Pineda *et al.* (2008) are represented by diamonds. The solid regression lines indicate the correlation between age and suppression for individuals with ASD and the dotted line indicates the correlation between age and suppression for control participants.

observation through examination of mu suppression as a putative index for mirror neuron functioning across childhood and adolescence in typically developing individuals and individuals with ASD. The current study pooled data across four published reports resulting in a much larger sample size than any other individual study. Additionally, we directly compared the size of the relationship between age and mu suppression in both ASD and control groups to establish whether this relationship was unique to ASD.

Understanding the developmental course of mu suppression in ASD is critically important. If differences in mu suppression between individuals with ASD and typically developing individuals are only present early in life but people with ASD exhibit pervasive difficulties throughout the lifespan, this would have clear implications for theoretical models of the role of body action processing and MNS dysfunction in the social and communication difficulties experienced by people with ASD. Similarly, if mu suppression normalizes with age, then

this would suggest that any interventions targeting the normalization of the mirror neuron system should be focused on younger individuals.

The results of the current analysis suggest that an increase in mu suppression across the age span is not restricted to individuals with ASD. Therefore, although it is tempting to interpret the finding that mu rhythm during body action observation becomes more suppressed with age reflecting a ‘normalization’ of the MNS over time in ASD, this does not appear to be the case. Instead, the current results quite clearly show that this increased suppression in mu rhythm across the age span is not different between individuals with ASD and controls. Perhaps, the increase in suppression over time in both groups represents a general improvement in the MNS across childhood with greater suppression indicating improved functioning in both groups. Consistent with this, others have reported a general developmental change in mu wave power at central sites. Specifically, mu power appears to increase from childhood to early adolescence (Benninger et al., 1984; Niedermeyer, 1997; Somsen et al., 1997).

Mu frequency may also become more selective for action observation in older childhood and adulthood as compared to younger children. Martineau and Cochin (2003) found that children aged 5–8 years show greater suppression in the theta frequency during action observation as compared to the mu frequency in adulthood. Southgate et al. (2009) similarly found attenuation in a 6- to 9-Hz frequency band in 9-month-old infants. Additionally, Hagne (1968) noted that the resting rhythm associated with motor activity oscillates at ~4–7 Hz in infants increasing to ~7 Hz at 1 year of age, 8 Hz by 18 months and 9 Hz by 4 years. In fact, this effect only appears to stabilize to the standard 8–13 Hz range in mid-adolescence. Interestingly, similar shifts in the specific EEG frequency bands that are sensitive to other types of stimuli or states have also been found to exhibit clear shifts with development (e.g. Marshall et al., 2002). Thus, examining frequencies outside of the 8–13 Hz range (perhaps in the theta range as has been found in younger children) in the youngest of children or defining person specific EEG ranges (as in Muthukumaraswamy and Johnson, 2004) may be a more accurate index.

Though the mu rhythm is thought to originate in primary motor cortex, its suppression could be influenced by activity in primary motor cortex, premotor regions such as IFG or other regions earlier in processing such as the posterior parietal cortex. Thus, it is possible that the greater mu suppression with age reflects a general change either in the extended motor system or in the relationship between motor functioning and mu suppression over the lifespan (as discussed earlier), independent of diagnosis.

In summary, we pooled data across several studies (ASD: $n = 66$, Control: $n = 51$) and conducted the first direct comparison of the developmental trajectory of mu rhythm modulation in individuals with ASD to the developmental trajectory of mu rhythm modulation in controls, in order to explore the specificity of developmental changes to action observation and action execution. We found no evidence for differential changes in mirror neuron functioning with age in ASD compared to a typically developing control group as indicated by nearly parallel regression lines. Our analysis of the relationship of mu rhythm suppression and age in ASD and controls suggests that the observed negative correlation most likely reflects a more generic developmental process in the mu rhythm as opposed to normalization of the mirror neuron system in ASD. Our findings are restricted to developmental changes in childhood and adolescence, however, and cannot therefore speak to the developmental trajectory in adulthood.

Future research should continue to utilize multiple neuroscientific techniques, as brain imaging tools and neurophysiological frequency analysis can provide complementary information. It will be important to continue to explore the neurological underpinnings of ASD, fully taking into account that the behavioral and likely neurological

phenotypes are extremely heterogeneous and, thus, group means may not be representative. It is also critical to explore all potential mediating factors that may influence the degree of dysfunction as it is clear by the discrepancies in literature that not all individuals with ASD will show an impairment in MNS functioning and different paradigms reveal the sensitivity of the system to various experimental manipulations. Finally, careful clinical phenotyping of individuals with ASD and other developmental disorders, including measures of severity in various domains of functioning, will be critically important for quantifying the contribution of various mediating factors in the observed variability of neurological indices, including mirror neuron dysfunction.

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Conflict of Interest

None declared.

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