The role of imitation in the observed heterogeneity in EEG mu rhythm in autism and typical development

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ABSTRACT

Dysfunction in an execution/observation matching system, or mirror neuron system, has been proposed to contribute to the social deficits observed in Autism Spectrum Disorder (ASD). Atypical activity in this system, as reflected in attenuation of the EEG mu rhythm, has been demonstrated in several studies; however, normative patterns of activity have been evident in other ASD samples. The current study sought to investigate this poorly understood heterogeneity in social perceptual brain function in ASD. EEG mu rhythm was recorded in a well-characterized sample of 19 children with ASD (mean age = 6.4; 1 female) and 19 age-matched typically developing peers (mean age = 6.9; 2 females) during execution and observation of goal-directed hand actions. Children were assessed on variables theoretically related to mirror neuron system function (MNS), such as ASD symptoms and imitation ability. Results indicated that MNS activity was associated with facial imitation ability, but not hand imitation ability, in children with ASD and typically developing individuals. Groups were comparable in terms of average MNS activity during both action observation and execution, but, in both groups, a subset of children showed absent or significantly reduced MNS activity during observation of action in conjunction with greater difficulty in imitation. These results emphasize the relationship between EEG indices of MNS function and imitative skill and suggest that dysfunction of the MNS is related to imitation ability in both clinical and typical populations, rather than representing a core deficit or universal impairment in ASD.

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1. Introduction

Autism Spectrum Disorders (ASDs) are defined by social-communication deficits and repetitive and restricted interests and behaviors. Deficits in the social domain are the hallmark domain of impairment characterized by reduced social reciprocity (American Psychiatric Association, 2000), decreased social attention and motivation (Dawson, Webb, & McPartland, 2005a), strained peer relationships (Kelly, Garnett, Attwood, & Peterson, 2008), and deficits in social cognitive abilities such as imitation (Williams, Whiten, & Singh, 2004), empathy (Sigman, Kasari, Kwon, & Yirmiya, 1992), and theory of mind (Baron-Cohen, Leslie, & Frith, 1985).

Evidence from multiple modalities suggests that atypical function of an execution/observation matching system, also described as a mirror neuron system (MNS), may underpin these social cognitive difficulties in ASD. First identified in non-human primates using single electrode recording (Rizzolatti & Craighero, 2004; Rizzolatti, Fadiga, Gallese, & Fogassi, 1996), an analogous MNS system has since been described in humans using multiple, convergent neurophysiological modalities, including transcranial magnetic stimulation (TMS; Fadiga, Fogassi, Pavesi & Rizzolatti, 1995), functional magnetic resonance imaging (fMRI; Iacoboni et al., 1999), and intracranial electrodes (Mukamel, Ekstrom, Kaplan, Iacoboni, & Fried, 2010). MNS activity has also been studied using electroencephalography (EEG) and is reflected in attenuation of the mu rhythm, evident during both the execution and observation of movement within one's behavioral repertoire (Cochin, Barthelemy, Roux, & Martineau, 2001; Muthukumaraswamy, Johnson, & McNair, 2004; Pineda, 2005). The EEG mu rhythm, first observed in the 1954 (Gastaut & Bert, 1954), is defined as oscillatory activity in the 8–12 Hz band recorded over sensorimotor cortex. Although EEG offers limited spatial information about the source of this oscillatory rhythm, TMS stimulation of brain regions that interact with the MNS (i.e., the inferior frontal gyrus) abolishes EEG mu rhythm (Keuken et al., 2011), suggesting this EEG marker reflects, at least in part, activity localized to the MNS.

To date, seven independent studies and a pooled analysis have examined MNS function using EEG in ASD. An initial study (Oberman et al., 2005) with individuals with ASD ranging in age...
from 6 to 46 years found evidence of intact mu suppression during the execution but not the observation of actions in ASD relative to an age and gender matched control group. This was replicated in a sample of adult males with ASD and age and IQ matched typical males, additionally demonstrating that the degree of mu attenuation in the adults significantly correlated with behaviorally assessed imitative abilities (Bernier, Dawson, Webb, & Murias, 2007). A third study of 5–7 year old children with ASD and age and gender matched control children showed suppression of mu during the observation of human action in the typical children but not the children with ASD (Martineau, Cochin, Magne, & Barthelmy, 2008). Other studies have partially replicated these results or found typical patterns of activity. In a study of 13 school aged children (aged 8–12 years) with ASD, atypical mu suppression was observed during observation of unfamiliar people while typical activity was noted when the observed action was performed by a familiar person (Oberman, Ramachandran, & Pineda, 2008). Raymaekers, Wiersema, and Roeyers (2009) reported no differences in mu attenuation during the observation of hand actions in 8–13 year old high functioning children with ASD and age and IQ matched peers; in this study, children with ASD, but not typical counterparts, showed a correlation between mu attenuation and age. Finally, in a sample of 11–26 year old individuals with ASD and 20 controls matched on age, gender, and IQ, no differences in EEG mu rhythm attenuation or visual attention to stimuli were observed between groups during the observation of hand actions (Fan, Decety, Yang, Liu, & Cheng, 2010). The authors also reported poorer imitative performance in the ASD group relative to the controls, despite intact mu attenuation, and that communicative ability of the ASD participants did correlate with mu attenuation.

Given wide individual variability in ASD (Jones & Klin, 2009), variation in samples has been proposed to account for the conflicting findings reported in the literature. These studies have varied in key elements, including diagnostic characterization of the sample. Only one study (Bernier et al., 2007) relied upon gold standard (outlined in Filipek et al., 1999) research diagnostic measures (Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter & Le Couteur, 1994), the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000), and DSM-IV criteria (American Psychiatric Association, 2000). Three studies used only the ADI-R (Fan et al., 2010; Martineau et al., 2008; Raymaekers et al., 2009), one study used only theADOS (Oberman et al., 2008), and one study relied on clinical judgment alone (Oberman et al., 2005). This variability in diagnostic characterization may have contributed to the variability observed in results.

Age, visual attentional characteristics, and communicative ability have also been posited to contribute to heterogeneity in brain studies of ASD. In research to-date, participants’ age has ranged widely from 5 to 46 years of age. Oberman et al. (2012) pooled EEG mu rhythm data from four previously published studies to examine developmental changes in the mu rhythm. A significant correlation between age and mu attenuation during the observation, but not the execution, of actions was observed for both groups. However, this correlation was similar for both groups; because MNS research in ASD has relied on age-matched comparison samples, this is unlikely to account for observed heterogeneity. Variability in visual attention to the observed stimuli has also been implicated as a potential factor influencing mixed findings in MNS research in ASD. If children with ASD attend to visual stimuli less than comparison groups, as might be expected given decreased visual attention to social stimuli in ASD (Klin, Jones, Schultz, Volkmar, & Cohen, 2002), this could result in group differences in mu attenuation. Fan and colleagues (Fan et al., 2010) found no differences in visual attention as defined by fixation number and duration but also noted intact mu attenuation in the ASD group providing some insight into this factor. Fan and colleagues have also suggested that that communicative ability may influence MNS activation. Though not de facto measured, communicative proficiency, as measured by parent interview on the ADI-R, correlated with degree of mu attenuation in their ASD sample, though these correlations included individuals that failed to meet ADI-R criteria for ASD.

Finally, variability in imitation ability within individuals with ASD may contribute to the conflicting findings. Imitation impairments have long been noted in ASD since DeMeyer and colleagues published the first report in 1972 (DeMeyer et al., 1972), although the extent of the deficit is unclear (Sevlever & Gillis, 2010). A deficit in self-other mapping has been proposed to account for the imitation deficits noted (Rogers, Bennetto, McEvoy, & Pennington, 1996) and the MNS has been proposed as a neurological substrate for self-other mapping (Williams, Whiten, Suddendorf, & Perrett, 2001). As a result, it is conceivable that an execution/observation matching system could contribute to imitative ability, and conversely, deficits in imitation might be linked to MNS dysfunction. Bernier and colleagues (Bernier et al., 2007) reported a significant correlation between mu rhythm and imitation ability, though Fan and colleagues (Fan et al., 2010) did not replicate this finding using a different methodology. The current study sought to inform understanding of heterogeneity in MNS function in ASD by (a) studying a rigorously characterized sample of children with ASD and (b) tightly matched typically developing controls while (c) carefully examining relationships with factors purported to influence MNS activity, including age, autism related communication deficits, and imitative skill.

2. Methods

2.1. Participants

The original sample consisted of 56 children, 29 with ASD and 27 with typical development. Exclusionary criteria for ASD and TYP participants included known genetic disorders, seizures, significant head injury and use of anti-convulsant or barbiturate medications. TYP participants were additionally excluded if there were use of any psychotropic medication. Participants with ASD were identified based on clinical judgment of DSM-IV TR (American Psychiatric Association, 2000) criteria by a clinician with specific expertise in the diagnosis of ASD and met research criteria on both the Autism Diagnostic Interview-Revised (ADI-R, Lord, Rutter & Le Couteur, 1994) and the Autism Diagnostic Observation Schedule-Generic (ADOS-G, Lord et al., 2000). Eight of the ASD children in the original sample did not meet criteria on the ADI-R orADOS at the time of the assessment and were therefore excluded. The original typical sample included 7 females. Of these 7, 2 were randomly selected to include in the final sample to equate gender distribution between groups. Five of the children (2 ASD, 3 TYP) were excluded due to an insufficient number of artifact free trials in the EEG data. The final sample of 38 children included 19 children with ASD (18 males, 1 female), and 19 neurotypical children (17 males, 2 females). Cognitive abilities were assessed in both groups using the Differential Abilities Scale – 2nd Edition (DAS-II; Elliot, 2007). 4 children with ASD had Full Scale IQ estimates falling below the average range. Table 1 presents the demographic and cognitive information of the participants.

2.2. EEG assessment

2.2.1. Recording

Electrical brain activity was recorded using a 128-electrode dense-array EEG system developed by Electrical Geodesics (EGI Inc., Eugene OR). Impedances were below 50 kΩ and signals were referenced to the vertex, analog filtered between .1 and 100 Hz.
amplified, and digitized at 500 samples/s. Potentials were re-referenced to the average offline, including manual and automated artifact detection, using NetStation 4.3 software provided by EGI. Segments were inspected for movement artifacts by automated algorithms in NetStation software identifying fast average amplitudes exceeding 200 μVs, differential average amplitudes exceeding 100 μVs, and zero variance across trial. Segments also underwent strict manual artifact rejection via visual inspection in Matlab to remove all trials with any movement. Trials with significant artifact were excluded from analysis. The rate of trial rejection did not differ between groups (ASD: 39.8%; TYP: 29.7%), but as anticipated, did differ between conditions. The execute (43.3%) condition had a greater rate of trial rejection due to movement artifact than the observe (34.1%) or resting (25.4%) conditions.

2.2.2. Paradigm

The EEG paradigm consisted of three conditions: observe, execute, and rest, adapted from the paradigm developed by Muthukumaraswamy et al. (2004). During the observe condition, the participant watched a video presented on the monitor of a person grasping a block of wood with a sensor on it, known as the manipulandum. Each observe trial lasted 6 s, with the grasp occurring at exactly 3 s. A photocell attached to the video monitor indicated and marked the precise time point the grasp occurred. Participants were monitored closely for visual attention to the display and trials in which children did not attend to the screen were discarded. During the execute condition, an identical manipulandum as shown in the observe condition was placed in front of the participant. The participant was instructed to grasp the manipulandum upon hearing a prerecorded auditory cue, in the same manner as the person in the observe condition video clip. Each execute trial lasted 6 s, which the auditory cue occurring at exactly 3 s. A sensor on the manipulandum indicated the precise time point the participant’s grasp occurred. Forty trials of the observe condition and twenty trials of the execute condition were presented in randomized blocks of ten trials. Throughout the observe and execute blocks, including between trials, the image of the manipulandum remained on the screen. During the rest condition, spontaneous EEG was collected while participants were asked to sit quietly and focus on a plus sign displayed on the screen.

2.2.3. EEG analysis

As per Muthukumaraswamy et al. (2004), the trials were segmented into 2 s epochs, 1 s of data pre and post grasp in the observe and execute conditions as marked by the photocell or manipulandum sensor, and 2 s epochs segmented from the rest condition. Fast Fourier transforms (FFTs) were performed in Matlab (version 7.11.0.584, R2010b, Natick, MA) on each segment. Following Muthukumaraswamy et al. (2004) and Bernier et al. (2007), a cluster of eight electrodes on each hemisphere surrounding the standard C3 and C4 positions were used for statistical analyses.

Power spectra were calculated based on the average across the included trials in each condition. Mu attenuation was defined as the log of the ratio of power in the 8–13 Hz range during the observation or execution of the grasp, relative to power during the rest condition in this range. The use of a ratio score accounts for individual variability in EEG power across this frequency range. The use of the log transformation accounts for the inherent non-normality of ratio data. This calculation results in a value with zero representing no attenuation of the EEG mu rhythm, a negative value indicating attenuation and a positive value suggestive of augmentation. An individual was defined as showing attenuation by having a calculated log ratio of negative value. A non-attenuator was defined as showing no mu attenuation (a zero log ratio value) or enhancement of mu power as reflected in a positive log ratio value.

To construct topographic plots, segmented participant data were grand-averaged across the 8–13 Hz frequency band and plotted using EEGLAB’s v10.2.2.4b; (Delorme & Makeig, 2004) spectroto function. To construct time course plots, power in the 8–13 Hz frequency band recorded from central electrode clusters surrounding C3 and C4 was grand averaged across all participants for each condition. Power across the 6 s execute and observe trials was compared to reference power to calculate relative power and demonstrate time course following (Pfurtscheller & Lopes da Silva, 1999).

2.3. Imitation assessment

Imitation ability was assessed and coded using a battery adapted from Rogers and colleagues’ Mature Imitation Task (Rogers, Cook, & Greiss-Hess, 2005). Participants were instructed to imitate 8 single-hand gestures and 8 single-face gestures displayed on a video screen. Each gesture video clip lasted 6 s, with the complete gesture being presented at 3 s for at least 3 s or until the participant completed his or her imitation of the gesture. No time limit was imposed upon subjects. The subjects were recorded during the imitation of these gestures, and accuracy of imitation was scored offline by a coder blind to subject status. Inter-rater reliability of .90 or above was maintained by coders with the first two authors. Following the Mature Imitation Task manual, accuracy of hand and finger position and orientation, accuracy of facial imitation and expression, and number of imitative errors such as extraneous movements, overshooting, repetition, and approximated attempts were coded, yielding imitation ability scores for hand imitation, face imitation, and number of errors for each type of imitation.

2.4. Communication impairments

Autism related communication impairments were estimated in the ASD group using the ADI-R communication domain subscale score following Fan et al. (2010). Through parent report, the ADI-R communication domain assesses ASD-related communication impairments following DSM-IV criteria and scores each

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Table 1

Characteristics of the participants by diagnostic group, including age, IQ, and imitation ability.

<table>
<thead>
<tr>
<th>Groups</th>
<th>N</th>
<th>Age [years] (mean, SD)</th>
<th>Full scale IQ* (mean, SD)</th>
<th>Verbal IQ* (mean, SD)</th>
<th>NonVerbal IQ* (mean, SD)</th>
<th>Imitation score [Face/Hand] (mean, SD)</th>
<th>Imitation errors [Face/Hand] (mean, SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TYP</td>
<td>19</td>
<td>6.4 (1.3)</td>
<td>118.3 (12.7)</td>
<td>124.0 (13.1)</td>
<td>112.0 (14.9)</td>
<td>12.05 (3.5)/11.11 (1.6)</td>
<td>3.8 (1.5)/9.2 (4.2)</td>
</tr>
<tr>
<td>AUIT</td>
<td>19</td>
<td>6.9 (1.5)</td>
<td>95.5 (13.9)</td>
<td>96.2 (14.6)</td>
<td>96.3 (14.5)</td>
<td>10.26 (2.3)/8.6 (3.8)</td>
<td>5.6 (2.5)/10.6 (4.6)</td>
</tr>
</tbody>
</table>

19 Children (18 males) with Autism Spectrum Disorder (ASD).
19 Children (17 males) with neurotypical development (TYP).
* Significant difference between groups p < .05.
** Significant difference between groups p < .01.
communication behavior on a scale of 0 to 2 with 0 representing no deficit and 2 representing autism like impairment.

3. Results

3.1. Mu rhythm attenuation

Fig. 1a and b shows the mu rhythm surface topography of the grand averaged execute and observe conditions highlighting the consistency across conditions while Fig. 1c shows the grand averaged time course of mu rhythm power during the execution and observation of hand actions across all participants.

As shown in Fig. 2, both groups showed attenuation of the EEG mu rhythm relative to baseline resting during the observation and execution of hand actions. Repeated measures ANOVA was conducted to examine group differences in mu rhythm attenuation during both execute and observe conditions. Analyses revealed no differences between groups (F(1, 36) = .01, p = .91, partial $\eta^2 = .000$), conditions (F(1, 36) = .31, p = .58, partial $\eta^2 = .009$), or interactions (F(1, 36) = 2.5, p = .12, partial $\eta^2 = .06$). All participants showed attenuation of the EEG mu rhythm during the execution of actions, but seven (5 TYP and 2 ASD) of the participants failed to show any attenuation of the EEG mu rhythm during the observation of actions. Mu rhythm was not correlated with either verbal or nonverbal IQ. Further the four children with ASD and cognitive abilities falling below the average range did not differ in degree of mu attenuation during the observe condition from the remaining children with ASD ($t(17) = .50, p = .62$) or from the TYP participants ($t(21) = .28, p = .78$).

![Fig. 1. Surface topography of grand averaged executed (1a) and observed (1b) goal directed hand actions for all participants for the mu rhythm (8-13 Hz band) and grand averaged time course (1c) of relative mu rhythm power recorded from central electrode clusters (marked in 1a & b with an x) during the execution and observation of hand actions across all participants.](image)

![Fig. 2. Mu attenuation during the observation and execution of goal directed hand actions for children with ASD and typical development (TYP).](image)
3.3. Relationship of mu rhythm to factors purported to influence MNS functioning: age, communication impairments, and imitation skills

Correlational analyses revealed significant associations between mu rhythm attenuation during the observation condition and facial imitation ability (r(36) = -.38, p < .0125), but not with hand imitation (p = .86), age (p = .13), or autism related communication impairments (p = .39). A stricter p value of .0125 was utilized to determine significance to account for multiple comparisons. Fig. 3 shows the relationship between mu rhythm attenuation and facial imitation ability. One way ANOVA was conducted to examine differences on behaviorally assessed hand and facial imitation abilities between participants who showed mu rhythm attenuation during the observation of actions displayed significantly impaired imitation abilities. We found no differences between children with ASD and age-matched typically developing peers. These findings suggest that differences in EEG mu rhythm in ASD may be a reflection of differences in imitative ability, rather than a corollary of ASD per se. Thus previous reports of atypical MNS functioning in ASD likely reflect imitation deficits commonly reported at higher rates in ASD groups than typical samples (Williams et al., 2004).

Significant variation of imitative ability across ASD samples is possible, as imitation deficits are not part of the current diagnostic criteria for ASD. Indeed, in this study we observed significantly poorer imitative performance on hand based imitative tasks and a greater number of errors when attempting facial imitation tasks, but no differences in overall performance on facial imitation tasks. While imitation deficits have often been noted in ASD (Williams et al., 2004), some studies have demonstrated conditions in which imitation related skills are intact in ASD (Sevlever & Gillis, 2010). For example, primed automatic imitation effects appear intact in ASD (Bird, Leighton, Press, & Heyes, 2007; Gowen, Stanley, & Miall, 2008; Press, Richardson, & Bird, 2010); action representation task performance is unimpaired (Hamilton, Brindley, & Frith, 2007); and during elicited facial imitation tasks, children with ASD perform similarly to typically developing children (McIntosh, Reischmann-Decker, Winkelmann, & Willbarger, 2006).

These findings are analogous to those in the literature regarding face perception, electrophysiology and ASD. The N170 event-related potential (ERP) component, a negative voltage reflection that occurs approximately 170 ms following the observation of a face, is considered to reflect activity underlying the structural encoding of faces (Bentin, Allison, Puce, Perez, & McCarthy, 1996). While many studies have reported amplitude and latency differences in the N170 in ASD (Dawson et al., 2005b; McPartland, Dawson, Webb, Panagiotides, & Carver, 2004; O’Connor, Hamm, & Kirk, 2007; Webb, Dawson, Bernier, & Panagiotides, 2006), no differences between individuals with ASD and typical development have been reported (Jemel, Mottron, & Dawson, 2006; Laharie et al., 2006; Webb et al., 2009). Because deficits in face perception, like imitation, are often noted in ASD, but not part of the core diagnostic criteria, differential activity in the N170 component would be expected in ASD, but as a function of the face perception deficits, not the diagnosis. The findings from this study suggest an analogous phenomenon with imitation and the EEG mu rhythm.

The findings from this study replicate the McIntosh et al. (2006) findings of intact elicited facial imitation ability, but differed from a previous study with adults using the same procedure (Bernier et al., 2007). While diagnostic methodologies were the same for the children and adults with ASD in both studies, because imitation is not a diagnostic criterion for ASD, variability in performance is likely. Children with ASD are currently provided with a much wider array of treatment opportunities than previously available. Further, many of the intensive behavioral interventions, such as the Early Start Denver Model (ESDM; (Dawson et al., 2010)), incorporate imitation training into the treatment. Indeed, in the current sample, four of the children with ASD participated in an NIH funded two-year ESDM intervention study, indicating that at least a quarter of the ASD group had received specific imitation training. This variability in imitation ability likely contributed to the discrepancy in findings between this study and the study with adults.

As expected, all of the participating children demonstrated mu attenuation during the execution of simple, goal-directed hand actions. The majority of the children also showed some degree of mu attenuation during the observation of actions, but seven of the children failed to show so. This variability in attenuation during the observation condition is likely reflected in the topography in which there is a lack of a clear maximally attenuated signal hovering around the observation of actions displayed significantly impaired imitative abilities.
around electrodes 37 & 105 (C3 & C4, respectively). This dispersed
topography could impact measures of attenuation resulting in the
observed rates of children failing to show significant attenuation.
Those seven children, five of whom were typically developing, per-
formed significantly worse on behaviorally assessed imitation
highlighting the role of imitation rather than clinical diagnosis in
modulation of the EEG mu rhythm.

An interesting finding is that while mu attenuation moderately
coincided with facial imitation, it did not correlate with hand
imitation. Further, children that failed to show mu attenuation
showed worse face imitation ability, but not hand imitation ability.
While it is possible that this finding of a relationship between mu
attenuation during the observation of hand actions and face imita-
tion, but not hand imitation, is coincidental, there are differences
in hand and facial imitation that could contribute to this result.
Facial imitation abilities develop earlier in life (Sevlever & Gillis,
2010), recruit different neural regions than hand imitation (Gold-
enberg & Karnath, 2006), and rely more heavily on an execution/
observation matching system because there is no visual feedback
(Bernier et al., 2007). As a result, differential neural contributions
and increased recruitment of an execution/observation system
would translate into greater mu wave attenuation because of the
desynchronization of the underlying cell assemblies. This could ac-
tcount for the stronger relationship between facial imitation and
mu wave attenuation.

The findings from this study both parallel and contradict the re-
sults obtained by Fan et al. (2010). Both studies failed to find differ-
ences between children with ASD and typical peers, however Fan
and colleagues did not observe a correlation between mu attenua-
tion and imitation ability. Significant methodological differences
may contribute to this discrepancy. While mu attenuation for both
studies was defined as the log of the ratio of power during obser-
vation relative to baseline, the observed stimuli differed. Partici-
pants in the Fan et al. study observed a hand manipulating a
chessman, while participants in this study observed a grasping ac-
tion closely following the paradigm of Muthukumaraswamy et al.
(2004). More relevantly, imitation ability was assessed in the cur-
rent study using a manualized coding system for 8 hand and 8 face
gestures (Rogers et al., 2005), yielding codes for accuracy of hand
and finger position and orientation, accuracy of facial imitation
and expression, as well as various imitative errors such as extrane-
ous movements, overshooting, repetition, and approximated at-
tempts. This provided extensive information with regard to var-
ious aspects of imitation. In the Fan et al. study (Fan et al.,
2010), coded behavior consisted of the participant’s manipulation
of the chessman. Imitation ability was defined as the number of
incorrect movements during the manipulation. Finally, in the pres-
ent study we restricted our analyses to individuals in the ASD
group who met diagnostic criteria for ASD. These methodological
differences could contribute to the discrepant findings.

This study differs from previous studies in a variety of ways.
This is the first paper to use the gold-standard diagnostic proce-
ure for characterizing ASD. This significantly reduces the variabil-
ity of the ASD group by providing clearly replicable diagnostic
parameters. Secondly, the sample included children with ASD with
impaired cognitive abilities and while this increased the heteroge-
eity of the ASD sample in this domain, it expanded the cognitive
range of participants. This increased range allows for the explora-
tion and generalization of mu attenuation findings in ASD to the
many individuals with ASD with cognitive impairments. The inclu-
sion of children with ASD with comorbid cognitive impairments
prevented us from matching our typical peers on cognitive ability,
the lack of a correlation between mu rhythm attenuation and IQ
suggests this is not a critical factor. Further, comparison of mu
attenuation between the 4 children with ASD with IQ estimates
falling below the average range and both children with ASD with
average cognitive development and typical children revealed
non-significant differences. Additionally, as mentioned above, the
use of a manualized imitation coding system allowed for the care-
ful examination of this social cognitive ability and its relation to
mu rhythm attenuation to the observation of goal directed hand
actions.

5. Conclusion

Given the differences in design from this study and previous
studies, these findings contribute to the complicated literature
examining the mu rhythm in ASD and highlight the contributions
diagnostic choice, behavioral heterogeneity, and methodology
as mediating factors contributing to mu rhythm attenuation. It also
emphasizes the clinical and neurological heterogeneity under the
umbrella of ASD. Further research relying on carefully defined phe-
notypic and genotypic subtypes and standardized behavioral
assessments to better account for heterogeneity, is necessary to
clarify the nature and extent of the mu rhythm variability in
ASD, and the functioning of underlying neurological systems.

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