

Video Article

EEG Mu Rhythm in Typical and Atypical Development

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URL: <https://www.jove.com/video/51412>

DOI: [doi:10.3791/51412](https://doi.org/10.3791/51412)

Keywords: Medicine, Issue 86, Electroencephalography (EEG), mu rhythm, imitation, autism spectrum disorder, social cognition, mirror neuron system

Date Published: 4/9/2014

Citation: Bernier, R., Aaronson, B., Kresse, A. EEG Mu Rhythm in Typical and Atypical Development. *J. Vis. Exp.* (86), e51412, doi:10.3791/51412 (2014).

Abstract

Electroencephalography (EEG) is an effective, efficient, and noninvasive method of assessing and recording brain activity. Given the excellent temporal resolution, EEG can be used to examine the neural response related to specific behaviors, states, or external stimuli. An example of this utility is the assessment of the mirror neuron system (MNS) in humans through the examination of the EEG mu rhythm. The EEG mu rhythm, oscillatory activity in the 8-12 Hz frequency range recorded from centrally located electrodes, is suppressed when an individual executes, or simply observes, goal directed actions. As such, it has been proposed to reflect activity of the MNS. It has been theorized that dysfunction in the mirror neuron system (MNS) plays a contributing role in the social deficits of autism spectrum disorder (ASD). The MNS can then be noninvasively examined in clinical populations by using EEG mu rhythm attenuation as an index for its activity. The described protocol provides an avenue to examine social cognitive functions theoretically linked to the MNS in individuals with typical and atypical development, such as ASD.

Video Link

The video component of this article can be found at <https://www.jove.com/video/51412/>

Introduction

Electroencephalography (EEG) is an effective, efficient, and noninvasive method of assessing and recording brain activity. As neurons fire in the brain, the resulting voltage can be amplified, recorded, and graphically represented. The temporal resolution of EEG allows for the analysis of even brief changes in the oscillation patterns of the brain, as well as the analysis of the brain's response to specific stimuli.

Despite being the oldest brain imaging technique, dating back to the late 19th century, EEG still has wide-ranging applicability. While functional magnetic resonance imaging (fMRI) has excellent spatial resolution, it has relatively poor temporal resolution. This represents a major limitation of fMRI assessment given the incredible speed at which processes occur in the brain. EEG has the ability to assess electrical brain activity at the millisecond level, providing potential insight into the phases of the brain's processing.

Evolving technologies have also expanded the applicability of EEG. An increase in the density of recording systems has allowed for the development of source localization techniques, mitigating some of EEG's limitations regarding spatial resolution. Additionally, modern systems have reduced the individual participant set-up time significantly, allowing for the assessment of previously unavailable populations, such as infant and clinical samples^{1-3,28-30}.

Given the excellent temporal resolution, EEG can be used to examine the neural response related to specific behaviors, states, or external stimuli. An example of this utility is the assessment of the mirror neuron system (MNS) in humans. Mirror neurons were originally identified in monkeys using single neuron recording⁴, evidencing a group of neurons that responded to both the execution and observation of motor actions. This direct recording method of placing electrodes in the brain is rarely utilized in humans, and only in dire clinical cases. EEG has provided a method for assessing the MNS by monitoring the EEG mu rhythm. This oscillation pattern in the 8-12 Hz range has been shown to attenuate EEG power in response to the execution and observation of motor actions, similar to the activation pattern observed in monkeys⁵⁻⁷. Similarly, stimulation of putative MNS brain regions through Transcranial Magnetic Stimulation (e.g. the inferior frontal gyrus) abolishes EEG mu rhythm⁸ and EEG mu rhythm suppression correlates with BOLD signals from fMRI in putative mirror neuron regions within subjects⁹, providing additional support that this rhythm indexes, at least in part, MNS activity. Assessment of the EEG mu rhythm has allowed for a noninvasive assessment of mirror neuron activity in humans.

EEG provides a unique methodology for examining brain activity and when combined with behaviorally based assays, it can be a powerful tool for elucidating aspects of social cognition, such as imitation, in clinical populations. Further, the applicability of EEG for use with populations with

cognitive or language impairments allows for insight into abilities of individuals for whom other imaging techniques or behavioral paradigms may be less successfully utilize. The described protocol provides an avenue to examine social cognitive functions theoretically linked to the mirror neuron system in individuals with typical and atypical development, such as Autism Spectrum Disorder.

Protocol

The following protocol adheres to the guidelines of the University of Washington institutional review board.

1. Electrophysiological Assessment

1. Preparation of Session
 1. Room preparation: place the manipulandum (see **Figure 1**), a wooden block with a sensor attached, which sends a time-stamped marker to acquisition software when it is grasped, on the table within grasping reach of the participant. Activate EEG acquisition software and begin "new session" (**Figure S1**).
 2. Net preparation: warm solution of distilled water (1 L), potassium chloride (1 tablespoon), and baby shampoo (1 teaspoon) to 104 °F. Soak 128-electrode dense-array EEG system in the warmed saline solution.
 3. Participant preparation: ensure that the participant is seated comfortably approximately 75 cm from the stimulus presentation monitor and fully in view of the video camera. Find and mark the vertex on the participant's head with a skin marker. Measure the vertex by finding the intersection of the midpoint between the nasion and the inion and the midpoint between preauriculars.
 4. Net application: Position the EEG cap on the participant's head such that the vertex electrode is placed directly over the vertex mark. Check impedances and ensure that impedances are below the threshold appropriate for the EEG system in use (**Figure S2**).
 5. Begin video taping session.
2. Recording setup: Reference signal to the vertex electrode. Analog filter between 0.1 and 100 Hz, amplify the signal, and digitize at 500 samples/sec.
3. Stimulus presentation: present participant with 3 conditions: observe, execute and rest, adapted from the paradigm developed by Muthukumaraswamy and colleagues⁵.
 1. Observe condition: Instruct participant to sit quietly and watch a video of a person grasping the manipulandum. Each trial should last 6 sec. Time the prerecorded video for the observe trials precisely to ensure that the observed grasp occurs at exactly 3 sec. Monitor participant's visual attention during the task, and mark trials during which they do not attend to the screen to be discarded during post-processing.
 2. Execute condition: Instruct participant to sit quietly with right hand resting just below the manipulandum and, upon hearing a prerecorded auditory cue, to imitate the manipulandum grab from the observe condition video clip. Each trial should last 6 sec. Ensure that the auditory cue is presented at exactly 3 sec by prerecording an auditory track that maintains a consistently timed execute cue and inter-trial interval. Utilize a sensor on the manipulandum to precisely record the time that the participant's grasp occurs (**Figure S3**).
 3. Rest condition: Instruct participant to sit quietly with eyes open and passively observe a small crosshair on the stimulus monitor. Record continuous EEG during the rest condition for 3 min.
 4. For both observe and execute conditions, present randomized blocks of ten trials, for a total of forty trials per condition. Ensure that the image of the manipulandum remains on screen throughout the observe and execute blocks, including between trials. Administer the rest condition at the completion of the observe and execute conditions.
4. Data Processing
 1. Following data collection, recheck impedances. Note any changes to impedance levels. End acquisition software recording.
 2. Post-processing: Rereference EEG signal to the average. Segment continuous EEG data into forty 6-sec trials for each condition (**Figure S4**).
 3. Conduct automated artifact detection. Use automated algorithms to inspect segments for movement artifacts by identifying fast average amplitudes exceeding 200 μ V, differential average amplitudes exceeding 100 μ V, and zero variance across a given trial (**Figure S5**).
 4. Conduct manual artifact detection by visually inspecting data and confirming with video review of the session to remove all trials in the observation condition contaminated with any movement artifact and all trials in the execution condition contaminated with any movement artifact unrelated to the grasp gesture. Exclude trials with significant artifact from analysis. Discard any trials that were flagged during acquisition as not attended. Examine and note rate of trial rejection for each diagnostic group under analysis.
5. Data analysis
 1. Per Muthukumaraswamy *et al.*⁵, segment cleaned trials into 2 sec epochs consisting of 1 sec of data before the grasp and 1 sec after for both the observe (as marked by the photocell) and execute (as marked by the manipulandum sensor) conditions. Segment cleaned 2 sec epochs from the rest condition.
 2. Fast Fourier transform (FFT) each segment. Select a cluster of eight electrodes on each hemisphere surrounding the standard C3 and C4 positions for statistical analyses (following Muthukumaraswamy *et al.*⁵ and Bernier *et al.*³) (**Figure 2**). For each condition, average the power across the included trials to calculate power spectra.
 3. Calculate mu attenuation by examining the average power during either the execution or observation of a motor action, relative to the average power during the resting condition, across the 8-13 Hz range. Use the log of this ratio to determine degree of attenuation. Note: a negative value represents attenuation during execution or observation, while a positive value represents augmentation. This methodology takes into account variability across individuals, and the non-normality of values expressed in ratio form. Note: This protocol was developed using a 128-electrode dense-array EEG system with Net Station software version 4.1. While the basic steps are similar across EEG systems, acquisition and analysis protocols may vary.

2. Sample Characterization

1. Identify potential patient population for participation in paradigm through research registries, previous participant listings, or referrals from area clinics and clinicians.
2. Screen potential participants for likelihood of meeting diagnostic criteria for clinical construct (e.g. Autism Spectrum Disorder) and to identify any exclusionary criteria, such as presence of head injury, tumor, seizure history, or use of anti-convulsant or barbiturate medication which may distort the electrophysiological signal.
3. Confirm diagnostic status of patient population through the use of gold standard diagnostic instruments (e.g. Autism Diagnostic Interview-Revised (ADI-R¹¹) and the Autism Diagnostic Observation Schedule-Generic (ADOS-G,¹²) administered by expert clinician following Diagnostic and Statistical Manual – 5th Edition (DSM-5) criteria¹³.
4. Identify control sample matched on relevant variables of interest, such as age, gender, cognitive ability, etc.

Representative Results

Typical adults, children and infants have consistently demonstrated mu rhythm during both the execution and observation of actions across a variety of paradigms and stimuli^{5,14-30}. Attenuation in this frequency band is consistently localized across central electrodes (**Figure 3**) indicating that this is not reduction of alpha power which is recorded at other scalp regions. Similarly, attenuation in this frequency during the observation of movement is limited to the observation of biologically based movement and suppression is not elicited simply from movement passing through the visual field, such as bouncing balls (**Figure 4**). Mu suppression in response to an event, such as the execution or observation of a goal directed grasping action, is demonstrated by the reduction in spectral power followed by a return to baseline levels (**Figure 5**).

There have been eight independent studies and one pooled analysis of the EEG mu rhythm and social cognition in the ASD population. While the findings of mu suppression during both the observation and execution of actions have been consistently observed in typically developing individuals, findings regarding the mu rhythm in ASD have been variable. An initial study³¹ of the EEG mu rhythm compared individuals with ASD between the ages of 6-46 years to an age and gender matched control group. The ASD group demonstrated mu attenuation only during the execution of actions, and not during observation. This same pattern was replicated in adult males with ASD compared to a group of age and IQ matched typical peers, and in this group the degree of mu attenuation was significantly related to imitative ability³. Similarly, a third study failed to find mu suppression during the viewing of human-performed actions in 5-7 year old children with ASD, but did in the age and gender matched typical peers³². A second study by Oberman *et al.*³³, found typical mu suppression in a sample of 13 8-12 year old children with ASD during the observation of actions displayed by familiar people (mothers), but not during the observation of actions performed by unfamiliar people³³. Three studies have failed to find group differences in mu suppression between individuals with ASD and control groups. During the observation of actions performed by human hands, no differences in mu attenuation were found between 8-13 year old children with ASD and age and IQ matched typically developing children³⁴ or between 11-26 year old individuals with ASD and age, gender, and IQ matched peers³⁵.

Finally, Bernier and colleagues³⁶ found no differences between children with ASD, meeting gold standard diagnostic criteria, and age and gender matched peers on mu attenuation during the observation of goal directed actions, but did find a significant relationship between the EEG mu rhythm and behaviorally assessed imitation abilities. This suggests that the differences in EEG mu attenuation that have been observed may reflect differences in the ability to imitate, rather than being a direct result of ASD³⁶.

These experiments suggest that examination of the EEG mu rhythm is a viable tool for elucidating mechanisms related to social cognition in both typical and clinical populations.

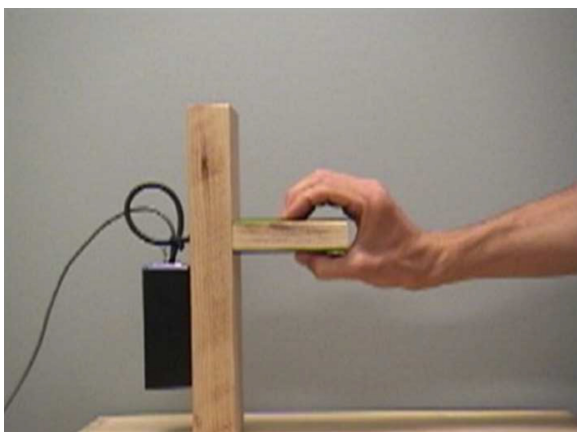


Figure 1. Manipulandum. In order to accurately examine the observation and execution of a goal-directed grasp, participants are instructed to execute a simple hand grasp of the manipulandum or observe a model grasping the manipulandum. When the manipulandum is grasped, a time locked signal is sent to the data acquisition computer for later, off-line segmenting of each trial.

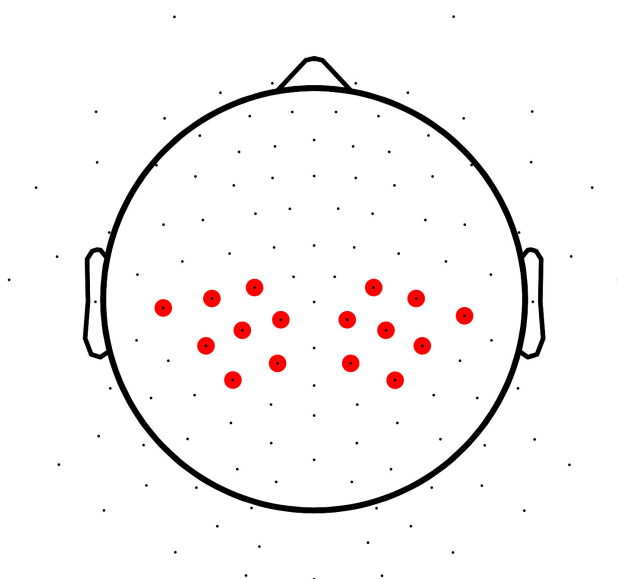


Figure 2. Dense electrode cap lead selection for capturing mu rhythm activity.

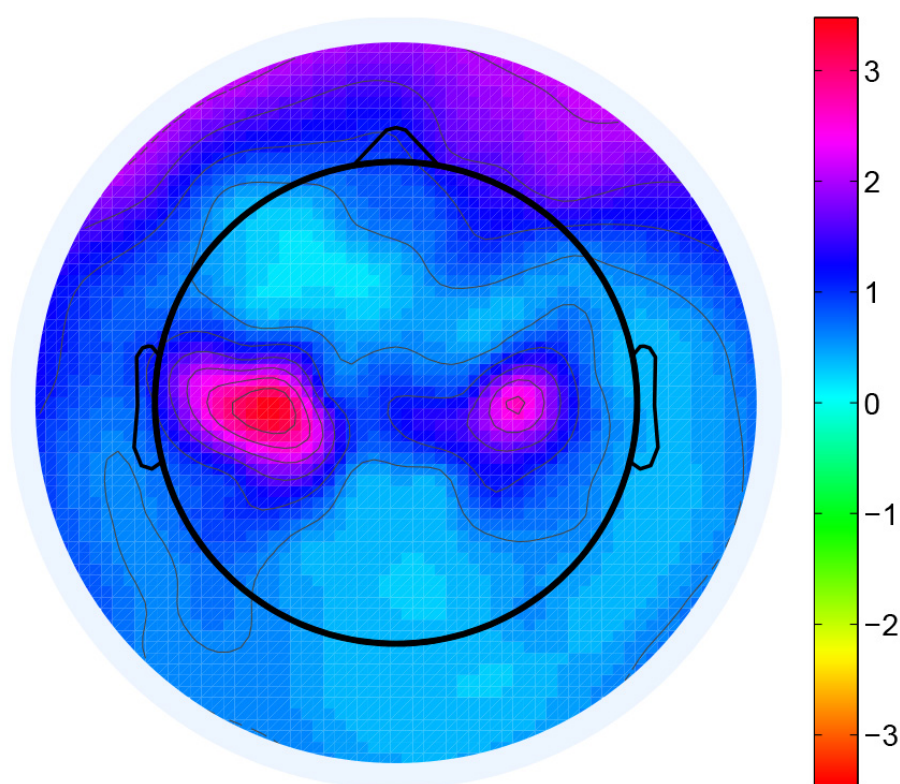


Figure 3. Characteristic topography of attenuation of the mu rhythm as demonstrated by individual with typical development (male, age 9.2 years) while observing goal directed grasping action. Mu rhythm attenuation is reflected in the scalp topography as reduced amplitude over centrally located electrodes.

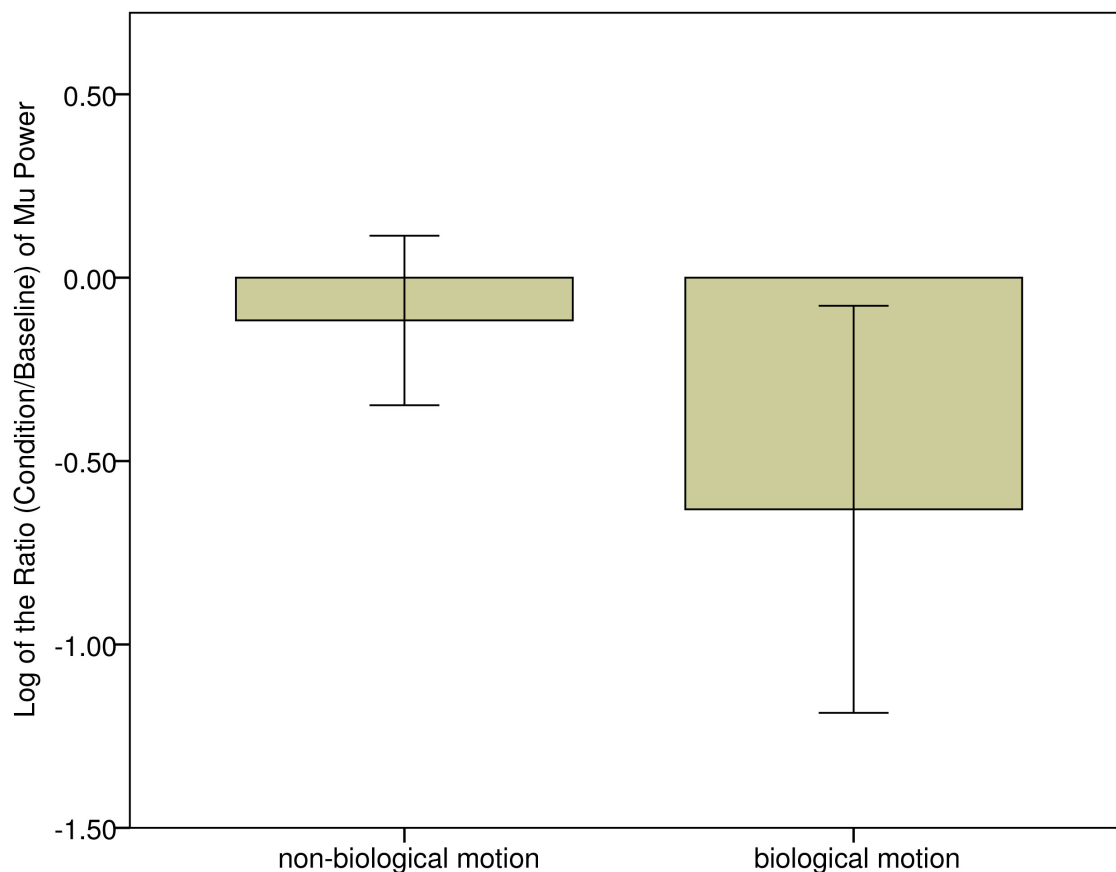


Figure 4. Spectral power during the observation of biological movement and nonbiological movement in 11 children (mean age 10.8 years (range = 8-15 years; 3 males, 8 females)). Averaged spectral power in the mu frequency (8-13 Hz) recorded from centrally located electrodes during the observation of biological movement (an animated dancer) is attenuated relative to baseline while mu power is not attenuated during the observation of nonbiological movement (an animated ball). The log transform of the ratio of power (microvolts²) for each condition over baseline indicates greater reduction of power in the biological movement condition through the more negative power value.

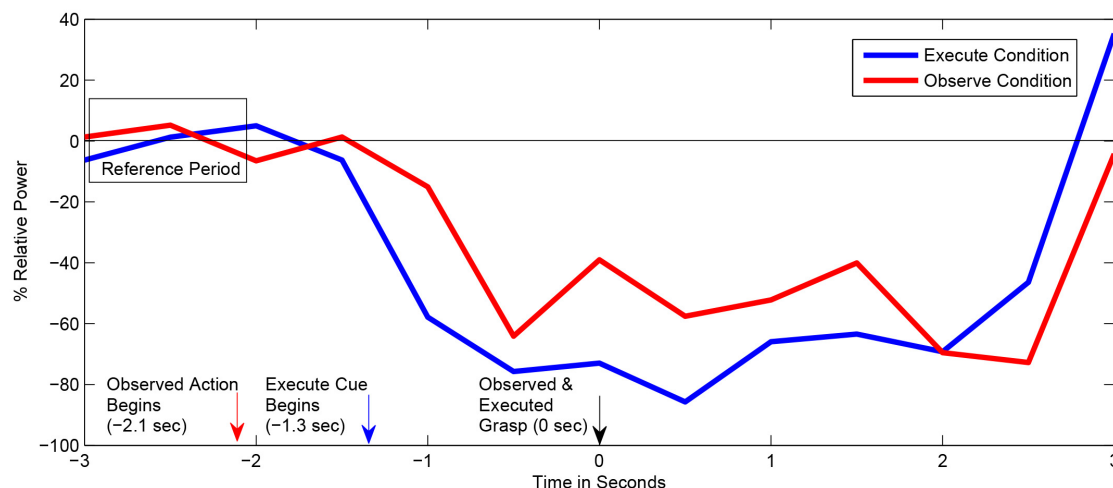


Figure 5. Event-related spectral power attenuation. Mu power (8-13 Hz from central leads) attenuates relative to baseline during the execution (averaged power over 20 grasp trials) and observation (averaged power over 30 observe trials) of actions in a typically developing 6 year old male.

Discussion

The successful acquisition, processing, and analysis of electrophysiological data related to the mu rhythm and the application to clinical populations requires 1) the application of EEG methodological tools, 2) careful artifact detection and data reduction, 3) accurate identification of the mu rhythm, and 4) accurate characterization of the clinical population and identification of appropriate control groups.

Appropriate EEG methodology requires properly functioning and integrated equipment, appropriate cap choice and placement, accurate amplification and timing of the signal, a clear, unimpeded, artifact-free signal, a properly referenced signal, appropriately segmented (if event-related) trials, carefully calculated power transformations, attention to the stimuli, and, of course, a paradigm and stimuli that elicit the cognitive capacity under investigation.

The mu rhythm is noted to be transient when examined during continuous EEG recording¹⁴ although can be clearly and reliably demonstrated during event-related analyses^{17,37}. Given the small signal to noise ratio, artifact can easily obscure changes in spectral power in this rhythm. As a result, careful artifact detection through automated programs or manual investigation of the contributing electrode data is necessary for the identification of the mu rhythm. Any differences between groups in the amount of artifact observed and removed must be recorded to ensure observed group differences are not as a result of data artifact or the artifact removal process. The final EEG data sample, in the case of event-related analyses such as described in this protocol, must contain artifact free trials and a sufficient number of trials to accurately capture the degree of attenuation, or lack thereof, for a given condition.

In order to accurately identify and index the mu rhythm, an analysis of the topography and event-related desynchronization is important to ensure the spectral power reduction is tied to the centrally located channels surrounding channels C3 and C4. If reduction is observed in other electrode clusters, this calls into question to the accurate identification of the rhythm. Additionally, the observed mu rhythm spectral power reduction should be limited to the execution and observation of actions within the behavioral repertoire. Reduction of this rhythm at rest or during the observation of nonbiological movement is suggestive of artifact, subtle movements in the observer, or inaccuracy in the assessment methods.

Development considerations are important for neurophysiological work. Attenuation of the mu rhythm has been recorded in response to the observation of goal directed actions in individuals from 8 months of age to adulthood^{28-29,38}. Importantly, while present, the degree of attenuation observed in infancy and childhood is much smaller than that noted in adults³⁹⁻⁴⁰. This pattern underscores the importance of considering developmental influences on neural rhythms and matching control populations to experimental groups on developmental level.

Finally, to conduct this work in clinical populations, the careful assessment of the clinical population is necessary to ensure groups are adequately defined. The inclusionary and exclusionary criteria for the clinical and comparison groups need to be clearly described and carefully considered. For example, the use of gold standard diagnostic instruments is necessary for establishing clinical populations. Without a clear diagnostic protocol, for heterogeneous clinical populations, the cognitive construct under study may differ vastly within a loosely defined clinical group. Tight, diagnostic definitions reduce that likelihood. If certain subpopulations of a clinical sample are excluded, that needs to be identified as it impacts the generalizability of the findings. For example, while the exclusion of individuals with epilepsy from a study of ASD helps to clean the EEG signal that could be obscured from the inclusion of individuals with comorbid seizures, this changes the generalizability of the findings given the high prevalence of seizures in individuals with ASD.

The protocol described above provides an avenue to examine an EEG index of the mirror neuron system in clinical populations in a noninvasive manner. It requires little communicative ability thereby allowing for the application to impaired individuals and is noninvasive, easily applied, and provides excellent resolution to understand cognitive constructs related to social abilities.

Disclosures

The authors declare no competing financial interests.

Acknowledgements

This work was supported by a grant from the Simons Foundation (SFARI #89638 to RB).

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