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Investigation of Action Prediction in ASD Individuals : an eye-movement guided EEG analysis

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Master's Thesis

To fulfill the requirements for the degree of
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Abstract

Autism Spectrum Disorder (ASD) is a lifelong developmental disorder characterized by difficulties in social interaction and communication, restricted interests, and repetitive behavior, according to the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). One popular assumption behind the core social characteristic of autism is that autistic individuals have difficulty understanding other people's intentions. This is assumed to be related to the reduced ability in predicting others' actions.

In the current study, we investigated the eye-tracking and neural correlates of action prediction in autistic individuals. We acquired eye-tracking data and electroencephalography (EEG) data from Ward et al. (2021). In Ward et al.'s study, she and colleagues collected EEG and eye-tracking data from both autistic and non-autistic teenagers when observing pre-recorded videos of multi-step day-to-day actions. In the experiment, each action in the video was divided into three steps and would become gradually clearer and more predictable.

Building on Ward et al.'s work, we analyzed neural information and eye-tracking data during action observation simultaneously. For eye-tracking analysis, we defined three metrics to measure the predictive behavior and its accuracy as indicated by the location of fixations. From the descriptive statistics, we observed a general higher accuracy rate for action prediction for the non-autistic group. The prediction patterns also appeared to be different between groups. For the autistic group, we observed no distinguishable difference across steps. While for the non-autistic group, more predictive fixations were made in the more predictable action steps. Further, we leveraged the eye-tracking results to 1) mark trial information and 2) set the onset for individual- and trial-dependent data segmentation for the EEG analysis. From analysis of the brain oscillation, we found more attenuation in mu power in central-parietal regions pre- and post-onset for the non-autistic group than the autistic group. We also observed a higher coherence between the occipital and parietal regions for the autistic group in the beta frequency band. According to Bayesian statistics, the between-group differences in mu power and beta coherence were not significant. However, the Bayes factors indicated that the rejection was not substantial.

In the current study, we found significant between-group differences in predictive fixation patterns and non-substantial rejection of the between-group differences in brain oscillation and connectivity. Our results contradicted the original study from Ward et al., in which the group effect was strongly rejected for both eye movements and mu/beta power. The reason for such different results is that we only included the participants with active predictive fixations while Ward et al. analyzed all available participants. The combined findings from these two studies indicate heterogeneity within the autistic group in eye-tracking and neural activities, which is also evident pathologically (Geschwind, 2009; Georgiades et al., 2012; Lenroot & Yeung, 2013). Therefore, we suggest for further studies to pre-select the autistic subjects and divide them into homogeneous subgroups before examinations.

1 Introduction

Autism Spectrum Disorder (ASD) is a lifelong developmental disorder characterized by difficulties in social interaction and communication, restricted interests, and repetitive behavior, according to the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). Various theories haven been developed to explained the underlying cause for autism (Baron-Cohen et al., 1985; Perkins et al., 2010; Pellicano & Burr, 2012; Van de Cruys et al., 2014; Sinha et al., 2014). Though these theories vary in different aspects, one common assumption behind the core social characteristic of autism is that autistic individuals have difficulty understanding other people's intentions. The difficulty can be translated into reduced ability in predicting others' actions in day-to-day life and would result in deficits in social interaction.

In the past decades, various studies were conducted to examine the action prediction in the autistic population from both neural and behavioral point of view. However, the findings were mixed. Some behavioral studies have found evidence for between-group differences. For example, when asked to arrange a set of shuffled pictures of daily actions into a logical order, autistic adolescents showed longer response time and a larger number of sequence error comparing to both the mentally retarded group and the normal control (Zalla et al., 2006). In another study by Zalla et al., autistic, mentally retarded and typical developing children were presented with short incomplete videos of an actor performing familiar and non-familiar daily actions. The children were asked to predict the possible outcomes from a selection of pictures after watching the videos. Autistic children had more prediction errors and less improvement for the familiar actions, as compared to the two other groups (Zalla et al., 2009). In an eye-tracking study, Schuwerk et al. used proactive eye movements to examine the underlying cognitive mechanism behind action prediction in both autistic and typically developed groups during repetitive animated action videos. They found that the autistic group showed less frequent anticipatory fixations and had difficulties in using statistical information to improve their action prediction (Schuwerk et al., 2016).

However, some other studies have shown evidence for similar patterns between autistic and non-autistic groups. For example, in a study by Tewolde et al., researchers found similar performance in autistic and typically developed groups in a motion prediction task. In that task, participants were asked to predict the endpoint of occluded objects with motions. As indicated by behavioral results, the autistic individuals predicted the motion as precise as the control group (Tewolde et al., 2017). In an eye-tracking study, high-functioning autistic and non-autistic adolescents were instructed to follow a moving target with their eye and predict its subsequent movement after a transient disappearance. Researchers found no significant between-group differences in occurrences and accuracy of predictive saccades (Ego et al., 2016).

As for neural correlates, the action prediction is thought to be implemented through motor system activation (Kilner et al., 2004) and can be measured by power attenuation in mu (8-12Hz) and beta (15-25 Hz) waves. According to the mirror neuron dsyfunction theory, the assumed deficits in action prediction should correspond to a reduction in motor system activation (i.e., less mu and beta power suppression) in the autistic individuals (Perkins et al., 2010). However, similar to the behavioral studies, evidence was mixed. Oberman et al. found significant mu suppression while observing another person's simple hand movements in the typically developed group but not in the high-functioning autistic group (Oberman et al., 2005). The results supported the mirror neuron dsyfunction theory. In a transcranial magnetic stimulation study, Enticott et al. found significantly reduced corticospinal ex-

citability in the ASD group during the observation of a transitive hand gesture comparing to the normal control group (Enticott et al., 2012). The result indicated that the ASD group had a reduced putative mirror neuron system activity within ventral premotorcortex/inferior frontal gyrus. In a meta-analysis of fMRI studies for action observation and imitation, Yang & Hofmann found that ASD individuals showed stronger effects in the anterior inferior parietal lobule, a part of the putative human MNS, as compared to the typical developed group. In addition, the ASD group demonstrated altered effects in the occipital cortex, dorsolateral prefrontal cortex, cingulate cortex, and insula (Yang & Hofmann, 2015).

There are also studies that show no between-group differences. For example, in an EEG study, Ward et al. examined the mu and beta power in the autistic and non-autistic adolescents during action observations. The mu and beta power attenuation showed no significant between-group differences. In a study similar to the paradigm in Oberman et al. (2005), high-functioning autistic group showed no different pattern of mu suppression when observing hand movements, as compared to the normal control group (Raymaekers et al., 2009).

In this non-exhaustive literature review, we noticed that the stimuli used in the above-mentioned studies were not alike, even though they were all studying action prediction in autism. Some studies examined the behavioral or neural patterns in autism using social-related tasks (Zalla et al., 2006, 2009; Ward et al., 2021). Others investigated the prediction of physical and sensory movements and its between-group differences (Tewolde et al., 2017; Raymaekers et al., 2009; Enticott et al., 2012; Schuwerk et al., 2016; Tewolde et al., 2017). We suspect that the different types of stimuli (social or non-social) could be a main reason for such mixed findings. Another important factor is the well-known heterogeneity within the autistic populations, which could also cause inconsistent results between two similar studies (Geschwind, 2009; Georgiades et al., 2012; Lenroot & Yeung, 2013).

In the current study, we used the experiment framework and data from Ward et al. (2021) and analyzed the data using a different approach. In Ward et. al.'s study, she and colleagues collected electroencephalography (EEG) and eye-tracking data from both autistic and non-autistic teenagers when observing pre-recorded videos of multi-step actions. In their experiment, each action in the video was divided into three steps and would become gradually clearer and more predictable. For example, the actor would first reach for the money, then put the money in the purse and lastly put the purse into a handbag. Ward et al. investigated the fixation behavior from the eye-tracking data and the motor system activation (i.e., mu and beta power reduction in central scalp regions) from the EEG signals. Based on Ward et al.'s results, beta power were modulated by the inherent uncertainty of the actions. As the action steps became gradually more predictable, both groups showed reduced beta power in the central scalp area. However, no evidence for between-group difference were found.
Building on Ward et al.'s work, we analyzed the eye-tracking and neural representations of action prediction in autism using a novel approach. Instead of analyzing the eye-tracking and EEG data separately, we performed a joined analysis. To be more specific, we aligned the EEG signals and eye-tracking recordings. Then, we analyzed the EEG signals using the metrics derived from simultaneous eye-tracking recordings. We used the eye-tracking data to 1) segment the EEG data based on individual eye movements; 2) label the trials based on eye-tracking performance and for later use in comparing EEG data between groups.

In Ward et al.'s experiments, the participants viewed the action videos passively with no response required. Because of no directly available response from the participants, it is difficult to extract the

EEG segments of our interests and correlate electroencephalographic patterns to action prediction. Thus, we could easily misread the EEG signals and extract random fluctuations as evidence for action prediction. By simultaneous analysis of eye movements and EEG data, we can better understand the internal states of the subjects through eye movements and pinpoint the onset of cognitive activities more accurately. Subsequently, we can analyze the EEG signals in an individual- and behavioral-catered manner.

After synchronization, for eye-tracking analysis, we derived predictive measurements from fixations using the eye movements coordinates. We examined the frequency and the accuracy of predictions for each subject and analyzed the group effect by Bayesian t-tests. We assumed the autistic group would have lower accuracy of prediction than the non-autistic group. For EEG analysis, we followed Ward et al.'s practice and investigated the mu and beta power attenuation in the central scalp regions. However, in contrast to Ward et al., we segmented the data using the eye-tracking data. We expected more mu and beta power attenuation from the non-autistic group than the autistic group. In addition, we examined the connectivity of brain oscillations in alpha and beta frequency band. In a visuomotor experiment, Tzagarakis et al. found the beta power desynchronization covaried with the degree of uncertainty of the tasks during the motor preparation stage (Tzagarakis et al., 2010). Therefore, we posited that the functional connectivity would also be modulated by inherent predictability of the actions and would differ between the autistic and non-autistic group.

The thesis includes three main sections: Method, Result and Discussion. In the Method section, I introduced the experiment materials and our analytical approaches in details, as well as the population statistics for the participants. In the Result section, I demonstrated results from our eye-tracking and brain oscillations analysis. Finally, I derived conclusions and proposed future work for this analysis in the Discussion section.

2 Methods

2.1 Participants and experimental materials

For the current analyses, experimental materials and the data were acquired from Ward et al. (2021).

In the original study, a total of 58 participants were recruited for the experiment and 56 of them contributed to the data (27 autistic; 29 non-autistic), among which 53 participants contributed to both EEG and eye-tracking recording (26 autistic and 27 non-autistic). All participants were aged between 12 and 18. The participants from the autistic group had a clinical diagnosis of Autistic Spectrum Disorder based on the DSM-5 (Association, 2013). Ward et al. (2021) only recruited autistic participants with no reported history of epilepsy and no other major psychiatric illness. Ward et al. (2021) also measured participants' IQ score, autistic symptoms and alexithymia level. A t-test was conducted to examine the between-group statistical difference for these measurements. The result indicated, under the significance level of 5%, no significant differences for age and IQ score for the two groups. The autistic symptoms and alexithymia level were significantly higher in the autistic group than in the non-autistic group.

Both autistic and non-autistic groups were asked to view 27 videos of a female actor performing daily activities, with no required response from the participants. The videos were an adapted version of the stimuli used in Braukmann et al. (2017). In general, each video consisted of three actions, of which the intention would become gradually clearer. At the beginning of each video, three objects (or targets) are presented on a table. The actor will first select and interact with one of the three targets, then reach for the next target. The videos would end either with the object at the actor's face or at an object on the table.

As the time progresses, there would be fewer remaining targets, hence, it would be easier for participants to predict the next actions. In addition, the actions are logically connected and based on a daily activity (e.g., put milk and oatmeal into a big bowl). Therefore, as the video unfolds, more information would be provided and participants would have a clearer view of the gist of the acts. In sum, later action would be easier to predict than earlier actions.

Figure 1 is an illustration of the actions in one of the videos. In this example, the video starts with the woman actor picking up a slice of ham (the first target), then placing it onto a piece of bread (the second target) and finally putting it into her mouth (the third target). The Areas of Interest (AOI) that will be used for the eye-tracking analysis are rectangular shapes covering the targets (see the first subfigure in Figure 1) and are defined according to Braukmann et al. (2017) andWard et al. (2021).

Each video was repeated four times: two times normally and two times with targets being occluded.

Figure 1: Screenshots of actions from an example stimulus.

In the current study, we only used the trials that presented stimuli normally, and took a total of 54 (27 different videos * 2) trials per subject. The presentation of the videos started with a fixation cross, lasting about 1250 ms, followed by 15 s of stimulus videos. The period of fixation cross was used as baseline in further analyses.

Electroencephalography (EEG) was recorded from 32 Ag/AgCl active electrodes in an EEG Acti-Cap (Brain Products, Munich, Germany) via BrainVision Recorder, and amplified by a BrainAmp amplifier (BrainVision Products, Munich, Germany). EEG signals at the left mastoid were taken as an online reference. Simultaneous eye-movements were collected with an SMI RED500 remote eye-tracker and iView software (SensoMotoric Instruments GmbH, Teltow, Germany) at a sampling frequency of 500 Hz. See Ward et al. (2021) for more details about the apparatus, presentation and recording.

2.2 Co-registration of Eye-movement and EEG data

2.2.1 Individual-dependent and universal time of interest

In Braukmann et al. (2017) and Ward et al. (2021), each of the three actions in the video were divided into two subsequent time windows: the *predictive time window* and the *reactive time window*. *The predictive time windows* begin when the actor's hand starts moving toward a target, and ends when she reaches an AOI. *The reactive time windows* mark the period in which the actor interacts with the target. Due to the nature of the video stimulus, the onset and duration of predictive and reactive time windows were different for all 27 videos. In Ward et al. (2021), the times of interest (TOI) were defined as 1200ms before the hand enters into a AOI (the end of the predictive window), which was dependent on the videos but universal across all subjects.

In the current study, we followed Braukmann et al.'s division of *predictive window* and used a different approach to define TOI which is dependent on both individuals and videos. In the experiment, participants watched the videos passively and no responses were required. In the passive viewing process, participants can decide for themselves when and whether they are engaged in the action prediction process. Furthermore, under the conceptual framework of this study, we hypothesized different action-prediction behavior and neural signatures between the autistic group and the non-autistic group. Ergo, we expect that the timing of action prediction could vary across participants in different groups. For these reasons, we used an individual-dependent method to define the time of interest. In addition, we implemented a universal TOI that is static for all the subjects, to compare with the individual-dependent method and to compare with the results from previous studies.

The individual-dependent TOI was defined using subjects' eye movements. In previous studies, Braukmann et al. (2017) and Ward et al. (2021) found that fixations were related to action prediction and were modulated by action steps. According to these results, fixations in the predictive time windows indicate predictive behavior in the corresponding trials. Therefore, in the current study, we used fixations in the predictive time windows to mark the start of the individual-dependent TOI for later analysis. More on TOIs will introduced in the Section 2.5.

2.2.2 Eye-tracking data and EEG data synchronizing

To use eye movements to mark TOI in EEG data, we first needed to connect and synchronize EEG data and eye-movement data. During the recording, trigger pulses were sent from the stimulation

computer to the EEG recorder. At the same time, short text messages containing stimulus information were sent to the eye-movement recording software. The triggers and messages are used as identifiers for the stimuli and to connect the two datasets. Due to the use of different software and jitters during recording, the starting time for the same stimuli in the EEG recordings and eye-tracking recordings can be off for tens of milliseconds. For a typical subject, the time difference was on average 55ms.

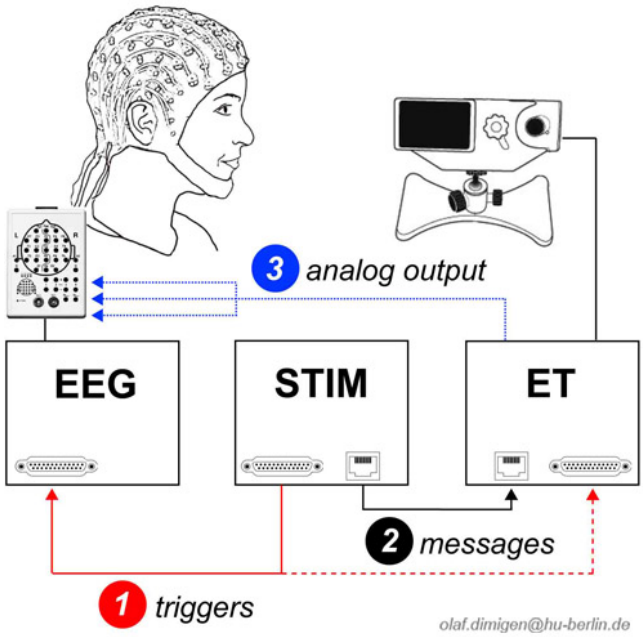


Figure 2: Connecting eye tracker & EEG (Dimigen, 2019).

In the current study, we used the EYE-EEG toolbox (Dimigen et al., 2011) and the open-source EEGLAB toolbox (Delorme & Makeig, 2004) to synchronize EEG data and eye-movement data. The EYE-EEG toolbox is a promising tool for simultaneous analysis of Eye Movements and EEG data. It was first developed to examine the effect of words' predictability on eye movements and Event-related potentials during natural sentence reading. It can be used in studying the dynamics of attention and cognition under free viewing conditions (Dimigen & Ehinger, 2021).

In the synchronization process, we first defined a communal starting and ending event, then linearly interpolated the eye-tracking data in between the start-event and end-event based on the sampling frequency of EEG data. Then, we evaluated the synchronization quality of remaining shared triggers/messages using the EYE-EEG toolbox (Dimigen, 2019). The synchronization results were generated in both figures and texts. For instance, for a typical subject 9, 293 out of 324 (90.4%) EEG events had a corresponding eye-tracking event within ± 8ms after synchronization. The tool also calculated the cross-correlation between the eye tracker and EOG channels as an evaluation method for synchronization. If the synchronization was good and EOG were highly corrected with eye-tracking data, the peak of the correlation should be around lag 0. Figure 3 shows the cross-correlation results for a typical subject (subject 9). According to Dimigen's standard, if the peak of the cross-correlation function is found within ± 4 samples (8 ms), the synchronization quality is acceptable. That is the case for the example subject. During synchronization, the fixations from eye-tracking data were also inserted as events into the EEG data accordingly for future use in EEG segmentation.

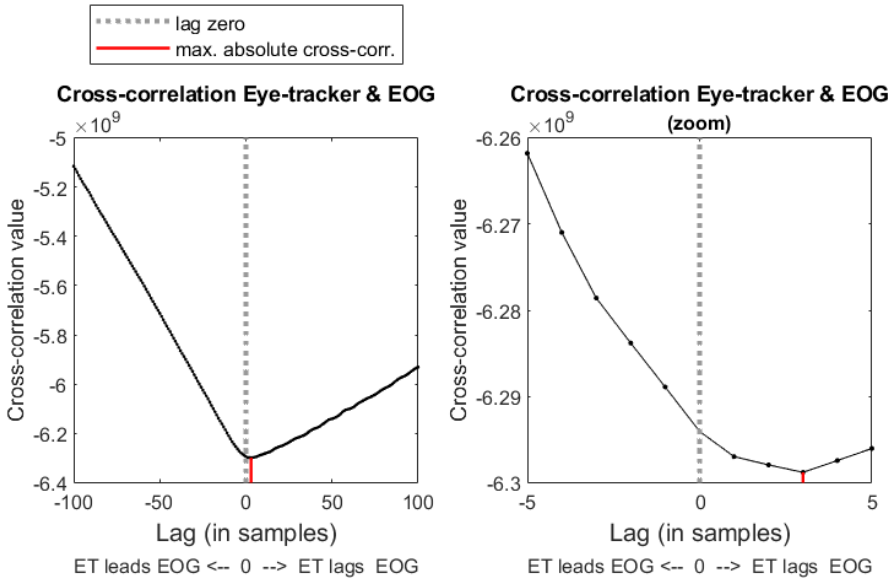


Figure 3: Synchronization quality of subject 9 (left: original plot, right: zoomed in at the peak).

2.3 Eye-tracking data analysis

2.3.1 Eye-tracking data preprocessing

The eye-tracking data (IDF file) were converted via iTools to text files. Following the protocol of Ward et al. (2021), we detected and extracted fixation data if the eye fixation had a minimum duration of 50ms and a peak velocity of 40o/s. We also removed bad eye fixations of which the locations were outside of the screen or less than 1 ms. After preliminary processing, we further transformed and analyzed the fixation data in MATLAB (R2021a, The Mathworks Inc., Natick, MA, 2021).

After manual inspection, we found systematic drifts to different degrees during recording. fig. 4 is a scatter plot of fixations during the baseline period (the fixation cross) for the example subject 9. As seen in the figure, the focus point of fixations was to the left of the center point (the fixation cross). To correct this drift, we calculated the median location of fixations during the baseline period for each consecutive 10 trials and computed its distance to the center location (the fixation cross). The difference between median location of fixation and the central location is then used to adjust the eye movement coordinates for each trial.

2.3.2 Eye-movement-derived conditions

Due to the nature of passive viewing, direct responses from subjects were not available. Therefore, defining trials and behavioral analysis is challenging. In previous studies, Ward et al. (2021) examined predictive indications in eye movements across the three steps in the videos and found more anticipatory behavior in later steps (e.g., longer duration of fixations during the predictive time window). Ergo, Ward et al. (2021) used the three action steps as the equivalent of trial conditions in EEG analysis.

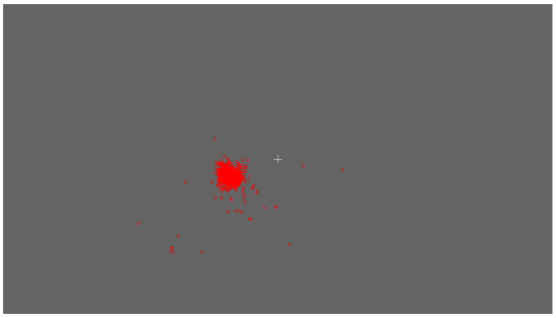


Figure 4: Locations of fixations during the baseline period for subject 9.

In the current study, we created prediction-related conditions and labeled trials based on the accuracy and occurrence of *predictive fixations*. Here, we defined the fixations during the predictive time window as *predictive fixations*. First, we added a field `pred_flag` and labelled it as 1 for the steps with at least one predictive fixation and otherwise as 0. The `pred_flag` field was used as an indication of predictive behavior.

Further, we examined the accuracy of predictive fixations. We calculated the distance between median locations and the center locations of the four AOIs respectively. The closest target to the median location(s) of the predictive fixation(s) was classified as the *predictive target*. Here, we used a distance measurement, instead of rigidly defining the predictive target as the AOI within which the focus of predictive fixations lied. During the predictive time window, the focus of fixation could jump back and forth between the current object and the predictive target, and happened in the bridge area between two adjacent targets. Figure 5 demonstrated the routes of predictive fixations (red dots) during observing one stimulus video for subject 9. Therefore, our definition of the predictive target is more tolerant for such unexpected eye movements and jitters during recording. Accordingly, we added two fields to reflect the accuracy of predictive fixations:

- `flag_correct` : 1 when the predictive target is the target of the current step, otherwise 0;
- `flag2_correct` : 1 when the predictive target is the target of the current or the previous step, otherwise 0;

Figure 5: Locations of fixations during predictive windows for subject 9.

2.3.3 Eye-tracking descriptive and statistical analysis

Ward et al. (2021) investigated three eye-movement related parameters: Predictive Looking Time, Predictive Gaze Onset and Predictive Count Ratio. Based on previous results, the effect of action steps was significant in all three measurements while the group effect was only significant in Predictive Gaze Onset.

In the current study, we based our behavioral analysis on the three action steps and calculated the following measurements for each subject in the autistic and non-autistic groups. Figure 6 show the relationship among trials with different definitions.

- `norm_sumDuration` : sum duration of predictive fixations divided by the length of corresponding predictive time window;
- `pct_pred` : number of action steps with predictive fixations divided by the total number of steps;
- `cnt_pred` : number of action steps with predictive fixations;
- `pct_correct` : number of correctly predictive steps (`flag_correct`) divided by `cnt_pred`;
- `pct_correct2` : number of correctly predictive steps (`flag2_correct`) divided by `cnt_pred`;
- `pct_correct_total` : number of correctly predictive steps (`flag_correct`) divided by the total number of steps;
- `pct_correct2_total` : number of correctly predictive steps (`flag2_correct`) divided by the total number of steps;

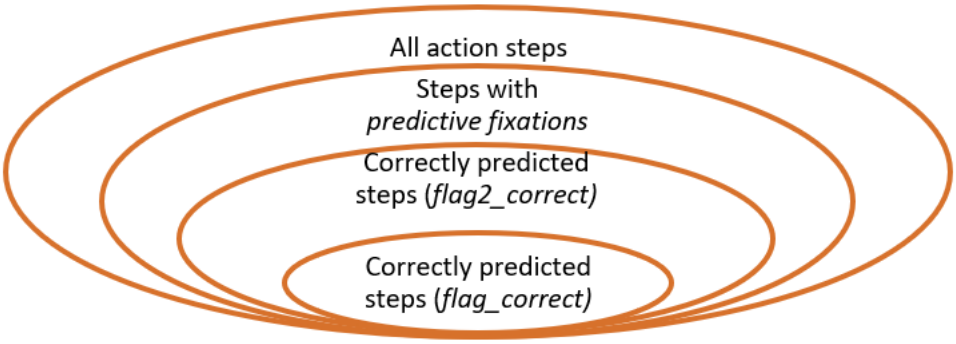


Figure 6: Venn diagram.

We aggregated the above measurements and produced a descriptive statistic table for the behavioral analysis. Further, we conducted Bayesian two-sample t-tests and Bayesian paired-sample t-tests to examine the between-group and across-step differences for the parameters. The Bayesian t-tests provided statistics to quantify the amount of evidence for the hypotheses and was conducted in RStudio (1.3.1073; RStudio Team, 2020) and Bolstad package (Curran & Bolstad, 2020). We expect to see a higher accuracy in later steps than earlier steps for both groups and different patterns of prediction and accuracy between the autistic and non-autistic group.

2.4 Participants selection

Among the 56 subjects that participated in the data collection, 1 subject was excluded due to excessive noise in EEG (Ward et al., 2021) and 8 subjects were excluded due to technical problems during recording or missing eye-tracking data. Further, we excluded 13 of the remaining 47 subjects because few eye movements were recorded during the viewing process. For these 13 subjects, more than 25% of the trials has no fixation in it. No fixations during the viewing process indicated less engagement in the videos, hence, undesirable for our analysis. This left a sample of 34 subjects.

In addition, we focused on discovering different behavioral and neural patterns of predictive behavior between the autistic group and non-autistic group. Therefore, we excluded 7 subjects that had less than 75% of the trials with predictive fixations. In the remaining 27 subjects, one subject was excluded due to bad data synchronization quality between eye-tracking and EEG data (0.6% synchronized events.).

In our final sample of 26 subjects, 10 were autistic and 16 were non-autistic. Ward et al. (2021) examined the IQ score, alexithymia level and autism symptoms. Table 1 lists the statistics and demographics of the final sample. As shown in the table, no significant difference of sample mean was found between the autistic group and the non-autistic group for age and IQ measurement. The range of IQ score is wider for the autistic group (78 – 127) than the non-autistic group (96-130). The autistic group has a significant higher level of autism symptoms as measured by Social Communication Questionnaire (SCQ). As indicated by two-sample t statistics of Bermond-Vorst Alexithymia Questionnaire (BVAQ), the difference of alexithymia level between groups is insignificant but closer to the 5% significant level.

Table 1: Participants characteristics: Mean (standard deviation)

Group	N	Gender (M: F)	Age	IQ
Autistic	10	7:3	14.94 (2.4)	110 (14.56)
Non-autistic	16	7:9	14.7 (1.73)	111.33 (8.52)
Statistic			$t(14.83) = 0.2713$, $p = 0.7899$	$t(11.35) = 0.2502$, $p = 0.8069$
Group			SCQ	BVAQ
Autistic			14.25 (7.36)	116.4 (18.35)
Non-autistic			3.4 (2.90)	106.06 (18.24)
Statistic			$t(8.18) = -4.0057$, $p = 0.0037$	$t(19.16) = -1.4008$, $p = 0.1773$

2.5 EEG data analysis

2.5.1 EEG data preprocessing

EEG data were processed using the EEGLAB toolbox (Delorme & Makeig, 2004) and MATLAB (R2021a, The Mathworks Inc., Natick, MA, 2021). Following the protocol of Ward et al. (2021), the EEG data were band-pass filtered between 1 and 45 Hz. All channels were visually inspected for major artefacts from non-neural sources. Channels were removed if they had excessive noise or

disconnected multiple times. On average, 1.1 bad channels were removed per subject. For ten subjects, less than 2 channels were removed. For two subjects, more than 5 bad channels were removed (5 and 7 respectively). Afterwards, major artefacts that influenced a substantial number of channels were manually detected and rejected before the independent component analysis (ICA). Further, we conducted ICA to remove the artefactual components of eye movement, eye blinks and muscles. On average, 6 components were removed per subject. After ICA, a second round of visual inspection and manual rejection were conducted for any remaining artefacts. Subsequently, we interpolated the data of previously rejected bad channels and re-referenced the EEG data to the average of all channels.

2.5.2 Dynamic and static segmentation

Following the protocol in Ward et al. (2021), every trial was segmented into one baseline (i.e., 1000ms after the onset of the fixation cross) and three other sections corresponding to each action step. In the current study, we implemented dynamic and statistic segmentation for action steps based on our previous work on data synchronization and eye movements analysis, as discussed above. For the static segmentation, we used the predictive time window and defined a universal TOI for all subjects. As introduced earlier, each action step had a predictive time window in which the actor moved her hand but not yet reached a target object. We assume the suspension is at its maximum during this period and the cognitive activities could be most active. Ergo, the action step segmentation was time-locked to the starting point of predictive time windows. The universal TOI were defined as 200ms before the start of the corresponding predictive time window to 800ms after. For dynamic segmentation of the action steps, we used the individual-dependent TOI and segmented trials based the predictive fixations of each subject. During the data synchronization process, we imported fixations as events in the EEG data. The segmentation is time-locked to the onset of first predictive fixation. The individual-dependent TOI were defined as 200ms before the onset to 800ms after. The EEG data in action step segments were then corrected to the temporal mean of the baseline period.

2.5.3 Time-frequency analysis

Following the protocol of Ward et al. (2021) and Braukmann et al. (2017), we applied Fast Fourier Transform to all the EEG data segments using a multi-taper frequency transformation. Specifically, Hanning tapers were used with a 2 Hz wide frequency smoothing and a fixed time window length of 0.5 s. The time frequency representations were computed and analyzed in the Fieldtrip toolbox (Oostenveld et al., 2011) and MATLAB (R2021a, The Mathworks Inc., Natick, MA, 2021). The output (i.e., power spectra) of action steps was then baseline-corrected by the average power of the fixation cross period. The baseline-correction was realized by taking the logarithm of the relative ratio between the power of the action step period and the fixation cross period. Following previous work by Ward et al. (2021), we focused on the average power spectrum in beta (15 to 25 Hz) and alpha (8 to 12 Hz) frequency bands at the channel Cz. For both dynamic and static segmentation of EEG data, we conducted a Bayesian one-tail one-sample t-test to examine whether the power spectrum was significantly different from the baseline period. Further, we set the trial conditions in the time-frequency analysis the same as trial definitions in previous eye-tracking analyses. We used the eye-movement-derived flag `pred_flag`, `flag_correct` and `flag2_correct` as conditions and investigated the between-group power differences by visualizations and Bayesian two-sample t-tests.

2.5.4 Connectivity analysis

For connectivity measurements, we calculated coherence spectra from the Fourier transform of pairs of signals. A cross-spectral density (CSD) was produced by multiplying the Fourier transform of one signal with the conjugate of the Fourier Transform of another signal. CSD is a function of frequency and considered as an equivalent to the cross-correlation in the time domain (Bastos & Schoffelen, 2016). Coherence coefficient is the normalized representation of averaged CSD between two signals at a given frequency. The coherence coefficient ranges from 0 to 1, with the higher value indicating a higher coherence between the two signals. In the current study, we examined the coherence between pairs of channels in central (C3, C4 and Cz), frontal (F3 and F4), parietal (P3, P4 and Pz), temporal (T7 and T8) and occipital (O1, O2 and Oz) regions. Figure 7 shows the display of channels in EEGLAB. Similar to the power spectrum analysis, we focused on beta (15 to 25 Hz) and alpha (8 -12 Hz) frequency bands and their connectivity between pairs of channels. Further, we investigated the coherence difference between the autistic and non-autistic groups for each pair of the above-mentioned channels. The computation is realized in the Fieldtrip toolbox (Oostenveld et al., 2011) and MATLAB (R2021a, The Mathworks Inc., Natick, MA, 2021). We also visualized the strength of connectivity in topoplots by a topoplot_connect function (Namburi, 2011) together with EEGLAB toolbox (Delorme & Makeig, 2004).

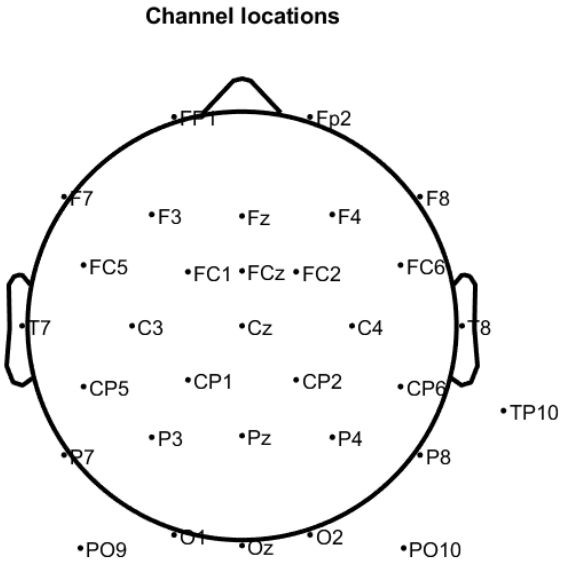


Figure 7: Channel locations.

3 Results

3.1 Eye-tracking analysis

3.1.1 Eye-tracking results

For the behavioral eye-movement analysis, we first computed the aggregated descriptive statistics in the autistic and non-autistic group. Table 2 displays the descriptive statistics of the parameters described in the section Eye-tracking descriptive and statistical analysis. The `sum Duration` metric in this table was the sum of `norm_sumDuration` parameter for each group divided by the total number of subjects in the group. As shown in the table, the ratio of trial with predictive fixations (i.e., `pct_pred`) and the sum duration of predictive fixations (i.e., `sum Duration`) were higher in the autistic group than the non-autistic group. On the contrary, the average accuracy rate (i.e., `pct_correct`, `pct_correct2`, `pct_correct2_total` and `pct_correct_total`) appeared to be higher in the non-autistic group than the autistic group.

Table 2: Eye-tracking statistics.

Group	Step	sum Dur.(s)	pct pred	pct cor.	pct cor.2	pct cor.2 tot.	pct cor. tot.
Autistic	1	918.63	87.03%	35.85%	35.85%	31.20%	31.20%
	2	905.54	87.97%	31.62%	61.32%	53.95%	27.82%
	3	891.59	87.59%	43.13%	77.25%	67.67%	37.78%
Non-autistic	1	686.05	80.14%	45.04%	45.04%	36.10%	36.10%
	2	661.57	84.11%	37.36%	70.56%	59.35%	31.43%
	3	663.97	84.46%	50.07%	84.51%	71.38%	42.29%

To quantify the between-group difference, we conducted a Bayesian two-sample t-test for the above parameters. As indicated in the Table 3, the between-group difference of prediction ratio (i.e., `pct_pred`) is significant for all three action steps, under the significance level of 1%. Bayes factors shows a strong evidence for between group differences as well. For the duration of fixation (i.e., `sumDuration`), the t-test result is not significant under the significance level of 5%, but the statistics is close to the significant threshold. Also, the Bayes factor indicates a not substantial acceptance for the null hypothesis (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 0.8$). For the accuracy measurements `pct_correct` and `pct_correct2`, the between-group difference is marginally significant with a $p = .055$ for step 1. The Bayes factor indicates the acceptance of the alternative hypothesis is not substantial (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 1.5424$). The between-group difference of `pct_correct` is also marginally significant for the step 3 with a not substantial Bayesian evidence (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 1.7052$). For accuracy measurements `pct_correct_total` and `pct_correct2_total`, the between-group differences are significant for step 1 and step 3 under significance level of 1%, and marginally significant for step 2 (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 1.3368$).

Additionally, we examined the eye movement patterns across the three action steps. As shown in the descriptive statistics, the ratios of predictive steps (i.e., `pct_pred`) were close to identical in the three action steps for the autistic group, but were different for the non-autistic group. This indicated that

Table 3: Bayesian two-sample t statistics between the autistic and non-autistic group

	sum Duration	pct pred
step1	$BF = 0.8253, t(24) = -1.4968, p = 0.1475$	$BF = 40129.36, t(24) = -7.1016, p < .001$ ***
step2	$BF = 0.8668, t(24) = -1.5432, p = 0.1359$	$BF = 16602.06, t(24) = -6.6859, p < .001$ ***
step3	$BF = 0.7852, t(24) = -1.4483, p = 0.1605$	$BF = 4161.083, t(24) = -6.0445, p < .001$ ***
	pct correct	pct correct2
step1	$BF = 1.5424, t(24) = 2.0184, p = 0.0549$ *	$BF = 1.5424, t(24) = 2.0184, p = 0.0549$ *
step2	$BF = 0.4772, t(24) = 0.8353, p = 0.4118$	$BF = 0.5145, t(24) = 0.9520, p = 0.3506$
step3	$BF = 1.7052, t(24) = 2.0918, p = 0.0472$ **	$BF = 0.7212, t(24) = 1.3621, p = 0.1858$
	pct correct total	pct correct2 total
step1	$BF = 5.4698, t(24) = -2.8359, p = 0.0091$ ***	$BF = 5.4698, t(24) = -2.8359, p = 0.0091$ ***
step2	$BF = 4.7529, t(24) = -2.7539, p = 0.011$ ***	$BF = 1.3368, t(24) = -1.9098, p = 0.0682$ *
step3	$BF = 55.9721, t(24) = -4.0464, p < .001$ ***	$BF = 8.9394, t(24) = -3.1117, p = 0.0048$ ***

Note: *** indicates significance under 1%; ** indicates significance under 5%; * indicates significance under 10%.

for autistic subjects, they asserted equal amounts of efforts for the three action steps, no matter what their inherited difficulties for action prediction are. While, for the non-autistic group, they invested less on the most difficult action step (i.e., step 1), but more on later steps. For accuracy measurements, we observed a clear across-step increasing trend for `pct_correct2` and `pct_correct2_total`. For measurements `pct_correct` and `pct_correct_total`, the general tendency is to increase with the difficulty level, except for the step 2. This could be due to misclassification for the close arrangements of objects on the table, which in turns prove the necessity to include a more tolerant accuracy measurements (i.e., `flag2_correct`).

We also conducted Bayesian paired-sample t-tests for the above measurements, and tested the equality of sample mean for two adjacent steps (i.e., step 1 with step 2, and step 2 with step 3), for the autistic and non-autistic group respectively. The results are in Table 4. Consistent with our observations in the descriptive statistics table, the `pct_pred` between step 1 and step2 is marginally significantly different for the non-autistic group with a p-value of .06. However, for the autistic group, the null hypotheses of equal means were accepted for both step 1 to step 2 and step 2 to step 3 comparison. The Bayes factor indicates the acceptance of the between-step differences in the non-autistic group is not substantial (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 1.5761$). Still, we observed a larger Bayes factor in the non-autistic group for step 1 and step 2 comparison. For the accuracy measurements `pct_correct2` and `pct_correct_total`, the across-steps differences are significant for both autistic and non-autistic groups.

3.1.2 Eye-tracking discussion

These results contradicted the discovery in Ward et al. (2021), which revealed strong evidence for an absence of group effects in Predictive Looking Time and Predictive Count Ratio. In our Bayesian two-sample t-test results, the duration of predictive fixations (i.e., `sumDuration`, an equivalent of Predictive Looking Time), though insignificant under 5%, did not have substantial evidence against the alternative hypothesis (the likelihood ratio of null hypothesis to the alternative hypothesis is 1

Table 4: Bayesian paired-sample t statistics across action steps.

Group	Step	sum Duration	pct pred
autistic	1-2	$BF = 0.4253, t(9) = 0.4378, p = 0.6718$	$BF = 0.4463, t(9) = -0.5731, p = 0.5806$
	2-3	$BF = 0.4146, t(9) = 0.3457, p = 0.7375$	$BF = 0.4031, t(9) = 0.2004, p = 0.8456$
non	1-2	$BF = 0.5146, t(15) = 1.0565, p = 0.3075$	$BF = 1.5761, t(15) = -2.0426, p = 0.0591$ *
autistic	2-3	$BF = 0.3367, t(15) = -0.0659, p = 0.9483$	$BF = 0.3407, t(15) = -0.1873, p = 0.8539$
Group	Step	pct correct	pct correct2
autistic	1-2	$BF = 0.5493, t(9) = 0.9620, p = 0.3612$	$BF = 100.2734, t(9) = -4.5933, p = 0.0013$ ***
	2-3	$BF = 3.961, t(9) = -2.7032, p = 0.0243$ **	$BF = 15.4356, t(9) = -3.5405, p = 0.0063$ ***
non	1-2	$BF = 1.9119, t(15) = 2.1722, p = 0.0463$ **	$BF = 172775.2, t(15) = -7.1911, p < .001$ ***
autistic	2-3	$BF = 16.4714, t(15) = -3.3503, p = 0.0044$ ***	$BF = 23741.93, t(15) = -6.3912, p < .001$ ***
Group	Step	pct correct total	pct correct2 total
autistic	1-2	$BF = 50.3101, t(9) = -4.2115, p = 0.0023$ ***	$BF = 0.5033, t(9) = 0.8201, p = 0.4334$
	2-3	$BF = 3.4446, t(9) = -2.6099, p = 0.0283$ **	$BF = 2.3527, t(9) = -2.3441, p = 0.0437$ **
non	1-2	$BF = 177076.2, t(15) = -7.2011, p < .001$ ***	$BF = 0.8067, t(15) = 1.5238, p = 0.1484$
autistic	2-3	$BF = 894.7174, t(15) = -5.0765, p < .001$ ***	$BF = 8.9337, t(15) = -3.0486, p = 0.0081$ ***

Note: *** indicates significance under 1%; ** indicates significance under 5%; * indicates significance under 10%.

: 0.8). In addition, noticeable between-group differences were found according to the descriptive statistics. Furthermore, in Ward et al. (2021), the Predictive Looking Time was found to be longer for the non-autistic group than the autistic group, which is the opposite of our discovery. For the predictive ratio (i.e., `pct_pred`, similar to Predictive Count Ratio), the between-group difference is significant for all three action steps in our study but not in Ward et al. (2021).

The discrepancy could be caused by the different analytical populations. In the current study, we only examined the subjects who showed active predictive eye movements. During the process of participants selection, we excluded a total of 21 subjects (12 autistics, 9 non-autistics) among the 47 participants of whom both EEG and eye-tracking data are available. While in Ward et al. (2021), all available subjects were included in the final analysis.

Combining the results from these two studies, we draw an interesting conclusion. On a general level, non-autistic individuals showed more anticipatory behavior (e.g., longer Predictive Looking Time) and more active engagement in predictive fixations than the autistic individuals. However, if only looking at the subjects with active predictive eye movements, the autistic individuals showed a significantly higher amount of fixations in predicting the actions, but have a significantly lower accuracy rate. The increasing amount in prediction and the smaller portion of active subjects in the autistic group canceled each other out, resulting in insignificant between-group results when comparing at a general level.

3.2 EEG analysis

Up till now, we aspired to examine the between-group differences for three eye-movement-derived conditions across three action steps in 32 EEG channels. However, the multiple simultaneous statistical tests raised the inevitable multiple comparison problem (MCP), which would increase the false

positive rate. To reduce the MCP, we only used the `flag2_correct` condition on a selection of channels for further EEG analysis. Based on the eye-tracking analysis results, the descriptive statistics of `flag2_correct` was closest to our expectations: the accuracy rate was generally higher in the non-autistic group and increased monotonically along with the action steps.

Furthermore, in the current study, we aimed to discover the between-group difference during the action prediction. In our eye-tracking analysis, the actions labelled as incorrect predictions could be caused by either attention drift or incorrect judgement. The two causes of incorrect predictions involved in different cognitive functions and could appear differently in EEG signals. Due to the nature of passive viewing, the attention drift and lack of engagement were not controlled and could happen often. Therefore, to avoid unwanted noises, we only included EEG segments labelled as correct predictions by `flag2_correct` in our analysis.

3.2.1 Time-frequency analysis results

In this section, we investigated the mu and beta power over the central scalp areas. The mu and beta power suppression in the sensorimotor cortex is thought to be related to the motor system activation and action prediction. We conducted analyses for both static and dynamic methods of segmentation. The results from the two methods were largely similar. For the simplicity of demonstration, we only discuss results from the dynamic methods in the following section. The output from the static method are presented in the appendix.

Mu power

The mu rhythm is defined as the EEG oscillation in the frequency band 8-12 Hz recorded over the motor cortex. Following Ward et al. (2021), we focused on the mu power recorded over the Cz channel. We first examined mu power differences between viewing the action steps and the fixation cross. Based on the results from Bayesian one-sample t-tests, the mu power during action predictions were statistically different from the baseline period for both autistic and non-autistic groups . In addition, the average mu power is negative, which indicated mu power was attenuated during the action viewing, giving our baseline correction method (i.e., logarithm of the relative ratio). The observations were consistent with Ward et al. 's results. The results from Bayesian t-tests are listed in Table 5

Table 5: Bayesian one-sample statistics for power comparison with the baseline (dynamic)

	Autistic	
Step	Mu	Beta
1	$BF = 1038.91, t(9) = -5.8849, p < .001$ ***	$BF = 5.5965, t(9) = -2.9267, p = 0.0169$ **
2	$BF = 55.1592, t(9) = -4.2627, p = 0.0021$ ***	$BF = 379.8678, t(9) = -5.3262, p < .001$ ***
3	$BF = 110.6218, t(9) = -4.6474, p = 0.0012$ ***	$BF = 6269.042, t(9) = -6.9127, p < .001$ ***
	Non-autistic	
Step	Mu	Beta
1	$BF = 238.2528, t(15) = -4.5325, p < .001$ ***	$BF = 3.0821, t(15) = -2.4685, p = 0.0261$ **
2	$BF = 273.1659, t(15) = -4.5895, p < .001$ ***	$BF = 130254.8, t(15) = -7.0764, p < .001$ ***
3	$BF = 171.0444, t(15) = -4.3934, p < .001$ ***	$BF = 62109.04, t(15) = -6.7772, p < .001$ ***

Note: *** indicates significance under 1%; ** indicates significance under 5%; * indicates significance under 10%.

Further, we computed the average power spectrum for each group and each action step respectively, and subtracted the power of the autistic group from the non-autistic group. For a clean demonstration, we only displayed the difference power spectrum of action step 3, for which the accuracy rate was the highest among the three and had more EEG data available (as we only included EEG segments with the correct prediction).

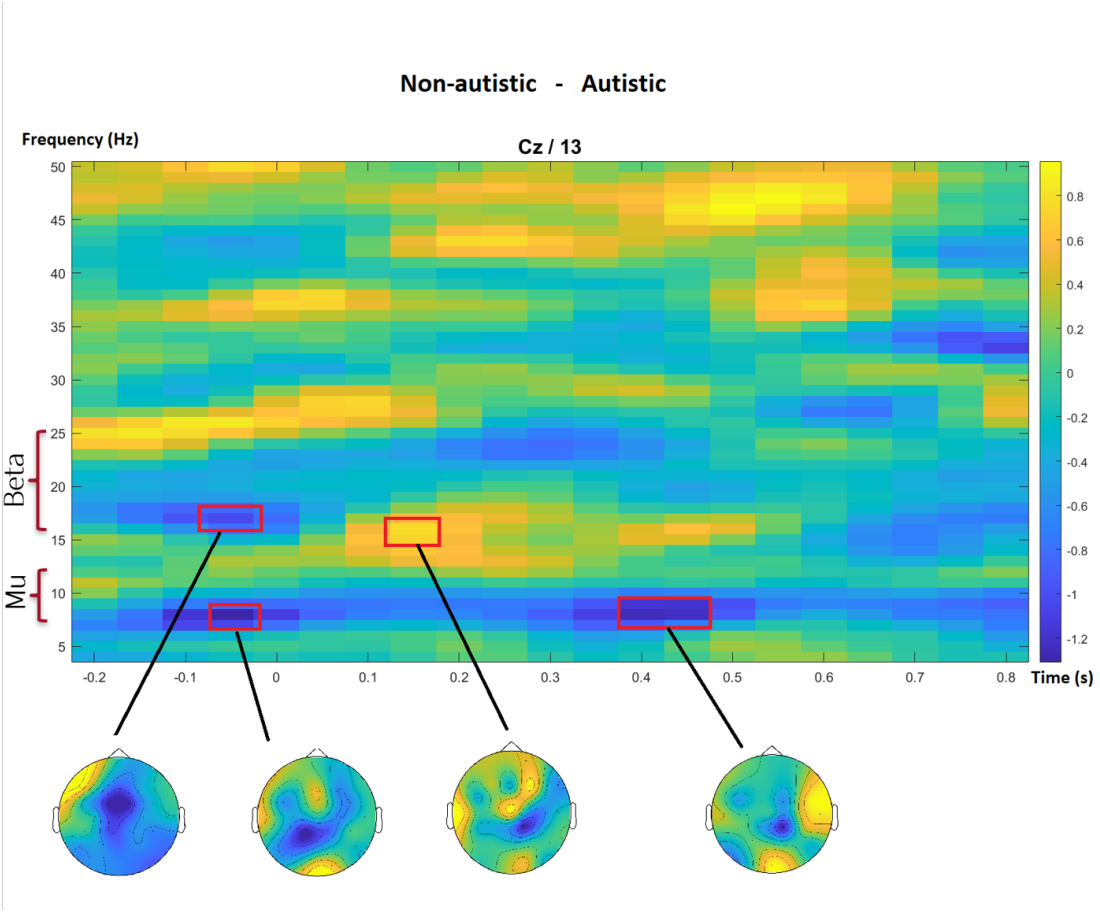


Figure 8: Power spectrum (non-autistic - autistic).

Figure 8 shows the difference power spectrum. In the figure, we marked the region with visually detectable power differences in red blocks and the topoplots of the marked region were displayed below the figure. As seen in Figure 8, the mu power appeared to be lower in the non-autistic group than the autistic group from -100 ms to 0 ms and from 380 ms to 480 ms. Because the power was negative, a lower mu power indicated more attenuation compared to the baseline period. Combined with the topoplots, we observed more mu attenuation for the non-autistic group in central-parietal regions in these two time windows. In addition, we conducted Bayesian two-sample t-tests to examine the statistical significance for such differences. The Bayesian statistics showed the group effect is not significant for mu power under the significance level of 5%. However, the statistical evidence for the null hypothesis is not substantial (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 0.6919$). Table 6 lists the Bayesian two-sample t-test results.

Figure 9 shows the distribution of mu power for each group and for three action steps. The mu power in the figure is a temporal average in frequency band 8 - 9 Hz, in which we observed the power difference in the previous power spectrum. As shown in the figure, the median mu power was higher in

the autistic group (blue) than the non-autistic (red) group, which translated to more power attenuation in the non-autistic group compared to the baseline period. This finding echoed with our previous observation from the power spectrum. We also observed a bigger variance and range (i.e., the length of the box) for the non-autistic group (red). The unequal variance could have effects on the Bayesian two-sample t-tests.

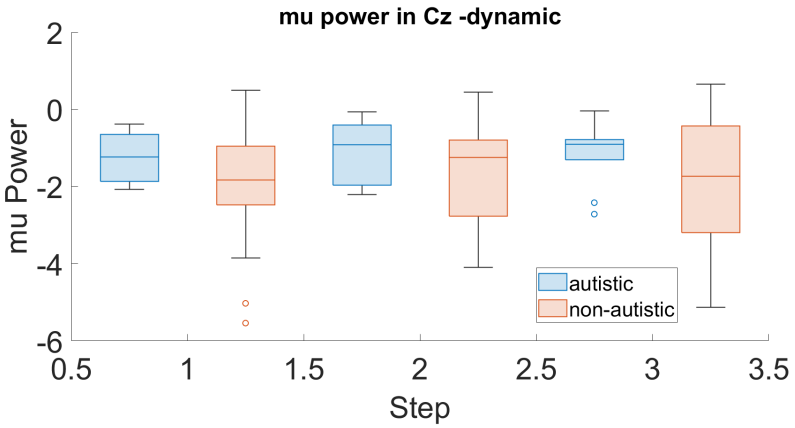


Figure 9: Boxplot for mu power in Cz channel (dynamic).

Beta power

Similar to the mu power analysis, we first studied the beta power attenuation during action predictions. The Bayesian statistics revealed that beta power (15-25 Hz) was attenuated significantly during action predictions over the Cz channel for both groups (see Table 5 for statistics). The observations were consistent with Ward et al.'s results. From the power spectrum (Figure 8), we observed a lower beta power from -100ms to 0ms and a higher beta power from 100ms to 200ms in the non-autistic group. The degree of difference in these time windows was less strong, as indicated by the moderate colors. We further conducted Bayesian two-sample t-tests for the group difference. Under significance level of 5%, the group effect is not significant for all three action steps. According to the Bayes factor, the statistical evidence for the null hypothesis is marginally substantial (the likelihood ratio of null hypothesis to the alternative hypothesis is $1:0.3938$). Figure 10 shows the distribution of beta power. The beta power in the figure is an temporal average in the frequency band 16 – 18 Hz, in which we observed a power difference in the previous power spectrum. As seen in the boxplot, the median beta power is almost identical for the two groups. We observed no visually detectable differences.

Table 6: Bayesian two-sample statistics for power comparison (dynamic)

Step	Mu	Beta
1	$BF = 0.6567, t(24) = 1.2609, p = 0.2195$	$BF = 0.3905, t(24) = -0.3851, p = 0.7036$
2	$BF = 0.5674, t(24) = 1.0859, p = 0.2883$	$BF = 0.4779, t(24) = -0.8377, p = 0.4105$
3	$BF = 0.6919, t(24) = 1.3183, p = 0.1998$	$BF = 0.3938, t(24) = 0.4140, p = 0.6825$

3.2.2 Time-frequency analysis discussion

In the time-frequency analysis, we compared the mu and beta power between the autistic group and non-autistic group. We observed more mu power attenuation in the non-autistic group over the cen-

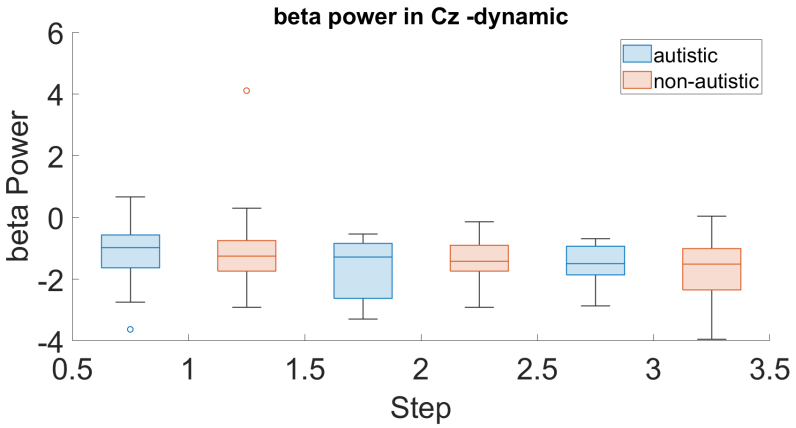


Figure 10: Boxplot for beta power in Cz channel (dynamic).

tral parietal regions from -100 ms to 0 ms and from 380 ms to 480 ms. From Bayesian statistics, the group difference is not significant, but the acceptance of the null hypothesis is not substantial. We observed similar mu power attenuation in the non-autistic group from the the boxplot. From Figure 9, the variance of the two populations appeared to be different, which could affect the power of Bayesian statistical tests and caused the insignificant results. For the beta power, the group effect is less obvious as shown in the power spectrum and the boxplot. The Bayesian statistics accept the null hypothesis strongly.

We conducted the same analysis for both static and dynamic methods of segmentation. The observations and findings were the same for the two methods. According to our methods, the onset (time 0 ms) of the static segments was marked by the beginning of the predictive time window, and the onset of dynamic segments was marked by the first fixations in the predictive window. We expect the two segments should have overlaps to different degrees.

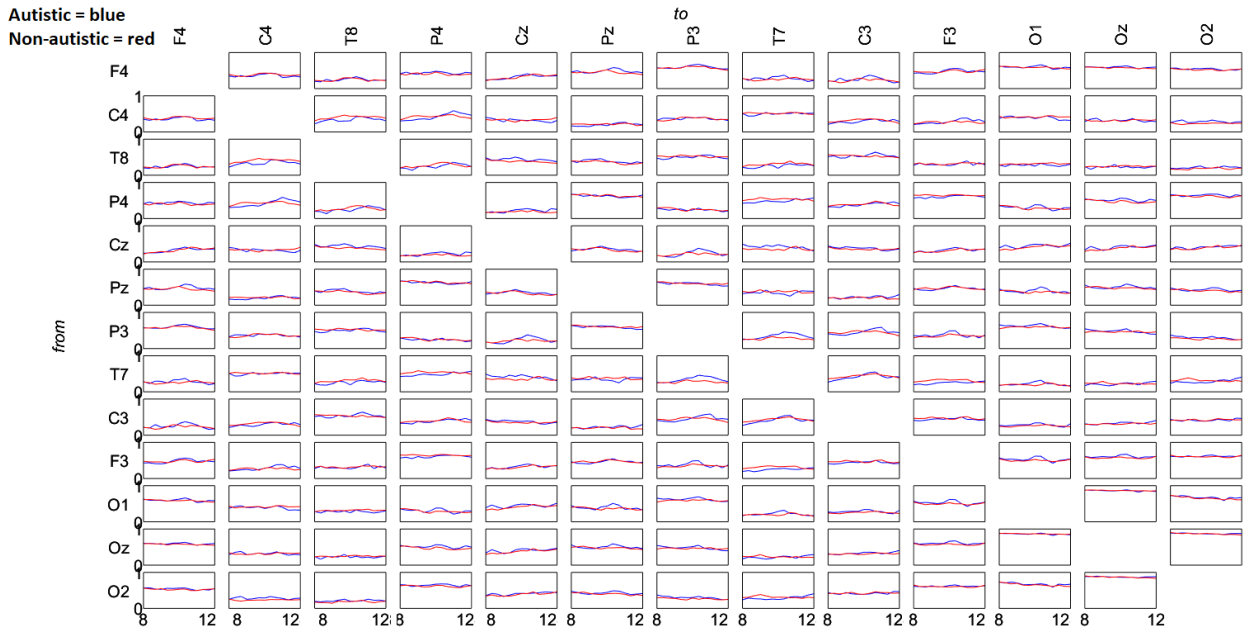
3.2.3 Connectivity analysis results

In this section, we examined the coherence between pairs of channels over central, frontal, parietal, temporal and occipital regions. Like the time-frequency analysis, we only include results from the dynamic methods in the following section. The output from the static method is presented in appendix, as the results are similar.

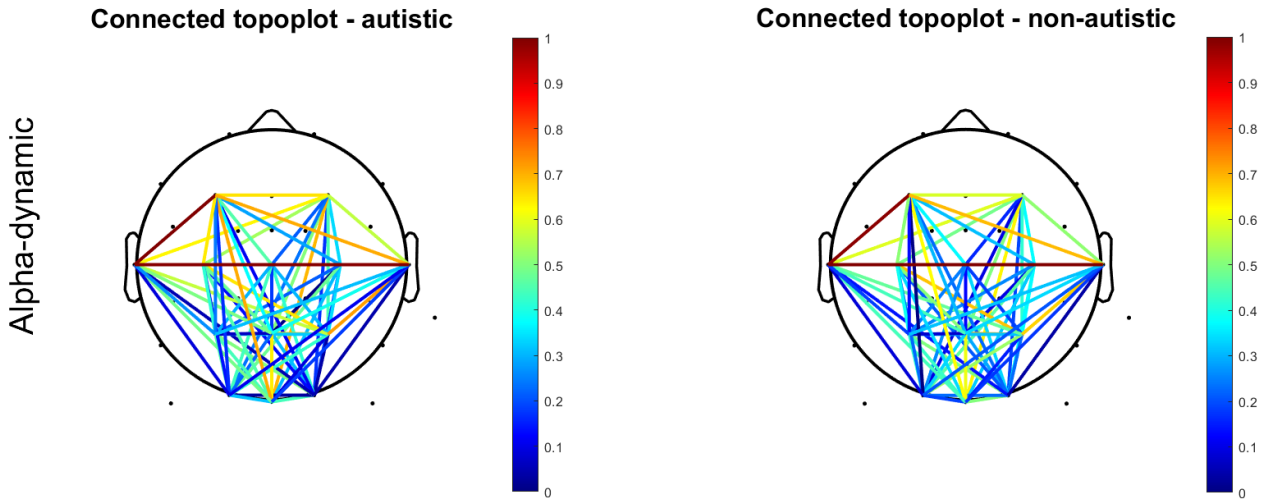
Alpha frequency band

Figure 11a is a connectivity plot between pairs of channels. Same as in the previous analysis, we displays the coherence plot of action step 3 as an example. In the figure, the x-axis represents frequencies from 8 to 12 Hz and the y-axis shows the coherence coefficient between each pair of channels. From the figure, we observed that the coherence curves of the autistic group (blue) and the non-autistic group (red) mostly overlapped. For channel pairs such as Cz-P3 and P3-T7, the autistic curve (blue) appeared to be higher than the non-autistic group at about 10 Hz. Figure 11b shows the topoplots for both groups (left: autistic; right: non-autistic) at 10 Hz, with the strength of coherence marked in color shades. From Figure 11b, we noticed a higher coherence between occipital regions (Oz) and frontal regions (F3 and F4) in the autistic group. The coherence between central regions (Cz) and parietal regions (P4) also appeared to be higher in the autistic group. We further conducted Bayesian

two-sample t-tests to examine the statistical significance of the differences. Table 7 lists the Bayesian statistics of the group difference. Under the significance level of 5%, none of the above-mentioned effect were significant.



(a) Coherence



(b) Topoplots

Figure 11: Connectivity in alpha frequency band (dynamic)

Beta frequency band

We conducted the same analysis for the beta frequency band. Figure 12a shows the coherence plot. In the figure, the x-axis represented the frequencies from 15 to 25 Hz. As seen in the figure, the coherence curves from two groups were mostly overlapping. For the channels between occipital (O1, Oz and O2) and parietal regions (Pz), the autistic group (blue) appeared to float slightly higher than the non-autistic group (red). Figure 12b shows the topoplots at 20 Hz. We saw a higher coherence in

Table 7: Bayesian statistics for coherence comparison in alpha band (dynamic)

	Channel Pair	
step	Oz - F3	Oz - F4
1	$BF = 0.4594, t(24) = 0.7704, p = 0.4486$	$BF = 0.3698, t(24) = -0.0050, p = 0.9961$
2	$BF = 0.4569, t(24) = 0.7603, p = 0.4545$	$BF = 0.4831, t(24) = 0.8558, p = 0.4006$
3	$BF = 0.7509, t(24) = 1.4037, p = 0.1732$	$BF = 0.4195, t(24) = 0.5866, p = 0.563$
step	Cz - P4	
1	$BF = 0.3699, t(24) = 0.0257, p = 0.9797$	
2	$BF = 0.4028, t(24) = 0.4825, p = 0.6338$	
3	$BF = 0.4597, t(24) = 0.7715, p = 0.4479$	

channel pairs Oz-F3 and Pz-Oz in the autistic group. Table 8 displays the Bayesian statistics for group comparison for above-mentioned channel pairs. As indicated by the statistics, the coherence between the O1 channel and Pz channel is significantly higher in the autistic group than the non-autistic group under the significance level of 5%. However, the acceptance of the between-group differences are not substantial (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 1.6665$)

Table 8: Bayesian statistics for coherence comparison in beta band (dynamic)

	Channel Pair	
step	Oz - F3	Oz - Pz
1	$BF = 0.5061, t(24) = 0.9280, p = 0.3626$	$BF = 0.4709, t(24) = 0.8132, p = 0.4241$
2	$BF = 0.3898, t(24) = 0.3783, p = 0.7085$	$BF = 0.5642, t(24) = 1.0786, p = 0.2915$
3	$BF = 0.3728, t(24) = 0.1489, p = 0.8829$	$BF = 0.7906, t(24) = 1.4551, p = 0.1586$
step	O1 - Pz	O2 - Pz
1	$BF = 0.3887, t(24) = 0.3683, p = 0.7159$	$BF = 0.37, t(24) = 0.0342, p = 0.973$
2	$BF = 1.8625, t(24) = 2.1547, p = 0.04144$ **	$BF = 0.3975, t(24) = 0.4432, p = 0.6616$
3	$BF = 1.6665, t(24) = 2.0752, p = 0.04885$ **	$BF = 0.7191, t(24) = 1.3591, p = 0.1868$

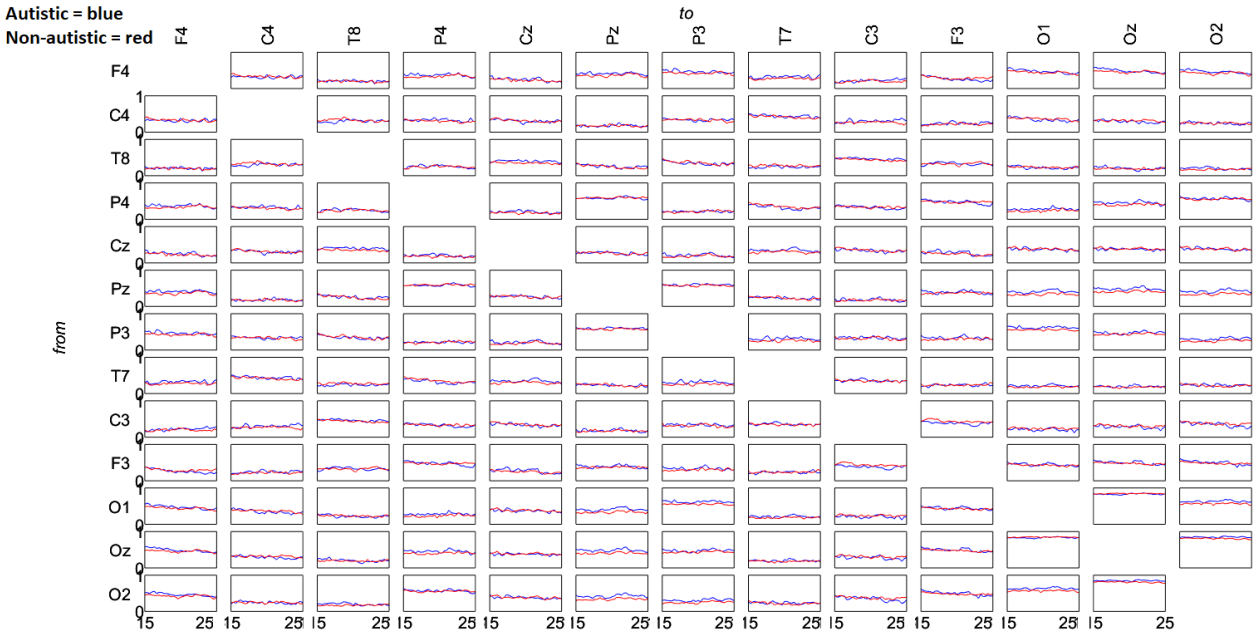
Note: ** indicates significance under 5%.

3.2.4 Connectivity discussion

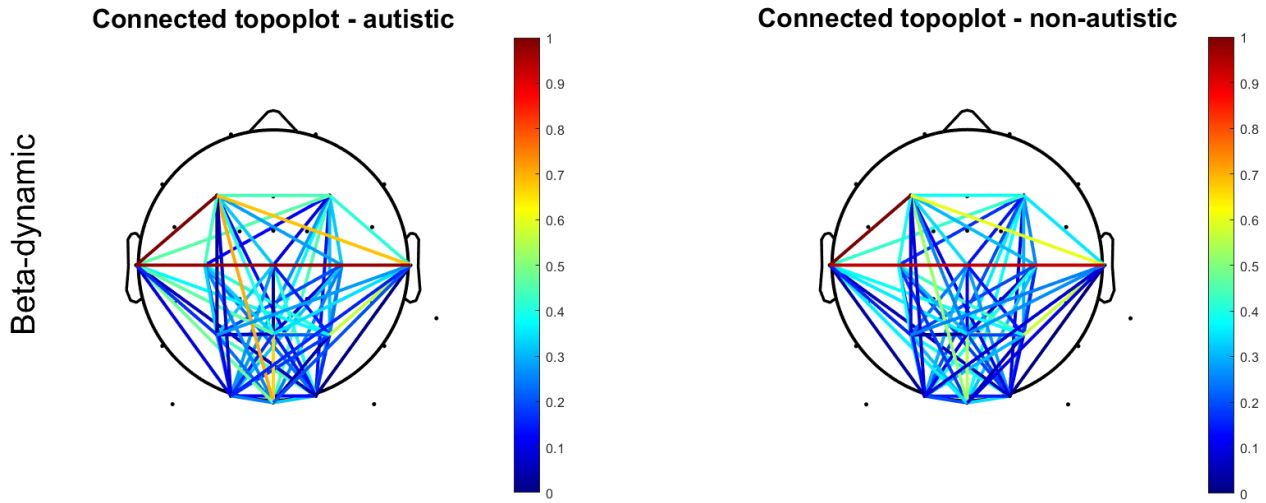
In this section, we computed and compared the coherence between pairs of electrodes for both the autistic group and non-autistic group. For the alpha frequency band (8 to 12 Hz), we observed a higher coherence between occipital and frontal regions and between central and parietal regions in the autistic group. According to the t statistics, the difference is not significant under 5%. For the beta frequency band (15-25 Hz), we observed a higher coherence between the occipital and parietal regions in the autistic group. The difference of coherence is marginally significant in O1-Pz for step 2 and step 3. For channel pair O2-Pz and Oz-Pz, the acceptance of the null hypothesis is not strong for step 3 (the likelihood ratio of null hypothesis to the alternative hypothesis is $1 : 0.7$).

We also noticed an increasing trend of Bayesian factors along with steps. In other words, there were less statistical evidence for the null hypothesis in later steps. In the analysis, we only included the EEG segments in which the eye-movement-derived metrics indicated correct prediction. Therefore, the inherent difficulty levels in different steps should not be a key trigger for differences in cognitive

activities. Instead, the increasing trend along with the action steps could be caused by the amount of information available in the corresponding step. The accuracy rate is higher in step 3 (84.51% for non-autistic and 77.25% for autistic) than step1 (35.85% for autistic, 45.04% for non-autistic) and step2 (61.32%for autistic, 70.56%for non-autistic). Consequently, we had more data for the analysis in later steps than the previous steps. The richness of information could be the cause for a higher Bayesian factor in step 3.



(a) Coherence



(b) Topoplots

Figure 12: Connectivity in beta frequency band (dynamic)

4 General Discussion

In the current thesis, we studied action prediction in autistic individuals from both eye-tracking and EEG aspects, as compared to the non-autistic group. We first synchronized the eye-tracking and EEG recordings and aligned the timestamp for stimulus in the two datasets. Subsequently, we computed the prediction and accuracy measurements for action prediction based on the coordinates of participants' fixations and the given AOIs. The results from eye-tracking analysis were then used for setting the individual-dependent EEG segments and as the condition of the EEG analysis.

The results from eye-tracking analysis showed significant between-group differences in accuracy and prediction measurements. In addition, the accuracy rate is generally higher in the non-autistic than the autistic group, which is in consistent with our assumptions. On the contrary, the amount of predictive fixations were significantly higher in the autistic group than the non-autistic group. One possible reason for this conflict is that autistic individuals could have put more efforts into prediction but had worse performance. However, we should not ignore the fact that most autistic individuals have unusual eye movements. According to DSM 5 manual, abnormalities in eye contact is listed as an example for deficits in nonverbal social-communicative behaviors in autism.

There are a large body of studies on eye-tracking and gaze patterns of autism (Frazier et al., 2017; Camero et al., 2021; Cañigueral & Hamilton, 2019; Guillon et al., 2014; Neumann et al., 2006). A meta-analysis of gaze difference between the autistic individuals and non-autistic individuals showed that the autistic individuals had greater attention (e.g., more fixation counts) to non-social cues and less visual attentions to eyes and faces (Frazier et al., 2017). However, most of the studies had limited focus and mostly used the non-social objects together with social facial expressions. For example, in a linguistic interaction task, researchers found that the autistic group had significantly more fixation counts and longer fixation duration for the object stimuli (Camero et al., 2021). In that study, a pseudo-object and a talking female face appeared on the screen and competed for the participants' visual attention. While in Ward et al.'s experiments, the face of the actor were occluded and the object of interactions had social meanings. Therefore, the results from the current gaze studies might not be directly transferable to our study. Nevertheless, we should mention that the longer duration and higher amount of predictive fixations could also be a result of the unusual eye movements of the autistic individuals and do not correlate with action predictions.

The results from our study and Ward et al.'s study are different. In Ward et al.'s study, she and colleagues did not found group effects in the duration and counts of predictive fixations. They also observed a longer duration of predictive fixation in the non-autistic group, which was contradictory to our results. As explained in the Result section, this might be caused by our participants selection and trial selection. In our analysis, we only included the subjects with active predictions and trials with correct predictions. While in Ward et.al.'s study, all the available participants and trials were included.

For the EEG analysis, we found more attenuation in mu power in central-parietal regions for the non-autistic group than the autistic group, which is also consistent with our assumptions. We also observed a higher coherence between the occipital and parietal regions for the autistic group in the beta frequency band. Through Bayesian t-tests, many of the above-mentioned difference were not significant under the significance level of 5%. However, the results can only accept the null hypothesis weakly. We also noticed an interesting increasing trend across the steps, which could be a result of increasing amount of available information in later steps. During the trial selection process, we excluded a large

proportion of trials (i.e., 60% for step1, 35% for step2, 20% for step3). One possibility is that we did not find significant statistical test results with the remaining data from earlier steps (especially for step 1). Therefore, we suspected the weak insignificance could be caused by the lack of data. Apart from that, we observed that the variance of mu and beta power were also different between subjects, which could have a negative effect on the credibility of Bayesian two sample t-test results.

Another important factor is that we excluded about half of the subjects during our participants selection process, with 10 autistic and 16 non-autistic subjects in our final sample. However, researchers suggest to have over 50 participants for a simple comparison of two within-participants conditions (Brysbaert, 2019). Because our analysis is based on previous studies, it is difficult for us to change the experiment setting and acquire new data. For future work, we would plan to include more participants and make the final subjects population more balanced. We would also insert catch questions during the action observation to eliminate the attention drift and reduce the number of trials to exclude. Hopefully, we would be able to derive substantial conclusions by then.

In the current thesis, we used two methods for EEG segmentation: universal (i.e., static) and individual-dependent (i.e., dynamic). Though the conceptualization for setting a dynamic segment is logically understandable, we did not detect differences between the results from the two segmentations. In the original study for combined EEG and eye-tracking data analysis, Dimigen et al. used saccades and fixations together with the event-related potential (ERP), which requires a higher temporal precision for segmentation. In our analysis, we also examined the Fixation-related potentials (FRP) (Baccino & Manunta, 2005), however, we observed no systematical change of potential from our data. The experimental setting is not suitable for ERP analysis due to its lack of interactive and active tasks. As an alternative, we analyzed the brain oscillations under dynamic segmentation. However, because of its calculation, the time-frequency analysis does not require an accurate temporal segment for EEG data. As a result, the two methods (static and dynamic) yielded similar patterns. For future work, we could redesign the experiment so that it is easier to incur ERP activities. The analytical framework of our study would be helpful by then.

In the current thesis, we examined the eye-tracking and neural patterns of autism. We re-analyzed the eye-tracking and EEG data from Ward et al. (2021) and found significant between-group differences in fixation patterns and non-substantial rejection of the between-group differences in brain oscillation and connectivity. On the contrary, the original study found strong evidence for the absence of group effect on the same dataset. The reason for such different results is that we only include the participants with active predictive fixations. The findings echo with the well-known heterogeneity in autism (Geschwind, 2009; Georgiades et al., 2012; Lenroot & Yeung, 2013). Participants with autism could have great individual differences. In our case, we select a subgroup of autistic individuals with a higher tendency for predictive fixations. The between-group difference in predictive fixations exist in this subgroup. When examining at the general level, several heterogeneous subgroups were mixed into one population, and possibly obscure the significance of each group effect. For future work, we would pre-select the analytical populations and ensure the homogeneity within each group.

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Appendices

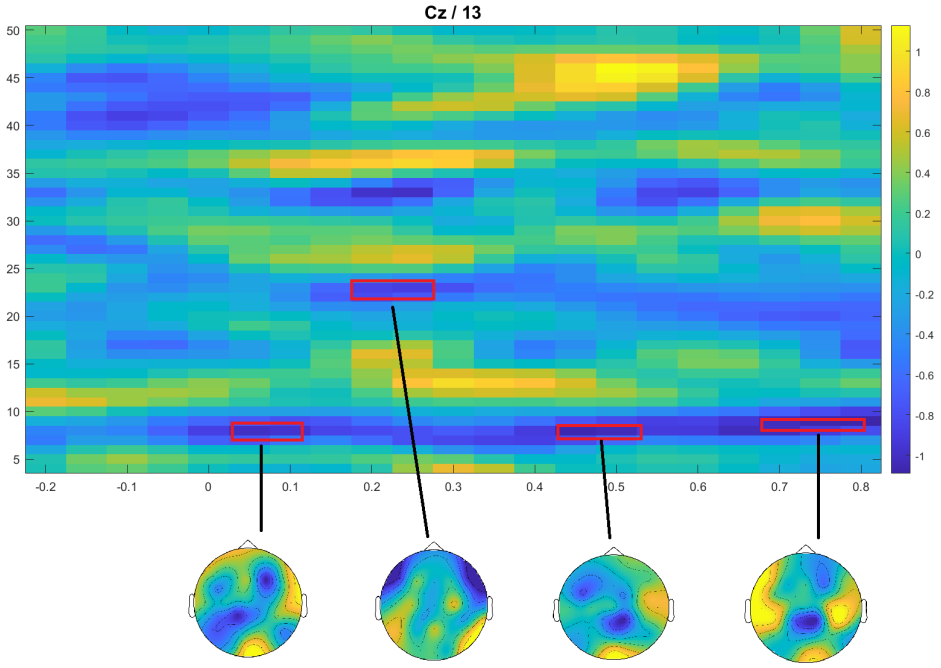


Figure 13: Power spectrum static (non-autistic - autistic).

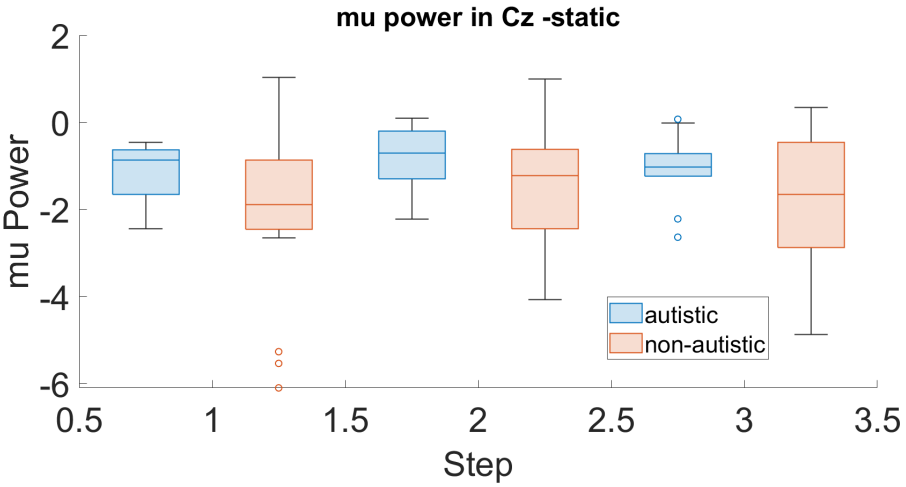


Figure 14: Boxplot for mu power in Cz channel (static).

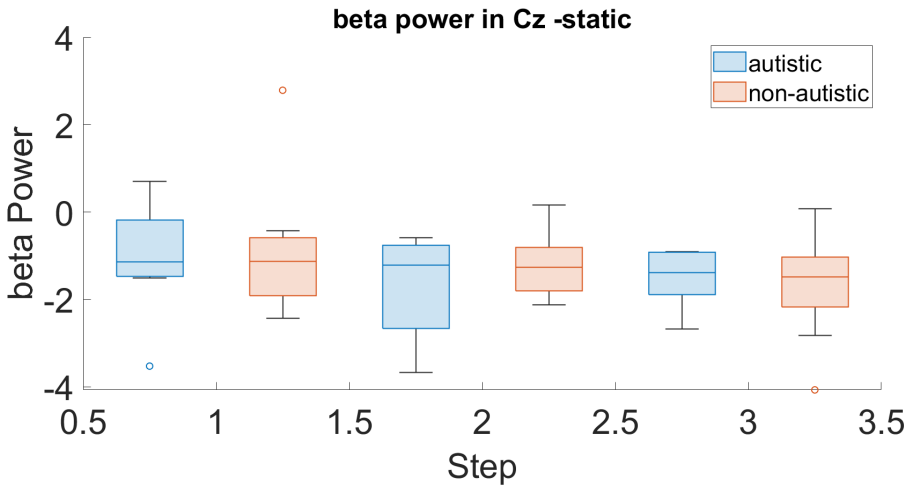


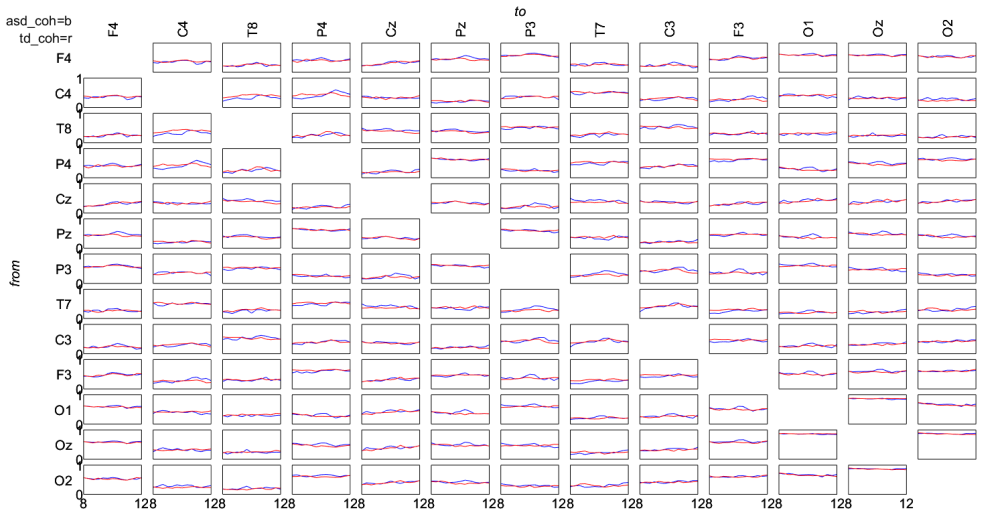
Figure 15: Boxplot for beta power in Cz channel (static).

Table 9: Two-sample Bayesian statistics for power comparison (static)

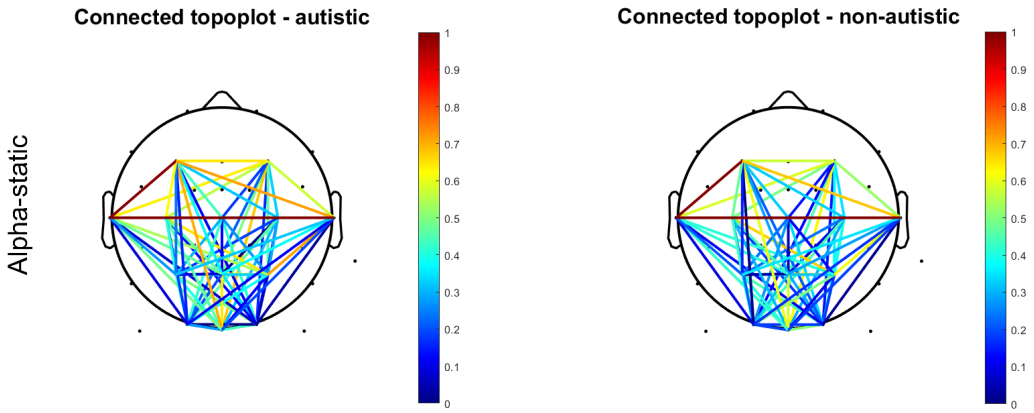
Step	Mu	Beta
1	$BF = 0.6930, t(24) = 1.32, p = 0.1993$	$BF = 0.3714, t(24) = 0.10747, p = 0.9153$
2	$BF = 0.6420, t(24) = 1.2353, p = 0.2287$	$BF = 0.6765, t(24) = -1.2939, p = 0.208$
3	$BF = 0.7086, t(24) = 1.3437, p = 0.1916$	$BF = 0.3892, t(24) = 0.37255, p = 0.7128$

Table 10: One-sample Bayesian statistics for power comparison with the baseline (static)

	Autistic	
Step	Mu	Beta
1	$BF = 424.8667, t(9) = -5.388, p < .001$	$BF = 4.1165, t(9) = -2.7286, p = 0.0233$
2	$BF = 15.5233, t(9) = -3.5438, p = 0.0063$	$BF = 171.3883, t(9) = -4.8882, p < .001$
3	$BF = 41.6765, t(9) = -4.1064, p = 0.0027$	$BF = 21918.44, t(9) = -7.6615, p < .001$
	Non-autistic	
Step	Mu	Beta
1	$BF = 88.8602, t(15) = -4.1138, p < .001$	$BF = 22.2955, t(15) = -3.4937, p = 0.0033$
2	$BF = 153.2264, t(15) = -4.3469, p < .001$	$BF = 121050.5, t(15) = -7.0467, p < .001$
3	$BF = 248.9316, t(15) = -4.5508, p < .001$	$BF = 35891.24, t(15) = -6.5568, p < .001$

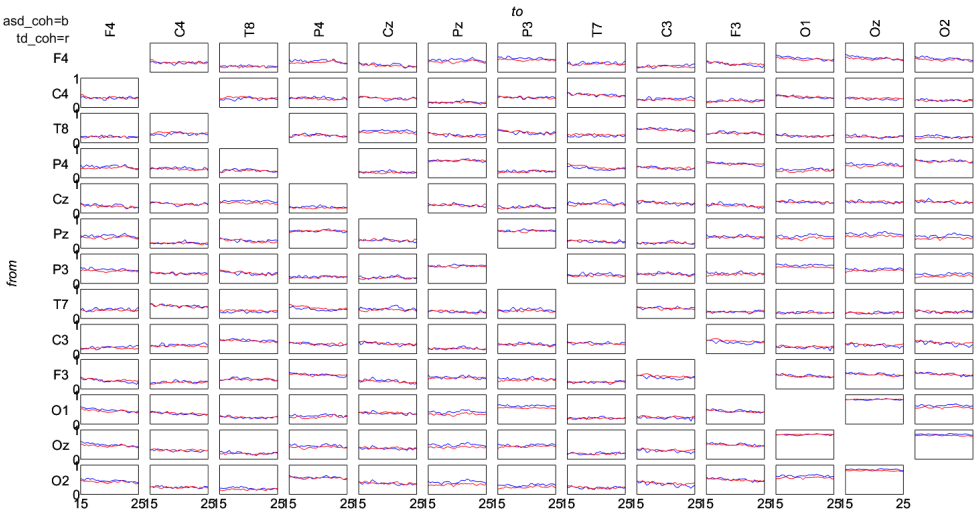


(a) Coherence

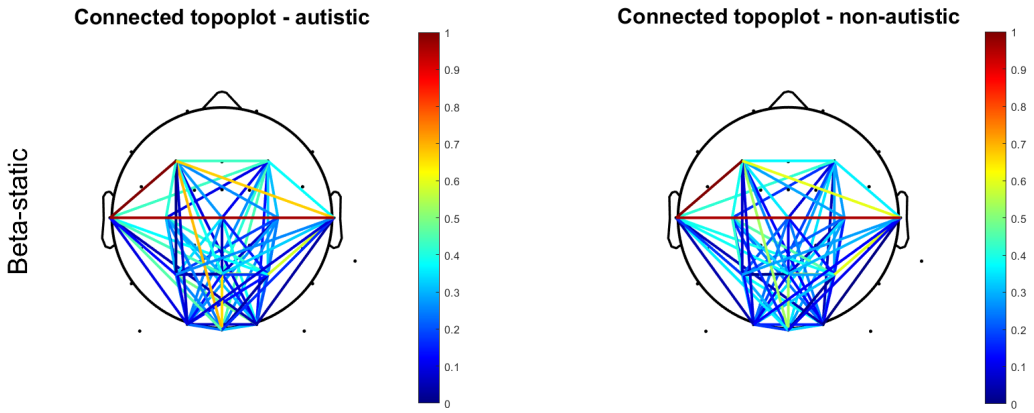


(b) Topoplots

Figure 16: Connectivity in mu frequency band (static)



(a) Coherence



(b) Topoplots

Figure 17: Connectivity in beta frequency band (static)