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Relationship Between Resting-State Frontal Alpha Asymmetry and Autism Characteristics Among Neurotypically Developing and Autistic Children and Adolescents

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Abstract

Autism is a heterogenous neurodevelopmental diagnosis associated with deficits in social communication and the presence of repetitive behaviors and sensory differences. While diagnostic criteria are behavioral, these behaviors are thought to arise from atypicalities in the brain. While many studies investigating autism have focused on understanding the neural processes underlying task performance, few have focused on understanding the brain at rest. This is critical, however, as the state of the brain before a stimulus is presented (i.e. its resting state) impacts how it responds to incoming information. Frontal alpha asymmetry, the comparison between alpha frequency power in the left and right frontal lobes, is one measure for assessing the resting-state of the brain. While several studies have investigated the relationship between frontal alpha asymmetry and autistic traits in those with an autism diagnosis, little research has examined the relationship between frontal alpha asymmetry and autistic traits regardless of diagnostic status. Thus, the purpose of this study was to examine the relationship between resting-state frontal alpha asymmetry and sensory seeking behaviors, social skills, attention to detail, and visual analytic skills among neurotypically developing and autistic children and adolescents. Results demonstrated no significant correlations between frontal alpha asymmetry and these autism characteristics. Bayesian analysis also failed to provide sufficient support in favor of either the null or alternative hypothesis when comparing the strength of these correlations between groups. Given the inconclusive nature of these results, future directions for this study, as well as the field of frontal alpha asymmetry EEG research, are discussed.

Keywords: frontal alpha asymmetry, autism spectrum disorder, autism traits

Relationship Between Resting-State Frontal Alpha Asymmetry and Autism Characteristics Among Neurotypically Developing and Autistic Children and Adolescents

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Contents

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Autism is a neurodevelopmental diagnosis that has large heterogeneity in its presentation (American Psychiatric Association, 2013). Difficulties with social communication and interaction as well as restricted and repetitive behaviors or interests are a few of the more common characteristics that are seen within autistic individuals (American Psychiatric Association, 2013). According to data collected by The Autism and Developmental Disabilities Monitoring Network, the prevalence rate of autism in children 8 years of age is approximately 1 in 36 (Maenner et al., 2023) resulting in it being one of the most common developmental disorders children are diagnosed with in the United States (Zablotsky et al., 2019). Due to autism still being poorly understood from both a genetic and behavioral perspective (Geschwind, 2008; Viding & Blakemore, 2007), the study of the underlying neurophysiology that may give rise to autism characteristics has provided new avenues for understanding autism.

Studies involving autism have frequently sought to investigate the underlying neurophysiology that differentiates autism from neurotypical development (Frith, 2003; Gliga et al., 2014; McPartland et al., 2011; Minshew & Williams, 2007). However, how these physiological differences map onto specific traits and characteristics in a more dimensional approach across diagnostic status has been understudied. This area of research is important because generating an understanding of the physiological processes that underly certain characteristics could provide us with valuable information about how trait heterogeneity in autism and neurotypical development emerges (Eigsti & Shapiro, 2003; Luckhardt et al., 2014). Given that difficulties with social communication and restricted and repetitive behaviors are normally distributed within the non-autistic population (Constantino & Todd, 2003; Ronald & Hoekstra, 2011; Skuse et al., 2005), understanding the neurological underpinnings of these characteristics would

be valuable for informing early intervention in both neurotypical and neuroatypical individuals alike. Should neurophysiological indicators that map on to social communication deficits or restricted and repetitive behaviors be found, this might allow for the provision of supports earlier on in development, ideally decreasing the impact that these characteristics may have later on.

Understanding Electroencephalography

One of the more common methods to studying neurophysiology in humans is electroencephalography (EEG) (Li et al., 2020). At its core EEG is a method for measuring the brain's electrical activity recorded at the scalp. EEG records postsynaptic potentials, which emerge after neurotransmitters bind to receptors in the postsynaptic membrane (Cohen, 2014; Luck, 2014; Xia & Hu, 2019). These postsynaptic potentials generate electric fields surrounding the neurons, and, when enough neurons in the same area are activated at the same time, they collectively generate a large enough electric field to be registered by the EEG recording device (Li et al., 2020). This differs from other common neuroimaging techniques such as functional magnetic resonance imaging (fMRI), which measures metabolic changes in the brain (Li et al., 2020). Given their different methods for measuring neural activity there exists several trade-offs between both methods. Most importantly for this study, however, is that since EEG directly measures the electrical activity of neurons, as opposed to measuring metabolic changes (Li et al., 2020), the recorded signal can be broken down into specific frequency bands such as the alpha frequency band that appears to be correlated with various autism characteristics (Burnette et al., 2011; Damiano-Goodwin et al., 2018; Simon et al., 2017; Sutton et al., 2005). Furthermore, due to the non-invasive and relatively inexpensive nature of EEG (Li et al., 2020) it is a far more accessible neuroimaging technique than fMRI, lending itself to being a more reasonable strategy in the future for early detection of autism characteristics.

Resting-State vs Task-Related EEG Research

The field of EEG research can be categorized into resting-state research, which measures neural activity when no specific task or stimulus is imposed, and task-related research, which measures neural

activity that is evoked by a specific task or stimulus (Li et al., 2020). Within the field of task-related EEG research, the EEG profiles of autistic and neurotypical individuals differ during a variety of cognitive processes (Jaime et al., 2016; Milne et al., 2009; Russo et al., 2009). However, to better understand how these differences emerge it can be important to evaluate any differences that exist between these two populations when they are not actively engaged in any specific task (Li et al., 2020). This is because during task-related research only neural responses time-locked to key events (also known as event-related potentials) are studied while most other spontaneous activity is considered background noise (Fox et al., 2006; Fox et al., 2007; Makeig et al., 2004). This background noise, however, can actually be impactful to the task-related EEG recording. For example, pre-stimulus EEG activity has been shown to predict event-related potential for both motor response (Mazaheri et al., 2009) and visual stimuli (Gruber et al., 2005). This implies that when external sensory information enters the brain it interacts with rather than determines the brain state. Thus, to fully understand how it is responding when external stimuli are presented, one must consider the resting state of the brain. If the neural activity between neurotypical and autistic individuals is fundamentally different at rest, it is possible that these differences help explain why brain activity during more active processes differs between these two populations. Essentially, without first understanding the differences that exist between neurotypical and autistic individuals at rest, it can be difficult to interpret the differences that emerge during active engagement (Wang et al., 2013).

EEG Frequency Bands

One way of characterizing EEG data is by breaking the overall waveform into groups of frequency ranges (Wang et al., 2013). Within EEG literature, the frequency ranges are usually defined as delta (1-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), beta (12-30 Hz), and gamma (>30 Hz) (Buskila et al., 2019; Khurana et al., 2018). Each of these frequencies have been linked to certain physiological processes. The delta frequency has been most commonly associated with deep sleep, the theta frequency has been most commonly associated with memory processes, the alpha frequency has been most

commonly associated with relaxed awake individuals (i.e. the resting state of the brain), the beta frequency has been most commonly associated with alertness and task engagement, and the gamma frequency has been most commonly associated with working memory matching and early sensory responses (Buskila et al., 2019; Khurana et al., 2018; Wang et al., 2013).

Frontal Alpha Asymmetry

Since alpha waves have been most frequently associated with the relaxed and awake brain, it is the frequency that has been of primary interest in past research that has sought to examine differences between neurotypical and autistic individuals at rest (Burnette et al., 2011; Fox et al., 1994; Fox et al., 2001; Gabard-Durnman et al., 2015; Schiltz et al., 2018; Sutton et al., 2005). While there are multiple ways to think about or measure alpha, frontal alpha asymmetry, the difference in alpha power (μV^2) between left and right frontal regions, has gained the most traction in autism research (Burnette et al., 2011; Gabard-Durnman et al., 2015; Schiltz et al., 2018; Sutton et al., 2005). This is because the trajectories of frontal alpha asymmetry during development differ between autistic and neurotypical children which has led to the hypothesis that alpha asymmetry may serve as a marker for cortical development (Fox et al., 1994; Fox et al., 2001). Additionally, greater right frontal alpha asymmetry has been correlated with social deficits and increased visual analytic skills among autistic children and their siblings (Burnette et al., 2011; Sutton et al., 2005). When including neurotypical individuals in these analyses, however, the results have been less consistent with some research indicating that neurotypical individuals tend to have greater left frontal alpha asymmetry than autistic individuals (Gabard-Durnman et al., 2015; Schiltz et al., 2018) and some research indicating that neurotypical individuals tend to have greater right frontal alpha asymmetry than autistic individuals (Damiano-Goodwin et al., 2018; Sutton et al., 2005). Therefore, it seems that frontal alpha asymmetry might not necessarily be a consistent method for distinguishing autistic and neurotypical individuals, but perhaps a reflection of certain behavioral characteristics that are often associated with autism.

Frontal Alpha Asymmetry and Autism Characteristics

In 2005 Sutton et al. were one of the first to associate frontal alpha asymmetry with autism characteristics. In their study they looked at resting state frontal alpha asymmetry within a group of 23 autistic children and 20 age- and verbal-IQ- matched peers between the ages of 9 and 14. They found that children who displayed greater right than left frontal alpha asymmetry, regardless of diagnostic status, exhibited more social impairments, as assessed by the Autism Spectrum Screening Questionnaire (Ehlers et al., 1999) and the Australian Scale for Asperger Syndrome (Robinson, 2013), than children who displayed greater left than right frontal alpha asymmetry. Additionally, while they did not conduct this analysis with the neurotypical children, they found that amongst autistic children those with greater right than left frontal alpha asymmetry had greater visual analytic skill (as measured by performance on the Block Design subtest of the *Weschler Abbreviated Scale of Intelligence Second Edition,* Wechsler & Hsiao-pin, 2011), a well-documented autistic strength (Belmonte et al., 2004; Bertone et al., 2005; Joseph et al., 2009; Samson et al., 2012), compared to those with greater left than right frontal alpha asymmetry. Taken together these results illustrate a potential relationship between frontal alpha asymmetry and autistic characteristics.

A portion of these findings was later supported by Burnette et al. (2011) when they found that children with greater right frontal alpha asymmetry tended to display increased social impairment than those that had greater left frontal alpha asymmetry. Interestingly, however, they found that both verbal IQ and diagnostic status significantly moderated this effect, with only autistic individuals who had relatively lower verbal IQs seeing a strong relationship between right frontal alpha asymmetry and social impairments. They also found that individuals with greater left frontal alpha asymmetry had parent reports indicating a later onset of autism characteristics compared to individuals with greater right frontal alpha asymmetry. This led the authors to conclude that individual differences in frontal alpha asymmetry may moderate the expression and development of autistic characteristics.

Given this finding by Burnette et al. that verbal IQ significantly moderates the relationship between frontal alpha asymmetry and social impairments, I wanted to see how frontal alpha asymmetry

and autistic characteristics would relate when looking at a subset of autistic and neurotypical individuals that have typical IQs. Matching autistic and neurotypical participants on IQ is a common methodology in this field of research and is used in an attempt to disentangle relationships that emerge as a result of meaningful differences between the diagnostic groups from relationships that emerge as a result of the large heterogeneity across various characteristics seen within autism, such as IQ (Burack et al., 2021; Mottron, 2004; Russo et al., 2021).

Along with social deficits (American Psychiatric Association 2013) and enhanced visual analytic skills (Belmonte et al., 2004; Bertone et al., 2005; Joseph et al., 2009; Samson et al., 2012), atypical sensory responsiveness has also been commonly associated with autistic individuals (Baranek et al., 2006; Crane et al., 2009; Dawson & Watling, 2000). Studies by Damiano-Goodwin et al. (2018) and Simon et al. (2017) both sought to investigate the relationship between frontal alpha asymmetry and sensory-related characteristics. In their studies they found that infants with an elevated likelihood of being diagnosed with autism, younger siblings of autistic individuals, who demonstrated greater left than right frontal alpha asymmetry had increased rates of sensory seeking behaviors (Damiano-Goodwin et al., 2018) and increased rates of sensory hyporesponsivity (Simon et al., 2017). Importantly, in the Damiano-Goodwin article (2018) they did not find a significant relationship between frontal alpha asymmetry and sensory seeking behavior for infants with a decreased likelihood of being diagnosed with autism, as defined by being a younger sibling of a neurotypical individual.

While this is an interesting finding, there is some reason to believe that a similar relationship between diagnostic status, frontal alpha asymmetry, and sensory seeking behaviors would not be found in older individuals. In their study in 2015 Gabard-Durnam et al. found infants with older autistic siblings (infants at an elevated likelihood of being diagnosed with autism later in life) had differing trajectories with respect to frontal alpha asymmetry when compared to infants with older neurotypical siblings (infants with a decreased likelihood of being diagnosed with autism later in life). When measured at 6 months infants with older neurotypical siblings had significantly more negative alpha

asymmetry compared to infants with older autistic siblings. Over the course of the following 12 months, the trajectories with regard to alpha asymmetry between these two groups also differed. While the infants with older neurotypical siblings on average shifted from a negative asymmetry score toward a positive asymmetry score, infants with older autistic siblings on average shifted from a neutral asymmetry score toward a negative asymmetry score. This study demonstrates that infants with older autistic siblings and infants with older neurotypical siblings have fundamentally different frontal alpha asymmetry profiles and trajectories early in life, which may partially explain why only infants with autistic siblings were found to have a relationship between frontal alpha asymmetry and sensory seeking behaviors in the study by Damiano-Goodwin et al. (2018). Since frontal alpha asymmetry profiles of older neurotypical and autistic individuals matched on age, sex, and IQ do not significantly differ (Lefebvre et al., 2018), it could be interesting to investigate whether or not this relationship between diagnostic status, frontal alpha asymmetry, and sensory seeking behaviors is exhibited in older adolescents. Investigating this question could be useful in moving beyond the focus on group differences to categorically differentiate autism and neurotypical development, to instead examine how specific autism characteristics relate to certain patterns of neural activity in a more dimensional approach.

The emerging literature on frontal alpha asymmetry in autism indicates that greater right than left frontal alpha asymmetry may be linked with increased social deficits, increased visual analytic skills, decreased sensory seeking behaviors, and decreased hyporesponsivity. Considering that these four features are all common autism characteristics and have opposing relationships with frontal alpha asymmetry it is understandable that the literature surrounding comparisons in frontal alpha asymmetry between autistic and neurotypical individuals has been inconsistent. It also opens up questions surrounding how frontal alpha asymmetry and all of these autistic characteristics interact and correlate with one another in a group of older children whose ages and IQs are comparable between groups. Here I approached this question by examining the relationship between frontal alpha asymmetry and autism characteristics through the Autism Quotient (Baron-Cohen et al., 2001; Baron-Cohen et al., 2006) and

Sensory Profile (Dunn, 1999; Dunn, 2014), continuous measures of traits that are generally normally distributed but are elevated in autism (Lundstrom et al., 2012; Metz et al., 2019; Robinson et al., 2011). Together these measures will allow for the examination of individual differences as they relate to frontal alpha asymmetry.

Project Goals and Hypotheses

The goals for this project were three-fold. Fist I aimed to determine if frontal alpha asymmetry values differed as a function of group. The idea was for this to add to the general literature on frontal alpha asymmetry differences between neurotypical and autistic groups in a manner that considered and accounted for potential differences in development and cognitive abilities as the participants were matched on age and IQ. I hypothesized that frontal alpha asymmetry would not significantly differ between the autistic and neurotypical groups. Lefebvre et al. (2018) previously found that the frontal alpha asymmetry profiles of autistic and neurotypical children do not significantly differ, and thus I aimed to replicate those findings here.

The second aim was to investigate the relationship between frontal alpha asymmetry and clinical characteristics that are common in autism, and for which previous research suggests a relationship. This was completed by examining whether frontal alpha asymmetry correlated with social, visual analytic, and sensory traits that are normally distributed in the population. Social challenges were measured by raw scores on the social skill subscale of the Autism Quotient, visual analytic skills were measured by the raw scores on the attention to detail subscale of the Autism Quotient, sensory seeking behaviors were measured by the raw scores on the sensory seeking quadrant of the Sensory Profile or the Sensory Profile 2, and sensory hyporesponsivity was measured by the raw scores on the low registration quadrant of the Sensory Profile or the Sensory Profile 2 for both autistic and neurotypical children. It was hypothesized that frontal alpha asymmetry would correlate positively with the social skills and attention to detail subscales of the Autism Quotient and correlate negatively with the sensory seeking and low registration subscales of the Sensory Profile. This was in line with previous research that found that

greater right than left frontal alpha asymmetry (a positive value) was associated with greater social deficits and increased visual analytic skills (Burnette et al., 2011; Sutton et al., 2005) and greater left than right frontal alpha asymmetry (a negative value) was associated with increased sensory seeking behaviors and greater sensory hyporesponsivity (Damiano-Goodwin et al., 2018; Simon et al., 2017).

The third aim was to determine whether the strength of correlations between autistic traits and frontal alpha asymmetry differed between the autistic and neurotypical groups. Previous work yielded inconsistent results with some evidence suggesting a significant correlation between frontal alpha asymmetry and social deficits for both neurotypical and autistic individuals (Sutton et al., 2005) and some evidence suggesting a significant correlation between frontal alpha asymmetry and social deficits only for autistic individuals with relatively low verbal IQs (Burnette et al., 2011). Since it seemed possible that verbal IQ could impact social abilities, and thus the relationship between frontal alpha asymmetry and social deficits, and our participants had generally average verbal IQs, I anticipated my findings would be more in line with those of Sutton et al. (2005), that is no significant difference between the autistic and neurotypical groups with regards to their correlations between frontal alpha asymmetry and social deficits. I also did not anticipate finding a significant difference between the autistic and neurotypical groups with regards to their correlations between frontal alpha asymmetry and sensory seeking behaviors or sensory hyporesponsivity. While the study by Damiano-Goodwin et al. (2019) only found significant correlations between frontal alpha asymmetry and sensory seeking behaviors for individuals at an elevated likelihood of being diagnosed with autism, their study was conducted with toddlers. Given the research suggesting that infants with older autistic siblings and infants with older neurotypical siblings have different development trajectories with regards to frontal alpha asymmetry during this period of life (Gabard-Durnam et al., 2015), along with the research that older autistic and neurotypical individuals do not have significantly different frontal alpha asymmetry profiles (Lefebvre et al., 2018) there was reason to believe that a similar relationship between diagnostic status, frontal alpha asymmetry, and sensory seeking behaviors would not be found in older individuals.

Therefore, I hypothesized that autistic and neurotypical children would not significantly differ with regard to their correlations between frontal alpha asymmetry and sensory seeking behaviors or sensory hyporesponsivity.

Method

Participants

30 age- and IQ-matched (see Table 1) neurotypical (*n*=15, ages 11-17) and autistic (*n*=15, ages 10-17) children and adolescents participated in this study. Full-scale IQs (FSIQ) were measured with the *Weschler Abbreviated Scale of Intelligence Second Edition* (WASI-II, Wechsler & Hsiao-pin, 2011). To confirm that all participants in the autistic group met the DSM-5 criteria for autism, both an Autism Diagnostic Observation Schedule-2 evaluation (ADOS-2, Lord et al., 2012) and an Autism Diagnostic Interview-Revised (ADI-R; Rutter et al., 2003) were conducted. Neither the ADOS-2, nor the ADI-R were conducted with the neurotypical participants. All participants or their caregivers completed an Autism Quotient questionnaire (Baron-Cohen et al., 2001; Baron-Cohen et al., 2006) and Sensory Profile questionnaire (Dunn, 1999; Dunn, 2014) to assess the rates at which participants exhibit autistic characteristics. Participants or their caregivers also completed a clinical interview and a questionnaire focused on developmental history. For the neurotypical group these measures along with the Autism Quotient (Baron-Cohen et al., 2001; Baron-Cohen et al., 2006) were used to ensure that they did not have a history of displaying nor currently displayed (defined as having an Autism Quotient score below 32) a significant number of autistic characteristics. The group of autistic participants scored significantly higher across the two subscales of interest on both the Autism Quotient and the Sensory Profile (illustrated in Table 1) as would be expected.

Of the 15 neurotypical children, 93.3% identified as non-Hispanic, 86.7% as White, 6.7% as Asian, and 6.7% as American Indian. Of the 15 autistic children, 100% identified as non-Hispanic, 86.7% as White, 6.7% as Black, and 6.7% as mixed race (Black and White). Of the 15 neurotypical

children, 66.7% were the products of full-term healthy pregnancies, 6.7% were born 6 weeks early, 6.7% were born 4 weeks early, 6.7% were born 2.5 weeks early, and 13.3% did not have pregnancy data (guardians did not know the details of their birth). Of the 15 autistic children, 73.3% were the products of full-term healthy pregnancies, 6.7% were born 14 weeks early, 6.7% were born 6 weeks early, and 13.3% were born 2 weeks early. No neurotypical or autistic children had a history of seizures.

There were a number of participants within both groups that were currently taking various medications including: Lexapro, Adderall, Fluoxetine, Norepinephrine, Singulair, Prozac, Concerta, Abilify, Risperdal, Luvox, Vistaril, birth control, and allergy medication. Previous research has indicated that common antidepressants (van der Vinne et al., 2019) and common ADHD medications (Chueh et al., 2021; Keune et al., 2011) do not significantly impact frontal alpha asymmetry. No data is available for the impact that birth control, blood pressure medications, or allergy medications have on frontal alpha asymmetry.

The two groups did differ significantly on gender (see Table 1), with the autistic group being primarily comprised of males (80%) and the neurotypical group being 33% male and 66% female ($p<.01$). While research has indicated that frontal alpha asymmetry does not differ based on sex assigned at birth (Glier et al., 2022; Stewart et al., 2011), both sex and age have been covaried for in the data analysis.

Clinical and Behavioral Measures

ADOS-2

The ADOS-2 (Lord et al., 2012) is a semi-structured, standardized measure that assesses early communication, social interaction, and play. It is one of the main tools frequently used by researchers as one piece in developing a profile to help diagnose individuals with autism. While the ADOS-2 has four modules in total, only module 3 (n=10), for younger individuals that are verbally fluent, and module 4 (n=5), for older individuals that are verbally fluent, were used during this project. All ADOS-2 modules were conducted by a trained graduate student and video-reviewed by a trained licensed psychologist. All

autistic participants have met the cut-off for autism as defined by clinical judgment and a total score of 7 or above on the ADOS-2 module 3 and 8 or above on module 4 (Lord et al., 2012).

The ADOS-2 has generally good internal consistency, with Cronbach's alpha values ranging from .47 to .92 across all 4 modules (McCrimmon & Rostad, 2014). The internal consistency is considered high for the Social Affect (SA) domain (Cronbach's alpha ranging from .75-.92) and moderate for the Restricted and Repetitive Behaviors (RRB) domain (Cronbach's alpha: .47-.66) (McCrimmon & Rostad, 2014). Across all modules interrater reliability is above 70% for item coding with most mean weighted kappas above .60 (McCrimmon & Rostad, 2014). In general, agreement in diagnostic classification is also high, ranging from 92%-98% (McCrimmon & Rostad, 2014).

With regard to content and construct validity, logistic regressions demonstrated that both the SA and RRB domains independently made significant contributions to the prediction of diagnosis (McCrimmon & Rostad, 2014). The total score (generated from combining SA and RRB domain scores) produced the highest predictive value, supporting its use in diagnostic decision-making (McCrimmon & Rostad, 2014). When looking at predictive validity the ADOS-2 is comparable to the original ADOS with sensitivity ranging from 60%-95% and specificity ranging from 75%-100% (McCrimmon & Rostad, 2014).

Autism Quotient

The Autism Quotient is a self-report or parent-report questionnaire that measures the presence of autistic characteristics in individuals with average or above-average IQs (Baron-Cohen et al., 2001; Baron-Cohen et al., 2006). Three different variations of the Autism Quotient were administered during this project depending on the participant's age: the parent-report Child Autism Quotient for ages 4-11 (neurotypical, n = 4; autistic, n = 2), the Adolescent Autism Quotient for ages 12-15 (neurotypical, n = 8; autistic, n = 8), and the self-report Adult Autism Quotient for subjects over the age of 16 (neurotypical, n = 3; autistic, n = 5) (Baron-Cohen et al., 2001; Baron-Cohen et al., 2006). Each Autism Quotient assessment consists of 50 items. While the items assess the same concepts across each

variation, they are adapted to the corresponding developmental level. For each item, the respondent is asked to rate the described trait on a 4-point scale ranging from “definitely agree” to “definitely disagree.” During scoring, each item is given a score of 0 (for responses considered not to be representative of common autistic characteristics) or 1 (for responses considered to be representative of common autistic characteristics). It should be noted that the parent-report Child Autism Quotient for ages 4-11 is traditionally scored from 0 (for responses considered not to be representative of common characteristics) to 3 (for responses considered to be representative of common autism characteristics). However, these scores can and were converted to be consistent with the Adolescent and Adult Autism Quotient measures. With this method, items with a response of definitely agree or slightly agree are given the same score and items with a response of definitely disagree or slightly disagree are given the same score. When added together this results in each participant having a total Autism Quotient score ranging from 0-50 where higher scores indicate a higher presence of autistic traits, such as poor social skills, poor communication skills, poor imagination, exceptional attention to detail, and poor attention-switching (Baron-Cohen et al., 2001; Baron-Cohen et al., 2006). Scores of 32 or greater are highly predictive of autism (Baron-Cohen et al., 2001). This, combined with its good discriminant and convergent validity, results in the Autism Quotient having strong overall validity (Woodbury-Smith et al., 2005). The Autism Quotient also has strong test-retest and interrater reliability (Baron-Cohen et al., 2001). The social skills and attention to detail subscale scores from the Autism Quotient have been correlated with frontal alpha asymmetry for this project.

Sensory Profile

The Sensory Profile is a standardized questionnaire containing 125 items that is completed by a caregiver to assess the extent to which sensory processing impacts a child’s behavior (Dunn, 1999; Dunn, 2014). For each item, the caregiver rates the corresponding behavior on a 5-point Likert scale where a score of 1 indicates the child responds in the outlined manner 100% of the time and a score of 5 indicates the child never responds in the outlined manner (Dunn, 1999; Dunn, 2014). Responses indicate

to what extent the child fits into each of the four behavioral patterns of sensory modulation: hyporesponsivity (low registration) hypersensitivity (sensory sensitivity), sensory seeking, and sensory avoiding (Dunn, 1999; Dunn, 2014). The Sensory Profile and Sensory Profile 2 have good internal consistency with Cronbach's alpha coefficients ranging from 0.47 to 0.91 across subscales (Dunn, 1999; Dunn, 2014). Good inter-rater reliability and test-retest reliability have also been reported (Dean et al., 2016; Houwen et al., 2022). The hyporesponsivity/low registration subscale score and the sensory seeking subscale score have been correlated with frontal alpha asymmetry for this project.

WASI-II

The WASI-II (Wechsler & Hsiao-pin, 2011) is an abbreviated cognitive intelligence assessment for individuals ranging from 6 to 90 years of age. The assessment consists of 4 subtests across two indices: Verbal Comprehension (Vocabulary and Similarities subtests) and Perceptual Reasoning (Block Design and Matrix Reasoning subtests). Upon completion one receives a Verbal Comprehension Index (VCI) score, a Perceptual Reasoning Index (PRI) score, and a Full-Scale IQ score (FSIQ, comprised of scores from all 4 subtests). The WASI is reported to have strong internal consistency, test-retest stability, interrater reliability, and validity (McCrimmon & Smith, 2013). For the purposes of this project, the WASI-II was primarily used to IQ-match autistic and neurotypically developing individuals on the basis of FSIQ. The participants for this study did not significantly differ in their VCI, PRI, or FSIQ scores.

EEG Data Acquisition

For all participants, the EEG recording took place in a dimly lit, sound-attenuated room. Continuous EEG was recorded with a high-density 128-channel Geodesic SensorNet using NetStation software 5.4. All participants sat in front of a computer screen and were asked to remain as still as possible throughout the experiment. During recording, participants were run through two consecutive 3-minute blocks where they were first asked to keep their eyes open and focused on a fixation cross in the middle of the screen and then asked to keep their eyes closed for the full 3-minute block. Using both

eyes-closed and eyes-open conditions is consistent with previous research investigating frontal alpha asymmetry in autistic and neurotypical children and adolescents (Burnette et al., 2011; Sutton et al., 2005) and is considered the most appropriate practice when running resting state EEG studies (Barry et al., 2007; Petro et al., 2022).

EEG Pre-Processing and Analysis

During acquisition EEG data was referenced to the vertex (Cz) and filtered between 0.1 and 100Hz. It was then exported and processed further using EEGLAB (Delorme and Makeig, 2004). A 60Hz notch filter was used to remove line noise and data was band-pass filtered between 1 and 80 Hz using a zero phase, 6^{th} order Butterworth filter. Two-second-long epochs with 50% overlap were extracted and each epoch was visually inspected across all channels by a trained graduate student to identify any outliers or bad channels. Any epoch or channel that was deemed an outlier was rejected. Prior to visual inspection, two EEGLAB plug-ins, *trimOutlier* (Lee & Miyakoshi, 2019) and *TBT* (Ben-Schachar, 2018), were used to further inspect the data and eliminate artifacts and bad channels. The *trimOutlier* plugin helped detect and remove any potential flat channels which were defined as those that fell below 1 μV. The *TBT* plugin marked any channels that exceeded a differential average amplitude of 250 μV for rejection. Any channels that exceeded this threshold on more than 30% of epochs and any epochs that had more than 10 channels above this threshold were rejected. These guidelines are consistent with previous EEG research (Duma et al., 2020; Duma et al., 2021; Duma et al., 2022). With these criteria one subject from the autism group was removed from the dataset completely due to all of their channels being deemed bad. Of the remaining subjects no more than 9 channels (M = 1.67, SD = 2.22) and 87 epochs (M = 22.24, SD = 22.01) were removed. After removing the detected outliers, the average of all channels was calculated, and the signals were re-referenced to the common average. Lastly, independent component analysis was used to detect and subtract any residual artifacts that are often embedded in EEG data (e.g. eye blinks, eye movements, muscle movements). Following ICA any removed channels were interpolated. EEG data was then filtered by applying a zero padded fast Fourier

transform filter for the alpha frequency band (8-12Hz). For each electrode alpha power was calculated by squaring the filtered EEG signals (μV^{2-}). These pre-processing and analysis methods are consistent with previous resting-state empirical articles (Damiano-Goodwin et al., 2018; Schwab et al., 2014; Sutton et al., 2005)

To calculate alpha asymmetry, the natural log of the alpha-band power for each participant at each electrode on the left hemisphere was subtracted from the natural log of the alpha-band power for the corresponding electrode on the right hemisphere. Negative values indicate more power on the left side of the head.

To isolate frontal alpha asymmetry the same method was used but only for a select group of electrodes on both the left and right hemispheres. The left frontal group consists of electrode 24 (F3) and the neighboring 5 electrodes (19, 20, 23, 27, and 28) and the right frontal group consists of electrode 124 (F4) and the neighboring 5 electrodes (3, 4, 117, 118, and 123). These groupings are consistent with previous resting state frontal alpha asymmetry research (Damiano-Goodwin et al., 2018; Gabard-Durnam et al., 2015). None of these 12 channels had to be removed or interpolated for the 29 participants kept in this dataset.

Proposed Analysis

Aim 1: to test the hypothesis that frontal alpha asymmetry does not significantly differ between the autistic and neurotypical groups. Analyses were implemented in a Bayesian framework. The Bayes factor (Jeffreys, 1961; Kass & Raftery, 1995) allows for calculating the probability of the observed data under the null hypothesis compared to the alternative hypothesis (Wetzels & Wagenmakers, 2012). Since I hypothesized no difference between the autistic and neurotypical groups with regards to their frontal alpha asymmetry, assessing the relative probability of the null hypothesis compared to the alternative hypothesis was critical. The Bayes factor provided this opportunity and the analyses were implemented by using a Bayesian two-sample t-test via JASP (JASP Team, 2022).

Aim 2: to assess the relationship between frontal alpha asymmetry and autism characteristics. Pearson's correlation coefficients ("Pearson's Correlation Coefficient", 2008) were used to examine the relationship between frontal alpha asymmetry and social deficits and attention to detail, as measured by the Autism Quotient, as well as between frontal alpha asymmetry and sensory seeking behaviors and sensory hyporesponsivity, as measured by the Sensory Profile and Sensory Profile 2. The significance of each correlation coefficient was calculated using a threshold of 0.003125. This value was settled on after correcting a threshold of 0.05 for multiple comparisons using Bonferroni's correction (Curtin & Schultz, 1998).

Aim 3: to assess whether the strength of the correlations between frontal alpha asymmetry and autism characteristics differ as a function of group. Since my hypothesis was again in support of the null hypothesis, I relied on the Bayes factor test recently proposed by Mulder & Gelissen (2021) that allows for multiple hypotheses based on commonly used measures of association (i.e. correlation) to be tested simultaneously. Essentially this method allowed me to calculate the probability of the observed correlation coefficients under the null hypothesis compared to the alternative hypothesis. Since I hypothesized that there would be no difference in the frontal alpha asymmetry to autism characteristic correlations between the autistic and neurotypical groups, it was critical that I could directly test the null hypothesis. These calculations were made via an R package Mulder & Gelissen (2021) created which has already been used and cited in multiple peer reviewed articles (Colominas-Ciuró et al., 2022; Matsumoto et al., 2022; Mulder et al., 2022).

Results

Between-Group Frontal Alpha Asymmetry

Prior to conducting a Bayesian independent samples t-test the data was reviewed to confirm it met assumptions of normality and equal variance. As documented in Table 3 all four conditions had Shapiro-Wilks Normality Test p-values of above 0.05. This suggests there is not enough evidence to

conclude that these datasets are non-normal. To further analyze normality Figures 1-6 show Q-Q plots, box plots, and visual representations of the distributions. These figures further demonstrate that all four datasets appear to be relatively normally distributed. With regards to variance, Table 3 illustrates that all variance and standard deviation values appear relatively similar. In addition, Levene's tests to assess for equal variance across groups were conducted for both conditions and produced *p*-values of above 0.05 (Table 4). This means that it cannot be concluded that the variances significantly differ.

Once this was completed the frontal alpha asymmetry values were compared between groups for both the eyes-open and eyes-closed conditions using a Bayesian independent samples t-test. In order to use a Bayesian statistical test a prior had to be established. Establishing priors generally derives from either previous data, and thus the existence of an actual distribution of previous effect sizes, or an estimated distribution given relevant prior research (Gronau et al., 2020). Based on the effect sizes found within the research conducted by Burnette et al. (2011), Damiano-Goodwin et al. (2018), Edmunds et al. (2023), Gabard-Durnman et al. (2015), Neuhaus et al. (2023), Sutton et al. (2005), Wang et al. (2013), along with data of effect sizes from other existing frontal alpha asymmetry research (Gollan et al., 2014; Peltola et al., 2014) as well as data for effect sizes from the field of psychology as a whole (Schäfer & Schwarts, 2019), it was estimated that there would be roughly a 50% probability that the effect size would lie between -0.5 and 0.5. This, in turn, suggests that a prior described by a Cauchy distribution centered around 0 with a width of magnitude 0.5 would be most appropriate (Gronau et al., 2020). Using this prior, it was determined that the data was neither sufficiently in favor of the null hypothesis nor the alternative hypothesis ($1/3 < BF_{01} < 3$) (Tables 5-6 and Figures 7-8). Even when considering other possible priors, Figure 9 illustrates that no matter what prior width was chosen, the results still would have been anecdotal in the eyes open condition suggesting no preference for either the null or alternative hypothesis. In the eyes closed condition if a wider prior was deemed more reasonable the results would have suggested moderate preference for the null hypothesis (Figure 10). Taken altogether, this indicates that more data should be collected before any meaningful conclusions can be made for whether or not

neurotypically developing and autistic children and adolescents differ with regards to frontal alpha asymmetry.

Given that the two groups significantly differed with regards to gender and had mean ages of nearly 1 year apart, it was deemed appropriate that both gender and age should be covaried for to determine if these factors impacted between-group frontal alpha asymmetry comparisons. To do so a Bayesian analysis of covariance (ANCOVA) was implemented via JASP (JASP Team, 2022) on frontal alpha asymmetry power with group (neurotypical or autistic) and gender assigned at birth (male or female) as fixed factors and age as a covariate. The Bayesian ANCOVA compares the relative predictive performance of a variety of models with differing predictors. In this case there were 10 models in both the eyes open (Table 7) and eyes closed (Table 9) conditions. In the eyes open condition, only the models Gender + Group ($BF_M = 2.161$), Group ($BF_M = 1.832$), Gender + Group + Gender*Group (interaction effect) ($BF_M = 1.056$), and the null model ($BF_M = 1.977$) had their model odds increase after accounting for the observed data. However, none of these models sufficiently distinguished themselves from the others as illustrated by the values documented in the BF_{10} column. This column outlines how likely the observed data is for each model comparatively to best performing model (in this case Gender + Group). For instance, the observed data is 0.930 times as likely under the Null model than the Gender + Group model. While this implies support against the null hypothesis, it is not strong enough to warrant any meaningful conclusions.

To account for model uncertainty, Bayesian model averaging was also used to assess the effects of all predictors in both the eyes open (Table 8) and eyes closed (Table 10) conditions. This method takes each effect (Group, Gender, Age, Group*Gender) across all models in which it is present and determines the probability that the effect is included in the true model (i.e. changes in the effect provide meaningful information about changes in the dependent variable). In the eyes open condition only the Group effect had its posterior probability increase resulting in an inclusion Bayes factor (BF_{incl}) of 1.184. This suggests the observed data was 1.184 times more likely under models that include Group

(neurotypical vs autistic) as a predictor. With regards to effects Age and Gender, both saw their posterior probabilities decrease resulting in inclusion Bayes factors of 0.361 and 0.744 respectively. While this suggests that the observed data was 0.361 times (for age) and 0.744 times (for gender) as likely under models that contain those respective effects, these are not sufficient enough to conclude that neither age nor gender are effects in the true model for this dataset.

As alluded to above, these outlined methods were also used for the eyes closed condition. In this condition the following models had their odds increase after accounting for the observed data: Gender ($BF_M = 1.895$), Age ($BF_M = 1.284$), the null model ($BF_M = 3.510$). Similar to the eyes open condition none of these models sufficiently distinguished themselves from the others. While the null hypothesis was most supported given the observed data, the observed data was still 0.620 as likely under the Gender model (the second most supported model) compared to the Null model again indicating that no model was sufficiently supported above the others.

Bayesian model averaging was also used for the eyes closed condition. In this scenario all effects saw their posterior probabilities decrease, suggesting that the observed data was less likely when Group, Gender, Age, or Gender*Group were included as effects. While this supports the notion that none of these effects play critical roles in the outcome of the dependent variable (i.e. frontal alpha asymmetry), none of them reach the moderate evidence threshold of below 0.3. Taken altogether this suggests that across both the eyes closed and eyes open conditions it cannot be concluded that age or gender were not significant covariates.

Frontal Alpha Asymmetry Correlations with Autism Characteristics

After reviewing how frontal alpha asymmetry values compared between groups, partial correlations were analyzed within group between frontal alpha asymmetry and various autism characteristics. Given the conclusions of the Bayesian ANCOVAs, it was deemed most appropriate to conduct the correlations while controlling for age and gender as it could not be deemed that they were not covariates, hence the use of partial correlations. Across both eyes open and eyes closed conditions

neither the neurotypical group (Table 11 – eyes open, Table 12 – eyes closed) nor the autistic group (Table 13 – eyes open, Table 14 – eyes closed) demonstrated statistically significant correlations between frontal alpha asymmetry and sensory seeking or hyporesponsivity scores from the Sensory Profile, or social skills and attention to detail scores from the Autism Quotient. Given the relatively small sample of this study, along with some correlations approaching or at moderate strength (frontal alpha asymmetry and social skills scores for both eyes open and eyes closed conditions in the neurotypical group and frontal alpha asymmetry and attention to detail scores for both eyes open and eyes closed conditions, and frontal alpha asymmetry and social skills scores and sensory seeking scores for just the eyes closed conditions in the autistic group) more data should be collected before determining if frontal alpha asymmetry correlates with these selected autism characteristics for neurotypical and autistic children and adolescents.

Strength of Frontal Alpha Asymmetry and Autism Characteristic Correlations Between Groups

Once correlations had been calculated for all conditions for each group the strength of the correlations was compared between groups using a Bayes factor test for measures of association (Mulder & Gelissen, 2021). Again, since it could not be determined if age or gender had a significant interaction with the dependent variable of frontal alpha asymmetry, both these factors were controlled for. Results are displayed in Table 15 and Figure 11 and indicate that for all conditions the strength of the correlations between frontal alpha asymmetry and the measured autism characteristics is not sufficiently better described by either the null or alternative hypothesis ($1/3 < BF_{01} < 3$) further indicating that more data should be collected before conclusions are finalized.

Discussion

This study aimed to investigate the relationship between frontal alpha asymmetry and autism characteristics among neurotypically developing and autistic children and adolescents. Results largely indicated no relationship for either group, with correlations being non-significant between frontal alpha

asymmetry and sensory seeking behaviors, hyporesponsivity, social skills, and visual analytic skills. Results comparing strengths of these correlations and frontal alpha asymmetry power across groups were also inconclusive with Bayesian statistical methods indicating no sufficient preference for either the null or alternative hypothesis. These results are largely disappointing as they provide no information as to whether they support or contradict previous findings and provide little opportunity to reflect on how they relate to my hypotheses.

Even if significant findings had been observed, it would have been difficult to reconcile them with the sheer number of contradictory results noted across studies researching frontal alpha asymmetry and its relation to autism or autism characteristics. One potential contributing factor to this collection of contradictory findings is the use of inconsistent methodologies during data acquisition and analysis. These inconsistencies include how EEG is preprocessed, which electrodes are chosen for frontal alpha asymmetry analysis, and the selected range of the frequency band. This is not a novel facet of EEG research. For years researchers have documented the disparities in preprocessing and analysis methods used by the EEG community (Bigdely-Shamlo, et al., 2016; Delorme, 2023; Robbins et al., 2020). These disparities are also significant because choice of preprocessing methodology has been documented to impact subsequent results and conclusions, especially when conducting analyses with low-frequency spectral data (Robbins et al., 2020). Thus, for this specific study, it is possible that the preprocessing methodologies selected, while supported by empirical research, may have influenced the data and subsequent conclusions in a different way than other studies analyzing similar comparisons between frontal alpha asymmetry and autism characteristics.

For example, as discussed previously, research has found inconsistent results when it comes to comparing frontal alpha asymmetry power between autistic and neurotypical individuals, with some research indicating that neurotypical individuals have greater left frontal alpha asymmetry than autistic individuals (Gabard-Durnman et al., 2015; Schiltz et al., 2018) and some research indicating that neurotypical individuals tend to have greater right frontal alpha asymmetry than autistic individuals

(Damiano-Goodwin et al., 2018; Sutton et al., 2005). Interestingly, Schiltz et al. (2018) and Sutton et al. (2005), who found contradictory findings, had differing preprocessing steps, with each study using a different reference electrode, high-pass filter, and method for detecting and removing artifacts. While it cannot be concluded that these differences led to the contradictory findings, these types of variations in preprocessing methodologies have been documented as impacting subsequent results and conclusions in broader EEG studies (Robbins et al., 2020).

Another potential factor contributing to the steady inconsistent findings within the field of frontal alpha asymmetry EEG research is the lack of standardization in electrode selection. While reviewing general frontal alpha asymmetry EEG studies published in 2023, at least four different groupings of electrodes were used to calculate frontal alpha asymmetry (Flasbeck et al., 2023; Shangguan et al., 2023; Wise et al., 2023; Yoon & Kim, 2023). While seemingly no research has been conducted on the influence of slightly varying electrode selection on subsequent analysis and results, the lack of standardization in using the same groupings of electrodes across similar EEG studies has previously been brought forward as a potential concern within this field of research (Farzan et al., 2017). This concern is intuitive as the data collected by each electrode reflects a different constellation of weighted neural activity. Thus, if electrode selection differs by study, it is likely that so to does the specific region of neural activity being reflected in the data.

Interestingly, in the studies listed earlier that had contradictory findings on how frontal alpha asymmetry compared between neurotypical and autistic individuals (Damiano-Goodwin et al., 2018; Gabard-Durnman et al., 2015; Schiltz et al., 2018; Sutton et al., 2005), all four used a different collection of electrodes to calculate their frontal alpha asymmetry. Again, while it cannot be deemed that these differences resulted in the differing conclusions, these choices ultimately add some level of noise to the data. It should be determined how much noise is added by changing the electrodes of interest and if this noise ultimately impacts the final results and conclusions.

There is also a lack of consistency in what is considered the alpha frequency range. Across the same four 2023 studies referenced above (Flasbeck et al., 2023; Shangguan et al., 2023; Wise et al., 2023; Yoon & Kim, 2023), the researchers either used 8-12Hz as their alpha band range or 8-13Hz. Similarly, in the Schiltz et al. (2018) and Sutton et al. (2005) papers that found contradictory results with regards to frontal alpha asymmetry power between neurotypical and autistic children and adolescents, Schiltz et al. (2018) used a range of 8-12Hz whereas Sutton et al. used a range of 8-13Hz. Similar to with the variations in electrode selection, there is no research reviewing if minor variations in the selected frequency range impact subsequent results and conclusions. Given that this research is yet to be conducted it is difficult to ascertain the impact that these changes have on subsequent data analysis. However, as documented previously, distinct physiological processes are believed to be related to different frequency ranges (Buskila et al., 2019; Khurana et al., 2018; Wang et al., 2013). Thus, opting to include or exclude the frequency range between 12-13Hz, an area that lies at the intersection of the alpha and beta frequency ranges, again introduces some level of noise into the data. Whether or not this noise significantly impacts findings is unknown, however it should be determined to better inform the field as to how these choices may influence data.

It should be noted that with regards to selected preprocessing methods, frequency range, and electrodes, studies often have minimal discussion as to why these selections are most appropriate beyond the indication that they are sufficiently popular. This gives off the impression that there is minimal sufficiently backed rationale for choosing one method over another and one simply must find empirical articles that have previously used their methods and analysis to justify their proposed research. While more complete guidelines for best practices in preprocessing EEG have been proposed in recent years (Keil et al., 2014; Pernet et al., 2020), the guidelines are quite broad, allowing researchers significant flexibility in their preprocessing methodologies (Robbins et al., 2020). This likely results in the continued use of variable methodologies which, as noted previously by Bidgely-Shamlo et al.

(2016), is an issue as it prevents the field from being able to conduct wide-scale analyses of EEG as well as compare the reliability of methodologies used.

With regards to this study in particular, there were also several limitations that restrict the overall conclusions that can be made. For starters this study had a relatively small sample size (n=29). In Bayesian statistics in order to rule out the possibility of a small but statistically significant effect size, a large enough sample is required to sufficiently support the null hypothesis. Therefore, it is possible that if more data was collected there would have been enough evidence for the null or alternative hypothesis. Since the sample size was small it increased the likelihood of getting anecdotal evidence which is what was found in this study.

Another limitation of this project was the lack of racial and gender diversity. It has been well documented that there exist disproportionate rates of autism diagnoses by race and gender (Loomes et al., 2017; Travers et al., 2013). While the disparities by race are decreasing with regards to rate of autism diagnoses (Pham et al., 2022), given the additional concerns of disparities in psychology research participation by race (Roberts et al., 2020), concerted effort has to be made to reduce the presence and impact of these disparities as much as possible. This includes limiting the extent to the conclusions from this project to only populations represented by our sample which for the neurotypical group was 86.7% white and 33% male and for the autistic group was 86.7% white and 80% male.

Several future directions come to mind when taking the limitations from this study and the concerns with standardization within this field into consideration. First, I strongly second the voices in the field that are already calling for more transparent records of EEG methodologies selected across studies as well as pushing for a greater focus on comparing the validity of various methodology pipelines. Without consistent documentation of practices used it can be difficult to develop standardized procedures for how data is preprocessed and analyzed. Subsequently, without standardized practices in how EEG data is preprocessed and analyzed it is nearly impossible to ascertain how choices made during these steps ultimately impact data and conclusions.

Second, this study should be replicated with a larger sample. As discussed earlier, as the sample size increases so too will the likelihood that the Bayesian analysis will find sufficient support for either the null or alternative hypothesis. Thus, if a significantly large enough sample is utilized, more conclusive results can be determined about whether or not frontal alpha asymmetry by itself or its relationship to autistic characteristics differs between neurotypical and autistic children and adolescents.

Lastly, a more concerted effort needs to be made to improve the diversity of subject pools within the field of EEG and autism related research. This starts with decreasing diagnostic disparities by a function of race and gender. While diagnostic disparities by race are decreasing, more research should be conducted to determine why females continue to be diagnosed with autism at a significantly reduced rate compared to males. Additionally, due to the continued limitation that EEG research has on its inability to accommodate coarse and curly hair (Etienne et al., 2020), which ultimately leads to Black individuals being disproportionately excluded from EEG research, future research should prioritize finding alternative methods for increasing inclusivity, such as braiding hair and using novel types of electrodes (Etienne et al., 2020). In the cases when improved diversity is not obtained, it should be clearly outlined by the researchers to ensure that conclusions are strictly restricted to populations represented by the sample.

Table 1.

Demographic characteristics of neurotypical and autistic participants

Characteristic	Neurotypical (n =15)		Autism (n = 15)		t(28)
	M	*SD*	*M*	*SD*	
Age	13.51	2.11	14.46	2.32	-1.18
Ethnicity – Hispanic	1		0		
Ethnicity – Non-Hispanic	14		15		
Gender – Females	10		3		
Gender - Males	5	-	12	-	2.82**
Race – American Indian	1		0		
Race – Asian	1		0		
Race - Black	0		1		
Race – Mixed	0		1		
Race - White	13		13		

**$p < .01$

Table 2.

Clinical characteristics of neurotypical and autistic participants

Characteristic	Neurotypical (n =15)		Autism (n = 15)		t(28)
	M	*SD*	*M*	*SD*	
FSIQ	111.13	10.38	107.40	12.07	0.91
VCI	108.6	10.92	102.67	14.64	1.26
PRI	110.53	9.47	110.93	10.47	.11
ADOS Severity Score		-	8.53	1.19	-
Autism Quotient - Social Skills	2.53	2.64	6.67	2.19	-4.66***
Autism Quotient - Attention to Detail	4.07	2.31	7.00	2.70	-3.20**
Sensory Profile – Sensory Seeking	34.93	8.30	46.80	13.97	-2.83**
Sensory Profile – Low Registration	22.07	7.58	41.73	13.37	-4.96***

Note. FSIQ, full-scale intelligence quotient; ADOS, autism diagnostic observation schedule.

**$p < .01$

***$p < .001$

Table 3.

Descriptive Statistics of Frontal Alpha Asymmetry Power by Group and Condition

Condition	*N*	*M*	*SD*	Variance	*Shapiro-Wilk*(54)[a]
Neurotypical_EC	15	-0.059	0.336	0.113	0.925
Neurotypical_EO	15	0.217	0.437	0.191	0.987
Autism_EC	14	-0.079	0.426	0.182	0.947
Autism_EO	14	-0.044	0.420	0.176	0.898

Note. EC = eyes closed condition; EO = eyes open condition.

[a] A Shapiro-Wilk test was conducted to test for normality for each group and condition. All p-values were >.05 indicating that the null hypothesis that the data was normally distributed could not be rejected.

Table 4.

Levene's Test for Equality of Variance Between Groups for Eyes Open and Eyes Closed Condition

Condition	F	df_1	df_2
Frontal Alpha Asymmetry Power – Eyes Open	0.096	1	27
Frontal Alpha Asymmetry Power – Eyes Closed	1.703	1	27

Note. The *p*-values for both tests were above .05 indicating the variances between groups did not significantly differ for either condition.

Table 5.

Bayesian Independent Samples T-Test for Frontal Alpha Asymmetry Scores - Eyes Open Condition

Variable	BF_{01}	error %
Frontal Alpha Asymmetry Power	0.961	0.001

Note. BF_{01} = Bayes factor in favor of the null hypothesis comparative to the alternative hypothesis; error % = estimate of the error during the computation of the Bayes factor

Table 6.

Bayesian Independent Samples T-Test for Frontal Alpha Asymmetry Scores - Eyes Closed Condition

	BF_{01}	error %
Frontal Alpha Asymmetry Power	2.252	6.545×10^{-4}

Note. BF_{01} = Bayes factor in favor of the null hypothesis comparative to the alternative hypothesis; error % = estimate of the error during the computation of the Bayes factor

Table 7.

Bayesian ANCOVA Model Comparison - Eyes Open Condition

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Group + Gender	0.100	0.194	2.161	1.000	
Null model	0.100	0.180	1.977	0.930	0.577
Group	0.100	0.169	1.832	0.873	0.577
Group + Gender + Group ✻ Gender	0.100	0.105	1.056	0.543	0.994
Gender	0.100	0.087	0.853	0.447	0.577
Group + Age + Gender	0.100	0.072	0.702	0.374	1.071
Age	0.100	0.063	0.608	0.327	0.577
Group + Age	0.100	0.060	0.576	0.311	0.994
Group + Age + Gender + Group ✻ Gender	0.100	0.039	0.369	0.203	1.247
Age + Gender	0.100	0.030	0.282	0.157	1.062

Note. P(M) = prior model probability; P(M|data) = posterior model probability; BF_M = change from prior to posterior model odds; BF_{10} = Bayes factor of the row-model against the best performing model (1^{st} row-model); error % = estimate of the error during the computation of the Bayes factor

Table 8.

Bayesian ANCOVA Analysis of Effects - Eyes Open Condition

Effects	P(incl)	P(excl)	P(incl|data)	P(excl|data)	BF_{incl}
Group	0.600	0.400	0.640	0.360	1.184
Age	0.500	0.500	0.265	0.735	0.361
Gender	0.600	0.400	0.527	0.473	0.744
Group ∗ Gender	0.200	0.800	0.144	0.856	0.675

Note. P(incl) = prior inclusion probability; P(excl) = prior exclusion probability; P(incl|data) = posterior inclusion probability; P(excl|data) = posterior exclusion probability; BF_{incl} = change from prior to posterior inclusion odds

Table 9.

Bayesian ANCOVA Model Comparison - Eyes Closed Condition

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model	0.100	0.281	3.510	1.000	
Gender	0.100	0.174	1.895	0.620	0.003
Age	0.100	0.125	1.284	0.445	0.001
Group	0.100	0.099	0.985	0.351	0.002
Gender + Age	0.100	0.075	0.727	0.266	0.826
Group + Gender	0.100	0.074	0.715	0.262	0.791
Group + Gender + Group ✻ Gender	0.100	0.068	0.656	0.242	0.895
Group + Age	0.100	0.044	0.410	0.155	0.906
Group + Gender + Age + Group ✻ Gender	0.100	0.032	0.298	0.114	1.159
Group + Gender + Age	0.100	0.030	0.279	0.107	1.850

Note. P(M) = prior model probability; P(M|data) = posterior model probability; BF_M = change from prior to posterior model odds; BF_{10} = Bayes factor of the row-model against the best performing model (1st row-model); error % = estimate of the error during the computation of the Bayes factor

Table 10.

Bayesian ANCOVA Analysis of Effects - Eyes Closed Condition

Effects	P(incl)	P(excl)	P(incl|data)	P(excl|data)	BF_{incl}
Group	0.600	0.400	0.346	0.654	0.352
Gender	0.600	0.400	0.452	0.548	0.551
Group ∗ Gender	0.200	0.800	0.100	0.900	0.444
Age	0.500	0.500	0.305	0.695	0.440

Note. P(incl) = prior inclusion probability; P(excl) = prior exclusion probability; P(incl|data) = posterior inclusion probability; P(excl|data) = posterior exclusion probability; BF_{incl} = change from prior to posterior inclusion odds

Table 11.

Pearson's r Correlations Between Neurotypical Group Frontal Alpha Asymmetry Power and Various Autism Characteristics When Controlling for Gender and Age – Eyes Open Condition

Variable	NT_EO	NT Seek	NT Hypo	NT AtD	NT SS
NT_EO	—				
NT Seek	0.265	—			
NT Hypo	0.177	-0.366	—		
NT AtD	0.157	0.095	-0.097	—	
NT SS	0.365	0.038	0.136	0.462	—

Note. NT_EO = neurotypical group frontal alpha asymmetry from the eyes open condition; NT Seek = neurotypical group sensory seeking scores on the Sensory Profile; NT Hypo = neurotypical group sensory hyporesponsivity scores on the Sensory Profile (Low Registration); NT AtD = neurotypical group visual analytic skills scores on the Autism Quotient (Attention to Detail), NT SS = neurotypical group social skills scores on the Autism Quotient

Table 12.

Pearson's r Correlations Between Neurotypical Group Frontal Alpha Asymmetry Power and Various Autism Characteristics When Controlling for Gender and Age – Eyes Closed Condition

Variable	NT_EC	NT Seek	NT Hypo	NT AtD	NT SS
NT_EC	—				
NT Seek	0.048	—			
NT Hypo	-0.159	-0.366	—		
NT AtD	-0.119	0.095	-0.097	—	
NT SS	-0.323	0.038	0.136	0.462	—

Note. NT_EC = neurotypical group frontal alpha asymmetry from the eyes closed condition; NT Seek = neurotypical group sensory seeking scores on the Sensory Profile; NT Hypo = neurotypical group sensory hyporesponsivity scores on the Sensory Profile (Low Registration); NT AtD = neurotypical group visual analytic skills scores on the Autism Quotient (Attention to Detail), NT SS = neurotypical group social skills scores on the Autism Quotient

Table 13.

Pearson's r Correlations Between Autism Group Frontal Alpha Asymmetry Power and Various Autism Characteristics When Controlling for Gender and Age – Eyes Open Condition

Variable	Autism_EO	Autism Seek	Autism Hypo	Autism AtD	Autism SS
Autism_EO	—				
Autism Seek	0.188	—			
Autism Hypo	0.103	0.509	—		
Autism AtD	0.454	0.390	0.094	—	
Autism SS	0.129	0.612	0.618	0.293	—

Note. Autism_EO = autism group frontal alpha asymmetry from the eyes open condition; Autism Seek = autism group sensory seeking scores on the Sensory Profile; Autism Hypo = Autism group sensory hyporesponsivity scores on the Sensory Profile (Low Registration); Autism AtD = Autism group visual analytic skills scores on the Autism Quotient (Attention to Detail), Autism SS = Autism group social skills scores on the Autism Quotient

Table 14.

Pearson's r Correlations Between Autism Group Frontal Alpha Asymmetry Power and Various Autism Characteristics When Controlling for Gender and Age – Eyes Closed Condition

Variable	Autism_EC	Autism Seek	Autism Hypo	Autism AtD	Autism SS
Autism_EC	—				
Autism Seek	0.323	—			
Autism Hypo	0.055	0.509	—		
Autism AtD	0.300	0.390	0.094	—	
Autism SS	0.339	0.612	0.618	0.293	—

Note. Autism_EC = autism group frontal alpha asymmetry from the eyes closed condition; Autism Seek = autism group sensory seeking scores on the Sensory Profile; Autism Hypo = Autism group sensory hyporesponsivity scores on the Sensory Profile (Low Registration); Autism AtD = Autism group visual analytic skills scores on the Autism Quotient (Attention to Detail), Autism SS = Autism group social skills scores on the Autism Quotient

Table 15.

Bayes factors in favor of the Null Hypothesis over the Alternative Hypothesis When Assessing the Difference in the Strength of the Correlations Between Autistic and Neurotypical Groups for Each Condition

Comparison Group	BF_{01}
BF_EO_Hypo	1.198
BF_EC_Hypo	1.241
BF_EO_Seek	1.26
BF_EC_Seek	1.217
BF_EO_SS	1.26
BF_EC_SS	1.2
BF_EO_AtD	1.184
BF_EC_AtD	1.267

Note. BF01 = Bayes factor in favor of the Null Hypothesis; EO, eyes open condition; EC = eyes closed condition; Hypo = Bayes factor for group comparisons of frontal alpha asymmetry correlations with hyporesponsivity scores; Seek = Bayes factor for group comparisons of frontal alpha asymmetry correlations with sensory seeking scores; SS = Bayes factor for group comparisons of frontal alpha asymmetry correlations with social skills scores; AtD = Bayes factor for group comparisons of frontal alpha asymmetry correlations with attention to detail scores

Figure 1.

Q-Q Plot for Neurotypical Eyes-Closed Frontal Alpha Asymmetry Dataset

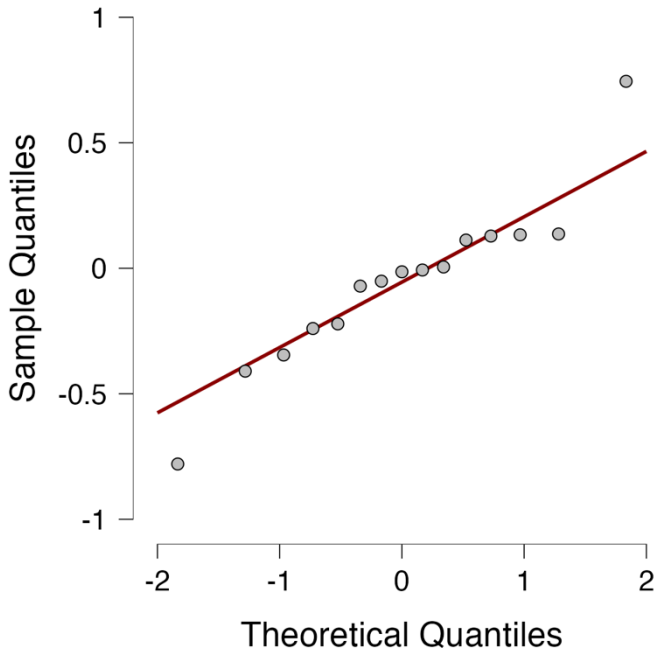


Figure 2.

Q-Q Plot for Neurotypical Eyes-Open Frontal Alpha Asymmetry Dataset

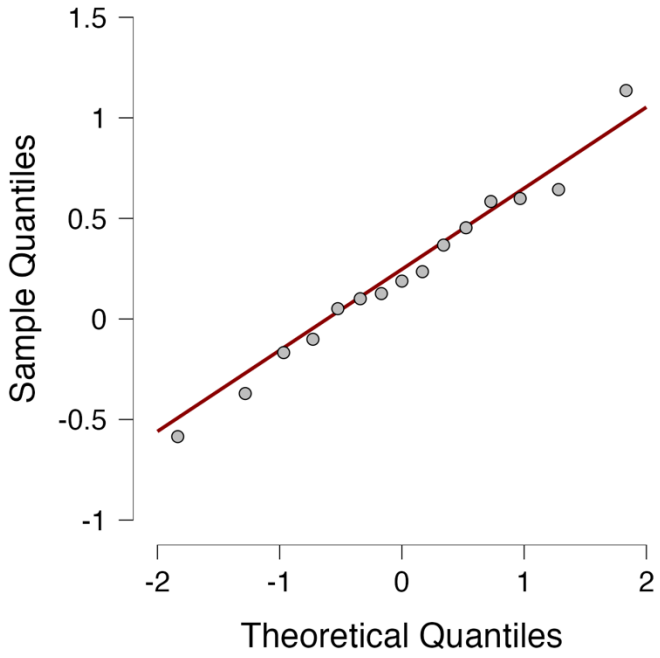


Figure 3.

Q-Q Plot for Autistic Eyes-Closed Frontal Alpha Asymmetry Dataset

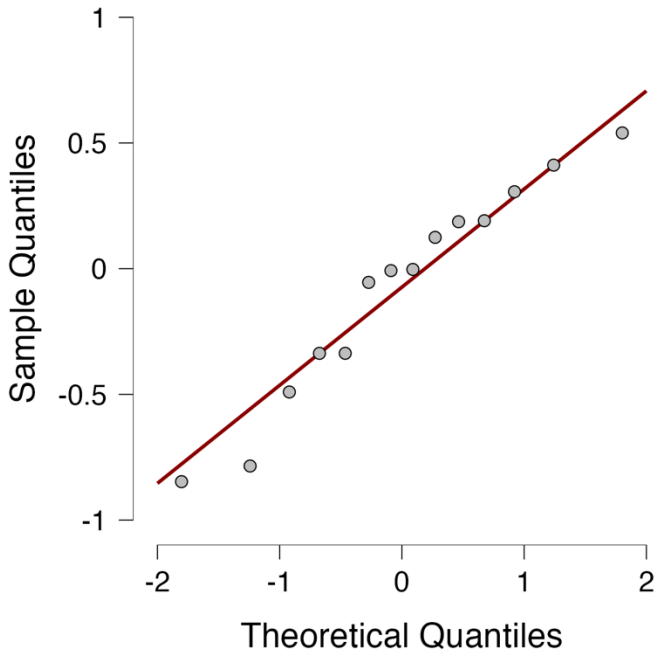


Figure 4.

Q-Q Plot for Autistic Eyes-Open Frontal Alpha Asymmetry Dataset

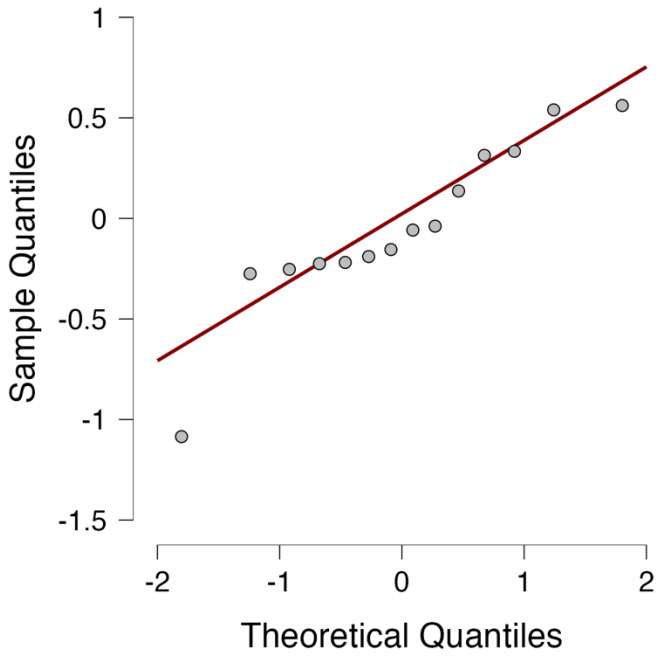
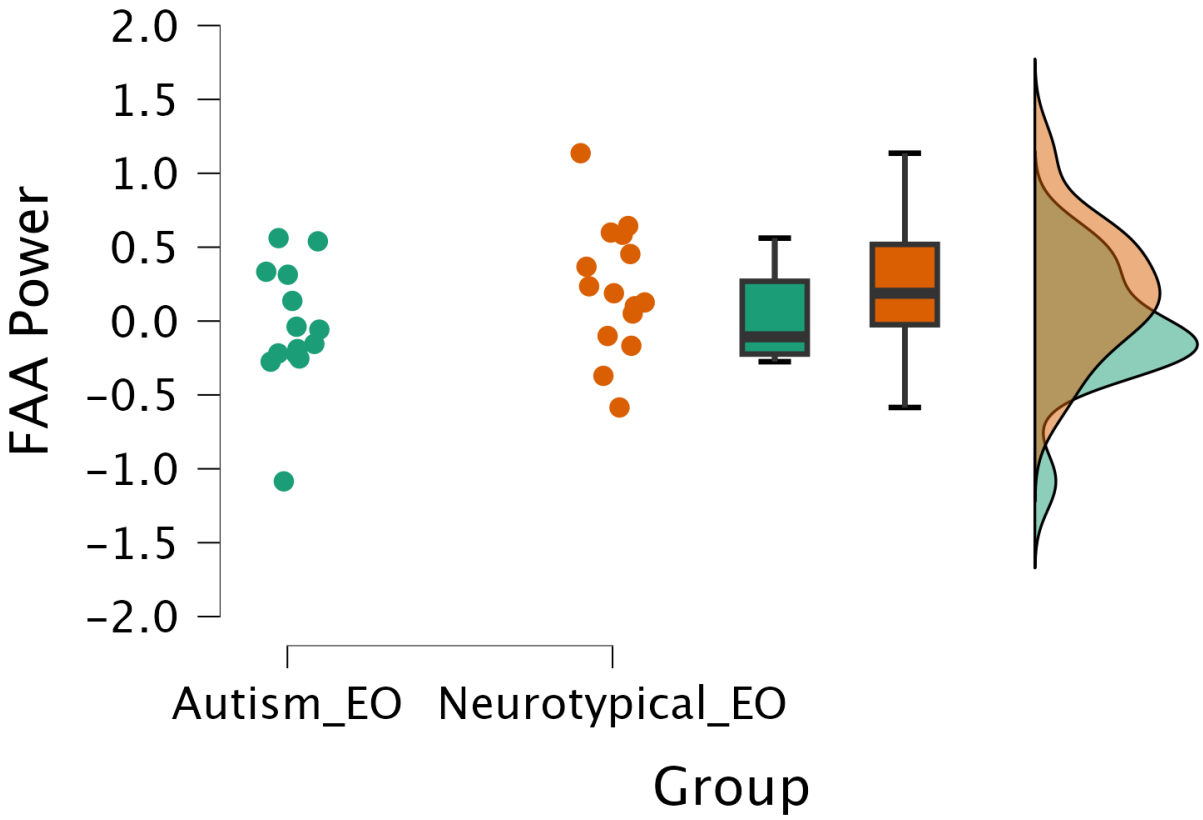


Figure 5.

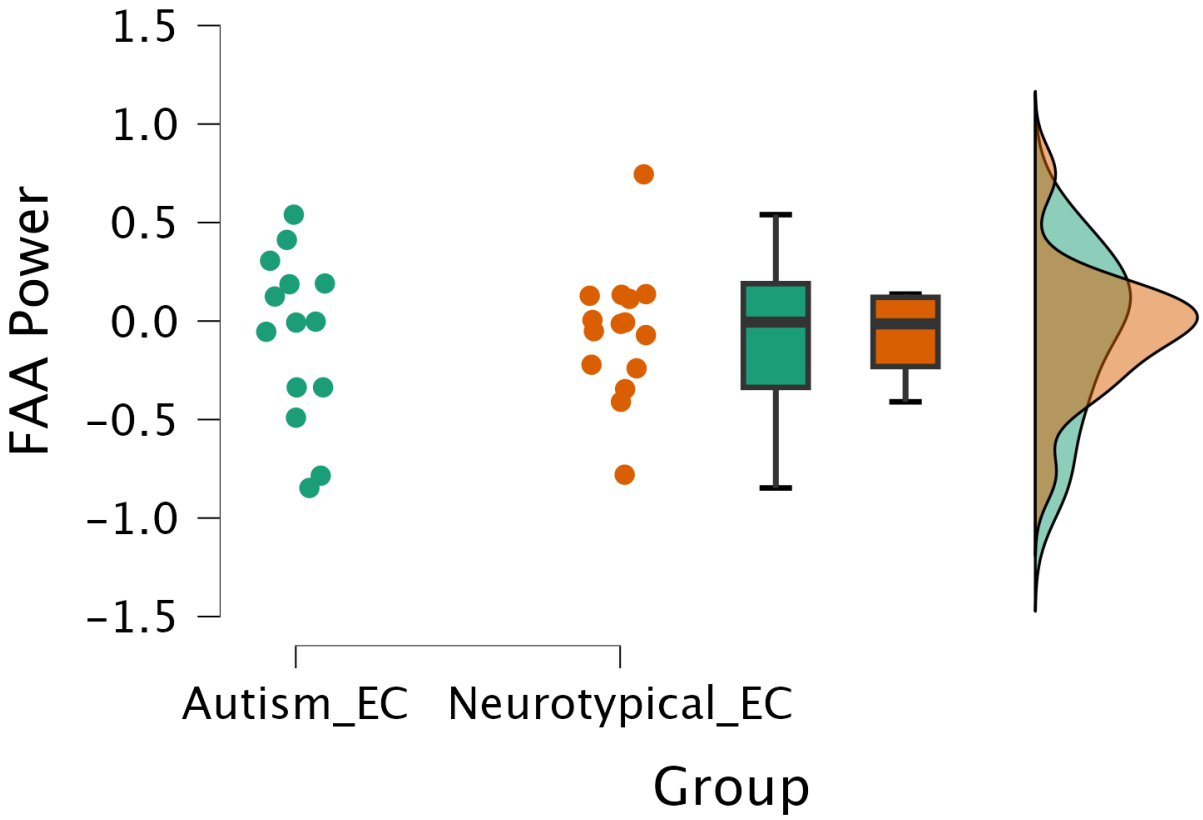
Distribution of Frontal Alpha Asymmetry Values by Group in the Eyes Open Condition



Note. FAA = frontal alpha asymmetry; EO = eyes open condition

Figure 6.

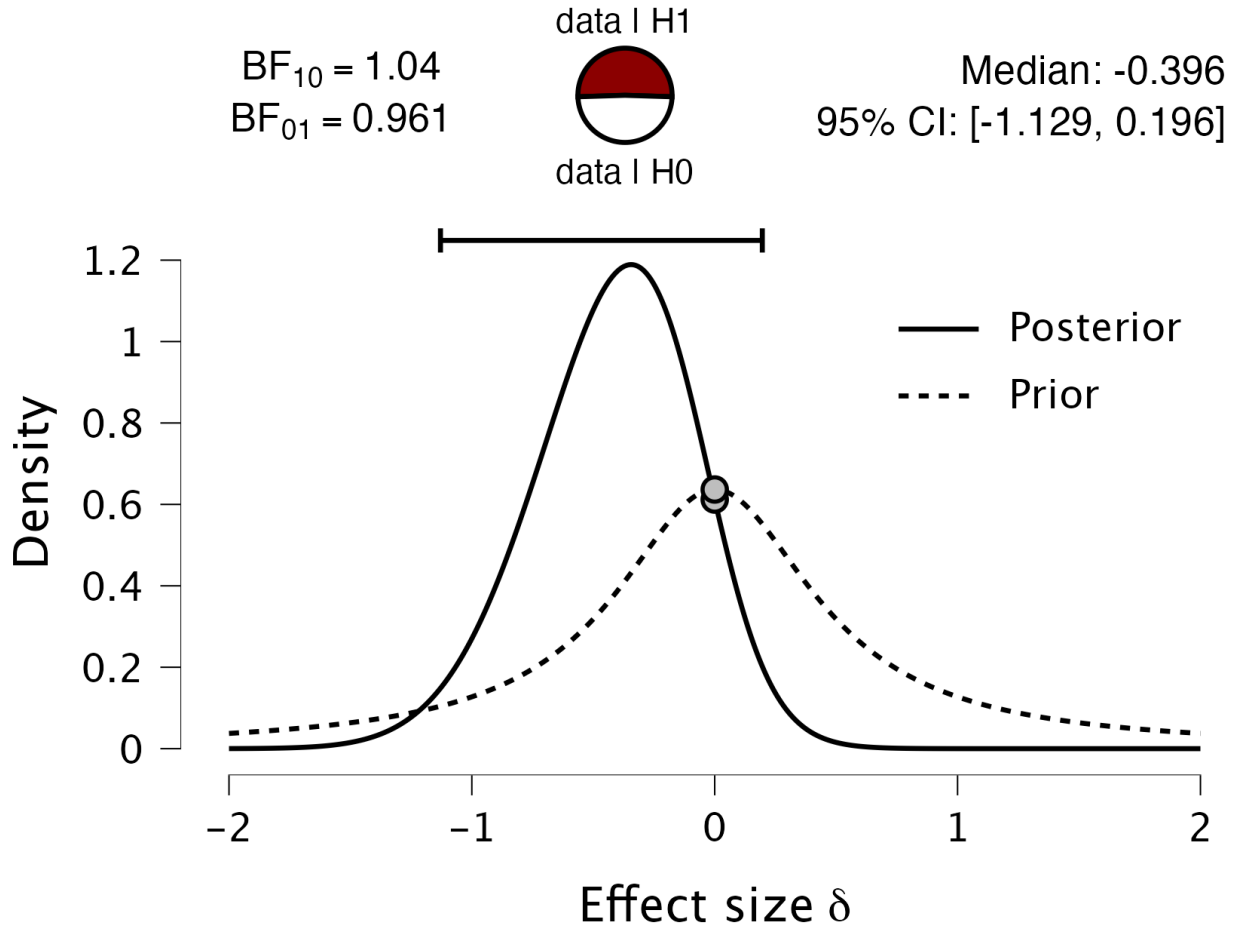
Distribution of Frontal Alpha Asymmetry Values by Group in the Eyes Closed Condition



Note. FAA = frontal alpha asymmetry; EC = eyes closed condition

Figure 7.

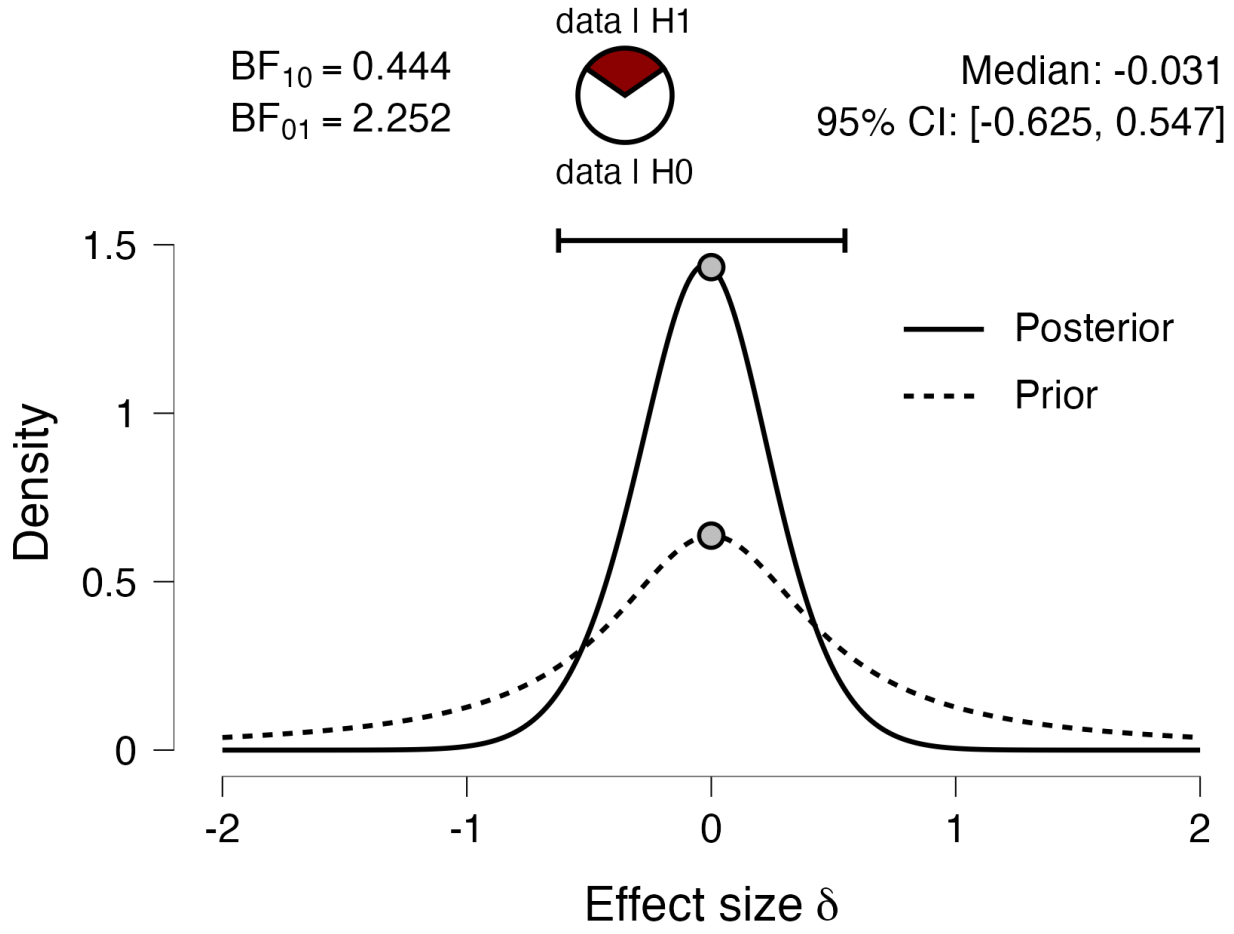
Prior and Posterior Distributions for Bayesian Independent Samples T-Test for Frontal Alpha Asymmetry Scores in the Eyes Open Condition



Note. This figure illustrates the prior and posterior distributions of the Bayesian independent samples t-test along with the corresponding Bayes factor illustrating the strength in favor of the alternative (BF_{10}) and null (BF_{01}) hypotheses.

Figure 8.

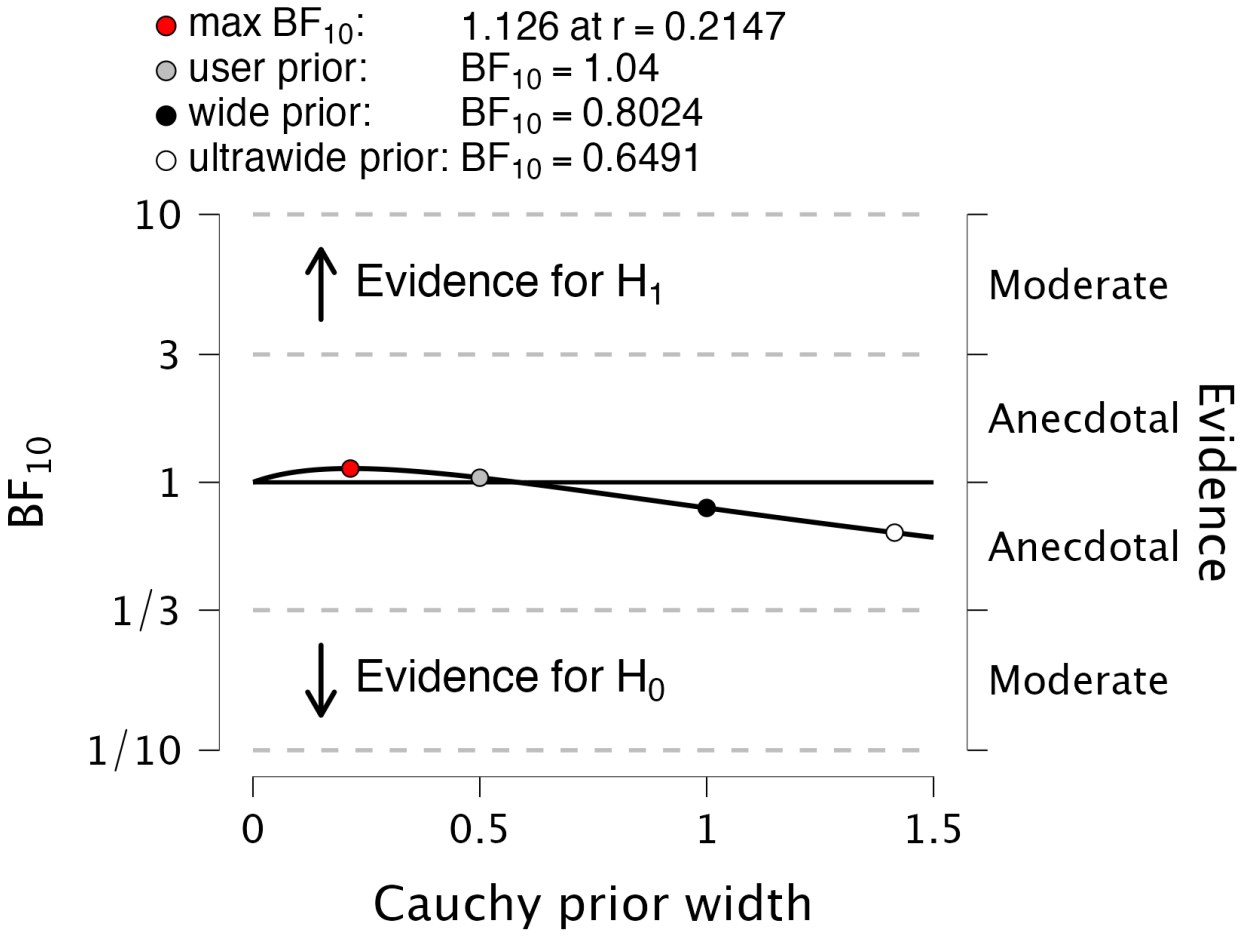
Prior and Posterior Distributions for Bayesian Independent Samples T-Test for Frontal Alpha Asymmetry Scores in the Eyes Closed Condition



Note. This figure illustrates the prior and posterior distributions of the Bayesian independent samples t-test along with the corresponding Bayes factor illustrating the strength in favor of the alternative (BF_{10}) and null (BF_{01}) hypotheses.

Figure 9.

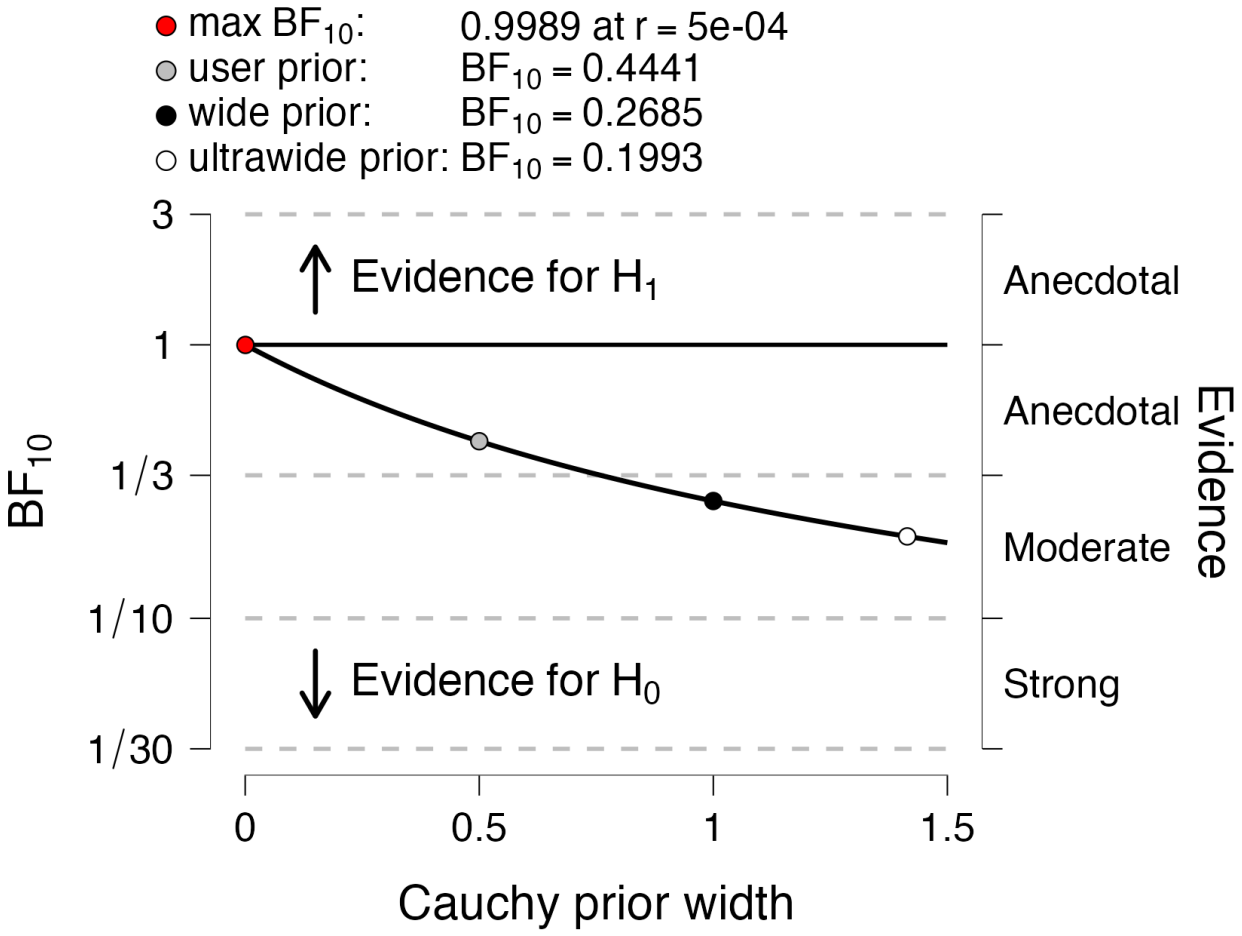
Bayes factor Robustness Check for Bayesian Independent Samples T-Test for Frontal Alpha Asymmetry Scores in the Eyes Open Condition



Note. This figure describes how the Bayes factor would compare if different widths of the prior distribution were used. BF_{10} = Strength of alternative hypothesis comparative to null hypothesis

Figure 10.

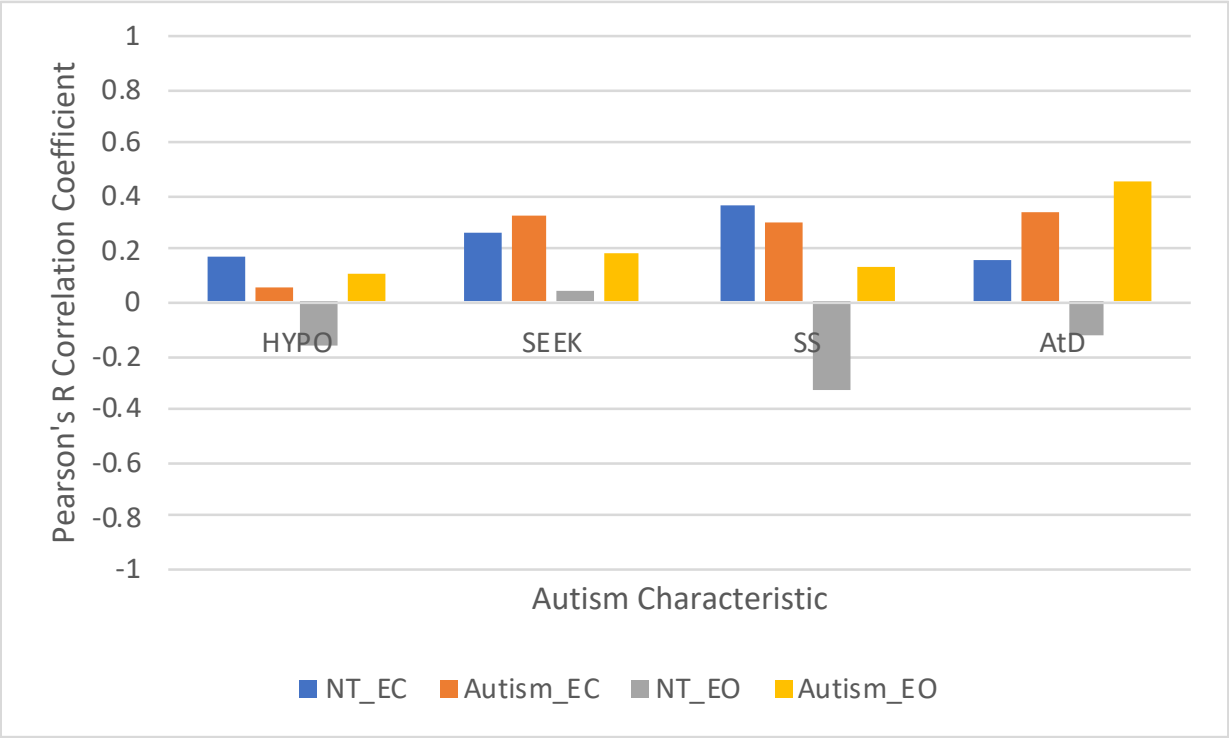
Bayes factor Robustness Check for Bayesian Independent Samples T-Test for Frontal Alpha Asymmetry Scores in the Eyes Closed Condition



Note. This figure describes how the Bayes factor would compare if different widths of the prior distribution were used. BF_{10} = Strength of alternative hypothesis comparative to null hypothesis

Figure 11.

Correlations Between Frontal Alpha Asymmetry and Autism Characteristics by Condition



Note. NT_EC = neurotypical group eyes closed condition; autism_EC = autism group eyes closed condition; NT_EO = neurotypical group eyes open condition; autism_EO = autism group eyes open condition. Hypo = frontal alpha asymmetry correlation with hyporesponsivity scores; Seek = frontal alpha asymmetry correlation with sensory seeking scores; SS = frontal alpha asymmetry correlation with social skills scores; AtD = frontal alpha asymmetry correlation with attention to detail scores.

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Curriculum Vitae

Jarryd Osborne

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EDUCATION

2021-Present

Graduate Student, School Psychology Program (APA accredited & NASP approved)
Syracuse University, Syracuse, NY
School Psychology, PhD

Primary Advisor: Natalie Russo, Ph.D., Licensed Psychologist
Master's Thesis: *Relationship Between Resting-State Frontal Alpha Asymmetry and Autism Characteristics Among Typically Developing and Autistic Children and Adolescents*

2014-2018

Bachelor of Arts with Honors in Neuroscience, Certificate in Cognitive Science
Princeton University, Princeton, NJ

Primary Advisor: Kenneth Norman, Ph.D.
Honors Thesis Title: *Examining how pattern matching for multiple distributions is impacted by time*

Cumulative GPA: 3.44

PUBLICATION

Antony, J. W., Stiver, C. A., Graves, K. N., **Osborne, J**., Turk-Browne, N. B., & Bennion, K. A. (2021). Spatial gist extraction during human memory consolidation. *Journal of Experimental Psychology: Learning, Memory, and Cognition*.

RESEARCH GRANTS AND AWARDS

Professional, Academic, and Creative Work Grant Travel Award
Syracuse University Graduate School Organization (GSO) - $300
2022

Travel Award
Syracuse University Interdisciplinary Graduate Neuroscience Concentration (IGNC) - $500
2022

CLINICAL EXPERIENCE

Graduate Student Assessment Technician
SUNY Upstate Medical University, Syracuse, NY
Supervisor: Deborah E. Spinks, Ph.D., Licensed Psychologist
2023-Present

Responsibilities:

- Conduct neuropsychological assessments for children and adults with histories of pediatric cancer, brain tumors, or other early developmental diagnoses expected to impact cognitive functioning
- Score and interpret assessments to inform diagnoses and recommendations
- Write integrated reports for families summarizing the results of the evaluation and providing recommendations
- Collaborate with a multidisciplinary care team including oncologists, teachers, and mental health professionals to try and ensure recommendations and support services are appropriately implemented for patients
- Utilize an electronic medical record software (i.e. EPIC) to manage patient records

Graduate Student Clinician
Center for Autism Research and Electrophysiology (CARE) Lab, Syracuse University
Supervisor: Natalie Russo, Ph.D., Licensed Psychologist
2021-Present

Responsibilities:

- Conduct cognitive and diagnostic evaluations for autism using the ADOS-2, WASI-II and Stanford Binet 5 in participants ranging in ages from 5 through adulthood
- Score and interpret assessments to inform diagnoses
- Write integrated reports for families summarizing the results of the evaluations and providing recommendations

Student Clinician
Psychological Services Center (PSC), Syracuse University
Supervisors: Afton Kapuscinski, Ph.D., Licensed Psychologist, Amy Goodrum, Ph.D., Licensed Psychologist, Amy Wedel, M.S., Madison Firkey, M.S., Natalie Russo, Ph.D., Licensed Psychologist
2022-2023

Responsibilities:

- Provide weekly empirically supported therapy (CBT, ERP, Psychodynamic, etc.) both in-person and via telehealth to children, adolescents, and adults experiencing depression, anxiety, OCD, eating disorders, ADHD, autism, and disruptive mood dysregulation disorder through a university-based clinic
- Provide caregiver training including psychoeducation, behavioural management, and emotional regulation strategies
- Conduct semi-structured intake interviews with adolescents and adults to gather information on their presenting concerns and relevant social, psychological, and medical histories

- Conduct psychological assessments to evaluate for the presence of neurodevelopmental and learning conditions
- Write integrated reports for individuals and families summarizing the results of the assessments and providing recommendations

Social Skills Group Facilitator
Department of Psychology, Syracuse University
Supervisor: Kevin Antshel, Ph.D., Licensed Psychologist
2022-2023

Responsibilities:

- Provide weekly social skills training for a group of children aged 7-12 with ADHD, autism, and anxiety and their caregivers

RESEARCH EXPERIENCE

Graduate Student
Department of Psychology, *Syracuse University*
PI: Natalie Russo, Ph.D., Licensed Psychologist
2021-Present

Responsibilities:

- Collect neurophysiological (i.e., EEG and ERP) data using EGI Net Station to compare how autistic and typically developing individuals perceive the world
- Program and pilot a variety of computer-based experiments using MATLAB
- Aid in task development for new studies
- Conduct independent research focused on assessing differences in alpha asymmetry between autistic individuals and typically developing individuals

Research Technician
Department of Neuroscience, *University of Pittsburgh*
PI: Caroline Runyan, Ph.D.
2018-2020

Responsibilities:

- Used mouse models and two-photon microscopy to investigate how excitatory and inhibitory neurons work together to control the flow of sensory information
- Trained mice on a variety of behavioral tasks and analyzed subsequent behavior data
- Performed viral injection surgeries and headplate surgeries to prepare mice for behavioral and imaging data collection
- Performed perfusions and brain-slicing to image mouse brain tissue
- Conducted administrative tasks including mouse colony management, ordering lab supplies, and scheduling

Summer Research Assistant
Department of Psychology, Queen's University,
PI: Jordan Poppenk, Ph.D.
2017

Responsibilities:

- Assisted in a research project investigating how the posterior and anterior regions of the hippocampus differed with regards to processing memory
- Conducted mock fMRI studies to prepare subjects for real fMRI experiences
- Categorized film stimuli to make markers for emotionally evoking events
- Qualitatively analyzed recorded subject responses to assess level of semantic and descriptive details associated with differing emotionally evoking memories

Undergraduate Research Assistant
Department of Psychology, Princeton University
PI: Nicholas Turk-Browne, Ph.D.
2016-2017

Responsibilities:

- Used MATLAB to track eye movements in infants in a study assessing attention shifting and visual habituation
- Shadowed a Post-Doctorate in the lab who was collecting and analyzing fMRI data using Python for a task investigating the role of the hippocampus in associative prediction for visual stimuli

Undergraduate Research Assistant
Department of Psychology, Princeton University
PI: Lauren Emberson, Ph.D.
2015-2017

Responsibilities:

- Scheduled and ran participants through a variety of MATLAB-based behavioral tasks aimed at investigating how learning mechanisms and the environment interact to shape how individuals learn language
- Developed stimuli for a study investigating how children and adults categorize language based on atypical versus typical exemplars
- Used Icoder to track eye movements in infants for a study investigating infant auditory habituation
- Organized and conducted various pilot studies through Amazon Turk

TEACHING AND UNDERGRADUATE MENTORING EXPERIENCES

Undergraduate Mentor
Department of Psychology, Syracuse University
2021-Present

Responsibilities:

- Train undergraduates on how to run participants through a variety of EEG and behavioral experiments
- Keep track of undergraduate hours in the lab and act as a resource they can use for any lab or school related questions or concerns

PSY205 Teaching Assistant
Department of Psychology, Syracuse University
Supervisors: Jennifer A Clarke, Ph.D., Meredith J Martin, Ph.D.
2021-2022

Responsibilities:

- Taught four weekly recitations to 25 students for an Introduction to Psychology undergraduate course
- Developed a syllabus, made lesson plans, and organized supplemental lecture material
- Graded students' writing assignments and acted as the point-person for any students struggling with coursework

PROEFESSIONAL EXPERIENCES

Level II Teacher
The New England Center for Children
2020-2021

Responsibilities:

- Taught individuals aged 16-22 diagnosed with autism, learning disabilities, and behavior disorders through an applied behavioral analysis framework with the goal of maximizing their skills and independence
- Analyzed data regarding students' academic and behavioral progress via ACE ABA Software System
- Acted as the manager on shift to ensure staff were completing necessary tasks and all students were receiving the care and attention they required
- Administered medication to the students after being trained by a Registered Nurse
- Acted as one student's case manager to ensure they were progressing towards their specific academic and behavioral goals
- Attended IEP meetings for one student to help establish goals for the subsequent school year

Squash Coach
Princeton Junior Squash Program
2014-2018

Responsibilities:

- Coached children of various levels aged 8-18 on how to improve their abilities in the sport of squash

Secretary
Bayridge Medical Center
2009-2014

Responsibilities:

- Completed general secretarial tasks including filing patient records, scheduling appointments, submitting daily billing fees, and bringing patients to the examination rooms

POSTER PRESENTATIONS

Osborne, J., Master, E., Matsuba, E. S. M., Watts, E., MacKenzie, C., Chen, X., Shuter, M., Soto, F. E., & Russo, N. (2023, April 7). *Preliminary Examination of Resting-State Frontal Alpha Asymmetry and Autism Characteristics Among Typically Developing and Autistic Children and Adolescents*. Poster presented at the Neuroscience Annual Research Day at Syracuse University.

Matsuba, E. S. M., Cary, E., Soto, F. E., Watts, E., MacKenzie, C., **Osborne, J.,** Chen, X., Shuter, E., & Russo, N. (2023, May 3-6). *Finding a Perfect Match: An ERP study comparing Age and IQ Matching*. Poster presented for the International Society for Autism Research Annual Meeting in Stockholm, Sweden.

Soto, F. E., Shea, N., Matsuba, E. S. M., MacKenzie, C., **Osborne, J.,** Chen, X., Shuter, E., Antshel, A., & Russo, N. (2023, May 3-6). *Do ADHD Symptom Clusters Mediate the Link between Autistic Traits & Error-Related Negativity?: An EEG/ERP Study*. Poster

Cary, E. L., Rodrigues, A., Masters, E., Matsuba, E. S. M., MacKenzie, C., **Osborne, J.,** & Russo, N. (2022, May 11-14). *Trauma mediates the relationship between autistic traits and sensory sensitivity and avoiding in adults.* Poster presented at the International Society for Autism Research Annual Meeting in Austin, Texas.

Cary, E. L., Rao, A., Matsuba, E. S. M., Masters, E., MacKenzie, C., **Osborne, J.,** & Russo, N. (2022, May 11-14). *Barriers to an autistic identity: How RRBs may contribute to the underdiagnosis of females.* Poster presented at the International Society for Autism Research Annual Meeting in Austin, Texas.

CONTINUED EDUCATION & TRAINING WORKSHOPS

Autism Diagnostic Observation Schedule, Second Ediction (ADOS-2); Clinical Training for Modules 1-4
2022

Citi Training: Human Subjects Research – Social/Behavioral
2021

CAMPUS INVOLVEMENT

Admissions Committee
Syracuse University, School Psychology Program
2021-Present

Varsity Athlete
Princeton Men's Squash Team
Princeton University

2014-2017

Membership Chair
The Cap & Gown Club
2017-2018

OTHER ACHIEVEMENTS

Captain of the Canadian Junior National Squash Team
World Junior Squash Championships, Namibia
2014

Member of the Canadian Junior National Squash Team
Pan American Junior Squash Championships, Brazil
2013

PROFESSIONAL AFFILIATIONS

International Society for Autism Research
Member
2022 - 2023

REFERENCES

Dr. Caroline Runyan, Professor of Neuroscience
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