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# Behavioral Inhibition and Activation as A Modifier Process in Autism Spectrum Disorder: Examination of Self-Reported BIS/BAS And Alpha EEG Asymmetry

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## Abstract

The Modifier Model of autism spectrum disorder (ASD) suggests that phenotypic variability within ASD is rooted in modifier processes, such as the behavioral inhibition system (BIS) and behavioral activation system (BAS). Among a sample of 53 adolescents with ASD, this study examined associations between (a) self-reported BIS/BAS and frontal and parietal alpha electroencephalogram asymmetry and whether these indices related to (b) ASD severity (via the Autism Quotient), and/or (c) co-occurring anxiety and attention-deficit hyperactivity disorder (via Youth Self Report and Child Behavior Checklist). Findings showed that alpha asymmetry was associated with self-reported BAS scores, such that greater BAS was related to greater right-frontal hemisphere activation and relatively greater left-parietal hemisphere activation. Additionally, associations emerged between ASD severity and self-reported BAS and alpha asymmetry, and between anxiety symptoms and self-reported BIS and alpha asymmetry. Furthermore, mediation analyses revealed that BAS mediated the association between asymmetry and autism severity. Therefore, alpha asymmetry and BIS/BAS activity may provide insight into how ASD presents in adolescence as well as who might be at greater risk for developing co-occurring psychopathologies. This study highlights the importance of considering motivational systems to elucidate individual differences among youth with ASD and working toward the longer term goal of better understanding differential responses to treatment.

## Lay Summary

Differences in the likelihood to avoid (behavioral inhibition system; BIS) or approach (behavioral activation system; BAS) situations are thought to relate to patterns of brain activity (via electroencephalogram asymmetry asymmetry). This study revealed that these tendencies may influence the presentation of autism spectrum disorder (ASD) and symptoms of anxiety in adolescents with ASD.

Despite a well-defined common core of symptoms in autism spectrum disorder (ASD), including challenges with communication, social relationships, and restricted/repetitive behaviors (APA, 2013), there is a great deal of heterogeneity in its presentation. The Modifier Model of ASD posits that

phenotypic variability within ASD stems from a combination of processes (Mundy, Henderson, Inge, & Coman, **2007**). Variability is commonly believed to result from syndrome-specific biological initial causal processes; modifier processes, however, are an additional source of variation nonspecific to ASD that are related to individual differences among all people. As discussed by Mundy et al. (**2007**), modifier processes interact with autism and contribute to variability in the manifestation of behavioral and psychological differences. Such processes may include differences in social awareness and attributions, self-monitoring, and, the focus of this study, motivational biases. Understanding these dimensions of variability is important for better identifying subgroups of people with ASD with the long-term goal of elucidating and, perhaps, predicting differential responses to treatment.

The behavioral inhibition system (BIS) and behavioral activation system (BAS) are ways of conceptualizing motivational biases and, as posed by the Modifier Model, may influence the presentation of ASD. Theory suggests that particular neural substrates and patterns of neurological activity underlie the BIS/BAS and, therefore, can be partly captured through lateralization of electroencephalogram (EEG) activity (i.e., EEG asymmetry) in the alpha band (e.g., Sutton & Davidson, **1997**). Here, the literature on BIS/BAS and EEG asymmetry in people with typical development is reviewed, anomalies in BIS/BAS-related processes among people with ASD are discussed, and gaps in the literature are identified. In particular, despite a relatively large body of research on both BIS/BAS and alpha asymmetry in typical development, it is unknown whether self-reported BIS/BAS and alpha asymmetry are associated in people with ASD. It is also unclear how indices of motivational bias (i.e., self-report of BIS/BAS and alpha asymmetry) may relate to factors of heterogeneity, including ASD symptom severity and co-occurring emotional and behavioral conditions. Finally, it has yet to be tested whether the effect of alpha asymmetry on such outcomes can be explained (i.e., mediated) by self-reported BIS/BAS. In light of these considerations, this paper aims to begin to address these gaps in the literature.

## BIS/BAS and Alpha Asymmetry in Typical Development

The BIS/BAS is suggested as a framework for understanding the intersection of regulation, personality, and motivation and, in extension, facets of psychopathology (Carver & White, **1994**; Gray & McNaughton, **2000**). The BIS is responsible for governing behavioral responses involving aversive motivation (i.e., withdrawal, inhibition, or avoidance) and is often associated with more painful emotions such as sadness. These inhibition-related processes are especially sensitive to punishment, negative reinforcement, and novelty (Carver & White, **1994**; McAdams, **2015**). A bias or sensitivity toward BIS activation would facilitate a tendency to withdraw from or avoid novel or undesired situations, people, or events as well as inhibition of behavior associated with aversive consequences. Conversely, the BAS contributes to approach-related behavior, is highly attuned to reward, and is often associated with more pleasurable emotions such as joy (Carver & White, **1994**; McAdams, **2015**), although it can also be related to painful emotions (Carver, **2004**). Bias toward BAS would promote a propensity for initiation and maintenance of goal-directed behaviors, especially for potentially rewarding situations. The BIS and BAS are relatively orthogonal systems and, therefore, all combinations of high and low levels of BIS and BAS likely exist (Gray, **1987**). A 24-item questionnaire, the BIS/BAS Scales (Carver & White, **1994**), is commonly used to assess activity in these systems and has been extensively used to examine individual differences in typically developing samples.

In addition to a questionnaire measure, researchers have examined the neurobiological systems thought to underlie the BIS/BAS, activity which can be captured using EEG (Sutton & Davidson, **1997**). The BIS is composed of noradrenergic and serotonergic pathways and, more specifically, a neuroanatomic network involving the amygdala, hypothalamus, and brainstem, as well as the hippocampus, anterior cingulate, and prefrontal cortex (Carver & White, **1994**; Gray, **1987, 1990**; McAdams, **2015**). Dopaminergic pathways play a central role in the BAS, which has a neural basis in the left dorsolateral prefrontal cortex and medial orbitofrontal regions (Gray, **1990**; McAdams, **2015**; Pizzagalli, Sherwood, Henriques, & Davidson, **2005**). Although the BIS/BAS is posited to involve a combination of cortical and subcortical brain regions, previous research suggests that BIS/BAS activity may be reflected by distinct patterns of EEG asymmetry, most notably in the alpha band. That is, a correlate of BIS/BAS activity is believed to be lateralized cortical activity in one hemisphere compared to the other, specific to alpha band oscillations (Sutton & Davidson, **1997**).

Research has revealed positive associations between BAS and greater left-relative-to-right frontal asymmetry (Amodio, Master, Yee, & Taylor, **2009**; Coan & Allen, **2003**; Harmon-Jones & Allen, **1997**; Sutton & Davidson, **1997**), as well as positive relations between higher BAS-relative-to-BIS activation and greater left-frontal hemisphere activation (Sutton & Davidson, **1997**). Thus, those with a tendency for approach-related behaviors may show increased lateralization of cortical activation to the left hemisphere. Alternatively, some studies have identified positive associations between BIS activity and greater right-frontal hemisphere activation (Sutton & Davidson, **1997**), while others have found no relation between BIS activity and alpha asymmetry (Coan & Allen, **2003**; Harmon-Jones & Allen, **1997**).

Nonetheless, unusually high levels of BIS and BAS activity and alpha asymmetry are believed to lay the foundation for the manifestation of emotional and behavioral difficulties. Alpha asymmetry reflecting greater-relative-right cortical activation in the frontal region and greater-relative-left cortical activation in the parietal region has been linked with internalizing symptoms (Blackhart, Minnix, & Kline, **2006**). In turn, symptoms such as depression and anxiety have also been associated with high levels of self-reported BIS activity (Johnson, Turner, & Iwata, **2003**). Findings also point to correlations between asymmetry indicative of stronger left-frontal cortical activity, approach behavior, and externalizing symptoms such as attention-deficit hyperactivity disorder (ADHD; Keune et al., **2011**); high levels of self-reported BAS activity have also been related to externalizing symptoms (Johnson et al., **2003**; Mitchell & Nelson-Gray, **2006**).

Although much of the research on motivational systems has focused on frontal alpha asymmetry, previous findings also indicate that parietal alpha asymmetry relates to individual differences, including personality traits, emotion processing, and psychopathology (De Pascalis, Cozzuto, Caprara, & Alessandri, **2013**; Hale et al., **2009**; Henriques & Davidson, **1990**; Jaworska, Blier, Fusee, & Knott, **2012**; Stewart, Towers, Coan, & Allen, **2011**). In particular, although evidence is mixed, some suggest an opposite pattern for parietal asymmetry compared to frontal asymmetry; greater leftward parietal activity, rather than rightward activity, seems to pose vulnerability for emotional disruption (Stewart et al., **2011**). Thus, both frontal and parietal asymmetry may be important for activity in the BIS/BAS.

Considering these findings in the typically developing literature, self-reported BIS/BAS and alpha asymmetry might serve as a foundation for better understanding heterogeneity in ASD, in terms of

both autism-specific symptoms as well as common co-occurring internalizing and externalizing psychopathologies (i.e., anxiety and ADHD).

## BIS/BAS and Alpha Asymmetry in ASD

Evidence in the literature points to a critical role of motivational systems in the presentation of ASD, theoretically, behaviorally, and neurologically. It is posited that challenges within the social communication domain of ASD have strong neurobiological underpinnings and are rooted in a lack of motivation to orient to the social world, coupled with a lack of reward associated with social interactions (Social Motivation Theory of Autism; Chevallier, Kohls, Troiani, Brodtkin, & Schultz, **2012**; Dawson, Webb, & McPartland, **2005**). Integrated with a Modifier Process perspective, the *degree* of social challenge in ASD may be, in part, linked with motivation-related processes. That is, in the context of the BIS/BAS, greater levels of autism symptoms are likely associated with particular neurobiological profiles through heightened behavioral inhibition and diminished behavioral activation.

Although research on questionnaire indices of behavioral motivation in ASD is modest in quantity, some studies have identified group-level anomalies in both self- and parent-report on the BIS/BAS scales (Althaus et al., **2015**; Larson, South, Krauskopf, Clawson, & Crowley, **2011**; South et al., **2015**; South, Dana, White, & Crowley, **2011**; South, Larson, Krauskopf, & Clawson, **2010**). Collectively, these studies suggest greater behavioral inhibition among those with ASD compared to typically developing peers, yet it is unclear how BIS/BAS relates to variability within the presentation of ASD. Moreover, although relations between BIS/BAS scales and alpha asymmetry have been examined in the general population (e.g., Sutton & Davidson, **1997**), it remains unknown whether self-reported BIS/BAS activity is linked with alpha asymmetry in ASD.

On a neurological level, Stroganova et al. (**2007**) identified a pattern of leftward parietal and temporal asymmetry in the alpha band among a sample of children with ASD (ages 3–8 years). Taking into account the inverse relation between power and brain activity for alpha band oscillations (see Davidson, Jackson, & Larson, **2000**) and given that greater left-hemisphere alpha activity was observed, this finding can be interpreted such that there may be a profile of greater right-hemisphere activation in ASD. In a sample of children and adolescents with ASD (ages 9–14 years), Sutton et al. (**2005**) found associations between ASD severity and frontal alpha asymmetry; paradoxically, the ASD group exhibited more left-sided-than-right-sided midfrontal activation compared to the controls but, within group, greater ASD severity was associated with more rightward frontal activation. Similar findings were replicated by Burnette et al. (**2011**) in a sample of children and adolescents (ages 8–15 years), such that fewer ASD symptoms were present in children with greater leftward frontal activation; however, these findings were uncovered only among those with an IQ below the median for the sample.

Together, these studies raise the possibility that BIS/BAS-related processes may play a critical role in the presentation of ASD and, in particular, the level of autism symptomatology. That is, although many of the aforementioned studies have identified mean-level differences in motivation-related constructs in ASD, the Modifier Model indicates that such processes may also be important for explaining individual differences within ASD (i.e., within-group variability). Furthermore, it is yet unknown

whether the role of alpha asymmetry, as it pertains to the behavioral presentations (e.g., autism symptom severity), can be explained by individual differences in BIS/BAS activity.

Moreover, the common expression of additional emotional and behavioral challenges that extend above and beyond core ASD symptoms further contributes to the heterogeneous nature of ASD. As previously mentioned, the BIS/BAS framework may help to better understand the presentation of internalizing and externalizing comorbidities within ASD. Anxiety is particularly common among people with ASD; evidence suggests that approximately 40% of youth with ASD also meet criteria for at least one anxiety disorder (van Steensel, Bögels, & Perrin, **2011**), with other estimates ranging from 11% up to 84% (White, Oswald, Ollendick, & Scahill, **2009**). Research suggests that anxiety exacerbates the core symptoms of ASD, such that greater anxiety may lead to social avoidance, greater social skills deficits, and negative social interactions (Bellini, **2006**; Duvekot, Ende, Verhulst, & Greaves-Lord, **2018**). Another especially common co-occurring disorder in ASD is ADHD, with prevalence rates of approximately 28%–30% (Gjevik, Eldevik, Fjæran-Granum, & Sponheim, **2011**; Leyfer et al., **2006**; Simonoff et al., **2008**). Symptoms of ADHD are also believed to add to the central social difficulties in ASD (Factor, Ryan, Farley, Ollendick, & Scarpa, **2017**). It might be that co-occurring anxiety and ADHD symptoms are also linked to patterns of neural activity and motivational tendencies in ASD, either as cause or consequence.

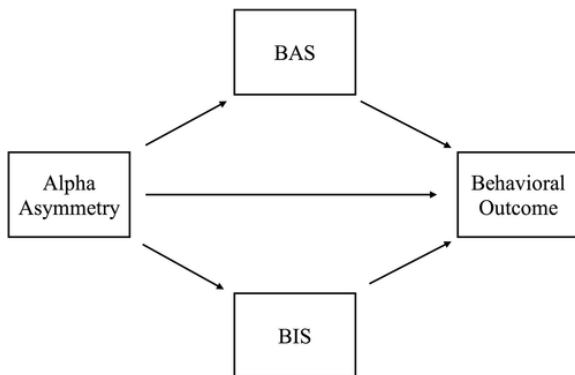
## Summary and Aims of This Study

Although research has suggested that people with ASD may demonstrate differences in alpha asymmetry from their typically developing peers and that motivational bias may explain variability within the ASD phenotype, no study to date has examined self-reported BIS/BAS and alpha asymmetry in ASD. It also remains unclear in what capacity there are relations between both neurological and self-reported measures of motivational biases and autism symptom presentation, as well as co-occurring anxiety and ADHD symptoms. Furthermore, although variability in behavioral presentation (i.e., ASD symptoms, anxiety, etc.) is believed to be linked with alpha asymmetry due to differences in BIS/BAS, this possibility has not been tested.

Given these gaps in the current literature, this study sought to first examine associations among self-reported BIS/BAS activity, alpha asymmetry, autism severity, and comorbidities. More specifically, (a) resting-state frontal and parietal alpha asymmetry with self-reported BIS/BAS scores among adolescents with ASD: it was hypothesized that greater self-reported BAS activity would be associated with greater left-frontal hemisphere activation and that greater BIS activity would be associated with greater right-frontal hemisphere activation, (b) parent-reported measures of autism symptomology with self-reported BIS/BAS activity and alpha asymmetry: it was hypothesized that greater autism severity would be related to greater BIS scores, lesser BAS scores, and greater right-frontal hemisphere activation, (c) self- and parent-reported co-occurring anxiety and ADHD symptoms with BIS/BAS and frontal alpha asymmetry: it was hypothesized that greater BIS scores and greater right-frontal hemisphere activation would be related to higher levels of anxiety symptoms and that greater BAS scores and relatively greater right-frontal hemisphere activation would be linked with higher levels of ADHD symptoms. Hypotheses are posed only for frontal asymmetry; given the relatively smaller foundation of research on parietal asymmetry, these analyses were exploratory in nature and thus, no a priori hypotheses were generated. Second, based on the results of the first aim, this study also



sought to test the mediating role of self-reported BIS/BAS in identified associations between alpha asymmetry and behavioral outcomes (i.e., autism severity, anxiety, or ADHD) (see Fig. 1 for conceptual model). It was hypothesized that links between alpha asymmetry and behavioral outcomes would be mediated by BIS and BAS.



**Figure 1** Conceptual parallel mediation model. *Note.* BIS, Behavioral Inhibition System Subscale; BAS, Behavioral Activation System Subscale.

## Method

### Participants

Participants represent a subsample taken from a randomized controlled trial (RCT) examining the influence of a social skills intervention on brain activity; this study is a post hoc analysis of existing data. For the purposes of this study, only data collected before the intervention were included. For complete details on inclusion criteria and sampling procedures for the RCT, please see Schohl et al. (2014). Fifty-three 11- to 16-year-old adolescents (age: Mean = 13.45, SD = 1.36) completed the measures used in this study, and a subset of 44 had EEG data collected. Two participants were missing self-report data on anxiety and ADHD symptoms and, thus, were omitted from those analyses (but were included in other analyses). Demographics are presented in Table 1. Participant IQ and age were unrelated to variables of interest and, therefore, were not included as a covariate in these analyses.

**Table 1.** Participant Demographics

Variables	Mean	SD	Range
Age	13.45	1.36	11–16
IQ	104.96	19.39	71–144
ADOS-G	10.3	2.93	7–18
Gender (% female)	9.4		
Race (% white)	84.3		
Ethnicity (% non-Latino)	86.8		
Household income (% per category)			
<25,000	13.2		
25,000–50,000	9.4		
50,000–75,000	17.0		
75,000–100,000	18.9		
>100,000	41.5		

*Note.* SD, standard deviation; IQ, composite intellectual quotient as measured by the Kaufman Brief Intelligence Test, Second Edition; ADOS-G, Autism Diagnostic Observation Schedule, Generic.

## Procedure

Approval for the overarching RCT was granted by the Institutional Review Board at Marquette University. Participants provided informed assent and a parent or legal guardian provided informed consent for participation in the RCT. Participants and their parent(s)/caregiver(s) attended a 2- to-3-hour research appointment wherein they completed demographic forms and a battery of questionnaires to assess autism symptomology, social behavior, social knowledge, and co-occurring internalizing and externalizing symptoms. All measures were completed by the adolescent or parent/caregiver themselves or read to them by a research assistant based on their preference. Adolescents were administered a brief measure of IQ, the Kaufman Brief Intelligence Test (KBIT-2; Kaufman & Kaufman, **2004**); participants with a full scale intelligence quotient greater than or equal to 70 were included in this study, per inclusion criteria required for participation in the intervention. The ADOS-G (Lord et al., **2000**), administered by graduate student researchers trained to research reliability within the laboratory, was used to confirm the presence of ASD; participants with a total score equal to or greater than 7 on the ADOS-G were eligible to participate in the study.

For the EEG administration, an appropriately sized 64-channel Electrical Geodesics electrode net was used with reference at Cz and a 1000 Hz sampling rate via Net Amps (Electrical Geodesics, Inc., Eugene, OR) equipment and Net Station data collection software; adjustments were made until impedances were at or below 40 k $\Omega$ . Participants completed a 3-minute eyes open (EO) and 3-minute eyes closed (EC) resting state procedure during which they were seated in a comfortable chair and asked to remain still. For the EO condition, participants were asked to focus on a crosshair presented on a 19" computer screen about 3 feet in front of them and were encouraged to blink as little as possible. For the EC condition, they were asked to keep their EC, stay relaxed, and not fall asleep.

## Measures

### *Behavioral inhibition and activation*

Adolescent self-report of Behavioral Inhibition and Activation Systems was assessed using the BIS/BAS scales (Carver & White, **1994**). The BIS/BAS scales have been shown to have good reliability and validity in both clinical (Campbell-Sills, Liverant, & Brown, **2004**) and nonclinical samples (Jorm, Christensen, Henderson, Jacomb, Korten, & Rodgers, **1998**) and have previously been used in the studies of ASD (South et al., **2011**). Total scores on the BIS (7 items) and BAS (13 items) subscales were used here; for ease of interpretation, items were coded such that high scores reflect high levels of the construct. Internal consistency for the present sample was acceptable for the BAS ( $\alpha = 0.78$ ) and marginally acceptable for the BIS ( $\alpha = 0.64$ ).

### *Co-occurring anxiety and ADHD symptoms*

Adolescents completed a well-validated and commonly used broadband measure to assess for internalizing and externalizing symptoms, the Youth Self Report (YSR; Achenbach, 1995), and parents completed its complement, the Child Behavior Checklist (CBCL; Achenbach & Rescorla, **2001**). The School-Aged form for ages 6–18 years was used. The YSR and CBCL have strong psychometric properties, including good test–retest reliability and validity (Achenbach, **1991**; Achenbach & Rescorla, **2001**) and have previously been used in samples of adolescents with ASD (Kanne, Abbacchi, &

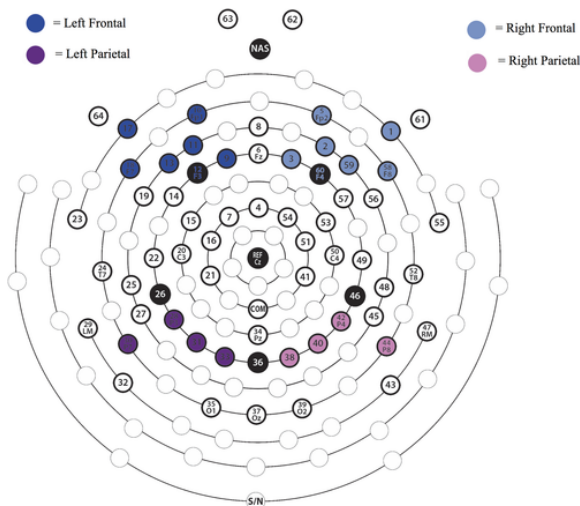
Constantino, **2009**; Mazefsky, Borue, Day, & Minshew, **2014**). The anxiety problems (nine items) and attention-deficit hyperactivity disorder (ADHD) problems (nine items) composite *t*-scores on the YSR and CBCL were used in this study. Internal consistency was acceptable for all subscales used in analyses (YSR anxiety problems  $\alpha = 0.71$ ; YSR ADHD problems  $\alpha = 0.79$ ; CBCL anxiety problems  $\alpha = 0.79$ ; CBCL ADHD problems  $\alpha = 0.79$ ).

#### *Autism symptomology*

A parent or primary caregiver of the adolescent completed a 50-item commonly used measure of autism severity, the Autism Quotient (AQ), which has shown good test–retest and interrater reliabilities (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, **2001**), as well as good convergent and diagnostic validity (Armstrong & Iarocci, **2013**; Woodbury-Smith, Robinson, Wheelwright, & Baron-Cohen, **2005**). The total score was used here; internal consistency was acceptable ( $\alpha = 0.76$ ).

#### *EEG analysis*

EEG data were filtered from 0.3 to 100 Hz, exported from Net Station software (Electrical Geodesics, Inc., Eugene, OR), and then analyzed in MATLAB (2012a, The MathWorks, Natick, MA) using custom scripts involving EEGLAB functions (Delorme and Makeig, **2004**). Data were re-referenced to a common reference (i.e., average reference). Before artifact screening, an 8th-order Butterworth zero-phase filter was used to pass-band filter low-frequency noise from 2 to 100 Hz and notch filter power line noise from 59 to 61 Hz. Data were then epoched into 1-sec periods and the `pop_autorej` function was used to reject epochs (EEGLAB) by first rejecting large fluctuations in potential (default of 1000  $\mu\text{V}$  was used), then data outside of a given SD (default of 5 was changed to 3), with a maximum percent of total trials rejected per iteration set at 10. The average number of epochs included in analyses for the EO condition was 130.02, with an SD of 20.63, and ranged from 69 to 167, and for the EC condition was 128.40, with an SD of 24.45 and ranged from 64 to 161. An adaptive mixture independent component analysis was then applied to the remaining epoched data (Palmer, Makeig, Kreutz-Delgado, & Rao, **2008**). Artifact components were identified using ADJUST (Mognon, Jovicich, Bruzzone, & Buiatti, **2011**), which is designed to use artifact-specific spatial and temporal features to identify eye blinks, vertical and horizontal eye movements, and generic discontinuities. A graduate student researcher trained in EEG visually inspected all data to ensure appropriate artifact component rejection. Remaining data were then Fast Fourier Transformed to calculate the average power spectral density for alpha (8–12 Hz) using Welch's Periodogram approach (1024 pt segments, 50% overlap). Mean power in frontal and parietal electrodes (Fig. 2) was calculated and then natural logarithm transformed to correct for violations of normality inherent in spectral power values. Measures of alpha asymmetry were then derived by subtracting the natural log power in the left from the right hemisphere ( $\ln[\text{right}] - \ln[\text{left}]$ ). Power in the alpha band is inversely related to activation (Davidson et al., **2000**); therefore, positive asymmetry scores indicate relatively greater power in the right hemisphere and, thus, relatively greater left-hemisphere activation, whereas negative asymmetry scores indicate relatively greater power in the left hemisphere and, thus, relatively greater right-hemisphere activation.



**Figure 2** Electrode channel diagram.

## Results

Following spectral and asymmetry analyses, data were imported into SPSS 24.0 for statistical analysis (IBM Corp., 2017). Data were screened for univariate outliers using boxplots and for normality using histograms. There were six outlying alpha asymmetry data points and one outlying BAS data point (0.86% of total data); outliers were winsorized to the next largest/smallest value (EO frontal asymmetry:  $-0.56$  to  $-0.33$  and  $1.25$  to  $0.86$ ; EC frontal asymmetry  $0.95$  and  $0.85$  to  $0.65$  and  $-0.58$  to  $-0.47$ ; EO parietal asymmetry  $-1.19$  to  $-0.78$ ; BAS 23 to 26) (Tabachnick & Fidell, **2013**). Data were subsequently rescreened and found to be within normal limits. Descriptives of EEG data are presented in Table 2. An alpha level of 0.05 was used as the significance criterion for hypothesis tests. *Post hoc* G\*Power analyses were calculated for a two-tailed test with an alpha level of 0.05 and a sample size of 44; results indicated that there was sufficient power (0.98) to detect significance of large effects, but that the study was underpowered to detect moderate (0.53) and small (0.10) effects.

**Table 2.** Descriptives of EEG Variables

	Raw		Ln	
	Mean (SD)	Range	Mean (SD)	Range
EO EEG data				
Left frontal alpha power	7.10 (24.21)	0.58–162.92	0.95 (1.03)	–0.55 to 5.09
Right frontal alpha power	7.81 (24.17)	0.50–162.34	1.13 (1.03)	–0.69 to 5.09
Left parietal alpha power	11.31 (25.76)	0.21–162.08	1.49 (1.24)	–1.55 to 5.09
Right parietal alpha power	11.41 (29.29)	0.39–193.40	1.49 (1.21)	–0.95 to 5.26
Frontal alpha asymmetry			0.18 (0.29)	–0.33 to 0.86
Parietal alpha asymmetry			0.01 (0.36)	–0.78 to 0.71
EC EEG data				
Left frontal alpha power	8.11 (8.61)	0.81–46.25	1.66 (0.95)	–0.21 to 3.83
Right frontal alpha power	9.39 (10.03)	1.20–53.37	1.80 (0.95)	0.18 to 3.98
Left parietal alpha power	28.51 (29.86)	0.70–124.71	2.74 (1.25)	–0.36 to 4.83
Right parietal alpha power	26.07 (23.03)	0.89–94.53	2.74 (1.20)	–0.12 to 4.55
Frontal alpha asymmetry			0.13 (0.28)	–0.47 to 0.65

Parietal alpha asymmetry			0.00 (0.51)	-0.93 to 1.18
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*Note.* EEG, electroencephalogram; EC, eyes closed; EO, eyes open; SD, standard deviation; Ln, natural log.

### Correlations

Pearson correlations were used to examine relations between (a) BIS/BAS scores and alpha asymmetry, (b) AQ scores and both BIS/BAS scores and alpha asymmetry, and (c) self- (YSR) and parent-report (CBCL) of anxiety and ADHD symptoms and both BIS/BAS scores and alpha asymmetry. Correlations among variables are presented in Table 3.

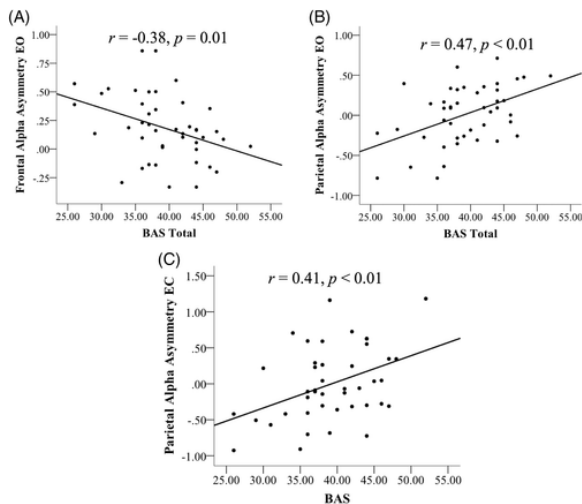
**Table 3.** Correlations and Descriptives of BIS/BAS, Asymmetry, Anxiety, and ADHD

Variables	Mean (SD)	1	2	3	4	5	6	7	8	9	10	11	12
1. BIS	18.75 (4.05)												
2. BAS	39.04 (5.63)	0.28*											
3. F Asym EO	0.18 (0.29)	-0.17	-0.38*										
4. F Asym EC		-0.23	-0.19	0.55**									
5. P Asym EO	0.01 (0.36)	0.09	0.47**	-0.19	-0.25								
6. P Asym EC		0.15	0.41**	-0.14	-0.42**	0.80**							
7. AQ	34.92 (5.83)	0.18	-0.35	0.37*	0.24	-0.30*	-0.31*						
8. YSR Anxiety	56.76 (7.39)	0.40**	0.19	-0.30*	-0.17	0.13	0.12	0.11					
9. YSR ADHD	58.69 (7.63)	0.24	0.17	-0.29	-0.17	0.04	0.10	-0.01	0.47**				
10. CBCL Anxiety	65.24 (9.00)	0.09	0.05	0.00	0.01	0.08	0.13	0.34*	0.37**	0.17			
11. CBCL ADHD	63.43 (7.62)	-0.17	0.09	0.06	0.18	-0.04	-0.04	0.09	0.26	0.49**	0.50**		
12. Age	13.45 (1.36)	0.19	0.15	0.00	-0.17	0.00	0.03	-0.16	0.07	-0.15	0.01	0.03	
13. IQ	104.96 (19.39)	0.01	-0.07	0.11	-0.22	0.04	-0.06	0.07	0.04	0.18	-0.12	0.11	-0.11

*Note.* BIS, Behavioral Inhibition System Subscale; BAS, Behavioral Activation System Subscale; SD, standard deviation; F Asym, Frontal Alpha Asymmetry; P Asym, Parietal Alpha Asymmetry; EC, eyes closed; EO, eyes open; AQ, Autism Quotient; YSR Anxiety, Youth Self Report Anxiety Problems Subscale; YSR ADHD, Youth Self Report Attention-Deficit Hyperactivity Problems Subscale; CBCL Anxiety, Child Behavior Checklist Anxiety Problems Subscale; CBCL ADHD, Child Behavior Checklist Attention-Deficit Hyperactivity Problems Subscale; IQ, Composite Intellectual Quotient as measured by the Kaufman Brief Intelligence Test, Second Edition.

### BIS/BAS scores and alpha asymmetry

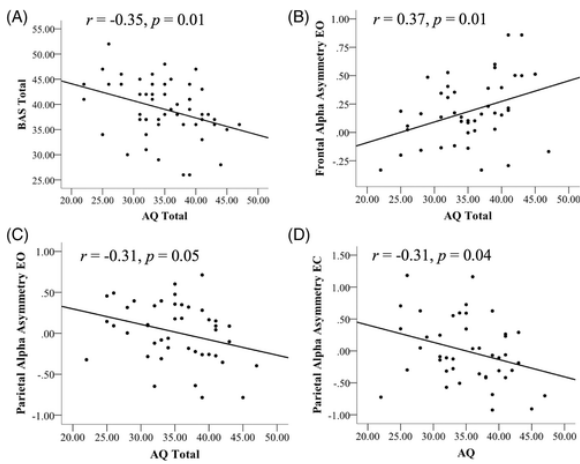
BAS scores were *negatively* associated with *frontal* alpha asymmetry scores ( $r = -0.38, p = 0.01$ ) (Fig. 3A) for the EO condition, thus, greater self-reported behavioral activation was linked with relatively greater right lateralized frontal activation. In contrast, BAS scores were *positively* associated with *parietal* alpha asymmetry scores for both conditions (EO:  $r = 0.47, p = 0.001$ ; EC:  $r = 0.41, p = 0.006$ ) (Fig. 3B, C); thus, greater self-reported behavioral activation was linked with relatively greater left-parietal activation. Scores on the BIS were not significantly associated with either parietal or frontal alpha symmetry in either condition (Table 3).



**Figure 3** Scatterplots showing the relation between the BAS total score and (A) frontal alpha asymmetry EO, (B) parietal alpha asymmetry EO, and (C) parietal alpha asymmetry EC. *Note.* Asymmetry,  $\ln(\text{Right}) - \ln(\text{Left})$ ; BAS, Behavioral Activation System Subscale; EC, eyes closed; EO, eyes open.

### Autism severity, BIS/BAS scores, and alpha asymmetry

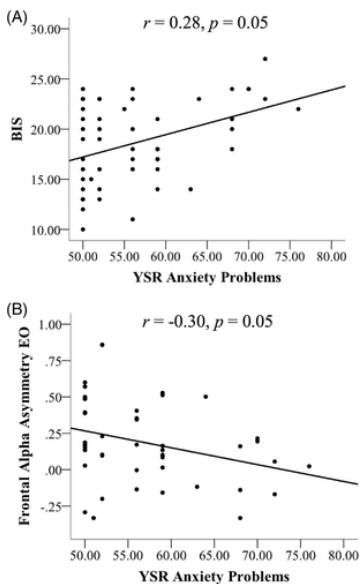
A negative relation emerged between BAS scores and AQ total scores ( $r = -0.35, p = 0.01$ ) (Fig. 4A); this finding indicates that greater self-reported behavioral activation was associated with fewer autism symptoms. There was no significant association, however, between the AQ total score and BIS score ( $r = 0.18, p = 0.18$ ). A significant *positive* link between AQ and *frontal* alpha asymmetry emerged in the EO condition ( $r = 0.37, p = 0.01$ ) (Fig. 4B), thus lesser autism symptomology was related to relatively greater left frontal activation. In contrast, a significant *negative* link between AQ and *parietal* alpha asymmetry emerged for both conditions (EO:  $r = -0.30, p = 0.05$ ; EC:  $r = -0.31, p = 0.04$ ) (Fig. 4C, D), thus, greater autism symptomology was related to relatively greater right parietal activation.



**Figure 4** Scatterplots showing the relation between the AQ total score and (A) BAS total score, (B) frontal alpha asymmetry EO, (C) parietal alpha asymmetry EO, and (D) parietal alpha asymmetry EC. *Note.* Asymmetry,  $\ln(\text{Right})-\ln(\text{Left})$ ; BAS, Behavioral Activation System Subscale; EC, eyes closed; EO, eyes open.

#### *Co-occurring symptoms, BIS/BAS scores, and alpha asymmetry*

There were no associations with parent-reported measures (CBCL) (Table 3). There was a significant positive association between the YSR anxiety problems subscale and BIS score ( $r = 0.28, p = 0.05$ ) (Fig. 5A); therefore, greater levels of self-reported anxiety symptoms were related to higher self-perceptions of behavioral inhibition. There was also a significant negative association between the YSR Anxiety Problems subscale and frontal alpha asymmetry for the EO condition ( $r = -0.30, p = 0.05$ ) (Fig. 5B), thus higher levels of self-reported anxiety symptoms were related to relatively greater right frontal activation.



**Figure 5** Scatterplots showing the relation between the YSR Anxiety Problems subscale and (A) total BIS score and (B) frontal alpha asymmetry. *Note.* Asymmetry =  $\ln(\text{Right})-\ln(\text{Left})$ ; BIS, Behavioral Inhibition System Subscale; YSR, Youth Self Report.

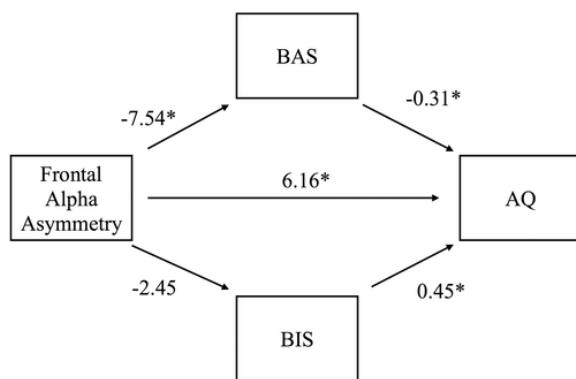


## Mediation analyses

PROCESS for SPSS version 2.16.3 (Hayes, **2013**) in SPSS version 24.0 (IBM Corp., **2016**) was used to test parallel multiple mediator models (Fig. 1). Given the potential of BIS and BAS to operate as both facilitators and antagonists of one another (Corr, **2002, 2004**), this parallel framework tests the independent effects of BIS controlling for BAS and vice versa. This approach also uses bootstrapping to estimate parameters, which is especially well suited for small samples. Based on correlation analyses described above, three mediation analyses were conducted to examine BIS and BAS as mediators of the association between (a) frontal alpha asymmetry (EO only) and autism severity, (b) parietal alpha asymmetry (EO and EC) and autism severity, and (c) frontal alpha asymmetry (EO only) and anxiety.

### *Frontal alpha asymmetry and autism severity*

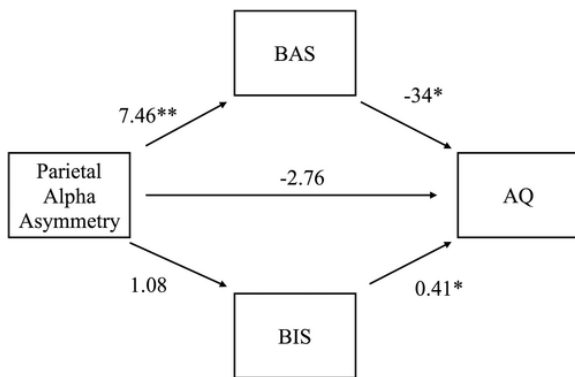
Figure 6 displays the findings for the model that includes EO frontal alpha asymmetry and autism severity. In parallel with correlational findings, EO frontal alpha asymmetry was a significant predictor of BAS ( $B = -7.54$ ,  $SE = 2.87$ ,  $p = 0.01$ ) but not BIS ( $B = -2.45$ ,  $SE = 2.23$ ,  $p = 0.28$ ). Additionally, EO frontal alpha asymmetry ( $B = 6.16$ ,  $SE = 2.94$ ,  $p = 0.04$ ), BAS ( $B = -0.31$ ,  $SE = 0.15$ ,  $p = 0.04$ ), and BIS ( $B = 0.45$ ,  $SE = 0.19$ ,  $p = 0.03$ ) demonstrated significant direct effects on autism severity, explaining 27.42% of the variance in autism severity;  $F(3, 40) = 5.04$ ,  $p < 0.01$ . Additionally, the indirect effect from frontal alpha asymmetry to autism severity through BAS was significant ( $B = 2.37$ , confidence interval [CI]: 0.48–6.17); the indirect effect through BIS, however, was not significant ( $B = -1.11$ , CI: -5.53 to 0.51).



**Figure 6** Parallel mediation model results for frontal alpha asymmetry and autism severity. *Note.* BIS, Behavioral Inhibition System Subscale; BAS, Behavioral Activation System Subscale; AQ, Autism Quotient.

### *Parietal alpha asymmetry and autism severity*

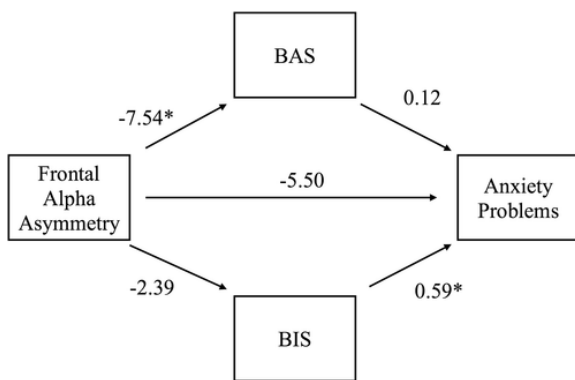
Figure 7 presents the results for the model that includes EO parietal alpha asymmetry and autism severity. BIS ( $B = 0.41$ ,  $SE = 0.20$ ,  $p = 0.03$ ) and BAS ( $B = -0.34$ ,  $SE = 0.17$ ,  $p = 0.05$ ) demonstrated significant direct effects on AQ, whereas the direct effect of EO parietal alpha asymmetry was not significant ( $B = -2.76$ ,  $SE = 2.55$ ,  $p = 0.00$ ); these predictors explained 21.78% of the variance in autism severity  $F(3,40) = 3.71$ ,  $p = 0.02$ . The indirect effect from EO parietal alpha asymmetry to autism severity through BAS ( $B = -2.54$ , CI: -6.58 to -0.22) but not BIS ( $B = 0.44$ , CI: -0.84 to 2.23) was significant. An identical pattern of results was evident for EC parietal alpha asymmetry.



**Figure 7** Parallel mediation model results for parietal alpha asymmetry and autism severity. *Note.* BIS, Behavioral Inhibition System Subscale; BAS, Behavioral Activation System Subscale; AQ, Autism Quotient.

### *Frontal alpha asymmetry and anxiety*

Figure 8 depicts the findings for the model for EO frontal alpha asymmetry and adolescent-reported anxiety symptoms. Direct effects were consistent with correlational findings such that a negative association between EO frontal alpha symmetry and a positive association between BIS and anxiety problems emerged. No significant indirect effects were identified.



**Figure 8** Parallel mediation model results for frontal alpha asymmetry and anxiety. BIS, Behavioral Inhibition System Subscale; BAS, Behavioral Activation System Subscale.

## Discussion

Evidence from this study provides partial support for the hypothesis that alpha asymmetry is related to self-report of motivation tendencies among adolescents with ASD, particularly for behavioral activation. For frontal alpha asymmetry, the direction of association with the BAS was opposite of expected, such that greater rightward frontal activation was associated with higher levels of self-reported BAS scores. This pattern was only evident in the EO condition, suggesting a nuanced role of neural activity specific to state. Perhaps the EO condition captures more of an “inactive state,” rather than a pure rest state due to the addition of visual processing and potential arousal. In fact, global alpha levels have been shown to decrease from EC to EO conditions among adults (Barry et al., 2007) and children (Barry et al., 2009), suggesting an increase in arousal. For parietal alpha asymmetry, however, greater leftward activation, for both EO and EC conditions, was associated with higher levels of self-reported BAS. Because of the stronger correlation between EO and EC conditions for parietal than frontal asymmetry, the greater consistency of findings in parietal across conditions is unsurprising. Together, these results suggest that alpha asymmetry, as it relates to perception of

motivational tendencies, may operate differently within ASD compared to typical development. Additionally, previous research has similarly demonstrated inverse patterns of activity for frontal versus parietal brain regions (i.e., greater right frontal activation and greater left parietal activation) related to depression (Blackhart et al., **2006**; Davidson, Schaffer, & Saron, **1985**; Henriques & Davidson, **1990**). Thus, although additional research is necessary to replicate these findings in comparison to a typically developing sample of adolescents, this inverse pattern of results for frontal and parietal brain regions may reflect roles of different neurofunctional systems.

In addition, consistent with previous literature that has failed to identify a neurological associate of the BIS (De Pascalis et al., **2013**; Wacker, Chavanon, & Stemmler, **2010**), in this study, self-reported levels of BIS were unrelated to alpha asymmetry. These null findings may be due to a number of factors, namely interpretive and psychometric limitations of the BIS scale. Perhaps this null finding is owing to the difficulty in interpretation of BIS. In particular, as an extension of the BIS/BAS theory, the revised Reinforcement Sensitivity Hypothesis refers to the aforementioned BIS and BAS, as well as the Fight–Flight–Freeze System (FFFS). The FFFS mediates reactions to both conditioned and unconditioned aversive stimuli (i.e., avoidance of threat) and is associated with the emotion of fear, whereas the BIS primarily relates to resolving goal conflict (i.e., inhibiting automatic conflicting behaviors) and is associated with the emotion of anxiety. Similarly, Amodio et al. (**2009**) also suggest that pure BIS may correspond to conflict monitoring rather than avoidance. Thus, the BIS scale of the Carver and White's BIS/BAS questionnaire fails to distinguish the FFFS (i.e., evading threat) from the BIS (i.e., resolve current conflict), and, therefore, may contribute to the null findings in the literature and this study for the BIS scale and potential neurobiological correlates.

Moreover, considering the questionable internal consistency of the BIS scale, it might be that self-reported BIS in this study of youth with ASD did not tap into the BIS as it might in typically developing samples. Internal consistency has not been reported upon in other studies of the BIS/BAS in ASD (Althaus et al., **2015**; Larson et al., **2011**; South et al., **2010**), nor has there been psychometric evaluation; therefore, broader generalizations about the use of the BIS/BAS with youth with ASD are not yet possible. Furthermore, the small positive correlation between BIS and BAS scales in this sample is also noteworthy; although these systems are generally orthogonal (all combinations of low and high BIS and BAS are possible), research on the BIS/BAS in non-ASD samples has reported nonsignificant correlations between these scales (Amodio et al., **2009**; Sutton & Davidson, **1997**). To the authors' knowledge, previous studies using the BIS/BAS scales in an ASD sample do not explore the possible correlation between BIS and BAS; thus, it is unclear whether this finding is or is not expected for those with ASD. One possible explanation is that the BIS/BAS may not hold up as truly orthogonal scales or as orthogonal constructs in ASD; tendencies toward inhibition and activation may present differently and thus be captured differently in this population. In fact, previous literature supports a pattern of greater inhibition (BIS) in this population compared to typical development (Althaus et al., **2015**; Larson et al., **2011**; South et al., **2010**; South et al., **2011**; South et al., **2015**). Alternatively, it is possible that there was a response bias in this sample; that is, a tendency for youth to exhibit either nonacquiescence bias (“nay-saying”) or acquiescence bias (“yea-saying”) would lead to consistently higher or lower scores across both subscales (i.e., BIS and BAS). Without a typically developing comparison sample, it is difficult to determine whether these findings are due to limitations of the measure itself or a different functional meaning of alpha symmetry in ASD.

Furthermore, results of this study provide mixed support for the hypothesis involving autism severity and the BIS/BAS. More specifically, elements of the BIS/BAS may modify the expression of ASD, such that greater self-reported BAS activation was related to lesser ASD symptom presentation. Therefore, self-perceptions of heightened behavioral activation were associated with lesser ASD symptoms, whereas self-perceptions of behavioral inhibition were unrelated to ASD symptoms in this sample. Again, the opposite predicted pattern was observed for frontal alpha asymmetry; there was an association between greater left frontal activation during EO and greater ASD symptom severity, as well as between greater right parietal activation during both conditions and greater ASD symptom severity. Although unexpected, this finding for frontal asymmetry is in line with the association between BAS and frontal alpha asymmetry; self-reported BAS and rightward frontal asymmetry both appear to be associated with lesser autism symptoms. The paradoxical finding for frontal asymmetry is also in line with previous work in which Sutton et al. (2005) identified that their ASD sample exhibited greater left dominant activation compared to typically developing youth.

Hypotheses were partially supported for co-occurring anxiety and ADHD symptoms. Self-report, although not parent-report, suggested that a greater tendency toward behavioral inhibition was related to self-reported anxiety symptoms. This pattern was also mirrored in the EEG data, as there was an association between greater right frontal hemisphere activation during the EO condition and heightened anxiety symptoms. This asymmetry finding parallels previous literature on anxiety and alpha asymmetry in typical development (Blackhart et al., 2006). Therefore, perhaps the role of frontal alpha asymmetry in anxiety is distinct from the role of frontal alpha asymmetry in motivational processes. The differences in findings for self- and parent-report may be due to the challenges parents face in accurately reporting upon internalizing symptoms, as they must infer internally experienced emotions (Sourander, Helstelä, & Helenius, 1999; Youngstrom, Loeber, & Stouthamer-Loeber, 2000).

Mediation analyses revealed that the effect of alpha asymmetry (both frontal and parietal) on autism severity was mediated by BAS. These findings are consistent with the theoretical foundation of the Modifier Model; alpha asymmetry may influence variability in autism presentation through differences in motivational tendencies. These data suggest that this effect is evident in particular in behavioral activation but not behavioral inhibition. That is, more rightward frontal asymmetry and leftward parietal asymmetry was related to greater BAS, and in turn, lesser autism symptoms. Interestingly, these analyses also revealed an instance of suppression such that the zero-order association of BIS with autism severity was nonsignificant but in turn became significant when controlling for BAS and asymmetry. Additionally, analyses also indicated that the relation between frontal alpha asymmetry and anxiety was not explained by BIS/BAS. Thus, the effect of BIS on anxiety may be relatively independent of asymmetry.

Together, these findings suggest that behavioral inhibition and activation processes are an important component in the heterogeneity of ASD. Perception of behavioral activation was related to lesser expression of ASD symptoms, whereas perception of behavioral inhibition appears to be related to greater anxiety. Alpha asymmetry may be associated with dampened or amplified expression of ASD and anxiety, with patterns specific to frontal and parietal brain regions. Although these findings may raise more questions than they answer, nevertheless, they suggest that both alpha asymmetry and BIS/BAS may provide insight into how autism severity presents in adolescence as well as who might be

at greater risk for developing co-occurring psychopathologies. Additionally, these individual differences within the present sample of adolescents with ASD may shed light on the disparate findings in the extant literature; depending upon the severity of ASD and presence of co-occurring conditions of the participants in a given sample, different rightward/leftward asymmetries may emerge at the group-wise mean level.

Although additional research is necessary to better understand these motivational processes, the observations from this study have implications for future basic and applied research in ASD. This study adds to a growing body of literature that suggests variability in neurological functioning among those with ASD contingent upon co-occurring symptoms of psychopathology (Herrington et al., **2017**; Herrington, Miller, Pandey, & Schultz, **2016**). Thus, consideration of individual differences in neural processes involved in approach and inhibiting behaviors may serve to facilitate more precise identification of subgroups of youth with ASD. Moreover, this dimension of variability has implications for treatment. Perhaps behavioral activation is an important target for intervention in adolescence. In parallel to interventions in early childhood, which incorporate child-directed play and target social motivation (e.g., Pivotal Response Training; Koegel & Kern Koegel, **2006**), it might be beneficial to follow an adolescent's lead, identify motivating factors, and promote situations in which approach behaviors become reinforcing. This approach may, in turn, enhance social engagement and ameliorate symptoms of ASD. Another possibility is that less symptomatic adolescents have a greater propensity to approach situations; thus, in this case, intervention targeting social skills may in turn enhance motivational for approach-related behaviors.

This study is not without its limitations. One limitation of this study includes the sample of participants in relation to size, age, IQ, and diversity; therefore, these findings may not generalize to the broader population of people with ASD. Although a sizable sample for EEG research, the sample was underpowered to detect significant moderate or small correlational effects; thus, lack of significant effects should be interpreted with caution. The participants in this study were adolescents—this stage of development is laden with dramatic changes in neurobiology (Dahl, **2004**). All participants also had an IQ greater than or equal to 70, and, thus, it is unknown whether these findings would be applicable to those with lower intellectual abilities. The present sample is limited in terms of SES as well as racial and ethnic diversity, which limits the generalizability of the findings to more diverse populations. Additionally, given that this study is cross-sectional and correlational in nature, causality cannot be inferred; additional longitudinal work is necessary to test directionality of these associations. The reliability of the BIS scale comes into question in terms of its psychometric properties in this sample. Finally, as this study does not include a typically developing control sample, it cannot be determined whether motivational activity in this sample is significantly different from that of a typically developing sample.

Overall, the findings from this study support the Modifier Model of ASD and provide insight into the neurobiological underpinnings of the heterogeneity in ASD, both in terms of ASD-specific symptomology and additional comorbidities. This work suggests the importance of considering motivational systems to better understand individual differences among youth with ASD, both for basic and applied research.

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## Conflict of Interest

The authors declared that they have no conflict of interest.

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