



Frontoparietal connectivity, Sensory Features, and Anxiety in Children and Adolescents with Autism Spectrum Disorder

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Abstract

Objectives Because atypical global neural connectivity has been documented in autistic youth, but only limited data are available regarding the association between generalized anxiety disorder (GAD), sensory features (SF), and neural connectivity between frontal and parietal brain regions, these links were investigated in a sample of male autistic children and adolescents.

Methods Forty-one autistic males aged between 6 and 18 years and their mothers were recruited as volunteer participants from Queensland, Australia. Participants underwent 3 min of eyes-closed and 3 min of eyes-opened electroencephalography (EEG) under resting conditions. EEG connectivity was investigated using Granger causality between frontal and parietal regions in alpha (8–13 Hz) and beta (13–30 Hz) bands.

Results There was a significant ($p < .01$) positive correlation between SF and GAD. GAD was associated with some characteristics of SF in the sample population. Additionally, there was a significant ($p < .01$) inverse correlation between directional frontoparietal connectivity and SF during the eyes-closed condition, specifically in relation to avoiding stimuli and sensitivity to the environment.

Conclusions Reduced frontoparietal connectivity in association with higher anxiety and SF may demonstrate reduced relaxation due to greater sensitivity to sensory input.

Keywords Sensory features · Anxiety · Brain connectivity · Behavior · Parietal

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by impairments in social communication and social interaction and the presence of restricted and repetitive behaviors (RRBs) (APA, 2013). A key feature listed under the RRB diagnostic criterion is the presence of sensory features (SF), which refer to hyper- or hypo-sensitivity to sensory input (Dunn, 1999; Schaaf & Lane, 2015). SF have a prevalence of 40 to 90% in autistic individuals (Baranek et al., 2014) and may occur in reaction to auditory, visual, somatosensory, oral, and multisensory stimuli (Baranek et al., 2014). The presence of SF in autistic individuals may be comorbid with affective disorders, particularly anxiety (Bitsika et al., 2016; Pfeiffer et al., 2005). Bitsika et al.'s (2016) study on autistic children and adolescents found that self and parent reports of their child's SF significantly were correlated with generalized anxiety

disorder (GAD), separation anxiety (specifically in regards to hyper-sensitivity to auditory, visual, and touch modalities), and social phobia. These findings are consistent with those from other studies (Ben-Sasson et al., 2009; Bitsika & Sharpley, 2015; Green et al., 2012; Pfeiffer et al., 2005). If left untreated, SF may have a continuous and detrimental impact on the anxiety states of autistic individuals.

One method of investigating the neurobiological bases of ASD is via electroencephalography (EEG), which measures neuronal oscillations over a targeted region of the brain (Schomer & Lopes da Silva, 2017). These oscillations have been typically associated with cognitive (e.g., attention, sensory perception) and motor functions. Based on the electrical signals received from specific brain regions, “functional connectivity” is a statistical dependence measure and evaluates the strength of the communication between, or within, two brain regions (Sporns, 2014). This is different from “power spectra,” which measure neural oscillations from an isolated brain region (Bowyer, 2016; Schomer & Lopes da Silva, 2017). Data on EEG connectivity collected from autistic children and adolescents have consistently

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demonstrated reduced connectivity in lower frequency bands (i.e., delta = 0.5–3.5 Hz, theta = 4–7.5 Hz, alpha = 8–12.5 Hz, beta = 13–30 Hz) and increased connectivity in the gamma (30–80 Hz) frequency band, compared to neurotypical control participants (O'Reilly et al., 2017; Schwartz et al., 2017; Wang et al., 2013). Additionally, several studies have found reduced long-range (> 90 mm) connectivity and increased short-range (< 30 mm) connectivity in autistic individuals (Catarino et al., 2013; Coben et al., 2008; Isler et al., 2010; Lazarev et al., 2015; van den Heuvel et al., 2012; Wang et al., 2013), although there have been some contradictory findings (Duffy & Als, 2012; Elhabashy et al., 2015; Mohammad-Rezazadeh et al., 2016; Murias et al., 2007; O'Reilly et al., 2017). However, much of this research has been focused on *global* connectivity, whereas examination of specific neural pathways that have been associated with anxiety, and their links with SF, have yet to be clearly reported. Investigation of the possible associations between a particular neural pathway, GAD, and SF might help understand how SF is linked with GAD in the autistic brain.

One such neural pathway is the frontoparietal network (FPN), which refers to the neural connections between the frontal region and the parietal region. The FPN is an adaptive cognitive control system which demonstrates extensive connectivity with other brain regions and is correlated with responses to task demands (M. Cole et al., 2014a, b; Genovesio et al., 2014; Ma et al., 2019; Marek & Dosenback, 2018; Sylvester et al., 2012). Therefore, dysfunction in the FPN may not only affect the frontal and parietal regions themselves, and their interaction, but may also disrupt cognitive processing and overall daily functioning. Because of this effect on cognitive functioning, and the important role that cognitive functioning plays in helping individuals deal with anxiety-provoking stimuli (Bishop et al., 2004), it is not surprising that disruption in the FPN is also associated with anxiety that emerges from an inability to solve problems and think through challenges, resulting in GAD in particular (Ball et al., 2013; Cui et al., 2016; Etkin et al., 2009; Liao et al., 2013; Sylvester et al., 2012). There is also evidence of an association between disruption of the FPN and SF (Marek & Dosenback, 2018; Ptak, 2011; Scolari et al., 2015) in the non-autistic population, plus some evidence for an altered FPN in the autistic population (Lin et al., 2019; May & Kana, 2020; O'Reilly et al., 2017; Yuk et al., 2020). However, these findings have primarily focused on executive functioning deficits, rather than on SF and GAD specifically.

In addition, most of these studies (in both autistic and non-autistic samples) used functional magnetic resonance imaging (fMRI) rather than EEG to measure connectivity between frontal and parietal regions. While fMRI is a viable methodology, it provides little information about the brain's electrical activity at varying frequency ranges. This is an important methodological issue because the alpha frequency

is directly associated with relaxation and the beta frequency is associated with cognitive or behavioral alertness (Schomer & Lopes da Silva, 2017). In autistic samples, these cognitive processes have been associated with anxiety (Hollocks et al., 2014; Zimmerman et al., 2017) and SF (Boyd et al., 2009). Measurement of alpha and beta frequency bands, particularly in the resting state (i.e., with minimal input from external stimuli), also enables the assessment of long-range connectivity (such as the FPN) (Hinkley et al., 2011; Marek & Dosenback, 2018). While some studies have demonstrated reduced alpha- and beta-band resting state connectivity between frontal, parietal, and temporal regions in children and adults with ASD as compared with neurologically intact controls (Carson et al., 2014; Coben et al., 2008; Hull et al., 2017; Murias et al., 2007), to the authors' knowledge, no studies have directly addressed the role of the FPN in relation to SF and anxiety in children with ASD.

Based on the strong behavioral association between SF and GAD in the autistic population, the relevance of frontoparietal (FP) connectivity in GAD and SF, and the focus on executive functioning deficits via almost exclusively fMRI methodologies that did not allow for exploration of alpha and beta connectivity between frontal and parietal regions, SF and GAD in the previous literature, the current study aimed to investigate the association between (a) GAD and SF and (b) GAD, SF, and EEG connectivity within the FPN in a sample of autistic children and adolescents. Several methodological decisions were made prior to undertaking the study in order to constrain the possible sources of external invalidity. These included (i) recruitment of autistic males due to the often-reported ratio of 4:1 (male:female) favoring males (APA, 2013); (ii) restricting the sample to males between the ages of 6 years and 18 years to focus on the school-aged group because this study is a discrete part of a larger research project investigating anxiety in autistic youth; (iii) setting an inclusion criterion of a minimum IQ of 70 so that the confounding effects of cognitive disability could be excluded; (iv) collecting SF and GAD data from the participants' parents about their sons because this is the most common procedure used in the literature (Brown & Dunn, 2002; Dunn, 2014; Kientz & Dunn, 1996; van Steensel & Heeman, 2017; van Steensel et al., 2011); (v) collecting EEG data only when the participants were at rest (i.e., not engaged in any specific activity) to exclude possible confounding effects; (vi) focusing on alpha wave and beta wave activity because of their association with anxiety and SF; and (vii) calculating EEG connectivity by Granger causality (Brovelli et al., 2004), one of the most commonly used methods that allows bi-directional connectivity indices to be calculated.

Thus, the specific research questions arising after applying the above methodological conditions were as follows: (i) Is there a significant association between GAD and SF in male children and adolescents with ASD? (ii) Is there

a significant association between GAD and characteristics of SF in male children and adolescents with ASD? (iii) Is there a significant association between GAD, SF, and FP connectivity in male children and adolescents with ASD? While it was reasonably expected that SF and GAD would be associated in similar ways as previously reported (Ben-Sasson et al., 2009; Bitsika & Sharpley, 2015; Green et al., 2012; Pfeiffer et al., 2005), both at the global SF level and in terms of its specific characteristics, expectations regarding the association between SF, GAD, and FPN connectivity were not easily enunciated due to the lack of previous studies that specifically investigated that association using EEG techniques. However, bearing that caveat in mind, it was tentatively expected that, extending the findings from some previous studies, FPN connectivity would be associated with GAD (Ball et al., 2013; Cui et al., 2016), and SF (Marek & Dosenback, 2018; Ptak, 2011; Scolari et al., 2015), although specific hypotheses could not legitimately be stated.

Methods

Participants

Participants were recruited on the Gold Coast, Australia, from responses to publicity delivered to autism support groups, as part of a larger study into the effects of anxiety in autistic youth (Bitsika & Sharpley, 2016), and have not been previously reported. Inclusion criteria were that participants were previously formally diagnosed with ASD, male, between 6 and 18 years of age ($M = 10.76$ years, $SD = 3.14$ years), and had an $IQ \geq 70$. To maintain EEG neural signal validity, exclusion criteria included a history of epilepsy or schizophrenia and intake of anticonvulsant medication.

Following a priori power analysis (described below), 41 male autistic participants aged between 6 and 17 years were recruited for the study. All participants had been diagnosed with ASD several years previously by a registered pediatrician or psychiatrist, and these diagnoses were confirmed during study recruitment using the *Autism Diagnostic Interview-Revised*. One parent of each child was also recruited as participants to provide diagnostic data on their child's ASD, SF, and GAD. Twenty-six of the 41 participants had a secondary diagnosis (primarily ADHD, OCD, anxiety, and depression), and 29 participants were medicated, seven for anxiety. All behavioral and neurophysiological data were collected at the Centre for Autism Spectrum Disorder at Bond University (BUHREC Approval Number: 15786). EEG signal processing and data analyses were conducted in the Behavioural Neuroscience Laboratory at the University of New England (UNE Human Research Ethics Committee Approval Number: HE17-208).

Procedure

During the first visit to the laboratory at Bond University, Australia, participants were administered the *WASI-II* by a research assistant (RA), while the participants' parents completed the *ADI-R* and *CASI-4* with another RA. Parents and their sons were also shown the EEG equipment and given an outline of the experiment to increase familiarity with the procedure. Consent (parents, boys aged 15 years or more) and assent (boys aged 6 years to 14 years) to the experiment were provided during this visit. Following confirmation of the boys' suitability for participation, parents and their sons (who will be referred to as "participants" in the following sections) attended the laboratory on a subsequent day for the EEG session.

Experimental Setting

Stimuli were presented to participants in a dimly-lit, 4 m × 5 m sound-attenuated laboratory. A PC monitor showing the resting EEG data instructions and stimuli was set approximately 0.90 m in front of the participants, with the EEG recording equipment set behind the participants. Participants were videotaped with a Logitech HD Webcam camera to monitor their overt anxious behavior during the experiment and to observe any physiological artifacts (e.g., eye blinks, muscle artifacts) during signal processing.

Experimenter

The procedure was conducted by a doctoral student (who sat behind the participant during the experiment to monitor his behavior and EEG recordings) with experience in working with autistic children.

Experimental Phases

Experimental conditions were always preceded by an adaptation period. In this adaptation period (approximately 15 min), participants were settled into the chair, had the EEG cap and external electrodes fitted, and engaged in minor conversation with the experimenter to ensure that they were calm and prepared for the rest of the protocol. Experimental conditions are described below.

1. Resting eyes-closed condition (3 min): Participants sat still in the chair with their eyes closed, as demonstrated in previous studies (Duffy & Als, 2012; Wang et al., 2013).
2. Resting eyes-opened condition (3 min): Participants were asked to look at the PC screen in front of them, which displayed a black screen with a white circle in the center of the screen. This condition was congruent

with those used in previous studies where participants looked at a dot on a blank screen or just a black screen (Machado et al., 2015; Mathewson et al., 2012) in order to keep participants' reactions to stimuli at a minimal level but also to direct their focus.

Data Acquisition and Pre-processing

Continuous EEG was recorded using a 40-channel NuAmps EEG amplifier from *Compumedics NeuroScan, Compumedics Ltd.* Thirty-four sintered Ag/AgCl electrodes (Quik-cap), four drop-down integrated electrodes, and two auricle electrodes were used with Cz as the chosen reference electrode. Signal pre-processing was conducted using Curry 7, the seventh version of the Curry Neuroimaging Suite also from *Compumedics Ltd.*, and included common average referencing (CAR), notch filter (50 Hz), and a bandpass filter (with default low and high pass frequencies ranging from 0 to 30 Hz) to avoid any power line noise. The sampling rate was 1 kHz. Impedances for the current study for 37 participants were at or below 5 k Ω , to a maximum of 10 k Ω for other participants. Due to sensory sensitivities that are characteristic in this group of participants, the experimenter was mindful to limit abrasion to the scalp.

EEG Signal Processing

All EEG data collected from the eyes-closed and eyes-opened experimental conditions were first processed using a constant baseline correction to eliminate any DC offsets. Filter parameters included the notch filter with harmonics (frequency: 50 Hz; slope: 1.5 Hz) and the bandpass with both low (frequency: 0.5 Hz; slope: 2 Hz) and high (frequency: 30 Hz; slope: 5 Hz) filter settings. Hann filter was used for data tapering to structure the continuous data stretch. The general artifact cleaning procedure for each participant included (i) visual inspection to identify and reject any bad blocks and (ii) using Curry 7 thresholds to identify artifacts in the integrated leads and auricle electrodes. Typical artifacts detected came from ocular (eye blinks, lateral or roving eye movements), electrode, and muscle sources. Depending on the number of detected artifacts for each dataset, one or more of the reduction techniques (i.e., subtraction, covariance, principal component analysis, and independent component analysis [ICA]) embedded within Curry 7 was implemented. For example, EEG data with consistent eye blinks and muscle artifacts required ICA, whereas subtraction may have been used for sparse eye blinks or cardiac artifacts. After applying artifact reduction and elimination techniques, data were extracted to be used for connectivity analyses.

Connectivity analyses were conducted using MATrix LABoratory (MATLAB) R2018b for academic use and two toolboxes: EEGLAB (Delorme & Makeig, 2004) and

FieldTrip (Oostenveld et al., 2011). EEGLAB was used to delete bad blocks that were not identified during Curry 7 artifact reduction and to convert the Curry 7 data format to a compatible version that was accepted by FieldTrip for Granger Causality (GC) analysis. GC (Brovelli et al., 2004) was used to calculate directional connectivity due to its wide usage in EEG effective connectivity research, and in research on the autistic population (Nolte et al., 2010; O'Reilly et al., 2017; Pollonini et al., 2010; Schwartz et al., 2017), and also because of the experimental design used in the current study, which collected stationary data. Relevant to the current study, GC predicts whether one brain region's electrical activity influences another brain region's electrical activity (Brovelli et al., 2004; Ding et al., 2006) and can therefore provide greater insight into the *function* of the connectivity. Other directed or effective connectivity measurements, such as phase slope index (PSI) and transfer entropy (TE), were considered because they may be more robust against field spread and identifying non-linear interactions compared to GC (Kaminski & Blinowska, 1991; Nolte et al. 2010; Nolte et al., 2008; Schreiber, 2000; Vinck et al., 2015). However, these methods have also been shown to be less accurate for detecting direction (Vinck et al., 2015) due to their generality in detecting interaction, which may limit the interpretation of EEG connectivity data (Bastos & Schoffelen, 2016). Because the current study was focused on the FPN in the alpha and beta frequency ranges based on stationary data, GC, as a measure of bivariate linear directional connectivity, was chosen to provide more meaningful information about the ways the selected brain regions interacted with, and were influenced by, each other.

FieldTrip was used to calculate connectivity via GC (Brovelli et al., 2004) due to its wide usage in EEG effective connectivity research, and in research on the autistic population (Nolte et al. 2010; O'Reilly et al., 2017; Pollonini et al., 2010; Schwartz et al., 2017). GC determines if one brain region's electrical activity influences another brain region's electrical activity (Brovelli et al., 2004; Ding et al., 2006) and can therefore provide greater insight into the *function* of the connectivity. Because the current study was focused on the FPN in the alpha and beta frequency ranges, a measure of bi-directional connectivity was determined to provide more meaningful information about the ways the selected brain regions interacted with and were influenced by each other.

The first step in using FieldTrip to calculate GC involved data pre-processing and redefining trials, where each dataset was used in its cleaned format and redefined as having 4-s epochs. The number of available epochs ranged from 35 to 40 4-s epochs, depending on the quality of data for each participant. The second step involved frequency analysis of the redefined dataset, where the multi-taper frequency transformation method (with 5 Hz smoothing) was chosen

to calculate the power spectra (Oostenveld et al., 2011). Fast Fourier transform was designated as the desired output. GC was calculated using a non-parametric bivariate spectral matrix factorization with frequency ranging from 0 to the Nyquist frequency, which is half the frequency of the sampling rate (Oostenveld et al., 2011). The frequency bands of interest extracted for this study were alpha (i.e., 8 to 13 Hz) and beta (i.e., 13 to 30 Hz). Spectrally resolved GC typically ranged from 0 to 1 and was loosely based on the version by Brovelli et al. (2004), as offered through the FieldTrip toolbox (Oostenveld et al., 2011). All relevant mathematical calculations were embedded within the FieldTrip toolbox and did not require user input.

Measures

Autism Diagnostic Interview – Revised (ADI-R)

The *ADI-R* is a standardized and semi-structured interview with the participant's parent to assess previous and current autistic symptoms, following ICD-10 and DSM-IV criteria (Lord et al., 1994). Several studies conducted after the development of the DSM-5 have used the *ADI-R* for research purposes (Isler et al., 2010; Machado et al., 2015; Magana & Vanegas, 2017; Simon et al., 2017). Lord et al. (1994) have demonstrated interrater reliability coefficients for many of the *ADI-R* items to be over 0.70, with no item coefficients below 0.60. Test–retest reliability for the *ADI-R* ranges from 0.93 to 0.97 (Lord et al., 1994). Satisfactory diagnostic, construct, and convergent validity using the *ADI-R* have also been demonstrated (Lecavalier et al., 2006; Saemundsen et al., 2003; Tuschija et al., 2013).

Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II)

The *WASI-II* is a standardized test of general cognitive functioning (Wechsler, 2011). Scores on four *WASI-II* subtests are summed to provide a measure of verbal comprehension and perceptual reasoning, which are then combined to provide a Full Scale IQ (FSIQ) (Wechsler, 2011). The *WASI-II* can be administered to children and adults between the ages of 6 and 90 years. Internal consistency coefficients for the subtests range from 0.87 to 0.91 for the child sample (ages 6 to 16 years) and 0.90 to 0.92 for the adult sample (ages 17 to 90 years) (Wechsler, 2011). Test–retest reliability coefficients for the *WASI-II* subtests range from 0.79 to 0.90 for children and 0.83 to 0.94 for adults; inter-scoring reliability coefficients range from 0.98 to 0.99 for the block design and matrix reasoning subtests, 0.97 for vocabulary, and 0.94 for the similarities subtests (Wechsler, 2011). The *WASI-II* has also been shown to have strong validity with the WISC-IV in autistic samples with an IQ ≥ 70 (Minshe et al., 2005). The

WASI-II was used for the current study to assess participants' FSIQ to ensure that all members of the sample had IQ ≥ 70 .

Child and Adolescent Symptom Inventory, Fourth Revision (CASI-4)

The *CASI-4* (Gadow & Sprafkin, 2010) checklist is a 173-item rating scale which may be completed by parents or other caregivers and is based on the diagnostic criteria for emotional and behavioral disorders outlined in the DSM-5 (APA, 2013). The *CASI-4* is intended to evaluate relevant symptoms in children between 5 and 18 years. The GAD subscale of the *CASI-4* contains eight items drawn from the DSM-IV (and which are current for the DSM-5) measuring the presence of concentration problems, severe worry, difficulties controlling worry, restlessness, irritability, tension, sleeping difficulties, and fatigue. Participants may respond to the *CASI-4* GAD items by ratings of 0 (never), 1 (sometimes), 2 (often), or 3 (very often) about their child's "overall behavior" (Gadow & Sprafkin, 2010), thus providing a measure of severity beyond that from categorical assessment procedures. Psychometric data are satisfactory (Gadow & Sprafkin, 2010) and include test–retest reliability of $r=0.67$ ($p<0.001$) over a six-week period, and internal consistency of 0.74 (Gadow & Sprafkin, 2010).

Child Sensory Profile 2 (CSP-2)

The *CSP-2* was used to measure SF in participants and includes 86 items suitable for the assessment of children and teenagers aged between 3 and 14 years (Dunn, 2014). The items are divided according to sensory sections, which include auditory processing, visual processing, touch processing, movement processing, body position processing, and oral processing. Scores on these sensory sections are combined to form four quadrants (1 = sensory seeking; 2 = sensory avoiding; 3 = sensory sensitivity; 4 = sensory registration). Although the *CSP-2* is intended for ages 3 to 14 years, some previous studies have used the earlier version of the Sensory Profile (SP; Dunn, 1999) (originally intended for ages 3 to 10 years) on adolescents up to the age of 17, with comparable results (Lidstone et al., 2014; Myles et al., 2004). Dunn (2014) has demonstrated that the *SP* and *CSP-2* are moderately to highly correlated ($r=0.39$ to $r=0.87$). The *CSP-2* has been used in several previous studies investigating sensory processing in autistic samples (Burns et al., 2017; Dunn, 2014; Kientz & Dunn, 1996).

CSP-2 items are answered by caregivers based on the frequency of their child's sensory experiences, scored on a scale of 1 to 5 (1 = almost never, 2 = occasionally, 3 = half the time, 4 = frequently, and 5 = almost always) (Dunn, 2014). Dunn (2014) reported that most sensory sections of the *CSP-2* had internal consistency (Cronbach's alpha) of

between 0.70 (adequate) and 0.90 (excellent) or above, with only the visual section of the *CSP-2* rated at 0.60. Test–retest reliability coefficients were reported as from 0.87 to 0.97 for all *CSP-2* quadrants, and interrater reliability coefficients for these quadrants were greater than 0.73 (Dunn, 2014). (Oostenveld et al., 2011). Fast Fourier transform was designated as the desired output. GC was calculated using a non-parametric bivariate spectral matrix factorization with frequency ranging from 0 to the Nyquist frequency, which is half the frequency of the sampling rate (Oostenveld et al., 2011). The frequency bands of interest extracted for this study were alpha (i.e., 8 to 13 Hz) and beta (i.e., 13 to 30 Hz). Spectrally resolved GC typically ranged from 0 to 1 and was loosely based on the version by Brovelli et al. (2004), as offered through the FieldTrip toolbox (Oostenveld et al., 2011). All relevant mathematical calculations were embedded within the FieldTrip toolbox and did not require user input.

Data Analyses

Statistical Package for Social Sciences (SPSS), version 25, was used for all statistical analyses. *G-Power* 3.1 power analysis was performed and showed that, for a correlational study (i.e., the major statistical procedure used to test for associations between SF, GAD, and FP connectivity in this study), a sample size of 40 was sufficient to detect a “moderate” effect (Cohen, 1988) of $r=0.30$ to 0.49 (i.e., accounting for between 9.0 and 24.0% of the variance) with $\alpha=0.05$ and power = 0.95. Appropriate Bonferroni corrections for family-wise error rate were conducted to reduce the likelihood of a type I error where applicable.

Electrodes of interest consisted of areas encompassing the prefrontal cortex and posterior parietal cortex, which have been considered as core components of the FPN (M. W. Cole et al., 2014a, b; Power et al., 2011). Therefore, FP

connectivity was defined as the connectivity between each of the seven Frontal electrodes, Fp1, Fp2, F7, Fz, F3, F4, and F8 with each of the three parietal electrodes P3, Pz, and P4, measured for alpha (i.e., 8–13 Hz) and beta (i.e., 14–30 Hz). Using GC, connectivity was calculated ipsilaterally and contralaterally between each frontal and parietal electrode, with midline electrodes (i.e., Fz, Pz) calculated with all other frontal and parietal electrodes. These connectivity indices were then averaged for each direction of association (i.e., $F \rightarrow P$ and $P \rightarrow F$) to encompass a directional frontoparietal network in alpha and beta frequency bands.

As per the research questions identified in the introduction to this paper, the research aims of the current study were to (i) investigate the relationship between GAD and SF; (ii) explore whether GAD is associated with certain SF; and (iii) identify any association between GAD, SF, and the FPN in the group of participants from the current study. Pearson correlations were used to explore research questions and aims (i) and (iii), and linear regression was used to explore research question and aim (ii).

Results

Descriptive Data

Table 1 presents the means and standard deviations of the *ADI-R*, *WASI-II* FSIQ, *CSP-2*, and *CASI-4* GAD total scores. Inter-item consistency (Cronbach’s alpha) was 0.814, and scale reliability (McDonald’s ω) was 0.823 for the eight GAD items. There were no significant correlations between *WASI-II* FSIQ scores and the *CSP-2* quadrant scores, nor between the *ADI-R* total score and any of the four quadrant scores. There was no significant correlation between participants’ *CSP-2* sensory quadrant scores and their age, or

Table 1 Means and standard deviations for participant age, *WASI-II* FSIQ¹, *ADI-R*² domains, *CASI-4* GAD³, and *CSP-2*.⁴ ($N=41$)

Instrument	Variable	<i>M</i>	SD
WASI-II	Participant age	10.76	3.14
	FSIQ-4	102.10	14.46
ADI-R	Social	19.12	5.11
	Verbal	14.49	4.78
	Restricted and repetitive behavior	6.71	2.35
CASI-4 GAD	GAD total scores	10.10	5.13
CSP-2 sensory quadrants	1: sensory seeking	46.27	18.04
	2: sensory avoiding	58.41	15.12
	3: sensory sensitivity	51.41	17.16
	4: sensory registration	56.80	16.23

¹Wechsler Abbreviated Scale of Intelligence – revised, full-scale score

²Autism Diagnostic Interview – revised

³Child and Adolescent Symptom Inventory, fourth revision, general anxiety disorder subscale total score

⁴Child Sensory Profile, second edition

between participants' *CASI-4* GAD scores and their age (all $p > 0.159$). There was no significant main effect for *CASI-4* GAD total scores or any of the four *CSP-2* quadrant scores, $F(5,35) = 1.252$ (Wilk's Lambda), $p = 0.306$, partial eta squared = 0.152, nor any significant univariate effects (all $p > 0.112$) for the participants who had a secondary diagnosis compared to those with no secondary diagnoses. There was also no significant main effect for participants who were taking medication vs. those who were not, $F(5, 35) = 1.066$, $p = 0.395$, partial eta squared = 0.129, nor any significant univariate effects (all $p > 0.064$).

Table 2 presents the means and standard deviations of the $F \rightarrow P$ and $P \rightarrow F$ connectivity in the alpha and beta frequency ranges for both eyes-closed and eyes-opened conditions.

Univariate normality testing was conducted for *CSP-2* quadrant scores, *CASI-4* GAD total scores, and EEG power values from cleaned data after signal processing across eyes-closed and eyes-opened conditions. The Kolmogorov–Smirnov (K-S) statistics for *CSP-2* quadrant scores and *CASI-4* GAD total scores were nonsignificant and therefore met the criteria for normality. Only 24.1% of EEG power spectra data met the criteria for normality after performing the K-S statistic. However, normalizing data or data transformation may potentially distort the original dataset and increase the likelihood of misinterpreting the results (Tabachnick & Fidell, 2014). In addition, for the current study, most of the data analyses undertaken were correlational, for which non-normality is not a necessary major source of confound (Norris & Aroian, 2004). Therefore, EEG data were not transformed for the current study.

GAD and SF in Children and Adolescents with ASD

To investigate any associations between GAD and SF in the current participant sample, those aspects of SF which

Table 2 Means and standard deviations for $F \rightarrow P^1$ and $P \rightarrow F^2$ directional connectivity under the eyes-closed condition in the alpha (8–13 Hz) and beta (13–30 Hz) frequency ranges

Connectivity direction	Frequency ranges		Eyes closed	Eyes opened
$F \rightarrow P^1$	Alpha	M	0.697	0.553
		SD	0.308	0.245
	Beta	M	0.410	0.388
		SD	0.177	0.174
$P \rightarrow F^2$	Alpha	M	1.764	0.919
		SD	1.166	0.566
	Beta	M	0.685	0.559
		SD	0.352	0.254

¹Frontal to parietal regions

²Parietal to frontal regions

were significantly associated with GAD were identified via Pearson correlations. After applying Bonferroni corrections ($0.05/4 = 0.0125$), there were statistically significant, positive Pearson correlations between participants' *CSP-2* quadrant (Q) scores and *CASI-4* GAD total scores (Q1, sensory seeking: $r = 0.429$, $p = 0.005$; Q2, sensory avoiding: $r = 0.627$, $p = 0.00001$; Q3, sensory sensitivity: $r = 0.508$, $p = 0.001$) but not for Q4, sensory registration ($r = 0.372$, $p = 0.017$). *CSP-2* quadrants 1, 2, and 3 were therefore identified as those which were significantly associated with anxiety and were combined via simple addition to form an overall measure of SF, labeled as Qc or the “combinatory variable” (i.e., $Q1 + Q2 + Q3$).

GAD and Characteristics of SF in Children and Adolescents with ASD

To identify any characteristics of SF (using the *CSP-2*) that may be associated with *CASI-4* GAD total scores in the current participant sample, linear regression analyses were conducted. Linear regression indicated that the previously calculated *CSP-2* combinatory variable made a significant contribution to the variance in the *CASI-4* GAD total score, $F(3,40) = 8.452$, $p = 0.0002$. When the effect of the combinatory variable was separated from the *CSP-2* Q1, Q2, and Q3 scores, Qc made a significant contribution to the variance in GAD individual scores ($F(1,39)$ for change = 21.621, $p = 0.00003$), but the addition of the three quadrants separately did not significantly add to this contribution, $F(2,37) = 1.558$, $p = 0.224$. Based on these results, which deemed the combinatory variable to be associated with GAD total scores, the combinatory variable was used for further analyses.

GAD, SF, and FP Connectivity in Children and Adolescents with ASD

Pearson correlations between the *CSP-2* combinatory variable (Qc), *CASI-4* GAD total scores, and both $F \rightarrow P$ and $F \rightarrow P$ GC in the alpha frequency under the eyes-closed condition are shown in Table 3. Applying the Bonferroni-adjusted p value of $0.05/4 = 0.0125$, one robust negative correlation was found between the *CSP-2* combinatory variable and $F \rightarrow P$ connectivity in the alpha frequency, $r = -0.419$, $p = 0.006$. There were no significant correlations between Qc and $F \rightarrow P$ connectivity, $r = -0.086$, $p = 0.593$, or between Qc and $P \rightarrow F$ connectivity, $r = -0.064$, $p = 0.689$, in the beta frequency.

Due to the variability in significant associations between $F \rightarrow P$ connectivity in the alpha frequency and $F \rightarrow P$ connectivity in the beta frequency, a further calculation was conducted to explore any associations between FP connectivity in alpha and beta frequency bands. FP

Table 3 Pearson correlations for $F \rightarrow P^1$ and $P \rightarrow F^2$ directional connectivity under the eyes-closed condition in the alpha frequency for CASI-GAD³ total scores CSP-2⁴ combinatory variables

Assessment variables		$F \rightarrow P$	$P \rightarrow F$
CASI-GAD total scores	<i>r</i>	-.381	-.327
	<i>p</i>	.014	.037
CSP-2 combinatory variables	<i>r</i>	-.419	-.152
	<i>p</i>	.006	.344

¹Frontal to parietal regions²Parietal to frontal regions³Child and Adolescent Symptom Inventory, generalized anxiety disorder⁴Child Sensory Profile, second edition

connectivity in the beta frequency was not significantly ($p > 0.05$) correlated with FP connectivity in the alpha frequency for both eyes-closed and eyes-opened conditions. Therefore, FP connectivity in the beta frequency was not calculated for further analyses.

For further analysis, Pearson correlations between the three CSP-2 sensory quadrants forming the previous combinatory variable (i.e., sensory seeking [Q1], sensory avoiding [Q2], sensory sensitivity [Q3]) and both $F \rightarrow P$ and $P \rightarrow F$ GC in the alpha frequency under the eyes-closed condition were conducted. These are shown in Table 4 (there were no statistically significant correlations between FP connectivity and sensory features in the alpha frequency under the eyes-opened condition). Applying the Bonferroni-adjusted p value of $0.05/6 = 0.0083$ (to allow for the three quadrants and two directions of FP connectivity), two robust negative correlations were found between the sensory avoiding (Q2) and $F \rightarrow P$ connectivity, $r = -0.466$, $p = 0.002$, and between sensory sensitivity (Q3) and $F \rightarrow P$ connectivity, $r = -0.448$, $p = 0.003$. There was no significant difference for the correlation between Q2 and GAD ($r = 0.627$) and Q3 and GAD ($r = 0.408$): $z = 0.77$, $p = 0.441$.

Table 4 Pearson correlations for $F \rightarrow P^1$ and $P \rightarrow F^2$ directional connectivity under the eyes-closed condition in the alpha frequency for CSP-2³ sensory quadrants

CSP-2 sensory quadrants		$F \rightarrow P$	$P \rightarrow F$
Quadrant 1: sensory seeking	<i>r</i>	-.191	-.050
	<i>p</i>	.231	.757
Quadrant 2: sensory avoiding	<i>r</i>	-.466	-.188
	<i>p</i>	.002	.240
Quadrant 3: sensory sensitivity	<i>r</i>	-.448	-.166
	<i>p</i>	.003	.300

¹Frontal to parietal regions²Parietal to frontal regions³Child Sensory Profile, second edition

Discussion

In this exploratory study, we investigated the relationship between SF, GAD, and the FPN under the eyes-closed and eyes-opened stimulus conditions in 41 participants with ASD aged between 6 and 17 years by asking several research questions. Based on our findings, there was a positive association between autistic participants' SF and generalized anxiety (as in several previous studies). The presence of GAD was also significantly associated with some aspects of SF for those participants who were considered as more sensory seeking, sensory avoidant, and sensory sensitive to their environment than those who were slower to register their environment. Based on the data collected from the eyes-closed condition, FP connectivity was inversely associated with SF in the alpha frequency band (i.e., participants who presented with higher SF typically had decreased FP connectivity in the alpha band), with the frontal region influencing the parietal region. Further exploratory analysis also demonstrated that participants who presented with increased SF characteristics in relation to sensory avoiding and sensitivity had significantly decreased FP connectivity.

In terms of the association between SF and GAD, it is relevant to note that the CSP-2 is based on a sensory processing framework which characterizes the four quadrants using two continua: neurological threshold and self-regulation (Dunn, 1999). The neurological threshold continuum reflects the threshold of how individuals may respond to sensory stimuli; those with a higher neurological threshold are more likely to be hypo-sensitive to their surroundings (Q1 and Q4), whereas those with a lower threshold are more likely to be hyper-sensitive (Q2 and Q3). The self-regulation continuum reflects how individuals may manage their needs; those who are passive may not respond to their environment immediately (Q3 and Q4), whereas those who are active may respond to their environment promptly (Q1 and Q2) (Dunn, 1999). Based on this framework and in relation to the current study, the quadrants may be said to refer to the degree to which a participant may obtain (Q1), avoid (Q2), detect (Q3), and miss (Q4) sensory input in his environment (Dunn, 1999). Only one quadrant (i.e., Q4 or sensory registration) of the four quadrants on the CSP-2 was not associated with CASI-4 GAD total scores after adjusting for multiple comparisons.

The finding that participants with higher Q2 and Q3 scores, encompassing low neurological threshold or hyper-responsivity to sensory stimuli, were more likely to be anxious agrees with previous studies (Ben-Sasson et al., 2009; Bitsika et al., 2016; Pfeiffer et al., 2005). The finding that low sensory registration (Q4) was not significantly correlated with anxiety (due to participants placed in this quadrant having a higher neurological threshold) has also been

found in previous studies (Green et al., 2012; Mazurek et al., 2013). Interestingly, although there has been no reported evidence of an established link between sensory seeking and anxiety (Schauder & Bennetto, 2016), the current study found Q1 to be associated with anxiety. Slightly deviating from the sensory processing framework, this finding may be linked to the “over-arousal hypothesis” which theorizes sensory seeking as a compensatory behavior to distract individuals from aspects of their environment that provoke anxiety (Liss et al., 2006). Applying the over-arousal hypothesis may place participants with higher sensory seeking scores as having a lower neurological threshold and making them more susceptible to anxious behaviors.

From the results of the research question (ii), the severity of GAD may predict some aspects of SF. For the current study, these aspects of SF included atypical sensory seeking, sensitivity, and avoiding. Previous literature has demonstrated an association between anxiety and sensory over-responsiveness in autistic toddlers and children, with sensory over-responsiveness predicting anxiety symptoms (Ben-Sasson et al., 2009; Green et al., 2012; Mazurek et al., 2013). These findings are contradictory to findings from the current study and may demonstrate a need for further research. However, it is important to note that previous studies focused on autistic toddlers (Ben-Sasson et al., 2009; Green et al., 2012) and as the profile of SF and anxiety in autistic individuals has been shown to change in adolescence and adulthood (Schauder & Bennetto, 2016; Uljarevic et al., 2020; van Steensel et al., 2011), it is plausible that the relationship between the two variables may change as well.

Finally, there was a significant inverse relationship found between Qc and F→P connectivity in alpha frequency for the eyes-closed condition. Previous studies have similarly demonstrated reduced alpha connectivity in relation to frontal and parietal regions in the ASD population (O'Reilly et al., 2017); however, no previous research has directly investigated the relationship between the characteristics of SF (via the CSP-2) and FP connectivity. As the FPN is responsible for cognitive flexibility and integration of information from other brain regions, reduced FP connectivity, particularly in the alpha band, may be associated with reduced executive functioning, alertness, and top-down processes such as attention (Padmanabhan et al., 2015; Sadaghiani et al., 2012; Urbain et al., 2016). The current study demonstrated an association between increased sensory sensitivity and sensory avoiding and reduced alpha FP connectivity, suggesting that atypical connectivity between frontal and parietal regions, even in resting conditions, may result in disrupted sensory processing in autistic children and adolescents.

Alpha (i.e., 8 to 13 Hz) frequency refers to a relaxation state (Schomer & Lopes da Silva, 2017). When a person transitions to any activity that requires a complex function

(such as transitioning from an eyes-closed to an eyes-opened condition), alpha waves typically change to asynchronous and higher bands such as beta (i.e., 13 to 30 Hz) waves (Garcia-Rill et al., 2016; Hall, 2016). Although the beta frequency band has been implicated in multisensory processing, anxiety, and cortical arousal (Engel et al., 2001; Hong et al., 2008; Kopell et al., 2000; Rangaswamy et al., 2002; Schomer & Lopes da Silva, 2017; Sheth et al., 2008), in the current study, there was no significant relationship between Qc and FP connectivity in beta frequency, in both eyes-closed and eyes-opened conditions. Furthermore, there was a positive but nonsignificant association between FP connectivity in alpha and FP connectivity in beta, implying that there may be no reciprocity between alpha and beta in the FPN. While further research is needed to validate these findings, targeting neurophysiological interventions, such as neurofeedback or transcranial direct current stimulation that is specific to SF, may help increase alpha connectivity in the FPN in children and adolescents with ASD.

Limitations and Future Research

One of the major limitations of this study was the lack of generalizability on gender, intellectual ability, geo-cultural areas, and research design factors. Although there are relatively few reports of differences in SF across male and female autistic youth (Calderoni, 2022), there are some indications of such differences (Bitsika et al., 2018; Osório et al., 2021). It may be hypothesized that there are female-specific SF profiles that may have specific frontoparietal connectivity and associations with GAD. Similarly, there was an intended age restriction placed upon participant recruitment, and these findings do not generalize to older or younger people with ASD. It is also important to mention that although 26 out of 41 participants with ASD presented with comorbid disorders, and 29 were taking medication for other disorders, there were no significant effects due to the presence of comorbidity or medication. Although deemed unnecessary for the current study (as there have been several comparisons previously), the inclusion of a control group may help with further comparisons that have not previously been made between autistic and non-autistic persons. GC (i.e., effective or directional connectivity method) has been criticized due to issues such as uncertainty in model order, reliability, and degraded quality of GC estimates due to down-sampling (Pagnotta et al., 2018; Stokes & Purdon, 2017).

Although a non-parametric multi-tapered version of GC was used in the current study to counter-balance any potential issues related to model order or down-sampling data, using a pairwise or bivariate approach on sensor-level EEG data may have limited the interpretability of GC in the current study (Dhamala et al., 2008; Pagnotta et al., 2018).

While the focus of the current study was to use bivariate connectivity between only the electrodes encompassing the PFC and PPC accounting for a uniform FPN, overlapping connections from other networks such as the default mode network may further limit the interpretability. GC (i.e., effective connectivity method) was used to reduce volume conduction inherent to data acquisition (Bastos & Schoffelen, 2016; Coben et al., 2014), but source localization was not used, potentially increasing the chances of volume conduction. The use of a 34-channel *QuikCaps*, *Compumedics NeuroScan*, *Compumedics Ltd*, with seven frontal sites and three parietal sites available may have limited the number of detailed analyses conducted on the FPN. Finally, although the current study focused on the association between SF and GAD, there are also data indicating a significant association between SF and depression in autistic male youth (Sharpley et al., 2016) and non-autistic depressed participants (Serafini et al., 2017), but no studies to date have examined the role of frontoparietal connectivity in that association, which might be hypothesized to be similar to that found for GAD in the current study.

The current study has extended previous research by demonstrating that autistic children and adolescents, especially those who have higher SF in relation to *CSP-2* sensory sensitivity and sensory avoiding quadrants, are likely to have reduced FP connectivity. This study also explored the relationship between SF and GAD in these participants, further indicating that the severity of GAD scores may predict some aspects of SF. Taken together, these findings demonstrate that autistic participants with higher severity of GAD and hypersensitivity to sensory input may present with reduced alpha connectivity in the FPN. Generalizing these findings, reduced alpha connectivity in the FPN in association with higher anxiety and SF may demonstrate reduced relaxation, executive functioning, and potentially longer cognitive processing speed due to more sensitivity to sensory input. Therefore, this study emphasizes the need for further exploration of the impacts of SF and anxiety on FP connectivity in children and adolescents with ASD.

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Author Contribution VB, KS, and CFS designed and executed the study and collected the data; KS reduced the EEG data, and CS analyzed the results; KS and CFS wrote the draft of the manuscript; VB, KS, and CFS revised the manuscript.

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Data Availability These data are part of a larger study of anxiety in autistic youth (Bitsika & Sharpley, 2016) and have not been previously reported. Data reported in this manuscript are available from the link to Bitsika and Sharpley (2016) given in the References.

Declarations

Ethics Approval Bond University Human Research Ethics Committee Approval Number: 15786; University of New England Human Research Ethics Committee Approval Number: HE17-208.

Consent to Participate Written consent was given by parents and their sons aged 15 years or more, and verbal assent was given by sons aged 6 to 14 years.

Conflict of Interest The authors declare no competing interests.

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