Neural Plasticity in Response to Intervention in Adolescents with Autism Spectrum Disorders

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NEURAL PLASTICITY IN RESPONSE TO INTERVENTION IN ADOLESCENTS WITH AUTISM SPECTRUM DISORDERS

by

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ABSTRACT
NEURAL PLASTICITY IN RESPONSE TO INTERVENTION IN ADOLESCENTS WITH AUTISM SPECTRUM DISORDERS

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Current theories of Autism Spectrum Disorders (ASD) suggest that they may develop from the transactional interaction between biological risk factors and environmental processes (Dawson et al., 2009). Due to the brain's experience-expectant nature, one's degree of social exposure may have a significant impact on their brain development and behavioral presentation. In addition to the primary critical neurodevelopmental period identified in early childhood, recent research has demonstrated a second period of substantial neurodevelopment during the adolescent period (Sisk & Foster, 2004). This study investigated the neural and behavioral impact of participation in an empirically validated behavioral intervention (The Program for the Education and Enrichment of Relational Skills; Laugeson & Frankel, 2010) during the adolescent years among individuals with ASD. Prior to intervention adolescents with ASD (n=21) differed from their neurotypical peers (n=24) with regard to amount of EEG spectral power across brain locations within the theta and beta frequency bands but not the delta, alpha or gamma frequency bands. Participation in the intervention resulted in increased EEG power in both frequency bands to a degree rendering adolescents with ASD statistically indiscernible from their typically developing peers. Waitlist control subjects (n=22) continued to differ statistically from their neurotypical peers at follow-up assessment. Behavioral change also was observed in response to the intervention, namely increased social exposure and social skills knowledge. No direct correlations could be drawn, however, between neural and behavioral outcomes, suggesting the presence of mediating factors not examined here. A secondary aim of the study was to examine new EEG methodology. Standard continuous EEG procedures complete data collection with subjects in a resting state with no stimuli present. A novel condition involving video and audio presentation of a neurotypical peer providing autobiographical information normally shared in social settings was examined here. No differences were noted between subjects with and without ASD during the novel condition that were not observed in the resting state condition. Taken together, results suggest continued use of standard EEG procedures in the assessment of neurodevelopment in ASD. They also point to adolescence as a crucial period of neural and behavioral development sensitive to behavioral intervention.

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Autism spectrum disorders (ASD) include a set of deficits in social communication and social interaction, and the presence of restricted or repetitive patterns of behavior, interests, or activities (American Psychiatric Association, 2013). The effects of ASD are severe and reach beyond social deficits to affect the emotional health of the individual diagnosed and the mental health and functioning of the entire family system (Benson & Karlof, 2009; Brobst, Clopton, & Hendrick, 2009; White, Oswald, Ollendick, & Scahill, 2009). Furthermore, current costs of raising a child with ASD are staggering and present a significant financial burden to parents and the community at large (Ganz, 2006). The prevalence rate of autism spectrum disorder (ASD) has been rising steadily, increasing over ten-fold over the last 20 years, and is currently estimated at approximately 1 in every 68 children (Centers for Disease Control and Prevention, 2014; Coben, Linden, & Myers, 2010). Furthermore, researchers have indicated that only 68% of the prevalence increase can be attributed to increased awareness and improved diagnostic assessments (Hansen et al., 2008), suggesting that this pattern of increasing prevalence will likely remain an issue for many years to come. ASD has clearly become a major societal health concern and therefore warrants the research attention of those involved in its diagnosis and treatment.

While there is no one known cause of ASD, there are a number of genetic risk factors, behavioral patterns, and environmental features believed to be potential contributors to its development. Dawson and colleagues (2009) have posited a developmental model of risk factors and risk processes leading to symptom emergence in ASD (Figure 1). According to this model, genetic risk factors lead to

reduced experiences of reward in response to social stimuli, causing decreased social motivation and thus reduced attention to social stimuli (Dawson, Sterling, & Faja, 2009). In the typical child, attention to the social environment is instinctual rather than deliberate, and is believed to play a significant role in the development of appropriate social behavior (Rochat & Striano, 1999) and brain development (as reviewed by Johnson, 2001). Researchers therefore suggest that the failure of children with ASD to attend appropriately to the social world may place them at risk for the development of abnormal social behavior. Further compounding these difficulties, many children with ASD are rejected, actively bullied, and isolated by their peers (Symes & Humphrey, 2010; Tse, Strulovitch, Tagalakis, Meng, & Fombonne, 2007), depriving them of the rich social experiences necessary for typical social maturation. These early and secondary environmental differences disproportionally affect the individual due to the vastly experience-expectant nature of brain development in the first half of the lifespan (Galvan, 2010; Greenough, Black & Wallace, 1987; Hebb, 1949; Pascual-Leone et al., 2005; Pascual-Leone et al., 2011; Warralch & Kelm, 2010).

Further, the importance of social experience on behavioral and neural development may differ based on the developmental period considered. In the neurotypical population, the brain is known to progress through a primary critical period during the early childhood years and to reach approximately 90% of its adult size by the age of six years (Casey, Jones, & Hare, 2008); however, recent research has demonstrated ongoing neurodevelopment throughout the lifespan, due in large part to synaptogenesis and pruning (Galvan, 2010). Adolescence, in particular, has

recently attracted attention as a second period of substantial neurodevelopment (Sisk & Foster, 2004). Adolescence is most commonly defined in the literature as that period between the onset of puberty and the acceptance of adult social roles (Dahl, 2004; Spear, 2000). In boys, this period typically occurs between 12-18 years of age (Falkner & Tanner, 1986). This time is one of immense behavioral change, involving an increased focus on socialization (Blakemore, 2008). Typically, adolescents also form increasingly complex peer relationships and become more aware of peer rejection at this stage of life (Brown & Larson, 2004; Steinberg & Morris, 2001). In addition, vast neurodevelopment occurs during this period, consisting of refinement/pruning of gray matter and increases in white matter (Casey, et al., 2008; Giedd et al., 1999; Giedd, 2004; Pfefferbaum et al., 1994; Purves, 1998; Steen, Ogg, Reddick, & Kingsley, 1997). Given the dynamic nature of this period, adolescence will be the primary focus of the current study.

Given interest in autism's neurodevelopmental nature this study will explore the extent to which a standard behavioral intervention can normalize brain development in ASD. Evidence suggesting that neural manipulation can be achieved with behavioral strategies would further support the importance of their implementation in this population. In light of this, we will review the measurement and nature of functional neural differences in ASD. A brief review of interventions for ASD and their effects on neural function will follow. The current study, which examined how neural activity changed due to a social-behavioral intervention for adolescents with ASD, will then be presented.

Functional Neural Differences in ASD: Measurement and Evidence

Mediating the relationship between genetic predisposition and behavioral tendencies in ASD is the central nervous system. Despite the heterogeneity within the ASD population, a number of consistent findings with regard to deviation from typical neural development and activity have been described (as reviewed by Pascual-Leone et al., 2011).

Measurement of Neural Activity via EEG. Direct neural activity (vs. metabolism) can be measured in a variety of ways, the most common of which are magnetoencephalogram (MEG) and electroencephalogram (EEG). The current study will utilize EEG methodology, thus this review focuses on that domain. EEG is a technique that provides information about electrical activity patterns within the brain. The EEG signal is a repetitive, oscillatory wave of activity, and is therefore studied with regard to its sinusoidal waveform components. The wave components discussed in EEG analysis include amplitude and frequency. Amplitude characterizes the wave's magnitude and is communicated in terms of electrical potential, or microvolts (μV). Typically, researchers present EEG findings as they relate to power (amplitude squared) and/or coherence levels (correlations in power levels between brain regions).

Frequency is defined as the wave's rate and is represented in Hertz (Hz), where one wavelength involves movement from baseline to peak, followed by a decrease to a trough, and finally a return increase to baseline. Waveforms of five different frequency ranges are normally discussed in the EEG literature: delta (0.5-4

Hz), theta (4-8 Hz), alpha (8-13 Hz), beta (13-30 Hz), and gamma (30-45 Hz; Blinkowska & Durka, 2006). The delta band has been recognized as a slow wave frequency appearing predominantly during deep sleep (Blinkowska & Durka, 2006; Rippon, 2006). Theta activity has been coined "the fingerprint of all limbic structures" and is believed to serve a gating role in the communication between the thalamus/hypothalamus and cortex during emotional states, and between the prefrontal cortex and posterior association cortex during memory tasks (Blinkowska & Durka, 2006; Lopes da Silva, 1992; Niedermeyer, 1999; Sarnthein, Rappelsberger, Shaw, & von Stein, 1998). Alpha activity, predominant during restful wakefulness (Blinkowska & Durka, 2006; Hughdahl, 1995; Pfurtscheller, Stancak, & Neuper, 1996), has a hypothesized role in memory functioning, as well as long-range communication essential for numerous cognitive processes (Petsche, Kaplan, von Stein, & Filz, 1997; von Stein & Sarnthein, 2000). Beta activity is most commonly associated with alertness and focused attention, and is typically noted during experiments with specific task demands (Blinkowska & Durka, 2006; Rippon, 2006). Beta waves occur in more focal, localized patterns than other waveforms and appear only on the cortex, which suggests a medium-distance 'binding' role for beta activity wherein it serves to synchronize activity between neighboring cortical areas such as the temporal and parietal lobes (von Stein & Sarnthein, 2000). Finally, gamma activity is believed to serve a short-distance communication role within localized cortical and thalamocortical areas (Urbano et al., 2012; von Stein & Sarnthein, 2000), especially during perceptual representations, memory, sensory-memory connections, and problem solving (Hermann, Munk, & Engel, 2004; Tallon-Baudrey,

Bertrand, Peronnet, & Pernier, 1998; Tallon-Baudry, Bertrand, Delpuech, & Pernier, 1997). Overall, the literature is sparse in the area of linking waveforms to the processing of social information.

Evidence for Differences in Neural Activity in ASD. Researchers have described a "social brain" network in the literature that includes the orbito-frontal cortex, temporal cortical areas, and several subcortical structures (Adolphs, 2001; Brothers, 1990). Activity within and between these areas is believed to contribute to typical social behavior, and abnormalities in the social brain network have been identified in ASD. One study performed with children, examining EEG power (i.e., activity) differences in ASD, revealed reduced power in the frontal and temporal regions, with differences more apparent in the left hemisphere (Dawson, Klinger, Panagiotides, Lewy, & Vastelloe, 1995). Lower functional connectivity between cortical regions has also been noted in childhood ASD studies investigating language, working memory, problem-solving, and social cognition (Just, Cherkassky, Keller, & Minshew, 2004; Kana, Keller, Cherkassky, & Minshew, 2006; Koshino, Carpenter, Minshew, Cherassky, Keller, & Just, 2005; Castelli, Frith, Happe, Frith, 2002), suggesting widespread hypoconnectivity in childhood autism. Studies examining adults with ASD have also shown communication deficits, namely local hyperconnectivity in some regions of the brain and medium- and long-range hypoconnectivity (Brock, Brown, Boucher, & Rippon, 2002; Brown, Gruber, Boucher, Rippon, & Brock, 2005; Coben et al., 2008; Murias et al., 2007). Studies examining the social brain network during the presentation of dynamic versus static facial images have revealed enhanced activation in neurotypicals, but not in adults with

ASD (Kilts, Egan, Gideon, Ely, & Hoffman, 2003; Pelphrey, Morris, McCarthy, & LaBar, 2007; Sato, Kochiyama, Yoshikawa, Naito, & Matsumura, 2004). In ASD, the inferior temporal gyri, regions typically mediating object perception, have demonstrated unexpected activation during face processing; meanwhile, the fusiform gyrus, typically responsible for face processing, has been less active than anticipated (Critchley et al., 2000; Schultz et al., 2000).

Another abnormality that appears to emerge in the ASD population is one of atypical functional lateralization. In an EEG study examining cerebral lateralization in children, adolescents, and adults with ASD, Dawson and colleagues (1982) demonstrated right-hemisphere dominance for both verbal and spatial functions in individuals on the autism spectrum. Consistent with this finding, Stroganova and colleagues (2007) have demonstrated a lack of expected leftward asymmetry of the EEG mu rhythm in children on the spectrum, and a study by Sutton and colleagues (2004) has demonstrated functional right-dominant asymmetry patterns in the EEGs of children on the spectrum exhibiting more severe symptomatology and social impairments. A more recent study by Van Hecke and colleagues (2013) also found evidence of right-dominant EEG lateralization in the ASD population during the adolescent years. Using other methodologies, researchers have also demonstrated decreased blood flow and activity levels in the left hemisphere in individuals with ASD (Chiron, Leboyer, Leon, Jambaque, Nuttin, & Syrota,1995), and increased activity in the right frontal and temporal lobes (Kleinhans et al., 2008) of those on the spectrum as compared to control subjects. These atypical functional patterns likely are related to underlying structural abnormalities, most notably the

lack of typical leftward asymmetry in ASD (Haznedar, Buchsbaum, Hazlett, LiCalzi, Cartwright, & Hollander, 2006; Lo et al., 2011; Wan, Marchina, Norton, & Schlaug, 2012), and documented enlargements of the right hemisphere (Herbert et al., 2005).

Intervention for ASD: Evidence Base and Effects on Neural Function

Given the numerous behavioral and neural differences in adolescents with ASD, research has also focused on how to remediate these differences. Evidence-based interventions for this developmental period, and studies of the effects of intervention on neural function in ASD, will be reviewed below.

Interventions for Adolescents with ASD. The establishment of empirically-validated treatments for ASD is a large focus in the field of autism research today. At this time, ASD interventions abound; very few, however, have shown strong research support. The only psychological treatment for ASD that currently meets criteria as a well-established and efficacious intervention and is recommended as evidence-based practice for clinicians by the U.S. Surgeon General (*Mental health: A report of the surgeon general,* 1998) is applied behavior analysis (ABA). ABA is an intensive behavioral treatment focused on the improvement of intellectual skills and adaptive functioning. Unfortunately, ABA is most readily used with young, lower-functioning individuals on the autism spectrum. Studies investigating the success of interventions targeting adolescents who are less cognitively impaired are more limited in number. In a review of the social skills treatment literature, Williams-White and colleagues (2007) examined 14 group-based social skills training programs for children and adolescents. Only one study used a randomized control

group design (Provencal, 2003) and demonstrated significant improvements in symptoms, social knowledge, and social skills, though no manual was utilized, sample size was small (N=20), and the study was not peer-reviewed and published. Only two interventions reviewed by Williams-White and colleagues used manualized treatments (Webb, Miller, Pierce, Strawser, & Jones, 2004; Barnhill, Cook, Tebbenhamp, & Byler, 2002) and no study had a sample size over twenty. Another group-based social skills intervention deserving of mention is Ozonoff and Miller's (1995) 14-week social skills treatment teaching adolescents interactional and conversational skills, as well as theory of mind (how to infer the mental state of others). Improvements in theory of mind were observed following participation in the intervention; however, no improvements were noted in social competence and no generalization of social skills was observed. A more recent study, by Tse and colleagues (2007) attempted to teach social skills to adolescents over a twelve-week period. Findings indicated gains in social competence and decreased problem behaviors; however, no control group was utilized. Finally, a more recent review of the social skills group intervention literature was completed by Reichow and colleagues (2012). The review searched literature spanning from 1948 to 2011 and identified five randomized control trials (RTCs) evaluating the effects of social skills groups in participants with ASD aged 6 to 21 years. Results of the review indicated evidence that social skills groups improve social competence and friendship quality. One of the RCTs included in the review by Cochrane and colleagues that has shown impressive behavioral outcomes is the Program for the Education and Enrichment of Relational Skills (PEERS; Laugeson & Frankel, 2010). PEERS is a manualized

treatment focused both on improving the social skills set of participants, as well as on enriching their social environments by expanding their social networks and increasing the frequency of their exposure to social interactions with peers. The first study examining outcomes of the PEERS intervention demonstrated improvements in social skills, knowledge of how to make and keep friends, quality of friendships, and increased social time with peers (Laugeson et al., 2009). A recent independent replication study also found increased knowledge of social rules, and increased social contact among participants (Schohl, Van Hecke, Carson, Dolan, Karst, & Stevens, 2013). Furthermore, research suggests maintenance of gains at 14-week follow-up after participating in PEERS (Laugeson et al., 2009) and at 1-5 years post-treatment (Mandelberg, Laugeson, Cunningham, Ellingsen, Bates, & Frankel, 2013). Given its success, the PEERS treatment manual has also recently been modified for use in the school setting as a teacher-assisted intervention, and has been translated for use with Korean adolescents. Research examining delivery of the PEERS treatment in the school setting shows similar effectiveness with regard to improving the social skills of teenagers with ASD (Laugeson, Ellingsen, Sanderson, Tucci, & Bates, 2014). The culturally modified and translated version of PEERS introduced to Korean adolescents also demonstrated significant improvements in a number of social domains as well as in co-morbid depressive symptoms (Yoo et al., 2014). Taken together, these findings show strong evidence of improvement secondary to involvement in the PEERS program and suggest a need for further research examining the mechanisms underlying the associated changes.

Developmental Plasticity and Effects of Intervention on Neural Function.

Underlying the behavioral changes of adolescence are drastic structural and functional neural modifications. Although many sources have been suggested as guiding forces in this ongoing neurodevelopment and subsequent shift in behavioral focus (e.g., genetics, nutrition, viruses; Giedd, 2004), many researchers believe that in addition to being under physiological control, changes are occurring on the basis of the "use it or lose it" principle (Giedd, 2004; Huttenlocher & Dabholkar, 1997; Shaw et al., 2008). According to this principle, the recurring stimulation of synapses results in the strengthening of neural synapses, while a lack of stimulation leads to the weakening or elimination of neural connections during synaptic pruning, rendering environmental experiences critically important in the course of healthy neurodevelopment (Galvan, 2010). In addition to neurogenesis and programmed cell death, this concept, also known as 'activity-dependent synaptic plasticity,' is one of the basic mechanisms believed to underlie the phenomenon of neural plasticity (Galvan, 2010). The nervous system's plastic response to environmental demands was first demonstrated in humans by studies noting brain differences in musicians and taxi drivers as compared to controls in areas related to their specialties (Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995; Maguire et al., 2000). Studies using neurofeedback, a technique wherein subjects are trained to willingly alter brain activation patterns via immediate visual feedback of electrical activity, have also put forth evidence of significant neural change as a result of intervention in a number of populations, including ASD (Coben, 2009; Coben et al., 2010; Cowan & Markham,

1994; Jarusiewicz, 2003; Kouijzer, van Schie, de Moor, Gerrits, & Buitelaar, 2010; Linden, 2004; Scolnick, 2005).

More recently researchers have begun to explore the effects of behavioral training programs on neural composition (Masterpasqua & Healey, 2003; May et al., 2007; Pascual-Leone et al., 2011) and have demonstrated neural change in response to treatment in ASD (Dawson et al., 2012; Russo, Hornickel, Nicol, Zecker, & Krauset al., 2010; Pardini et al., 2012; Bolte, Hubl, Feineis-Matthew, Prvulovic, Dierks, & Poustka, 2005; Faja et al., 2012; Vaughan Van Hecke et al., 2013). In very young children with autism, increased EEG activity in response to faces was shown in those who participated in an intensive behavioral treatment (Dawson et al., 2012). In a study examining efficiency of brainstem responses to sound, children with ASD who had received an auditory intervention also showed improvements (Russo et al., 2010). Additionally, a correlational study found an association between white matter integrity and onset and duration of early intervention in children with autism (Pardini et al., 2012). Two studies have also demonstrated short-term neural change in adults with autism in response to social training programs (Bolte et al., 2006; Faja et al., 2012). With regard to adolescents, a recent study demonstrated correction in resting whole brain cerebral asymmetry patterns in those with ASD following intervention (Van Hecke et al., 2013).

As noted in Dawson's model of the risk processes associated with ASD (Dawson et al., 2009), a lack of attention paid to social information may result in the development of abnormal social behavior. During the early childhood years, this

decreased social information processing is largely attributed to a lack of orienting and attention to the stimuli; however, as those with ASD age into adolescence and begin to become isolated from their peers, the question of how impoverished social environments may also impact their development arises. Given the phenomenon of activity-dependent synaptic plasticity, it is reasonable to assume that both a failure to attend to social information as well as decreased exposure to the social world may result in the over-pruning of areas that are normally of great importance for appropriate social behavior. Due to the heightened sensitivity now known to exist in the adolescent brain, it may be imperative that adolescents capitalize on their highly plastic state and the environment's simultaneously enhanced focus on socialization to guide their neurodevelopment to a more typical maturational course and improve their social behaviors.

The Current Study

The current study aims to examine neural responses to intervention in the ASD population during the adolescent period. To this end, this study will explore neural change in response to the evidence-based Program for the Education and Enrichment of Relational Skills (PEERS: Laugeson & Frankel, 2010b). Here, we will examine changes in neural activity as they relate to the PEERS intervention. We will then attempt to correlate those changes with behavioral improvements also resulting from participation in the intervention.

In addition to the collection of EEG data using standard procedures, this study will examine the use of a novel condition in EEG data collection. Standard

continuous EEG procedures complete data collection while the subject is in a resting state with no stimuli present. The novel condition to be investigated in the current study will address this limitation, and will involve a video and audio presentation of a neurotypical peer providing autobiographical information normally shared in social settings. This condition is intended to enhance our understanding of the meaning of EEG abnormalities, specifically, their behavioral implications for the processing of social information.

Given the widespread developmental changes in the brain during the adolescent years, and numerous neural abnormalities noted in the ASD population, a number of brain regions will be investigated via EEG in this study, including the frontal, temporal, and parietal regions. Due to hemispheric differences known to exist in both typical and clinical populations, the left/right separation of these regions is required for accurate assessment of neural activation in the brain regions of interest.

The *first aim* of this study was to examine whether differences in patterns of neural activity existed, at baseline, between adolescents with ASD and their typically developing peers. *The second aim* of the study was to investigate the neural effects of the relationship intervention by determining whether neural activity would differentially change in adolescents with ASD who participated in the PEERS program, and specifically, whether it would bring the neural activity patterns of those adolescents with ASD who participated in the program closer to those patterns seen in typically developing teenagers. The *third aim* of the study was to examine the meaning of any neural changes noted in adolescents who participated

in the intervention by correlating these changes with changes in their behavioral presentations. The *fourth and final* aim of this study was to explore the value of collecting EEG data in this population during a social viewing condition rather than simply during a resting eyes open condition.

Method

Data collection for this study was approved by the Marquette University Internal Review Board (IRB). Data was collected with collaboration from Amy Van Hecke's, Ph.D., laboratory, which included financial support from the Autism Society of Southeastern Wisconsin (ASSEW).

Participants

A total of 140 families were recruited for this longitudinal, randomized controlled trial study (see Figure 2). Recruitment of participants with ASD was completed through local intervention agencies, autism support groups, community advertisements, and an in-house waiting list for the PEERS treatment. Prior to the first appointment, adolescents with ASD were randomly assigned to either the Experimental (EXP) or Waitlist Control (WL) group. These participants completed two research appointments. EXP families began the PEERS treatment immediately after the first research appointment, and completed a second follow-up research appointment at the end of the intervention. Families in the WL group completed an initial research appointment followed by a second follow-up appointment 13 weeks later. Subsequent to their second appointment, WL families entered the PEERS treatment. The PEERS treatment was provided free of charge and included 10 or fewer adolescents and their caregivers per treatment group. Typically developing participants (TYP) for this study were recruited via community advertisements and were seen on only one occasion.

Inclusion criteria for adolescents in the EXP and WL groups were: a) adolescent was between 11-16 years of age at intake, b) adolescent had a verbal and full scale IQ of 70 or greater as measured by the Kaufman Brief Intelligence Test-Second Edition (KBIT-2: Kaufman & Kaufman, 2005), c) adolescent and participating caregiver spoke English fluently, d) adolescent did not have neural, physical, hearing, or visual impairments that prohibited participation in a classroom setting, e) adolescent did not have co-morbid diagnoses of bipolar disorder or schizophrenia, f) adolescent met autism or autism spectrum diagnosis on Module 3 or 4 of the Autism Diagnostic Observation Schedule- Generic (ADOS-G: Lord, Rutter, Dilavore, & Risi, 1999), g) adolescent expressed an interest in receiving the PEERS treatment, and h) adolescent attended at least 12 of the 14 weekly PEERS sessions.

Inclusion criteria for participants in the TYP group included a) through e) listed above, scores under 13 on the Autism Spectrum Screening Questionnaire (ASSQ: Ehlers, Gillberg, & Wing, 1999) and receiving a t-score of 65 or under on all scales of the Child Behavior Checklist (CBCL: Achenbach & Rescorla, 2001).

Given known gender differences in brain development and striking differences in the gender makeup of our sample groups all female subjects were dropped from inclusion in the current study. Furthermore, differences were noted in the number of left- and right-handed subjects among groups within our sample. Previous investigation of neural change in response to the PEERS intervention has demonstrated significant lateralization changes in response to the treatment (Van Hecke et al., 2013). Differences in lateralization patterns have been demonstrated

among individuals with left hand dominance (Guadalupe et al., 2014; Luders E. et al., 2010; Steinmetz, Volkmann, Jancke, & Freund, 1991), therefore, left-handed subjects also were dropped from inclusion in the study.

The final sample included 67 adolescents: 24 TYP subjects, 21 EXP subjects, and 22 WL subjects. Racial background included 92.5% Caucasian, 3.0% Asian American, 3.0% biracial, and 1.5% unspecified. The average participant age was 13.42 (*SD*= 1.60). All subjects demonstrated an IQ of 70 or higher, with an average IQ of 105 (*SD*= 16.44) as assessed on the KBIT-2. Confirmatory diagnostic evaluation of adolescents with ASD on the ADOS-G indicated a mean communication score of 3.56 (*SD*= 1.32), a mean social score of 6.30 (*SD*= 1.70) and a mean total score of 10.00 (*SD*= 2.79). No adolescents received additional psychological therapies for anxiety or depression at or between the research collection sessions. See Table 2 for additional demographic information including data on parental age, education, and income. No significant differences on demographic variables were noted between the EXP, WL, and TYP groups.

With regard to concurrent pharmacological intervention, all adolescents in the TYP group were un-medicated. Of those in the WL ASD and EXP ASD groups, 41.9% were un-medicated during experimentation, 34.9% were receiving one medication, 16.3% two medications, and 6.9% five or more medications. Among subjects receiving medications, 39.13% were receiving antidepressants, 78.26% stimulants, 17.39% atypical antipsychotics, 8.70% alpha-2a receptor agonists, 13.04% mood stabilizers, and 43.48% other medications. Exploratory analyses

indicated only minimal differences in findings with the removal of medicated subjects. Furthermore, removing medicated subjects substantially reduced the study's sample size (N=42) and power. Consideration was given to including medication as a covariate in analyses, however, the use of covariates in repeated measures ANOVA is highly controversial in the literature (Miller & Chapman, 2001) and would provide a skewed statistical picture of group differences given the heterogeneity in medication classes and doses among subjects. Medicated subjects were therefore retained and medication use was not considered further.

Procedures

Families expressing interest in the present study were screened prior to participation via phone or email. Screening involved confirmation of the adolescent's age, diagnostic history, school history, English language ability, motivation to participate in treatment (WL and EXP groups only), and ability to attend weekly PEERS sessions (WL and EXP groups only). Following this screening, families were scheduled for a laboratory intake appointment. Written informed consent and assent were obtained, and participation criteria confirmed. The adolescent and at least one caregiver then completed a number of self-report questionnaires, and an EEG was recorded for the adolescent. Compensations of $30 were given to adolescents in the TYP group at the end of this appointment. Those in the EXP and WL groups received their incentive upon completion of the PEERS treatment. EXP group participants were immediately enrolled in the PEERS program, and WL subjects received no treatment for 13 weeks. Subsequent to

PEERS or a 13-week waiting period, participants returned to complete a second research appointment during which self-report questionnaires were presented for a second time and the adolescent completed another EEG.

Measures

Screening and intake. At the intake visit, caregivers were asked to complete a demographic questionnaire and a questionnaire concerning their adolescent's medical status and list of past and current medications. The cognitive abilities of all adolescent participants were examined using the KBIT-2 (Kaufman & Kaufman, 2005). Adolescents with ASD were interviewed regarding their interest in participating in the group using the Mental Status Checklist (Laugeson & Frankel, 2010a). Diagnoses of ASD were confirmed in EXP and WL teenagers using the ADOS-G, Module 3 or 4, the gold standard among autism diagnostic tools (Tanguay, 2000), and ruled out in TYP subjects using the ASSQ (Ehlers, Gillberg, & Wing, 1999). ADOS-G screenings were completed by trained graduate students. Training required establishing $\geq$ 80% inter-rater reliability on three consecutive administrations with a more experienced gradate student examiner. Other psychopathology also was ruled out in TYP participants using the CBCL (Achenbach & Rescorla, 2001).

Questionnaires. To track behavioral change, caregivers completed a number of questionnaires. The Social Skills Improvement System Rating Scales (SSIS: Gresham & Elliot, 2008) was administered to caregivers. This measure offers an assessment of a variety of social skills such as communication, cooperation, empathy and self-control, as well as competing problem behaviors such as bullying,

externalizing and those characteristic of ASD. The SSIS provides scores on two subscales: Social Skills and Problem Behaviors with higher scores reflecting more of the behavior measured. The Social Skills Score was used in this study. Research has demonstrated that the SSIS has strong reliability and validity on both scales (Gresham, Elliott, Cook, Vance, & Kettler, 2010; Gresham, Elliott, Vance, & Cook, 2011). In the current study, the Cronbach alpha coefficient for the Social Skills scale was .93. The Adolescent Autism Spectrum Quotient (AQ: Baron-Cohen, Hoekstra, Knickmeyer, & Wheelwright, 2006) also was completed by caregivers. The AQ assesses for autistic traits and ultimately provides a Total score in addition to five subscale scores: Social Skills, Attention Switching, Attention to Detail, Communication, and Imagination. The Total score was used for the current study. The AQ has good internal consistency within the five domains as well as fair construct validity, as evidenced by Cronbach's alpha coefficients ranging from 0.6-0.9 for all areas examined, and has demonstrated good test-retest reliability and success differentiating typically developing adolescents from those with ASD (Baron-Cohen et al., 2006). In the current study, the Cronbach alpha coefficient for the Total score was .75. Finally, the Quality of Socialization Questionnaire- Revised (QSQ-R: Laugeson et al., 2012) also was administered to caregivers. This rating form assesses the quantity and quality of the adolescent's get-togethers with peers. One score, a Contact score, was drawn from this measure. Contact scores are the sum of two items on the questionnaires: number of peer get-togethers planned by the adolescent in the last month, and number of peer get-togethers the adolescent was invited to in the last month. Higher scores indicate more social contact. In addition

to caregiver rating forms, adolescents completed the Test of Social Skills Knowledge (TASSK: Laugeson et al., 2012). The TASSK was specifically developed to measure adolescent learning and retention of the lessons taught in PEERS, and yields a Total score indicative of social skills and knowledge. Due to the wide range of topics covered on the TASSK and the lack of subscales, Cronbach's reliability alpha was not evaluated by the creators of the instrument.

Electroencephalogram session. Following completion of self-report questionnaires, participants and their caregivers were taken to the EEG laboratory. At this time non-sedated neural data was collected from a 64-electrode EGI HydroCell Sensor Net (Electrical Geodesics, Inc., Eugene, OR) selected on the basis of individual head circumference and adjusted to ensure no individual electrode impedance measurements above 50 kOhm. The EEG signal was amplified and sampled at a rate of 1000 Hz using Netamps 300 (Electrical Geodesics, Inc., Eugene, OR). Recordings were collected with the adolescent seated in a comfortable chair positioned approximately 19-inches from a computer monitor during two conditions. During the first condition (at rest, eyes open; EO), the adolescent was instructed to sit quietly while focusing on a fixation point on the computer monitor in front of them for a period of 3 minutes. During the second condition (monologue; MONO) the adolescent was told to sit quietly and to focus on a video playing on the computer monitor. The video was 3 minutes in length and included video and audio of a typically developing adolescent delivering a monologue. The monologue was a loosely scripted presentation of personal information detailing such things as name, school, family makeup, and an array of likes and dislikes (e.g., favorite book, favorite

movie, least favorite subject, etc.). All participants were monitored for alertness via live video feed during all EEG data collection.

Intervention

ASD Intervention (Program for the Education and Enrichment of Relational Skills, PEERS: Laugeson & Frankel, 2010). PEERS is a 14-week outpatient, empirically-supported, manualized intervention that aims to assist adolescents with ASD in establishing and maintaining developmentally appropriate friendships (for more information, see Laugeson et al., 2009; Laugeson et al., 2010 Laugeson et al., 2012; Schohl et al., 2013). Weekly meetings are 90-minutes in length and consist of separate but simultaneous adolescent and parent meetings. Group size is maintained at approximately 10 adolescents and 10-15 parents. PEERS sessions are led by trained doctoral students in a clinical psychology program and undergraduate assistants. Adolescent group leaders were required to have a Master's degree or higher. A Socratic teaching method is employed in the sessions, which consist of homework review, a didactic lesson, and behavioral rehearsals. Each week the previous session's assigned homework is reviewed and appropriate feedback is provided. Following homework review a new skill is introduced and explained in detail (Table 1). Finally, group leaders perform role-plays and adolescents rehearse skills before homework is assigned. Group leaders offer additional feedback during skill rehearsal. Undergraduate assistants monitored for adherence to the treatment protocol in adolescent sessions via completion of weekly fidelity check sheets.

Outtake session. Following participation in the PEERS program (EXP group) or a 13-week waiting period (WL group), families returned for an outtake session. At this time, a variety of intake measures were repeated (AQ, SSIS, QSQ, TASSK, EEG) in the same manner as at intake. The ADOS-G, K-BIT-2, Mental Status Checklist, demographic, and medication questionnaires were not repeated at the time of outtake.

EEG Data Analysis

EEG data was filtered from 0.3 to 100 Hz and exported from NetStation (Electrical Geodesics, Inc., Eugene, OR) software to MATLAB (2012a, The MathWorks, Natick, MA). Custom MATLAB scripts using EEGLAB functions (Delorme & Makeig, 2004) was used for offline data analysis. Data was re-referenced to a common reference. Low frequency noise was bandpass filtered from 2 to 100 Hz and power line noise notch filtered from 59 to 61 Hz using an 8th order, Butterworth, zero-phase filter. Data was broken down into 1-second epochs from which artifact resulting from large movements was rejected using the *pop_autorej* function (EEGLAB). An adaptive mixture independent component analysis (AMICA; Palmer, Makeig, Kreutz-Delgado, & Rao, 2008) was then used to decompose the epoched data and artifact components were identified using ADJUST (Mognon, Jovicich, Bruzzone, & Buiatti, 2010) and visual inspection. Average power spectral density was then calculated for each electrode using Welch's method (1024pt segments, 50% overlap), and spectral power calculated for the delta (0-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), beta (12-30 Hz), and gamma (30-50 Hz) bands by

calculating the area under the average spectrums. Finally, the data was transformed using the natural-logarithm transform to correct for violations of normality inherent in power analyses. Given expected hemispheric differences, average power within each band was calculated for the frontal, parietal, and temporal regions in the left and right hemispheres separately (see Figure 3 for electrode locations). For power measurements in the delta, theta, beta and gamma bands, higher numbers describe increased activity levels. For power measurements in the alpha band, higher numbers are associated with decreased activity levels, as alpha has been associated with the inhibition of cortical activation (Pfurtscheller et al., 1996; Rippon, 2006). Subjects in the TYP group had data calculated at one time point only. Those in the EXP and WL groups had data from their initial intake appointment (PRE) and from their follow-up appointment 13 weeks later (POST).

Results

Data Screening

All statistical analyses were completed in the SPSS 22.0 program (IBM, 2013) and analyzed at $p < .05$. Descriptive statistics for power values at pre- and post-test (if applicable) for the EXP, WL, and TYP groups are presented in Tables 3 and 4. Distributions were examined for problems with the assumptions of normality, homogeneity of variance and outlying values. Outlying values identified in the EEG spectral power output and the pre- and post-test questionnaire scores were replaced with the next most extreme value in the distribution (Winsorization: Howell, 2012). 1.8% of questionnaire data was winsorized. 7.3% of EEG data was winsorized. Violations of sphericity and corrections applied are noted in Appendix A.

To reduce the amount of data in statistical tests, exploratory analyses were completed. RM-ANOVAs within each of the five frequency bands (delta, theta, alpha, beta, gamma) for all aims of the study revealed findings of interest only within the theta, alpha, and beta bands. Statistics are therefore only presented for findings within these three bands.

Aim I. Neural Differences between ASD and TYP

Repeated measures ANOVAs for Aim 1 were performed to examine neural differences between adolescents with and without ASD (DX: ASD, TYP) within the selected frequency band across EEG conditions (CONDITION: EO, MONO) and brain

locations (LOCATION: Left frontal, left parietal, left temporal, right frontal, right parietal, right temporal) at baseline.

Theta Band. The between-subjects effect, **DX, was significant**, F (1, 65) = 5.78, $p < .05$; partial η^2 =.08, observed power = .66 (TYP= 1.63 $\pm$ .11 (mean $\pm$ standard error, here and elsewhere), ASD= 1.31 $\pm$ .08). The main effect of **CONDITION was significant**, F (1, 65) = 8.84, $p < .05$; partial η^2 =.12, observed power = .84 (EO= 1.39 $\pm$ .07, MONO= 1.55 $\pm$ .08). The main effect of **LOCATION also was significant**, F (5, 61) = 31.84, $p < .05$; partial η^2 =.72, observed power = 1.0 (see Table B1 for LOCATION descriptive statistics & Table B2 for LOCATION pairwise comparisons). Two- and three-way interactions were not significant.

Alpha Band. The between-subjects effect, DX, was not significant. The main effect of **LOCATION was significant**, F (5, 61)= 27.48, $p < .05$, partial η^2 =.69, observed power = 1.0 (see Table B1 for LOCATION descriptive statistics & Table B3 for LOCATION pairwise comparisons). The main effect of CONDITION and higher-order interaction effects were not significant.

Beta Band. The between-subjects effect, **DX, was significant**, F (1, 65)= 11.98, $p < .05$, partial η^2 =.07, observed power = .58 (TYP= 1.76 $\pm$ .09, ASD= 1.50 $\pm$ .07). The main effect of **CONDITION was significant**, F (1, 65) = 17.57, $p < .05$, partial η^2 =.21, observed power = .99 (EO= 1.55 $\pm$ .06, MONO= 1.71 $\pm$ .06). The main effect of **LOCATION was significant**, F (5, 61) = 4.13, $p < .05$, partial η^2 =.25, observed power = .94 (see Table B1 for LOCATION descriptive statistics & Table B4 for LOCATION pairwise comparisons). The interaction effect **CONDITION by**

LOCATION also was significant, F (5, 61) = 2.99, p < .05, partial η^2 =.20, observed power = .83. All other interactions were not significant.

To follow-up the significant interaction CONDITION by LOCATION, the file was split by LOCATION and six additional paired samples t-tests were conducted, with Bonferroni corrected alpha level of .025, to examine spectral power within the beta frequency band between conditions (EO, MONO), averaged across diagnostic groups. **Significant differences were found within each location for all participants, whereby more activity was observed during the MONO condition than during the EO condition at baseline** (see Table B5 for descriptive statistics and Table B6 for paired t-test statistics).

Aim II. Neural Changes in ASD

a.) *PEERS involvement and neural change.*

Repeated measures ANOVAs for Aim 2a were performed to examine neural differences between adolescents with ASD (GROUP: EXP, WL) within the selected frequency band across EEG conditions (CONDITION: EO, MONO), brain locations (LOCATION: Left frontal, left parietal, left temporal, right frontal, right parietal, right temporal), and time (TIME: PRE, POST).

Theta band. The main effects of the between-subjects variable GROUP and the within-subjects variables CONDITION and TIME were not significant. There was a **significant main effect of LOCATION**, F (5, 37)= 21.23, p < .05, partial η^2 =.74, observed power = 1.0 (see Table C1 for descriptive statistics & Table C2 for

LOCATION pairwise comparisons). The interaction effect of **CONDITION by TIME also was significant**, F (1, 41) = 7.25, p < .05, partial η^2 =.15, observed power = .75. All other interactions were not significant.

To follow-up the significant interaction CONDITION by TIME, the file was split by TIME and two paired samples t-tests, with Bonferroni corrected alpha level of .025, were conducted comparing spectral power, within the theta band, averaged across GROUP and LOCATION, between the two experimental conditions (EO, MONO). **At *pre-treatment,* significantly more activity was observed in response to the MONO condition than to the EO condition**, t (42)= -2.97, p <.025 (EO= 1.19 $\pm$ .08, MONO= 1.42 $\pm$ .10). At *post-treatment*, no significant difference was noted.

Alpha band. The between-subjects effect of GROUP was not significant. The main effect of **CONDITION was significant**, F (1, 41) = 4.27, p < .05, partial η^2 =.09, observed power = .52 (EO=1.24 $\pm$ .10, MONO=1.03 $\pm$ .08). There was also a **significant main effect of LOCATION**, F (5, 37) = 29.27, p < .05, partial η^2 =.80, observed power = 1.0 (see Table C1 for descriptive statistics & Table C3 for pairwise comparisons). The main effect of TIME was not significant. There was a **significant interaction effect of CONDITION by TIME**, F (1, 41) = 8.30, p < .05, partial η^2 = .17, observed power = .81. All other interactions were not significant.

To follow-up the significant interaction CONDITION by TIME, the file was split by TIME and two paired samples t-tests, with Bonferroni corrected alpha level of .025, were conducted comparing spectral power, averaged across GROUP and LOCATION, between the two experimental conditions (EO, MONO). At *pre-treatment*,

no significant difference was noted. At *post-treatment*, **significantly more alpha power was observed in response to the EO condition than to the MONO condition**, t (42)= 2.49, p <.025 (EO= 1.35 $\pm$ .11, MONO= .91 $\pm$ .12).

Beta band. The main effect for the between subjects variable GROUP was not significant. There were no significant main effects of CONDITION or TIME. There was a **significant main effect of LOCATION**, F (5, 37)= 3.95, p < .05, partial η^2 =.35, observed power = .91 (see Table C1 for descriptive statistics & Table C4 for LOCATION pairwise comparisons). The interaction effect **CONDITION by TIME was significant**, F (1, 41)= 6.16, p < .05, partial η^2 =.13, observed power = .68. The interaction **LOCATION by TIME by GROUP** also neared significance, F (5, 37)= 2.37, p = .058, partial η^2 =.24, observed power = .69. All other interactions were not significant.

To follow-up the significant interaction CONDITION by TIME, the file was split by TIME and two paired samples t-tests, with Bonferroni corrected alpha level of .025, were conducted comparing spectral power, averaged across Groups and Locations, between the two experimental conditions (EO, MONO). **At *pre-treatment*, significantly more activity was observed in response to the MONO condition than to the EO condition**, t (42)= -3.76, p <.025 (EO= 1.40 $\pm$ .08, MONO= 1.61 $\pm$ .07). No significant difference was observed at *post-treatment.*

To follow-up the interaction LOCATION by TIME by GROUP, the file was split by LOCATION and six additional RM-ANOVAs were conducted, examining spectral power averaged across conditions within the beta band at each of six brain

locations, as it differed between treatment groups from pre- to post-treatment. No significant findings were noted within any of the brain locations examined.

b.) *Post-treatment ASD/TYP neural differences.*

Repeated measures ANOVAs for Aim 2b were performed to examine neural differences between adolescents with and without ASD (GROUP: TYP, EXP, WL) within the selected frequency band across EEG conditions (CONDITION: EO, MONO) and brain locations (LOCATION: Left frontal, left parietal, left temporal, right frontal, right parietal, right temporal) at post-treatment.

Theta band. The main effect for the between subjects variable, **GROUP, was significant**, F (2, 64) = 4.82 $p < .05$; partial $\eta^2 = .13$, observed power = .78. Follow-up comparisons revealed significantly **more activity within the TYP** (TYP= 1.63 $\pm$ .09) **group than the WL** (WL= 1.24 $\pm$ .10) **group. No significant differences were noted between the EXP** (EXP= 1.31 $\pm$ .10) **group and either the TYP or WL groups**. The main effect for the within-subjects variable, **LOCATION, was significant**, F (5, 60) = 27.91, $p < .05$; partial $\eta^2 = .70$, observed power = 1.0 (see Table D1 for LOCATION descriptive statistics & Table D2 for LOCATION pairwise comparisons). There was a **significant CONDITION by GROUP interaction**, F (2, 64) = 3.78, $p < .05$; partial $\eta^2 = .11$, observed power = .67. There was also a **significant interaction CONDITION by LOCATION by GROUP**, F (5, 60) = 2.40, $p < .05$; partial $\eta^2 = .17$, observed power = .93. The main effect of CONDITION and all other interactions were not significant.

To follow-up the significant interaction CONDITION by GROUP, the file was split by GROUP and three paired samples t-tests, with Bonferroni corrected alpha level of .025, were conducted comparing spectral power, within the theta band, averaged across LOCATION, between the two experimental conditions (EO, MONO). No significant differences were noted between conditions within any of the groups.

To follow-up the significant interaction CONDITION by LOCATION by GROUP the file was split by LOCATION and six additional RM-ANOVAs were conducted, examining spectral power within the theta band at each of six brain locations, as it differed between treatment groups in the eyes open and monologue conditions. **A significant main effect of GROUP was found within the left parietal region,** F (2, 64)=4.87, $p < .05$; partial $\eta^2 = .13$, observed power = .78 (TYP= 1.71 $\pm$ .11, EXP= 1.32 $\pm$ .12, WL= 1.71 $\pm$ .11. **Significant main effects of GROUP also were found within the left frontal and right frontal regions. However, these main effects were qualified by significant GROUP by CONDITION interactions in both the left frontal,** F (2, 64)=8.31, $p < .05$; partial $\eta^2 = .21$, observed power =.96, **and right frontal,** F (2, 64)=6.11, $p < .05$; partial $\eta^2 = .16$, observed power = .87 **regions. A significant interaction effect of GROUP by CONDITION also was found within the right temporal region,** F (2, 64)=3.47, $p < .05$; partial $\eta^2 = .10$, observed power = .63. These interaction effects were followed up by splitting the file by GROUP and running paired samples t-tests with Bonferonni corrected alpha levela of .025 to compare spectral power within the theta band between the two experimental conditions (EO, MONO) for the specified region. Significantly more activity was observed during the eyes open condition than the monologue condition within the

EXP group but not the WL or TYP groups in the left frontal, *t* (20)= 2.96, p <.025 (EO= 1.83 ± .25, MONO= .99 ± .09) and right frontal, , t (20)= 2.35, p <.025 (EO= 1.70 ± .14, MONO=1.13 ± .08) regions. No significant group differences were noted between conditions with regard to theta spectral power in the right temporal region in follow-up analyses.

Alpha band. The main effect for the between subjects variable GROUP was not significant. The main effect for the within-subjects variable, **CONDITION was significant**, F (1, 64) = 9.20, $p < .05$; partial $\eta^2 = .13$, observed power = .85 (EO= 1.40 ± .08, MONO= 1.05 ± .09). The main effect of **LOCATION also was significant,** F (5, 60) = 40.95, $p < .05$; partial $\eta^2 = .77$, observed power = 1.0 (see Table D1 for LOCATION descriptive statistics and Table D3 for LOCATION pairwise comparisons). All interactions were not significant.

Beta band. The main effect for the between subjects variable, **GROUP, was significant**, F (2, 64) = 4.39, $p < .05$; partial $\eta^2 = .12$, observed power = .74. Follow-up revealed significantly **more activity within the TYP group** (1.76 ± .08) **than the WL** (1.45 ± .08) **group**. **There was no significant difference between the EXP (1.47 ± .09) and TYP or WL groups.** The main effect for the within-subjects variable, **LOCATION was significant**, F (5, 60) = 7.08, $p < .05$; partial $\eta^2 = .37$, observed power = 1.0 (see Table D1 for descriptive statistics & Table D4 for pairwise comparisons). The main effect of CONDITION was not significant. There was a **significant interaction CONDITION by GROUP**, F (2, 64) = 3.83, $p < .05$; partial $\eta^2 = .11$, observed power = .68. All other interactions were not significant.

To follow-up the significant interaction CONDITION by GROUP, the file was split by GROUP and three paired samples t-tests, with Bonferonni corrected alpha level of .025, were conducted examining spectral power within the beta band between conditions (EO, MONO). No significant differences between conditions were noted within any of the groups.

Aim III. Relations Between Neural Changes and Behavioral Change in ASD

a.) Behavioral Changes.

Repeated measures ANOVAs for Aim 3 were performed to examine changes in behavioral rating questionnaires before and after treatment (PRE, POST) in adolescents with ASD who did and did not receive the PEERS intervention (GROUP: EXP, WL).

Autism Spectrum Quotient- Parent. The between-subjects variable GROUP was not significant. There was a **significant main effect of TIME**, F (1, 40)= 4.15, p< .05; partial η^2 = .09, observed power= .51 (PRE= 34.05 $\pm$ .93, POST= 31.38 $\pm$ 1.57). There was no significant interaction between TIME and GROUP.

Social Skills Improvement System- Parent. The main effect for the between subjects variable, GROUP, was not significant. The main effect for the within subjects variable, TIME, also was not significant. The interaction TIME by GROUP was not significant.

Quality of Socialization Questionnaire-Revised. The main effect for the between subjects variable, **GROUP, was significant**, F (1, 38) = 8.68, p < .05, partial

η^2 =.19, observed power = .82 (EXP= 2.58 ± .38, WL= 1.00 ± .38). The main effect of **TIME also was significant**, F (1, 38) = 7.41, p < .05; partial η^2 =.16, observed power = .76 (PRE= 1.33 ± .29, POST= 2.25 ± .35). Main effects were qualified by the **significant interaction TIME by GROUP**, F (1, 38) = 5.89, p < .05; partial η^2 =.13, observed power = .67. Post hoc paired t-tests, with a Bonferroni corrected alpha level of .025, splitting the file by GROUP, revealed that **EXP QSQ-R Contact scores significantly increased over time**, t (19) = -3.73, p < .025 (PRE= 1.70 ± .43, POST= 3.45 ± .57). In contrast, QSQ-R Contact scores in the WL group did not significantly change over time. These results suggest that the ASD group that received PEERS showed an increase in social contacts via hosted and invited get-togethers over time, whereas the ASD group that did not receive PEERS did not show a change in reported social contacts over time.

Test of Adolescent Social Skills Knowledge. The main effect for the between subjects variable, **GROUP, was significant**, F (1, 41) = 25.94, p < .05; partial η^2 = .39, observed power= 1.0 (EXP= 17.24 ± .58, WL= 13.10 ± .57). The main effect for the within subjects variable, **TIME, was significant**, F (1, 41) = 77.88, p < .05; partial η^2 = .66, observed power= 1.0 (PRE= 13.19 ± .42, POST= 17.14 ± .51). Main effects were qualified by a **significant interaction between TIME and GROUP**, F (1, 41) = 81.50, p < .05; partial η^2 = .67, observed power= 1.0. Post hoc paired t-tests, with a Bonferonni corrected alpha level of .025, splitting the file by GROUP, revealed that **EXP TASSK scores significantly increased over time**, t (20) = -11.89, p < .025 (PRE= 13.24 ± .61, POST= 21.24 ± .89). In contrast, TASSK scores in the WL group did not change over time. These results suggest that the ASD group that received

PEERS between the PRE and POST appointments showed an increase in their knowledge of PEERS concepts, while the ASD group that did not receive the intervention did not show a change in their knowledge of the concepts.

b.) Neural and Behavioral Correlations.

Neural and behavioral data were selected for inclusion in correlational analyses based on outcomes from previous aims in order to preserve power. Given directional differences in overall neural change from pre- to post-treatment across groups and locations between experimental conditions (EO, MONO), correlations were conducted for activity within each condition separately. No significant correlations were noted between overall changes (average across all brain locations) in spectral power within the theta or beta bands and changes within the TASSK total score or QSQ contact score from pre-to post-treatment in either of the experimental conditions.

Aim IV. Effects of Incorporating a Novel Condition on Neural Findings

Results of analyses investigating differences in spectral power between experimental conditions are described throughout results of previous aims.

Discussion

Autism spectrum disorders have been classified as neurodevelopmental disorders, reflecting literature indicating significant neural differences between individuals exhibiting symptoms consistent with an ASD diagnosis and their neurotypical peers. Given our understanding of ASD as stemming from neural atypicalities, there has been a recent push within the field for behavioral therapies driven by our understanding of the neural basis of ASD (Dawson et al., 2012). In light of this imperative, the current study investigated outcomes of the PEERS social skills intervention from both behavioral and neural perspectives.

To begin, EEG spectral power in adolescents with ASD was compared to that of their neurotypical peers to gain information about baseline differences in neural functioning. Previous literature investigating differences in EEG activation patterns has largely focused on children or adults. To our knowledge, only one study (Van Hecke et al., 2013) has directly examined EEG activity in ASD during the adolescent years, rendering this analysis of great importance. Results of the first aim revealed significant differences between diagnostic classes. Specifically, average spectral power across brain locations examined was higher in neurotypicals than in adolescents with ASD within the theta and beta frequency bands. Previous studies investigating the EEG activation patterns of individuals with ASD as they compare to those of neurotypicals have demonstrated reduced EEG spectral power during the childhood years (Dawson, et al., 1995). Furthermore, widespread hypoconnectivity has been reported in children on the autism spectrum and weak medium and long-range connectivity levels have been demonstrated in adults on the autism spectrum

(as reviewed by Minshew & Williams, 2007). Given that theta band activity has been associated with long-range communication within the brain and beta has been tied to medium-range communication, it is not surprising that underactivation was noted within these frequency bands in adolescents with ASD in the current study. The decreased neural activation noted in ASD has been associated with difficulties processing information in a neurotypical manner, particularly with difficulties synthesizing and processing complex information from the environment (Barnea-Goraly, et al., 2004; Frith, 1989; Frith & Harpe, 1994; Happe, 1999; Minshew & WIlliams, 2007). This type of deficit in neural processing may be tied to social difficulties due to the limitations it places on one's ability to process social information in an expected way and behave accordingly, particularly given our understanding of theory of mind tasks requiring complex information processing (Baron-Cohen, Leslie, & Frith, 1985). Differences were not observed between adolescents with ASD and their neurotypical peers within the delta, alpha, or gamma frequency bands. These findings are initially somewhat unexpected. Further consideration, however, provides potential interpretations of these results. Delta band activity is predominantly present during deep sleep (Blinkowska & Durka, 2006; Rippon, 2006). Given that the current study's data was collected with subjects in an alert, wakeful state, limited delta band activity should be observable, making it more difficulty to identify group differences in patterns of activation.

Further, during the childhood years, hypoconnectivity is widespread within the brains of individuals with ASD (Minshew & Williams, 2007), suggesting that we might expect lower activity levels within the gamma frequency band, in light of

gamma's role in short-range communication. It is important to note, however, that the opposite finding has been shown in adults with ASD. That is, adults on the autism spectrum have demonstrated local hyperconnectivity in some regions of the brain (Murias, et al., 2007). It follows, therefore, that during the adolescent years individuals on the autism spectrum must be experiencing changes with regard to short-range communication, from underactivation to overactivation. It is possible that with the current study's focus on adolescents, the lack of significant differences within the gamma frequency band between individuals with ASD and neurotypicals is reflective of a period of normalized short-range communication during a broader transition from local hypoconnectivity to hyperconnectivity.

With regard to alpha band activity, findings of the current study suggest that individuals with ASD experience typical cortical inhibition. Again, given the transition individuals with ASD experience from global hypoconnectivity to medium and long-range hypoconnectivity but local hyperconnectivity, it is possible that changes are occurring that impact the level of cortical inhibition experienced by individuals with ASD, explaining the lack of significant findings in the alpha band in this investigation.

Variation was noted in spectral power between locations throughout the brain, though this variation was consistent between diagnostic groups, indicating that differences in activation between groups do not differ by brain region. This finding is inconsistent with previous literature, which has typically demonstrated differences in patterns of activation which have varied by neuroanatomical location (Dawson et al., 1995; Murias et al., 2007; Van Hecke et al., 2013). An inherent

limitation of EEG studies, however, is poor spatial specificity. Combining this limitation with the lack of consistent EEG data collection methods (e.g., varied electrode locations and electrode choices for regional estimates), comparisons between studies with regard to spatial findings must be interpreted with caution.

The second aim of the study was to directly examine the impact of the PEERS intervention on neural functioning. To this end, adolescents with ASD who had participated in the PEERS treatment were compared to teens with ASD who had not received the intervention. No significant differences were noted between the two ASD groups. This finding suggested no identifiable neural change in response to the intervention. Interestingly, however, at the time of second measurement, individuals with ASD who had not participated in the intervention continued to differ significantly from their same-aged peers, from a neural perspective, while those who had received the treatment no longer differed statistically from their neurotypical peers. In essence, after participating in the PEERS intervention, the amount of overall spectral power within the theta and beta bands in adolescents with ASD approximated that of adolescents without ASD. A note of caution, however: even though experimental group adolescents with ASD no longer differed significantly from typically developing adolescents, the amount of activation in the experimental group of adolescents with ASD remained descriptively (but not statistically) less than their neurotypical peers. This is in contrast to the waitlist group, who remained descriptively and statistically less active than the neurotypical group. It is therefore somewhat dramatic to describe those in the experimental group as having "normalized" neural activation post-treatment; however, it is

encouraging to note that participation in PEERS assisted in bringing teens with ASD closer to their typically developing peers from a neural perspective. The current study did not involve long-term follow-up of participants, however, it will be important for future studies to explore to what degree this trend toward "normalized" neural patterns in theta and beta EEG activity persists over time. In sum, these findings are indicative of neural plasticity in response to a behavioral intervention in ASD. They also suggest that adolescence may represent a second crucial developmental period for intervention targeting neural abnormalities in this population.

The current results are of great scientific value. To our knowledge, outside of our laboratory only one other research group has reported neural change in response to intervention among individuals with autism (Dawson et al., 2012). In their seminal paper, published in 2012, Dawson and colleagues reported normalized patterns of brain activity associated with a developmental behavioral intervention in young children with autism. The study, however, did not include pre-treatment neural data and was therefore unable to clearly demonstrate meaningful change in direct response to treatment, as questions of baseline group differences were not first answered. In this study, pre-treatment neural data showed significant group differences that were no longer present at post-treatment. Given the design of this study, we are confident that our findings represent a unique, innovative contribution to the field's exploration of neural change in response to behavioral intervention.

Although neural change in response to intervention is interesting, behavioral outcomes are paramount in demonstrating the functional utility of any intervention. The third aim of this study explored behavioral change in response to PEERS. Findings indicated that adolescents with ASD who participated in the intervention gained a significant amount of social skills knowledge from pre- to post-treatment, while those teens who did not receive the treatment did not experience an increase in knowledge. Furthermore, participation in PEERS resulted in significantly more social exposure for adolescents on the autism spectrum in the form of more frequent get-togethers with same-aged peers. Although traits of ASD did not decrease significantly in response to the intervention and observable improvements in social skills were not noted, we are hopeful that, over time, with increased social skills knowledge and more social opportunities, those with ASD who participated in the treatment will begin to demonstrate improved social ability.

Attempts to correlate changes in neural and behavioral presentations in response to the intervention were unsuccessful. This suggests that there may be a mediating variable that was overlooked during this investigation. It will be important for future studies to explore potential mediating factors such as mood or motivation for social contact.

While we were unable to provide a direct link between neural change and behavioral improvement, we can hypothesize about the clinical utility of our neural findings given the patterns observed. These patterns included increased activity within the theta and beta frequency bands, suggesting improved medium- and long-range communication within the brains of adolescents who received the

intervention. This enhanced communication may underlie significant improvements in information processing abilities, specifically, individuals' ability to integrate information from many sources within their environments. At a broad clinical level, this may translate to improved theory of mind capabilities. At a more narrow level, it is possible that these neural changes could result in improved face processing due to improvements in the integration of information and, subsequently, more appropriate social behavior in response to others due to improvement in long-range communication allowing better information transfer between posterior face processing regions and frontal regions of the brain which allow for behavioral regulation.

In addition to investigating neural plasticity in response to the PEERS intervention, the current study explored the benefits of including a new testing condition in EEG data collection. Given the fundamental difficulties individuals on the autism spectrum experience with social interaction, in addition to collecting EEG data during a traditional eyes open, resting state condition, we collected data during a 'monologue' condition. This condition involved having the subject watch and listen to a neurotypical peer deliver a short narrative focused on personal information typically shared in a social setting. While this condition would not capture brain activity differences involved in social behavior, we hypothesized that it would offer information about the processing of social information. Results indicated that, at pre-treatment, significantly more activity was observed during the monologue condition than during the eyes open condition within the theta and beta bands across brain locations analyzed. This pattern was consistent across diagnostic

groups. In other words, including the new condition did not reveal neural differences between diagnostic groups that were not already apparent during the eyes open condition. These findings indicate that including the novel condition did not contribute meaningful findings with regard to comparisons between diagnostic groups at baseline beyond what was already obtained using standard methods of data collection. At post-treatment, spectral power did not differ between conditions for experimental or waitlist group subjects within the alpha or beta bands, and within the theta band significantly more activity was observed in the left and right frontal regions during the eyes open condition than during the monologue condition. It is unclear why this may be the case. Possible interpretations could include decreased attention to the stimulus or decreased anxiety at post-test, given the subjects' comfort level with the test setting at second testing. More optimistically, it is possible that these findings reflect enhanced efficiency of communication within the brain. This improved efficiency could simply be a result of development. It is also possible that treatment enhanced this developmental process within the experimental group, leading to the region-specific findings in the theta band.

Although this study demonstrates new information regarding neural plasticity in response to a behavioral intervention in adolescents with ASD, there are several important limitations to be addressed. First, it remains unclear how the increases in spectral power noted in this study will impact those with ASD in a functional manner. Although behavioral changes were shown in response to the treatment, no direct correlation between the neural and behavioral changes could

be made. Furthermore, no long-term follow-up analyses were completed as part of this investigation, therefore, we cannot know if changes will continue to occur or persist after the therapy's termination. For these reasons, it will be important for long-term follow-up studies to continue to track neural and behavioral changes, and how they may relate, in this sample. We must also address the fact that data on neurotypical adolescents was only collected at one time-point. The inclusion of a waitlist control group reduces the possibility that changes noted in the experimental group are naturally occurring, developmental changes unrelated to intervention. The limitation remains, however, that we compared data from adolescents with ASD at post-treatment, to neurotypical data without a 13-week lapse, assuming that natural developmental changes are not occurring within the neurotypical group at a rate differing from that of changes occurring in the ASD group. This is a large assumption given that ASD is associated with developmental delays by definition. An additional limitation deserving of mention is that medication usage was not controlled for in the current study. Given the heterogeneity within the ASD sample with regard to their medication classes and doses, this was not feasible. Excluding subjects using medications also was not an option due to high rates of medication use in this population. Future studies would benefit from controlled, randomized studies investigating the usefulness of combining the PEERS intervention with selected medications. Finally, it is worth mentioning that significant heterogeneity is noted within the ASD sample. While all neurotypical adolescents in the study did not meet criteria for mental health diagnoses, those with ASD had a number of co-morbid conditions (e.g., ADHD, anxiety, depression) that likely also impacted their

neural patterns. Furthermore, symptom severity differed greatly among those on the autism spectrum.

In conclusion, despite previously discussed limitations, findings from the current study are potentially of great clinical value. Researchers and clinicians are beginning to note the importance of selecting treatments for neurodevelopmental disorders that can directly impact neurodevelopment. The results of this study indicate that neural change in response to behavioral intervention is possible, to the point of rendering patterns of brain activity of those with ASD indiscernible from that of their typically developing peers. Moving forward it will be intriguing to explore how these changes occur (e.g., do changes in EEG activity levels reflect a slowing down of pruning in ASD?). Imaging studies examining structural changes in the brain in response to the intervention may offer more information in this regard. Additionally, EEG techniques offer limited spatial specificity and no access to functional information about subcortical structures. Future studies using fMRI techniques may shed more light on changes in activation levels in response to the intervention occurring in specific cortical areas and within deeper structures. Although a number of questions remain, the importance of this study should not be dismissed. This investigation provides additional support for studies examining neural plasticity in response to behavioral intervention, particularly during the sensitive developmental period of adolescence.

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Table 1

PEERS Sessions & Associated Didactic

Session	**Didactic**
1	Conversational Skills I: Trading Information
2	Conversational Skills II: Two-way Conversations
3	Conversational Skills III: Electronic Communication
4	Choosing Appropriate Friends
5	Appropriate Use of Humor
6	Peer Entry I: Entering a Conversation
7	Peer Entry II: Exiting a Conversation
8	Get-togethers
9	Good Sportsmanship
10	Rejection I: Teasing and Embarrassing Feedback
11	Rejection II: Bullying & Bad Reputations

12	Handling Disagreements
13	Rumors & Gossip
14	Graduation & Termination

Note. Reproduced with permission of the authors.

Table 2

Sample Demographic Characteristics

Characteristic	**EXP M(SD)**	**WL M(SD)**	**TYP M(SD)**	***p***
Age (years)	13.48(1.54)	13.86(1.73)	12.96(1.46)	*ns*
IQ (points)	108.05(18.64)	101.64(15.62)	107.54(15.06)	*ns*
ADOS Total score	10.38(2.58)	9.64(2.99)	--	*ns*
Communication score	3.71(1.35)	3.41(1.30)	--	*ns*
Social score	6.57(1.75)	6.05(1.65)	--	*ns*
Mother's age (years)	45.43(3.79)	46.55(3.75)	44.54(3.95)	*ns*
Father's age (years)	47.57(4.34)	48.21(4.45)	46.33(4.15)	*ns*
Race (percentage)				
Asian	0	9.1	0	
African-American	0	0	0	
Biracial	0	4.5	4.2	
Caucasian	100	81.8	95.8	
Unreported	0	4.5	0	
Income (percentage)				
Under 50k	14.3	22.7	12.5	
50k-75k	28.6	4.5	12.5	
75k-100k	9.5	9.1	12.5	
100k plus	47.6	63.6	62.5	
Unreported	0	0	0	
Parent Education				

(percentage)	2.5	4.76	6.3	
High School	5.0	19.0	2.1	
Vocational/Technical	25.0	11.9	14.6	
Some College	2.5	0	2.1	
Junior College	50.0	35.7	22.9	
B.A./B.S.	10.0	21.4	33.3	
M.A./M.S.	5.0	7.1	18.8	
Ph.D/M.D./J.D.				

Table 3

Descriptive statistics for EEG spectral power values at pre- and post-treatment within eyes open (EO) condition

	Pre			**Post**	
Band	**EXP** **M(SD)**	**WL** **M(SD)**	**TYP** **M(SD)**	**EXP** **M(SD)**	**WL** **M(SD)**
Delta	1.73(.13)	1.52(.12)	1.88(.12)	2.06(.17)	1.66(.16)
Theta	1.29(.12)	1.09(.12)	1.58(.11)	1.55(.14)	1.22(.14)
Alpha	1.27(.15)	.98(.15)	1.47(.14)	1.62(.15)	1.10(.16)
Beta	1.50(.10)	1.28(.10)	1.70(.10)	1.64(.11)	1.37(.11)
Gamma	.58(.15)	.42(.14)	.43(.14)	.50(.12)	.50(.12)

Note. EXP = Experimental Group, WL = Waitlist Group, TYP = Typically Developing Group (measured on only one occasion). M= mean, SD = standard deviation. Pre= average spectral power across all brain locations at pre-treatment, Post = average spectral power across all brain locations at post-treatment.

Table 4

Descriptive statistics for EEG spectral power values at pre- and post-treatment within monologue (MONO) condition

	Pre			**Post**	
Band	**EXP** **M(SD)**	**WL** **M(SD)**	**TYP** **M(SD)**	**EXP** **M(SD)**	**WL** **M(SD)**
Delta	1.80(.15)	1.77(.15)	1.87(.14)	1.59(.11)	1.59(.11)
Theta	1.47(.13)	1.38(.13)	1.68(.12)	1.07(.12)	1.27(.12)
Alpha	1.31(.15)	1.01(.13)	1.33(.13)	.88(.16)	.94(.16)
Beta	1.72(.10)	1.52(.10)	1.81(.10)	1.30(.12)	1.53(.11)
Gamma	.71(.14)	.74(.14)	.62(.13)	.48(.14)	.50(.14)

Note. EXP = Experimental Group, WL = Waitlist Group, TYP = Typically Developing Group (measured on only one occasion). M= mean, SD = standard deviation. Pre= average spectral power across all brain locations at pre-treatment, Post = average spectral power across all brain locations at post-treatment.

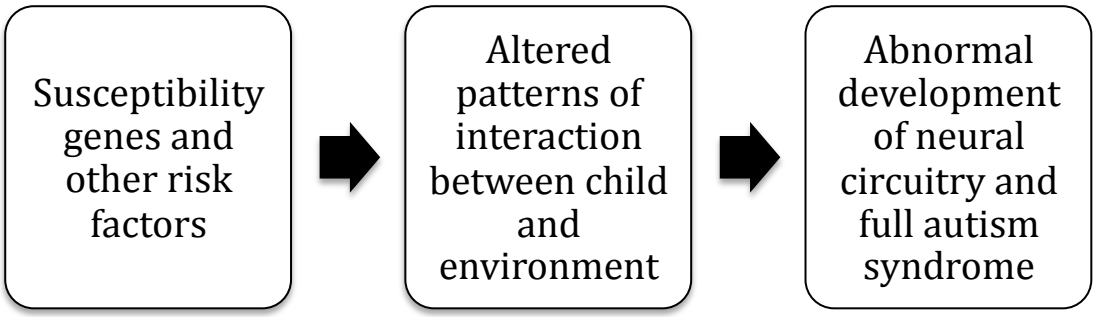

Figure 1. Dawson et al. (2009) experience-based risk processes model of autism.

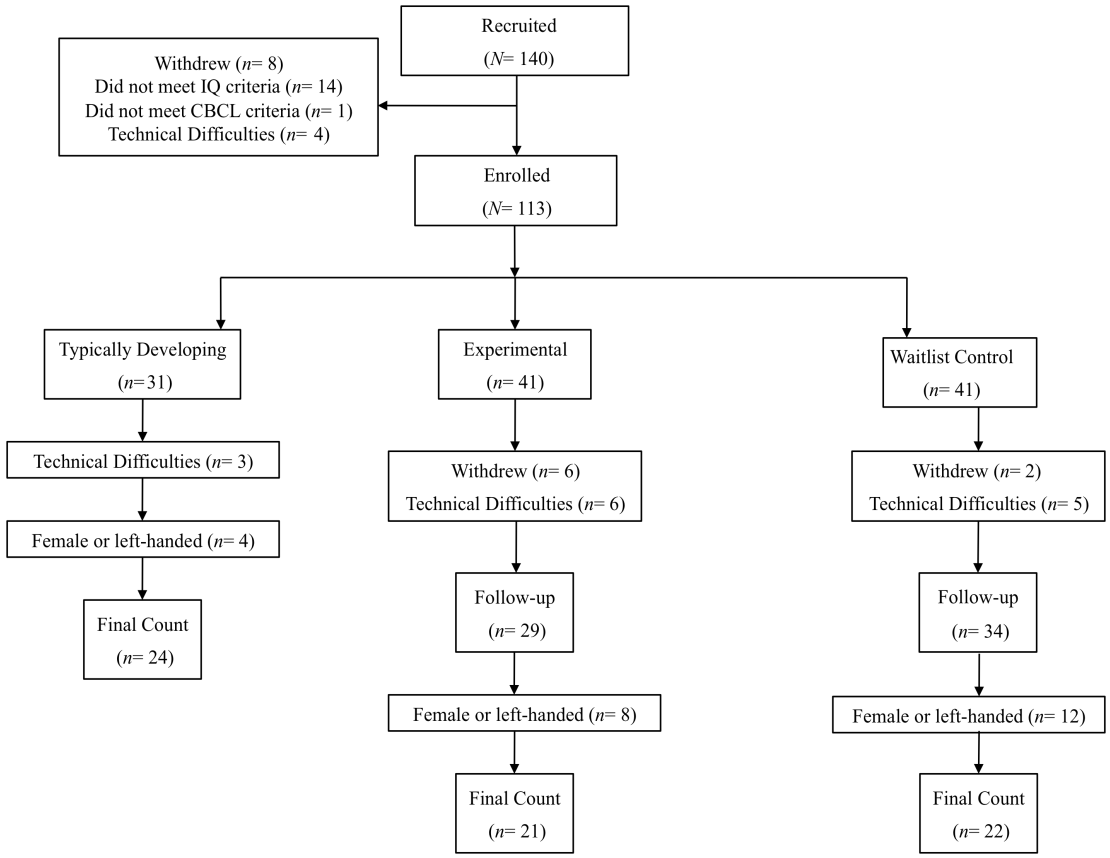

Figure 2. Consort chart.

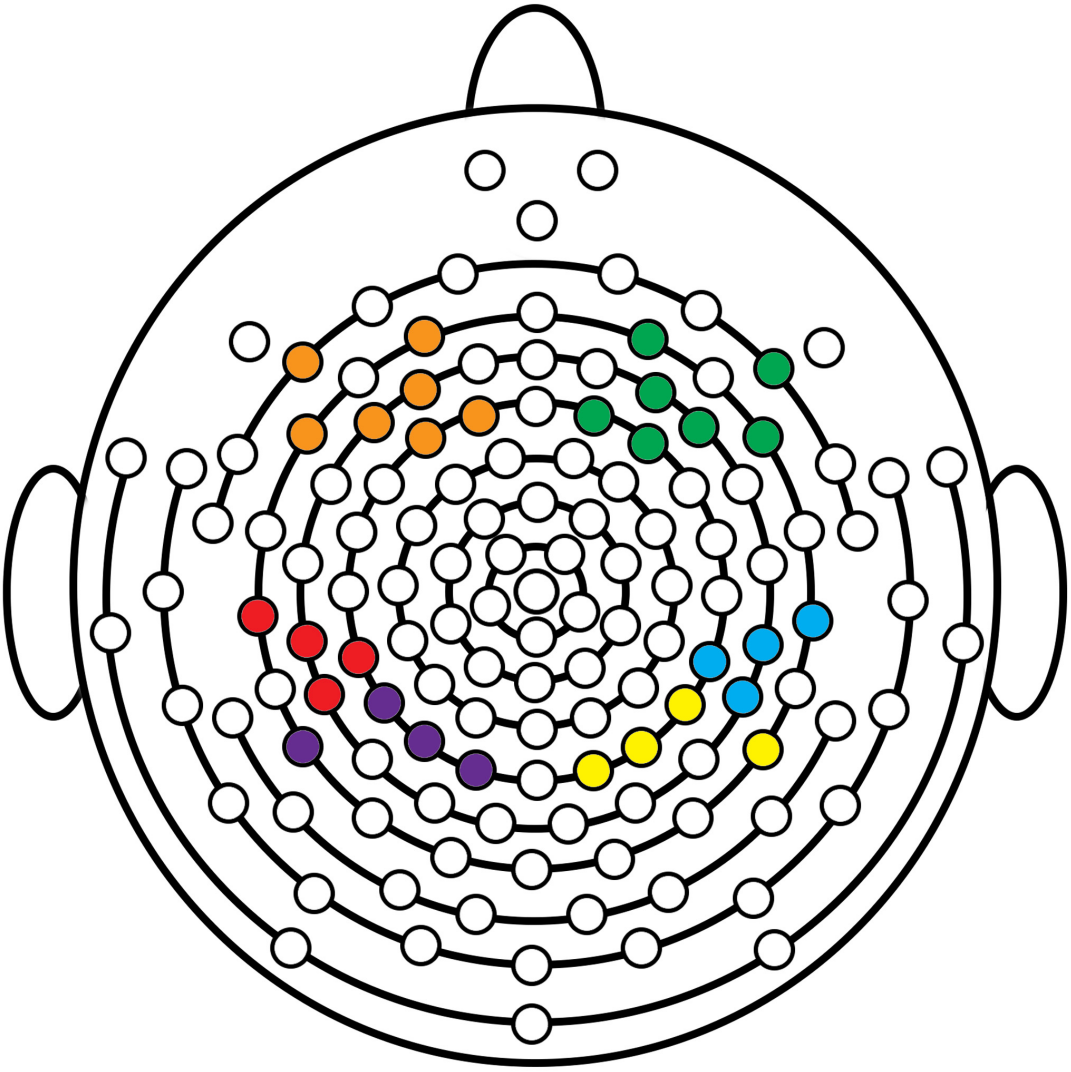

Figure 3. Geodesic Sensor Net Hydrocell 64-channel pediatric medium, large, adult small, and adult medium nets, based on standard sizing for head circumference (Electrical Geodesics: Eugene, OR). Electrodes broken down by region to be examined. Orange= Left Frontal. Red= Left Temporal. Purple= Left Parietal. Green= Right Frontal. Blue= Right Temporal. Yellow= Right Parietal.

Appendix A

Violations of Sphericity and Corrections Applied

Table A1

Violations of Sphericity for Aim 1

Variable	Mauchley's *W*	ε
Theta		
Location	.41*	.81
Location by Condition	.40*	.80
Alpha		
Location	.34*	.79
Location by Condition	.27*	.73
Beta		
Location	.40*	.76
Location by Condition	.38*	.84

*$p < .05$

df=14

Note. Wilks' Lambda multivariate statistics cited for all Aim1 statistics. Values for variables not violating assumptions of sphericity not listed.

Table A2

Violations of Sphericity for Aim 2a

Variable	Mauchley's *W*	ε
Theta		
Location	.42*	.83
Location by Condition	.42*	.86
Location by Time	.34*	.80
Location by Condition by Time	.48*	.94
Alpha		
Location	.37*	.84
Location by Condition	.20*	.75
Location by Time	.26*	.75
Location by Time by Condition	.52*	.92
Beta		
Location	.49*	.87
Location by Condition	.48*	.89
Location by Time	.31*	.73
Location by Condition by Time	.52*	.94

*$p < .05$

df=14

Note. Wilks' Lambda multivariate statistics cited for all Aim2a statistics. Values for variables not violating assumptions of sphericity not listed.

Table A3

Violations of Sphericity for Aim 2b

Variable	Mauchley's *W*	ε
Theta		
Location	.35*	.74
Location by Condition	.44*	.86
Alpha		
Location	.30*	.78
Location by Condition	.35*	.82
Beta		
Location	.40*	.76
Location by Condition	.51*	.87

*$p < .05$

df=14

Note. Wilks' Lambda multivariate statistics cited for all Aim2b statistics. Values for variables not violating assumptions of sphericity not listed.

Appendix B

Means and Follow-Up Pairwise Comparisons for Significant Main Effects of Location in Aim 1

Table B1

Spectral Power by Location Across Conditions and Diagnostic Groups at Pre-Treatment, Descriptive Statistics

	OVERALL (ASD & TYP)	
Variable	Mean	SE
Theta		
Left Frontal	1.52	.071
Left Parietal	1.48	.076
Left Temporal	1.33	.071
Right Frontal	1.69	.073
Right Parietal	1.29	.069
Right Temporal	1.50	.073
Alpha		
Left Fontal	1.06	.075
Left Parietal	1.46	.091
Left Temporal	1.18	.085
Right Frontal	1.13	.076
Right Parietal	1.55	.090
Right Temporal	1.24	.084
Beta		
Left Frontal	1.65	.060

Left Parietal	1.59	.064
Left Temporal	1.56	.065
Right Frontal	1.75	.069
Right Parietal	1.62	.061
Right Temporal	1.61	.063

Note. See Figure 3 for electrode groupings within each location.

Table B2

Pairwise Comparisons for Main Effect of Location in Theta Band Across Conditions and Diagnostic Groups at Pre-Treatment (Aim I), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	.041	.195*	-.162*	.024	.236*
Left Parietal	--	--	.154*	-.203*	-.017	.195*
Left Temporal	--	--	--	-.357*	-.170*	.042
Right Frontal	--	--	--	--	.186*	.398*
Right Parietal	--	--	--	--	--	.212*

Note. * = $p < .05$. N = 67.

Table B3

Pairwise Comparisons for Main Effect of Location in Alpha Band Across Conditions and Diagnostic Groups at Pre-Treatment (Aim I), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	-.397*	-.123	-.073	-.487*	-.176*
Left Parietal	--	--	.274*	.324*	-.090	.221*
Left Temporal	--	--	--	.050	-.364*	-.053
Right Frontal	--	--	--	--	-.414*	-.103
Right Parietal	--	--	--	--	--	.311*

Note. * = $p < .05$. N = 67.

Table B4

Pairwise Comparisons for Main Effect of Location in Beta Band Across Conditions and Diagnostic Groups at Pre-Treatment (Aim I), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	.065	.093	-.103	.031	.039
Left Parietal	--	--	.028	-.168*	-.034	-.026
Left Temporal	--	--	--	-.196*	-.061	-.054
Right Frontal	--	--	--	--	.134*	.142*
Right Parietal	--	--	--	--	--	.007

Note. * = $p < .05$. N = 67.

Table B5

Spectral Power Within the Beta Band by Location and Condition Across Diagnostic Groups

	OVERALL (ASD & TYP)			
	EO		MONO	
Variable	Mean	SE	Mean	SE
Left Frontal	1.53	.062	1.77	.067
Left Parietal	1.53	.066	1.64	.067
Left Temporal	1.49	.067	1.63	.070
Right Frontal	1.66	.077	1.85	.074
Right Parietal	1.56	.067	1.68	.062
Right Temporal	1.51	.070	1.71	.069

Table B6

Paired Samples T-Tests Comparing Spectral Power Within the Beta Band Between Conditions (EO and MONO) Within Locations Across Diagnostic Groups (Aim I)

Location	*t*
Left Frontal	-5.83*
Left Parietal	-3.18*
Left Temporal	-3.55*
Right Frontal	-3.94*
Right Parietal	-3.64*
Right Temporal	-3.95*

Note. * = $p < .05$ (two-tailed). df=66. N= 67.

Appendix C

Means and Follow-Up Pairwise Comparisons for Significant Main Effects of Location in Aim IIa

Table C1

Spectral Power by Location Across Treatment Groups, Time and Conditions, Descriptive Statistics

	OVERALL (EXP & WL)	
Variable	Mean	SE
Theta		
Left Frontal	1.36	.062
Left Parietal	1.27	.075
Left Temporal	1.13	.070
Right Frontal	1.48	.063
Right Parietal	1.37	.074
Right Temporal	1.16	.060
Alpha		
Left Fontal	.934	.071
Left Parietal	1.30	.083
Left Temporal	1.07	.077
Right Frontal	1.03	.070
Right Parietal	1.37	.088
Right Temporal	1.11	.079
Beta		
Left Frontal	1.51	.061

Left Parietal	1.44	.057
Left Temporal	1.42	.056
Right Frontal	1.59	.061
Right Parietal	1.48	.059
Right Temporal	1.49	.056

Note. See Figure 3 for electrode groupings within each location.

Table C2

Pairwise Comparisons for Main Effect of Location in Theta Band Across Time, Conditions and Treatment Groups (Aim IIa), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	.084	.227*	-.123*	-.010	.195*
Left Parietal	--	--	.143*	-.207*	-.094	.111
Left Temporal	--	--	--	-.350*	-.237*	-.032
Right Frontal	--	--	--	--	.113	.317*
Right Parietal	--	--	--	--	--	.205*

Note. * = $p < .05$. N = 43.

Table C3

Pairwise Comparisons for Main Effect of Location in Alpha Band Across Time, Conditions and Treatment Groups (Aim IIa), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	-.369*	-.136*	-.100*	-.434*	-.178*
Left Parietal	--	--	.233*	.269*	-.065	.191*
Left Temporal	--	--	--	.036	-.298*	-.042
Right Frontal	--	--	--	--	-.332*	-.078
Right Parietal	--	--	--	--	--	.256*

Note. * = $p < .05$. N = 43.

Table C4

Pairwise Comparisons for Main Effect of Location in Beta Band Across Time, Conditions and Treatment Groups (Aim IIa), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	.071	.089	-.079	.031	.021
Left Parietal	--	--	.018	-.150*	-.040	-.050
Left Temporal	--	--	--	-.168*	-.058	-.068
Right Frontal	--	--	--	--	.110	.100
Right Parietal	--	--	--	--	--	-.010

Note. * = $p < .05$. N = 43.

Appendix D

Means and Follow-Up Pairwise Comparisons for Significant Main Effects of Location in Aim IIb

Table D1

Spectral Power by Location Across Treatment Groups and Conditions at Post-Treatment, Descriptive Statistics

	OVERALL (EXP, WL & TYP)	
Variable	Mean	SE
Theta		
Left Frontal	1.45	.061
Left Parietal	1.43	.065
Left Temporal	1.25	.065
Right Frontal	1.56	.052
Right Parietal	1.46	.066
Right Temporal	1.22	.055
Alpha		
Left Fontal	.991	.059
Left Parietal	1.45	.079
Left Temporal	1.16	.075
Right Frontal	1.11	.063
Right Parietal	1.48	.074
Right Temporal	1.16	.070
Beta		

Left Frontal	1.57	.055
Left Parietal	1.54	.053
Left Temporal	1.49	.054
Right Frontal	1.67	.052
Right Parietal	1.56	.053
Right Temporal	1.52	.052

Note. See Figure 3 for electrode groupings within each location.

Table D2

Pairwise Comparisons for Main Effect of Location in Theta Band Across Conditions and Treatment Groups (Aim IIb), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	.018	.195*	-.116*	-.011	-.230*
Left Parietal	--	--	1.77*	-.135*	-.030	.211*
Left Temporal	--	--	--	-.311*	-.206*	.035
Right Frontal	--	--	--	--	.105	.346*
Right Parietal	--	--	--	--	--	.241*

Note. * = $p < .05$. N = 67.

Table D3

Pairwise Comparisons for Main Effect of Location in Alpha Band Across Conditions and Treatment Groups (Aim IIb), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	-.457*	-.173*	-.122*	-.486*	-.169*
Left Parietal	--	--	.284*	.335*	-.028	.289*
Left Temporal	--	--	--	.051	-.313*	.004
Right Frontal	--	--	--	--	-.363*	-.046
Right Parietal	--	--	--	--	--	.317*

Note. * = $p < .05$. N = 67.

Table D4

Pairwise Comparisons for Main Effect of Location in Beta Band Across Conditions and Treatment Groups (Aim IIb), Mean Differences

Location	Left Frontal	Left Parietal	Left Temporal	Right Frontal	Right Parietal	Right Temporal
Left Frontal	--	.030	.089	-.096*	.011	.059
Left Parietal	--	--	.059	-.126*	-.019	.029
Left Temporal	--	--	--	-.185*	-.078	-.030
Right Frontal	--	--	--	--	.107	.155*
Right Parietal	--	--	--	--	--	.048

Note. * = $p < .05$. N = 67.