Examining the Durability of PEERS for Adolescents with ASD: Maintenance of Neurological and Behavioral Effects

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EXAMINING THE DURABILITY OF PEERS FOR ADOLESCENTS WITH ASD: MAINTENANCE OF NEUROLOGICAL AND BEHAVIORAL EFFECTS

by

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ABSTRACT

EXAMINING THE DURABILITY OF PEERS FOR ADOLESCENTS WITH ASD: MAINTENANCE OF NEUROLOGICAL AND BEHAVIORAL EFFECTS

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To date, there are no known published studies that have assessed the maintenance of treatment effects in the context of neurological changes and their relationship to behavioral outcomes following a social skills intervention for adolescents with Autism Spectrum Disorder (ASD). The few studies that have incorporated long-term assessment into their design have focused exclusively on sustained behavioral responses to treatment. Individuals with ASD across the lifespan exhibit aberrant neural activity, which is thought to underlie social skill deficits noted in persons on the spectrum. Thus, this study sought to examine the impact of a social skills intervention, the Program for the Education and Enrichment of Relational Skills (PEERS: Laugeson, Frankel, Mogil, & Dillon, 2009), on the maintenance of neural plasticity and treatment gains in social functioning. Neural activity was assessed via electroencephalography (EEG) in terms of spectral power and asymmetry, which also was compared to a cohort of typically developing adolescents. Additionally, behavioral outcomes, examining a variety of social domains, at pre, post, and 6-month follow-up, were investigated for their relationship to changes in EEG activity. Results revealed that adolescents with ASD demonstrated a decrease in gamma activity in the right temporal region following PEERS, which was maintained at 6-month follow-up. This sustained neural change related to fewer problem behaviors and improved social cognition, which highlights the role of neural plasticity as a mechanism for maintaining improvements in behavioral presentation following intervention.

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I. INTRODUCTION

Autism Spectrum Disorder (ASD) is a pervasive, developmental, and neurologically based disorder with rising prevalence rates (Matson & Kozlowsi, 2011). While the etiology of ASD remains unknown, the literature suggests that abnormalities in brain structure and function account for the social deficits observed in ASD. Furthermore, researchers have demonstrated that these neural substrates in ASD significantly differ from their counterparts in typically developing individuals. Social impairments represent a key feature of ASD, which have serious implications for academic achievement, occupational success, emotional well being, and mental health throughout development. Researchers emphasize the importance of early and continued intervention in remediating social impairments in individuals with ASD.

One important intervention opportunity consists of targeting social skill improvement in *adolescence* for individuals with ASD. First, social skill deficits associated with ASD do not improve or resolve with age (White, Keonig, Scahill, & 2007), which poses a problem in adolescence because teenagers place greater emphasis on social affiliations and friendships (Mitchel, Regehr, Reaume, & Feldman, 2010). Additionally, adolescence marks a period of rapid brain development (Sisk & Foster, 2004). Thus, social skill intervention has the potential to capitalize on neural changes, improve social behavior, and create a foundation for sustainable change.

Research examining social skills training groups rarely examines sustainability of treatment effects, and the few studies that have looked solely at behavioral responses to intervention. While research on the neural basis for response to intervention for persons with ASD is limited (Ventola, Oosting, Anderson, & Pelphrey, 2013), a few studies have

demonstrated significant changes in the brain following intervention (Dawson et al., 2012; Van Hecke et al., 2013; Voos et al., 2013).

To the author's knowledge, no known study has examined neural plasticity as a possible underlying mechanism for maintenance of treatment effects for adolescents with ASD. Neural plasticity occurs throughout the lifespan, and given the burst of brain development occurring in adolescence (Sisk & Foster, 2004), it is equally important to understand how treatment impacts and changes the brain of this age group of individuals with ASD. Behavior and environmental change alone may not adequately explain the maintenance of treatment effects. Thus, an important next step, aside from incorporating the collection of follow-up data into study design, is to understand the mechanisms driving maintenance (Lerner, White, & McPartland, 2012; Lord et al., 2005). One social skills intervention that has received extensive empirical support, the Program for the Education and Enrichment of Relational Skills (PEERS: Laugeson, Frankel, Mogil, & Dillon, 2009), creates an experience-driven opportunity for adolescents with ASD, which may translate into additional neural development and change, and thus sustained treatment improvements.

This manuscript will begin by reviewing social skill challenges for adolescents with high functioning ASD. Discussion of an intervention to address these impairments, PEERS, and research on social skill maintenance outcomes for adolescents with ASD will follow. Next, neural development and function in this population will be discussed, with an emphasis on electroencephalography (EEG) findings, as this method was used in the present study. Lastly, the current study, which aims to expand the existing PEERS

literature by investigating the durability of the program in the context of neurological changes and relations to behavioral improvements, and its findings, will be discussed.

A. Social Skills Challenges in ASD

Social skills enable individuals to interact appropriately with other people (Radley, Jenson, Clark, & O'Neill, 2014). People with well-developed social skills are typically liked and accepted by their peers, while individuals with underdeveloped social skills often experience rejection, feelings of loneliness, and low self-esteem (Patrick, 2008). Additionally, having proficient social skills affords acceptance in integrated settings (i.e., occupational) and the ability to live more independently (Wang & Spillane, 2009).

Social skills challenges are among the most commonly identified difficulties in ASD. Individuals with ASD struggle with social pragmatics (e.g., engaging in turn-taking in the conversation) and initiating social interaction, exhibit odd speech prosody (e.g., speaking in a monotone voice and lacking inflection), perseverate on special interests, and have difficulty with interpreting non-literal forms of language (e.g., sarcasm; Krasny, Williams, Provencal, & Ozonoff, 2003; Rao, Beidel, & Murray, 2008). For children with ASD, these difficulties with socialization negatively impact academic, emotional, and social development, which ultimately impedes their achievement of developmental milestones (Rao et al., 2008).

Beginning in preschool, children with ASD exhibit markedly impaired social skills, as compared to their typically developing peers. Elementary school leads to significant peer relational problems, and by adolescence, these problems manifest in outright peer rejection and ridicule (Church, Alisanski, & Amanullah, 2000). The picture

is equally bleak for adults with ASD; poor social skills translate to under- or unemployment and dissatisfying social relationships (Venter, Lord, & Schopler, 1992).

Individuals with high-functioning ASD possess average to superior levels of intelligence and perhaps struggle with and suffer the most from having difficulty socializing. Given their typical to high levels of intellectual functioning, they tend to recognize their deficits in this area during adolescence (Rao et al., 2008). One study highlighted this distressing insight, as children with high-functioning ASD rated their social skills significantly below that of their typically developing peers (Knott, Dunlop, & McKay, 2006).

Not surprisingly, social skill deficits among individuals with ASD often result in social ostracism and isolation, which translates into withdrawal and perpetual aloneness. Habitual isolation diminishes social motivation, which may exacerbate symptoms of depression (e.g., feeling irritable or hopeless). The latter is particularly concerning given the high rates of comorbid depression (Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006) and suicide (Hannon & Taylor, 2013) noted within the ASD population. Barnhill and Myles (2001) explain that by adolescence, 80% of persons with high-functioning ASD have been treated with antidepressant medication. Meanwhile, research supports the importance and benefits of friendship (Buhrmester, 1990). Specifically, friendships buffer the negative effects of difficult life events (e.g., divorce, loss of a relative, etc.), help ameliorate symptoms of depression, and improve independence and self-esteem (Buhrmester, 1990).

Ultimately, these social skill deficits foster a negatively reinforcing feedback loop, with negative peer interactions potentially leading to avoidance of social interaction

altogether and/or anxiety accompanying interacting with others. With this negative feedback loop in motion, social skill impairments, understanding of peer etiquette, and anxiety compound upon each other as social demands become more complex in adolescence and adulthood (Frankel et al., 2010). Not surprisingly, this negatively reinforcing loop impacts adolescents' ability to process social information, produce an appropriate response, and integrate with peers (Yeates et al., 2007). Furthermore, social amotivation is well documented in ASD, which undoubtedly affects social learning (Lerner et al., 2012), and negative social interactions likely continue to dampen social motivation.

B. PEERS

Given the marked social challenges adolescents with ASD face and the detrimental consequences of prolonged isolation on mental health and well being, intervening during this developmental period presents an opportunity to reverse this trajectory. Several research teams have sought to examine the efficacy of programs designed to target and improve social skill impairments in adolescents with ASD (see Kaat & Lecavalier, 2014; Mitchel et al., 2010; Rao et al., 2008; Reichow & Volkmar, 2010; Schreiber, 2011; Tse, Strulovitch, Tagalakis, Meng, & Fombonneet, 2007; Wang & Spillane, 2009; White et al., 2007, for reviews). While typically developing individuals rely on observational learning to acquire social skills, individuals with ASD have difficulty with interpreting others' perspectives and mental states. Thus, individuals with ASD benefit more from learning social etiquette via direct instruction, guided observation, and constant practice (Patrick, 2008). The social skills training group approach provides structure and teaches social skills during didactic instruction, coupled with role-plays,

behavioral rehearsals, and constructive feedback (Frankel et al., 2010; Rao et al., 2008; Schreiber, 2011).

PEERS (Laugeson et al., 2009) is a 14-week, empirically validated, manualized, outpatient treatment program designed to teach motivated adolescents with ASD the social skills required to make and maintain friendships (Laugeson et al., 2009). The program contains 14 modules that teach a variety of foundational social skill concepts (e.g., having a two-way conversation, initiating conversation, and handling arguments and disagreements; Laugeson et al., 2009). Each module consists of a didactic lesson that hones in on a particular social skill (e.g., the rules for trading information) and presents the content in simplified, concrete steps. See Table 1 for a listing of PEERS didactics and descriptions. The leader of the adolescent group demonstrates the highlighted skill through appropriate and/or inappropriate role-plays and asks the adolescents with ASD specific questions about how the rules were either implemented or broken. After observing the role-plays, the adolescents participate in behavioral rehearsals with one another to practice these skills. During the behavioral rehearsals, the leader of the adolescent group listens and provides coaching and feedback. To conclude each session, adolescents and their parents reunite to briefly review the didactic lesson and discuss the upcoming homework assignment. Homework assignments provide an opportunity for adolescents to generalize newly learned skills to their social hobbies and extracurricular activities. The parent group meets simultaneously in a separate room, discussing the previous week's homework assignment, and the group leader works with parents to troubleshoot any obstacles or problems that arose while adolescents completed the assignment. The parent group leader reviews and explains the content from the didactic

lesson, which helps parents understand what their adolescent is learning and allows them to serve as a "social coach" for their adolescent (Laugeson et al., 2009). Parents also learn techniques and strategies for providing constructive, positive feedback and identifying appropriate friend groups for their adolescent (Laugeson et al., 2009). The developers of PEERS found that the treatment group hosted and attended more get-togethers, exhibited improved knowledge of social skills, and demonstrated greater social responsiveness and fewer symptoms related to ASD, as per parents' report (Laugeson et al., 2009).

A study at an independent research facility replicated these findings (Schohl et al., 2013). Additionally, Dolan and colleagues (2016) examined *in vivo* social skills and noted that adolescents completing PEERS demonstrated significant improvement in vocal expressiveness and overall quality of rapport during a social interaction with an unfamiliar typically developing peer, as compared to adolescents in the waitlist control group. Taken together, these findings further support the program's efficacy. PEERS also has been culturally modified for use in Korea (Yoo et al., 2014). Yoo and colleagues' sample exhibited enhanced social skill knowledge and interpersonal skills, as well as a decrease in symptoms related to depression and ASD (Yoo et al., 2014), which parallels results from both the pilot study (Laugeson et al., 2009) and the replication (Schohl et al., 2013). This study highlights how the PEERS program, with modest cultural adjustments, is efficacious, and Yoo and colleagues' work represents one of the only trials of an empirically supported social skills intervention receiving cross-cultural validation. Validating efficacious social skills intervention programs for other cultures is of upmost importance, as ASD affects children globally, and understanding if and how interventions

work beyond the United States allows for providers around the world to implement empirically supported treatments.

PEERS is arguably the only extensively researched and empirically validated social skills intervention for adolescents with high-functioning ASD (Dolan et al., 2016; Laugeson et al., 2012; Mandelberg et al., 2014; Reichow, Steiner, & Volkmar 2013; Schohl et al., 2013; Van Hecke et al., 2013; Yoo et al., 2014).

C. Maintenance Outcomes for Social Skills Interventions

PEERS has garnered extensive empirical support from its site of development, as well as independent research sites. An important next step is to more fully examine maintenance of treatment gains following PEERS. Demonstrating treatment maintenance suggests that an intervention truly works, as the goal of treatment is for skills to generalize beyond the treatment setting, and perhaps most importantly, last into the future.

Broadly, there is limited evidence demonstrating long-term outcomes for social skills training groups designed for adolescents with ASD. To this author's knowledge, only three published studies (Beaumont & Sofronoff, 2008; Lerner et al., 2011; Mandelberg et al., 2014) examining social skills training groups for adolescents with ASD have incorporated evaluation of maintenance into their study design. Findings from these studies will be discussed in the paragraphs that follow.

Beaumont and Sofronoff (2008) examined the Junior Detective Training Program (JDTP), which is an 8-week social skills training group, consisting of small group sessions, computer games, parent training sessions, and teacher handouts. The researchers collected data not only at pre- and post- intervention, but also at 6 weeks post-treatment and 5 months following the program's completion (Beaumont & Sofronoff, 2008). The

investigators examined parent-report of their children's social skills, and the children with ASD also completed measures assessing emotion recognition and emotion management strategies. Clinically significant improvements in terms of parent-reported social functioning were maintained at both 6-week and 5-month follow-up time points. Notably, parent-report of social skills was the only outcome that maintained at the 5-month follow-up appointment, suggesting maintenance of treatment effects in the home environment (Beaumont & Sofronoff, 2008).

Lerner and colleagues (2011) also reported on 6-week follow-up data from the Socio-Dramatic Affective-Relational Intervention program (SDARI: Lerner & Levine, 2007). At 6 weeks post-treatment, parents whose adolescents participated in the SDARI program reported greater social assertiveness. Likewise, on a task requiring the adolescents to identify emotions in adult voices, adolescents in the SDARI group exhibited a decrease in errors, which maintained at long-term follow-up. As previously mentioned, the adolescents' maintenance of parent-reported social assertiveness along with improved ability to detect emotion in adult voices may be related to increased social confidence as a result of possessing greater interpretive accuracy of social situations, which may assist in decreasing problematic social interactions over time (Lerner et al., 2011). The authors also note that maintenance measurements were taken at the start of a new school year, which is promising that adolescents likely generalized these skills to peers and social situations at school (Lerner et al., 2011).

The developers of PEERS retrospectively examined long-term treatment outcomes of participants who had completed the program (Mandelberg et al., 2014). Fifty-three past participants who had completed the program 1-5 years prior, with an

average of 29 months post-PEERS completion, completed the same questionnaire measures at follow-up in order to make comparisons to pre- and post- treatment time points (Mandelberg et al., 2014). Overall, results at follow-up revealed that all outcome variables significantly improved from baseline. Specifically, total social skills and problem behaviors, which at post-treatment demonstrated significant improvements, maintained at follow-up (Mandelberg et al., 2014). Parent-reported social responsiveness also revealed maintained improvements at follow-up (Mandelberg et al., 2014). Notably, these improvements were not only statistically significant in terms of changing over time, but also, were in the same range to that of typically developing adolescents based on normative data. Interestingly, the authors note that past research utilizing these measures has not shown results in which children with ASD naturally improve and normalize over time (Mandelberg et al., 2014), which may suggest that the PEERS program has the ability to shift adolescents' social skill behavior much closer to typical limits than treatment as usual. The latter finding highlights that it is less likely that maturation alone accounts for improvements in this domain. Adolescents also demonstrated maintained treatment effects in terms of knowledge of PEERS concepts, as adolescents' scores on a knowledge questionnaire were significantly greater than their baseline scores. Additionally, the frequency of get-togethers, which had significantly improved immediately following the PEERS intervention, maintained at follow-up, suggesting that the adolescents were arranging and attending get-togethers with friends, which is a fundamental skill heavily emphasized in the PEERS program (Mandelberg et al., 2014). The fact that get-togethers remained significantly improved at follow-up points to experience-driven processes at play. That is, as the adolescents in PEERS learn skills,

practice them in their own environment, and encounter positive feedback and success, they continue to utilize the skills, which in turn reinforces their desire for social interaction, and thus, their willingness to approach other peers increases. Furthermore, data from this study suggested that adolescents who received PEERS and demonstrated maintenance of treatment effects were more likely to be accepted by their peers, given that instances of adolescents being invited to get-togethers by other adolescents also increased over time (Mandelberg et al., 2014).

Gresham, Sugai, and Horner (2001) describe effective strategies for teaching social skills, which include use of behavioral modeling and role-plays, behavioral rehearsal, and coaching with constructive feedback within a small-group setting. The studies outlined earlier (Beaumont & Sofronoff, 2008; Lerner et al., 2011; Mandelberg et al., 2014) all utilized each of the aforementioned methods, which highlight some of the positive features and elements of each intervention that likely contributed to the ultimate maintenance of effects. In terms of assessing maintenance, all of the studies incorporated a multi-method (i.e., at least two validated measures) and multi-informant (i.e., parent-, teacher-, and/or self- report) approach to assessing behavioral outcomes. Clearly, more treatment maintenance research for social skills training groups needs to be conducted; however, these studies add to the minimal literature in this area of intervention research. Interestingly, none of these studies mentioned the importance of examining the neural substrates of treatment maintenance when discussing future directions. Examining how the brain changes in response to treatment and understanding if these neural modifications maintain at long-term follow-up may explain a critical ingredient for how treatment gains last into the future.

D. Neural Activity in ASD: Evidence from EEG

It is clear that individuals with ASD experience challenges in social skills and interactions. Less clear, however, is exactly how these impairments come to be. Social impairments, often targeted in interventions for ASD, are thought to stem from atypical brain development and function. Broadly, when comparing individuals with ASD to their typically developing counterparts, brain activity looks quite different. Researchers have theorized that over-connectivity of local brain regions, coupled with aberrant and dysfunctional connectivity between long-range networks (Wang, Barstein, Ethridge, Mosconi, Takarae, & Sweeney, 2013), particularly in areas implicated in the "social brain" (i.e., frontal and temporoparietal regions; Volkmar, 2011), serve as a possible neurological underpinning of social skill deficits seen in ASD.

However, given the heterogeneity in clinical presentation of persons on the spectrum, it is not surprising that neuroimaging studies have reported discrepant findings. Furthermore, differences in neuroimaging techniques can make it challenging to compare and interpret results across studies. Nevertheless, there have been several consistent *structural* findings from magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI) that have suggested children with ASD experience brain overgrowth in the first few years of life (Cicchetti & Curtis 2006). White matter (i.e., myelinated axons that facilitate communication between functional networks) appears responsible for the early, abnormal brain overgrowth (Courchesne et al., 2001). By adolescence, teens and young adults with ASD exhibit a pattern of white matter reduction (Alexander et al., 2007; Blakemore & Choudhury, 2006; Courchesne, 2004; Keller, Kana, & Just, 2007). This reduced volume of white matter in adolescents with ASD contrasts sharply to the pattern

of white matter increase in healthy adolescents (Paus, 2010). Other research has noted that relative to controls, persons with ASD exhibit worse white matter integrity in structures involved in social engagement (e.g., frontal lobe, ventromedial prefrontal cortex, superior temporal sulcus, anterior cingulate cortex, and the amygdala; Barnea-Goraly et al., 2004). Clearly, ASD appears to impact multiple neural regions and networks (Cicchetti & Curtis, 2006).

In contrast to MRI and DTI methods, EEG noninvasively assesses neural *function* by using electrodes adhered to the scalp, or arranged in net or cap, to measure electrical changes in the postsynaptic activity of cortical neurons oriented perpendicular to the scalp (Wang et al., 2013). EEG data acquisition yields activity in five bands that oscillate at different frequencies and amplitudes, measured in hertz (Hz): 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). Research suggests that unique cognitive processes underlie each of the frequency bands (see Wang et al., 2013 for a review). For instance, delta waves are associated with sleep, theta is related to memory processes, alpha waves correspond to inhibition, beta waves have been linked to motor behavior and task engagement, and gamma waves are associated with higher order cognitive functions, such as sensory processing (Wang et al., 2013). While the literature suggests various associations between EEG frequency bands and cognitive processes, this remains an area of current scientific inquiry, especially in terms of examining the brain "at rest."

Resting-state EEG allows researchers to monitor neural oscillations in the absence of stimuli, which affords an opportunity to examine how the brain operates intrinsically (Wang et al., 2013). Resting-state EEG is indicative of the coordinated and organized

"idling" neural activity that may be necessary as a starting point for complex cognitive processes (e.g., social interaction; Cornew, Roberts, Blaskey, & Edgar, 2012). EEG research in ASD has examined the coordination of brain activity between electrode pairings (coherence) or the magnitude of activity at single predetermined regions (spectral power). Complex neuroanatomical homeostatic networks (i.e., brainstem, cortico-cortical, and cortico-thalamic) underlie EEG power within each of the frequency bands, which involve the brain's neurotransmitters (e.g., gamma-Aminobutyric acid (GABA) and glutamate; Billeci et al., 2013). A typically developing brain exhibits coordination of these systems and neurotransmitters, whereas persons with ASD demonstrate dysfunctional regulation (Billeci et al., 2013). Further, spectral EEG power has the potential to examine abnormalities unique to each specific frequency band (Wang et al., 2013).

While some discrepancies exist in the literature, spectral power analyses across multiple studies in ASD have revealed a few consistent findings. Individuals with ASD across development (i.e., childhood through adulthood) exhibit *excessive power* in the **delta** (Chan, Sze, & Cheung, 2007; Clarke et al., 2016; Stroganova et al., 2007;), **theta** (Clarke et al., 2016; Coben, Clarke, Hudspeth, & Barry, 2008), **beta** (Coben et al. 2008), and **gamma** (Orekhova et al., 2006) bands but *decremented activity* in the **alpha** band (Dawson, Klinger, Panagiotides, Lewy, & Castelloe, 1995) in comparison to their neurotypical peers. It is important to note that increased alpha activity represents inhibition of cortical activation in neural networks (Klimesch, Sauseng, & Hanslmayr, 2007; Wang et al., 2013). Thus, lower levels of alpha observed in persons with ASD suggest a lack of neural inhibition and over activation. Taken together, EEG assessment

indicates that individuals with ASD exhibit a neural profile of overactivity and dysregulation across frequency bands. These findings have been observed in a variety of brain regions. For instance, Coben and colleagues (2008) noted elevated theta activity specific to the right posterior region, while a study by Clarke et al. (2016) observed the same increases globally (i.e., across all regions). While differences in frequency band activity across scalp regions may be the product of methodological differences in data acquisition (e.g., variations in electrode groupings) and analysis (e.g., different processing programs), these findings highlight the likely involvement of multiple brain structures and networks implicated in atypical brain activity that underlies social skills deficits in ASD.

EEG asymmetry is an additional modality for assessing spectral power, and it provides a measure of neural activity in the left hemisphere as compared to the right. Researchers compute asymmetry by taking the spectral power within a predetermined region of interest (e.g., frontal lobe) or entire hemisphere and subtracting the corresponding values from the contralateral hemisphere (e.g., right frontal lobe – left frontal lobe). In subtracting left hemispheric activity from the right hemisphere, a more negative score would suggest greater left hemisphere activity in resting-state EEG, whereas a positive value would indicate greater right hemispheric contribution.

Research has suggested that abnormal EEG asymmetry may explain some of the impairments seen in ASD (Dawson, 1983). Specifically, right hemisphere behavioral asymmetries have been noted in several studies, in which participants with ASD demonstrate impairments in skills typically ascribed to the left hemisphere (e.g., verbal abilities) while tasks related to the right hemisphere appear advantaged and largely intact

(e.g., visuo-spatial skills; Ashwin, Wheelwright, & Baron-Cohen, 2005; Dawson, 1983; Gunter, Ghaziuddin, & Ellis, 2002; Rinehart, Bradshaw, Brereton, & Tonge, 2002). Anatomical asymmetry has also been noted: Floris and colleagues (2013) examined rightward asymmetry of several subregions of the corpus callosum, a large fiber tract connecting the two hemispheres, and discovered that rightward asymmetry of the posterior midbody of the corpus callosum was positively related to symptom severity (i.e., greater rightward asymmetry was correlated with more severe ASD symptoms). It should be noted that rightward structural asymmetries were observed for Floris et al.'s comparison group of typically developing males; however, the degree of the rightward asymmetry for the ASD group was more profound.

Other research has sought to interpret hemispheric asymmetries as relating to motivational systems of approach and withdrawal (Davidson, 1992; Fox, 1994). Davidson (1998) proposed that an approach-related, positive affective style is associated with left frontal hyperactivity, while a withdrawal-related, negative affective style is linked to increased activity of the right frontal hemisphere. Left frontal asymmetry also is related to positive peer interactions (Henderson, Marshall, Fox, & Rubin, 2004) while right frontal asymmetry correlates with behavioral inhibition, withdrawal, and depression (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001; Henderson et al., 2004). A majority of these findings are based on younger populations (i.e., preschool age); however, they have been replicated in older populations of school-age children and adults (Gray, 2001; Muris, Meesters, de Kanter, & Timmerman, 2005). Sutton and colleagues (2004) investigated resting state EEG in anterior regions (i.e., frontal lobe) in children with high-functioning ASD, as compared to typically developing children. In accordance with the

asymmetry literature, the investigators found that participants who exhibited right frontal asymmetry exhibited greater social impairments, as compared to their typically developing counterparts who displayed left frontal dominance (Sutton et al., 2004). Interestingly, a subgroup of children with ASD displayed increased left frontal activation, which was accompanied by greater insight into their social challenges (Sutton et al., 2004). It can be inferred that adolescents with ASD likely exhibit a neural profile consistent with social avoidance and withdrawal (i.e., rightward asymmetry) because social interactions are anxiety provoking and/or have led to negative outcomes (i.e., peer rejection) in the past. The latter indicates that social withdrawal in ASD, coupled with differences in neural activity, might have long-reaching effects on social development.

It is important to note that the aforementioned studies examining spectral power and asymmetry in individuals with ASD have assessed neural activity at one time point. That is, little work has examined neural change following social skills intervention for adolescents. Only one study, to this author's knowledge, by Van Hecke and colleagues (2013), examined EEG asymmetry following a social skills intervention (PEERS: Laugeson et al., 2009) and reported that the experimental group demonstrated a shift from right to left hemispheric EEG asymmetry post-intervention. These neural changes related to improved social skill knowledge and greater social contacts (i.e., get-togethers) at post-treatment. These findings were unique to the experimental group, as the waitlist group remained relatively unchanged from baseline to post-assessment. While these results require replication, the findings are promising, suggesting that the intervention elicits significant neural change. Aside from this study, little is known about neural plasticity in response to a social skills intervention for adolescents. Further, a critical

future direction is to investigate if these neural effects maintain following completion of PEERS.

E. Summary and Current Study

The variety of findings on neural function and structure in ASD in the literature attests to the heterogeneity of the disorder, as well as its theorized global impact on brain function. Contributions of left hemispheric dysfunction and impairments in connectivity likely impair the integration of information from various systems (Courchesne & Pierce, 2005), which impacts social functioning on a behavioral level. Furthermore, research examining EEG spectral power and asymmetry indicates that persons with ASD exhibit atypical neural oscillations, which appears related to over activation of neural networks, especially those recruited for social processing.

If the environment and its demands do not compensate for the unique profile of strengths and struggles observed in ASD, it may reinforce secondary psychopathology associated with ASD. An unaltered environment might foster additional neurological anomalies in activity, which highlights the effect and impact of neurological functioning on all levels of social development. The latter underscores the importance of and argument for social skill intervention in ASD, as non-compensatory environments might further prevent functional neuronal communication and connections, and thus, negatively affect social presentation (Cicchetti & Curtis, 2006). Neural plasticity may serve as a viable mechanism facilitating not only brain changes, but also, behavioral maintenance of treatment effects.

In summary, given that neural activity is disrupted in individuals with ASD, it is important to examine the impact of social skills intervention on neural plasticity and

improving neural network communication, and consequently, improving symptom presentation. Furthermore, if neural change ensues and maintains following a social skills intervention, it is equally important to examine how this impacts social behavior.

F. Aims of the Current Study

The specific aims of this study were to:

I. Examine whether neural activity changes for adolescents in PEERS over three time points (baseline before treatment, after treatment, and at long-term follow-up), as assessed via resting EEG spectral power.

II. Examine whether patterns of neural activity change for adolescents in PEERS over three time points (before treatment, after treatment, and at long-term follow-up), as assessed via resting EEG asymmetry.

III. Examine if spectral power and asymmetry change observed in adolescents receiving PEERS approximates that of typically developing adolescents at a maintenance time point, 6 months after completion of treatment.

IV. Examine if behavioral change for adolescents with ASD seen at PEERS treatment completion maintains 6 months after completion.

V. Explore the relationship between symptom improvement and neural change in response to PEERS at 6 months following treatment completion.

The hypotheses that were tested in the current study were as follows:

I. At 6-month follow-up, adolescents who received PEERS would demonstrate a significant change in EEG spectral power from baseline.

II. At 6-month follow-up, adolescents who received PEERS would demonstrate a significant change in EEG asymmetry from baseline.

III. At 6-month follow-up, the ASD group who received PEERS would approximate (i.e., not significantly differ) a typically developing adolescent group in terms of EEG spectral power and asymmetry.

IV. At 6-month follow-up, the ASD group who received PEERS would show significant improvement on all behavioral measures from their pre-treatment baseline assessment.

V. Based on the outcomes of hypotheses I-IV, neural findings and the behavioral measures that indicated a statistically significant change would be significantly related at 6-month follow-up.

II. METHOD

A. Participants

Sixty-three adolescents, ages 11-16, were recruited for participation in this study: 32 typically developing participants and 31 participants with ASD who completed PEERS. Typically developing adolescents were recruited via flyers and online advertisements. For inclusion in this study, typically developing teens did not have a history of ASD or a sibling with ASD. Additionally, their caregiver completed the Autism Spectrum Screening Questionnaire (ASSQ: Ehlers, Gillberg, & Wing, 1999) and Child Behavior Checklist (CBCL: Achenbach & Rescorla, 2001) to confirm the absence of symptoms consistent with ASD, as well as other behavioral concerns. Specifically, typically developing adolescents included in the present study received scores below 13 (raw score) on the ASSQ and 65 (T-score) on the CBCL, as per caregiver report.

Adolescents with ASD were recruited from Milwaukee-area schools and organizations, such as Easter Seals and the Autism Society of South Eastern Wisconsin (ASSEW). Participants who came in for the intake appointment but did not meet eligibility to participate in the study were compensated with a $30 Target gift card. Adolescents needed to meet the following criteria to be eligible for participation in the study: 1) 11-16 years old; 2) parent identified that adolescent has social problems/deficits; 3) is fluent in English; 4) has a parent or another family member who speaks English and was willing to participate in the study; 5) had no history of major mental illness (e.g., schizophrenia, bipolar disorder); 6) had no history of significant hearing, visual, or physical impairments that would hinder his or her ability to fully

participate in PEERS activities; 7) had a previous or current diagnosis of Autism Spectrum Disorder confirmed via the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al., 1999); 8) had a Verbal IQ of 70 or greater assessed via the Kaufman Brief Intelligence Test, Second Edition (KBIT-2: Kaufman & Kaufman, 2005); and 9) showed motivation and interest in participating in a class that teaches adolescents how to make and keep friends. To reduce attrition for eligible participants, families were given the PEERS intervention free of charge, and the adolescents received a prize at completion of the intervention.

Participants with ASD were randomly assigned to either the experimental or waitlist control group after meeting eligibility criteria for participation in the larger, randomized controlled trial study. Experimental group participants completed measures and began the 14-week intervention immediately, after which they completed the measures again. Waitlist group participants completed initial measures at the same time as the experimental group and then completed the measures again 14 weeks later. Within three months after completing the 14-week follow-up measurements, the waitlist group entered the 14-week intervention. Adolescents in the experimental group were the only participants asked to complete the measures for a third time point, six months following treatment. Given the study's aims and hypotheses to examine the durability of PEERS, only the adolescents from the experimental group who completed 6-month follow-up data and the typically developing adolescents were included in the analyses of this study.

In terms of demographic information, racial background for the experimental group consisted of 83.3% Caucasian, 6.7% Asian, 3.3% African American, 3.3% Pacific

Islander, and 3.4% did not disclose racial background. For the neurotypical group, racial background was comprised of 96.9% Caucasian and 3.1% endorsed biracial background.

Mean age for the adolescents with ASD was 13.61 years (*SD* = 1.38) and 13.12 years (*SD* = 1.41) for the typically developing teens. For the experimental group, 87.1% were male and right-handed, respectively. The typically developing group was comprised of 93.8% male and 90.6% right-handed participants. General cognitive abilities were in the average range for the experimental group (*M* = 104.7; *SD* = 18.02) and typically developing group (*M* = 107.94; *SD* = 13.55). ASD diagnoses confirmed via the ADOS-G revealed a mean communication score of 3.61 (*SD* = 1.10), a mean social score of 7.35 (*SD* = 2.14), and a mean total score of 10.97 (*SD* = 2.82).

Examining concurrent pharmacological intervention in the ASD group, 61.3% were currently prescribed medication, 35.5% were never prescribed medication, and 3.2% were formerly prescribed medication. In terms of specific medication classes, 35.7% were prescribed stimulants, 25% mood stabilizers, 21.4% selective serotonin reuptake inhibitors, 10.7% selective α_{2A} receptor agonists, 3.5% tricyclic antidepressants, and 3.5% serotonin and norepinephrine reuptake inhibitors. All typically developing adolescents were un-medicated. See Table 2 for complete demographic information, as well as information regarding parental age, education, and income.

B. Procedure

Data collection took place at Marquette University in the Center for Psychological Services and the Marquette Autism Project (MAP) laboratory. For the typically developing adolescents, there was only one session for data collection (approximately 2 hours). At this appointment, neurotypical teens and their caregivers provided informed

assent and consent, respectively. Additionally, caregivers of the typically developing participants completed behavioral measures regarding their adolescents' social and emotional functioning, and adolescents completed the resting-state EEG recording (see the Electroencephalogram Session section below for details).

There were three points of data collection for adolescents with ASD in the experimental group: one at the intake before the PEERS intervention (approximately 3.5 hours), a second, outtake, after the PEERS intervention ended, and a third session six months following the completion of PEERS. Interested participants on an in-house registry list for treatment were randomly assigned to either the experimental group or the waitlist control group. If the adolescent met inclusion criteria during the intake (see above), adolescents and their parents provided informed assent and consent, respectively, and completed a variety of self-report measures on social, emotional, and adaptive functioning. Then, at that same appointment, adolescents and their caregivers were escorted to the MAP laboratory for the adolescents to complete a neurophysiological assessment, which included the resting-state EEG recording (see the Electroencephalogram Session section below for details).

Adolescents assigned to the experimental group began the PEERS intervention approximately two weeks after they completed the intake. For adolescents in the experimental group, PEERS met for 14 sessions for approximately 1.5 hours each session. Following the completion of the 14-week intervention, outtakes were scheduled and consisted of completing the same self-report measures for both adolescents and parents and adolescents' neurophysiological measures. This process was repeated for adolescents in the experimental group that participated in the 6-month follow-up session.

All participant data was stored on a password protected hard drive. All data were de-identified, as participants were assigned a unique ID number at the time of their intake. Only graduate students on the research team and the faculty supervisor, Dr. Amy Vaughan Van Hecke, had access to any identifying information. Any paper materials (e.g., consent and assent forms) were stored in a locked file cabinet in the laboratory. Data collection for this study was reviewed and approved by the Marquette University Institutional Review Board (IRB). All procedures performed protected human subjects and were in accordance with Marquette's IRB ethical standards and the 1964 Helsinki declaration and its later amendments.

C. Measures

Electroencephalogram Session. Adolescents sat in a comfortable chair, facing a 19-inch computer monitor located approximately four feet away. Adolescents' caregivers were seated in an adjacent room so as to reduce any distraction during the EEG recording. Based on the adolescents' head circumference, an appropriately sized 64-channel electrode net (Electrical Geodesics, Inc., Eugene, OR) was selected and positioned, following standard capping procedures. All impedances were at or below 50 kOhm. Continuous resting-state EEG during an eyes open condition was collected for three minutes. Electrical activity was amplified and sampled at 1,000 Hz using a Netamps 300 (Electrical Geodesics, Inc., Eugene, OR). The graduate research assistant instructed adolescents to look straight ahead at a black cross (e.g., a fixation point) on a gray background on the computer monitor while remaining as still and relaxed as possible. Adolescents' alertness and attention to the fixation point were simultaneously videotaped in order to assess for potential movement artifacts (e.g., excessive blinking, head and neck movement, etc.). EEG is non-invasive and flexible, which is particularly conducive for research in this population, as EEG recordings do not require reclining in a confined space or being exposed to loud noises (e.g., scanning tube in MRI).

EEG Data Analysis. EEG recordings were filtered from 0.3 to 100 Hz. The EEG files were then exported from NetStation software (Electrical Geodesics, Inc., Eugene, OR) to MATLAB and processed using custom scripts, as well as EEGLAB functions (Delorme & Makeig, 2004). EEG data were re-referenced to an average reference, including the reference electrode. Low frequency noise was band-pass filtered from 2 to 100 Hz. Power line noise was notch filtered from 59 to 61 Hz using an 8th order,

Butterworth, zero-phase filter. Data were epoched into one-second intervals and large movement artifact was automatically rejected using the *pop_autorej* function (EEGLAB; Delorme & Makeig, 2004). To correct for additional artifacts, the remaining epoched data were broken down via an adaptive mixture independent component analysis (AMICA: Palmer et al., 2008). The artifact components were identified using ADJUST (Mognon, Jovicich, Bruzzone, & Buiatti, 2010) and custom scripts. After completion of the aforementioned procedures, the remaining data were used to calculate the average power spectral density using Welch's method (1024pt segments, 50% overlap) for each of the 64 electrodes. Lastly, spectral powers were 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 computing the area under the average spectrums. Power values were averaged across electrodes in the left and right frontal, temporal, and parietal regions, respectively, within each frequency band. See Figure 1 for spectral power electrode groupings. Additionally, power values for asymmetry calculations were computed by averaging power across all electrodes in the left and right hemispheres, respectively, and then subtracting average right minus average left hemispheric activity. Thus, positive asymmetry scores indicate relatively more right-hemisphere activity, whereas negative asymmetry scores indicate relatively more left hemisphere activation. See Figure 2 for asymmetry electrode groupings. All data were natural-logarithm transformed to correct for violations of normality innate in spectral power values.

Quality of Socialization Questionnaire-Revised (QSQ-R: Laugeson et al., 2012). Caregivers completed the QSQ-R, which measures the frequency of adolescents' get-togethers with peers. The questionnaire asks caregivers to identify how many get-

togethers their adolescent initiated, as well as how many get-togethers their adolescent was invited to by peers, within the past month. The present study combined these two variables to assess adolescents' total social contacts (i.e., sum of organized and invited get-togethers). Cronbach's alpha coefficient for total social contact was .85. The QSQ-R was administered to caregivers at pre, post, and 6-month follow-up.

Social Responsiveness Scale (SRS: Constantino, 2005). The SRS is a caregiver-report, assessing global and specific characteristics of ASD. The measure consists of 65 items, assessing social awareness, reciprocal social communication, social anxiety, social information processing, and traits associated with ASD. The SRS produces scores for five subscales: Social Awareness, Social Cognition, Social Communication, Social Motivation, and Autistic Mannerisms. Higher scores on the SRS indicate greater symptom severity and impairment. The SRS has good established internal validity and reliability, with all scales reporting $\alpha > .70$ (Constantino et al., 2003). In the present study, Cronbach's alpha coefficient was acceptable across all domains: Social Awareness ($\alpha = .71$); Social Cognition ($\alpha = .77$); Social Communication ($\alpha = .81$); Social Motivation ($\alpha = .74$); and Autism Mannerisms ($\alpha = .77$). Caregivers for adolescents in the experimental group completed the SRS at pre, post, and 6-month follow-up.

Social Skills Improvement System (SSIS: Gresham & Elliott, 2007). The SSIS is a 65-item caregiver rating scale designed to assess individuals' social skills and problem behavior. The Social Skills domain includes items pertaining to communication, cooperation, assertion, responsibility, empathy, engagement, and self-control. The Problem Behaviors scale measures behaviors that interfere with the acquisition or performance of socially appropriate behaviors. The SSIS has good established internal

validity and reliability, with all scales reporting $\alpha > .70$ (Gresham & Elliott, 2007). Cronbach's alpha coefficient in the present study was .88 for the Social Skills domain and .90 for the Problem Behaviors subscale. Caregivers completed the SSIS at pre, post, and 6-month follow-up.

Test of Adolescent Social Skills Knowledge (TASSK: Laugeson & Frankel, 2006). The TASSK consists of 26 items assessing adolescents' knowledge about the specific social skills taught during PEERS. The measure consists of two items from each of the 13 didactic lessons. Items on the TASSK consist of sentence stems with two possible answers. Total scores range from 0 to 26, with higher scores reflecting greater knowledge of the social skills taught in PEERS. Given the range of topics and lack of subscales on this questionnaire, Cronbach's reliability alpha was not computed for the TASSK. Adolescents receiving PEERS completed the TASSK at pre, post, and 6-month follow-up.

D. Data Analytic Plan

Hypothesis I. To examine whether EEG spectral power changed for adolescents with ASD in PEERS over time, a 3 x 5 x 6 repeated-measures analysis of variance (ANOVA) was conducted. Specifically, in order to examine a maintenance effect over time, TIME (3 levels: pre, post, and 6-month follow-up), frequency BAND (5 levels: delta, theta, alpha, beta, and gamma), and scalp LOCATION (6 levels: left and right frontal, temporal, and parietal, respectively) served as within-subject factors. Any significant main effects and interactions were followed with appropriate simple effects tests, controlling for Type 1 error rate. This hypothesis would be minimally supported if there was a main effect of time, with post hoc analyses indicating either 1) a significant

mean difference in EEG power between time 1 (pre-treatment) and time 3 (6-month follow-up), or 2) a significant mean difference, with time 1 differing from both time 2 and time 3. Significant interactions with time as a factor, and follow-up tests indicating one of the two patterns of mean differences in time above, also would indicate support for hypothesis I.

Hypothesis II. To examine whether EEG asymmetry changes for adolescents with ASD in PEERS over time, a 3 x 5 repeated-measures ANOVA was conducted, with TIME (3 levels: pre, post, and 6-month follow-up), and frequency BAND ASYMMETRY (5 levels: asymmetry in delta, theta, alpha, beta, and gamma, respectively) as the within subjects factors. First, asymmetry variables (right hemisphere average spectral power – left hemisphere average spectral power) for each of the frequency bands were computed for all time points (i.e., asymmetry at pre, post, and 6-month follow-up). More negative asymmetry scores indicate greater relative left hemisphere activity/dominance. Any significant main effects and interactions were followed with appropriate simple effects tests, controlling for Type 1 error rate. Similar to Hypothesis I, this hypothesis would be minimally supported if there was a main effect of time, with post hoc analyses indicating either 1) a significant mean difference in EEG asymmetry between time 1 (pre-treatment) and time 3 (6-month follow-up), or 2) a significant mean difference, with time 1 differing from both time 2 and time 3. Significant interactions with time as a factor, and follow-up tests indicating one of the two patterns of mean differences in time above, also would indicate support for hypothesis II.

Hypothesis III. To examine if the neural patterns observed in adolescents with ASD who received PEERS approximated that of typically developing adolescents, two analyses were conducted. First, to examine if the adolescents receiving PEERS approximated typically developing adolescents in terms of EEG spectral power, independent samples t-tests were computed to compare adolescents in the experimental group at baseline and 6-month follow-up, respectively, to the typically developing adolescents at baseline. Scalp locations included in the analyses to examine spectral power differences between groups were determined based on results from Aim I. Secondly, similar t-test comparisons between groups utilized EEG asymmetry as the dependent variable, comparing adolescents with ASD at baseline and 6-month follow-up, respectively, to the neurotypical adolescents at baseline. This hypothesis would be supported if the ASD and typically developing groups did *not* show statistically significant differences in EEG power and asymmetry at 6-month follow-up.

Hypothesis IV. To examine the maintenance of behavioral change in response to PEERS for adolescents with ASD, a repeated-measures multivariate analysis of variance (MANOVA) was conducted with time (pre, post, and 6-month follow-up) as the repeated factor and each behavioral measure (i.e., QSQ-R, SRS: Social Awareness, Social Cognition, Social Communication, Social Motivation, Autism Mannerisms; SSIS: Social Skills, Problem Behaviors; TASSK) as the dependent variables. This hypothesis would be supported if a significant effect of time were found, with post hoc tests indicating that time 1 significantly differed from time 3, and/or that time 1 differed from both time 2 and time 3.

Hypothesis V. Finally, to explore the relationship between symptom improvement and neural change in response to PEERS for adolescents with ASD, outcomes from hypotheses I-IV informed a set of exploratory bivariate correlation analyses. These analyses examined the relations amongst the change scores, from time 1 to time 3, in spectral EEG power, asymmetry, and the behavioral measures. Variables were chosen for inclusion based on patterns of significance in Hypotheses I-IV (i.e., measures that did not show change in the expected direction were not included in the correlation matrix).

III. RESULTS

A. Data Screening

All statistical analyses were completed using SPSS 22.0 (IBM, 2013) and analyzed at $p < .05$. All data were screened for normality and outliers. Outlying values were assessed at pre, post, and follow-up assessment. 2.4% and 1.9% of the EEG and behavioral data, respectively, were winsorized by replacing the outlying value with the next most extreme value in the distribution (Howell, 2012). Violations of sphericity are noted in Appendix A. All repeated-measures ANOVAs cite Huynh-Feldt corrected values when sphericity was violated. Descriptive statistics for sample characteristics are located in Table 2. Exploratory analyses were conducted in order to evaluate any influences of gender or handedness, which did not yield any significant differences in results. Thus, to preserve power, participants who were female and/or left-handed were retained in the analyses that follow.

B. Aim I: Changes in EEG Spectral Power

A 3 x 5 x 6 repeated-measures ANOVA with TIME (pre, post, 6-month follow-up) x BAND (delta, theta, alpha, beta, gamma) x LOCATION (left and right frontal, parietal, and temporal regions, respectively) as the within-subjects factors was conducted to examine whether EEG spectral power changes for adolescents with ASD in PEERS over time.

There was a **significant main effect for TIME**, F (1.41, 42.17) = 4.22, p = .034, partial η^2 = .12, observed power = .61. Bonferroni pairwise comparisons at each time point across band and location indicated that neural activity at 6-month follow-up was

significantly lower than at post-PEERS. Table B1 contains means and standard error for the omnibus main effect for TIME. See Table B2 for pairwise comparisons for TIME.

There was a **significant main effect for BAND**, F (2.57, 77.19) = 46.67, p < .001, partial η^2 = .61, observed power = 1.00. Bonferroni pairwise comparisons indicated that neural activity, across time and location, in **delta** was significantly greater than theta, and **gamma** activity was significantly lower compared to all of the bands. Table B3 contains means and standard error for the omnibus main effect for BAND. See Table B4 for pairwise comparisons for BAND.

There also was **a significant main effect for LOCATION**, F (2.57, 76.99) = 10.26, p < .001, partial η^2 = .26, observed power = .99. Bonferroni pairwise comparisons indicated that there were significant neural differences in the following locations collapsed across time and band: **left frontal** activity was significantly greater than left and right temporal, respectively, but lower than right frontal activation; **left temporal** activity was significantly lower than left parietal, right frontal, and right parietal activation; **left parietal** activity was significantly lower than right temporal activity; **right frontal** activity was significantly greater than right temporal activity; and **right temporal** activity was significantly greater than activity in the right parietal region. Table B5 contains means and standard error for the omnibus main effect for LOCATION. See Table B6 for pairwise comparisons for LOCATION.

There were no statistically significant interactions for TIME x BAND, F (4.72, 141.53) = 1.48, *ns*, or TIME x LOCATION, F (6.93, 207.81) = .81, *ns*. There was a **significant interaction for BAND x LOCATION**, F (5.14, 154.05) = 26.10, p < .001, partial η^2 = .26, observed power = 1.00. To examine this interaction, a simple effects test

was conducted, splitting the file by BAND and examining LOCATION as the within-subjects factor. A Bonferroni corrected alpha level of .009 was used to adjust for multiple comparisons. There was **a significant main effect for LOCATION in the delta band**, F (2.33, 69.92) = 93.55, $p < .001$, partial $\eta^2 = .76$, observed power = 1.00. There was **a significant main effect for LOCATION in the theta band**, F (2.25, 67.54) = 16.07, $p < .001$, partial $\eta^2 = .35$, observed power = 1.00. There was **a significant main effect for LOCATION in the alpha band**, F (3.01, 90.26) = 26.46, $p < .001$, partial $\eta^2 = .47$, observed power = 1.00. There was **a significant main effect for LOCATION in the beta band**, F (3.74, 112.12) = 5.34, $p < .001$, partial $\eta^2 = .15$, observed power = .96. Within each band, most locations significantly differed from one another. The greatest number of differences between locations emerged within the delta, theta, and alpha bands. Conversely, within the beta band, only left frontal activation was significantly lower than right frontal activity, and right frontal activity was significantly greater than right temporal activation. Table B7 contains means and standard error for the simple effects test, examining the omnibus interaction for BAND x LOCATION. See Table B8 for corresponding pairwise comparisons. There were no significant main effects for LOCATION in the gamma band, F (2.52, 75.56) = 3.31, *ns*.

The significant omnibus main effects for TIME, BAND, and LOCATION and two-way interaction for BAND x LOCATION were qualified by **a significant three-way interaction for TIME x BAND x LOCATION**, F (10.98, 329.29) = 1.91, $p = .038$, partial $\eta^2 = .06$, observed power = .86. This three-way interaction was followed by a test of simple interaction effects, which is described in the paragraphs that follow.

For the test of simple interaction effects, the file was split by LOCATION in order to assess TIME x BAND. A Bonferroni corrected alpha level of .009 was used to adjust for multiple comparisons. There were no significant main effects for TIME within the left frontal, F (1.80, 54.10) = 2.09, *ns*; left temporal, F (1.39, 41.79) = 5.93, *ns*; left parietal, F (1.46, 43.65) = 2.01, *ns*; right frontal, F (1.97, 59.01) = 3.99, *ns*; right temporal F (1.54, 46.32) = 4.61, *ns*; or right parietal regions, F (1.56, 46.71) = 2.75, *ns*.

There were significant main effects for BAND within all six locations: **left frontal**, F (2.47, 74.16) = 40.80, $p < .001$, partial $\eta^2 = .58$, observed power = 1.00; **left temporal**, F (2.79, 83.55) = 34.76, $p < .001$, partial $\eta^2 = .54$, observed power = 1.00; **left parietal**, F (2.47, 73.99) = 61.19, $p < .001$, partial $\eta^2 = .67$, observed power = 1.00; **right frontal**, F (2.70, 80.85) = 40.93 $p < .001$, partial $\eta^2 = .58$, observed power = 1.00; **right temporal**, F (2.49, 74.55) = 33.91, $p < .001$, partial $\eta^2 = .53$, observed power = 1.00; **right parietal,** F (2.61, 78.14) = 51.99, $p < .001$, partial $\eta^2 = .63$, observed power = 1.00. Table B9 contains means and standard error for locations that demonstrated a main effect for BAND. See Table B10 for corresponding pairwise comparisons.

There were no interactions for TIME x BAND within the left frontal, F (4.14, 124.17) = 1.65, *ns*; left temporal, F (5.03, 150.87) = 1.82, *ns*; left parietal, F (4.73, 142.02) = .67, *ns*; right frontal, F (3.96, 118.80) = 2.08, *ns*; or right parietal regions, F (4.16, 124.73) = .85, *ns*. There was a **significant interaction for TIME x BAND within the right temporal region**, F (4.85, 145.46) = 2.76, $p = .007$, partial $\eta^2 = .08$, observed power = .81. To follow this interaction, simple effects tests were conducted, splitting the file by BAND within the right temporal region and assessing the within-subjects effect of TIME. A Bonferroni corrected alpha level of .01 was used to adjust for multiple

comparisons. Results revealed that there was **a significant main effect for TIME within the gamma frequency band in the right temporal region**, F (1.59, 47.69) = 7.76, p = .002, partial η^2 = .21, observed power = .89. Specifically, Bonferroni pairwise comparisons revealed that adolescents receiving PEERS significantly decreased in gamma activity in the right temporal region from pre- to post-treatment, and this effect maintained at 6-month follow-up. See Table B11 for the means and standard error for the main effect of TIME within the right temporal region and gamma band. See Table B12 for pairwise comparisons within the gamma band in the right temporal region, examining the main effect of TIME. There were no significant main effects for TIME for delta, F (1.78, 53.51) = 3.59, *ns*; theta, F (1.53, 45.77) = 4.28, *ns*; alpha, F (1.60, 47.94) = 1.19, *ns*; or beta, F (2, 60) = 3.34, *ns*, in examining neural activity in the right temporal region.

C. Aim II: Changes in Neural Asymmetry

A 3 x 5 repeated-measures ANOVA with TIME (pre, post, and 6-month follow-up) and BAND ASYMMETRY (asymmetry in delta, theta, alpha, beta, and gamma, respectively) as the within-subjects factors was conducted. There were no significant main effects for TIME, F (2, 60) = .38, *ns*, or BAND ASYMMETRY, F (1.83, 54.83) = 3.09, *ns*. The interaction for TIME x BAND ASYMMETRY also was not significant, F (2.49, 74.76) = .42, *ns*. See Table B13 for means and standard deviations for band asymmetry values at each time point.

D. Aim III: Comparison to Typically Developing Adolescents

To examine if neural patterns at 6-month follow-up in adolescents with ASD who completed PEERS approximated that of typically developing adolescents, two sets of

independent sample t-tests were conducted. One set of analyses compared gamma spectral power in the right temporal region between the two groups, given that this was a significant finding for the adolescents with ASD. Gamma spectral power in the right temporal region at pre-treatment and 6-month follow-up for adolescents with ASD were separately compared to the typically developing adolescents' baseline assessment. A Bonferroni corrected alpha level of .025 was used to adjust for multiple comparisons. Independent samples t-tests comparing right temporal gamma activation at pre-treatment did not reveal any significant differences between the two groups, t (61) = .33, *ns*. Similarly, there were no significant differences noted when comparing right temporal gamma band activity at 6-month follow-up for adolescents with ASD to the typically developing controls' initial assessment, t (61) = 1.86, *ns*. Refer to Tables B14 and B15 for means and standard deviations.

Although there were no significant changes in asymmetry over time for adolescents who completed PEERS, asymmetry differences between the two groups were still explored. Adolescents' with ASD data at pre-treatment and 6-month follow-up, respectively, were compared to the baseline assessment of the typically developing adolescents within each of the five bands for asymmetry. For the second set of t-test comparisons, a Bonferroni corrected alpha level of .01 was used to adjust for multiple comparisons. No significant differences were noted for neural asymmetry within any of the bands between the two groups at either baseline or 6-month follow-up. See Tables B16 and B17 for means, standard deviations, and t-test values.

E. Aim IV: Maintenance of Behavioral Findings

Given the large number of behavioral measures, all dependent variables were entered into a repeated-measures omnibus MANOVA with TIME (pre, post, and 6-month follow-up) as the within-subjects factor. Results revealed a **significant main effect for TIME** for the combined outcome measures, F (18, 80) = 15.05, $p < .001$, partial $\eta^2 = .77$, observed power = 1.00. Each outcome measure was subsequently analyzed at the univariate level via one-way repeated-measures ANOVA with TIME (pre, post, and 6-month follow-up) as the within-subjects factor and the behavioral measure/domain as the dependent variable. Outcomes that demonstrated a significant main effect for TIME were followed up with Bonferroni pairwise comparisons to determine which time points significantly differed. Each behavioral measure is described in the paragraphs that follow.

QSQ-R. There was a **significant main effect for TIME**, F (2, 48) = 12.97, $p < .001$, partial $\eta^2 = .35$, observed power = 1.00. Adolescents who completed PEERS demonstrated a significant increase in social contacts (i.e., hosted and invited get-togethers) post-PEERS and 6 months following treatment completion, as compared to baseline. See Table B18 for means and standard deviations and Table B19 for pairwise comparisons.

SRS. The **main effect of TIME for Social Awareness was significant**, F (2, 48) = 6.00, $p = .004$, partial $\eta^2 = .21$, observed power = .87. Adolescents significantly improved in caregiver-reported social awareness at post-PEERS and 6-month follow-up as compared to baseline. See Table B20 for means and standard deviations and Table B21 for pairwise comparisons.

The **main effect of TIME for Social Cognition was significant**, F (1.68, 40.31) = 15.62, $p < .001$, partial $\eta^2 = .39$, observed power = 1.00. Caregivers' report of adolescents' social cognition improved at post-treatment and maintained 6 months later. See Table B22 for means and standard deviations and Table B23 for pairwise comparisons.

The **main effect of TIME for Social Communication was significant**, F (2, 48) = 12.89, $p < .001$, partial $\eta^2 = .35$, observed power = 1.00. Caregivers reported that adolescents' social communication significantly improved from pre- to post-intervention and maintained at 6-month follow-up. See Table B24 for means and standard deviations and Table B25 for pairwise comparisons.

The **main effect of TIME for Social Motivation was significant**, F (2, 48) = 8.86, $p = .001$, partial $\eta^2 = .27$, observed power = .96. Adolescents in PEERS demonstrated a significant improvement in social awareness at post-treatment, and this effect maintained at 6-month follow-up. See Table B26 for means and standard deviations and Table B27 for pairwise comparisons.

The **main effect of TIME for Autism Mannerisms was significant**, F (2, 48) = 7.68, $p = .001$, partial $\eta^2 = .24$, observed power = .94. Adolescents who completed PEERS demonstrated a significant improvement in Autism Mannerisms at post-treatment and maintained this treatment effect at 6-month follow-up. See Table B28 for means and standard deviations and Table B29 for pairwise comparisons.

SSIS. There was a **significant main effect for TIME in the Social Skills domain**, F (2, 48) = 9.14, $p < .001$, partial $\eta^2 = .28$, observed power = .97. Adolescents who received PEERS exhibited improvements in caregiver-reported social skills on the

SSIS, with post-treatment and 6-month follow-up being significantly different from baseline. See Table B30 for means and standard deviations and Table B31 for pairwise comparisons.

There also was **a significant main effect for TIME in the Problem Behaviors domain**, F (1.63, 41.50) = 6.00, p = .007, partial η^2 = .20, observed power = .82, with caregivers rating improvements in problem behaviors (i.e., lower scores indicate improvement) at 6-month follow-up compared to baseline. See Table B32 for means and standard deviations and Table B33 for pairwise comparisons.

TASSK. There was **a significant main effect for TIME**, F (2, 48) = 241.72, p < .001, partial η^2 = .91, observed power = 1.00. Adolescents demonstrated significant improvement in knowledge of PEERS concepts at post-treatment, and this effect maintained at 6-month follow-up. See Table B34 for means and standard deviations and Table B35 for pairwise comparisons.

F. Aim V: Relations between Symptom Improvement and Neural Change

Bivariate Pearson product-moment correlations were conducted to examine the relationship between symptom improvement and neural changes. Change scores (pre minus 6-month follow-up) were computed for all of the behavioral measures and gamma band activity in the right temporal region. There was **a significant negative, moderate correlation with right temporal gamma activity and SSIS Problem Behaviors**, r (30) = -.40, p = .027. Specifically, a significant decrease in gamma activity was related to improvements in caregiver rated problem behaviors. Additionally, there was **a significant negative, moderate correlation with right temporal gamma activity and SRS Social Cognition**, r (28) = -.40, p = .033. Significant improvements in caregiver reported social

cognition related to decreases in gamma band activity in the right temporal region. See Table B36 for the correlation matrix.

IV. DISCUSSION

To date, there is a paucity of literature that has examined the maintenance of treatment effects for adolescents with ASD receiving social skills intervention. Given that ASD is a neurologically-based condition and one study demonstrated neural plasticity following the PEERS intervention (Van Hecke et al., 2013), the current study investigated maintenance of neural change in response to PEERS and examined if these changes related to behavioral presentation of adolescents with ASD. Specifically, EEG was used as a proxy for measuring neural change and its relationship to improvements in social behavior.

The first aim of the current study investigated the maintenance of neural plasticity in EEG spectral power 6 months following treatment. Results supported the hypothesis for Aim I, in that adolescents who completed PEERS demonstrated a significant decrease in gamma band activity in the right temporal region post-PEERS, and this finding maintained at 6-month follow-up. Examination of bands oscillating at higher frequencies is relatively new in the literature. Technological advances in the amplification and analysis of higher-frequency bands with small amplitudes have allowed for examination of gamma (Herrman & Demiralp, 2005).

Previous EEG studies indicate that individuals with ASD possess elevated gamma band activity at rest (Cornew et al., 2012; Machado et al., 2015; Orekhova et al., 2006; Stroganova et al., 2011). Studies have observed these elevated gamma oscillations over the midline (Coben et al., 2012; Machado et al., 2015), occipital, and parietal (Murias, Webb, Greenson, & Dawson, 2007), and posterior (Cornew et al., 2012; Orekhova et al., 2007) regions. The current study's finding from the first aim suggests that participation in

PEERS might normalize or reverse a trajectory of excessive gamma band activity typically seen in individuals with ASD across childhood and adulthood.

Researchers have conceptualized excessive gamma oscillations as an imbalance between excitatory and inhibitory neurotransmitters (Wang et al., 2013). Rojas and Wilson (2014) explain that pyramidal glutamatergic (excitatory) neurons input to GABAergic (inhibitory) interneurons, which leads to a recurrent inhibition of glutamate. In turn, this inhibition allows for synchronized pyramidal neuronal output, which creates gamma band oscillations (Rojas & Wilson, 2014). Researchers have theorized that deficits in GABAergic systems (i.e., reduced GABA) correspond to the neural abnormalities noted in ASD (Wang et al., 2013). Specifically, reduced GABA concentration affects the inhibition of glutamate, which may lead to over excitation of neurons. This increased neural excitability has implications for one's ability to appropriately process information, and in ASD this mechanism likely contributes to difficulties in processing social input. Specifically, deficiencies in processing elements of a social interaction likely elicit awkward and/or inappropriate social responses.

Fatemi and colleagues (2009) conducted a postmortem study of persons with ASD and age-matched controls to examine the GABAergic system. The authors found that individuals with autism exhibited a significant reduction in GABA in the cerebellum, superior frontal cortex, and parietal regions (Fatemi et al., 2009). This evidence of lower GABA concentration across multiple brain regions, combined with GABA's role in eliciting gamma oscillations, suggests that elevated gamma band activity may relate to poor neural control. Results from the current study indicated that gamma band activity decreased over time, and importantly, maintained six months following treatment. This

neural change to intervention suggests that adolescents who receive PEERS experience greater neural regulation following treatment that persists beyond the program's completion.

As described earlier, behavioral and environmental change alone may not adequately explain maintenance of treatment effects. The clinical manifestation of ASD consists primarily of social skill challenges; however, if an intervention like PEERS can remediate the social isolation and difficulties that accompany ASD, then perhaps the pathogenesis of ASD can be altered (Cramer et al., 2011). By transforming adolescents' environment via involvement in a new extracurricular activity centered on their interests, the teens with ASD have an opportunity to practice their newly acquired social skills with a group of potential friends in order to develop a relationship. This study provides evidence for modified pathogenesis in adolescents with ASD in that they demonstrated a significant decrement in gamma activity at post-treatment and long-term follow-up.

In the present study, adolescents' continual use and practice of PEERS skills changed neural activity in the right temporal region, which participates in "perceiving socially relevant stimuli" (Adolphs, 2001, p. 231). The temporal lobe then projects to various structures of the brain directly implicated in social cognition: amygdala (processes the relevance and value of socioemotional stimuli), fusiform gyrus (processes facial expressions), and right somatosensory cortices (processes the mental states of others; Adolphs, 2001). Aberrant, dysfunctional gamma oscillations may contribute to the lack of coordination between the temporal lobes' connection to deeper brain structures. The present study found a maintained decrease in gamma activity unique to the right temporal region following PEERS, which significantly related to improved caregiver-

reported social cognition and fewer problem behaviors. Although this study did not examine EEG coherence (i.e., how well different brain regions communicate with one another), perhaps better neural control in this region translates into greater neural efficiency of the right temporal lobe to communicate with other regions (e.g., right somatosensory cortex) or subcortical structures (e.g., amygdala) involved in social cognition.

Additionally, the decreased EEG spectral power in the right temporal region within the gamma band may explain Van Hecke and colleagues' (2013) report of EEG asymmetry in the gamma band for the experimental group following PEERS. The authors examined neural asymmetry by examining the entire right and left hemispheres, respectively. While the authors observed a significant shift to left hemisphere dominance in gamma for adolescents who completed PEERS, the authors were unable to report on whether the finding was a global, lateralized effect, or if it was generated by a particular region (e.g., frontal, temporal, or parietal lobes). The shift seen in leftward EEG asymmetry at post-treatment for the experimental group in the Van Hecke et al. (2013) study may have been facilitated by a decrease in right temporal gamma activity. However, given that the current study did not see any asymmetry effects over time, it makes it difficult to attribute the asymmetry shift noted by Van Hecke and colleagues (2013) to a decrease in right temporal gamma activity observed in the present investigation.

The second aim examined if there was a maintenance effect over time of EEG asymmetry following PEERS. This hypothesis was not supported, as adolescents who completed PEERS did not demonstrate any significant shifts in asymmetry over time. This finding was initially surprising given the results from the study by Van Hecke and

colleagues (2013); however, there are key differences that may explain the lack of continuity between the two studies. First, the present study utilized a different MATLAB script for manually inspecting components, which allowed for a more conservative approach to rejecting artifacts (e.g., head, neck, and shoulder movement). Secondly, the present study's sample and that of Van Hecke and colleagues' (2013) did not include the same participants. Inclusion in the present study hinged upon having complete EEG and behavioral data at all three time points, and thus, the difference in sample composition may have impacted the present study's ability to note the same changes in asymmetry. While a mixed, repeated-measures ANOVA was computed in both studies to examine a time by band asymmetry interaction, the current study examined a third time point with a smaller sample, which in turn affected power of the omnibus repeated-measures ANOVA. Thus, lack of power due to the smaller sample of the present study may have impacted the ability to see any statistically significant changes in asymmetry. Encouragingly, examination of estimated marginal means, while not statistically significant, does show a pattern of increased leftward asymmetry over time across all of the bands. While this observation cannot be interpreted due to the lack of statistical significance, it suggests that with a larger sample size, perhaps significant asymmetry findings across bands could emerge.

The third aim of the present study compared neural activity in adolescents with ASD who completed PEERS to a group of same-age typically developing counterparts. In comparing right temporal gamma band activity, adolescents with ASD did not significantly differ from the neurotypical adolescents at pre-treatment or at 6-month follow-up. While it is encouraging that the adolescents who completed PEERS did not

significantly differ from their typically developing counterparts at the maintenance time point, it is important to note that gamma band activity in the right temporal region did not significantly differ at baseline either. Similarly, adolescents with ASD and the typically developing adolescents did not significantly differ in terms of EEG asymmetry over time. Even though this hypothesis was not supported, it is reassuring that, six months following PEERS, activity in this region within the gamma band remained similar to that of the typically developing adolescents, rather than showing a markedly different pattern at that time point. While the present study's sample is relatively large in comparison to most EEG studies examining individuals with ASD, having a greater number of participants would increase statistical power, and thus the potential for significant differences in spectral power and asymmetry between the two groups to emerge. Unfortunately, since the neurotypical adolescents were only assessed at one time point, it is impossible to compare any potential effects of maturation or development. This will be described in greater detail when discussing limitations of the present study.

In examining the literature, there are several EEG studies that report similar null findings when comparing individuals with ASD to typically developing peers. Catarino and colleagues (2011) examined group differences in EEG spectral power in a group of adults with ASD to typically developing adults. None of the frequency bands revealed significant differences between the adults with ASD and typically developing participants. In a different study by Chan et al. (2007), the authors sought to establish EEG profiles for a large sample (n = 66) of children with ASD, as compared to neurotypical children. Examining spectral power, the two groups did not significantly differ in their theta, alpha, or beta activation (Chan et al., 2007). Similarly, a recent study by Clarke and colleagues

(2016), examining 20 male children with Asperger's syndrome compared to 20 age-matched typically developing peers, indicated that the two groups did not significantly differ in spectral alpha or beta power, nor in total power (i.e., activation across all bands; Clarke et al., 2016). Coben et al. (2008) reported on EEG power and coherence in a group of children with ASD, as compared to a neurotypical control group. Significant differences did not emerge between the two groups for neural activity in the theta, alpha, or beta bands or total power. The aforementioned findings mirror the lack of activation differences between the ASD and typically developing groups in the present study. One explanation for the lack of spectral power differences may be a result of local hyperconnectivity noted in ASD, which may explain higher baseline gamma activity in the right temporal region that normalized over time. Additionally, long-range hypoconnectivity (i.e., poor communication between brain regions) cannot be examined in spectral power. Spectral power may not highlight complexities in neural activity. Alternatively, EEG coherence may reveal group differences in neural activity in ways that spectral power was not sensitive to in the present study.

The fourth and fifth aims explored if behavioral changes following treatment maintained six months later and if symptom improvement related to neural change. Hypotheses for Aim IV were supported, as evidenced by global improvement and maintenance of these effects at long-term follow-up in multiple domains of social functioning. Specifically, adolescents continued to host and attend get-togethers, demonstrate understanding of concepts taught in PEERS, exhibit better social skills and fewer problem behaviors, and presented with fewer core symptoms related to ASD. These behavioral changes are in accordance with the developers' of PEERS long-term

follow-up data (Mandelberg et al., 2014) and provide a level of independent replication of this work.

The hypothesis for Aim V also was supported, as caregivers reported improved social cognition and fewer problem behaviors, which corresponded to a decrease in right temporal gamma band activity at 6-month follow-up. This finding is of extremely significant scientific importance. Few studies have examined efficacious social skill interventions for adolescents, and even fewer have incorporated assessment of whether treatment gains maintained following termination of intervention. Findings from the present study not only evidenced maintenance of treatment effects behaviorally but also demonstrated a maintained neuroplasticity effect, and these two findings significantly related to one another. To this author's knowledge, no known published study has examined PEERS for adolescents in the context of treatment maintenance biomarkers and behavioral relationships.

One study by Maxwell et al. (2013) investigated the relationship between EEG spectral power and behavioral measures in a sample of adolescents with ASD and compared them to age-matched typically developing controls. Specifically, the authors examined resting state spectral power in the gamma band. The authors observed significantly lower gamma activity in the right temporal region in adolescents with ASD, as compared to neurotypical teens, and this level of gamma activity related to worse ASD symptoms, as rated by caregivers on the SRS (Maxwell et al., 2013). Maxwell et al. (2013) interpreted this correlation between decremented gamma activity and SRS ratings as a possible biomarker for ASD. While these authors' claims might appear contradictory

to the current investigation's findings, there are several key differences between the two studies that may have contributed to the opposing interpretations.

To begin, the present study assessed adolescents' with ASD *response to intervention* in the context of neural and behavioral findings across multiple time points. While the adolescents with ASD in Maxwell's (2013) sample might have significantly differed from neurotypical teens, it is difficult to apply their interpretation to the present study given that this investigation focused on maintenance of neural change as a result of a social skills treatment. It is possible that had the sample in Maxwell's (2013) study received intervention, participants may have continued to exhibit reduced gamma and improved social functioning as rated by caregivers on the SRS, given the results from the current investigation.

Another major point to address is a methodological difference in acquisition of EEG data. The present study's electrical activity assessed via EEG was amplified and sampled at 1,000 Hz, whereas Maxwell et al. (2013) sampled at a rate of 250 Hz. Sampling rate is an important consideration in EEG data collection because the rate at which data is sampled determines the highest frequency signal that can be recorded reliability without aliasing (i.e., corrupting) the data (Luck, 2005). In EEG data acquisition, the highest possible frequency that current technology can capably record is approximately 100-125 Hz. Thus, sampling at a rate of 250 Hz is doubling the highest frequency component that is capable of being recorded at the scalp (Luck, 2005). In other words, sampling at a much higher frequency, as seen in the present sample, provides a layer of precaution that data was reliably recorded, especially data in the higher frequency ranges, such as gamma. The difference in the sampling rate between the

present study and Maxwell et al.'s (2013) work is an important consideration when comparing results between each study, as it is possible that gamma results from the present study were recorded more reliably.

While the researchers in Maxwell et al. (2013) also note decremented gamma oscillations in the right lateral region, it is worth mentioning that these analyses did not survive Bonferroni correction when accounting for multiple comparisons. Nevertheless, lower gamma activity was associated with poorer functional outcomes on the SRS (Maxwell et al. 2013). However, examining this one time point in isolation makes it difficult to determine if the level of gamma activity observed in the Maxwell et al. (2013) study would change as a result of intervention. Overall, differences between Maxwell and colleagues' (2013) and the present study reflect the lack of agreement on the EEG features of ASD (Stroganova et al., 2007). Moreover, the inconsistencies highlight the importance of furthering research in this area in order to fully understand EEG profiles in individuals with ASD across development, as well as potential biomarkers for response to intervention.

It is encouraging that multiple studies across developmental periods have observed elevated gamma oscillations, which aligns with results of the present study in that gamma activation decreased over time in response to a social skills intervention, which significantly related to fewer problem behaviors and improved social cognition. The latter relationship is particularly exciting given the right temporal lobe's involvement in social cognition. In other words, better neural regulation in a region of the brain that processes social information explains improvements in caregiver-reported functioning in

this domain, as adolescents are able to appropriately and effectively implement social skills taught in PEERS.

A. Limitations of the Present Study

Although the current study revealed exciting information about neural change and maintenance in response to PEERS, it is not without its limitations. Although the sample for the ASD group was relatively large (n = 31), especially in comparison to other EEG studies in the literature (n = ~15-20), the current sample size may still place limitations on power, and thus the ability for differences in neural asymmetry and group differences in neural activity to emerge.

Furthermore, the typically developing adolescents only completed the EEG assessment at one time point, which limits the present study's ability to understand and draw conclusions about the developmental time course of gamma band activity. It seems important for future work to include a waitlist control group of adolescents who have not completed PEERS to understand the oscillatory patterns over time in the absence of intervention. Inclusion of a waitlist control group and assessing the neurotypical teens at repeated time points would allow for an examination of potential maturation effects. Even if developmental processes are at play, it is important to understand if PEERS accelerates developmental change in adolescents completing the program.

Another important limitation to address is the fact that many adolescents in the ASD group were on medication at the time of their EEG assessments. Given that the typically developing group was entirely un-medicated, it is possible that medications taken in the ASD group washed out the ability to see any meaningful differences between the groups. It is not surprising that the majority of adolescents completing PEERS were

receiving medication for mood or attentional concerns, given the high rates of Attention-Deficit/Hyperactivity Disorder (ADHD; Gadow, DeVincent, & Pomeroy, 2006) and depression (Stewart et al., 2006) within the ASD population. It was not possible in the present study to exclude adolescents based on medication status for ethical reasons, as well as to preserve power; however, it seems important for future work to examine response to interventions like PEERS with more controlled samples.

Lastly, the present sample included mostly Caucasian males from relatively higher earning households, which makes the findings less generalizable to more diverse samples. In future studies, a larger, more diverse (i.e., gender, ethnicity, and socioeconomic status) sample should be included.

B. Future Directions and Conclusions

Despite these limitations, the current study's findings contain substantial scientific value. Adolescents with ASD demonstrated a decrease in gamma activity in the right temporal region following PEERS, which was maintained at 6-month follow-up. Perhaps the most exciting finding from this study was the relationship between functional outcomes—fewer problem behaviors and greater social cognition—and neural change, which highlights the role of neural plasticity as a mechanism for maintenance of improved behavioral presentation following intervention. While these findings require replication, they represent a promising biomarker for neural response to treatment and maintenance of gains. As mentioned earlier, additional work is warranted to expand the field's understanding of neural activity in ASD and elucidate the nature of neural oscillation patterns that underlie the disorder. While EEG is a viable option for adolescents on the spectrum given its flexibility and non-invasive properties, it does not

provide the same spatial resolution as other neuroimaging techniques, such as MRI or DTI. Thus, future work would benefit from examining the specific neural structures, such as white matter, underlying cortical functioning. Examination of deeper, subcortical structures would provide clarity about the specific neural assemblies and networks that respond to intervention. While there are exciting avenues of future research, the current study adds to the minimal literature examining not only neural response to intervention, but also the maintenance of these effects and their behavioral correlates.

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TABLES AND FIGURES

Table 1

PEERS Sessions with Descriptions

Session	Didactic lesson	Description of the lesson
1	Introduction & Conversational Skills I: Trading Information	Trading information during conversations with peers in order to find common interests
2	Conversational Skills II: Two-way Conversations	Having two-way conversations with peers. Parents identify teen activities leading to potential friendships
3	Conversational Skills III: Electronic Communication	Appropriate use of voicemail, email, text messaging, instant messaging, and the Internet in developing pre-existing friendships. Parents taught the social structure of school peer groups
4	Choosing Appropriate Friends	Pursuing teen extra-curricular activities leading to friendships. Teens taught the social structure of school peer groups and identify groups they might fit in with
5	Appropriate Use of Humor	Appropriate use of humor in same-age peer interactions. Parents taught strategies to provide feedback to their teen about their use of humor
6	Peer Entry I: Entering a Conversation	Steps involved in joining conversations with peers
7	Peer Entry II: Exiting a Conversation	How to assess receptiveness during peer entry and how to

		gracefully exit conversations when not accepted
8	Get-togethers	Planning and having successful get-togethers with friends. Appropriate parent monitoring and intervention during teen get-togethers
9	Good Sportsmanship	The rules of good sportsmanship during games and sports
10	Rejection I: Teasing and Embarrassing Feedback	Appropriate responses to teasing. Differentiating between teasing and negative feedback and using appropriate responses to the latter
11	Rejection II: Bullying & Bad Reputations	Strategies for handling bullying and changing a bad reputation
12	Handling Disagreements	Resolving disagreements with peers
13	Rumors & Gossip	Strategies for handling rumors and gossip
14	Graduation & Termination	Graduation party and ceremony. Maintaining gains in teen friendships after termination

Table 2

Sample Characteristics

	EXP (*n* = 31) *M* (*SD*)	TYP (*n* = 32) *M* (*SD*)
Age (years)	13.61 (1.38)	13.12 (1.41)
KBIT-2		
FSIQ (standard score)	104.71 (18.02)	107.94 (13.55)
VIQ (standard score)	103.29 (17.61)	109.28 (11.14)
NVIQ (standard score)	101.0 (24.9)	104.16 (15.29)
ADOS total score	10.97 (2.82)	Not administered
Communication score	3.61 (1.1)	--
Social score	7.35 (2.14)	--
Mother's age (years)	46.29 (5.98)	44.72 (4.03)
Father's age (year)	47.74 (6.07)	46.97 (4.25)
Gender (percentage)		
Male	87.1	93.8
Female	12.9	6.2
Handedness (percentage)		
Right	87.1	90.6
Left	12.9	9.4
Race (percentage)		
Asian	6.7	0
African-American	3.3	0
Biracial	0	3.1
Caucasian	83.3	96.9
Hawaiian/Pacific Islander	3.3	0
Unreported	3.4	0
Ethnicity (percentage)		
Hispanic	9.7	6.3
Not Hispanic	87.1	87.5

Unreported	3.2	6.2
Parent education (percentage)		
High school	9.7	3.1
Vocational/tech	6.5	0
Some college	16.1	18.8
Junior college	3.2	0
B.A./B.S.	45.2	34.4
M.A./M.S.	12.9	37.5
Ph.D./M.D./J.D.	6.5	6.3
Unreported	0	0
Household income (percentage)		
Under 50 k	25.7	9.3
50-75 k	19.4	18.8
75-100 k	19.4	15.6
100 k plus	35.5	56.3
Medication status (percentage)		
No medication	38.7	100
Taking medication	61.3	0

Note. EXP = experimental group; TYP = typically developing group; KBIT-2 = Kaufman Brief Intelligence Test, Second Edition; FSIQ = full scale IQ; VIQ = verbal IQ; NVIQ = nonverbal IQ; ADOS = Autism Diagnostic Observation Schedule, Generic.

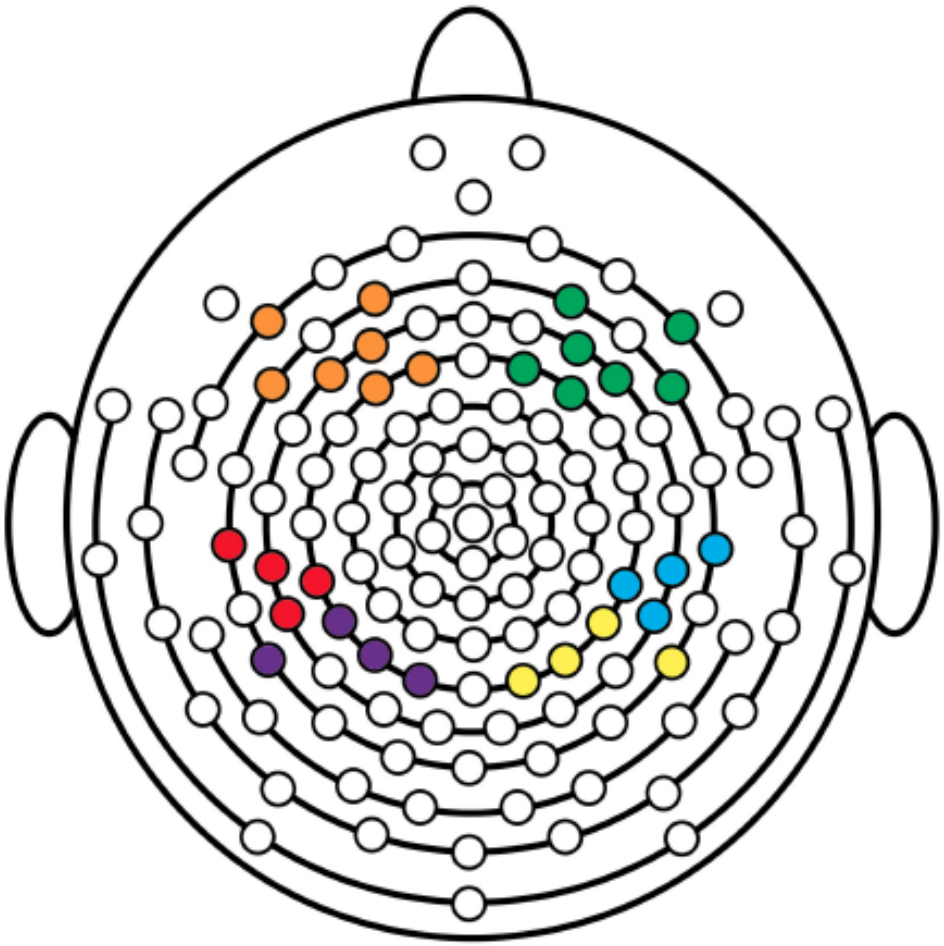

Figure 1. 64-Channel Geodesic Electrode Net. Colored electrode regions represent scalp topography assessed in analyses for spectral power. Orange = Left Frontal; Red = Left Temporal; Purple = Left Parietal; Green = Right Frontal; Blue = Right Temporal; Yellow = Right Parietal.

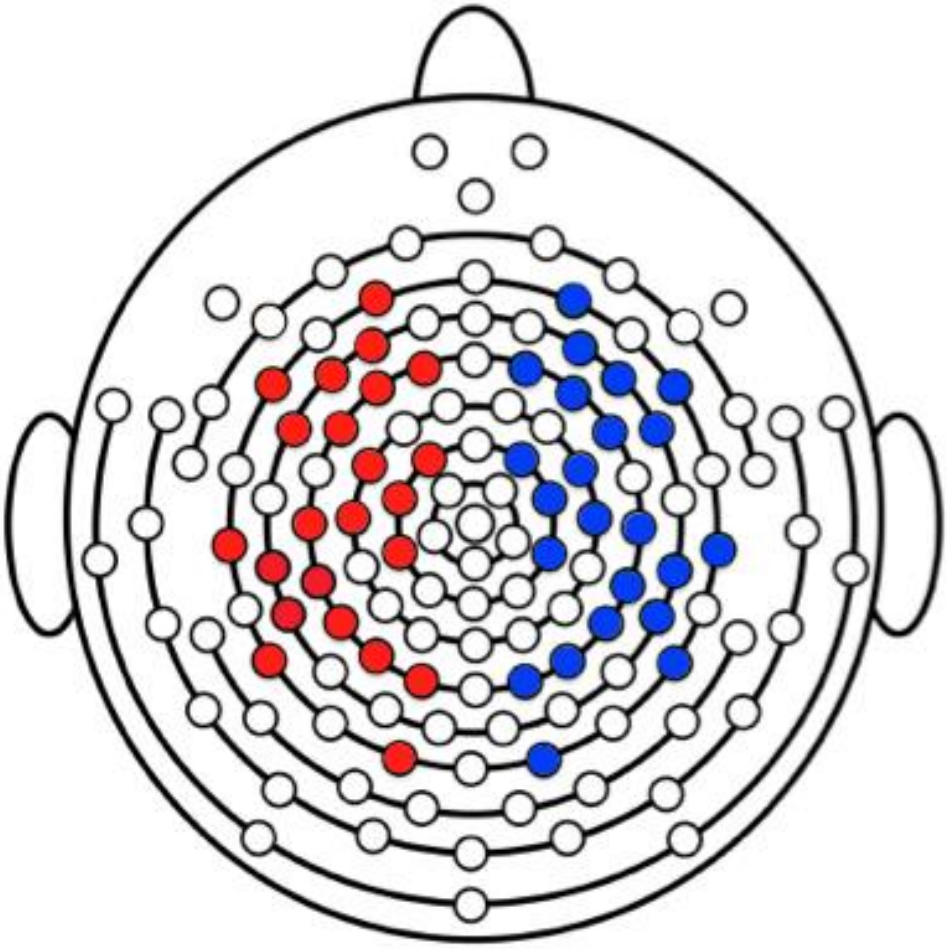

Figure 2. 64-Channel Geodesic Electrode Net. Colored electrode regions represent scalp topography assessed in analyses for asymmetry. Red = Left Hemisphere; Blue = Right Hemisphere.

APPENDIX A

Violations of Sphericity

Table A1

Violations of Sphericity for Aim I: Omnibus Three-Way Repeated-Measures ANOVA

Within-Subjects Effect	df	Mauchley's *W*	ε
TIME	2	.533***	.703
BAND	9	.154***	.643
LOCATION	14	.060***	.513
TIME x BAND	35	.005***	.590
TIME x LOCATION	54	.018***	.693
BAND x LOCATION	209	.001***	.257
TIME x BAND x LOCATION	819	.001***	.274

*$p < .05$, **$p < .01$, ***$p < .001$

Note. Huynh-Feldt corrected values are cited for all Aim I statistics for cases in which sphericity was violated. Values for variables not violating assumptions of sphericity not listed.

Table A2

Violations of Sphericity for Aim I: Simple Effects Test for BAND x LOCATION Interaction, File Split by BAND, Assessing LOCATION

Within-Subjects Effect	df	Mauchley's *W*	ε
Delta			
LOCATION	14	.019***	.466
Theta			
LOCATION	14	.038***	.450
Alpha			
LOCATION	14	.068***	.602
Beta			
LOCATION	14	.149***	.747
Gamma			
LOCATION	14	.047***	.504

*$p < .05$, **$p < .01$, ***$p < .001$

Note. Huynh-Feldt corrected values are cited for all Aim I statistics for cases in which sphericity was violated. Values for variables not violating assumptions of sphericity not listed.

Table A3

Violations of Sphericity for Aim I: Simple Interaction Test, File Split by LOCATION, Assessing TIME x BAND

Within-Subjects Effect	df	Mauchley's *W*	ε
Left Frontal			
BAND	2	.126***	.618
TIME x BAND	35	.002***	.517
Left Temporal			
TIME	2	.521***	.697
BAND	9	.156***	.696
TIME x BAND	35	.011***	.629
Left Parietal			
TIME	2	.578***	.727
BAND	9	.138***	.617
TIME x BAND	35	.009***	.592
Right Frontal			
BAND	9	.919***	.983
TIME x BAND	35	.002***	.495
Right Temporal			
TIME	2	.653***	.772
BAND	9	.098***	.621
TIME x BAND	35	.011***	.606
Right Parietal			
TIME	2	.663***	.778
BAND	9	.145***	.651
TIME x BAND	35	.007***	.520

*$p < .05$, **$p < .01$, ***$p < .001$

Note. Huynh-Feldt corrected values are cited for all Aim I statistics for cases in which sphericity was violated. Values for variables not violating assumptions of sphericity not listed.

Table A4

Violations of Sphericity for Aim I: Simple Effects Test, File Split by BAND, Assessing Main Effect of TIME in Right Temporal Region

Within-Subjects Effect	df	Mauchley's *W*	ε
Theta			
TIME	2	.638***	.763
Alpha			
TIME	2	.695***	.799
Gamma			
TIME	2	.688***	.795

*$p < .05$, **$p < .01$, ***$p < .001$

Note. Huynh-Feldt corrected values are cited for all Aim I statistics for cases in which sphericity was violated. Values for variables not violating assumptions of sphericity not listed.

Table A5

Violations of Sphericity for Aim II: Omnibus Two-Way Repeated-Measures ANOVA

Within-Subjects Effect	df	Mauchley's *W*	ε
BAND	9	.060***	.457
TIME x BAND	35	.001***	.312

*$p < .05$, **$p < .01$, ***$p < .001$

Note. Huynh-Feldt corrected values are cited for all Aim II statistics for cases in which sphericity was violated. Values for variables not violating assumptions of sphericity not listed.

Table A6

Violations of Sphericity for Aim IV: Behavioral Measures

Behavioral Measure	df	Mauchley's *W*	ε
SRS – Social Cognition	2	.739*	.841
SSIS – Problem Behaviors	2	.771*	.865

*$p < .05$, **$p < .01$, ***$p < .001$

Note. Huynh-Feldt corrected values are cited for all Aim IV statistics for cases in which sphericity was violated. Values for variables not violating assumptions of sphericity not listed.

APPENDIX B

Means and Pairwise Comparisons for Significant Main Effects and Interactions

Table B1

Means and Standard Error for the Omnibus Main Effect of TIME (Aim I)

Time	*M* (*SE*)
Pre	1.08 (.12)
Post	1.01 (.09)
6-month follow-up	.84 (.11)

Table B2

Pairwise Comparisons for the Omnibus Main Effect of TIME (Aim I), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	.065	--	--
6-month follow-up	.236	.171*	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B3

Means and Standard Error for the Omnibus Main Effect of BAND (Aim I)

Band	*M* (*SE*)
Delta	1.30 (.10)
Theta	1.05 (.11)
Alpha	1.15 (.15)
Beta	1.27 (.09)
Gamma	.13 (.11)

Table B4

Pairwise Comparisons for Omnibus Main Effect of BAND (Aim I), Mean Differences

Band	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--
Theta	.251***	--	--	--	--
Alpha	.149	-.102	--	--	--
Beta	.032	-.219	-.117	--	--
Gamma	1.165***	.914***	1.016***	1.133***	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B5

Means and Standard Error for the Omnibus Main Effect of LOCATION (Aim I)

Time	*M* (*SE*)
Left Frontal	1.01 (.09)
Left Temporal	.87 (.10)
Left Parietal	1.01 (.11)
Right Frontal	1.12 (.09)
Right Temporal	.85 (.10)
Right Parietal	1.01 (.11)

Table B6

Pairwise Comparisons for Omnibus Main Effect of LOCATION (Aim I), Mean Differences

Location	LF	LT	LP	RF	RT	RP
LF	--	--	--	--	--	--
LT	.142*	--	--	--	--	--
LP	-.004	-.145**	--	--	--	--
RF	-.114*	-.256***	-.110	--	--	--
RT	.160*	.018	-.163**	.274***	--	--
RP	-.005	-.147*	-.002	.109	.165*	--

*$p < .05$, **$p < .01$, ***$p < .001$

Note. LF = left frontal; LT = left temporal; LP = left parietal; RF = right frontal; RT = right temporal; RP = right parietal.

Table B7

Means and Standard Error for Simple Effects Test to Examine Omnibus LOCATION x BAND, Bands that Demonstrated a Main Effect for LOCATION (Aim I)

Location	*M* (*SE*)
Delta	
LF	1.60 (.11)
LT	1.04 (.10)
LP	1.23 (.10)
RF	1.66 (.11)
RT	1.01 (.10)
RP	.06 (.12)
Theta	
LF	1.16 (.11)
LT	.88 (.12)
LP	1.08 (.13)
RF	1.25 (.10)
RT	.85 (.12)
RP	1.06 (.14)
Alpha	
LF	.91 (.14)
LT	1.06 (.15)
LP	1.41 (.17)
RF	1.00 (.14)
RT	1.08 (.16)
RP	1.44 (.18)
Beta	
LF	1.18 (.09)
LT	1.24 (.10)
LP	1.31 (.11)
RF	1.38 (.09)
RT	1.21 (.09)
RP	1.28 (.10)

Note. LF = left frontal; LT = left temporal; LP = left parietal; RF = right frontal; RT = right temporal; RP = right parietal.

Table B8

Pairwise Comparisons for Simple Effects Test to Examine Omnibus BAND x LOCATION, Bands that Demonstrated a Main Effect for LOCATION (Aim I), Mean Differences

Delta	LF	LT	LP	RF	RT	RP
LF	--	--	--	--	--	--
LT	.560***	--	--	--	--	--
LP	.370***	-.191***	--	--	--	--
RF	-.059	-.619***	-.428***	--	--	--
RT	.589***	.029	.219***	.648***	--	--
RP	1.542***	.982***	1.173***	1.601***	.953***	--
Theta	LF	LT	LP	RF	RT	RP
LF	--	--	--	--	--	--
LT	.282***	--	--	--	--	--
LP	.079	-.203	--	--	--	--
RF	-.089	-.371	-.168	--	--	--
RT	.309***	.027	.230***	.398***	--	--
RP	.105	-.177	.026	.194	-.204***	--

Alpha	LF	LT	LP	RF	RT	RP
LF	--	--	--	--	--	--
LT	-.147	--	--	--	--	--
LP	-.493***	-.347***	--	--	--	--
RF	-.082	.065	.412***	--	--	--
RT	-.162	-.015	.332***	-.080	--	--
RP	-.525***	-.378***	-.032	-.443***	-.363***	--
Beta	LF	LT	LP	RF	RT	RP
LF	--	--	--	--	--	--
LT	-.056	--	--	--	--	--
LP	-.124	-.068	--	--	--	--
RF	-.199***	-.143	-.075	--	--	--
RT	-.029	.028	.095	.170**	--	--
RP	-.100	-.043	.025	.100	-.071	--

*$p < .05$, **$p < .01$, ***$p < .001$

Note. LF = left frontal; LT = left temporal; LP = left parietal; RF = right frontal; RT = right temporal; RP = right parietal.

Table B9

Means and Standard Error for Simple Interaction Effects Test: TIME x BAND, Locations that Demonstrated a Main Effect for BAND (Aim I)

Location	*M* (*SE*)
Left Frontal	
Delta	1.60 (.11)
Theta	1.16 (.11)
Alpha	.91 (.14)
Beta	1.81 (.09)
Gamma	.18 (.12)
Left Temporal	
Delta	1.04 (.10)
Theta	.88 (.12)
Alpha	1.06 (.15)
Beta	1.24 (.10)
Gamma	.11 (.12)
Left Parietal	
Delta	1.23 (.10)
Theta	1.08 (.13)
Alpha	1.41 (.17)
Beta	1.31 (.11)
Gamma	.03 (.12)
Right Frontal	
Delta	1.66 (.11)
Theta	1.25 (.10)
Alpha	.99 (.14)
Beta	1.38 (.09)
Gamma	.32 (.13)
Right Temporal	
Delta	1.01 (.10)
Theta	.85 (.12)
Alpha	1.08 (.16)
Beta	1.21 (.09)
Gamma	.09 (.11)
Right Parietal	
Delta	1.23 (.10)
Theta	1.06 (.14)
Alpha	1.44 (.18)
Beta	1.28 (.10)
Gamma	.06 (.12)

Table B10
Pairwise Comparisons for Simple Interaction Effects Test: TIME x BAND, Locations that Demonstrated a Main Effect for BAND (Aim I), Mean Differences

LF	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--
Theta	.442***	--	--	--	--
Alpha	.689**	.248	--	--	--
Beta	.422*	-.020	-.268	--	--
Gamma	1.423***	.981***	.733***	1.001***	--
LT	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--
Theta	.164*	--	--	--	--
Alpha	-.018	-.182	--	--	--
Beta	-.195	-.359**	-.177	--	--
Gamma	.931***	.767***	.949***	1.126***	--
LP	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--

Theta	.151	--	--	--	--
Alpha	-.174	-.325	--	--	--
Beta	-.072	-.223	.102	--	--
Gamma	1.203***	1.052***	1.377***	1.275***	--
RF	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--
Theta	.412***	--	--	--	--
Alpha	.667**	.255	--	--	--
Beta	.281	-.130	-.385**	--	--
Gamma	1.340***	.928***	.673***	1.058***	--
RT	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--
Theta	.162*	--	--	--	--
Alpha	-.061	-.223	--	--	--
Beta	-.196	-.358**	-.135	--	--
Gamma	.923***	.762***	.985***	1.119***	--

RP	Delta	Theta	Alpha	Beta	Gamma
Delta	--	--	--	--	--
Theta	.173	--	--	--	--
Alpha	-.209	-.382*	--	--	--
Beta	-.051	-.225	.158	--	--
Gamma	1.169***	.996***	1.378***	1.220***	--

*$p < .05$, **$p < .01$, ***$p < .001$

Note. LF = left frontal; LT = left temporal; LP = left parietal; RF = right frontal; RT = right temporal; RP = right parietal.

Table B11

Means and Standard Error for Simple Interaction Effects Test: TIME x BAND, Examining Significant Main Effect of TIME within the Right Temporal Region and Gamma Band (Aim I)

Time	*M* (*SE)*
Pre	.33 (.14)
Post	.001 (.10)
6-month follow-up	-.06 (.14)

Table B12

Pairwise Comparisons for Simple Interaction Effects Test: TIME x BAND, Examining Significant Main Effect of TIME within the Right Temporal Region and Gamma Band (Aim I), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	.331*	--	--
6-month follow-up	.395*	-.064	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B13

Means and Standard Deviations for BAND ASYMMETRY at Each Time Point (Aim II)

Band Asymmetry Value	Pre *M* (*SD)*	Post *M* (*SD)*	6-month follow-up *M* (*SD)*
Delta	.004(.17)	-.02(.16)	.001(.16)
Theta	.01(.17)	-.05(.17)	-.01(.16)
Alpha	.0001(.18)	-.06(.17)	-.05(.18)
Beta	-.04(.17)	-.07(.18)	-.06(.25)
Gamma	-.08(.27)	-.07(.34)	-.08(.39)

*$p < .05$, **$p < .01$, ***$p < .001$

Table B14

Independent Sample T-Test, Comparing Right Temporal Gamma Power between ASD and Typically Developing Groups at Baseline (Aim III)

Variable	EXP (n = 31) *M* (*SD)*	TYP (n = 32) *M* (*SD)*	*t*	*p* value
Right Temporal Gamma Power	.33(.77)	.27(.63)	.33	.74

*$p < .05$, **$p < .01$, ***$p < .001$

Note. EXP = experimental group. TYP = typically developing group.

Table B15

Independent Sample T-Test, Comparing Right Temporal Gamma Power between ASD Group at 6-Month Follow-Up and Typically Developing Group at Baseline (Aim III)

Variable	EXP (n = 31) *M* (*SD*)	TYP (n = 32) *M* (*SD*)	*t*	*p* value
Right Temporal Gamma Power	-.06(.79)	.27(.63)	-1.86	.07

*$p < .05$, **$p < .01$, ***$p < .001$

Note. EXP = experimental group. TYP = typically developing group.

Table B16

Independent Sample T-Tests, Comparing Band Asymmetry between ASD and Typically Developing Groups at Baseline (Aim III)

Band Asymmetry	EXP (n = 31) *M* (*SD)*	TYP (n = 32) *M* (*SD)*	*t*	*p* value
Delta	.004(.17)	-.04(.17)	1.11	.27
Theta	.01(.17)	-.03 (.15)	.98	.33
Alpha	.0001(.18)	-.08(.17)	1.90	.06
Beta	-.04(.17)	-.07(.17)	.69	.49
Gamma	-.08(.27)	-.12(.40)	.37	.71

*$p < .05$, **$p < .01$, ***$p < .001$

Note. EXP = experimental group. TYP = typically developing group.

Table B17

Independent Sample T-Tests, Comparing Band Asymmetry between ASD Group at 6-Month Follow-Up and Typically Developing Group at Baseline (Aim III)

Band Asymmetry	EXP (n = 31) *M* (*SD)*	TYP (n = 32) *M* (*SD)*	*t*	*p* value
Delta	.001(.16)	-.04(.17)	1.07	.29
Theta	-.01(.16)	-.03 (.15)	.40	.69
Alpha	-.05(.18)	-.08(.17)	.84	.40
Beta	-.06(.25)	-.07(.17)	.09	.93
Gamma	-.08(.39)	-.12(.40)	.41	.69

*p < .05, **p < .01, ***p < .001

Note. EXP = experimental group. TYP = typically developing group.

Table B18

Means and Standard Deviations for QSQ (Aim IV)

Time	*M* (*SD)*
Pre	.88 (1.42)
Post	3.80 (2.58)
6-month follow-up	2.40 (2.63)

Table B19

Pairwise Comparisons QSQ (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	-2.92***	--	--
6-month follow-up	-1.52*	1.40	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B20

Means and Standard Deviations for SRS – Social Awareness (Aim IV)

Time	*M* (*SD*)
Pre	69.92 (10.50)
Post	63.72 (12.37)
6-month follow-up	64.32 (10.37)

Table B21

Pairwise Comparisons SRS – Social Awareness (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	6.20*	--	--
6-month follow-up	5.60*	-.60	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B22

Means and Standard Deviations for SRS – Social Cognition (Aim IV)

Time	*M* (*SD)*
Pre	79.76 (8.02)
Post	68.16 (11.11)
6-month follow-up	68.64 (12.84)

Table B23

Pairwise Comparisons SRS – Social Cognition (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	11.60***	--	--
6-month follow-up	11.12***	-.48	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B24

Means and Standard Deviations for SRS – Social Communication (Aim IV)

Time	*M* (*SD*)
Pre	80.84 (7.60)
Post	71.92 (10.19)
6-month follow-up	68.56 (12.38)

Table B25

Pairwise Comparisons SRS – Social Communication (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	8.92***	--	--
6-month follow-up	12.28***	3.36	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B26

Means and Standard Deviations for SRS – Social Motivation (Aim IV)

Time	*M* (*SD*)
Pre	78.20 (10.88)
Post	70.88 (11.55)
6-month follow-up	68.52 (12.12)

Table B27

Pairwise Comparisons SRS – Social Communication (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	7.32**	--	--
6-month follow-up	9.68**	2.36	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B28

Means and Standard Deviations for SRS – Autism Mannerisms (Aim IV)

Time	*M* (*SD*)
Pre	81.60 (9.20)
Post	74.44 (12.58)
6-month follow-up	73.04 (14.84)

Table B29

Pairwise Comparisons SRS – Autism Mannerisms (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	7.16**	--	--
6-month follow-up	8.56**	1.40	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B30

Means and Standard Deviations for SSIS – Social Skills (Aim IV)

Time	*M* (*SD)*
Pre	110.56 (9.01)
Post	117.72 (9.59)
6-month follow-up	118.76 (10.08)

Table B31

Pairwise Comparisons SSIS – Social Skills (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	-7.16**	--	--
6-month follow-up	-8.20**	-1.04	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B32

Means and Standard Deviations for SSIS – Problem Behaviors (Aim IV)

Time	*M* (*SD)*
Pre	153.7 (7.69)
Post	150.52 (10.17)
6-month follow-up	146.68 (9.61)

Table B33

Pairwise Comparisons SSIS – Problem Behaviors (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	3.16	--	--
6-month follow-up	7.00**	3.84	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B34

Means and Standard Deviations for TASSK (Aim IV)

Time	*M* (*SD)*
Pre	12.84 (2.70)
Post	22.04 (2.57)
6-month follow-up	21.80 (3.81)

Table B35

Pairwise Comparisons TASSK (Aim IV), Mean Differences

Time	Pre	Post	6-month follow-up
Pre	--	--	--
Post	-9.16***	--	--
6-month follow-up	-8.87***	.29	--

*$p < .05$, **$p < .01$, ***$p < .001$

Table B36

Correlations Examining the Relationship between Symptom Improvement and Neural Changes

	RT Gamma Activity
RT Gamma Activity	1
QSQ – Social Contacts	.199
SRS – Social Awareness	-.240
SRS – Social Cognition	-.403*
SRS – Social Communication	-.205
SRS – Social Motivation	-.282
SRS – Autism Mannerisms	-.331
SSIS – Social Skills	.180
SSIS – Problem Behaviors	-.404*
TASSK	-.009

*$p < .05$, **$p < .01$, ***$p < .001$

Note. RT = right temporal.