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The Relative Efficacy of Connectivity Guided and Symptom Based EEG Biofeedback for Autistic Disorders

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Abstract Autism is a neurodevelopmental disorder characterized by deficits in communication, social interaction, and a limited range of interests with repetitive stereotypical behavior. Various abnormalities have been documented in the brains of individuals with autism, both anatomically and functionally. The connectivity theory of autism is a recently developed theory of the neurobiological cause of autisic symptoms. Different patterns of hyper- and hypo-connectivity have been identified with the use of quantitative electroencephalogray (QEEG), which may be amenable to neurofeedback. In this study, we compared the results of two published controlled studies examining the efficacy of neurofeedback in the treatment of autism. Specifically, we examined whether a symptom based approach or an assessment/connectivity guided based approach was more effective. Although both methods demonstrated significant improvement in symptoms of autism, connectivity guided neurofeedback demonstrated greater reduction on various subscales of the Autism Treatment Evaluation Checklist (ATEC). Furthermore, when individuals were matched for severity of symptoms, the amount of change per session was significantly higher in the Coben and Padolsky (J Neurother 11:5–23, 2007) study for all five measures of the ATEC. Our findings suggest that an approach guided by QEEG based connectivity assessment may be more efficacious in the treatment of autism. This permits the targeting and amelioration of abnormal connectivity patterns in the brains of people who are autistic.

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Introduction

Autistic spectrum disorders (ASD) are a group of pervasive developmental disabilities characterized by deficits in communication, social interaction and restricted repetitive behavior. The spectrum includes Autistic Disorder, Rett's Disorder, Childhood Disintegrative Disorder, Asperger Disorder, and Pevasive Developmental Disorder Not Otherwise Specified (PDD-NOS) (Tidmarsh and Volkmar 2003). The prevalence of these disorders appears to be on the rise, with studies indicating that about 1 out of 150 children will be diagnosed with an ASD (Center for Disease Control and Prevention 2006).

Autistic Disorder is characterized by impaired social interaction, delay or total lack of spoken language and communication, as well as repetitive stereotyped behaviors, interests or activities (APA 2000; *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision; DSM-IV-TR*). Asperger's Disorder is often associated with high cognitive function, literal pedantic speech, difficulty comprehending implied meaning, problems with fluid movement, and inappropriate social interaction. PDD-NOS refers to the category of deficits in language and social skills that do not meet the criteria for other disorders. In contrast, Childhood Disintegrative Disorder and Rett's Disorder are characterized by intervals of normal early development followed by loss of previously acquired skills. Although communication and social skill deficits are common among these conditions, there remains a substantial degree of variability in terms of onset and severity of symptomatology within the Autistic Spectrum of Disorders (Attwood 1998;

Hamilton 2000; McCandless 2005; Sicile-Kira 2004; Siegel 1996).

The exact cause of autism is unknown, though many studies have noted differences in the structure and function of the brains of individuals with autism. Abnormal brain morphology in autism was first noted when Kanner (1943) observed an enlargement of the heads of children diagnosed with autism. These anecdotal findings were then corroborated by more controlled studies which showed that macrocephaly was present in approximately 20% of individuals with autism (Bailey et al. 1993; Courchesne et al. 2003; Davidovitch et al. 1996) and is supported by neuroimaging studies (Courchesne et al. 2001; Filipek et al. 1992; Piven et al. 1996) as well as findings of increased brain weight (Bailey et al. 1998; Courchesne et al. 1999). Specifically, there may be an increase in temporal, parietal, and occipital lobe volume, but not the frontal lobe (Piven et al. 1996). This pattern may be interpreted as either abnormal posterior brain enlargement, or a frontal lobe abnormality in which the frontal lobes lag behind the rest of the brain in their development. While numerous studies suggest there is an increase in total brain volume in Autism, this anomaly does not appear to be present at birth. Rather, during the first 2 years of life there is overgrowth, followed by a decrease in the normal growth process (Courchesne et al. 2001, Courchesne 2004). It has been suggested that the reason for this abnormal growth process is that there may be dysfunction in the normal pruning process (Frith 2003). In addition to the early synaptogenesis in childhood, the pruning process eliminates faulty connections, which are not strengthened through long term potentiation.

Some studies have found increased cell packing density and reduced cell size in various brain regions (Kemper and Bauman 1998), while other studies have shown a decrease in Purkinje cell density in the cerebellum of autistic cases (Kemper and Bauman 1998; Bailey et al. 1998). In an attempt to localize cerebral dysfunction in Autism, various functional neuroimaging studies have been conducted. Schmitz et al. (2006) found that individuals with ASD had significantly increased brain activity associated with the left inferior and orbital frontal gyrus (associated with motor inhibition), left insula (regulating interference inhibition), and parietal lobes (required for set shifting). Increased frontal gray matter density in areas of increased functional activation was also observed. Increased frontal metabolite levels have been associated with obsessional behavior in Asperger Syndrome (Murphy et al. 2002), and have been reported in the amygdala-hippocampal regions in ASD (Page et al. 2006). Others have found significant bilateral temporal hypoperfusion in the superior temporal gyrus and superior temporal sulcus in children with Autism (Boddaert et al. 2002).

Structural changes linked to Autism have indicated a significant reduction in total grey matter volume, particularly within fronto-striatal and parietal networks, along with increased cerebral spinal fluid (CSF) volume, and reduced white matter in the cerebellum, left internal capsule, and fornices (McAlonan et al. 2005). MRI based morphometric analysis found that overall, whole brain volume was moderately increased (Herbert et al. 2003). Factor analysis, however, showed significant heterogeneity of brain differences in autism and demonstrated the difficulty in looking for a specific brain area to be implicated in the disorder. Rather, it has been suggested that their may be abnormalities in the pathways, and that pervasive core processing deficits, impaired complex information processing, or weak central coherence in Autism may be associated with abnormal white matter.

Deficits in cross-modal information processing and corticocortical connections may be linked to behavior and communication impairment in Autism (Herbert et al. 2004). Cell minicolumn anomalies of the cerebral cortex representing connectivity linking afferent, efferent, and interneuronal connections have been reported in Autism (Casanova et al. 2002), as well as reduced white matter concentration (Chung et al. 2004), including regions of the corpus callosum (Piven et al. 1997). The corpus callosum is the most robust white matter fiber tract in the brain and connects most of the two cerebral hemispheres. Thus, it plays a major role in interhemispheric neural connectivity. Several studies now have found this pathway to be aberrant in Autism (Alexander et al. 2007; Boger-Megiddo et al. 2006; Chung et al. 2004; Courchesne et al. 1993; Vidal et al. 2006).

The aforementioned research and multiple brain regions implicated in Autism provide support for cerebral connectivity deficits in Autism. In the 1980s, Uta Frith suggested that autistic behaviors may be explained by the individual's lack of ability to integrate information due to an obsessive focus on details. She attributed this to a lack of communication between frontal brain areas which would typically integrate the information with more posterior areas (Wickelgren 2005). Since that time, much research has been conducted in support of this connectivity deficit hypothesis.

Research utilizing fMRI has reported a pattern of underconnectivity in Autism (Just et al. 2007). A decreased degree of synchronization between frontal and parietal areas of activation was noted during an executive function task, suggesting that cortical underconnectivity is associated with a deficit in the neural and cognitive integration of information. Others have found anomalies in connectivity (associated with inter-regional grey matter correlations) of limbic-striatal social brain systems in Autism (McAlonan

et al. 2005). Functional underconnectivity associated with reduced cortical activation and synchronization during a sentence comprehension task (Just et al. 2004), and even during the resting brain state has been found in Autism (Cherkassky et al. 2006).

Autism has also been classified as a disorder of arousal-modulating systems associated with atypically increased functional connectivity, in addition to areas of underconnectivity. Research utilizing fMRI bold oxygen level dependent (BOLD) signal during simple visuomotor coordination has indicated greater thalamocortical functional connectivity in Autism. Excessive connectivity was noted in the left insula, right postcentral, and middle frontal regions. Increased thalamocortical functional connectivity may be associated with excessive synaptic generation and reduced pruning which may be linked to brain enlargement in Autism (Mizuno et al. 2006).

Courchesne and Pierce (2005) described a pattern of over-connectivity (hyperconnectivity) within the frontal lobe, with long-distance disconnection (hypoconnectivity) between the frontal lobe and other brain regions associated with ASD. Reduction of long-distance cortical to cortical reciprocal activity and coupling disrupts the integration of information from emotional, language, sensory, and autonomic systems (Courchesne and Pierce 2005).

By dividing cerebral white matter with a white matter parcellation technique, Herbert et al. (2004) found that the increase in white matter was in the radiate (outer) zones of all cerebral lobes and longer myelinating regions. In contrast, inner zone white matter volumes showed no difference compared to a control group. Since deeper myelination occurs earlier on, the authors interpreted this finding as supporting a postnatal disturbance which disrupts primarily cortico-cortical connections. In a review of neuropathological findings in Autism, Herbert (2005) indicated that neuroinflammation is present in Autism and also contributes to the increased cranial volume. The overall increase in volume may result in dysfunction of the ability to integrate information between different parts of the brain. Herbert further speculated that disconnectivity may result in specific dysfunction, not just pervasive, nonspecific deficits. Therefore, domains most likely to be affected by the inflammatory response are those which require more coordination and communication between brain areas, such as language and executive functioning.

The connectivity theory of autism has become an empirically supported theory describing the neurobiological basis of Autism, with evidence suggesting that it is an overgrowth of white matter during the first 2 years of life, followed by a retardation of growth thereafter which leads to disordered connectivity (Hughes 2007). Because EEG measures electrical activity across the brain with high temporal resolution, it lends itself well to the investigation of connectivity through EEG coherence measurement.

Computerized EEG analyses have indicated that children with Autism have significantly greater coherence between hemispheres in the beta band than typically developing children. They also have been found to have higher coherence in the alpha band than normal controls, and less inter- and intrahemispheric asymmetry than either children who are developing typically or who have mental handicaps (Cantor et al. 1986).

Murias et al. (2007) assessed functional connectivity with EEG coherence during an eyes closed resting state. Relative to controls, adults with ASD showed long range alpha band coherence reductions in frontal-occipital and fronal-parietal areas. The alpha band represents more globally dominant functions, which are more dependent on corticocortical and callosal fibers (Nunez 1995; Nunez and Srinivasan 2006). Adults with ASD also showed increased coherence at temporal recording sites between 3–6 Hz, reflecting intact locally dominant cortical activity. These findings support the hypothesis of a weak connection between frontal and other areas.

Coben et al. (2008), using quantitative EEG (QEEG), found that children who were autistic showed decreased intrahemispheric coherences across short-medium as well as long inter-electrode distances within delta and theta bands. In addition, there were reduced interhemispheric coherences in the alpha band in temporal regions, and reduced interhemispheric coherences in beta in central, parietal, and occipital regions. Greater relative theta was especially prevalent in the right posterior region, while lower beta was noted across the right hemisphere, especially over the right frontal region.

At least two critical issues result from the aforementioned findings. First, through scientific investigation, we must learn how to prevent these problems from taking place. Second, we must improve the evaluation and treatment of connectivity disturbances after they occur. The EEG appears to be good candidate for the evaluation of neural connectivity in Autism, based on coherence analyses. Specifically, we propose that EEG biofeedback can be utilized to remedy aberrant coherence patterns.

Although there have been only a few studies investigating the use of neurofeedback in the treatment of autism, there is ample evidence documenting the efficacy of neurofeedback for various other neuropsychological disorders, including ADHD (Fuchs et al. 2003; Heinrich et al. 2004; Lubar and Lubar 1984), epilepsy (Lubar et al. 1981; Monderer et al. 2002; Sterman 2000; Sterman and Friar 1972; Walker and Kozlowski 2005), traumatic brain injury (TBI) (Byers 1995; Hoffman et al. 1996; Keller 2001; Schoenberger et al. 2001; Walker et al. 2002), anxiety disorders (Moore 2000), and

substance abuse disorders (Trudeau 2005). Furthermore, neurofeedback (NF) appears to have long lasting effects, something that pharmacological therapies often lack (Ayers 1995). The majority of these studies have utilized symptom based neurofeedback protocols, which has been the traditional form of treatment.

Quantitative electroencephalograph guided neurofeedback studies have recently demonstrated efficacy for treating obsessive-compulsive disorder (Hammond 2003), behavioral difficulties found in children who have been abused and/or neglected (Huang-Storms et al. 2007), post-traumatic symptoms (Walker et al. 2002) of traumatic brain injury; as well as learning disabilities (Thornton and Carmody 2005). These accumulated studies are adding evidence in support of the efficacy of QEEG guided neurofeedback protocols. We have been unable to find any published studies directly comparing the efficacy of symptom based neurofeedback and QEEG guided neurofeedback. Although there has been some research documenting the efficacy of neurofeedback in ASD, these two distinct approaches have not been compared in this population.

Cowan and Markham (1994) conducted the first case study of neurofeedback with Autism. QEEG analysis, performed on an 8 year old girl diagnosed with high functioning Autism during an eyes open and at rest state, indicated an abnormally high amount of alpha (8–10 Hz) and theta (4–8 Hz) activity predominately in the parietal and occipital lobes. Based on these results, a neurofeedback protocol was designed to suppress the ratio of theta and alpha (4–10 Hz) to beta (16–20). Following 21 sessions, the child showed increased sustained attention, decreased autistic behaviors (inappropriate giggling, spinning), and improved socialization based on parent and teacher reports. Attention improved substantially, as assessed by the Test of Variables of Attention (TOVA), and this was maintained at a 2 year follow-up.

Two controlled studies have been published that have investigated group differences in the efficacy of neurofeedback for autistic spectrum disorders. Jarusiewicz (2002) administered between 20 and 69 sessions of neurofeedback to a group of 12 autistic children. They were matched for age, sex, and disorder severity to a control group of autistic children. Treatment efficacy was determined by scores on the Autism Treatment Evaluation Checklist (ATEC). Her neurofeedback protocols were selected based on the individual child's symptoms and were determined by the Othmer Assessment (1997). Initial protocols provided reward for activity at site C4, referenced to the contralateral ear, in the 10–13 Hz range. Fifty-four percent of the sessions utilized this protocol. Children with vocalization problems had an F7 electrode placement with right ear reference. Rewards were for 15–18 Hz and inhibits were at 2–7 and 22–30 Hz. If no signs of overstimulation were shown after 5 min, additional 5 min increments were added, up to a maximum of 30 min. This protocol accounted for 15% of sessions.

For children who required help with socialization and communication, a bipolar F3-F4 electrode placement was utilized with 7–10 and 14.5–17.5 Hz rewards and 2–7 and 22–30 Hz inhibits. This protocol was used 12% of the time and was discontinued if inappropriate laughing or giggling were noted in the child. Children with emotional instability were given a T3–T4 placement, beginning with 9–12 Hz rewarded and 2–7 and 22–30 Hz inhibited. Protocol frequencies were increased or decreased depending on whether children were sad, anxious, or hyperactive. Training sessions were generally given one to three times per week, with two sessions being the most common.

Neurofeedback resulted in all children showing improvement, as based on ATEC scores, with significant improvements noted in 8–56%, or an average 26% reduction of symptoms. Specifically, improvement was noted in the areas of sociability (33%), speech/language/communication (29%), health (26%), and sensory/cognitive awareness (17%). These results stand in contrast to a 3% overall reduction in the control group. Furthermore, parents reported behavioral improvements in socialization, vocalization, anxiety, schoolwork, tantrums, and sleep. Only minimal changes were noted in the control group.

In contrast to the study by Jarusiewicz, Coben and Padolsky (2007) utilized assessment guided neurofeedback on 37 patients over the course of 20 sessions, compared to a wait-list control group. The training protocol was based on several measures including ratings scales, neuropsychological data, several neurobehavioral rating scales, and primarily QEEG. The focus was on reducing hyperconnectivity, principally in posterior-frontal to anterior-temporal regions, and was based on regions of maximal hyperconnectivity. Hyperconnectivity was chosen as an early training goal based on our perception of its prevalence and priority within our connectivity theory of autism (Coben et al. 2008). It was also shown to be effective in previous studies (see Coben and Padolsky 2007, for a review). For example, Coben (2007) reported a case study of a boy who was autistic who showed a 45% reduction in autistic symptoms, improvement on various neuropsychological measures, and reductions in connectivity in theta, alpha, and beta bands. This example shows how protocols are designed based on this connectivity approach. This protocol remained constant throughout all 20 sessions, and were conducted twice per week. Eighty-nine percent of the 37 patients had sequential (bipolar) versus unipolar montages. Ninety-four percent of the sequential (bipolar) montages included frontal or temporal electrode sites including F8-F7, Ft8-Ft7, T4-T3, or F7-F8. In one case, F6-F5 was applied and in the other F4-F3. Reward bands ranged anywhere from 5–16 Hz. A delta inhibit protocol as

low as 1–2 Hz, ranging to as high as 6 Hz, was utilized for 92% of the patients. In 100% of patients, a high beta inhibit protocol was applied ranging from 18–50 Hz with the greatest overlap at 18–30 Hz. A third inhibit ranging within a 7–14 Hz range was utilized for 68% of the patients.

Following neurofeedback, parents reported symptom improvement in 89% of the experimental group, compared to the control group in which 83% of subjects remained unchanged. Neuropsychological improvement was noted in the areas of attention, visual perceptual functioning, language, and executive functioning. We (Coben and Padolsky 2007) found a 40% reduction in core ASD symptoms as rated by the ATEC total scores, along with decreased hyperconnectivity in 76% of the experimental group as assessed by follow-up QEEG. These results suggest that decreased hyperconnectivity results in improvement in treatment outcomes measures in autism.

In this study, we hypothesized that QEEG connectivity guided neurofeedback, would have greater relative efficacy when compared to symptom based neurofeedback. Specifically, we expect to see greater improvement in symptom severity, over the course of fewer sessions when comparing these two approaches. Additionally, because there were differences in both the number of participants and severity of symptoms of autism between these studies, we predicted that symptom severity would not impact the greater efficacy seen with QEEG connectivity guided neurofeedback.

Methods of Comparison

In order to investigate whether there are any differences in the effectiveness of QEEG connectivity guided and symptom based neurofeedback, we compared the results of Jarusiewicz's (2002) study to those of Coben and Padolsky (2007). Both of these studies utilized neurofeedback with the methods of the two studies described above. The main difference between the two studies is that Coben and Padolsky (2007) utilized a QEEG assessment guided neurofeedback protocol based on abnormal connectivity, while Jarusiewicz (2002) administered neurofeedback protocols based on the individual child's symptoms as determined by the Othmer Assessment (1997).

Because the sample size of Coben and Padolsky's study was larger ($n = 37$) and displayed less severe autistic symptomotology, separate analyses were conducted with and without equal sample sizes. To equate the groups in terms of sample size and symptom severity (as measured by ATEC scores), 25 children from Coben and Padolsky's study with the lowest scores on the ATEC were removed. An independent groups t-test was conducted to examine group differences in both pre- and post-treatment ATEC subtest scores (Speech/Language Communication, Sociability, Sensory/Cognitive Awareness, Health/Physical/Behavior) and total scores. The difference in scores (pre-treatment score minus post-treatment score) and percent change scores (post-treatment score divided by pre-treatment score) were also examined between the two groups. A regression analysis was then performed to determine if age predicted outcome. Another independent groups t-test was performed to examine differences in the amount of change that occurred per session, defined as the amount of change that occurred pre-post neurofeedback divided by the number of sessions of NF administered.

Results

As noted in their original papers, both studies showed significant improvement in symptoms of autism as measured by ATEC scores. When comparing the two study groups there were no significant differences in race or gender. Data on handedness, IQ and medication were unavailable for Jarusiewicz's (2002) group. While Jarusiewicz's group was significantly older statistically [$t\ (22) = -2.743$, $p = .012$], this difference is not believed to be clinically significant (less than a 3 year difference between groups). When sample sizes were not equated, significant differences were found between the two data sets in the total score at post-treatment [$t\ (40) = 3.028$, $p = .003$] and percent change (see Table 1) that occurred between groups [t ($df = 32.8$ with equal variances not assumed) $= -2.122$, $p = .041$]. However, the subjects in Jarusiewicz's (2002) study were significantly more impaired at pre-treatment as well [$t\ (41) = 2.480$, $p = .017$]. Therefore, differences between groups were further examined with equal sample sizes, which permitted comparisons considering equivalent severities by removing 25 subjects with the most severe symptoms of autism.

On the ATEC, there were no significant group differences in any of the pretreatment scores (see Table 2). Significant differences were found on the Sensory/Cognitive Awareness scale when comparing post-treatment scores [$t\ (22) = 3.068$, $p = .006$], difference scores [$t\ (22) = -2.249$, $p = .035$] (see Fig. 1), and percent change scores [$t\ (22) = -2.442$, $p = .023$] (see Table 3 for a listing of all percent change scores). Although there were no significant differences in post-treatment Health/Physical/Behavior subtest scores, the percent change score was significant [$t\ (22) = -2.099$, $p = .047$]. Significant differences were also found between groups when comparing both the total ATEC difference scores [$t\ (22) = -3.032$, $p = .006$] (see Fig. 2) as well as percent change scores for the Sensory/Cognitive Awareness [$t\ (22) = -2.442$, $p = .023$], Health/Physical/

Table 1 Percent change between pre- and post-treatment scores—all subjects

	N	Mean	SD	*t*	*df*	Sig. (2-tailed)
Speech/lang/comm						
Jarusiewicz (2002)	12	34.17	26.10	.312	39	.757
Coben and Padolsky (2007)	29	22.41	128.62			
Sociability						
Jarusiewicz (2002)	12	30.33	30.79	−.954	40	.346
Coben and Padolsky (2007)	30	39.90	28.81			
Sens/cog awareness						
Jarusiewicz (2002)	12	16.08	9.28	−3.148	34.48*	.003
Coben and Padolsky (2007)	30	40.52	39.23			
Health/phys/behavior						
Jarusiewicz (2002)	12	22.75	19.36	−.912	40	.367
Coben and Padolsky (2007)	30	32.97	36.64			
Total						
Jarusiewicz (2002)	12	26.17	14.39	−2.122	32.80*	.041
Coben and Padolsky (2007)	30	38.83	23.48			

* df for equal variances not assumed

Behavior [t (22) = −2.099, p = .047], and total scores [t (22) = −2.853, p = .009] (see Fig. 3). Regression analysis using age as a predictor was not significant for any of the subscales of the ATEC, indicating the differences in age between the two groups could not account for the differences in treatment outcomes.

It is also important to note that Jarusiewicz (2002) used a significantly greater number of sessions [t (22) = 3.160, p = .005] than Coben and Padolsky (2007) to achieve their outcomes. When the amount of change which occurred per session was compared, Coben and Padolsky's (2007) study demonstrated significantly greater change on all subscales of the ATEC (see Table 4; Fig. 4), including the total score (see Fig. 5). Specifically, greater change was noted in the areas of Speech/Language Communication [t (22) = −3.092, p = .005], Sociability [t (22) = −2.608, p = .016], Sensory/Cognitive Awareness [t (11.9) = −2.947, p = .012], Health/Physical/Behavior [t (22) = −3.471, p = .002]), and total autistic symptoms [t (22) = −4.471, p < .001]. We found a threefold improvement per session (ATEC Total percent change per session; 0.84 vs. 2.31%) in the QEEG based study as compared to the symptom based study. Thus, more efficacious results were demonstrated in fewer treatment sessions.

Table 2 Pre-treatment ATEC scores

	N	Mean	SD	*t*	*df*	Sig. (2-tailed)
Speech/lang/comm						
Jarusiewicz (2002)	12	13.65	5.83	0.78	22	0.446
Coben and Padolsky (2007)	12	11.42	8.08			
Sociability						
Jarusiewicz (2002)	12	14.85	6.22	−1.54	22	0.138
Coben and Padolsky (2007)	12	18.92	6.72			
Sens/cog awareness						
Jarusiewicz (2002)	12	17.67	4.02	1.45	22	0.162
Coben and Padolsky (2007)	12	15.08	4.70			
Health/phys/behavior						
Jarusiewicz (2002)	12	18.69	10.82	−1.22	22	0.236
Coben and Padolsky (2007)	12	23.67	9.12			
Total						
Jarusiewicz (2002)	12	64.86	21.08	−0.63	22	0.537
Coben and Padolsky (2007)	12	69.92	18.29			

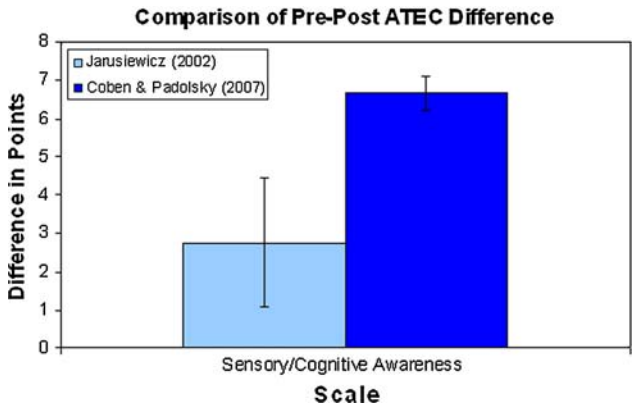

Fig. 1 Significant differences were found between Jarusiewicz (2002) and Coben and Padolsky (2007) when comparing pre-treatment scores on the sensory/cognitive awareness scale of the ATEC with post-treatment scores

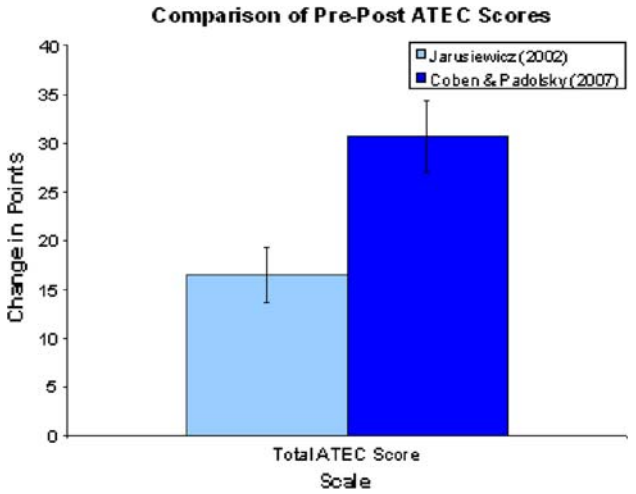

Fig. 2 Significant differences were found between Jarusiewicz (2002) and Coben and Padolsky (2007) when comparing pre-treatment total ATEC scores with post-treatment total ATEC scores

Discussion

One of the ongoing debates among neurofeedback providers is whether treatment should be assessment based or symptom based (Hammond et al. 2004). However, few empirical studies have been conducted to examine differences in the efficacy of these approaches. This was the first attempt to compare assessment (QEEG) and symptom guided neurofeedback protocols in an autistic population, albeit not a direct comparison.

It is important to note that both studies have provided evidence suggesting that neurofeedback is an effective form of intervention for autism. Jarusiewicz (2002) demonstrated a 26% average reduction in symptoms of autism following neurofeedback. Coben and Padolsky (2007) demonstrated a 40% reduction in symptoms of autism in addition to improvement on various neuropsychological and neurophysiological measures post-neurofeedback. However, when comparing the two studies, the assessment guided neurofeedback resulted in significantly lower scores on measures of Sensory/Cognitive Awareness and Health/Physical/Behavior, as well as total treatment effectiveness. The Sensory/Cognitive Awareness scale assesses an individual's responsiveness to their environment, understanding of explanations and events, and demonstrating imagination and interest in things. The Health/Physical/Behavior scale assesses health functioning, such as gastrointestinal issues, sleep, diet; level of physical activity (i.e., hyperactive,

Table 3 Percent change between pre- and post-treatment scores—equal sample sizes

	N	Mean	SD	*t*	*df*	Sig. (2-tailed)
Speech/lang/comm						
Jarusiewicz (2002)	12	34.17	26.10	−1.86	22	0.076
Coben and Padolsky (2007)	12	56.50	32.25			
Sociability						
Jarusiewicz (2002)	12	30.33	30.79	−1.15	22	0.262
Coben and Padolsky (2007)	12	42.83	21.62			
Sens/cog awareness						
Jarusiewicz (2002)	12	16.08	9.28	−2.44	22	0.023
Coben and Padolsky (2007)	12	42.33	36.07			
Health/phys/behavior						
Jarusiewicz (2002)	12	22.75	19.36	−2.10	22	0.047
Coben and Padolsky (2007)	12	41.00	23.07			
Total						
Jarusiewicz (2002)	12	26.17	14.39	−2.85	22	0.009
Coben and Padolsky (2007)	12	46.25	19.69			

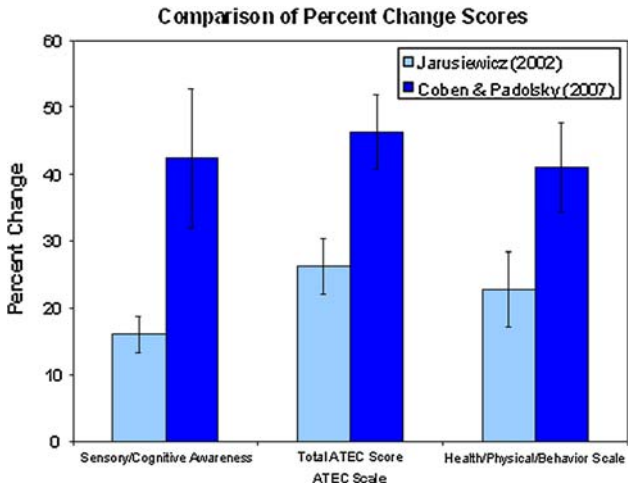

Fig. 3 Significant differences were found between Jarusiewicz (2002) and Coben and Padolsky (2007) when comparing the percent of change that occurred between pre- and post-treatment scores on the sensory/cognitive awareness scale, health/physical/behavior scale, and total ATEC

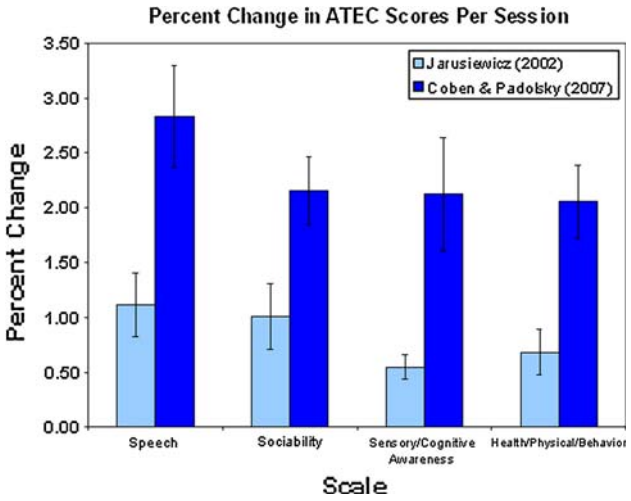

Fig. 4 The amount of change which occurred per session in Coben and Padolsky (2007) was significantly greater than the amount of change which occurred per session in Jarusiewicz (2002) for all subscales of the ATEC

lethargic); and overall behavior including anxiety, mood, repetitive movements and speech, agitation, and sensitivity to sounds and pain. These findings could not be accounted for by differences in age between the two groups.

There was a large disparity in the number of sessions required to produce the changes observed in these studies. Whereas Coben and Padolsky (2007) used 20 sessions of neurofeedback for each subject, Jarusiewicz (2002) used between 20 and 69 sessions (mean of 36 sessions). Coben and Padolsky (2007) administered significantly fewer sessions, which resulted in significantly greater change per session on all scales of the ATEC. Not only was greater improvement noted in their group, but it was accomplished more quickly. We found a threefold improvement per session (ATEC Total percent change per session; 0.84 vs. 2.31%) in the QEEG based study as compared to the symptom based study. Thus, greater results were demonstrated in much fewer treatment sessions. This is particularly important considering that individuals who are autistic often have difficulty sitting through extensive treatment sessions, and so reducing the number of sessions needed would be particularly beneficial to this group.

Our reanalysis suggest that neurofeedback guided by a QEEG assessment may be more efficacious than a

Table 4 Percent change per session

	N	Mean	SD	*t*	*df*	Sig. (2-tailed)
Speech/lang/comm						
Jarusiewicz (2002)	12	1.12	1.03	−3.092	22	0.005
Coben and Padolsky (2007)	12	2.83	1.62			
Sociability						
Jarusiewicz (2002)	12	1.01	1.06	−2.608	22	0.016
Coben and Padolsky (2007)	12	2.15	1.08			
Sens/cog awareness						
Jarusiewicz (2002)	12	.55	.37	−2.947	22	0.012
Coben and Padolsky (2007)	12	2.12	1.80			
Health/phys/behavior						
Jarusiewicz (2002)	12	.68	.74	−3.471	22	0.002
Coben and Padolsky (2007)	12	2.05	1.15			
Total						
Jarusiewicz (2002)	12	.84	.57	−4.471	22	0.000
Coben and Padolsky (2007)	12	2.31	.98			

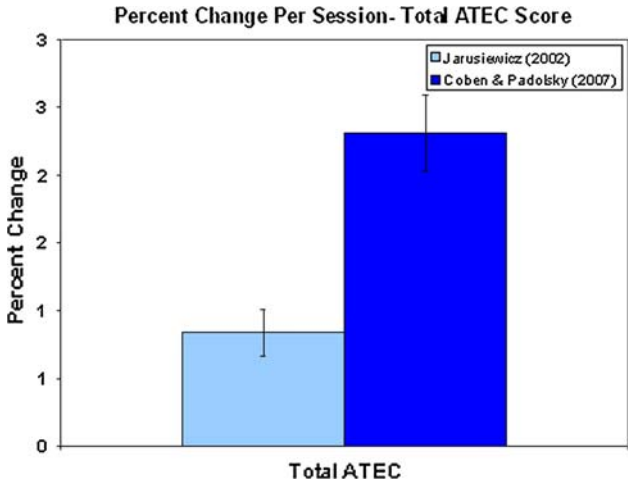

Fig. 5 The amount of change in Total ATEC scores per session was significantly greater in Coben and Padolsky (2007) than the amount of change per session in Jarusiewicz (2002)

symptom based approach to neurofeedback. However, there are different approaches even within QEEG guided neurofeedback, including both power and coherence training protocols. The number of electrode locations also can vary between assessments. However, a full QEEG assessment allows the clinician to pinpoint the area of abnormality and train it accordingly. Coben and Padolsky (2007) produced their results by reducing hyperconnectivity in 76% of the experimental group, which led to improved treatment outcomes. In fact, this was the first published study to demonstrate the effectiveness of coherence training for reducing the symptoms of autism.

Recently, evidence has been accumulating in support of a connectivity theory of autism (Alexander et al. 2007; Boger-Megiddo et al. 2006; Coben et al. 2008; Coben and Myers 2009; Chung et al. 2004; Courchesne and Pierce 2005; Courchesne et al. 1993, 2005; Just et al. 2007; Murias et al. 2007; Vidal et al. 2006). Coherence training is a direct application to address these findings, which are backed by numerous empirical studies.

Despite the promising results of our comparison, there were several limitations in this analysis. Perhaps most importantly, it did not involve a direct comparison of the two groups. The studies took place 5 years apart, in different locations, and with different sample sizes. Although the samples were equated in both number of subjects and symptom severity, the sample sizes were relatively small. Although these findings must be viewed with caution as a result, our levels of significance suggest the group differences are meaningful.

Future studies should be conducted to directly compare these two methods of neurofeedback treatment, both in individuals who are autistic as well as in the treatment of other psychological/neuropsychological disorders, with larger sample sizes. In this analysis, our results were based on coherence training to correct areas of abnormal connectivity. Future studies should investigate whether this method of treatment is superior to other QEEG assessment based protocols that may focus more on changing EEG frequency and amplitude. As a follow up to our study, it would also be interesting to see if individuals treated by these different approaches varied with respect to long-term maintainence of gains following the completion of neurofeedback.

The type of coherence training shown to be of value here (for hyperconnectivity principally in posterior-frontal to anterior-temporal regions) is based on just one of the many abnormalities noted on QEEG that may be ameliorated by neurofeedback training. Coben and Myers (2009) outlined seven patterns of abnormal connectivity in autistic spectrum disorders; some hyper- and some hypo-connected. Theoretically, any individual may present with between one and seven of these abnormalities. It is likely that treatment of all abnormalities present would lead to the highest reduction of symptoms. Other types of abnormalities in Mu rhythm (Bernier et al. 2007), excessive theta (Coben et al. 2008), and higher theta and beta 1 power (Murias et al. 2007) among others have also been documented. No studies have been conducted, to our knowledge, that have addressed all abnormalities in patients. Furthermore, it would be difficult to parse out the effects of one protocol from another to demonstrate differential efficacy. Future research may address if sequential or simultaneous treatment of EEG abnormalities is more effective.

Ideally, there should be randomized controlled trials (RCTs) in order to demonstrate the efficacy of neurofeedback as a validated treatment (LaVaque et al. 2002). Future research should be conducted with double blind, placebo controlled trials for both neurofeedback approaches. The connectivity guided model should be further investigated, in particular with individuals diagnosed as autistic.

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