1-1-2011

Effects of Neurofeedback on Neuropsychological Functioning in an Adult with Autism

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2012

Abstract

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Dissertation Submitted in Partial Fulfillment

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Doctor of Philosophy

Psychology

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Abstract

Autism spectrum condition (ASC) is a complex neurodevelopmental disorder that impacts physiological processes, cognition, functional behaviors, social-communication, and often has comorbidities. One approach gaining empirical support for ASC treatment is neurofeedback. Neurofeedback uses operant conditioning to normalize cerebral activity through auditory and visual reinforcement. Live Z-score Training (LZT) has become the latest advancement in neurofeedback. There is no published research to date on LZT neurofeedback in adulthood ASC. The purpose of this study was to evaluate LZT's impact on neuropsychological measures in an adult with ASC. A multiple baseline single-case research design was used with a convenience sample of one adult with ASC to evaluate the effects of 20 LZT sessions using the Conservative Dual Criterion visual inspection method as the primary form analysis. ADHD, mood stability, anxiety, depression, and ASC symptoms were significantly reduced according to the Neuropsych Questionnaire. The participant improved significantly on the CNS Vital Signs (CNVS) Neurocognitive measures of executive function, cognitive flexibility, reaction time, and complex attention. Also, the participant increased intelligence as measured by the Test of Nonverbal Intelligence. Lastly, the participant had changes in brain function according to quantitative electroencephalography and low-resolution brain electromagnetic tomography. CNVS processing speed was the only measure that did not significantly change. No adverse effects were reported. This study may lead to positive social change by providing a technologically advanced intervention for adults with ASC, which may improve their overall quality of life and promote self-sufficiency through adulthood.

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Dedication

First and foremost, this dissertation is dedicated to my sweet Melissa and our doggies, Helios and Cash, for their love and support in allowing me to dedicate these years toward obtaining my doctorate. Melissa, you are my best friend and twin flame for whom I owe so much. I would have never been able to do this without your unconditional love and words of encouragement that empowered me to continue. Thank you for being you- I love you so much. Secondly, this project is devoted to my mother and father who have provided me with the foundation and encouragement to pursue higher education and always strive to learn more. Since my first day of kindergarten, I still remember their mantra every morning, "Do good in school." This dissertation reflects the product of all those years you both dedicated in helping me succeed- thank you. Finally, this dissertation is dedicated to adults with autism who struggle with the condition, and their caregivers who work so hard to find the best treatment. You are admired for your strength, energy, and advocacy. I would especially like to thank the participant in this study and his mother for playing a key role in advancing the research in this area. The findings in this study exemplify that there can be significant improvement in many areas for an adult with autism. Finding the cure should not be goal. The goal should be to improve the overall quality of life of people with autism, so that they can be happier and achieve more goals than ever expected. There is always hope!

Acknowledgments

First and foremost, I am in debt to Dr. Lisa Scharff's guidance throughout this process. She provided thorough reviews of countless rough drafts and offered the continuous support I needed to complete this project. She was always available either by phone, conferencing or email. You have really helped me to work through the many obstacles that might have otherwise set me back or abandoned the project completely. One last time- thank you! I would like to also acknowledge the important perspectives of Dr. Patricia Loun. You provided the expertise for advancing the methodology and allowing me the freedom to use the single case research method as a viable way to test my hypotheses. You gave key recommendations to this study for which I am so grateful. Also, to Dr. Brian Ragsdale, as the University Research Reviewer, you ensured I met the high standards of Walden University. This project would not have been completed if you did not provide this necessary feedback. Thank you for supporting and approving this project. Next, I am greatly appreciative of Dr. Andrew Sahara's mentoring and encouragement. Sharing your experience provided me the motivation I needed to accomplish this great task. Finally, I would not have been able to complete this study without Linda Walker's mentoring, partnership, guidance, and assistance. You gave me the opportunity to explore the efficacy of neurofeedback in autism while maintaining the highest level of professional standards set forth by the field. Thank you so much Linda for your many hours, materials, and equipment that you donated to this study. I look forward to our future collaboration.

Table of Contents

List of Tables vi

List of Figures vii

Chapter 1: Introduction 1

Introduction to the Study 1

Background of the Study 2

Problem Statement 3

Purpose of the Study 4

Nature of Study 4

Research Questions and Hypotheses 5

Theoretical Basis 7

Definition of Terms 8

General Terms 8

Neurofeedback Terms 9

Assumptions 11

Limitations 12

Delimitations 14

Significance of the Study 15

Social Change Implications 16

Summary and Transition 16

Chapter 2: Literature Review 18

Introduction to the Literature Review 18

Overview of Autism Spectrum Conditions 19

Diagnosis Background of Autism Spectrum....19
Neurocognitive Theories....21
Genetic & Neurochemical Abnormalities....26
Neuroimaging Patterns in ASC....29
Neurological Patterns in ASC....33
Prevalence Rates & Costs....38
Autism in Older Adolescence and Adulthood....39
Neurofeedback Background....40
Overview of Electroencephalography....40
Theoretical Background of Neurofeedback....43
Neuroplasticity and Neurofeedback....46
Brief History of Neurofeedback....47
Efficacy of Neurofeedback in ASC....49
Single-Case Research....49
Quasi-Experimental Research Studies....54
Experimental Research Study....58
Longitudinal Research Studies....60
Research Specific to Neurofeedback LZT....60
Number of Sessions Variability in Neurofeedback Research....65
Neurofeedback Adverse Effects....66
Gap in the Neurofeedback Research on Autism....67
Summary and Transition....69

Chapter 3: Research Method....71
Introduction to the Research Method....71
Research Design Overview....71
Setting and Participant Recruitment....73
Research Setting....73
Participant....74
Sample Size....77
Data Collection and Analysis....79
Instrumentation & Materials....83
Computer Equipment....83
Software Programs....84
Neuropsych Questionnaire....84
CNSVS Neurocognitive Test....86
Test of Nonverbal Intelligence....87
Quantitative Electroencephalogram....89
Monitoring of Side Effects Scale....90
Overview of Dependent Variables....91
Procedure....92
Phase A....92
Phase B....94
Research Questions & Hypotheses....95
Variables....99
Protection of Participant....99

Summary and Transition 100
Chapter 4: Results 101
Introduction to the Results 101
Overview of Sampling 101
Recruitment 101
Sample 101
Research Question Analysis 102
Research Question 1 102
Research Question 2 103
Research Question 3 106
Research Question 4 109
Research Question 5 110
Side Effect Scales 115
Summary Tables 116
Conclusion 117
Chapter 5: Discussion 118
Introduction to the Discussion 118
Summary of Research Findings 119
Limitations of the Study 123
Generalization 125
Implications for Social Change 126
Implications for Future Research and Practice 127
Conclusion 128

References 130

Appendix A: Wave Frequency Descriptions 155

Appendix B: Cover Letter to Potential Participant 156

Appendix C: Advertisement 157

Appendix D: Informed Consent 158

Appendix E: Confidential Agreement 164

Appendix F: Neurofeedback Volunteer Registration and Screening Form 166

Appendix G: Authorization to Disclose PHI for Research 168

Appendix H: Neuropsych Questionnaire Long Form 169

Appendix I: Monitoring of Side Effects Scale 171

Appendix J: Letters of Permission 172

Curriculum Vitae 175

List of Tables

Table 1. Dependent Variables 92

Table 2. Mean Change for Self-Reports 116

Table 3. Mean Change for Neurocognitive Abilities 116

Table 3. Mean Change for Intelligence 117

Table 4. Mean Change for Side Effect Profiles 117

List of Figures

Figure 1. The 10-20 International System of Electrode Placement 42

Figure 2. LZT Neurofeedback Screen 65

Figure 3. Example of visual inspection with significant effect 82

Figure 4. Example visual inspection with no significant effect 82

Figure 5. Example of QEEG maps' summary of absolute power 83

Figure 6. Brainmaster flash player Brain Planets game 94

Figure 7. Asperger's index displays a significant improvement 103

Figure 8. ADHD index displays a significant improvement 104

Figure 9. Anxiety index displays a significant improvement 104

Figure 10. Depression index displays a significant improvement 105

Figure 11. Mood Stability index displays a significant improvement 105

Figure 12. Executive Functioning displays a significant improvement 106

Figure 13. Processing Speed index displays no significant effect 107

Figure 14. Cognitive Flexibility index displays a significant improvement 108

Figure 15. Complex Attention index displays a significant improvement 108

Figure 16. Reaction Time index displays a significant improvement 109

Figure 17. Nonverbal IQ displays a significant improvement 110

Figure 18. Baseline Eyes Closed QEEG Neuroguide connectivity maps 111

Figure 19. QEEG absolute power maps display significant effects 112

Figure 20. LORETA maps display normalization of absolute power in 18 Hz 114

Figure 21. MOSES Psychological side effect profile 115

Figure 22. MOSES Neurological side effect profile 115

Chapter 1: Introduction

Introduction to the Study

The purpose of this study is to explore the efficacy of neurofeedback Live Z-score Training (LZT) in improving overall neuropsychological functioning in an adult who has an autism spectrum condition (ASC). The American Psychiatric Association Diagnostic (APA) Statistical Manual Fourth Edition Text Revised (DSM-IV-TR; 2000) categorized ASC in the following mental disorders: autistic disorder, Asperger's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified. Most recently, the APA (2010) has considered autism as a spectrum disorder, including the subtypes under one unified label. The proposed change will still maintain the two core symptoms of autism: stereotypic behaviors/obsessive narrowed interests and social-communication impairment. Although diagnosed in childhood and considered a part of the childhood mental disorders in the DSM-IV-TR, autism is a neurodevelopmental disability with impairments that persist in adulthood (APA, 2000, 2010). Despite the lifelong impact of ASC, intervention researchers largely focused on children and adolescents and have not been validated in adult samples (Roy, Dillo, Emrich, & Ohlmeier, 2009).

The principal investigator plans to evaluate the effects of neurofeedback on measures of neuropsychological functioning in an adult with ASC, an area that has not been well researched (Coben, Linden, & Myers, 2010). Neurofeedback has demonstrated effectiveness in treating children with ASC through improved neurophysiological functioning, executive functioning, and decreased autistic symptoms (Coben & Padolsky, 2007); however, there has been only one published neurofeedback study that included

adults with ASC (Thompson & Thompson, 2010). Thompson and Thompson published this retrospective report reviewing the effect of a combination of neurofeedback and other interventions in 12 adults with ASC (Thompson, Thompson, & Reid, 2010b). Earlier, the same authors published qualitative case studies of adults with ASC in a textbook on neurofeedback (Thompson & Thompson, 2003). It is necessary to further investigate neurofeedback in adults with ASC in order to better understand the efficacy of neurofeedback in older age groups, with consideration for changes of neuroplasticity with age.

Quantitative research on neurofeedback in adults with neurodevelopmental disorders will be an important step to more fully evaluate the effects of such interventions in different stages of life. Further, there has not been a quantitative study investigating change related to a specific type of neurofeedback, LZT, which uses a normative database in real-time to individualize sessions (Collura, Guan, Tarrant, Bailey, & Starr, 2010; Thatcher, 2008; Thatcher & Lubar, 2008).

Background of the Study

Since the mid-1980s, rates of ASC have continued to rise. From only .4 in 1,000 children being diagnosed in 1985 to 9 in 1,000 children by 2006 this increase identifies a great need for comprehensive evaluations and interventions early in childhood (Centers for Disease Control and Prevention [CDC], 2007, 2009). Providing such services may reduce the scope of lifelong needs for services and aid in obtaining social independence. However, early interventions are often not available due to financial reasons, access to care, or treatment resources available (Symon, 2001). Adults with ASC in particular are left with few options, especially for noninvasive interventions that address both the

behavioral and neurological aspects of autism (Minshew, Sweeney, Bauman, & Webb, 2005). Adulthood interventions are further lacking in areas like assessment and for research that addresses the ongoing deficits that impact global functioning such as ability to obtain and maintain employment (Shea & Masibov, 2005). The need to validate effective interventions for adults with ASC is critical to offsetting pervasive service needs throughout adulthood (Wolf & Paterson, 2010).

Problem Statement

The main research problem addressed in this study is that autism researchers have mainly focused treatment for ASC in childhood (Roy et al., 2009; Wolf & Paterson, 2010). Specifically, Coben et al. (2010) suggested that neurofeedback is effective in children with ASC; however, what is missing in the literature are data regarding how neurofeedback may be related to neuropsychological change in adults with ASC. No researchers have focused specifically on ASC in this age group despite the need for long-term interventions. Adults with ASC, who are often in need of lifelong supports because of developmental delays, are largely ignored in the literature (APA, 2000; Wolf & Paterson, 2010). Problems like gainful employment, independent living, relationships, and comorbid conditions such as anxiety disorders complicate the concerns for adult individuals with ASC (Shea & Masibov, 2005). Most importantly, researchers need to determine if interventions found effective in childhood are associated with neuropsychological change in adulthood (Coben et al., 2010). Also, interventions may be associated with improvement in neuronal development throughout a lifetime (Jones, 2004; Malkowicz & Martinez, 2009; Pinel, 2008). Furthermore, neuroplasticity in adulthood is a critical area to explore particularly for adults with ASC to determine if the

same interventions effective in childhood are related to improved neuronal functioning and neurocognitive abilities in adults.

Purpose of the Study

The purpose of this single-case research study was to evaluate the effect of neurofeedback LZT on neuropsychological symptoms, core autistic symptoms, neurocognitive abilities, intelligence, and neurophysiological functioning in an adult with ASC identified through a local neurofeedback clinic. Few studies investigating change associated with interventions specifically addressing ASC symptoms in adults have been conducted (Roy et al., 2009), and it may be expected that treatments found effective in children and adolescents may be associated with similar changes in adulthood. However, there have been no studies evaluating neurofeedback LZT for adults with ASC. Lastly, this study may enhance the need to explore use of single-case research as an effective methodological approach for smaller and unique populations like adults with ASC, particularly in rural areas.

Nature of the Study

The single-case research study will consist of a convenience sample of an adult participant, over the age of 18, diagnosed with ASC recruited from rural northern Michigan neurofeedback clinic. The clinic was responsible for distributing the advertorial to prospective clients who met the research criteria. The participant had a preexisting diagnosis of ASC identified by a qualified healthcare/educational professional. The clinic was responsible for providing a minimum of 16 sessions of neurofeedback LZT to the prospective client with ASC. The research study consisted of the assessment of neuropsychological and autistic symptoms, neurocognitive abilities,

nonverbal intelligence, quantitative electroencephalogram (QEEG), and observation of side effects, which were conducted during a baseline phase and neurofeedback phase, also referred to as an AB approach through visual inspection analyses.

Research Questions and Hypotheses

1. Is neurofeedback LZT related to the change in the core symptoms of autism in an adult with ASC?

$H_0 1$: $\mu_1 = \mu_2$ –There will be no significant differences in autism symptoms as measured by the Neuropsych Questionnaire, Long Form (NPQ-LF) during the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

$H_1 1$: $\mu_1 > \mu_2$ –There will be a significant decrease in autism symptoms as measured by the NPQ-LF between the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

2. Is neurofeedback LZT related to a significant reduction in neuropsychological symptoms associated with attention, impulsivity, anxiety, depression, and mood stability of an adult with ASC?

$H_0 2$: $\mu_1, \mu_2, \mu_3, \mu_4 = \mu_6, \mu_7, \mu_8, \mu_9$ –There will be no significant differences in ADHD, Anxiety, Depression, and Mood Stability indices as measured by the NPQ-LF during the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

$H_1 2$: $\mu_1, \mu_2, \mu_3, \mu_4 > \mu_6, \mu_7, \mu_8, \mu_9$ –There will be significant decreases in ADHD, Anxiety, Depression, and Mood Stability indices as measured by the NPQ-LF between

the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

3. Is neurofeedback LZT related to a significant improvement in neurocognitive abilities in executive functioning and processing speed in an adult with ASC?

$H_0$3: $\mu_1, \mu_2 = \mu_3, \mu_4$ –There will be no significant differences in executive functioning and processing speed as measured by the CNS Vital Signs (CNSVS) Neurocognitive Test between the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

$H_1$3: $\mu_1, \mu_2 < \mu_3, \mu_4$ –There will be significant increase in executive functioning and processing speed as measured by the CNSVS Neurocognitive Test between the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

4. Is neurofeedback LZT related to significant improvement in overall nonverbal intelligence in an adult with ASC?

$H_0$4: $\mu_1 = \mu_2$ –There will be no significant difference in general intelligence as measured by the Test of Nonverbal Intelligence (TONI) between baseline and post-test quotient scores in a participant who receives 20 sessions of neurofeedback LZT.

$H_1$4: $\mu_1 < \mu_2$ –There will be a significant increase in general intelligence as measured by the TONI from baseline to post-test quotient scores in a participant who receive 20 sessions of neurofeedback LZT.

5. Is neurofeedback LZT related to normalization in QEEG measures in an adult with ASC?

$H_0 5$: $\mu_1 = \mu_2$ –There will be no significant differences in neurophysiological functioning as measured by QEEG based on the Applied Neuroscience, Inc. (ANI) Dynamic Link Library (DLL) and low brain resolution electromagnetic tomography (LORETA) statistical software in a participant who receives 20 sessions of neurofeedback LZT.

$H_1 5$: $\mu_1 < \mu_2$ – There will be significant changes in neurophysiological functioning as measured by QEEG based on the ANI DLL and LORETA statistical software in a participant who receives 20 sessions of neurofeedback LZT.

Theoretical Base

Neurofeedback is the newest biofeedback modality that utilizes an electroencephalogram (EEG) in order to modify brain states for improved psychological, neurocognitive, and neurophysiological functioning (ISNR Board of Directors, 2009). Neurofeedback is based on modifying brainwaves in the learning theory paradigm, specifically in operant and classical conditioning (Thompson & Thompson, 2003). Operant conditioning is considered the main behavioral learning approach in the core textbooks on neurofeedback (Demos, 2005; Thompson & Thompson, 2003). Operant conditioning is essentially the concept that a reinforcing stimulus increases the likelihood of the temporally associative behavior occurring again (Skinner, 1935, 1937, 1948, 1950). The Law of Effect is the basis for operant conditioning and most learning theories in that behavior increases when associated with a reinforcement or reward. Specifically, neurofeedback is based on contingent reinforcers consisting of visual and auditory rewards (ISNR Board of Directors, 2009). The feedback is temporally associated with EEG brainwave patterns that are specifically chosen to improve brain function. An

example might be to increase higher frequency bandwidths like beta waves to improve attention. Neurofeedback is also representative of behavioural classical conditioning (Thompson & Thompson, 2003). For instance, pairing the training (unconditioned stimulus) and elicited brainwaves (unconditioned response) with a desired behavior such as reading (conditioned stimulus) will promote optimal brainwave activity (conditioned response) during this behavior when auditory or visual stimuli is no longer present (Thompson & Thompson, 2003). In summary, neurofeedback consists of teaching the individual to self-regulate brainwaves through auditory and visual feedback. The theoretical and historical influences of neurofeedback will be explored in more detail in Chapter 2.

Definition of Terms

General Terms

Autism spectrum condition (ASC): Autism spectrum condition will be used throughout this dissertation in place of the diagnostic label of autism spectrum disorder (Baron-Cohen et al., 2005).

Hyperserotonemia: Elevated serotonin levels identified in ASC (Anderson, Horne, Chatterjee, & Cohen, 1990).

Neurophysiology: Study of physiological processes in neurons (Pinel, 2008).

Neuroplasticity: Adaptation of neuronal connections in the central nervous system across the lifespan (Gynther, Calford, & Sah, 1998).

Neurotransmitters: Chemical transmission from a neuron to a target cell through the synaptic cleft such as noradrenaline, dopamine, serotonin, and cholinergic and anticholinergic systems (Pinel, 2008).

Quasi-experimental: Research approach that tests causal hypotheses through the comparison of the manipulation of an experimental group and absence of the manipulation in the control group using pretest and posttest measures without random assignment (Shadish, Cook, & Campbell, 2002).

Theory of Mind: The ability to understand or predict the mental states in others (Leslie, 1987).

Weak Central Coherence: Inflexible, maintaining sameness, the inability to draw information together, recalling details but not the whole context, or failing to understand changes in the context for appropriate behavior (Thompson et al., 2010a).

Neurofeedback Terms

Asymmetry: A type of EEG connectivity measure that identifies the differences between signal amplitudes normalized to the sum of their amplitudes (Collura, 2008).

Amplitude: Height of the wave measured in microvolts-the variable that is changeable in neurofeedback (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007).

Bandwidth frequencies: The component bands for the Dynamic Link Library database consists of: Delta = 1-4 Hz, Theta = 4-8 Hz, Alpha = 8-12.5 Hz, Beta = 12.5-25.5 Hz, Beta 1 = 12-15.5 Hz, Beta 2 = 15-18 Hz, Beta 3 = 18-25.5 Hz, and Gamma = 25.5-30.5 (Collura, 2007).

Coherence: A type of EEG connectivity measure that calculates the cross correlation of shared activity and morphology between frequencies of two or more sites (Thompson & Thompson, 2003).

Connectivity: A complex mathematical equation that calculates the similarity

between various parts of the cerebral cortex (Collura, 2008).

Electroencephalography (EEG): A physiological recording measured with microvolts of post-synaptic potentials from pyramidal cells within the cerebral cortex to assess or use as a biofeedback intervention in neurological conditions such as epilepsy (Collura, 2008; Rowan & Tolunsky, 2003).

Frequency: Number of cycles per second measured in Hertz (Hz; Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007).

Hemoencephalography: The measurement of voluntarily-controlled regional blood flow in the brain through audio/visual feedback (Limsila et al., 2003).

Hertz: Measurement of each cycle of EEG wave per second (Demos, 2005; Hammond, 2006; Thompson & Thompson, 2003; Townsend, 2007).

Live Z-score Training (LZT): Software developed by Brainmaster using the Neuroguide database to compute z-scores in real time for assessment and neurofeedback training (Collura et al., 2010).

Mu rhythms: Frequency band of 8-13 Hz over the sensorimotor cortex that is consistent with mirror neuron system with reduced mu power being associated with performing and observing actions critical for imitation and understanding other's behaviors (Oberman et al., 2005).

Neurofeedback: A form of biofeedback that uses an EEG amplifier to measure electrical activity from the cortex to monitor and change brain function related to behavioral, cognitive, and subjective experiences through audio and visual reinforcement (ISNR Board of Directors, 2009).

Phase: A type of EEG connectivity measure that calculates the covariance and

when morphologically the same waves occur at the same time in two different sites (Thompson & Thompson, 2003).

Quantitative EEG (QEEG): The processing of an EEG recording of typically 19 sites and processed through statistical methods like Fast-Fourier Transformation (FFT) to quantify the power for each bandwidth or comparison of the record with a normative database which present results that show power, coherence, symmetry, or phase (Collura, 2008; Hughes, & John, 1999).

Z score: A metric standardization that compares a score or measure with a population mean through the number of standard deviations from the mean (Collura, 2007).

10-20 International System of Electrode Placement: The EEG system developed in the 1950s by Dr. Herbert Jasper to provide accurate measurements of the skull and landmarks using 10% and 20% of the total measurement to identify the 19 placements of EEG electrodes on three planes sagittal, coronal, and horizontal (Rowan & Tolunsky, 2003).

Assumptions

The following assumptions apply to this study:

- The use of neurofeedback will promote neuroplasticity of brain function in adulthood and serve as a way to develop new neuronal growth for complex neurological conditions like ASC (Malkowicz & Martinez, 2009).
- Changes in the dependent variables assessed during baseline and neurofeedback phases are assumed to be related to the neurofeedback.

Limitations

The limitations of this proposed study are the specificity of the research questions, type of neurofeedback training, methodology and design, and experimenter bias. Neurofeedback has been argued as having several problems in methodology related to few randomized controlled trials, small sample sizes, and few longitudinal studies that support this approach (Rojas & Chan, 2005). Within this study, all these concerns are present including assessing an individual participant rather than a whole sample, the inability of longitudinal assessment, and the lack of a control group. Further, researchers have felt that there were few neurofeedback studies that provided adequate statistical analyses of effect size changes in cognitive, behavioral, and EEG measures (Rojas & Chan, 2005). The limitations of neurofeedback research has been in large part due to preexperimental case study reports that have not utilized consistent measures that are reliable and valid for assessing the effect of neurofeedback. For this study, however, a more stringent single-case research approach that provided multiple subjective and objective measures was used. For instance, there was repeated baseline and treatment phase measures consisting of questionnaires rating symptom severity, neurocognitive testing, intelligence, and brain maps. The variety of measures allowed for an in-depth exploration of changes in overall functioning. This was the first neurofeedback LZT study to specifically evaluate broad neuropsychological changes in ASC in adulthood.

Given this was a single-case research design there are a number of methodological concerns regarding the type of analysis and threats to external validity. Visual inspection served as the primary method to determine efficacy. The use of visual inspection and identifying results in individual participants could raise the concern of

threats to internal validity. For instance, change may be the result of exposure to the assessment process in baseline and neurofeedback phases. Also, visual inspection has been historically considered largely subjective; therefore, for this study an expectation to have a more reliable means to validating findings was required. Therefore, the conservative dual criterion provided an improved visual inspection method, which has guarded against Type I and II error rates and higher power levels than statistical procedures (Fisher, Kelley, & Lomas, 2003). Additionally, external validity of the findings may have been impacted by the $N = 1$ design in the study, pretest sensitization, type of setting, and awareness of symptoms that are being assessed with the expectation of benefit from the neurofeedback (Kazdin, 1982).

Also, there was the possibility that the participant was highly motivated and had the expectation to see change through neurofeedback, which could then lead to elevated placebo responses. This was guarded against using objective measures such as the QEEG and neurocognitive measures that are not influenced by subjectivity. Finally, the participant was drawn from an inherently biased sample of European American males based on both the gender specificity in ASC and the rural area that will be sampled. The researcher encouraged the neurofeedback clinic to use nonbiased recruitment by not discriminating against any participants based on gender, race, or ethnicity. However, the participant was a European American male.

These limitations were detailed in Chapter 5 of this study. The most critical element was the ability to generalize findings to the larger ASC population. The issues with sample selection and the use of a single participant limited the exposure of the findings to other individuals with ASC. Discussing the need for larger sample studies

will be important to explore in neurofeedback LZT. In addition, the sample from the area in which it was collected was explored in more detail. This was considered a preliminary study into the role that it might play in adults with ASC. In addition, the visual inspection method might be considered a limitation in the analysis of the data if the data is not clearly depicted. For instance, the data in baseline may not have been stable, so it may have resulted in skewed regression line. Concerns related to the analysis will be important to explore for future researchers who intend to use visual inspection as a primary analysis. The principal investigator provided a minimum of five baseline measures to ensure adequate baseline and treatment phase data are provided, which provides enough data to determine significance based on the binomial formula (Fisher et al., 2003). This may assist in future research in reducing the time or number of testing administrations.

Delimitations

The scope of this study is delimited by the potential to assess neuropsychological changes associated with neurofeedback LZT and generalize findings to the larger population of adults with ASC. Single-case research avoids averaging group processes in the study, thus decreasing the risk of Type I errors (Kazdin, 1982). Further, this study provides specific information on individual changes over time using neurofeedback LZT through visual inspection analyses. Visual inspection has been argued as a more powerful way to show effects over statistical analyses that may find effects in very small changes (Kazdin, 1982). It may be more applicable to the larger population if the participant is treated as their own control with a baseline and an experimental phase clearly delineating the changes between phases (Kazdin, 1982). These phases consisted

of multiple measures including neuropsychological symptoms, autistic symptoms, neurocognitive performance, intelligence, and brain function. The repeated measures approach provided a stronger case for generalizing this research because there will be significantly more evidence to support change over time rather than simply pre and postmeasurements that are typical in between group studies.

Significance of the Study

The significance of the study lies in providing support in the literature for technological advancement of procedures that are being newly developed and implemented. Most importantly, it supports the need to address developmental deficits across the lifespan within this ASC population. A definite need has been identified to explore and validate interventions in adulthood as opposed to continuing only to provide research in earlier stages of development. This study helps support the need to explore viable interventions like neurofeedback into adulthood and may also aid in the development of research that is able to employ more stringent randomized controlled treatment studies. There have been no studies to date in research on the effects of neurofeedback LZT that utilize a quantitative approach in an adult with ASC. To date, only qualitative case studies and retrospective reports with mixed interventions (e.g., metacognitive strategies and neurofeedback combined) have been researched (Thompson & Thompson, 2003, 2010). Also, there is a great need for community mental health, hospitals and private clinics to provide and implement cutting edge interventions for ASC to more comprehensively address the complexity of the condition. Through this study, the research will promote utilization of tools to help adults with ASC optimize their life.

Social Change Implications

The social change implications for this study are twofold. First, the principal investigator's intention was to evaluate the effect of neurofeedback on neuropsychological measures. The research will help to expand the literature regarding whether neurofeedback interventions are associated with neuropsychological change in adult ASC. Second, there is a need to identify interventions that address the neuropsychological complexity of ASC in adults through behavioral and neurophysiological methods like neurofeedback. It will assist in exploring issues such as adult neuroplasticity, the ability to generalize studies from children to adults, and better understand changes associated with neurofeedback. The findings may also provide research-based rationale for insurance reimbursement consideration for neurofeedback services; neurofeedback is currently not covered by most insurance providers, forcing patients to pay out of pocket for these services.

Summary and Transition

The purpose of this single-case research study was to evaluate the effect of neurofeedback LZT on neuropsychological symptoms, core autistic symptoms, neurocognitive abilities, intelligence, and neurophysiological functioning in an adult with ASC identified through a neurofeedback clinic. The single-case research study consisted of a convenience sample of an adult participant, over the age of 18, diagnosed with ASC recruited from rural northern Michigan by a local neurofeedback clinic. The research findings will help to expand the literature regarding whether neurofeedback interventions are associated with neurophysiological change in an adult with ASC.

Chapter 2 is a review of the literature on autism such as the diagnostic background, cognitive theories, genetic and neurophysiological issues, neuroimaging and neurological patterns, prevalence and costs associated with autism and autism in adulthood. Chapter 2 is also an exploration of research on neurofeedback's theoretical background, research of neurofeedback in ASC, longitudinal research, neurofeedback LZT case reports, number of sessions to identify an effect, potential adverse effects, and the need for further research in specific areas of neurofeedback in ASC. Chapter 3 provides a background in the single-case research design, the study's setting, participant inclusion and exclusion, informed consent, confidentiality, data collection and analysis, instrumentation and materials, procedure in Phase A and Phase B, research questions and hypotheses, variables, and protection of the participant. Chapter 4 is a review of the results, and Chapter 5 includes the conclusion and future directions.

Chapter 2: Literature Review

Introduction to Literature Review

The literature review presents themes consisting of an overview of ASC with issues related diagnosis, neurocognitive deficits, neurophysiological phenotypes, prevalence rates, costs associated with level of care needs, and adult-related issues, particularly the lack of research. Next, the history, background, and efficacy of neurofeedback for treating ASC, and LZT as a specific neurofeedback approach are addressed. Research began in 2007 and continued into July 2010. Articles were derived from the electronic database EBSCO HOST in PsycINFO, PsycARTICLES, PsycBOOKS, SocIndex, Military & Government Collection, CINAHL Plus with Full Text, MEDLINE, Academic Search Complete, Academic Search Premier, and ERIC. Other collection methods included the search engine Yahoo using a modified search in advanced settings to collect only Adobe PDF files. The key terms were used in the Boolean format: "AND," "OR," or quoted text (e.g., "Asperger's syndrome"). Terms used were: *autism*, *autistic*, *spectrum*, *Asperger's syndrome*, *disorder*, *PDD*, *neurofeedback*, *neurotherapy*, *neurobehavioral therapy*, *EEG biofeedback*, *hemoencephalography or HEG*, *EEG*, *QEEG*, *fMRI*, *PET*, *blood perfusion*, *mental health*, *costs*, *financial*, *frontal lobe*, *temporal lobe*, *frontotemporal*, *epileptiform*, *epilepsy*, *dietary*, *nutrition*, *gastrointestinal*, *allergies*, *psychopharmacology*, *neuroplasticity*, *neurotransmitters*, *serotonin*, *dopamine*, *theory of mind*, *empathy*, *weak central coherence*, and *executive functioning*. Supplemental information was found from reference lists. Finally, the principal investigator also collected journal articles and texts in the area of autism and neurofeedback.

Overview of Autism Spectrum Conditions

Diagnostic Background of Autism Spectrum

According to the CDC (2009), children with ASC are most accurately identified at around age 36 months. ASC includes the DSM-IV-TR (2000) categories of autistic disorder, Asperger's disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS). The DSM-IV-TR also includes the diagnoses of childhood disintegrative disorder and Rett's disorder. Historically, these conditions were considered a form of childhood psychosis because of the similarities between schizophrenia and autism, including idiosyncratic behaviors, obsessive rumination, poor social interrelatedness, and flat affect (APA, 2000; Asperger, 1945; Kanner, 1943, 1944; Wing, 1981). Kanner (1943, 1944) was the first to identify and conceptualize ASC in 11 children who had similar deficits in social interaction, communication, and stereotypic behaviors. Later, Asperger (1945) identified similar symptoms noted by Kanner but without language delays. Nearly 40 years later, Wing (1981) further differentiated high functioning autism by identifying children who had autistic symptoms with normal language development up to age 3, a condition he called Asperger's disorder. Underlying symptoms of autism consist of social-communication deficits, repetitiveness, obsessions, stereotypies, or restricted patterns of behaviors (APA, 2000). However, by including those with and without language impairments and providing the PDD, NOS label, many researchers in the field are concerned that the DSM has resulted in an overabundance of ASC diagnosis (Volkmar & Klin, 2005). The concerns are partly due to the wide range of subjective interpretation that could result when making a diagnosis of PDD, NOS.

The APA (2010) presented proposed changes for autism in the upcoming DSM-V

with the recommendation to include all of the PDD conditions under one unified label, autism spectrum disorder (ASD). Autistic disorder, childhood disintegrative disorder, Asperger's disorder, and PDD, NOS are currently understood as a spectrum of varying levels of deficits associated with two main areas of dysfunction: social-communication and fixated interests or repetitive/stereotypic behaviors (APA, 2010). Rett's disorder will be considered a separate medical condition and not included as a part of the spectrum (APA, 2010). Although delays in language were formerly viewed as a differentiation between higher functioning and lower functioning autism, the new DSM will consider autism as a varying disorder on a spectrum rather than distinct categories between Asperger's and autistic disorder.

An additional change advocated for by APA (2010) is the age of diagnosis. Despite DSM-IV-TR expectations of a diagnosis in early childhood, social delays in ASC may present as late as adolescence (APA, 2010). Psychosocial demands are less apparent in infancy and early childhood and may only be evident at a later date when the social demands exceed the adolescent's abilities (APA, 2010). These changes in diagnostic criteria may improve the understanding of what ASC truly is.

Fundamentally, some of the core symptoms of ASC are associated with problems in nonverbal and verbal forms of social-communication and social-emotional reciprocity (APA, 2000, 2010). These symptoms are apparent in limited appropriate peer-relationships and interactions. In the other core area, symptoms are associated with idiosyncratic behaviors such as stereotypic motor mannerisms (e.g., finger-wringing), repetitive verbal behaviors (e.g., echolalia), sensory behaviors (e.g., spinning), adherence to ritualistic or routine regiments, or fixated/narrowed areas of interests (APA, 2000;

2010). Other comorbid symptoms consist of problems with poor attention, hyperactivity, self-injurious behaviors, sensory integration, aggression, abnormal eating habits, and neurocognitive deficits (APA, 2000).

Contradictory to the DSM-IV-TR, ASC is often comorbid with other symptoms found in exclusionary disorders such as obsessive-compulsive tendencies, receptive-expressive communication deficits, flat affect similar to schizophrenia, hyperactivity/impulsivity, and attention deficits (Volkmar & Klin, 2005). Despite similar attributes to other disorders, the current diagnostic practice is to not diagnose conditions like attention-deficit hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD), communication disorders, or schizophrenia in the presence of ASC (APA, 2000). Nevertheless, researchers have historically found that pharmacological interventions such as neurostimulants have been effective in treating individuals with ASC who also struggle with poor attention, difficulty concentrating, and hyperactivity (Aman & Langworthy, 2000; Tsai, 1999). There are also high rates of anxiety, depressive, and bipolar disorders among individuals with ASC, further complicating treatment course (Raja & Azzoni, 2008; Shtayermman, 2008). The current practices for differentiation recommended by the DSM have inherent problems in recognizing the complexity of ASC beyond the core symptoms with the trend of comorbidity in disorders like ADHD and OCD (Volkmar & Klin, 2005), social anxiety (Bellini, 2006), and depressive disorders (Shtayermman, 2008).

Neurocognitive Theories

ASC has been often associated with neurocognitive deficits that contribute to the autistic symptoms. Researchers have found that individuals with ASC uniquely differ

from normative samples with impediments in empathy or theory of mind (TOM) tasks (APA, 2000; 2010; Baron-Cohen et al., 2005; Baron-Cohen, Leslie, & Frith, 1985, 1986; Lawson, Baron-Cohen, & Wheelright, 2004; Leslie, 1987; Leslie & Frith, 1990; Thompson et al., 2010a), weak central coherence (APA, 2000; 2010; Happe, 2005; Thompson et al., 2010a), and executive functioning (APA, 2000; Best, Moffat, Power, Owens, & Johnstone, 2008; Hill & Bird, 2006; Kouijzer, de Moor, Gerrits, Buitelaar et al., 2009; Knezevic, Thompson, & Thompson, 2009, 2010; Koshino et al., 2005; Kouijzer, de Moor, Gerrits, Congedo et al., 2009; Lawson et al., 2004; Pineda et al., 2008; Rinaldi et al., 2008; Thompson et al., 2010a). These problems are often associated with deficits in anterior regions of the cortex, which is associated with higher cognitive abilities like emotional reciprocity, seeing the whole picture, and executive functioning (Thompson et al., 2010a). Because of these problems, individuals with ASC are in need of interventions like neurofeedback that improve neurocognitive functioning (Thompson, Thompson, & Reid, 2010b).

According to the APA (2000, 2010), one of the primary symptoms of ASC is a qualitative impairment in social interactions related to mutual interest, understanding others intentions, empathy, emotional reciprocity, and the underlying concepts of TOM. Empathizing deficits are consistent with problems in reciprocating communication, difficulty in predicting the thoughts and feelings of others, interpreting abstract emotions of others, difficulty in predicting the thoughts and feelings of others, and an appearance of social insensitivity (Baron-Cohen et al., 2005; Lawson et al., 2004). Empathy and TOM are critical issues for individuals with ASC in regard to difficulty with pretend play, creating different attributions in inanimate objects, imaging the emotions and

actions of others, or maintaining social responses to others based on mental states (Baron-Cohen et al., 1985, 1986; Lawson et al., 2004; Leslie, 1987; Leslie & Frith, 1990).

Developmentally, toddlers with ASC have problems with shared nonverbal communication and reflective facial expressions as early as 12 to 14 months (Baron-Cohen et al., 2005). Normal developing infants and toddlers from 18 to 24 months are able to understand emotional expression from others through intonation, facial expressions, and other nonverbal communication skills associated with empathy and TOM. Later in childhood, children develop a basis of early attachment through caregivers and eventually engage in larger contextual socialization outside their family of origin with peers (Premack & Woodruff, 1978; Wimmer & Perner, 1983). Even children with Down's syndrome are more capable than children with ASC in completing tasks of empathy and identifying mood states (Baron-Cohen, 1989); children with autism are unable to commit to unreal or imaginative aspects of cognition, such as drawing an unreal house (Scott & Baron-Cohen, 1996). Advanced levels of TOM include the ability to process intonation and nonverbal facial cues of emotion (Hobson, 1986 a,b), and these are specific deficits in ASC that persist from childhood into adulthood (Kleinman et al., 2001). TOM does add to the social referencing model of attachment theories and provides a perspective on ASC core symptom (Leslie & Frith, 1990).

Another major neurocognitive deficiency in ASC is described by weak central coherence (WCC; APA, 2000; 2010; Happe, 2005; Thompson et al., 2010a). The WCC theory suggests that individuals with ASC are more apt to focus on details rather than integrating information as a whole (Happe, 2005). Recall will tend to have unessential details as opposed to the whole concept of the situation (Thompson et al., 2010a), and is

likely the result of hyperfocused areas of interest or seeing only the parts rather than the whole picture (Baron-Cohen et al., 2005). Focusing on details may result in rigidity and obsessive behaviors where individuals are unable to switch attention to another area of focus, and it also accounts for the inability to understand or shift to different rules when the context changes (Thompson et al., 2010a). WCC explains the specialized skills individuals with ASC tend to have, such as memorization of numbers or musical inclination (Happe, 2005). Individuals with autism are often seen to have interest in system details and pursue careers in engineering, building, clocks, machines, puzzles, or computers, which are often obsessive interests in ASC (Baron-Cohen, et al. 2005). There is also a need for structure, routine, and regimented activity (Lawson et al., 2004).

Although TOM and WCC provide frameworks for the cognitive styles of ASC, there are few testing methods outside of checklists to evaluate them (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). Alternatively, executive functioning has a variety of testing measures that have been assessed in ASC, which have also helped to evaluate the efficacy of interventions like neurofeedback (Knezevic et al., 2009. 2010; Kouijzer , de Moor, Gerrits, Buitelaar, et al., 2009; Pineda et al., 2008). Significant problems have been found in cognitive flexibility, speed of processing, goal setting, attentional control, and other executive functioning areas in neuropsychological testing in participants with ASC (Knezevic et al., 2009, 2010; Kouijzer, de Moor, Gerrits, Buitelaar, et al., 2009). Neurocognitive impairment in executive functioning tasks like response initiation, intentionality, planning, impulse control, working memory, and cognitive flexibility have been extensively researched in ASC (APA, 2000; Hill & Bird, 2006; Kouijzer, de Moor, Gerrits, Buitelaar et al., 2009; Kouijzer, de Moor, Gerrits,

Congedo et al., 2009; Knezevic et al., 2009. 2010; Koshino et al., 2005; Pineda et al., 2008; Rinaldi et al., 2008). The diagnostic formulation for ASC identifies a high comorbidity of symptoms related to executive functioning such as attention deficit, impulsivity, mood dysregulation, and cognitive inflexibility (APA, 2000). For individuals with high functioning ASC, functional magnetic resonance imaging (fMRI) research suggests that while completing mental rotation tasks participants showed impaired performance in attention, cognitive control, and visual-spatial processing deficits compared to controls (Silk et al., 2006).

Research methods have included the Tower of London (TOL; Just, Cherkassky, Keller, Kana, & Minshew, 2007; Knezevic et al., 2009, 2010) and Test of Variable Attention (TOVA; Pineda et al., 2008). The TOL measures executive functioning through assessing problem solving skills. Individuals with ASC were impaired in their performance on the TOL and significantly different from a normative control group in functional brain imaging (Just et al., 2007). Another study using the TOL concluded that individuals with ASC were significantly impaired in planning efficiency, working memory, cognitive flexibility, and inhibition (Knezevic et al., 2009, 2010). Pineda et al. (2008) found that a sample of children with ASC performed poorly in attention and cognitive control on the Test of Variable Attention (TOVA), a continuous performance executive functioning task. There is evidence that male participants tend to have executive functioning deficits in areas like cognitive flexibility and strengths in analyzing systems and disembedding tasks (Best et al., 2004). In this regard, autism has also been considered an extreme form of the male brain. Overall, these studies exhibit significant problems related to anterior cortical processes associated with executive functioning

neurocognitive deficits.

Genetic and Neurochemical Abnormalities

Autism is a complex neurodevelopmental condition consisting of multiple factors that influence the degree of impairment of the core autistic symptoms. From genetic abnormalities (Caglayan, 2010; Cook, 1998) to irregular neurochemical processes (Anderson, & Hoshino, 2005), the heterogeneous nature of ASC has evolved over the past 20 years with advancement in neuroimaging and biogenetics technology.

Heredity has been a major factor in setting the stage for autism. The risk rates for monozygotic twins as high as 60%, and siblings having a 45 to 90 times greater likelihood of developing an ASC noted in one study (Cook, 1998). A more recent review has found that there are rates as high as 95% in monozygotic twins (Caglayan, 2010). First degree relatives were found to have psychiatric disorders such as depression, OCD, and other anxiety disorders that are associated with serotonin abnormalities (Devlin et al., 2005), which are also comorbid in ASC (APA, 2000).

Given this familial tendency, genes likely play a significant role in the development of autistic symptoms. For the past decade, independent study groups have been formed to define chromosomal abnormalities in ASC and to develop phenotypes that categorize variants on the spectrum (Cook, 1998). Genetic research reviews have concluded that several genes may account for autism, such as single chromosome abnormalities in Fragile X or Turner's syndrome (Cook, 1998), and other genetic disorders comorbid with ASC like Klinefelter, Rett, Prader-Willi, Timothy, Phenylketonuria, and Angelman syndromes (Caglayan, 2010). The X chromosome may be the key to higher rates in males compared to females in the expression of the condition

(Klin, McPartland, & Volkmar, 2005). Chromosomal abnormalities with 17q11.1-q12 impair tryptophan synthesis, resulting in hyperserotonemia and abnormal metabolic processes that are critical for normal neuronal development (Devlin et al., 2005; Tordjman et al., 2001). Abnormalities with the short allele on the serotonin transporter gene will result in increased severity of social-communication deficits (Tordjman et al., 2001). Besides serotonin, there are other polymorphisms that impair dopamine and norepinephrine transmissions on chromosome 9q34, which results in the deficiencies seen in autism (Polleux & Lauder 2004).

Genetic influences impact both neurochemical processes and neuronal development in ASC (Anderson & Hoshino, 2005). Atypical neurologic development is concordant with the dysregulation of serotonin, dopamine, hypothalamic-pituitary-adrenal functioning, and complications can arise from chemicals like exorphins (Anderson & Hoshino, 2005). Neurochemical aspects of ASC were researched as early as 50 years ago by Schain and Freedman (1961), who identified abnormally high levels of serotonin or hyperserotonemia in autism with up to one-third of the ASC sample having this abnormality (Anderson, Horne, Chatterjee, & Cohen, 1990). Hyperserotonemia has been found to trigger an autoimmune response that results in high titers of autoantibodies furthering abnormal neuronal development (Burgess, Sweeten, McMahaon, & Fujinami, 2006). Children with ASC have been found to have elevated epinephrine and norepinephrine plasma levels, and lower platelets for epinephrine, norepinephrine, and dopamine (Launay, Burszteijn, Ferrari, & Dreux, 1987). The abnormal levels of neurotransmitters may result in the comorbid symptoms of ASC: hyperactivity/impulsivity, inattention, neurocognitive deficits, poor motor control,

perceptual distortions, obsessiveness, and social cognitive problems (Anderson & Hoshino, 2005; Ernst, Zamatkin, & Lancet, 1997). Other neurochemicals related to ASC consists of components of the neuroendocrine system including glucocorticoid cortisal, gamma-aminobutyric acid, histidinemia, and phenylketonceria (Anderson & Hoshino, 2005). In addition, oxytocin has been identified as being dysregulated in ASC as well, and is related to the underlying problems in early attachment and social bonding (Hollander et al., 2007).

With these genetic and neurochemical influences, the underlying structural development of the brain in autism is associated with problems such as accelerated neuronal development in the frontal lobe in as early as 28 weeks of life (Minshew et al., 2005). Because serotonin is critical for creation of synapses and neuronal differentiation, hyperserotonemia in autism leads to irregular sleep, body temperature, appetite, hormones, mood, and diminished neuroplasticity (Anderson & Hoshino, 2005; Chugani, 2002; Tsai, 1999). Specifically, serotonin synthesis from ages 2 to 11 has been calculated to be around 1.5 times more than typical adult levels of serotonin, which ultimately hinders growth in thalamocortical, sensory cortices, and subcortical structures such as the hippocampus and amygdala; these all are critical for communicating with the cerebral cortex in social memory and social language processing (Baron-Cohen et al., 1999; Chugani, 2002). Abnormal white matter and pyramidal cell growth are also apparent within the corpus callosum, left planum temporal, left inferior prefrontal gyrus, frontal lobe, hippocampus, medial nucleus septum, and mamillary body (Minshew et al., 2005). The frontal lobe demonstrates abnormal neural connectivity throughout the cortical and subcortical areas in particular (Coben, 2009b; Minshew et al., 2005).

Neuroimaging Patterns in ASC

Over 20 years ago, researchers speculated that autism is related to impaired neural connectivity resulting in functional deficits in cortical and subcortical information processing (Horwitz, 1988). Only in recent years has research confirmed the neural connectivity hypothesis through measuring metabolic processes with positron emission tomography (PET) or single photon emission computed tomography (SPECT; Ohnishi et al., 2000) as well as cerebral blood flow (CBF) in real time using fMRI (Wicker et al., 2008). These approaches provide indications of abnormal glucose metabolism, hyperperfusion (i.e., excess blood flow), or hypoperfusion (i.e., diminished blood flow). Such research is critical for evaluating the phenotypes in ASC and for establishing the need to offer interventions associated with regulating these processes (Coben & Myers, 2008).

Extensive research has been conducted in exploring deficient CBF especially within and around the frontal lobe (Limsila et al., 2003; Ohnishi et al., 2000; Chandana et al., 2005). ASC specifically has abnormal CBF within the medial prefrontal cortex, anterior cingulate gyrus, and right medial temporal lobe, which reflect deficits in TOM, obsessive behaviors, and need for sameness (Ohnishi et al., 2000). Further, autism spectrum is differentiated by abnormal CBF in the bilateral insula, superior temporal gyri, and left prefrontal cortices suggestive of global cognitive impairments in language, executive functioning, and sensory integration. In a large sample (n = 117) of children with ASC, researchers found that these children had abnormal PET scans indicative of serotonin synthesis in the right and left hemispheres with no asymmetry in

temporoparietal lobes associated with severity of language impairments (Chandana et al., 2005). Chandana et al. (2005) suggested that this results in the development of disorganized microcircuitry and more tightly packed columns impeding neurotransmission.

There has been a growing body of neuroimaging research in autism using fMRI to evaluate the neuronal pathways for problems related to social-cognitive abilities like TOM (Baron-Cohen et al., 1999; Iacoboni & Dapretto, 2006; Koshino et al., 2005; Rinaldi, Perrodin, & Markram, 2008; Silk et al., 2006; Vollm et al., 2006; Welchew et al., 2005). fMRI imaging has explored the connectivity theory in people with ASC and it has been found that when presented with emotional expressions there is indication of abnormal functional connectivity in medial temporal lobe areas, specifically in the amygdala, hippocampus, and parahippocampal gyrus (Welchew et al., 2005). Other fMRI research suggested that there is hypoconnectivity or lower synchronization amongst anterior regions and increased processing information in the right (Koshino et al., 2005). This right side processing is opposed to the primarily left side processing in normative controls (Koshino et al., 2005). In the prefrontal cortex, hyper-connectivity has been found that may lead to deficits in higher order functioning such as with socialization, attentional deficits, and cognitive inflexibility or repetitive behaviors (Rinaldi et al., 2008). Adults with ASC utilize primarily linguistic and memory functions when processing nonverbal forms of communication rather than emotional centers in the brain such as the amygdala or left prefrontal region (Baron-Cohen et al., 1999). Wicker et al. (2008) also explained that there was hypoconnectivity between the ventrolateral and dorsolateral prefrontal cortices, and superior temporal sulcus which is critical for

attentional emotional processing as well as affective emotional expression.

TOM and empathy are identified in areas of the medial prefrontal cortex, temporoparietal junction, and middle and inferior temporal gyri (Vollm et al., 2006). The same research has found differences between TOM and empathy where empathy activated the cingulate and amygdala and TOM activated the orbitofrontal cortex, middle frontal gyrus, cuneus, and superior temporal gyrus. Empathy was linked to frontal lobe areas such as with Broca's area or pars opercularis and bilateral dorsal and ventral premotor areas (Leslie, Johnson-Frey, & Grafton, 2003). In another study on TOM, there was bilateral damage to the orbito-frontal cortex resulting in deficits in more complex TOM tasks like faux pas or mistakes in TOM, apparent particularly in high functioning ASC (Stone, Baron-Cohen, & Knight, 1998). Further, individuals with unilateral damage did not show deficits with TOM tasks. Lastly, sociopaths and ASC, although both sharing underlying problems in empathy, are distinguished in fMRI literature by the differences in processing TOM with sociopaths having no perceived deficit in orbitofrontal cortex and temporopareital cortices (Blair, 2008).

In high functioning autism, the research on fMRI functional connectivity has found less activation in lateral and medial premotor cortex, dorsolateral prefrontal cortex, anterior cingulated gyrus, and caudate nucleus when undergoing mental rotation tasks, which is a result of problems in executive functioning and working memory (Silk et al., 2006). The caudate nucleus is the link to frontoparietal networks for attention, cognitive control, and visuospatial processing (Silk et al., 2006). In another article, individuals with high functioning autism demonstrated hypoconnectivity in frontoparietal areas when completing the Tower of London task, and the functional deficiency is linked to the high

occurrence of decreased size of the genu in the corpus callosum (Just et al., 2007). Adults with high functioning autism or Asperger syndrome compared to a normative sample were significantly different in processing of angry and happy faces compared to a normative sample in a recent study using a 3-T whole body imager, a type of fMRI (Wicker et al., 2008). Wicker et al. (2008) showed a lack of activation in the dorsomedial prefrontal cortex and right ventrolateral prefrontal cortex in individuals with ASC. Those areas are associated with comprehending social significance of emotional facial features. The medial prefrontal region is particularly an important integrator of information from cortical and subcortical systems like the amygdala, another deficient area of ASC (Wicker et al., 2008). In addition, the occipital cortex showed little interaction in the fusiform gyrus, an area that is responsible for social/emotional perceptual networks (Wicker et al., 2008).

One specific theory explaining the main deficit in autism has been the mirror neuron system, which influences social cognitive functioning in areas such as nonverbal and social communication (Iacoboni & Dapretto, 2006). Iacoboni and Dapretto (2006) provided a thorough overview of the mirror neuron system and its relation to TOM. They point to the interconnections between the superior temporal cortex, inferior parietal cortex, and inferior frontal cortex through white matter tracts alongside the arcuate fasciculus. Frontal and parietal network, particularly the agranular frontal cortex, provides the basis for movement of body parts into actions (Iacoboni & Dapretto, 2006). Mirror neuron circuitry within the frontal regions is found at the inferior frontal gyrus and ventral premotor cortex, which are interconnected with the inferior parietal cortex. The connections also take place at the posterior superior temporal sulcus, creating the core

circuitry for imitation. The frontal mirror neuron system is important for the "goal of the action" (p. 943). The pars opercularis found within Broca's area is the location of a large majority of the MNS activity, and suggests the evolutionary basis for language in imitation and social interconnectedness (Coben, 2009b; Iacoboni & Dapretto, 2006). The pars opercularis is important for reflecting and predicting an observed model's movement. Connectivity with the temporal, parietal, and frontal networks is critical for imitative learning and social mirroring. The MNS network, amygdala, and insula are critical for the complex sensorimotor processing especially when interpreting and understanding the intentions of others as well as the perception of self. The neural substrates for TOM consist of fronto-temporal, supplementary motor, and bilateral temporal and parietal areas (Baron-Cohen et al., 1999). There is also a subcortical involvement found in the left sides of the amygdala, hippocampal gyrus, and striatum as well as a bilateral involvement in the insula.

Neurological Patterns in ASC

Quantitative EEG (QEEG) is the statistical analysis of raw EEG data through comparison of the EEG spectrum (e.g., theta/beta ratios of 3:1 or greater indicative of ADHD) or comparative normative databases in order to identify standard deviations of brainwave activity (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007). It may either be represented in the form of color-coded maps of the 10-20 sites or else in statistical quantitative data utilizing a measure such as z-scores or standard deviations for comparison with a normative EEG sample. QEEG has been argued to be the most effective method of assessing brain function and differentiating autism with normative groups in its evaluation of seizure disorders, abnormal EEG oscillations such as inability

to suppress mu rhythms, connectivity irregularities, and elevated theta and delta waves (Coben, 2009b). Also, QEEG has been used to differentiate ASC and control groups with up to 95.2% accuracy, and these differences were consistent over a 3-month period (Chan & Leung, 2006).

The functional neuroimaging approaches previously noted are considered invasive procedures over QEEG because they require injections, consumption of radioisotopes, or exposure to radiation in order to assess brain function (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007). In addition, although the use of fMRI is less invasive than SPECT or PET, the equipment is limited to high-tech research labs and can cost hundreds of thousands of dollars and even more if there is the use of higher field magnets which increase sensitivity (Wilkie, 2009). Also, the temporal resolution of fMRI is limited due to the problem that blood oxygenation changes within seconds whereas thought processes change within milliseconds (Wilkie, 2009). QEEG is the least invasive measure for brain function, has the best temporal resolution, and is more readily available because of the affordability and portability of the equipment and software (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007). In addition, the portability has special implications for outpatient clinical settings or remote rural areas far from hospitals or universities where access to technologically advanced equipment is limited.

In an early case study on QEEG in ASC, the researchers found that an adult with autism was found to have higher amplitude brainwave activity in the right anterior area suggestive of behavioral symptoms associated with aprosodia of speech, impulsiveness, and difficulty with social behaviors (Harrison, Demarre, Shenal, & Everhart, 1997). The findings indicated that individuals with ASC may exhibit higher amplitudes in delta,

theta, alpha, sensorimotor, and beta, as well as higher theta/beta ratios, and unstable absolute amplitude. Another QEEG study found high delta and low alpha power, which was able to differentiate autism from a normative population (Chan, Sze, & Cheung, 2007).

In addition to abnormal EEG oscillations and localization, connectivity measures are driving much of the EEG research on autism providing a much more complex understanding of the condition (Coben, 2009a,b; Coben & Myers, 2008; Coben & Myers, 2010; Coben & Padolsky, 2007; Minshew et al., 2005; Thatcher et al., 2008). One thought is that neural hypoconnectivity is in part due to reduction in the corpus callosum, left planum temporal lobe, inferior prefrontal gyrus (Minshew et al., 2005). Further, the corpus callosum may play a critical role in connectivity in individuals with ASC because of its physiological basis for connectivity in brain function (Coben & Myers, 2008; see also Coben, 2009b; Just et al., 2007). In a study consisting of 54 children with ASC, researchers found significantly shorter phase shift duration particularly for alpha 1 (8-10 Hz) and longer phase lock duration in alpha 2 (10-12 Hz) in the occipitoparietal regions (Thatcher et al., 2008). The study reflected prior research that children with ASC have reduced thalamo-cortical connections attributed to GABA inhibitory deficiencies. Coben and Myers (2008) presented cases and summarized research suggesting that ASC consists generally of hyperconnectivity in frontotemporal and left hemisphere intrahemispheric regions and hypoconnectivity in orbitofrontal, right posterior, frontal-posterior, and left hemispheric regions. These areas represent executive functioning, social reasoning, emotional recognition, social pragmatics, and informational processing. Coben and Myers suggested phenotyping subtypes of ASC utilizing QEEG connectivity measures

rather than simply basing the diagnosis on subjective reports or observations.

Researchers summarizing EEG phenotypes in individuals with ASC have been complex, with issues like a lack of interhemispheric communication (Thompson & Thompson, 2003) and epileptiform activity particularly in temporal regions (Hughes & Roy, 1999; Minshew et al., 2005). Children with ASC were identified as having high rates of at least 32% of EEG recordings showing epileptiform activity (Akshoomoff, Farid, Courchesne, & Haas, 2007), and in a review of EEG subclinical epileptic activity (i.e., no behavioral observations of seizures) in individuals with autism, studies showed a high occurrence of seizures from 20-30% on average and epileptiform activity ranging from 10.3% to as high as 72.4% (Kagan-Kushnir, Roberts, & Snead, 2005). Kagan-Kushnir et al. (2005) suggested a definite neurological basis for treating ASC through neurofeedback in addressing EEG abnormalities such as epileptiform activity.

Other researchers have found EEG patterns for anterior sites that are asymmetric in children with high functioning autism (Sutton et al., 2005). Specifically, those with right frontal asymmetry were more socially aloof and less capable of managing social interaction, but the intent and motivation was considered more active. On the other hand, children with greater left midfrontal activity had higher social anxiety and more withdrawn due in part to anxiety (Sutton et al., 2005). Sutton et al. (2005) explained that this is in contrast to other research that suggests right hemispheric asymmetry, rather than left, is more suggestive of anxiety. Specifically, these EEG patterns may suggest the need to address frontal lobe asymmetry in order to accommodate for anxiety and social motivation issues common in ASC. One example of this has been an intervention study that found hypercoherence frontally with lower frequency bandwidths in children with

ASC (Pineda et al., 2008).

Another area that has been gaining attention in ASC is that of the mirror neuron system (MNS), which requires functional connectivity between left and right hemispheres (Iacobini & Dapretto, 2006). The MNS is important in imitating and performing social interactions in the frontal and parietal regions of the cortex. The inferior frontal cortex and connections to the superior temporal sulcus through the arcuate fasciculus is the connectivity of the mirror neuron system and considered the network important for social imitation (Aziz-Zadeh, Koski, Zaidel, Mazziotta, & Iacoboni, 2006). The F5 site, left frontal lobe, shows a unique Mu wave activity which is thought to be consistent with ASC mirror neuron system, which impacts imitation of observed behaviors and emotional affect (Coben, 2009b), but other researchers have found C4 as being linked with mu rhythm (Oberman et al., 2005). Bernier, Dawson, Webb, and Murias (2007) found that adults (n = 15) with high functioning ASC had reduced attenuation of Mu rhythms when observing movement, indicative of problems in imitation abilities. The mu rhythms are a sensorimotor processing function of frontoparietal networks suggestive of mirror neurons, which are suppressed during self or observed movements, and mu suppression has been found in individuals with ASC to be typically present only in self movement and not in observed movements, indicating that there is a disconnect between the mirror neuron and sensorimotor systems (Coben, 2009b; Oberman et al., 2005; Pineda, 2005). Mu suppression in children with ASC has been found to be improved through 15 hours of neurofeedback (Pineda et al., 2008). Other case reports identified individuals with ASC as consisting of high slower wave amplitudes areas of the brain that are associated with Asperger syndrome, and associated

Mirror Neuron System (Thompson & Thompson, 2003).

Prevalence and Costs

Prevalence rates have been looked at closely in ASC, as opposed to observing simply incident rates, since ASC is considered a long-term disorder that is better assessed in specific time points and places (CDC, 2007, 2009). Initially, the prevalence for ASC was rare, with approximately .4 to .5 per 1000 children identified in 1985 (CDC, 2007). From 1991 to 1999, the CDC (2007) identified a 500% increase in the prevalence rates of ASC. To address the problem of varying and questionable survey methods, the CDC (2007) established stringent methodological criteria over a broad multisite review of ASC, and the CDC developed the Autism and Developmental Disabilities Monitoring (ADDM) Network to oversee the consistency of diagnostic formulation and reporting of prevalence amongst 8 year old children. The ADDM data concluded in 2002 that 1 in 152 children are diagnosed with ASC. From 2002 to 2006, they continued this evaluation across 11 of the 14 ADDM sites areas in the United States finding 2,757 of 307,790 or 1 in 110 children diagnosed with ASC, which represents an average increase of 57% since 2002 (CDC, 2009). These results also varied from site to site, with New Jersey having the highest prevalence rate of 1 in 100 children being diagnosed with ASC. Furthermore, there was a 60% increase in boys and a 48% increase in girls with a male to female ratio of 4.5:1. The cause for the increase has been debated in research and has not come to a specific cause (CDC, 2009). One rationale has been that the label PDD from the DSM has allowed for a wide opening of variants of autism, which have led to this increase (Volkmar & Klin, 2005). Higher functioning forms of autism like Asperger's syndrome has only been identified as a mental disorder since the DSM-IV in 1994. Therefore,

many children that had milder symptoms of autism and high educational performance were not identified as having autism.

The cost of treating ASC has been estimated at around $35 billion per year nationwide to cover expensive interventions and educational needs (Ganz, 2006). For each individual diagnosed with ASC, this represents approximately $3.2 million in costs over the course their lifetime including $29,000 per year for medical treatments associated with comorbid medical conditions, behavioral therapy, and medication (Ganz, 2006). Medical costs alone range from $4,110 to $6,200 per year (Shimabukuro, Grosse, & Rice, 2008). These costs can rise to $43,000 per year when including severe forms of ASC and expenses associated with special education and child care (Ganz, 2006). Despite these high rates of expenditures in providing assessment and interventions for ASC, Ganz (2006) showed a disparity in ASC funding of $100 million per year compared to other developmental disabilities such as cognitive impairments where government spends close to $51 billion.

Autism in Older Adolescence and Adulthood

The majority of research on ASC intervention research has been conducted primarily in infancy to early childhood with minimal research in adolescent, adulthood, and elderly populations (Coben et al., 2010; Roy et al., 2009; Wolf & Paterson, 2010). Because it is a neurodevelopmental disorder according to the APA, individuals with ASC continue to have problems beyond early childhood and fall further behind peers with limited access to gainful employment and specific work abilities that restrict them (Shea & Masibov, 2005). Further, autism is a life-long condition that continues into adulthood at some level (Wolf & Paterson, 2010), and individuals with ASC continue to show

neurological deficits compared to adults without ASC in processing of social-communication (Baron-Cohen et al., 1999). Many adults with ASC will need supported housing or live within group home settings, separated from mainstream society despite the fact that some of these individuals demonstrate average to above average intellectual abilities (Shea & Masibov, 2005). Individuals with ASC are often unable to maintain gainful employment or stable relationships and adults with this diagnosis are more likely to be victimized due to their social cognitive deficits (Shea & Masibov, 2005). Despite all these challenges, research for adults with ASC is minimal, especially in regard to empirically supported interventions that may offset ongoing support services throughout a lifetime (Wolf & Paterson, 2010). Nevertheless, there are researchers beginning to explore areas for interventions in adulthood particularly in employment (Howlin, Alcock, & Burkin, 2005).

Neurofeedback Background

Overview of Electroencephalography

Neurofeedback begins with understanding the utility of electroencephalography in measuring and changing brain function. Berger (1929) conducted the first human EEG and was the first researcher to analyze the raw EEG through a statistical procedure called fourier transform, the origin of quantifying EEG (i.e., QEEG) through mathematical analyses (for history, see Thatcher & Lubar, 2008). Electroencephalography neurology has been widely beneficial across assessments and interventions in neurological conditions (e.g., Demos, 2005; Hughes & Roy, 1999; Rowan & Tolunsky, 2003; Thompson & Thompson, 2003). A review of literature has found that the EEG has correlated with neuroimaging approaches that assess brain perfusion or cerebral blood

flow (Gunkelman & Johnstone, 2005). Although EEG measurement occurs from the surface of the outer cortex, frequency bandwidths of the EEG spectrum also are indicative of subcortical electrical activity (Hughes & Roy, 1999). Specifically, alpha rhythm is associated to pacemaker neurons projected from the thalamus, theta is produced primarily from GABA release within the nucleus reticularis, delta waves correlate with oscillator neurons within the thalamus, and beta waves are produced from cortical as well as thalamocortical electrical activity during higher information processing (Hughes & Roy, 1999).

The 10-20 International System of Electrode Placement is the standard of EEG sensor placements identified through skull landmarks (e.g., nasion and inion) for initial measurements and determining 19 sites through 10% and 20% of the total measurement across the sagittal, coronal, and horizontal planes (Demos, 2005; Hughes & John, 1999; Jasper, 1958; Rowan & Tolunsky, 2003; Thompson & Thompson, 2003). The placements consist of each region of the cerebral cortex including the frontal (F sites), sensorimotor (C sites), temporal (T sites), parietal (P sites), and occipital (O sites) cortices. Even numbers are associated with right hemisphere locations, odd numbers are associated with left hemisphere locations, and z is the zero line associated with the central split between left and right hemispheres. Figure 1 below provides the EEG site locators: the 10-20 sites.

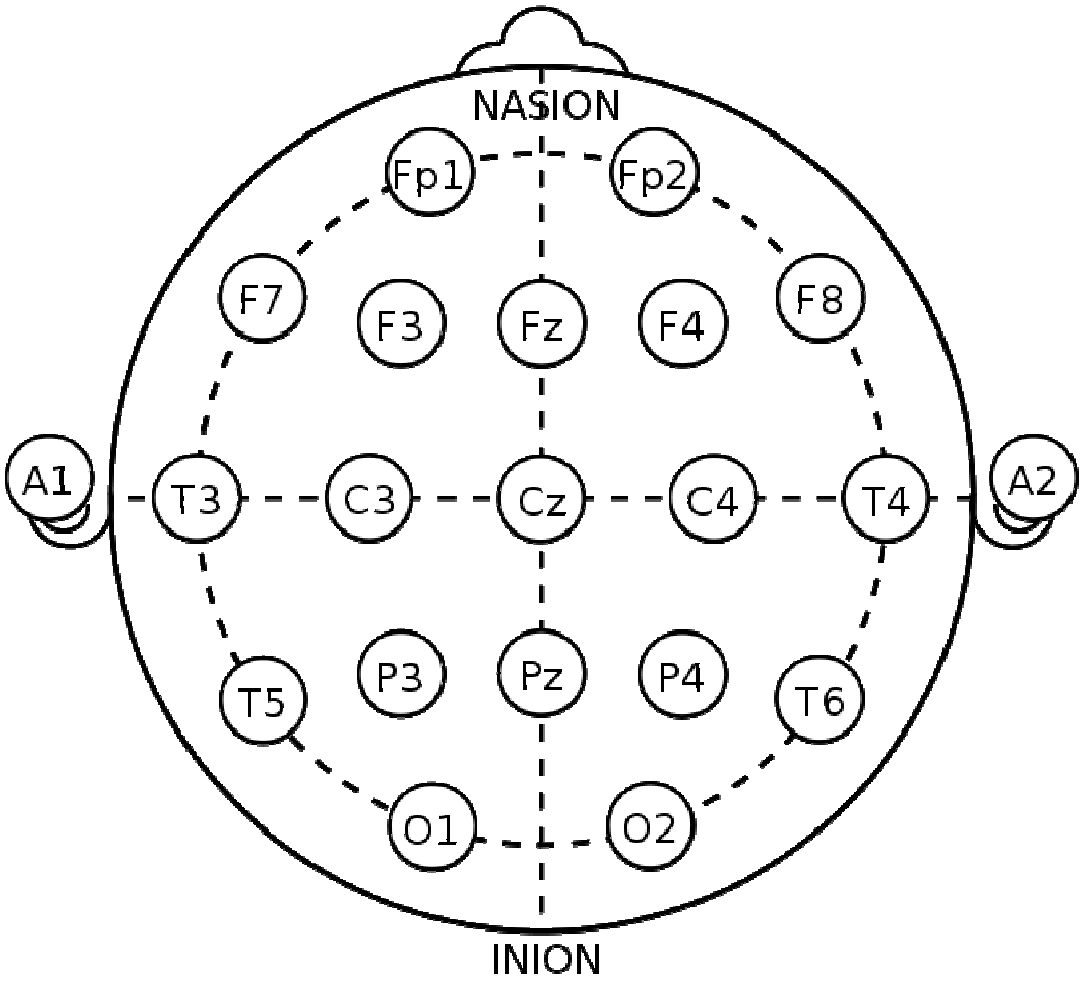


Figure 1. The 10-20 System (permission by Wikipedia, 2010).

Sensor sites record brainwave frequencies measured by hertz (Hz), cycles per second, and amplitude in microvolts (μV), height of the wave (Demos, 2005; Rowan & Tolunsky, 2003; Thompson & Thompson, 2003; Townsend, 2007). Active sensors measure the sites noted above, and in addition to active sensors, there are reference placements that help to cancel common extraneous electrical noise like electromyography. For the purpose of this paper, bandwidth frequencies in Dynamic Link Library (DLL) database consists of: Delta = 1-4 Hz, Theta = 4-8 Hz, Alpha = 8-12 Hz, Alpha 1 = 8-10 Hz, Alpha 2 = 10-12 Hz, Beta = 12-25 Hz, Beta 1 = 12-15 Hz, Beta 2

= 15-18 Hz, Beta 3 = 18-25 Hz, Gamma 1= 30-35 Hz, Gamma 2 = 35-40 Hz, and Gamma 3 40-50 Hz (Collura, Thatcher, Smith, Lambos, & Stark, 2009). However, it should be noted that depending on the database for EEG software frequency bandwidths will vary (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007).

Presently, EEG acquisition is the least invasive and least costly compared to all other current assessments of temporal brain function (Demos, 2005; Gunkleman & Johnstone, 2005; Thompson & Thompson, 2003; Wilkie, 2009). PET and fMRI scans are typically only available in larger hospitals and university centers. On the other hand, EEG is more readily available due to a broad range of practitioners due to the affordability and decreased exposure to neurochemicals and radiation. In addition, it has the best temporal resolution of all the neurological assessments with relay of information within milliseconds (Demos, 2005; Gunkleman & Johnstone, 2005; Thompson & Thompson, 2003).

See Appendix A for information on each frequency wave bandwidth, description of cognitive states, function, morphology, and disorders associated with each wavelength (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007; Wikipedia, 2005).

Theoretical Background for Neurofeedback

Neurofeedback is essentially EEG biofeedback, and allows individuals to learn to modify brainwave activity to alter and improve states of cognitive processes such as alertness, attention, calmness, internal focus, or flexibility (Demos, 2005; Thompson & Thompson, 2003). The theoretical basis for neurofeedback comes from the Law of Effect and learning theories that propose that rewarding a specific behavior will increase the likelihood of that behavior occurring again (Thompson & Thompson, 2003). With the

Law of Effect, learning theories such as operant conditioning have found that successive approximations toward a desired behaviour through positive reinforcement will increase the likelihood of the behaviour reoccurring (Skinner, 1935, 1937, 1948, 1950). Conditioning is the influence of changing the direction of behavior through a reinforcing stimulus that is temporally related with the order of stimulus and reward strengthened through correlation or contingency (Skinner, 1950). The operant conditioning paradigm set the implications that contingent reinforcement is the most basic form of behavior even before classical conditioning (Skinner, 1935). Initially, the process of neurofeedback presents much like the incidental learning of pigeons trained with superstitious behaviors (Skinner, 1948), and conditioning maybe completed through complex operant behaviors that result in reinforcement through successive approximation (Skinner, 1937).

Neurofeedback may involve other learning approaches, such as classical conditioning that influence the improvement of brain function. Thompson and Thompson (2003) suggest that through neurofeedback the desired brain state becomes a conditioned response over time in completing homework assignments. However, neurofeedback is based primarily on operant conditioning through auditory and/or visual rewards that result when EEG frequencies reach specified amplitude thresholds (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007). The temporal relationship between EEG patterns and auditory/visual rewards successively approximates the brain behavior toward increased performance. The individual who participates in training becomes increasingly self-aware of what brain behaviors are expected and is also able to generalize this learning to real-life situations such as in school or work (Demos, 2005; Thompson & Thompson, 2003). Neurofeedback may be summarized as an intervention

or training technique that helps individuals to learn to modify neural activity in order to balance arousal levels and self-awareness of various cognitive states (Demos, 2005; Thompson & Thompson, 2003). There are disorders like ADHD that have specific EEG phenotypes identified as having theta/beta ratios greater than 3:1 in frontocentral regions associated with inattention and poor concentration, and suggest the need to inhibit slow wave frequencies while increasing sensorimotor and Beta 1 (Demos, 2005; Thompson & Thompson, 2003).

There was a need to provide a formal definition of neurofeedback in order to express to the general public and other professionals what neurofeedback is and how it works. The International Society for Neurofeedback and Research (ISNR) (2009) provided this definition for consistency in the literature and research on neurofeedback:

> Like other forms of biofeedback, neurofeedback training (NFT) uses monitoring devices to provide moment-to-moment information to an individual on the state of their physiological functioning. The characteristic that distinguishes NFT from other biofeedback is a focus on the central nervous system and the brain. NFT has its foundations in basic and applied neuroscience as well as a data-based clinical practice. It takes into account behavioral, cognitive, and subjective aspects as well as brain activity. NFT is preceded by an objective assessment of brain activity and psychological status. During training, sensors are placed on the scalp and then connected to sensitive electronics and computer software that detect, amplify, and record specific brain activity. Resulting information is fed back to the trainee virtually instantaneously with the conceptual understanding that changes in the feedback signal indicate whether or not the trainee's brain

> activity is within the designated range. Based on this feedback, various principles of learning, and practitioner guidance, changes in the brain patterns occur and are associated with positive changes in physical, emotional, and cognitive states. Often the trainee is not consciously aware of the mechanisms by which such changes are accomplished although people routinely acquire a 'felt sense' of these positive changes and often are able to access these states outside the feedback session. (para. 1-2)

Neuroplasticity and Neurofeedback

The neuroscientists Ernesto Lugaro in 1909 and Jean Demoor in 1896 were the first to explore the central nervous system as being plastic and the ability to regenerate and grow new neuropathways (Jones, 2004). Neuroplasticity has been developed in neurotransmission for excitatory and inhibitory pathways such as with GABA and flutamate (Gynther, Calford, & Sah, 1998). Cognitive retraining, pharmacotherapy, stimulating environments, and other approaches may actually regenerate and promote growth in neuronal connectivity through increasing dendrites and creating larger synapses (Beauregard & Lévesque, 2006; Jones, 2004; Malkowicz & Martinez, 2009; Pinel, 2008). Further, neuroplasticity proves that adulthood is not the end to the development of neuropathways; rather it is a lifelong process (Jones, 2004). It is particularly important for adults with ASC who tend to have less brain weight and abnormal brain circuitry that develops well into adulthood (Minshew et al., 2005).

Kaiser (2008) identified that connectivity patterns increased from ages five to 35 coinciding with increased anterior myelination in the brain in normally developing adults. It may be a critical basis for ongoing interventions for individuals with

neurodevelopmental disorders to improve their level of functioning through neurofeedback. Malkowicz and Martinez (2009) explained that modifying the thalamocortical oscillatory EEG activity is an indication of neuroplasticity. Malkowicz and Martinez explain that the process of neurofeedback results in changes of EEG activity which is related with functional aspects of the brain such as neuromodulation of neurotransmitters, metabolic activity, and other processes related to structural changes as well.

Lévesque, Beauregard, and Mensour (2006) conducted one of the only studies to look at the effects of neurofeedback through fMRI. The researchers found significant changes in children with ADHD after neurofeedback sensorimotor rhythm (SMR) training with significant increases in metabolic activity in the striatum. Furthermore, there are long-term effects of neurofeedback in children with ASC on improving behaviors and neuropsychological functioning, which is suggestive of structural long-term changes as opposed to short-term treatment effects (Kouijzer et al., 2009a; Coben, 2009a). It would logically follow that research in neurofeedback during adulthood is necessary to explore efficacy in improving neurocognitive functioning, because it may be another opportunity to improve and promote neuroplasticity especially in adults with ASC.

Brief History of Neurofeedback

The history of neurofeedback has several major ground breaking research studies that built upon each other identifying that brain behavior can be modified based on the operant learning approaches. In 1963, Joseph Kamiya opened the door to neurofeedback by demonstrating that people can change their brain waves through alpha enhancement

training, which was not thought to be possible (Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007). However, by 1968 at the University of California Los Angeles, Maurice Barry Sterman went further than merely controlling brainwave patterns through the application of neurofeedback in the medical arena. He developed the concept of sensorimotor rhythm or SMR, which is the frequency of 12-15 Hertz. Through operant conditioning, he trained 10 cats to increase SMR activity. He was later asked by NASA to study the exposure of hydrazine or rocket fuel. For the experiment, he gathered 50 cats including the 10 SMR trained cats, and all 40 of the cats that were not trained in SMR had seizures, while the remaining 10 were seizure resistant. He later provided operant conditioning to increase SMR in human patients with epilepsy and found that it decreased the frequency, severity, and duration of seizures. Sterman, without foresight, had inadvertently stumbled on the remarkable benefits of neurofeedback (Demos, 2005; Thompson & Thompson, 2003).

Neurofeedback was more thoroughly explored in the 1960s and 70s when it was identified that individuals could both control specific frequencies and identify mental states associated with the frequencies (Hammond, 2006). Beta has been identified as an outward focus, attention, and concentration; alpha is seen as a state of relaxation, idling, and disengaged; theta is viewed as a day-dream state and inefficient mental processes; and delta are typically experienced in sleep. Through the use of computer technology, an individual is able to view the changes in brainwave activity during different states of mind and identify ways to manipulate them through coaching and practice improving cognitive efficiencies, flexibility, resting, awareness, and control (Hammond, 2006). In order to make substantial change in EEG activity, neurofeedback requires anywhere from

10 to 60 sessions (Hammond, 2006). The brain consists of short and long-range connections throughout subcortical structures, primarily the thalamus, and across a number of cortical centers, synchronization of pyramidal cells (Collura, 2008). Normalizing EEG power and connectivity is considered the most validated approach in neurofeedback to date (Collura, 2008). The connectivity measures associated with LZT training and considered the most frequently used are phase, coherence, and asymmetry (Collura, 2008).

Efficacy of Neurofeedback in Autism Spectrum Condition

Case Study Research

There have been a number of researchers who have explored neurofeedback as a viable intervention for ASC through case study research (e.g., Beaumont & Montgomery, 2005; Coben & McKeon, 2009; Collura et al.,, 2010; Cowan & Markham, 1994; Othmer, 2007; Rutter, 2009; Sichel, Fehmi, & Goldstein, 1995; Thompson & Thompson, 2003a, b; Thompson et al., 2010b). The first publications on neurofeedback efficacy for treating ASC was by Cowan and Markham (1994) regarding an 8-year-old girl with high functioning autism. They found elevated alpha-theta wave amplitudes in the parietal and occipital lobes, and set up training with a bipolar montage inhibiting theta-alpha (4-10 Hz) ratios and rewarding beta (16-20 Hz). The girl showed observed improvements in autistic behaviors, increased attention, and improved social and academic functioning. Many other clinicians have reported protocols similar to Cowan and Markham's such as Beaumont and Montgomery (2005) with a 7-year-old with ASC in inhibiting theta (2-8 Hz) and rewarding beta (16-20 Hz). They conducted 33 sessions and identified gains within 17 sessions, specifically with normalization of QEEG data. The authors also

noted improved parental reports according to the Childhood Autism Rating Scale, Autism Behavior Checklist, and Vineland Adaptive Behavior Scales.

Other neurofeedback investigators such as Thompson and Thompson (1995, 2003a, 2003b), Linden (2004), and Othmer (2007) have reported case studies indicating that neurofeedback is effective improving attention, behavioral problems, socialization, sleep, obsessive symptoms, speech, and sensory integration for individuals with ASC. At the Annual Meeting of the Association for Applied Psychophysiology and Biofeedback, Thompson and Thompson (2003a) presented 60 case reviews of individuals with ASC with training in frontal and parietal sites especially right hemispheric training for individuals with high functioning ASC. Thompson and Thompson (2003b) presented case details of four children and adults diagnosed with ASC receiving 40-100 sessions with improved neuropsychological functioning. One of the cases was a 13 year-old boy with ASC who received training on the sensorimotor cortex (i.e., C2 and C4) with rewarding 13-15 Hz and inhibiting 3-10 Hz. The authors reported that the child improved in emotional regulation, decreased anxiety and impulsivity, and improved educational performance with sustained results in an eight year follow up. Othmer (2007) presented positive results in case study research with neurofeedback in children who have ASC that led to decreased need for special education services and autistic symptoms through training SMR and calming overall arousal in the right hemisphere and frontotemporal lobes for stabilizing epileptiform activity and social cognition (e.g., P4, T3, T4, Fp1, F2). The sessions ranged from 28 to close to 100 sessions of 20-30 minute training.

The first peer-reviewed article published reported on an 8-year-old boy with mild

autism who showed observed improvement in behaviors and movement toward normalization of brain function (Sichel et al., 1995). Utilizing 19-site QEEG measures, they found theta to beta ratios greater than three similar to profiles of ADHD, and focused on reducing theta and rewarding SMR along the sensorimotor strip and parietal lobe with reference to ears. After 31 sessions, the boy had observed improvement for social behaviors, improved sleep, a reduction in self-stimulation, and an increase in appropriate eye contact. QEEG results found decreased power ratios across 15 sites. Sichel, Fehmi, and Goldstein helped to set the stage for conducting meaningful research and submitting it to the still developing *Journal of Neurotherapy*, which has provided a venue to report research on the efficacy of neurofeedback.

Recently, Coben and McKeon (2009) released a single-subject case report of a young boy who had 165 epileptiform paroxysmal discharges, and reported specifically on utilizing QEEG-guided neurofeedback. The protocol consisted of temporal-occipital sites (i.e., O1 and T3) using 2-channel coherence training, rewarding 1-7 Hz and inhibiting 1-4 HZ and 8-13 Hz. Coben and McKeon found that this improved neuronal regulation across regions as opposed to just the focal epileptiform activity. They also found significant improvement on the Autism Treatment Evaluation Checklist (ATEC) showing an 82% overall improvement after one year of training. In another study addressing the neurological problems associated with ASC, Coben and Hudspeth (2006) explored NFT in mu rhythms (i.e., suggestive of mirror neurons and social interactions). They found a significant reduction in mu activity and increased social functioning for 14 children with ASC.

There have been three case series studies unpublished in peer-reviewed literature

on hemoencephalography (HEG) neurofeedback (Berman, Sudol, Miller, & Berman, 2005; Coben, 2006; Limsila et al., 2004). Hemoencephalography measures blood flow dynamics and cellular metabolism, and because of this, HEG neurofeedback provides the functional capability to have direct control over the prefrontal lobe's cerebrovascular system, a critical site for conditions like ADHD, depression, and migraines (Carmen, 2001). Further, the use of HEG neurofeedback has the benefit of minimizing artifacts and is less invasive procedurally when compared to EEG neurofeedback (Carmen, 2001). The largest HEG neurofeedback case series was conducted by Limsila et al. (2004) with 180 children who were diagnosed with ASC. They found that there was improvement after 40 sessions of prefrontal HEG training as indicated by improvement in average values of blood oxygenation, grade point averages, and positive reports by parent, teacher, therapist, and psychiatrist reports. Berman, Sudol, Miller, and Berman (2005) found similar results with a child age 14 at a charter school that gained five points in nonverbal intelligence, increased 22 points for Stroop testing, and improved hand writing legibility. The authors reported that five out of the six original participants for HEG training were unable to complete pre or post testing data, so they were excluded from the study. Finally, Coben (2006) presented the most comprehensive research on HEG in ASC with 28 children who received either near infrared or passive infrared HEG for 20 sessions. Compared to a wait-list control of 12 children, there were statistically significant reduction of autistic symptoms as measured by the ATEC and other behavioral rating scales, improved neuropsychological performance with executive functions, language, visuospatial, and attention indices, increased temperature based on Flir infrared imaging, and QEEG connectivity measures in the children with ASC.

Coben reported that there were no differences between the two types of HEG. There have been no peer-reviewed articles in this area of neurofeedback for children or adults with ASC, and these three studies have had limitations relative to past treatment, drop-out rates, and limited reports on specific methodology and statistical analyses.

Thompson et al. (2010b) provided a comprehensive overview of 159 clients with Asperger's syndrome or autistic disorder over a 15-year period. This may have included the previous mentioned case studies in other research (Thompson & Thompson, 2003b). They evaluated the efficacy of neurofeedback in combination with metacognitive training and respiration, electrodermal, and heart rate variability biofeedback. The sessions ranged from 40-60 sessions and a majority of training consisted of decreasing slow wave activity (3-7 Hz) and beta spindling (23-35 Hz), and increasing SMR (12-15 or 13-15 Hz). They primarily used central and frontocentral sites for training. The authors found significant improvements for psychological assessments that included questionnaires that assessed the core symptoms of Asperger's syndrome, Conners' Global Index, DSM-IV criteria for ADHD, and psychological testing like TOVA and IVA, achievement, and intelligence testing. Interestingly they found an average Full Scale IQ score gain of nine points. Thompson et al. further found a significant improvement in EEG ratios.

Overall, qualitative case studies are problematic because they do not generalize well and create standards for practice. One confounding variable could disrupt the entire study or lead to type I or type II errors. For example, Beaumont and Montgomery (2005), in their case study, reported the confounding variable of neurostimulant medication being added during the neurofeedback intervention, making a type I error likely. Thompson and Thompson (2003b) and Thompson et al. (2010b) used

diaphragmatic breathing and metacognitive strategies in addition to neurofeedback in many training sessions, and this confounds their findings. Case studies in neurofeedback has lacked sound methodology with vague reports of pre and post measure findings, retrospective rather than prospective collection, tendency for researcher biases, and not establishing a stable baseline of functioning (e.g., one pretest measurement as opposed to more than one). Single-case research would improve methodology over qualitative case studies because it offers time-series measurement, quantitative data collection, experimental control within the individual, and use of statistical and/or visual inspection analyses of the hypotheses (Blampied, Barabasz, & Barabasz, 1996). Neurofeedback may also be better evaluated through single-case research because it provides in-depth detail of the impact of the intervention, conditions like autism in the DSM-IV vary so greatly in symptoms that it would be dismissed to assume a sample of individuals with ASC will be equivalent, and regardless, each case becomes in and of itself an individual research study with a control (i.e., baseline) and experimental condition (Blampied et al., 1996).

Quasi-experimental Research Studies

Neurofeedback has advanced as an effective tool in treating ASC in the last decade with quasi-experimental research, initially explored in pilot studies to evaluate both the intervention as well as testing tools to evaluate its efficacy (Jarusiewicz, 2002; Knezevic et al., 2009, 2010; Scolnick, 2005). Jarusiewicz (2002) and Scolnick (2005) explored the efficacy of neurofeedback in children with ASC, setting the need for further exploration of efficacy by other researchers (Coben & Padolsky, 2007; Kouijzer et al., 2009b). Jarusiewicz (2002) conducted the first pilot-study of neurofeedback in ASC with

a nonrandomized experimental-control matched control group of 24 participants. With an average of 36 sessions at 30 minutes per session, the study found efficacy in utilizing symptom-based protocols which consisted of sensorimotor, frontal, and temporal sites to improve socialization, arousal, emotional stability, and expressive communication. The protocol also consisted of inhibiting 2-7 Hz and 22-30 Hz because of high amplitudes for slower and faster bandwidths. Results included statistically significant improvement ($p <$ .001) in parent-ratings on the ATEC for the ASC group compared to the control group, specifically in speech/language communication, sociability, sensory/cognitive awareness, and health categories. In the second pilot study, Scolnick (2005) conducted a less stringent single group study with a high recidivism rate of five youth with ASC out of an initial group size of 10 dropping out before completing 12 sessions, and did not achieve statistically significant results in pre and posttest QEEG. However, the researcher noted that the QEEG of the five students who completed 24 sessions appeared to normalize, and parents and teachers reported that the children had improved in behaviors. Despite the findings of limited efficacy, this was the first peer-reviewed article outside of a biofeedback specific journal such as the Journal of Neurotherapy or Applied Psychophysiology and Biofeedback Journal.

Later, Knezevic, Thompson, and Thompson (2009, 2010) conducted a neurofeedback pilot study with 19 participants ages 7 to 21 years who were diagnosed with Asperger's syndrome to evaluate the utility of the Tower of London-Drexel (TOL^{DX}) in assessing the efficacy of neurofeedback. They conducted the single channel Cz EEG measure ToL^{DX} for pre and post measures after 40 sessions of neurofeedback and metacognitive strategies to maintain alertness and focused state. Through the use of

computer game software, the participants were asked to be attentive to the feedback in the form of points and to use their own method or approach to remain alert. With three to four-minute training intervals and approximately 40 minutes each session, clients were rewarded at Cz for SMR (13-15 Hz) and/or problem solving (15-18 Hz) and for inhibiting slower frequency bandwidths (e.g., 4-8 Hz, or 3-10 Hz). They found statistically significant improvement in a paired samples t-tests for pre and post measures of executive functioning, the ToL^{DX}. However, the study established a set protocol that required 40 to 60 sessions of neurofeedback, and in addition to another intervention, metacognitive training, which is a significant confounding variable in terms of methodology and efficacy of neurofeedback as a treatment alone. Nevertheless, Knezevic, Thompson, and Thompson included adult participants with ASC, which has helped to explore possible efficacy of neurofeedback in adults.

In another nonrandomized control group study, neurofeedback improved QEEG normalization, executive functioning, and parents' observations in seven children with ASC ages 8 to 12 through inhibiting theta and rewarding beta over 40 sessions during a three-month period (Kouijzer et al., 2009b). The sessions were conducted with 3 minutes baseline, 3 minutes feedback, and one-minute rest intervals. The multiple outcome measures included the Children's Communication Checklist, AUT-R, theta/beta ratios in QEEG, and neurocognitive testing such as the stroop and symbol digit coding tests. The authors found statistically significant changes on post-test measures via a MANOVA (as high as $p < .001$) among ASC compared to the control group. The researchers suggest that these findings are indicative of improved flexibility in the anterior cingulate cortex (ACC), which is an important aspect of the default mode network (DMN). The main

pattern found in DMN for ASC appears to be the lack of deactivation in ACC, so Kouijzer, et al. (2009b) assumes that the reduction of theta activity may improve network flexibility to perform better on attention control tasks such as the stroop and symbol digit coding. The two main limitations to this study are the small sample size and that the protocol was not individualized (i.e., neurofeedback training based on QEEG).

The most comprehensive and well-formulated quasi-experimental research to date was conducted by Coben and Padolsky (2007) with the largest sample size of 37 children with ASC ages 4 to 14 for the experimental condition, and 12 matched controls placed on a wait list. The experimental condition consisted of 20 sessions of QEEG-guided neurofeedback protocols conducted three times per week. Treatment efficacy was measured by comprehensive pre and post measures consisting of the ATEC, Gilliam Asperger Disorder Scale (GADS), Gilliam Autism Rating Scale (GARS), Behavior Rating Inventory of Executive Function (BRIEF), Personality Inventory for Children (PIC-2), baseline measures of neuropsychological functioning, QEEG, and Infrared (IR) Imaging. The researchers provided individualized neurofeedback protocols for each participant using bipolar montages. The analysis of QEEG identified hyperconnectivity for frontal-temporal sites. For one participant, the researchers rewarded alpha frequency and inhibited low and higher bandwidths at F8 and F7 to reduce hyperconnectivity with a majority of training being in F8-F7, Ft8-Ft7, T4-T3. Results of the experimental condition consisted of statistically significant results ($p < .01$) with 76% decrease in hyperconnectivity patterns, improvement in language functions, regulation of thermal activity according to the IR imaging, and 40% reduction in core symptoms of ASC according to the ATEC, which shows consistency across multiple areas that were

measured from subjective reports to testing to neurophysiology. There were no reports of symptoms worsening. The authors analyzed the benefit to harm ratio as determined by parents as being 89:1 which surpassed all current therapies or treatment for ASC (e.g., behavioral, chelation, risperidone).

Experimental Research Study

There has been only one neurofeedback article reporting on treatment of individuals with ASC that consisted of an experimental research design, and the report included both the initial pilot study and the actual follow-up study (Pineda et al., 2008). The pilot consisted of a single-blind randomized controlled trial (RCT) with an experimental and placebo condition (both $n = 8$) of boys with high functioning ASC who had an IQ > 80 ages 7-17, and the follow-up study consisted of a double-blind RCT with males and females (ages 7-17) and a larger sample size ($n = 10$ in the placebo group; $n = 9$ in the treatment group; Pineda et al., 2008). The neurofeedback providers were not aware of whether the participants were provided feedback or placebo because they were preset prior to the sessions by separate clinicians. Pre and post measures consisted of QEEG using Mini-Q software by Brainmaster, Mu Suppression Index (i.e., assessing the changes in mu power in response to observation of movement), and Test of Variables of Attention (TOVA).

In the placebo condition, the participants received an artificially generated mu rhythm and trapezius electromyography (EMG) or muscle activity to allow control over EMG artifacts, thus allowing placebo participants to believe that they were receiving EEG feedback when they were really receiving EMG feedback. For the experimental condition, participants in both the pilot and actual study received 15 hours of training in

30-minute sessions three times a week for 10 weeks. Neurofeedback consisted of site C4 with feedback for mu rhythm (Oberman et al., 2005 for reduced mu power in ASC), and inhibiting trapezius EMG activity (30-60 Hz). The feedback for both conditions consisted of computer games with two feedback bars indicating EEG and EMG activity. The experimental group would proceed in the game when the conditions were met for reaching 8-13 Hz at C4 and 30-60 Hz at the trapezius muscle, while the placebo would only receive feedback from the EMG activity. Along with this feedback, experimenters provided verbal reinforcement by praising participants for paying attention and proceeding through the games.

Pineda et al. (2008) used repeated measures ANOVA within and between for QEEG, Apraxia Imitation Scale, ATEC, and mu power, and they used a paired-sample t-test (two-tailed) for TOVA. For the pilot study, they found a significant difference ($p <$ .05) in the experimental group compared to control group with changes in decreased amplitude coherence and differences in mu and delta frequency bands, where as the placebo condition showed increases in coherence. The larger scale study found similar significant findings ($p <$ .01 to .05) for QEEG measures. For the TOVA, there was a significant difference found in the pilot and larger scale study ($p <$ .02 for both comparisons) for overall ADHD score and errors of commission with improvement of up to 70% on their TOVA scores in the experimental group. There were significant differences between the experimental and placebo group for both the pilot and larger scale study, with improvement in ATEC scales ($p <$ .05), and no within group differences were noted in this study. The Apraxia Imitation Scale was also improved for movement and accuracy in both studies ($p <$ .01 and .03). Despite the added participants for the

larger study, the number of participants is smaller than the quasi-experimental design completed by Coben and Padolsky. In addition, the neurofeedback was a set protocol in order to institute the double-blind procedure, which does not allow for more individualized treatment interventions.

Longitudinal Research Studies

The research studies reviewed above highlight the significant short-term effects of neurofeedback in reducing symptoms of ASC. Kouijzer et al. (2009a) and Coben (2009a) evaluated long-term neurofeedback efficacy through follow-up studies conducted at 12 months and up to 24 months after treatment. Coben reported statistically significant long-term improvement in 20 individuals with 12 and 24-month follow-ups. He found that children with ASC who received QEEG connectivity guided neurofeedback with at least 35 sessions were shown to maintain statistically significant improvements ($p < .01$) with neuropsychological and educational measures along with stabilized QEEG patterns. Kouizer et al. (2009a) found statistically significant (at least $p < .05$) improvements in a 12 month follow up for executive functioning including auditory selective attention, inhibition of verbal responses, inhibition of motor responses, set shifting, concept generation, and planning ability (Kouijzer et al., 2009b). Kouijzer et al. (2009b) also found sustained benefits from neurofeedback in behavioral domains as indicated by observers in areas like general communication, pragmatics, social interaction, communication, and typical behavior.

Research Studies Specific to Neurofeedback LZT

For a complex condition like ASC, research is clearly indicated for individualized neurofeedback approaches based on multimodal assessments including

neuropsychological performance, behavioral and self-report measures, and neurophysiological measures like QEEG in order to obtain the best results (Coben & Padolsky, 2007). Specifically, LZT utilizes QEEG post-processing software with Joint-Time-Frequency-Analysis (JTFA) through a comparative database using Gaussian validated norms to assess and train neurofeedback in real time (Collura et al., 2010; Thatcher, 2008; Thatcher & Lubar, 2008). The use of LZT provides a basis for using a single measure of analysis, Z-scores, for a variety of statistical analyses of EEG activity like coherence, ratios, phase delays, power, amplitude, and asymmetry (Collura, 2008a, 2008b; Thatcher, 2008). By identifying normality through Z-scores, the individual is capable of matching the state of mind to comparative age-based norms to normalize functioning. Further, there is the possibility of whole-head normalization by utilizing posterior to anterior EEG sites during LZT (Collura, 2008b).

The benefits consist of within and between subject variance within a set age that is analyzed by complex demodulation rather than Fourier transform to provide instant power and phase analyses (Thatcher & Lubar, 2008). An example is the Applied Neuroscience, Inc. (ANI) Dynamic Link Library (DLL) statistical software which consists of 625 people ages two months to 82 years old and has FDA registration (Collura & Thatcher, 2006; Collura et al., 2009; Thatcher & Lubar, 2008). It is capable of comparing the individual to the normative database based on age, whether collected under eyes open or eyes closed conditions. The Z scores are computed every 33 milliseconds to show the NeuroGuide coherences normative Z scores.

The Z score neurofeedback approach was first utilized in traumatic brain injury to help participants reach EEG normalization based on Z score comparisons (Thatcher,

2000). It has now developed into providing EEG metrics, specifically absolute, relative power, power ratio, asymmetry, coherence, and phase, and the number of potential targets for a 4 channel EEG amplifier will analyze 248 z-scores, 104 power and 144 connectivity (e.g., coherence) EEG metrics (Collura, 2007; Collura et al., 2009). Further, training options consist of training frequencies up or down, creating ranges such as all Z scores within +/- 1 standard deviations, and percentage of Z scores that approach the mean or zero. Z Scores are differentiated by color with yellow being +1 to 1.5 SD, orange +1.5 to 2.0 SD, red +2 SD and above, green -1.0 to -1.5 SD, blue-green/cyan -1.5 to -2.0 SD, and blue -2 SD and below.

Despite the multiple benefits of LZT, researchers still feel that conventional QEEG is indicated in properly assessing and determining the type of feedback protocol and a clinician is still needed to determine the type or scope of appropriate neurofeedback to be provided (Collura et al., 2009; Collura et al.,, 2010). With that said, the use of LZT simplifies the process of neurofeedback in that it provides a Gaussian distribution for individualized training protocols, single metric with Z-scores, instantaneous modification of reward and inhibit according to between and within subject variance depending on age and eyes closed or eyes open (Collura et al., 2009).

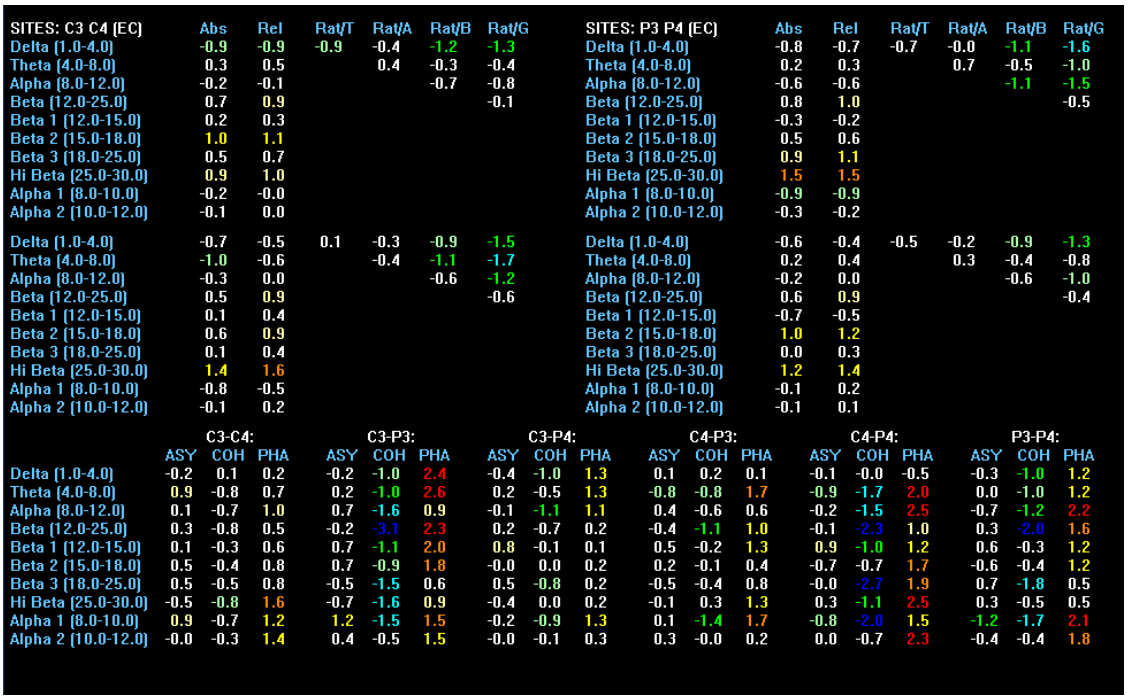
Gismondi and Thatcher (2009) reported on the efficiency and newly developing z-score training that allows for real-time normative database mathematical transforms for power and connectivity variables that are related to the theoretical concept of the hubs and modules that are functional and not merely one central area or location. The use of LZT helps to improve balance and regulatory improvement in brain function in neurodevelopmental disorders. For instance, Gismondi and Thatcher explain that

hypercoherence is essentially a cortical compensation in the loss of functional efficiency, which is common in neurodevelopmental disorders like autism. The use of this intervention in reducing hypercoherence will normalize brain function in real time.

Rutter (2009) published the first case study report of a child with profound autism using LZT NFT for Brainmaster based on the Z-score DLL from Thatcher's Neuroguide EEG analysis software. She conducted a QEEG that resulted in identification of elevated alpha hypocoherence, high beta (23-27), excess beta asymmetry, and phase activity in the fronto-central lobes, whereas there were low delta amplitude and high beta amplitude at the sensorimotor strip. They utilized the "Percent ZOK" Z-score training protocol with 40-60% reward adjusted during the session using linked-ear reference and ground behind right ear on frontal and sensorimotor cortex sites based on the most significant dysregulation found on the QEEG. They had musical tones or visual activated reinforcement for the client. He required desensitization to the experience but was able to cooperate with neurofeedback within the initial session lasting 24 minutes with sessions ranging from 5-40 minutes. She found that 10 sessions resulted in less aggression, and improved nocturnal enuresis, but increased restlessness and activity, but after 20 sessions she noted calmer behavior with less agitation and tics, and he was able to sit still and engage in the visual and musical feedback. Beyond 20 sessions, Rutter noted that he was more verbal, improved eye contact, addressing peers voluntarily, responded to external stimuli, and improved behaviors at school with social-communication, in addition to the functional changes in EEG toward the normative database. Rutter's research helped to highlight both the potential for neurofeedback in profound autism as well as the quick response to connectivity Z-score training within 10-

20 sessions despite the complex neurological problems associated with participant. This study, however, did not provide adequate baseline measures or data analyses that objectively evaluated the efficacy of LZT. For instance, the observations were not structured or objective. There needs to be more studies that provide measurable and operationalized behaviors that are being tracked throughout the study.

The first peer-reviewed publication on the relative efficacy of LZT was completed by Collura, Guan, Tarrant, Bailey, and Starr (2010) who reported the results of 19 submitted case studies, of which three were individuals with ASC. The montages were relatively similar using F3/,/P3/P4, F3/F4/C3/C4, or F7/F8/T5/T6, and all the case studies used the "Percent ZOK" program, which rewards the trainee when they maintain Z-scores (e.g., -1 SD to 1 SD) within a set percentage (e.g., 60% to 80%). All the participants showed reduction in abnormal z-scores and improved overall functioning. However, the research did not provide statistical analyses and simply provided qualitative reports from clinicians who submitted cases. Figure 2 below provides a screen shot from the Training Control Screen showing the z-scores available for viewing.



SITES: C3 C4 (EC)	Abs	Rel	Rat/T	Rat/A	Rat/B	Rat/G
Delta (1.0-4.0)	-0.9	-0.9	-0.9	-0.4	-1.2	-1.3
Theta (4.0-8.0)	0.3	0.5		0.4	-0.3	-0.4
Alpha (8.0-12.0)	-0.2	-0.1			-0.7	-0.8
Beta (12.0-25.0)	0.7	0.9				-0.1
Beta 1 (12.0-15.0)	0.2	0.3				
Beta 2 (15.0-18.0)	1.0	1.1				
Beta 3 (18.0-25.0)	0.5	0.7				
Hi Beta (25.0-30.0)	0.9	1.0				
Alpha 1 (8.0-10.0)	-0.2	-0.0				
Alpha 2 (10.0-12.0)	-0.1	0.0				
Delta (1.0-4.0)	-0.7	-0.5	0.1	-0.3	-0.9	-1.5
Theta (4.0-8.0)	-1.0	-0.6		-0.4	-1.1	-1.7
Alpha (8.0-12.0)	-0.3	0.0			-0.6	-1.2
Beta (12.0-25.0)	0.5	0.9				-0.6
Beta 1 (12.0-15.0)	0.1	0.4				
Beta 2 (15.0-18.0)	0.6	0.9				
Beta 3 (18.0-25.0)	0.1	0.4				
Hi Beta (25.0-30.0)	1.4	1.6				
Alpha 1 (8.0-10.0)	-0.8	-0.5				
Alpha 2 (10.0-12.0)	-0.1	0.2				

SITES: P3 P4 (EC)	Abs	Rel	Rat/T	Rat/A	Rat/B	Rat/G
Delta (1.0-4.0)	-0.8	-0.7	-0.7	-0.0	-1.1	-1.6
Theta (4.0-8.0)	0.2	0.3		0.7	-0.5	-1.0
Alpha (8.0-12.0)	-0.6	-0.6			-1.1	-1.5
Beta (12.0-25.0)	0.8	1.0				-0.5
Beta 1 (12.0-15.0)	-0.3	-0.2				
Beta 2 (15.0-18.0)	0.5	0.6				
Beta 3 (18.0-25.0)	0.9	1.1				
Hi Beta (25.0-30.0)	1.5	1.5				
Alpha 1 (8.0-10.0)	-0.9	-0.9				
Alpha 2 (10.0-12.0)	-0.3	-0.2				
Delta (1.0-4.0)	-0.6	-0.4	-0.5	-0.2	-0.9	-1.3
Theta (4.0-8.0)	0.2	0.4		0.3	-0.4	-0.8
Alpha (8.0-12.0)	-0.2	0.0			-0.6	-1.0
Beta (12.0-25.0)	0.6	0.9				-0.4
Beta 1 (12.0-15.0)	-0.7	-0.5				
Beta 2 (15.0-18.0)	1.0	1.2				
Beta 3 (18.0-25.0)	0.0	0.3				
Hi Beta (25.0-30.0)	1.2	1.4				
Alpha 1 (8.0-10.0)	-0.1	0.2				
Alpha 2 (10.0-12.0)	-0.1	0.1				

	C3-C4:			C3-P3:			C3-P4:			C4-P3:			C4-P4:			P3-P4:		
	ASY	COH	PHA	ASY	COH	PHA	ASY	COH	PHA	ASY	COH	PHA	ASY	COH	PHA	ASY	COH	PHA
Delta (1.0-4.0)	-0.2	0.1	0.2	-0.2	-1.0	2.4	-0.4	-1.0	1.3	0.1	0.2	0.1	-0.1	-0.0	-0.5	-0.3	-1.0	1.2
Theta (4.0-8.0)	0.9	-0.8	0.7	0.2	-1.0	2.6	0.2	-0.5	1.3	-0.8	-0.8	1.7	-0.9	-1.7	2.0	0.0	-1.0	1.2
Alpha (8.0-12.0)	0.1	-0.7	1.0	0.7	-1.6	0.9	-0.1	-1.1	1.1	0.4	-0.6	0.6	-0.2	-1.5	2.5	-0.7	-1.2	2.2
Beta (12.0-25.0)	0.3	-0.8	0.5	-0.2	-3.1	2.3	0.2	-0.7	0.2	-0.4	-1.1	1.0	-0.1	-2.3	1.0	0.3	-2.0	1.6
Beta 1 (12.0-15.0)	0.1	-0.3	0.6	0.7	-1.1	2.0	0.8	-0.1	0.1	0.5	-0.2	1.3	0.9	-1.0	1.2	0.6	-0.3	1.2
Beta 2 (15.0-18.0)	0.5	-0.4	0.8	0.7	-0.9	1.8	-0.0	0.0	0.2	0.2	-0.1	0.4	-0.7	-0.7	1.7	-0.6	-0.4	1.2
Beta 3 (18.0-25.0)	0.5	-0.5	0.8	-0.5	-1.5	0.6	0.5	-0.8	0.2	-0.5	-0.4	0.8	-0.0	-2.7	1.9	0.7	-1.8	0.5
Hi Beta (25.0-30.0)	-0.5	-0.8	1.6	-0.7	-1.6	0.9	-0.4	0.0	0.2	-0.1	0.3	1.3	0.3	-1.1	2.5	0.3	-0.5	0.5
Alpha 1 (8.0-10.0)	0.9	-0.7	1.2	1.2	-1.5	1.5	-0.2	-0.9	1.3	0.1	-1.4	1.7	-0.8	-2.0	1.5	-1.2	-1.7	2.1
Alpha 2 (10.0-12.0)	-0.0	-0.3	1.4	0.4	-0.5	1.5	-0.0	-0.1	0.3	0.3	-0.0	0.2	0.0	-0.7	2.3	-0.4	-0.4	1.8

Figure 2. Neurofeedback LZT screen with 248 z-scores including connectivity, absolute and relative power, and ratio measures (permission by Collura & Thatcher, 2010).

Number of Sessions Variability in Neurofeedback Research

Clinically, the number of sessions for neurofeedback varies greatly depending on the type of condition, severity, and procedure used. In research on individuals with ASC, neurofeedback has ranged from just 20 sessions (Coben & Podolsky, 2007) to as many as 100 sessions (Thompson & Thompson, 2003b). When symptom-based protocols were implemented (i.e., training based on symptom self-reports), the number of sessions reported is greater than those that implemented comprehensive neuropsychological testing and QEEG-guided protocols (Coben & Podolsky, 2007). Single channel approaches, such as training at C3 or C4, also require the greatest number of sessions in order to exhibit improvement (Thompson & Thompson, 2003b). In one study regarding neurofeedback LZT, the child with lower functioning autism demonstrated improvement

after 10 sessions, but received over 20 sessions to make significant gains (Rutter, 2009). Coben and Podolsky explained that the fewer sessions were needed due to the use of individualized neurofeedback approaches using bipolar protocols (i.e., one active sensor site and one reference site located over a specified brain site) as opposed to unipolar protocols (i.e., one active sensor over a brain region and a reference to the ear). The use of neurofeedback LZT may provide enhanced training over four active sites instead of two sites along with individualized training in real-time. Therefore, neurofeedback LZT may provide efficacy in a shorter period of time.

Neurofeedback Adverse Effects

Neurofeedback has a high benefit-risk ratio (89:1) compared to other interventions like psychopharmacological interventions or dietary supplements for ASC (Coben & Padolsky, 2007). According to parent reports, neurofeedback provided the most benefits and minimal to no adverse effects compared to all other interventions for ASC (Coben & Padolsky, 2007). However, there are always potential risks in changing brain function when it is not individualized using multimodal assessment strategies to determine appropriate site locations and feedback protocols (Hammond & Kirk, 2008). Most recently there is a trend for psychologists and researchers in being more assertive in identifying and reporting negative iatrogenic effects of therapeutic techniques (Barlow, 2010). The need for developing systems for monitoring adverse effects and randomized-controlled trials (RCT) are critical for examination of potential side effects as a result of psychological interventions (Dimidjian & Hollon, 2010).

With regard to LZT training, a compilation of case studies found that there were no abreactions (Collura et al., 2010). However, Collura, et al. (2010) suggested that Z-

score training with a wide threshold (e.g., +/- 3 SD) could potentially lead to abreactions and unnecessary training. Also, Rutter (2009) identified initial worsening of symptoms such as increased activity when using LZT, but in later sessions, she found a significant decrease in symptoms. Among reports from NFT on an internet list serve, clinicians providing neurofeedback noted adverse effects such as vocal/motor tics, muscle twitches, somatic complaints, enuresis, incontinence, epileptiform activity, fatigue, anxiety, agitation/irritability, obsessive-compulsiveness, depression, mania, cognitive inefficiencies, inattention, poor concentration, insomnia/hypersomnia, regression, and seizures (Hammond & Kirk, 2008). Also, it was identified that poorly planned interventions may create a decrease in executive functioning performance (Knezevic et al., 2009, 2010). However, these reports were subjective and had no specific tool to assess adverse effects or research method to adequately support these findings. Further, the main factor in adverse effects were a result of using protocol-based neurofeedback as opposed to individualized neurofeedback training that incorporates a comprehensive evaluation to determine the best course of treatment. Hammond and Kirk (2008) suggested that many of these protocols served to reinforce certain bandwidths that may have exacerbated symptoms rather than inhibiting EEG activity that is seen as problematic. Therefore, monitoring these symptoms is essential during neurofeedback.

Gap in the Neurofeedback Research on Autism

The biofeedback monograph was created to assess the level of efficacy according to standards of research for evidenced based practice of biofeedback (LaVaque et al., 2002). At the time, the monograph cited that autism was considered *insufficiently investigated* because there was only one publication (Sichel et al., 1995), and

neurofeedback was still in the process of developing standards for research and identifying efficacy in various neuropsychological disorders like ASC. However, since that time, there have been multiple case studies, case series, presentations, quasi-experimental studies, and double blind experimental studies that support a level of *probably efficacious* in treating ASC (Coben & Padolsky, 2007; Jarusiewicz, 2002; Knezevic et al., 2009, 2010; Pineda et al.,, 2008; Yucha & Montgomery, 2008). Still, there is a lack of empirical support for interventions in adults with ASC that have been found effective in reducing symptoms in children with ASC (Roy et al., 2009; Shea & Masibov, 2005; Wolf & Paterson, 2010). For example, neurofeedback has shown success in treating symptoms associated with ASC through multiple case studies and controlled trials, but majority of participants consisted of young children to early adolescents (Coben et al., 2010).

Some researchers like Thompson and Thompson (2003b) have found subjective improvement with psychosocial functioning into adulthood indicated by improved college and employment performances. When comparing if there were differences in the efficacy of neurofeedback for age or level of intellectual functioning, Knezevic, Thompson, and Thompson (2009, 2010) found no statistically significant differences on the ToL^{DX}, a test of executive functioning. These results suggest that varying ages and level of intellectual functioning show equally positive results with NFT. In a retrospective case series study, Thompson et al. (2010b) evaluated a combination of neurofeedback, metacognitive strategies, and traditional biofeedback in 159 participants, of which 12 were adults, and they found that neurofeedback improved neurocognitive abilities, self and other reports, intelligence, and achievement. However, there has yet to

be a prospective quantitative study that specifically evaluates the level of efficacy with adults for neurofeedback LZT.

Lastly, although there have been multiple case studies mentioned in this review, they have not followed quantitative experimental formats recommended by Blampied, Barabasz, and Barabasz (1996) or Kazdin (1982) such as multiple baseline AB designs, and because of this, it has led to subjective interpretations and qualitative reports. Further, Kazdin (1982) discussed biofeedback and psychophysiological studies are at an advantage in single-case research because automated measurement devices like EEG recordings are optimally objective, acquired in repeated measures, and reliable for data acquisition, which are easily evaluated in visual inspection formats. Despite the benefit of single-case research designs in neurofeedback research, none of the case studies noted above included baseline measures or quantitative procedures that allowed for causal inferences and the ability to reject or accept the null hypotheses. Another rationale is that applied research settings like local clinics and private practices often have imitations associated with access to larger sample sizes, so single-case research seems to be the preferred method for evaluating efficacy of interventions like neurofeedback. In addition, given the fact that ASC carries such a wide variety of social and behavioral symptoms (APA, 2000), the samples used in the larger studies are likely heterogeneous with great variability in each participant's symptom profile. Therefore, there is a need for increased utility of single-subject research in ASC to evaluate individual characteristics and changes in the participant's profile (Shadish et al., 2002).

Summary and Transition

This literature review establishes the theoretical and evidentiary groundwork for

the rationale of this proposal. Specifically, ASC is a neurodevelopmental disorder with challenges that maintain into adulthood and the rising prevalence rates and costs associated with ASC only increase the need for effective interventions beyond childhood. The literature review covered autism research that identified significant and broad impairments in neurophysiological functioning particularly EEG connectivity, neurocognitive deficits in information processing and executive functioning, impaired empathy and comprehension of the intentions of others, rigidity in routines such as fixed areas of interests, obsessive stereotypic behaviors, and along with a number of comorbid symptoms such as inattention, impulsivity, depression, anxiety, and mood instability. With all these concerns in ASC, there is a need for preliminary investigations of newly developing interventions like neurofeedback LZT particularly with measures that comprehensively explore its detailed effects. Although there are a number of neurofeedback studies finding significant improvement in children with ASC, there are only a few retrospective studies that have evaluated it in adulthood. Further, there are presently no quantitative research studies evaluating the effects of neurofeedback LZT. Chapter 3 will use this research as a direction for the proposed methodology in exploring neuropsychological changes of neurofeedback LZT in adulthood ASC.

Chapter 3: Research Method

Introduction to the Research Method

The purpose of this study was to evaluate whether neurofeedback LZT in an adult with ASC would result in a reduction of autistic and neuropsychological symptoms and improvement in general intelligence, neurocognitive abilities, and brain function as measured by QEEG and LORETA. In Chapter 3, the principal investigator provided the research design overview, setting, participant recruitment, sample size, data collection, analysis, instrumentation, materials, procedure, research questions and hypothesis, overview of dependent variables, and protection of the participant will be detailed.

Research Design Overview

Efficacy of neurofeedback in children diagnosed with autism has been well researched in qualitative case studies, with results indicative of improved neuropsychological and neurophysiological functioning (Beaumont & Montgomery, 2005; Cowan & Markham, 1994; Rutter, 2009; Sichel et al., 1995; Thompson & Thompson, 2003). Although qualitative and retrospective case study research is important and offers support for clinical utility, more rigorous research designs such as mixed methods, repeated measures single-case research, quasi-experimental, and experimental research designs are needed to further validate and identify clinical efficacy of neurofeedback through quantitative analyses and causal inferences (Blampied et al.,1996; Coben et al., 2010). The most important future direction for validation of neurofeedback in ASC is to evaluate the specificity of the effects in neuropsychological symptoms, neurocognitive abilities, and brain function in autism (LaVaque et al., 2002).

Recently, researchers have explored the efficacy of neurofeedback in children

with ASC in quasi-experimental research studies with either QEEG guided or symptom-based protocols (Coben & Padolsky, 2007; Jarusiewicz, 2002; Kouijzer et al., 2009b). There has been only one study to date in which researchers used randomized double-blind research (Pineda et al., 2008). Within the specific approach of neurofeedback LZT, there have been only two peer-reviewed articles investigating the efficacy of LZT in children with neurodevelopmental disorders using qualitative case study research (Collura et al., 2010; Rutter, 2009). These articles consisted of qualitative observer reports and pre and post QEEG data, but the researchers did not implement more rigorous research methods, such as repeated measures single-case research using validated and reliable report measures. Also, because ASC is heterogeneous in symptoms and functional level (APA, 2000), it made sense to evaluate the effects in a single case particularly because of the potential predictive variables—age and intellectual level (Coben et al., 2010).

The study was structured as a multiple baseline AB research design to evaluate changes associated with neurofeedback in an adult with ASC (Creswell, 1994; Kazdin, 1982; Shadish et al., 2002). The first phase consisted of recruiting volunteer adults who have been diagnosed with autism, a convenience sample from a local neurofeedback clinic. The participant who qualified was welcomed into this study and provided information on the informed consent process, provider's qualifications form, release of information allowing disclosure to his medical and therapist providers, limits to confidentiality, IRB research consent and disclosure, and consented to assessments and testing. Following the initial consents, the participant commenced with five baseline assessments of neuropsychological and core autistic symptoms, five baseline measures of

a neurocognitive battery, three baseline measures of intelligence, and pretreatment QEEG and LORETA maps. Neurofeedback LZT training consisted of 20 sessions in a clinical setting, and was within the number of sessions of neurofeedback LZT that has shown a treatment effect—as low as 10 sessions have demonstrated significant improvement in ASC (Collura et al., 2010; Rutter, 2009). The neurofeedback intervention was conducted by a neurofeedback clinic in rural Michigan and was separate from this research study. Testing occurred throughout treatment. Visual inspection was used to assess change and clinical significance between the baseline phase and neurofeedback phase with trending data point graphs (Blampied, 2000; Fisher, Kelley, & Lomas, 2003; Kazdin, 1982). Given that neurofeedback researchers have demonstrated long-term effects on neuropsychological functioning (Kouijzer et al., 2009a; Coben, 2009a); using another single-case research design such as ABAB was not applicable to this research study.

Setting and Participant Recruitment

This section includes details regarding the type of research setting, participant inclusion and exclusion criteria, rationale for sample size, informed consent process, and confidentiality. The sample size is explored in detail regarding investigators who support single-case research in conditions like autism and interventions like neurofeedback. The overall purpose of this section was to provide an overview of the research project's environment and participant.

Research Setting

The setting was in a rural community of the Lower Peninsula in northwest Michigan. The research was conducted at a local neurofeedback clinic with the necessary equipment for conducting testing and assessment procedures. It was equipped with

adequate lighting, handicap accessible space, and maintained a temperature of approximately 20 °C to prevent sweating during sensor placement and to reduce artifact during administration of neurofeedback. Neurofeedback clinic consultants volunteered and donated supervision, neurofeedback services, consultation, and direction for the research study. The community partnership with a neurofeedback clinic was essential because it provided the neurofeedback, acquisition of QEEG, and other data important to the study. The principal investigator had no prior or existing business relationship with the neurofeedback clinic that might be considered a conflict of interest for this study.

The clinicians of the neurofeedback clinic and the principal investigator are Board Certified in Neurofeedback (BCN) by the Biofeedback Certification International Alliance (BCIA). The BCN certification requires completion of 36 hours of didactic education in neurofeedback, coursework in physiology, 25 hours of mentoring, 100 client sessions, case conferences, and passing the written certification examination. The rigorous standards set by the BCIA are critical for professional competency and ethical practice in the application of neurofeedback in research for this research study.

Participant Recruitment

The convenience sample consisted of a single participant who was recruited by a continuous 2-week advertorial by the neurofeedback clinic. Only prospective neurofeedback clients with ASC who meet the inclusion criteria were offered information about the study. Further, only participants who sought neurofeedback at the clinic were considered. The neurofeedback clinic consultants were responsible for screening and selecting the potential participants for consideration without any input from the principal investigator. The solicitation consisted of a statement (see Appendix B), and an

advertisement (see Appendix C). Following the screening and when the interested participant was identified, he was referred to initiate the study. The research study consisted only of the interview, testing, and assessment procedures; the neurofeedback itself was provided as a clinical service separate from the research procedures.

Inclusion criteria. The potential participants consisted of individuals of either sex or any ethnicity over the age of 18. They needed to be taking less than three medications-no specific medications were part of the exclusionary criteria (Coben & Padolsky, 2007; Johnstone, Gunkelman, & Lunt, 2005; Townsend, 2007). Other inclusion criteria were at of least average intellectual functioning or a 100 IQ within a standard deviation of 15, and competent to consent to research participation. The candidate needed to have a diagnosis from a healthcare professional of an ASC, which included autistic disorder, Asperger's disorder, childhood disintegrative disorder, and PDD NOS (APA, 2000, 2010). Due to the high male-to-female ratio (APA, 2000; CDC, 2009; Klin, McPartland, & Volkmar, 2005), the prospective participants were all males, and although all races and ethnicities were included, the participants were European American because of the population demographics of this specific rural area in Michigan. The research procedures did not exclude a participant based on ethnicity, race, sex, religion, or education.

Exclusion criteria. The participants excluded were those who were under the age of 18, and/or those who were prescribed more than three medications, and/or those with a level of intellectual functioning below average or lower (i.e., 85 IQ) and who were not competent to consent to research participation. Due to the proposed changes for DSM-V (APA, 2010), Rett's disorder was considered a separate medical condition that

did not qualify as an ASC. Individuals who were non-English speaking, pregnant, elderly (ages 65 years or older), or who lived in a residential facility were excluded from this study. Individuals who scored in the severe range on the Neuropsych Questionnaire for Depression would have been referred for psychological treatment services; however this did not occur. Exclusion criteria were provided in the advertisement. Following the initial discussion of the advertorial, one participant was excluded because he was being incarcerated and was provided the following statement, "Unfortunately due to the specific nature of the study, I am only able to accept people who meet set criteria. Thank you for offering your time and considering this project."

Informed consent. The informed consent process was ongoing throughout the study in order to allow for continuous dialogue with the participant regarding the research study. At the initial session as well as at each research-related testing appointment the participant was informed that this was a clinical research project and he was only consenting to testing, assessment, and interviews for the study. The consent form was presented, read, and signed by the participant during the first session (see Appendix D). The principal investigator read the consent form aloud and addressed the participant's questions and concerns in order to ensure comprehension. The participant had an advocate, which was his mother, who acted as a witness during the informed consent. During the sessions, the participant was assessed for adverse effects and informed of his ability to retract consent and terminate participation at any time. The informed consent process also included providing information on neurofeedback, but it was made clear that the research study was investigating changes associated with the clinical training, and that neurofeedback was not a part of the study itself.

Confidentiality. The participant was assigned a case number to preserve confidentiality. The case number was used on all study-related documentation as well as computer files, which were kept on a secured password protected computer. IRB guidelines for consent and disclosure of data are provided in Appendix E. All original research documents including the ID key that associates case numbers with names were stored in a locked filing system and within a secured password protected computer. Identifying information was destroyed when data collection was completed.

Sample Size

The proposed study utilized a single-case research design that consisted of one participant. The decision for single-case research was based on the ability to provide causal inferences with a rejection of the null hypothesis, visual inspection of effects through graphs, and replication to develop reliable and consistent findings in multiple cases or larger sample studies (Blampied, 2000; Kazdin, 1982). Single-case research allows for preliminary investigations regarding the effects of an intervention prior to more rigorous research methodology such as randomized controlled trials. Researchers like Skinner (1948) have used single-case research to develop learning theories that have been generalized from animals to people in applied research settings. Further, neurofeedback is based on operant conditioning and other learning theories that have used single-case research as the primary research approach. It is an important method in psychological research, because it focuses on the individual rather than averaging group processes, and single-case research has been often used in research with biofeedback interventions (Kazdin, 1982). Single-case research is particularly important when considering the heterogeneity of groups like ASC that vary in functional level and

symptom profiles. Some researchers have suggested that studying ASC in a group analysis actually reveals little information due to the variety of differences in cognitive functioning (Towgood, Meuwese, Gilbert, Turner, & Burgess, 2009). However, when the analysis is based on single-case research, Towgood et al. (2009) found more informative details regarding cognitive profiles amongst individual participants with ASC offering more data to support the complexity of ASC. Therefore, using single-case research avoids the averaging and loss of information that might be found in this particular study.

Secondly, neurofeedback studies have historically consisted of smaller sample sizes (Rojas & Chan, 2005), and despite these small samples, the researchers have demonstrated high effect sizes for neurofeedback in neurodevelopmental conditions like ADHD and ASC (Arns, Ridder, Strehl, Breteler, & Coenen, 2009; Coben, 2009; Monastra, Monastra, & George, 2002; Thornton & Carmody, 2008). Monastra et al. (2002) reported large effect sizes of 2.22 for treating inattention and 1.36 for hyperactivity. In a recent meta-analysis research of neurofeedback in ADHD, Arns et al. (2009) identified effects sizes that averaged around .81 for inattention, .69 for impulsivity, and .40 for hyperactivity. In treatment for traumatic brain injuries (TBI), Thornton and Carmody (2008) reported effect sizes for neurofeedback protocols at .55, and when quantitative EEG assessments guided training, the effect size was large at around 2.61 for treating symptoms related to the condition. Coben (personal communication, July 21, 2009) calculated a large effect size of 1.05 in a cumulative sample of 92 research participants with ASC using QEEG-guided neurofeedback, which included neurocognitive measures, symptom-based measures, and neurophysiological

measures. Therefore, neurofeedback researchers have consistently found high effect sizes despite these smaller sample sizes.

Another rationale in choosing a single participant is that it protects against Type II error rates by analyzing multiple baseline measures of dependent variables on an individual level and provides an opportunity to more fully analyze changes associated with neurofeedback on subjective and objective measures. For these reasons, single-case research has multiple advantages and was the best approach for the specificity of this research. Further, empirically supported interventions for ASC have been validated through the single-case research designs for several decades (Smith, 2008). Almost 90% of behavioral interventions for ASC have been evaluated through single-case research (Matson, Matson, & Rivet, 2007). The benefit of such designs is that they have the individual become his own experiment with a baseline control and experimental phase to identify an effect. Lastly, single-case research has validated approaches like Applied Behavioral Analysis as well as invalidated approaches like facilitated communication (Smith, 2008). Thus, single-case research is a method that is effective in evaluating the null hypotheses and has been used to effectively validate interventions in ASC.

Data Collection and Analysis

The data collection began with demographic and descriptive information including gender, age, handedness, level of education, race/ethnicity, medications, supplements, and alcohol/drug use including caffeine and cigarettes. The participant was provided a screening form found in Appendix F. A release form in Appendix G was signed to allow the primary investigator to contact the participant's health care professional for confirmation of ASC diagnosis and supporting testing data. The reports

and records retrieved were kept in a locked cabinet. The collection process began by phone, mail, or direct face to face interviews.

Although there are statistical methods such as autocorrelation in single-case research, researchers have found that these techniques in single-case research are problematic and tend to skew effect sizes because they violate assumptions of statistical techniques (Parker et al., 2005). The other main analysis for single-case research is visual inspection, which has been viewed as an effective and accurate way to analyze the effects of neurofeedback (Kazdin, 1982). Visual inspection offers the ability to determine changes of performance through data pattern analysis over time through exploring consistency in changes. A recent study found high interrater agreement for visual inspection when considering mean shift, variability, and trend across phases indicating consistency in interpretation of single-case data (Kahng et al., 2010). Related to this study, researchers have utilized visual inspection analysis effectively and validly in psychophysiological research (Schoen, Miller, Brett-Green, & Hepburn, 2008) as well as new and innovative interventions for ASC (Taylor et al., 2009). In addition, visual inspection is considered a conservative approach compared to statistical analyses that tend to identify significant changes when there are minimal or slight differences in data (Fisher, Kelley, & Lomas, 2003; Kahng et al., 2010). For these reasons, visual inspection was used to plot data points across time through connecting lines through each data point by phase, and the graph was evaluated visually for the slope trend and mean baseline compared to the neurofeedback phase (Kazdin, 1982).

Fisher et al. (2003) developed a structured technique called the conservative dual criterion (CDC) for visual inspection. The dual criterion (DC) technique calls for

evaluating treatment efficacy when a set number of data points that fall above the linear regression trendline based on the binomial test and the same number of data points also had to fall above or below the mean line of the baseline data. The CDC went further by raising the two criterion lines, mean and trendline, by .25 standard deviations calculated from the baseline data. Fisher et al. applied the Monte Carlo Validation of CDC and found that it was the only visual inspection procedure that guarded against Type I and II error rates with and without autocorrelation and higher power levels than statistical procedures. The authors also found that applying a dual criterion provided greater improvement in determining an accurate treatment effect based on this method much better than other ways such as the split-middle technique proposed by Kazdin (1982). Recently, Stewart, Carr, Brandt, and McHenry (2007) found that the CDC improved substantially improved visual inspection accuracy over traditional subjective interpretations data trends. When the CDC lines were removed, students had increased false alarm rates suggesting the need for methods like CDC in visual inspection to prevent Type I errors. Keller (2007) found that the CDC had consistency with statistical process control of up to 54%, and that the CDC was more conservative when determining treatment effects in single subject studies.

Figures 3 and 4 below depict examples of significant and not significant effects, respectively, using the CDC method on the dependent variable NPQ-LF Asperger's index. The combination of the mean and linear regression lines of the baseline data with a .25 SD modification provides two strict criterions to evaluate the effect of neurofeedback on this dependent variable. Based on the binomial formula, all five data

points in this study needed to fall above or below the modified mean and regression lines in order for a significant effect to occur (Fisher et al., 2003).

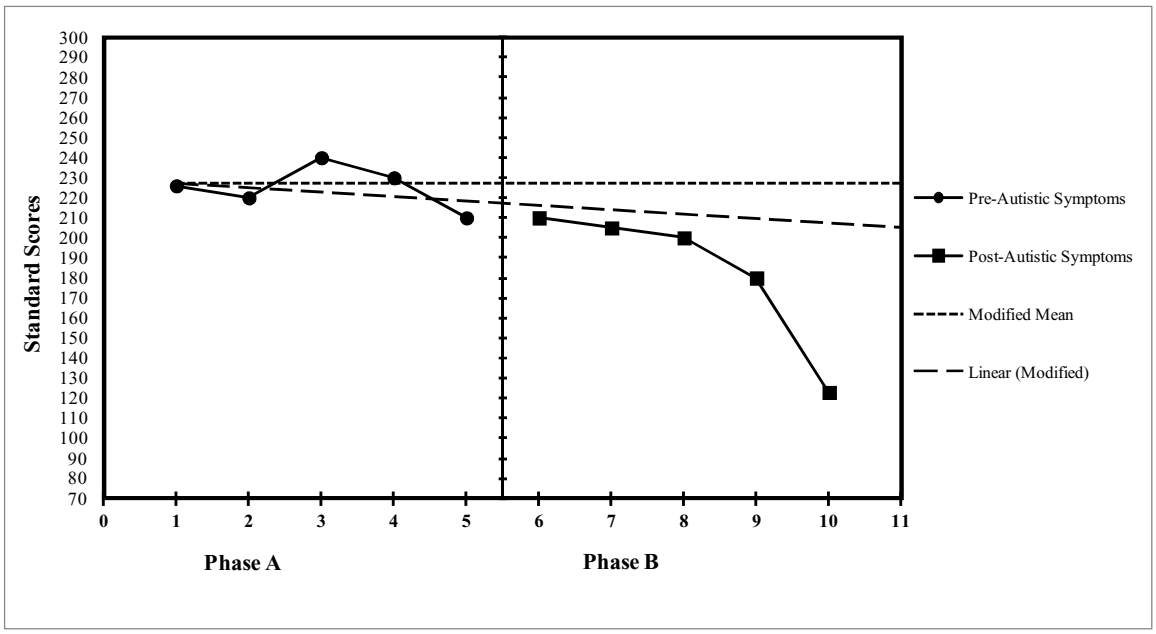


Figure 3. Example of visual inspection with a significant effect.

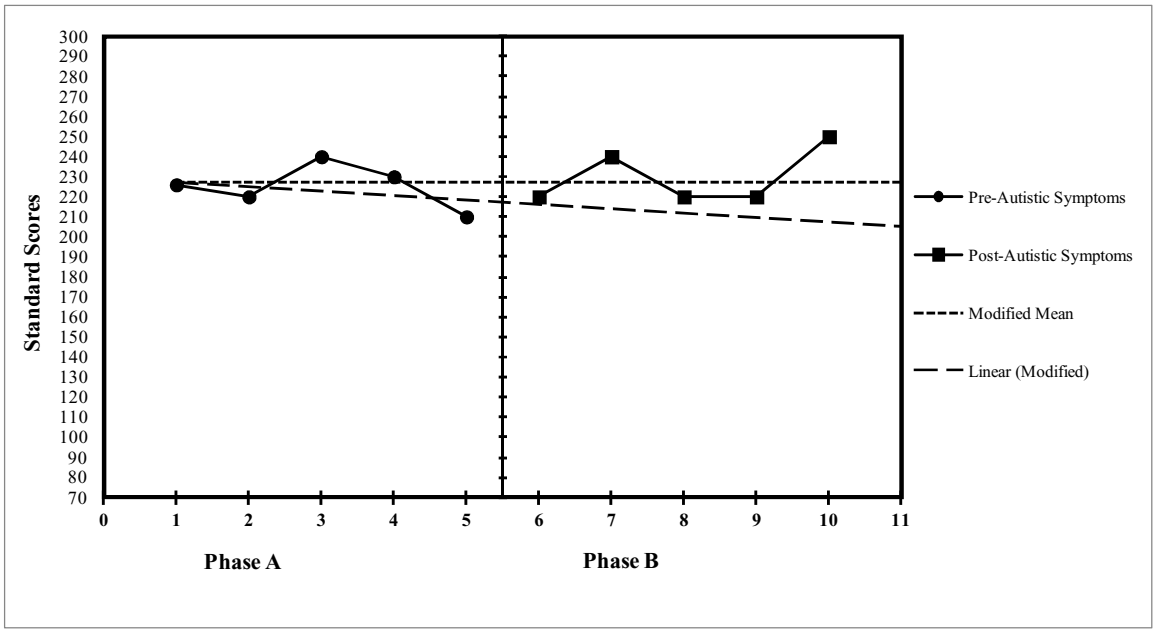


Figure 4. Example of visual inspection with no significant effect.

Lastly, pre and postdata were charted for the TONI and QEEG maps for visual inspection of change. A simple line graph plotting three baseline and three treatment phase data points were used for the TONI. A significant change in the TONI was analyzed by visual inspection with an observable change from the baseline mean and trendline to the treatment phase (Kazdin, 1982). For the QEEG maps, data were reviewed via Neuroguide software for changes in power and other measures. Changes in absolute power or relative power values were explored through visual inspection using Neuroguide's Neurostat software program of pre and post-QEEG maps. Figure 5 is an example of a summary Z-scored FFT QEEG maps of absolute power, which is the square of the magnitude indicating the amount of energy across the frequency bandwidths. In addition to serving as a tool for change, these maps were used to guide the neurofeedback practitioner for determination of which sites to choose.

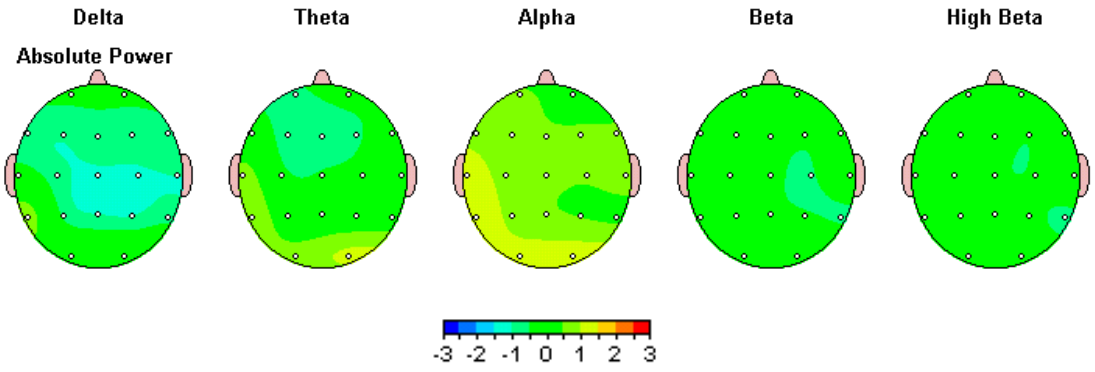


Figure 5. Example of QEEG maps' absolute power by the principal researcher.

Instrumentation and Materials

Computer Equipment

The computer used was a Hewlett-Packard HP Pavilion dv7 Notebook PC with a

17.3” HD+ Bright View LED Display, AMD Phenom II N850 Triple-Core processor 2.20 GHz, 4.00 GB RAM, 640 GB hard drive, 64-bit operating system, Windows 7 software, and Blu-Ray and DVD disc drives. It has a Fingerprint Reader that enhances protection of confidentiality and privacy of the data. The computer-based neurocognitive test has a standardized keyboard in order to improve the reliability of test administration in the same method that was used in the validity and reliability research studies.

Software Programs

The CNS Vital Signs (CNSVS; 2010) was the software used for the Neuropsych Questionnaire (NPQ; Gualtieri, 2007) and CNSVS Neurocognitive Test. This software requires at minimum Windows based software, 2 GH Pentium Class Machine, 256 MB RAM, 15 MB hard disc space, and 32 bit Super VGA. The software provides access to data collection of the CNSVS NPQ and Neurocognitive Test for each participant as well as summaries in Adobe formatted files. The participant was provided a series of tests on the computer screen that consisted of questions with multiple response options, shapes, words, and directions with count downs prior to each testing section.

Neuropsych Questionnaire

The NPQ (Gualtieri, 2007) is a computer-based neuropsychological screening instrument consisting of the NPQ-Long Form (LF) 207 questions, and Short Form (SF) that has 45 questions. For the purpose of this research, the NPQ-LF was used for baseline and postmeasures (see Appendix H). The rationale for using the NPQ-LF in this research project was that it offers ranges that are sensitive to treatment effects and consists of Asperger’s, autism, ADHD, anxiety, depression, and mood stability indices, and evaluates ASC symptoms and the comorbid symptoms of ASC simultaneously (APA,

2000; Bellini, 2006; Shtayermman, 2008; Volkmar & Klin, 2005). The index scores are categorized as not significant at 74 or lower, mild scores are in the 75-149 range, moderate scores fall in the 150 to 224 range, and severe scores fall in the 225 to 300 range.

Validity. The questions on the NPQ were developed through matching up to 75% similarity with common screeners used in clinical practice such as the Brief Psychiatric Rating Scale, Beck's checklists, and others. Each symptom is rated on a Likert scale from zero to three reflective of "not a problem," "a mild problem," "a moderate problem," or "a severe problem" respectively (Gualtieri, 2007, p. 4). The Beta version was administered to 814 adults, aged 18-80 years, 45% male, and 90% European American. Although this is not reflective of a multicultural sample, many of the questions were similar to scales that were normed using a more multicultural sample. Also, this research project was conducted in a rural area that has a majority European American sampling population, so the norms would generalize to the participant in this study.

Reliability. Gualtieri (2007) found that there was high internal consistency for the scales included in the final version of the NPQ, test-retest reliability for 74 patients in a 3 month period was significant ($p = .0001$) for an average $r = .74$ interrater reliability on the same day. Two different observer reports were also significant ($p = .0001$ to .002) for an average $r = .54$, and sensitivity to treatment with studies including pre and postinterventions and experimental-control studies showing changes for those that received interventions. The correlation between the NPQ-LF and NPQ-SF on the cluster

scales ranges from r = .33 to .96, test-retest reliability ranges from r = .53 to .82, and interrater reliability ranges between r = .39 to .74.

CNSVS Neurocognitive Test

The CNSVS Neurocognitive Test (Gualtieri & Johnson, 2008; Gualtieri, Johnson, & Benedict, 2004) was developed to detect neurocognitive impairments and is comprised of the Verbal and Visual memory, Finger Tapping, Symbol Digit Coding, Stroop, Shifting Attention, and the Continuous Performance Tests, which provide 17 primary scores and five domain scores. The indices are based on standard scores averaging at 100 with a standard deviation of 15 (Gualtieri & Johnson, 2008; Gualtieri et al., 2004). The subtests in the CNS VS consist of Symbol Digit Coding, Shifting Attention, Finger Tapping, Stroop Test, Continuous Performance Test, and Visual and Verbal Memory tests.

Validity. The norms were validated using 489 normal individuals ages 9-89, and standardized against other computerized tests (Gualtieri et al., 2004). The CNSVS also provides differential diagnostic categorizations between ADHD, traumatic brain injury, and dementia for over 1,000 patients. In a cross-sectional naturalistic study of 141 brain injury patients ages 18-65 years, the CNSVS was able to differentiate between level of traumatic brain injury (TBI) especially with regard to psychomotor speed and cognitive flexibility (Gualtieri & Johnson, 2008). The domain score were statistically significant (p < .05) for distinguishing TBI with control participants.

Reliability. The CNSVS is considered reliable between a 12-day interval retest (r = .45-.84, N = 155; Gualtieri et al., 2004). Recently, CNSVS has been used to determine the efficacy of neurofeedback for improving attentional control using the core battery

such as the CPT, ST, verbal and visual memory, and SDC (Kouijzer, de Moor, Gerrits, Buitelaar, et al., 2009a; Kouijzer, de Moor, Gerrits, Congedo, et al., 2009b). The researchers showed that there were statistically significant differences between the treatment and control groups on three separate administrations with a pre/postmeasure over 3 months and again after 12 months, demonstrating sustained for the experimental group. Therefore, it was a useful measure for testing neurocognitive changes in the participant of this study during the course of neurofeedback.

Test of Nonverbal Intelligence

Brown, Sherbenov, and Johnsen (2010) developed the Test of Nonverbal Intelligence (TONI) as an overall cognitive ability measure for those who have sensory deficits or have language difficulties or differences, which are concerns for individuals with ASC (APA, 2000, 2010; CDC, 2007, 2009). The TONI-4 is the latest edition, which reduced biases and increased validity and reliability associated with certain demographics such as gender and ethnicity (Johnsen et al., 2010). The TONI was developed through 307 items that were reviewed by experts in psychological testing and presently contains 60-items for each form. This measure was selected for use in this study based on an unpublished study by Berman, Sudol, Miller, and Berman (2005) with 10 children with ASC who were provided neurofeedback. Berman et al. (2005) found statistically significant improvement ($p < .005$) in pre and post TONI-3 scores for participant. Neurofeedback may improve global intelligence scores in children with ASC, and was considered a measure that may be helpful in evaluating the efficacy of neurofeedback in adults.

Validity. The TONI was normed across two time periods and administered to a total of 3,451 participants (Brown et al., 1997), and the most recent version TONI-4, was normed on a sample of 2,272 people in 32 states and included stratification of the sample (Brown et al., 2010). The TONI is largely representative of the U.S. population by geography, gender, community type, ethnicity/race, disability, and socioeconomic status, and age groups ranging from ages 6-0 to 89-11. The TONI is significantly correlated to the Wechsler Adult Intelligence Scale-Revised, with correlations of .57 and .58 for verbal and .75 and .76 for performance on the A and B forms respectively, correlation for general aptitude was a median of .52 (Brown et al., 1997). The item analyses was conducted using a point-biserial correlation, and the TONI was identified having .33 or higher and p value mean of .50. The construct validity consisted of six types of evidence: observed relationship between TONI and intelligence, correlation to school performance, performance ranges from gifted to significantly impaired individuals coincided with what was expected, strong predictor of full battery of intelligence testing, indicated by a single strong factor, and item point biserials by age group of .49 for form A and .50 for form B. The correlation between the TONI-4 and TONI-3 is very large with correlation coefficients of 74.

Reliability. Reliability was evaluated in four ways for the TONI-4: coefficient alpha, alternate forms, test-retest, and interscorer. The TONI-4 maintained high reliability at an average of 96 coefficient alpha on both forms with a standard error of measure from two to four (Brown et al., 2010). Alternate-forms correlation averaged .84 for all subjects. The test-retest correlations with 1 to 2-week separation correlation coefficient averaged 87 for both forms and across the sample. Finally, the interscorer

reliability held near-perfect .99 correlation coefficient. The TONI has maintained reliability since early versions of the test (Brown et al., 1997).

Quantitative Electroencephalogram

The participant received a QEEG assessment prior to neurofeedback training and at the conclusion of training. QEEG assessment is a measurement of real-time EEG function at multiple locations on the scalp simultaneously (Thatcher & Lubar, 2008). In addition to a measurement of functional patterns at each individual location, a measure of the interplay among the sites, including timing and similarity, is obtained (Thatcher & Lubar, 2008). EEG measures include absolute power, relative power, power ratio, coherence, asymmetry, and phase within eight bandwidths and individual bins (Thatcher & Lubar, 2008).

The QEEG assessment was implemented as directed by the standards set forth by Hammond and Gunkelman (2001). Prior to the EEG assessment, the participant was provided with information on how the assessment was done and given instructions on how to prepare for the assessment. He was instructed to avoid alcohol and over-the-counter medications prior to the assessment, to get at least 8 hours of sleep the night before, to thoroughly wash his hair with shampoo the morning of the assessment and to avoid the use of hair products. During the pre and postassessments, an appropriately sized elastic cap fitted with EEG electrodes (ECI Electro-Cap, Electro-Cap International, Eaton, OH) was placed on the participant's head and adjusted for symmetry and proper electrode placement. The electrodes were filled with conductive gel using a syringe and impedances were 5 Kohms or below.

EEG data were recorded with a Lexicor 24-channel digital EEG recording device using Neurolex™ software. The EEG recorded at 256 samples per second with high pass filter in the off position in two conditions–eyes closed for 10 minutes and eyes open for 10 minutes. The clinician paused periodically to ensure participant alertness and comfort. The EEG records were visually and automatically edited for artifact and processed using the Neuroguide Deluxe software and the Lifespan Normative Database (Applied Neuroscience, Inc.). This software has been normed with 625 individuals ages 2 months to 82 years with EEG acquisition eyes open or closed (Collura & Thatcher, 2006; Collura et al., 2009; Thatcher & Lubar, 2008). In addition to Neuroguide software, low resolution brain electromagnetic tomography (LORETA) is a functional brain imaging method that statistically maps neurophysiological processes through a three-dimensional anatomical generic model of the brain (Pascual-Marqui, Michel, & Lehmann, 1994). The LORETA and QEEG maps were used to guide the training protocol and track changes in neurophysiological functioning. These measures included probability measures of .001 to .06 ranges for significance. Significant changes were noted by visual inspection in changes of color.

Monitoring of Side Effects Scale

The face validity for the Monitoring of Side Effects Scale (MOSES; Kalachinik, 2001) was derived from peer-reviewed articles for side effects of psychopharmacologic and anticonvulsant medications. The MOSES was helpful in comparing side effects relative to medication interventions in autism. The scales range from zero (none) to four (severe), and are divided in the following categories: Ears/Eyes/Head, Mouth, Nose/Throat/Chest, Musculoskeletal/Neurological, Urinary/Genital, Gastrointestinal,

Skin, and Psychological. The procedure takes up to 5 minutes, maintains a high sensitivity for identifying side effects, and low specificity or false negatives/positives. For the purpose of this study, the only areas that were evaluated were the sections on Neurological and Psychological side effects each week during the baseline and neurofeedback phases (see Appendix I).

Overview of Dependent Variables

Table 1 provides the overview of the DV that were examined in this study. It is important to note that Chapter 2 provides a detailed account of the complexity of ASC. Included in the literature review, autism has symptoms associated with deficits in social-communication, ADHD, mood dysregulation, executive functioning, processing of information, and neurophysiology. These DVs provide a comprehensive assessment of neurofeedback's effect on ASC symptoms and related issues. Coben and Padolsky (2007) and other researchers in neurofeedback have used similar multiple baseline measures in order to adequately cover the broad deficits in ASC. Unique to this study was that this was the first neurofeedback research in ASC to use measures assessing comorbid disorders of autism and self-reports by the participant rather than other reporters like parents and teachers. This provided the opportunity for the participant to quantify changes in overall mental health related issues particularly in areas like depression and anxiety.

Table 1

Dependent Variables

	Variable	Assessment
Core Autism Symptoms	Asperger's	NPQ-LF
Neuropsychological Index 1	ADHD	NPQ-LF
Neuropsychological Index 2	Mood Stability	NPQ-LF
Neuropsychological Index 3	Anxiety	NPQ-LF
Neuropsychological Index 4	Depression	NPQ-LF
Neurocognitive Ability 1	Executive Function	CNSVS
Neurocognitive Ability 2	Processing Speed	CNSVS
Intelligence Measure	Nonverbal Intelligence	TONI
Neurophysiological Function	QEEG	Neuroguide
Adverse Effects	Neurological/Psychological	MOSES

Procedure

Phase A

Phase A consisted of the initial convenience-criterion sample selection of a volunteer participant solicited by the neurofeedback clinic. The principal investigator had no input to the manner in which the participants was identified. The adult participant with ASC accepted into the study was willing to participate in testing and assessment procedures while he received neurofeedback by the clinic. It was assumed that the participant hoped to receive benefit from the neurofeedback in improving symptoms. If he was in need of more comprehensive treatment services (e.g., medication intervention,

crisis assessment), then he would have been referred as needed by clinic. This was not the case for the participant who consented for the study.

The initial step required a verification of the ASC diagnosis by a licensed healthcare professional, and a release form was used to access only the evaluation confirming the diagnosis, which was confirmed. The pretest or baseline consisted of gathering pertinent background information and pretesting measures. At the initial session, the participant provided informed consent and an explanation of the assessment and testing, which consisted of the measures listed below. The initial evaluation required a total of 90 to 120 minutes and was separated in three individual sessions to prevent fatigue from the testing and assessment procedures.

The measures and administration time included

1. The NPQ- LF (approximately 15 minutes, baseline consisted of five administrations, and reassessed five times during the neurofeedback phase).
2. CNSVS Neurocognitive Test (approximately 15 minutes, baseline consisted of five administrations, and reassessed five times during the neurofeedback phase).
3. TONI (approximately 15 minutes, three times in baseline and three times in the neurofeedback phase).
4. QEEG (approximately 90 minutes, consisted of pre-treatment and post-treatment records).
5. MOSES (approximately 5 minutes, baseline consisted five administrations, and reassessed five times during the neurofeedback phase).

During the first week, the participant was administered the testing and

assessments three to five times to establish a baseline. The participant was also frequently assessed for emotional distress such as psychotic symptoms or risk of harm to self or others using the subscales on the NPQ.

Phase B

In phase B, the neurofeedback clinic provided 30-minute neurofeedback LZT sessions, four times per week, for approximately 5 weeks. The neurofeedback clinic provided all neurofeedback services separately from the research activities, which were the principal investigator's responsibilities. The LZT consisted of viewing a computer screen with video and audio feedback. In Figure 6, the participant attempted to create more planets in the solar system by meeting the reward threshold for EEG activity.

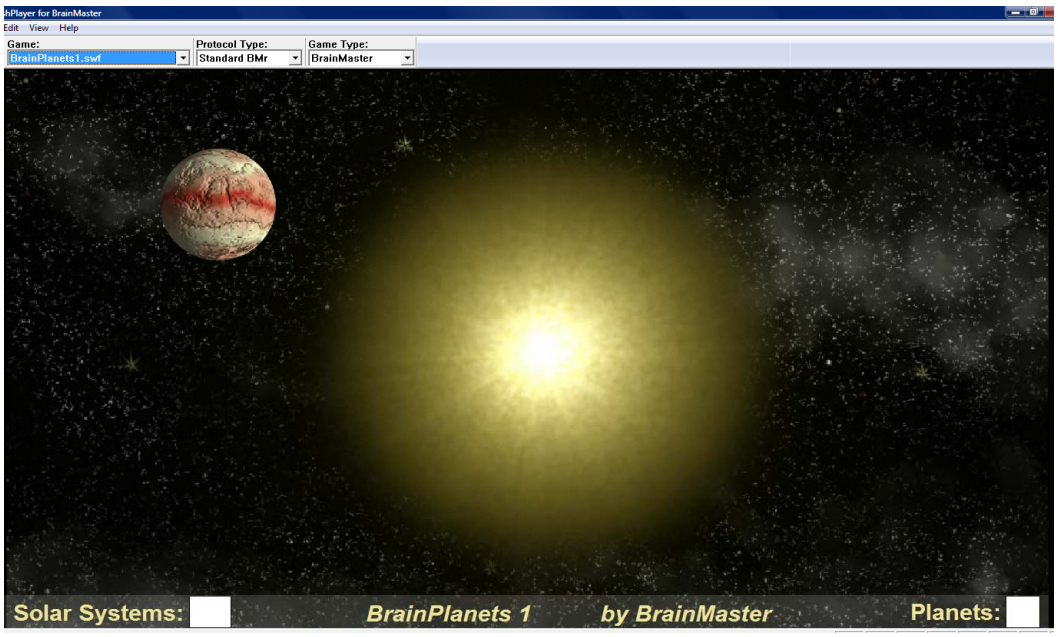


Figure 6. Brainmaster flash player Brain Planets

The neurofeedback clinic scheduled ahead for the prospective participant to conduct sessions at the same time of day to control for ultradian and circadian effects (Kaiser, 2008). The EEG recording had continuous real-time impedance checking to

maintain impedance below 10 K ohms (Coben & Padolsky, 2007).

At the end of each treatment week, the NPQ-LF, CNSVS Neurocognitive Test, and MOSES were administered. After the participant completed 20 sessions of neurofeedback training he completed all postmeasures including the CNSVS, NPQ-LF, TONI, QEEG, and MOSES. It required approximately 2 hours for posttesting. Phase B also provided dissemination of findings to the participant and his mother by phone and in person. The participant was asked to provide any subjective reports with a perspective on each research question and purpose of the study. Provisions of additional referrals were provided to local community mental health providers. If the participant had dropped out of the study before the 20 sessions then he would have been asked to offer feedback regarding early termination and provided any additional referrals if requested to local service providers. However, he completed 20 sessions without any adverse events. He was provided mileage reimbursement of 50 cents per mile according to the latest federal rate in the IRS Publication 17, Chapter 26.

Research Questions and Hypotheses

1. Is neurofeedback LZT related to a change in the core symptoms of autism in an adult with ASC?

$H_0 1$: $\mu_1 = \mu_2$ –There will be no significant differences in ASC symptoms as measured by the Neuropsych Questionnaire, Long Form (NPQ-LF) during the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

$H_1$1: $\mu_1 > \mu_2$ –There will be significant reduction in ASC symptoms as measured by the NPQ-LF between the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

The analysis consisted of the CDC visual inspection method. The CDC method provides two superimposed criterion lines in the neurofeedback phase to determine an effect. The criterion lines were established by calculating the trend line and baseline data's mean, which was then modified by lowering both lines by .25 standard deviations of the baseline data. According to the binomial test, all five data points in the neurofeedback phase must fall below both the CDC trend line and baseline in order to show a reliable effect.

2. Is neurofeedback LZT related to a significant reduction in neuropsychological symptoms associated with ADHD, anxiety, depression, and mood stability of an adult with ASC?

$H_0$2: $\mu_1, \mu_2, \mu_3, \mu_4 = \mu_6, \mu_7, \mu_8, \mu_9$ –There will be no significant differences in ADHD, Anxiety, Depression, and Mood Stability indices as measured by the NPQ-LF during the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

$H_1$2: $\mu_1, \mu_2, \mu_3, \mu_4 > \mu_6, \mu_7, \mu_8, \mu_9$ –There will be significant reduction in ADHD, Anxiety, Depression, and Mood Stability indices as measured by the NPQ-LF between the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

The analysis consisted of the CDC visual inspection method. The CDC method provides two superimposed criterion lines in the neurofeedback phase to determine an

effect. The criterion lines were established by calculating the trend line and baseline data's mean, which was then modified by lowering both lines by .25 standard deviations of the baseline data. According to the binomial test, all five data points in the neurofeedback phase must fall below both the CDC trend line and baseline in order to show a reliable effect.

3. Is neurofeedback LZT related to a significant improvement in neurocognitive abilities in executive functioning and processing speed in an adult with ASC?

$H_0 3$: $\mu_1, \mu_2 = \mu_1, \mu_2$ –There will be no significant differences in executive functioning and processing speed as measured by the CNS Vital Signs (CNSVS) Neurocognitive Test during the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

$H_1 3$: $\mu_1, \mu_2 < \mu_1, \mu_2$ –There will be significant increase in executive functioning and processing speed as measured by the CNSVS Neurocognitive Test between the baseline and neurofeedback phases in a participant who receives 20 sessions of neurofeedback LZT.

The analysis consisted of the CDC visual inspection method. The CDC method provides two superimposed criterion lines in the neurofeedback phase to determine an effect. The criterion lines were established by calculating the trend line and baseline data's mean, which is then modified by raising both lines by .25 standard deviations of the baseline data. According to the binomial test, all five data points in the neurofeedback phase must fall above both the CDC trend line and baseline in order to show a reliable effect.

4. Is neurofeedback LZT related to significant overall improvement in nonverbal intelligence in an adult with ASC?

$H_0$4: $\mu_1 = \mu_2$ –There will be no significant difference in general intelligence as measured by the TONI between baseline and post-test quotient scores in a participant who receives 20 sessions of neurofeedback LZT.

$H_1$4: $\mu_1 < \mu_2$ –There will be a significant increase in general intelligence as measured by the TONI from baseline to post-test quotient scores in a participant who receives 20 sessions of neurofeedback LZT.

A simple line graph plotting three baseline and three treatment phase data points was used. A significant change in the TONI was analyzed by visual inspection with an observable change in mean performance above the standard error of measurement between baseline and treatment phase data points.

5. Is neurofeedback LZT related to normalization in QEEG measures in an adult with ASC?

$H_0$5: $\mu_1 = \mu_2$ –There will be no significant differences in neurophysiological functioning as measured by QEEG based on the ANI DLL and LORETA statistical software in a participant who receives 20 sessions of neurofeedback LZT.

$H_1$5: $\mu_1 < \mu_2$ – There will be significant changes in neurophysiological functioning as measured by QEEG based on the ANI DLL and LORETA software in a participant who receives 20 sessions of neurofeedback LZT.

QEEG and LORETA maps provided visual inspection of changes in brain function toward normalization. There were also paired-sample *t*-test analyses that provided areas of statistically significant change ranging from .06 to .001. The maps

were chosen based on the most significant changes and to illustrate main themes of findings.

Variables

IV – Time (i.e., baseline and neurofeedback sessions)

DV –Self-reported autism symptoms (i.e., NPQ-LF)

DV –Self-reported symptoms (i.e., ADHD, Anxiety, Depression, Mood Stability)

DV – Neurocognitive measures (i.e., executive functioning, processing speed)

DV – Intelligence measure (i.e., nonverbal IQ)

DV – Neurophysiological measure (i.e., QEEG difference)

DV – Adverse effect measure (i.e., psychological and neurological areas)

Protection of Participant

The following procedures were followed to protect the rights and best interests of the participant: introduction of and discussion of the background of the study, informed consent process throughout the study, and disclosures. Information was provided regarding the purpose of the study, the voluntary nature of the study, participant rights to confidentiality, the limits of confidentiality, the participant's ability to discontinue the study at any time, and details about the benefits versus risks. Testing and questionnaires are considered minimally invasive. Nevertheless, the study also provided continued monitoring of any potential adverse effects using the MOSES. The MOSES helped to provide an integration of both the principal investigator's observations and the participant's self-reports, and was used as a continued dialogue of informed consent and whether the participant wished to continue the study. The participant was offered the ability to opt out of the research project at any time. The principal investigator completed

the National Institutes of Health Office of Extramural Research web-based training course "Protecting Human Research participants;" certification number 67892. This course provided a basis for the development of the methodology and ethical approach toward the development of tools such as the informed consent that is in compliance with federal standards. The Walden Institutional Review Board approved this study (Approval number 09-08-11-0072997).

Summary and Transition

The purpose of Chapter 4 was to construct the procedures of this study including the research questions, hypotheses, recruitment of the participant, data collection, measurements, and data analysis. The neurofeedback clinic was responsible for identifying the volunteer participant and providing the neurofeedback, which was separate from the proposed research study that included testing and assessment procedures only. The proposal for a single-case design was defended based on the need for improved quantitative research methods in neurofeedback and the benefits of exploring the effects of it from an idiographic perspective. Further, the sample size is adequate to determine an effect based on the historically large effect sizes found in neurofeedback research, number of repeated measures to regularly assess temporal change, and broad analysis across various domains of functioning. Single-case research has been a major contributor to the literature on applied behavioral research particularly in autism.

Chapter 4: Results

Introduction to Results

The purpose of this study was to examine the effects of neurofeedback LZT in an adult with ASC with neurocognitive and neuropsychological functioning. This section will provide an overview of the sampling method and participant. The data collection and research question analysis will be provided to evaluate each hypothesis and null hypothesis. All the research questions will have data graphed for visual inspection along with a review of the results obtained from the study and review of findings.

Overview of Sampling

Recruitment

A convenience sample was conducted at a local neurofeedback clinic through an advertorial and introductory letter (see Appendices A and B), as described in Chapter 3. One potential participant was interested in the study but was unable to devote the time involved as outlined in the advertorial and letter and so declined participation. Another prospective participant was interested in the study but was being transferred to a facility, which was an exclusion criterion. A third prospective participant met all the criteria for the study, agreed with the informed consent materials, and committed to participation in the study.

Sample

The participant was a 22 year-11 month old right-handed single European American male without children and living with his biological parents. His diagnostic formulation consisted of Asperger's disorder, bipolar disorder, expressive communication disorder (i.e., apraxic speech), impulse control disorder not otherwise specified, and

anxiety disorder not otherwise specified. Medical concerns consisted of a Chiari malformation, a left thalamic mass and static lesion, and partial complex seizures. He had obtained a high school diploma, was able to read all forms, and answered all comprehension questions. He performed in the average range of intelligence, 96 Nonverbal IQ on the TONI. During the baseline phase, his depression index scores according to the NPQ-LF fell in the moderate range, which was below the cut off for this study (i.e., severe range). He was taking the following medications: Abilify 12.5 mg tabs QHS, sertraline HCL 100 mg QAM, and divalproex SOD ER 1750 mg.

Research Question Analysis

Research Question 1

Is neurofeedback LZT related to a change in the core symptoms of autism in an adult with ASC?

H_1: $\mu_1 > \mu_2$ –There will be a significant reduction in ASC symptoms as measured by the NPQ-LF between the baseline and neurofeedback phases in the participant who received 20 sessions of neurofeedback LZT.

The null hypothesis was rejected. The participant's reported Asperger's symptoms were significantly reduced between baseline and neurofeedback phase according to the CDC method with all five data points in the treatment phase falling below the modified linear regression line and mean baseline. During the baseline phase, the Asperger's index confirmed the participant's diagnosis of ASC with a self-reported moderate level of impairment ($M = 171$). During the course of treatment, this index reduced significantly to the mild range of impairment ($M = 129$).

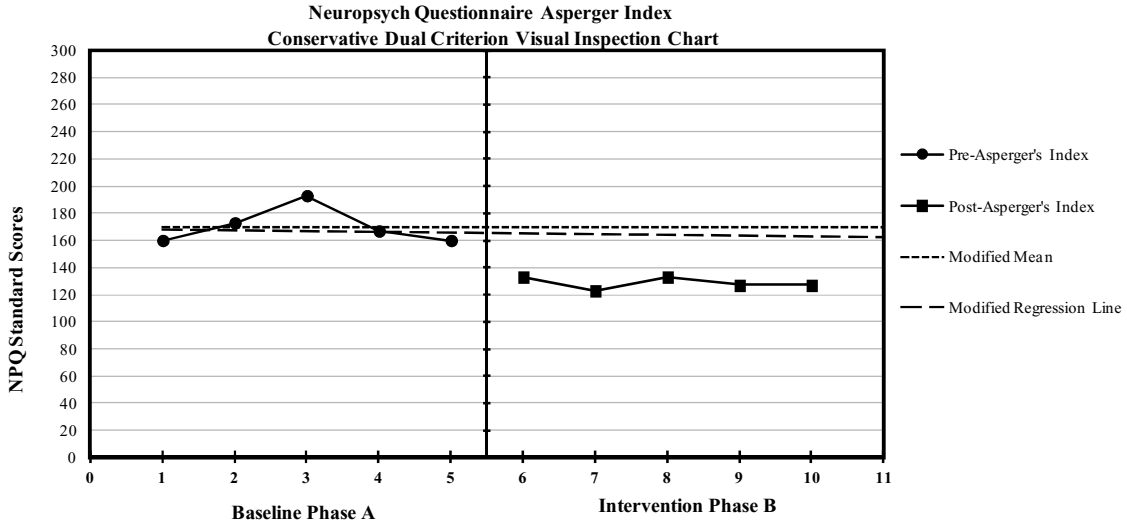


Figure 7. Asperger's index displays a significant improvement.

Research Question 2

Is neurofeedback LZT related to a significant reduction in neuropsychological symptoms associated with ADHD, anxiety, depression, and mood stability of an adult with ASC?

H_2: $\mu_1, \mu_2, \mu_3, \mu_4 > \mu_6, \mu_7, \mu_8, \mu_9$ –There will be a significant reduction in ADHD, Anxiety, Depression, and Mood Stability indices as measured by the NPQ-LF between the baseline and neurofeedback phases in the participant who received 20 sessions of neurofeedback LZT.

The null hypothesis was rejected. Figures 8, 9, 10, and 11 indicate a significant reduction in ADHD, Anxiety, Depression, and Mood Stability according to the CDC method with all five data points in the treatment phase falling below the modified linear regression line and mean baseline in all of the measures. Mood stability fell from moderate (M = 197) to mild (M = 128) range, anxiety reduced from moderate (M = 190) to mild (M = 128) range, depression had the greatest decrease from moderate (M = 182)

to mild (*M* = 108) range, and although ADHD did not change in level of severity, the scores significantly reduced (baseline *M* = 233; treatment *M* = 164).

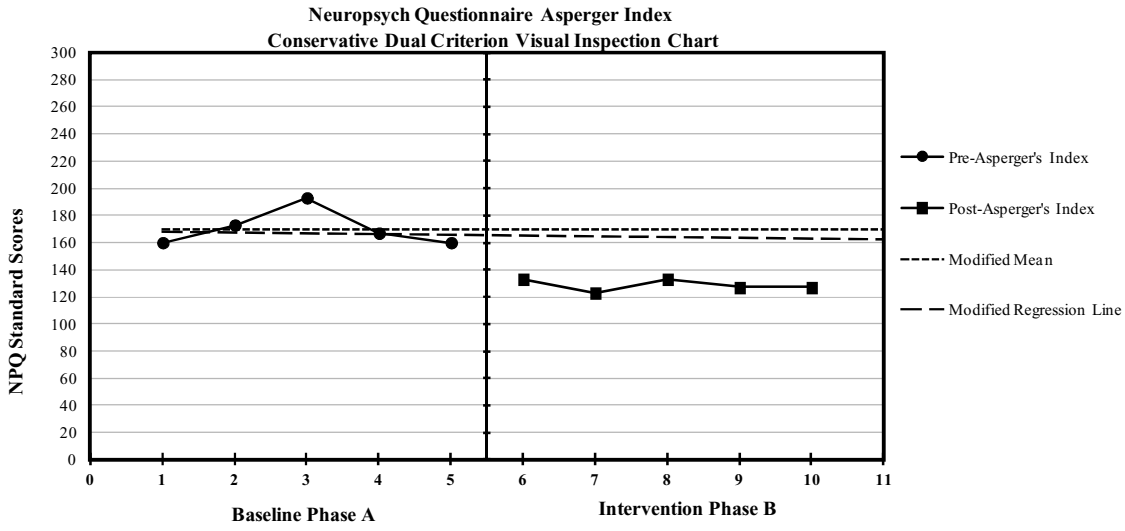


Figure 8. ADHD index displays a significant improvement.

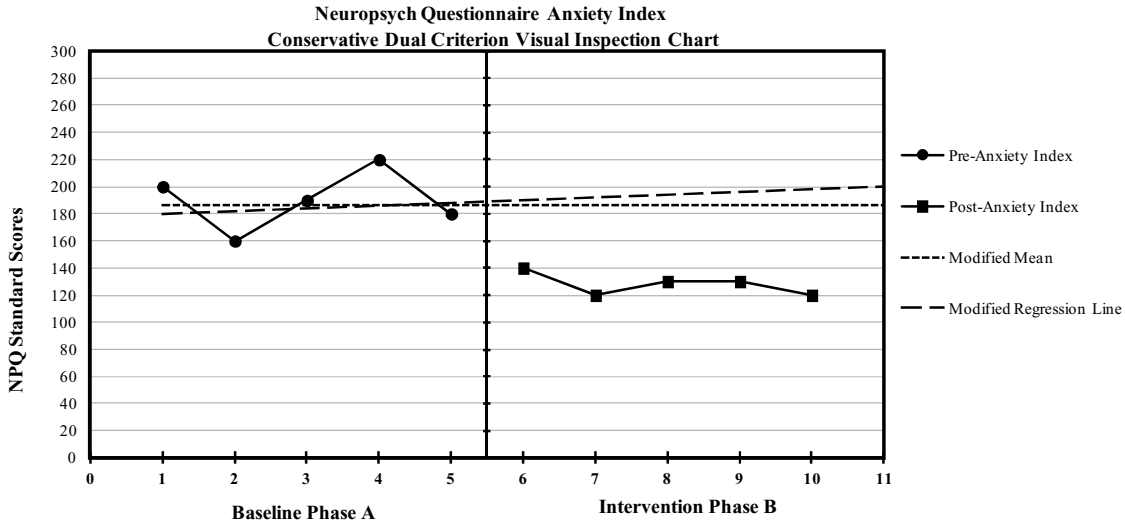


Figure 9. Anxiety index displays a significant improvement.

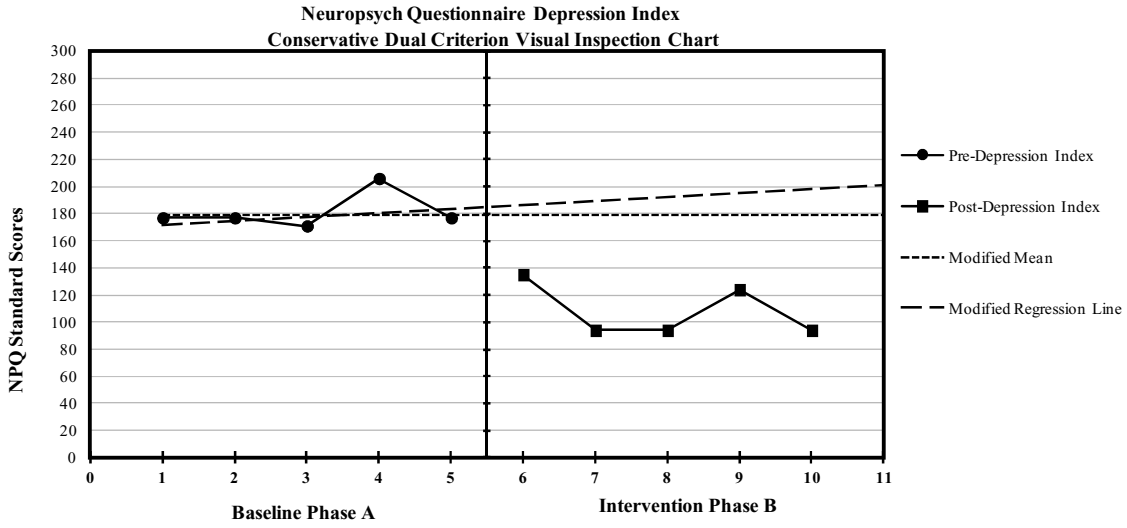


Figure 10. Depression index displays a significant improvement.

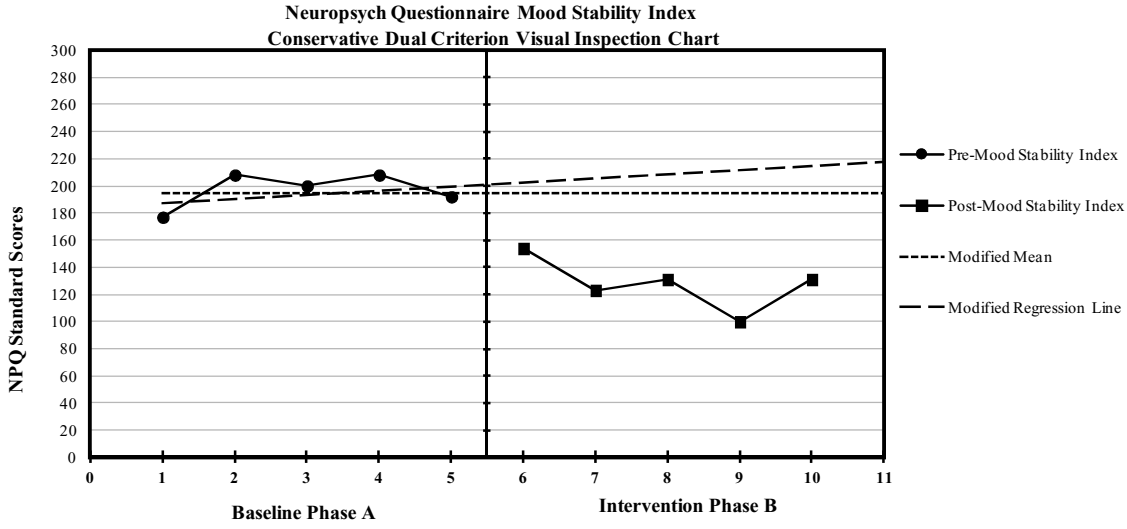


Figure 11. Mood Stability index displays a significant improvement.

Research Question 3

Is neurofeedback LZT related to a significant improvement in neurocognitive abilities in executive functioning and processing speed in an adult with ASC?

H_3: $\mu_1, \mu_2 < \mu_1, \mu_2$ –There will be a significant increase in executive functioning and processing speed as measured by the CNSVS Neurocognitive Test between the baseline and neurofeedback phases in the participant who received 20 sessions of neurofeedback LZT.

The null hypothesis was rejected for executive function, but was failed to reject the null hypothesis for processing speed. Figure 12 indicates a significant improvement in the CNSVS Executive Function index score according to the CDC method with all five data points in the treatment phase above the modified linear regression line and mean baseline. Executive function increased from borderline (M = 76) to low average (M = 88) range of functioning in the treatment phase.

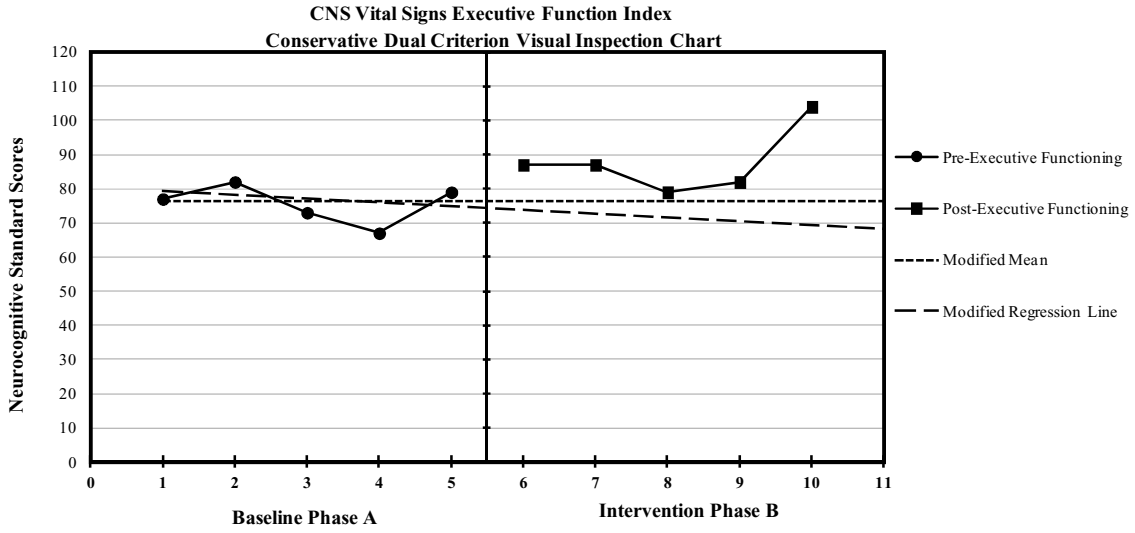


Figure 12. Executive Function displays a significant improvement.

There were, however, no appreciable differences in processing speed as measured by the CNSVS Neurocognitive Test between the baseline and neurofeedback phases. One of the data points fell below the modified trendline and baseline. However, there was a trend toward improvement and score increased from borderline (*M* = 79) to low average (*M* = 84) range of abilities in the treatment phase.

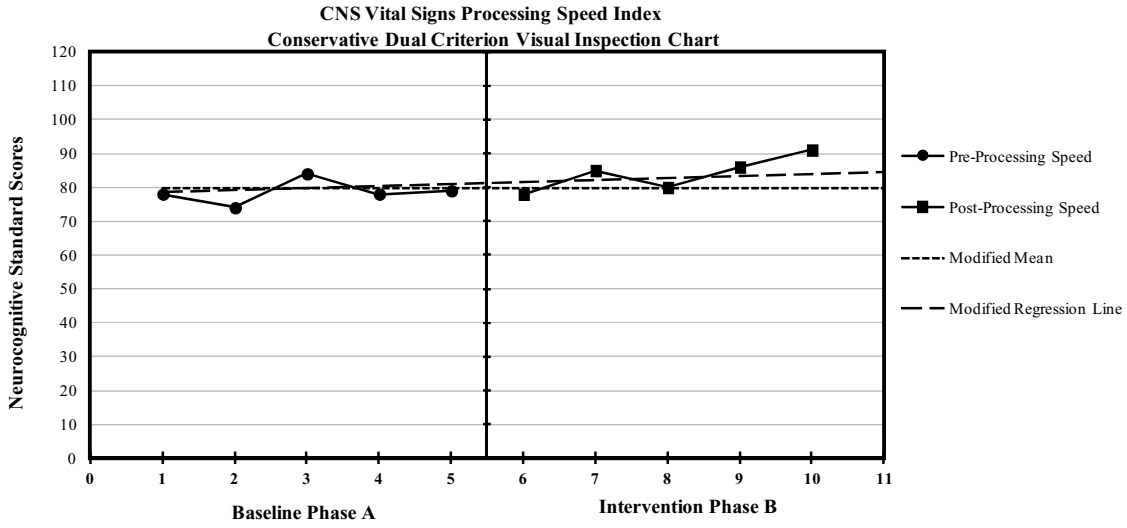


Figure 13. Processing Speed index displays no significant effect.

There were additional findings not included in the hypotheses that provided further evidence of neurocognitive changes associated with executive functioning and processing speed. Cognitive flexibility, complex attention, and reaction time indices significantly improved as indicated by the CDC method with all five data points above the modified linear regression line and baseline mean in all of the measures below.

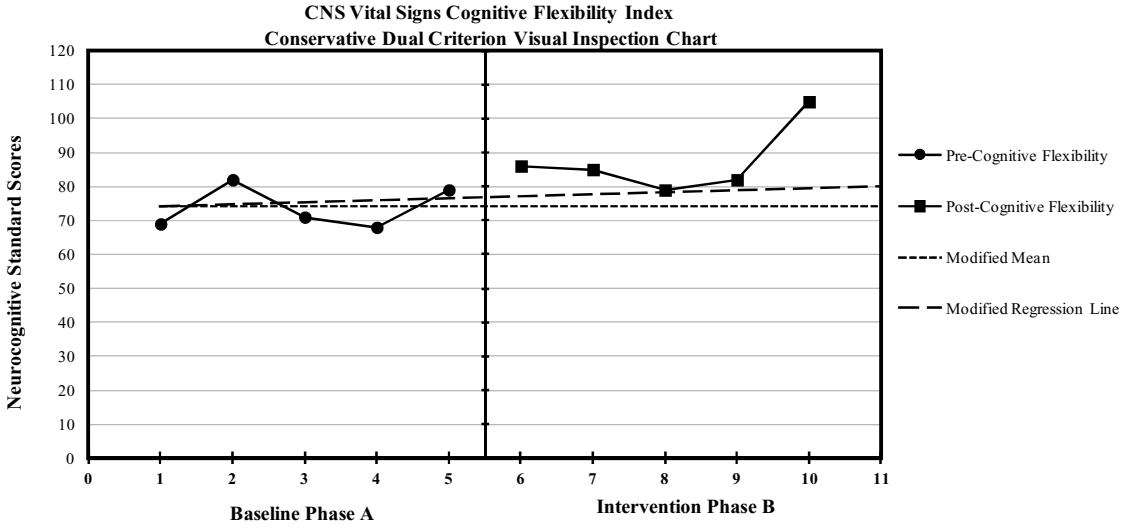


Figure 14. Cognitive flexibility index displays a significant improvement.

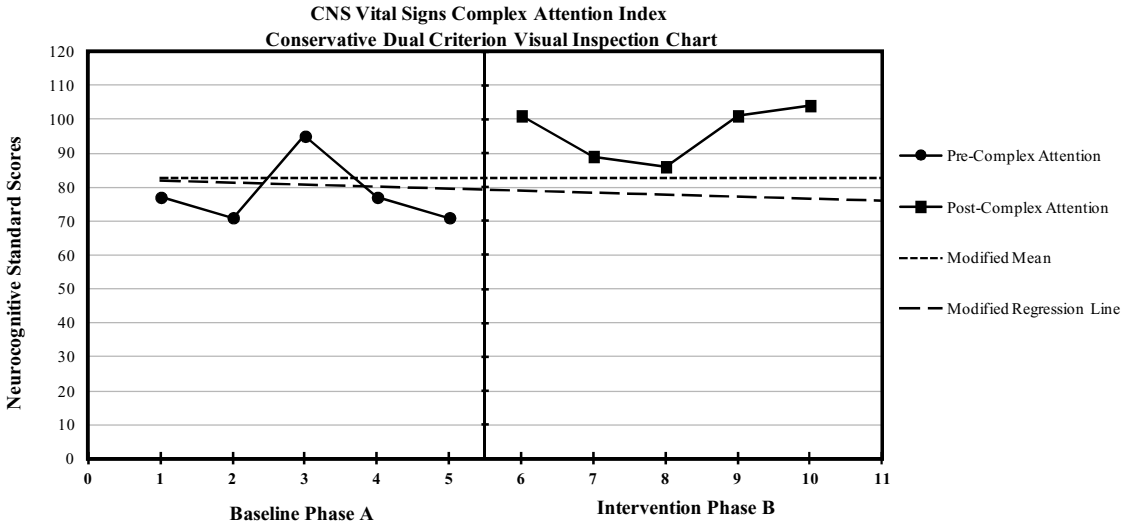


Figure 15. Complex attention index displays a significant improvement.

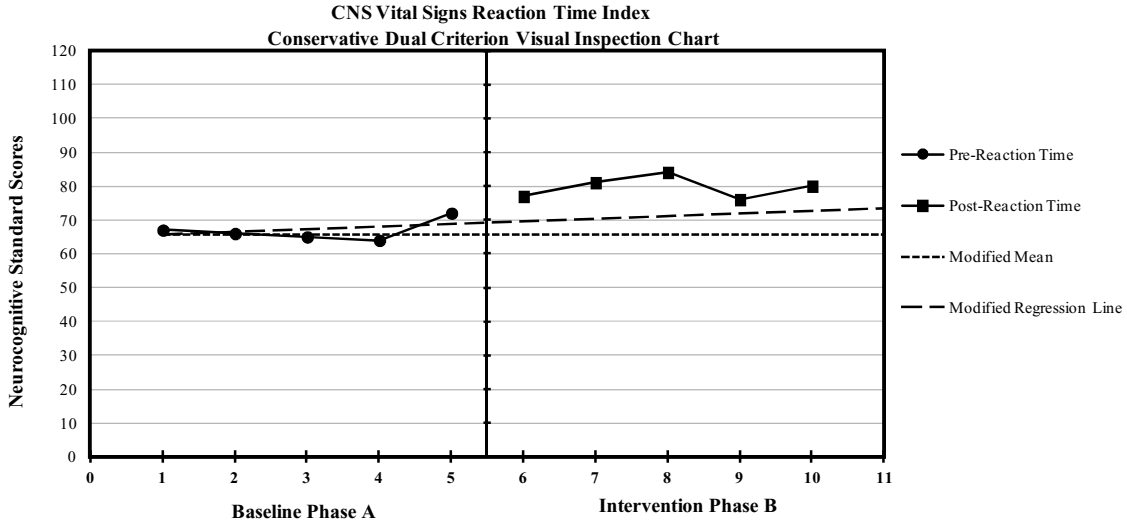


Figure 16. Reaction Time index displays a significant improvement.

Research Question 4

Is neurofeedback LZT related to significant overall improvement in nonverbal intelligence in an adult with ASC?

H_4: $\mu_1 < \mu_2$ –There will be a significant increase in general intelligence as measured by the TONI-2, TONI-3, and TONI-4 from baseline to intervention phase quotient scores in the participant who received 20 sessions of neurofeedback LZT.

The null hypothesis was rejected. The chart below indicates a significant improvement in the TONI IQ. Visual inspection depicts a significant change in mean performance from baseline nonverbal IQ scores (M = 97 NIQ) to treatment phase nonverbal IQ scores (M = 108). The change in performance was outside the standard error of measurement for the TONI (i.e., +/- 4), which further indicates a significant effect.

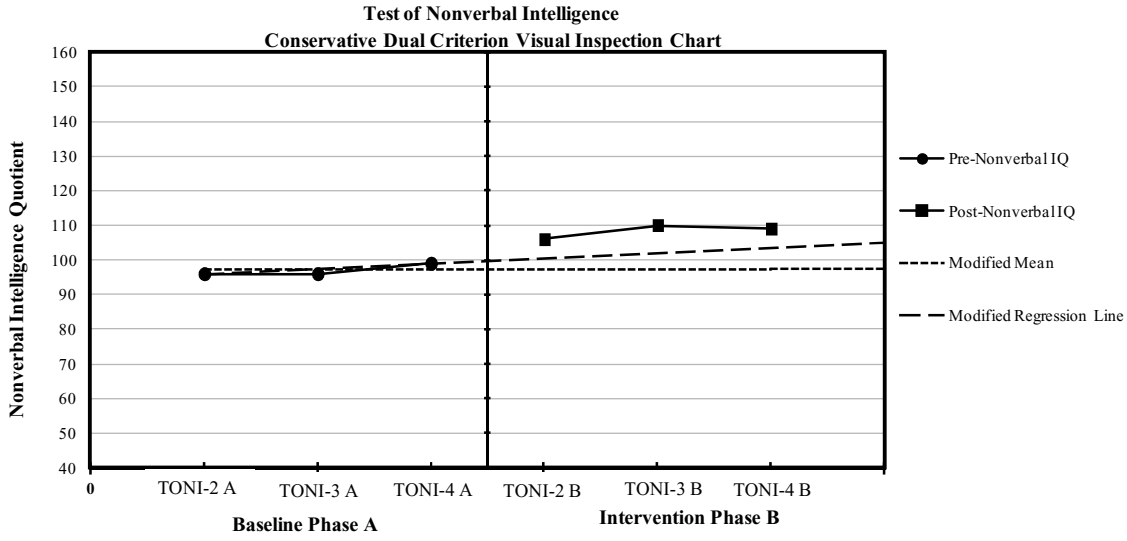


Figure 17. Nonverbal IQ displays a significant improvement.

Research Question 5

Is neurofeedback LZT related to normalization in QEEG measures in an adult with ASC?

H_5: $\mu_1 < \mu_2$ – There was significant changes in neurophysiological functioning as measured by QEEG based on the ANI DLL and LORETA statistical software in a participant who receives 20 sessions of neurofeedback LZT.

Hypothesis 5 was supported by the data and brain maps. There were changes in neurophysiological functioning according to pre and post-EEG recordings.

Baseline. During baseline, the participant presented with abnormal findings for his EEG with regard to absolute and relative power, connectivity, and paroxysmal waves. Specifically, he had excess 8 Hz and generally low voltage throughout his EEG record, and his peak alpha frequency was 9 Hz. Although paroxysmal waves were noted, they were not interictal or sustained and waves were transient. For connectivity measures in Figure 18, he presented with significant frontotemporal hypercoherence in beta and high

beta bandwidths. Phase lags existed in delta and theta for eyes closed frontal, temporal, and occipital sites. Amplitude asymmetry is depicted in right frontotemporal alpha and frontal beta.

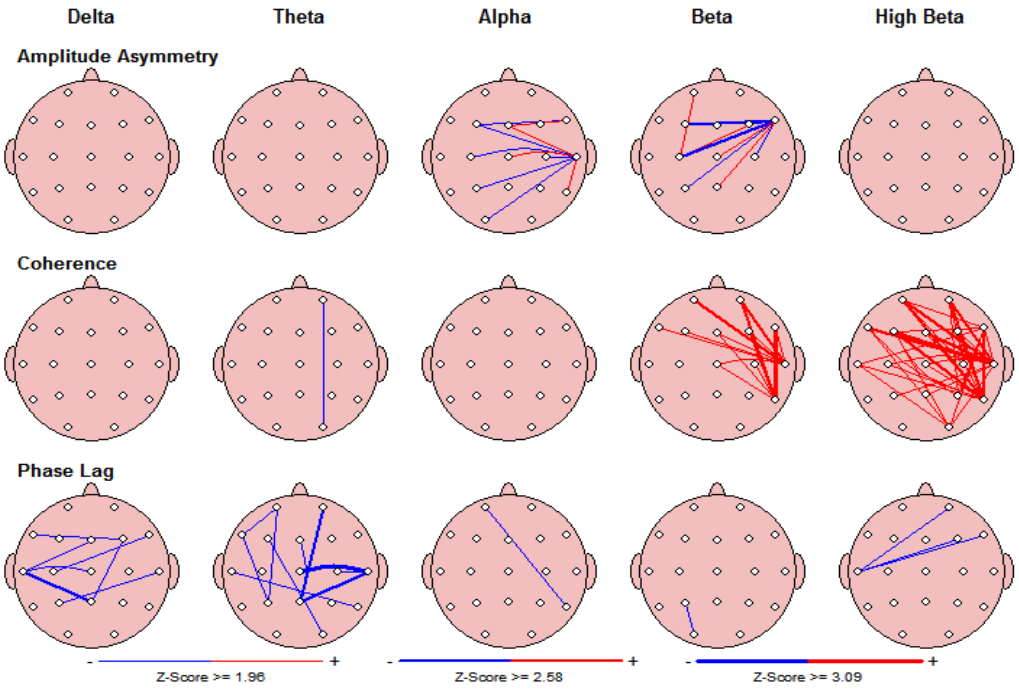


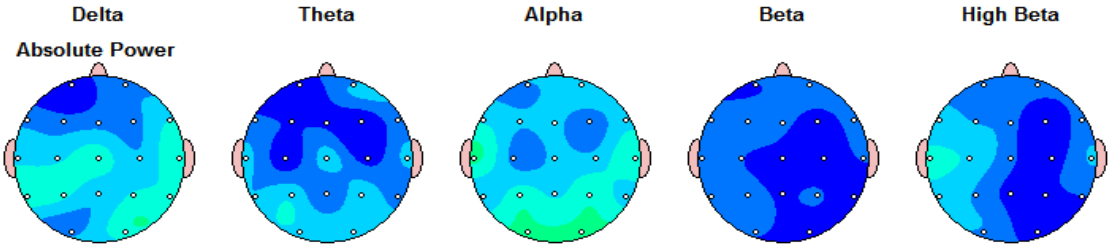
Figure 18. Baseline Eyes Closed QEEG Neuroguide connectivity maps.

Visual Inspection of Absolute Power. The following EEG acquisition maps were with eyes closed only using a Laplacian montage due to medication effects, which provides a reanalysis in difficult to interpret recordings (Rowan & Tolunsky, 2003). It should be noted that the posttreatment assessment was impacted by low-grade muscle tension and fatigue because the participant stayed up later than usual to celebrate a holiday festivity the prior evening. He denied any alcohol or other substance use. Therefore, the posttreatment EEG records should be interpreted with a measure of caution. The EEG recording was edited for artifact to reduce noise by a minimum of 60 seconds of artifact free data by a combination of manual selection and software-assisted

processing to eliminate EEG contamination by eye movement, muscle tension, and fatigue in order to obtain a sample of the EEG record representative of the client's overall functioning. The data exceeded commonly used standards in EEG analyses dictating a minimum split-half reliability above .95 and test-retest reliability exceeding .90.

Figure 19 provides pre and post-QEEG Neuroguide Z scored FFT summaries of absolute power maps for each frequency bandwidth. His posttreatment showed significant increases in theta, alpha, and beta power in frontal regions. This is noticeable by gradient shift from dark blue (representing three standard deviations below the norm) to light blue-green in the posttreatment maps below. Movement from blue to green represents a shift toward a more normalized and efficient state of cortical function and performance when compared to an age-matched normative group.

Pre-treatment



Post-treatment

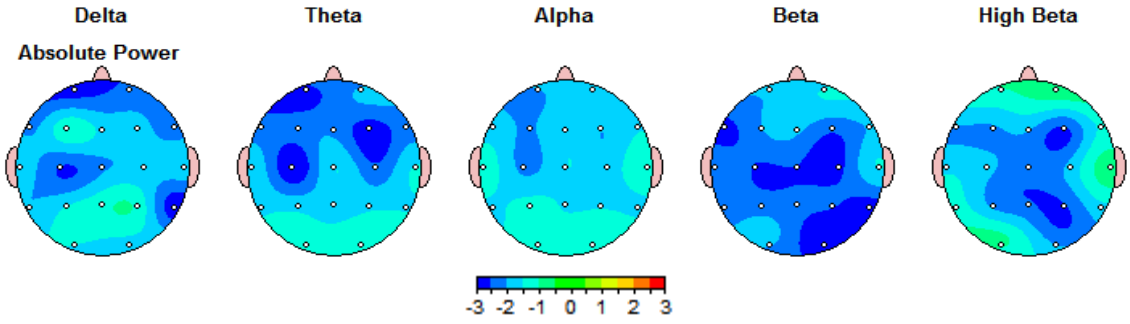
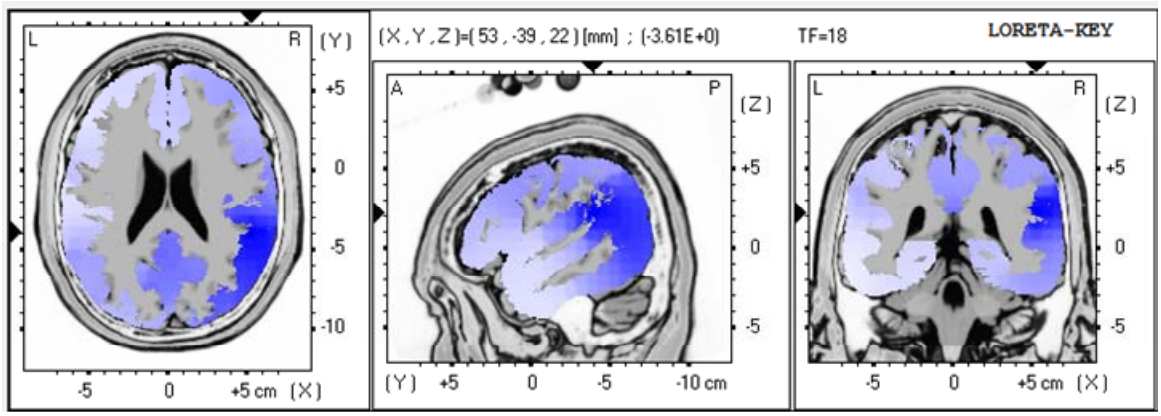


Figure 19. QEEG absolute power maps display significant effects.

LORETA Maps Comparison. In coordination with the neurofeedback clinic consultants, I examined frequency ranges between 1-40 Hz. They identified voxels indicative of significant change relevant to this study. Specifically, changes in beta frequency band were noted, and 18 Hz was chosen to depict normalization in the EEG. Each slide provides the Cartesian vector fields with coordinates on the *x*, *y*, and *z* axes depicting slices of a generic brain. Figure 20 provides the pretreatment in the top row and posttreatment in the bottom row. Each row consists of three individual maps consisting of the horizontal, sagittal, and coronal views of the brain respectively. The darker the gradient blue the more indicative of lowered absolute power in beta 18 Hz (i.e., abnormal brain function), whereas the areas with the light blue or no color signifies areas of normalized brain function. Figure 20 shows significant EEG normalization with the apparent gradient shifts from darker blue to lighter blue or no color in posttreatment LORETA. There is noticeable improvement particularly in the frontal, temporal, and parietal areas of the brain (i.e., Brodmann areas 21, 6, 17 and 18). Overall, the pre and posttreatment LORETA provide additional evidence of normalization in beta frequency, which further supports improvement in higher cognitive processing abilities like executive functioning and complex attention.

Pre-treatment



Post-treatment

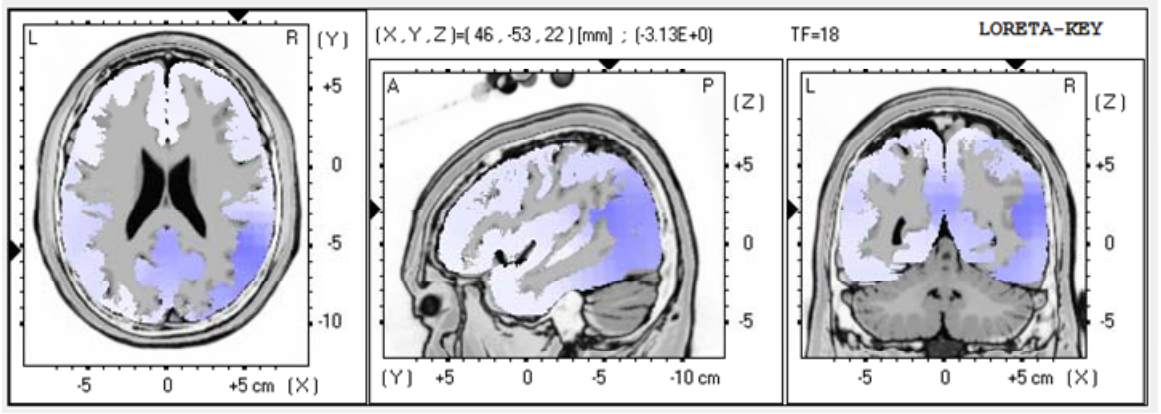


Figure 20. LORETA maps display normalization of absolute power in 18 Hz.

Side Effect Scales

The MOSES provided monitoring of side effects. The client indicated no change in neurological side effects symptoms. There was improvement in the psychological side effects, which is consistent with improved psychological symptoms in the NPQ-LF.

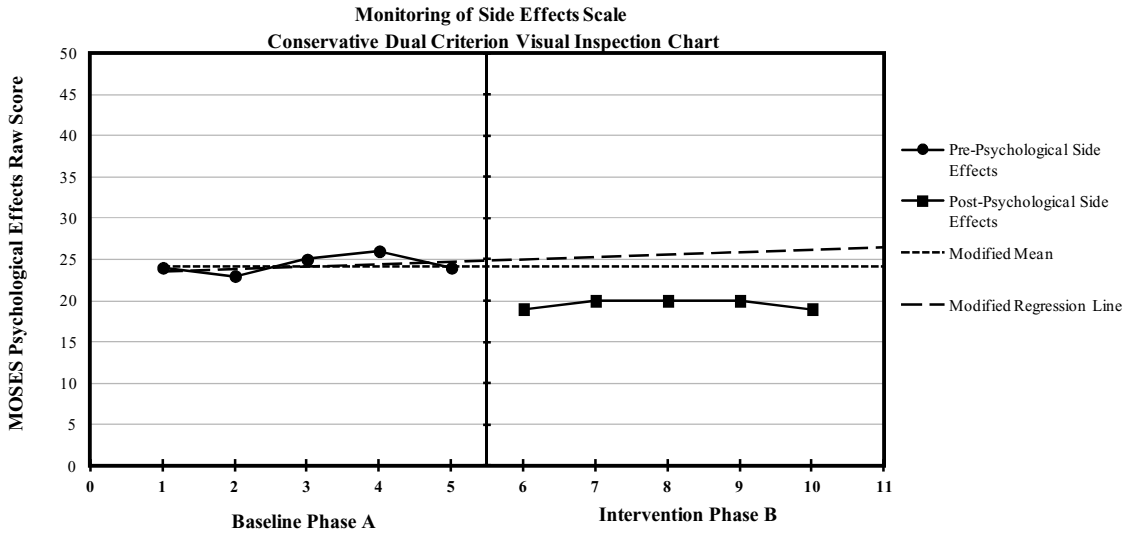


Figure 21. MOSES Psychological side effect profile.

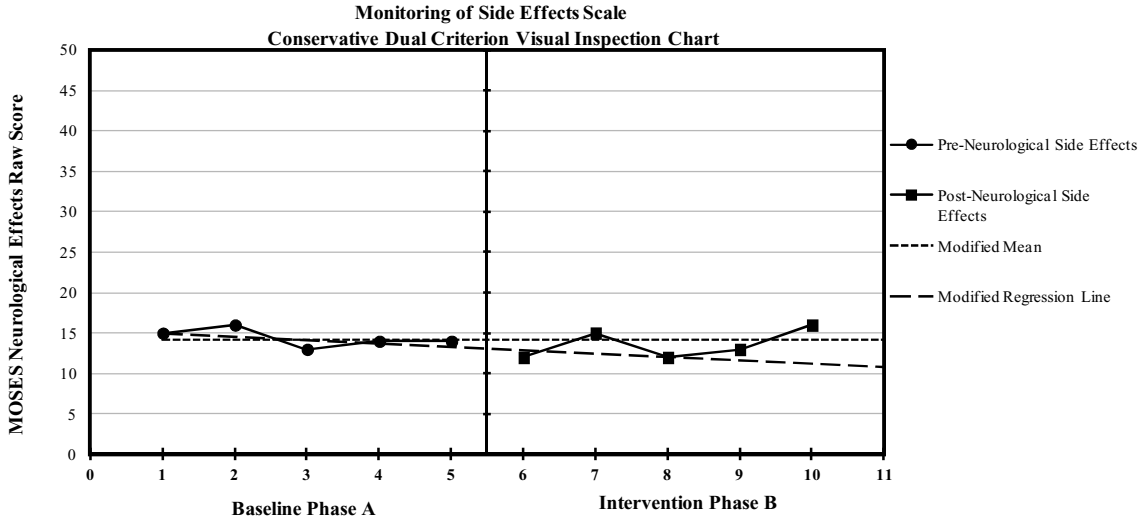


Figure 22. MOSES Neurological side effect profile.

Summary Tables

Tables 2, 3, 4, and 5 summarize the findings in self-report, neurocognitive, and side effect measures. Each table consists of the mean percentage change between baseline and treatment phases.

Table 2

Mean Change for Self-Reports

Dependent Variables	Baseline *M*	Treatment *M*	% Change
Asperger's NPQ Index	171	129*	25%
ADHD NPQ Index	233	164*	30%
Anxiety NPQ Index	190	128*	33%
Mood Stability NPQ Index	197	128*	35%
Depression NPQ Index	182	108*	41%

*Statistically significant improvement by the Conservative Dual Criterion.

Table 3

Mean Change for Neurocognitive Abilities

Dependent Variables	Baseline *M*	Treatement *M*	% Change
Processing Speed CNSVS	79	84	6%
Executive Function CNSVS	76	88*	16%
Cognitive Flexibility CNSVS	74	87*	18%
Reaction Time CNSVS	67	80*	19%
Complex Attention CNSVS	78	96*	23%

*Statistically significant improvement by the Conservative Dual Criterion and visual inspection methods.

Table 4

Mean Change for Intelligence

Dependent Variables	Baseline *M*	Treatement *M*	% Change
TONI	97	108*	12%

*Statistically significant improvement by the Conservative Dual Criterion and visual inspection methods.

Table 5

Mean Change for Side Effects

Dependent Variables	Baseline *M*	Treatment *M*	% Change
Neurological MOSES	14	14	0%
Psychological MOSES	24	20*	17%

*Statistically significant improvement by the Conservative Dual Criterion.

Conclusion

This concludes Chapter 4 results section. The results are consistent with previously published research on neurofeedback in autism. Specifically, this provides evidence that the participant benefitted from neurofeedback LZT with improved neuropsychological symptoms, neurocognitive abilities, intelligence, and neurological processes as measured by QEEG and LORETA maps. Further, neurofeedback depicted a favorable side effect profile with no changes in neurological adverse effects and decreased psychological adverse effects (e.g., agitation, insomnia).

Chapter 5: Discussion

Introduction to Discussion

Over 25 years ago, autism was considered a rare condition with a prevalence rate of 1 in 2,000 children (CDC, 2007). However, in recent years, there has been an exponential growth in the prevalence rate with 1 in 110 children being diagnosed with some form of autism (CDC, 2009). The cost of services to treat ASC may be as high as $43,000 per year, yet historically ASC research and interventions have been significantly underfunded compared to other developmental disabilities (Ganz, 2006). In addition to the core symptoms, autism is often comorbid with other psychiatric disorders such as anxiety, depression, ADHD, and bipolar disorders (Bellini, 2006; Raja & Azzoni, 2008; Shtayermman, 2008; Volkmar & Klin, 2005). Such comorbidity is also implicated by ASC's research on pathophysiological processes of ASC in exorphins, serotonergic, dopaminergic, and hypothalamic-pituitary-adrenal systems (Anderson & Hoshino, 2005). Specifically, hyperserotonemia in ASC has been thoroughly researched in ASC (Schain & Freedman, 1961), and may explain the development of mood disorders in autism. The complexity of the disorder often warrants a multimodal intervention approach to maximize functioning especially in adulthood where issues such as employment, independent living, and self-sufficiency are critical. However, a vast majority of research in autism has been focused on treating children (Coben et al., 2010; Roy et al., 2009; Wolf & Paterson, 2010). This study provides vitally needed exploration into the area of ASC within adults, as well as to evaluate the effect of neurofeedback LZT in a comprehensive repeated measures approach.

Summary of Research Findings

The participant met the study's criteria and was reflective of the complex neuropsychological issues reported in the literature review. He has a diagnostic history of Asperger's disorder, bipolar disorder, expressive communication disorder (i.e., apraxic speech), impulse control disorder not otherwise specified, and anxiety disorder not otherwise specified, which are congruent with research on comorbidities in ASC (Bellini, 2006; Raja & Azzoni, 2008; Shtayermman, 2008; Volkmar & Klin, 2005). Further, he also has neurological issues associated with a Chiari malformation, left thalamic mass and partial complex seizures. He presented with impaired gross motor coordination and abnormal QEEG measures in posterior regions, which may be associated with the Chiari malformation, a neurological defect in the cerebellum and brainstem affecting balance and coordination (National Institute of Neurological Disorders and Stroke, 2011). The left thalamic mass and static lesion may also have played a role in the significantly low amplitude relative and absolute powers shown in the QEEG because the thalamocortical connection is a critical pacemaker for EEG activity on the cortex (Hughes & Roy, 1999; Thompson & Thompson, 2003; Townsend, 2007). Further, there are high rates of neurologic conditions like seizure disorders and EEG paroxysmal discharges in ASC (Coben & McKeon, 2009; Kagan-Kushnir et al., 2005), and this participant had a history of partial complex seizure disorder and EEG paroxysmal discharges. Overall, he presented with a complex neuropsychological profile typical of individuals with ASC in research.

Despite the complexity of this participant neurofeedback LZT resulted in overall improvement across multiple domains: this included improvement in both subjective and

objective measures. The participant reported significant improvement in Asperger's symptoms and other symptoms associated with ADHD, mood stability, depression, and anxiety. He also reported significant reduction in Asperger's symptoms from a moderate to a mild level of severity as assessed by the NPQ-LF. Coben and Padolsky (2007) indicated that there was a 40% improvement in ASC symptoms in their sample of children with ASC in comparison to the present study that found a 25% improvement in those symptoms. For this study, the participant's ASC symptoms stabilized and showed little change over the treatment phase, which may suggest a limited treatment effect and the need for additional neurofeedback sessions. Further, he reported significant improvement in the NPQ-LF psychological indices with an average reduction of symptoms in ADHD by 30%, Mood Stability by 33%, Anxiety by 35%, and Depression by 41%. The participant had been diagnosed with significant symptoms in these areas, and neurofeedback appeared to lead to a favorable response on his related psychological symptoms. Hammond (2005) explored neurofeedback research in treating depression and found that there is a signature frontal asymmetry alpha, which was similar to our participant's QEEG frontal asymmetry. It is interesting to note that the most significant reduction was reported in depressive symptoms, and this may suggest that the targeted sites in the frontal lobe were responsible for the significant improvement, because neurofeedback training in the frontal lobe has been found to reduce depression (Hammond, 2005).

Consistent with past research on neurofeedback (Berman et al., 2005; Thompson et al., 2010), the participant gained in nonverbal intelligence score from baseline to intervention. Further, executive functioning, cognitive flexibility, complex attention, and

reaction time significantly improved from baseline to intervention phase, which are consistent with his self-reported ADHD, anxiety, depression, and mood stability. This further supports both the overall intellectual improvement that is dependent on executive functioning. Other researchers have found improvement in neurocognitive measures like Stroop, ToL^{DX}, and TOVA tests after neurofeedback interventions (Berman et al., 2005; Knezevic et al., 2009, 2010; Pineda et al., 2008). There are theories that might suggest the reason for such improvement especially with the participant's training protocol, which included fronto-temporal sites. For instance, frontal lobe deficits particularly with executive functioning, weak central coherence, and TOM and empathy are critical issues for treatment and might offset abnormal development found in the frontal lobe (Minshew et al., 2005; Thompson et al., 2010a). Furthermore, areas such as the pars opercularis within Broca's area influences mirror neuron activity and social interconnectedness and other areas along the prefrontal cortex and temporal lobes (Coben, 2009b; Iacoboni & Dapretto, 2006; Vollm et al., 2006). In this study, processing speed was not significantly impacted by neurofeedback, which might be the result of the site locations used in the neurofeedback training. Specifically, the training did not consist of parietal and occipital lobe sites which are linked to with processing speed in research (Peers et al., 2005). However, the chart did show gradual improvement over the 20 sessions, which suggests further training may have resulted in significant improvement.

Similar to this participant, past research has found that ASC is associated with right hemispheric asymmetry and hypercoherence particularly in the frontal lobe, which suggests anxiety and social motivation issues in ASC (Coben, 2009a; Coben & Myers, 2008; Just et al., 2007; Pineda et al., 2008; Sutton et al., 2005). The participant also had

paroxysmal discharges, which were noted within the record and consistent with past research (Coben & McKeon, 2009; Kagan-Kushnir et al., 2005). He had excess 8 Hz and generally low voltage throughout his EEG record. For the resulting changes of pre and posttreatment maps, the findings were interpreted with caution because of low grade muscle tension, medication effects, and fatigue during the posttreatment EEG acquisition. To counteract these variables, the EEG recording was edited for muscle artifact, included only eyes closed condition to reduce noise, and Laplacian montage to cancel out medication effects. Overall, posttreatment QEEG and LORETA maps showed increased EEG power across bandwidths particularly in the frontal and temporal lobes. Specifically, Brodmann areas 21, 6, 17, and 18 had significantly increased beta power, which is important for higher information processing such as executive functioning. Increasing power was the primary need for neurofeedback training to enhance brain function.

Finally, as with past research on neurofeedback (Coben & Padolsky, 2007), no significant adverse effects were reported by the participant, and associated with general improvement in neuropsychological symptoms. With regard to potential side effects, I purposely utilized a multimodal assessment strategy to evaluate neuropsychological measures and neurophysiological measures to identify the most effective neurofeedback protocol, which is the recommended standard of practice (Hammond & Kirk, 2008). This hoped to offset potential risks of training associated with changing brain function such as seizures, fatigue, or agitation. The participant's side effect profile revealed reduced psychological side effects when measured by the MOSES, a commonly used side effect measure for individuals with developmental disabilities. His neurological side effect profile during baseline and treatment phase was not significant showing that there were

no adverse incidents such as an increase in extrapyramidal symptoms or seizures. This is reflective of past research finding a high benefit to risk ratio compared to other procedures like psychotropic medication and no abreactions particularly with neurofeedback approaches like bipolar montage and LZT (Coben & Padolsky, 2007; Collura et al., 2010). Neurofeedback might actually reduce psychological side effects (e.g., agitation, anxiety) more expediently, while neurological side effects might require a longer course of training.

Overall, these results suggest that neurofeedback might be helpful in mitigating and stabilizing symptoms associated with adulthood ASC and that longer-term training is indicated due to the developed neural networks. However, research in neuroplasticity has suggested that neurodevelopment continues throughout adulthood with methods that challenge neurocognition (Beauregard & Lévesque, 2006; Jones, 2004; Malkowicz & Martinez, 2009; Pinel, 2008). Neurofeedback research has evidence of neuroplasticity indicated by normalizing the neural pathways based on changes in EEG activity and neuroimaging (Lévesque, Beauregard, & Mensour, 2006; Malkowicz & Martinez, 2009). Although long-term effects of neurofeedback were not explored in this study, there has been research that supports maintained treatment effects in children with ASC up to a year, which is indicative of neuroplasticity and lasting changes in brain function (Coben, 2009a; Kouijzer et al., 2009a).

Limitations of the Study

The limitations of this study consist of the specificity of the population and research question, limited generalizability due to the sample size of one, practice effects, neurofeedback LZT training, methodology, and the principal investigator's biases. The

sample of one participant consisted of a European American male, which limits the generalization to this individual. As noted, ASC is a heterogeneous diagnosis, and the multiple comorbid diagnoses present in this individual demonstrate that heterogeneity. Sampling issues and randomized controlled research have been historically a problem for neurofeedback research (Rojas & Chan, 2005). The present study was purposefully a single-case research design, which did not allow for a larger sample or randomized assignment of participants. However, by electing to conduct a single subject design, the participant was able to act as his own control with a baseline and intervention phase. The visual inspection aspect of the methodology might be questionable when compared to more stringent approaches such statistical procedures and subjective reports by the participant (Kazdin, 1982). The participant, by being involved in the baseline measures, might be predisposed to report greater concerns initially, but in the intervention phase report decreased symptoms because of being involved in the study and expectations of change or simply as an effect of regression to the mean. However, the results of self-report measures were compared against results of ability measures that are not subjective in nature. The participant's exposure to repeated measures may also lead to practice effects over time and to improvement in those measures (Kazdin, 1982). An additional limitation is that the participant had multiple mental health conditions, which is common in ASC, but makes it difficult to isolate the effects of neurofeedback in relation to ASC. Lastly, the principal investigator is biased having researched neurofeedback and acquired a Biofeedback Certification in Neurofeedback with strong opinions about the effectiveness of neurofeedback treatment. This bias may have influenced subjective reports by the participant.

Generalization

This research adds to the literature of technologically advanced neuropsychological interventions for adulthood ASC. This is the first quantitative study that evaluated neurofeedback LZT in an adult with autism using a multiple baseline single-case research approach. Currently, the research in the field is lacking in investigations of the efficacy of neurofeedback in adulthood (Coben et al., 2010). For adulthood ASC, treatment efficacy research has historically been lacking as with psychosocial rehabilitation and psychotherapy (Roy et al., 2009; Shea & Masibov, 2005; Wolf & Paterson, 2010). Knezevic, Thompson, and Thompson (2009, 2010) found that age or intellectual functioning did not show any significant differences in the level of improvement by neurofeedback. This suggests that neurofeedback may have equal benefit in adulthood as it does in childhood in individuals with ASC.

Furthermore, single-case research in neurofeedback has mainly consisted of qualitative approaches with subjective or interpretive reports that do not provide quantitative changes in pre and posttest measures (Beaumont & Montgomery, 2005; Collura et al., 2010; Othmer, 2007; Rutter, 2009; Sichel, Fehmi, & Goldstein, 1995; Thompson & Thompson, 2003a, 2003b). There are few studies published with quantitative experimental formats and single-case investigations such as AB designs in adults with ASC (Blampied, Blampied, Barabasz, & Barabasz, 1996; Kazdin, 1982). The current study results demonstrate a further need to explore this treatment in a larger sample of adults with ASC. It provides clear evidence of neurofeedback LZT's effect on neuropsychological symptoms and neurocognitive performance in one adult with ASC. These results indicate the role neurofeedback may play in neuroplasticity later in life.

Implications for Social Change

The results of this study may be used to advance social change by helping adults with ASC to improve their overall quality of life through neurofeedback: an empirically supported intervention for treating autism. By providing evidence that neurofeedback is beneficial in multiple symptoms domains it may also help providers to implement or suggest neurofeedback as an adjunct treatment. These findings advance research in neurofeedback on neuropsychological functioning in ASC, and provide support for the use of interventions in adults with ASC. The findings indicate the possibility of neuroplasticity through neurofeedback LZT in improving neurocognitive abilities, reduction of neuropsychological symptoms, and improved neurophysiological functioning. Research on neurofeedback in individuals with ASC has largely focused on children and adolescents rather than adults (Roy et al., 2009; Wolf & Paterson, 2010). Autism is considered a developmental disorder with continuous delays in social-communication and problems related to obsessive interests or repetitive behaviors (APA, 2000). Later in life, adults with ASC have problems across various psychosocial domains, which lead to further psychological symptoms related to anxiety and mood disorders (Shea & Masibov, 2005). This study provides support for the use of neurofeedback in improving overall functioning including measures of psychological symptoms, neurocognitive and intellectual abilities, and neurophysiological processes into adulthood.

This research may also advance social change by encouraging more research in rural areas because the context of this study was in northwestern Michigan, a rural area approximately 4 hours from the closest urban county. According to the U.S. Department

of Agriculture, Economic Research Service (2000), Michigan is a predominantly rural state with around 66% of Michigan being rural along with 15% of households being impoverished. Rural areas are especially challenging for access to care and services for individuals with ASC. Problems with limited resources, a 30-40 mile drive to the closest provider, and a waiting list for specialists extending several months are common (Hutton & Carron, 2005). In addition, rural areas present many challenges for professionals and primary care providers in addressing and coordinating referrals to the multiple specialty services that are needed for individuals with ASC and their families (Symon, 2001). Rural poverty also has led to poorer health, less education, and other problems associated with decreased agriculture and profitability in rural communities (Judd et al., 2002). These are issues that are indicated in the backdrop of this study, which further advocates for research and better access to care in rural areas.

Implications for Future Research and Practice

The need for technologically advanced interventions in ASC is critical particularly in adulthood. Given that ASC is a lifelong condition; it will require more research to validate empirically interventions that work. Ongoing research in adulthood ASC will be important in guiding clinical practice toward improving and promoting overall wellbeing in adults with ASC. This is the first neurofeedback LZT study for an adult with ASC providing psychometric and neurophysiological findings supporting its effect. However, further research is needed in larger samples and additional single-case research design. This study could be replicated to determine if effects are consistent for other participants. Ninety percent of behavioral interventions for ASC have been single-case research designs and have been able to validate and invalidate different treatment

approaches for ASC (Matson, Matson, & Rivet, 2007; Smith, 2008). It is encouraged that outpatient clinic settings employing neurofeedback in their practice use single-case research designs (Blampied, Barabasz, & Barabasz, 1996; Kazdin, 1982).

An unintended finding and important issue for future neurofeedback research in adults with ASC is the need to have accurate pre and post-EEG acquisition due to the various neuropsychological complications with this population. For instance, it is likely due to high comorbidities with other disorders that most adults with ASC will have some form of psychopharmacological or neurological interventions such as a mood stabilizer, vagal nerve stimulator, or anticonvulsant medication. This will impact the findings of QEEG and LORETA imaging. Also, participants with ASC have repetitive behaviors and stereotypies that make it difficult to edit and prevent artifacts. This participant represented a typical adult with ASC who had difficulties in a wide range of areas, medications to address neuropsychological complexities, and artifacts that impacted the pre and post-EEG records. Consideration might be made ahead of time for multiple baseline and post-EEG recordings to average pre and posttreatment to improve consistency of findings.

Conclusion

This study found that an adult with ASC benefited from 20 sessions of neurofeedback LZT following a comprehensive evaluation of both neuropsychological functioning and neurophysiological processes. The single-case research design offered a unique ability to evaluate the trend of data points between a control phase and treatment phase on a number of neuropsychological, neurocognitivie, and psychological variables. This allowed for detailed results of how neurofeedback affects each area evaluated,

which included psychological symptoms, neurocognitive abilities, intelligence, and neurophysiological functioning. The consistency in these results allowed for validation by both objective and subjective measures that neurofeedback LZT was effective in this adult with ASC. These findings provide evidence that neurofeedback LZT may be beneficial in improving developmental deficits into adulthood. Future research is needed to validate findings in more single-case research studies as well as larger group studies.

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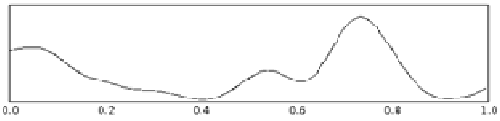
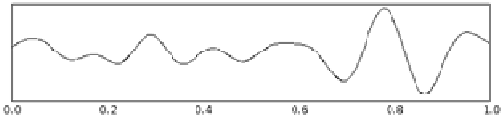
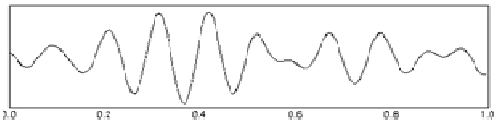
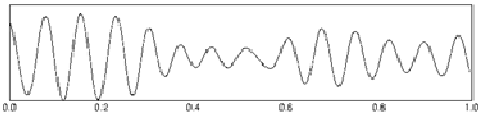
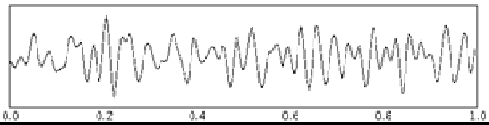
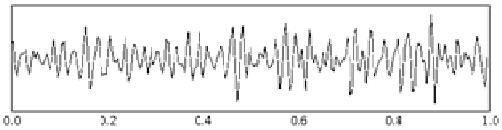
Appendix A

Wave	*Frequency*	*Brief Description & 1 Epoch Picture*
Delta	1-4 Hz	**Function:** Sleep, rest, drowsy states, or problem solving. **Morphology:** Rhythmic or arrhythmic. **Disorder:** TBI or stroke- focal, ADHD, ASC, or LD- widespread. 
Theta	4-8 Hz	**Function**: Spacey, working memory, deep states, insight, creativity. **Morphology**: Square top or sinusoidal rhythm rhythmic or arrhythmic. **Disorders**: ADHD- theta:beta ratio 3-6:1; depression, anxiety, ASC 
Alpha **Alpha 1** **Alpha 2**	8-12 Hz 8-10 Hz 10-12 Hz	**Function:** Alertness, readiness, meditation-relaxed, not processing. **Morphology:** Sinusoidal wave; mu rhythms. **Disorder:** Depression-high amplitude in anterior cortex; ADD, ASC. 
SMR	12-15 Hz	**Function:** Internally oriented, mental alertness, relaxation- C3, Cz, C4. **Morphology:** Similar to Beta 1. **Disorder:** Epilepsy, hyperactivity- low SMR; ASC- Mu rhythm. 
Beta **Beta 1** **Beta 2** **Beta 3**	12-32 Hz 12-15 Hz 15-18 Hz 18-25 Hz	**Function:** Processing, analytic, externally oriented, focus, attention. **Morphology:** Rhythmic activity. **Disorder:** OCD, sleep disorders, LD, anxiety, depression, ASC. 
Gamma	30-50 Hz	**Function:** Cognitive processing, learning, problem solving tasks. **Morphology:** Synchronous bursts. **Disorder:** Low in LD or cognitive impairment. 

Note: Information compiled from Demos, 2005; Thompson & Thompson, 2003; Townsend, 2007; permission for wavelength frequency pictorials by Wikipedia, 2005.

Appendix A

Thank you for considering this research project. A client with autism who is considering neurofeedback at [redacted] is invited to participate in this study, which will include 5 assessments over the course of 1-2 months. Mr. Lucido, principal investigator, is seeking an adult-18 years or older, who has been diagnosed with autism spectrum disorder, pervasive developmental disorder, or Asperger's syndrome. If you are pregnant, older than the age of 65, have intellectual disability, diagnosed with Rett's disorder, non-English speaking, taking more than three medications, or in a facility, then you will be unable to qualify for the study. This research study will explore the impact of your regularly scheduled neurofeedback sessions with [redacted]. The research study itself will involve only the assessment that will take place before, during, and after your neurofeedback at the clinic. The participant will need to be able to understand and independently consent to participation in the study, and complete a series of evaluations that will take at least 6-8 sessions with 1-2 hours of testing at each session.

To find out more about the study, please contact Michael Lucido, principal investigator [redacted] Mr. Lucido is conducting this research as a part of his doctoral program at Walden University. Dr. Lisa Scharff will be the Committee Chair, and overseeing the project. He is presently also an internship student with [redacted]. If you are related or presently working with Mr. Lucido, you are ineligible for the study. He will submit informed consent information, additional background information, and contact information for consideration with participating in the study. He will also be happy to review, read, or offer any information for consent.

Appendix C

RESEARCH STUDY SEEKING AN ADULT CLIENT WITH AUTISM SPECTRUM CONDITION TO PARTICIPATE IN AN INTERVIEW, TESTING, AND ASSESSMENTS WHILE THEY ARE RECEIVING NEUROFEEDBACK. This study will include an interview, testing, and assessments that will measure changes while you are receiving neurofeedback by [redacted]. If you are pregnant, older than the age of 65, have an intellectual disability, diagnosed with Rett's disorder, non-English speaking, taking more than three medications, or in a facility, then you will be unable to qualify for the study. One individual will be selected and will need to be able to read and provide consent to completing interview, testing, and assessments involved in the study. CONSENT FOR THIS STUDY ONLY INCLUDES CONSENT FOR THE INTERVIEW, TESTING, AND ASSESSMENTS CONDUCTED BY THE INVESTIGATOR FOR ANALYSES. It requires attendance for 6-8 sessions for 4-6 weeks. To find out more please contact:

Michael Lucido at [redacted] He is a conducting this research as a part of his doctoral program at Walden University. If you are related or presently working with Mr. Lucido, you are not eligible for this study. Please call for more information.

Appendix D

Informed Consent

Introduction to the Study

Prospective clients of [redacted] who have been diagnosed with autism are invited to participate in this study. This study will consist of an ongoing evaluation, which includes an interview, testing, and assessments. The consent is only for these procedures. Consent for neurofeedback is completely separate and through [redacted] alone. You are invited to voluntarily participate because you have a diagnosis of autism and meet the requirements of the study (English speaking, average intelligence, between 18-64 years old, and currently taking less than three medications).

Research Approach

The research will consist of interviews, assessments, and testing that take place during the process of receiving neurofeedback. Participants would be asked to complete 6-8 sessions that include 5 assessment procedures for a total of 4-6 weeks while they are doing regularly spaced neurofeedback sessions. The interview, assessments and testing are components of the research study, the neurofeedback is not. Self-reports will consist of questions related to symptoms of autism, depression, anxiety, mood, attention, and impulsivity. The tests will measure a part of intelligence, mental flexibility, and speed.

Why is this research being done?

The present study is important for finding out if there are any changes in the assessments and testing during neurofeedback in an adult with autism. It will also complete a part of Michael Lucido's education at Walden University.

How many people will take part in this research study?

There will be one participant for this study.

How long will you take part in this research study?

There will be 1-2 hours for each meeting and at least 6 meetings over 4-6 weeks.

What do we do if we can be in this study?

Contact the Michael Lucido to schedule a meeting.

What will happen to the results of the study?

Your results (any changes in the testing material from the first testing to the second testing) will be given to you in the final meeting in a one page summary. All the information will be kept within a password protected computer. No information such as names or addresses will be kept after all of the information is collected.

Is there a payment with being involved?

There is no payment for participating in this research. However, for being involved, traveling costs will be provided at the current Federal government mileage rate.

Volunteering:

Being involved in the study is completely voluntary, which means everyone will respect your decision of whether or not you want to be in the study. No one will treat you differently if you decide not to be in the study. If you decide to join the study now, you can still change your mind during the study and stop participating. If you feel stressed during the study you may stop at any time. You may skip any questions that you feel are too personal.

Researcher Disclosure:

Michael Lucido is not getting money or in a business related to this study.

Privacy and Confidentiality Procedures:

When results of the study are reported in meetings and journals, no one will be given any information that could identify the individual enrolled in the study such as names. Michael Lucido will not release any information about your research involvement without your written consent.

Benefits Being in the Study:

The primary benefit is to help research in autism. No one knows if neurofeedback is related to changes in brain functioning in adults with autism. Articles are available upon request about neurofeedback.

Possible Side Effects:

The main side effect for the study is fatigue during the tests and personal questions. Also, this study includes a detailed evaluation and approach that has been used in many neurofeedback studies. Regardless, participants will be closely monitored for potential side effects during the assessments and testing meetings.

- I have read and can understand the above information.
- I understand that this study only includes testing and assessment procedures while I am receiving my neurofeedback through [redacted].
- I give my consent voluntarily and I was not forced to enter this study.
- I am willing to participate in this research study.
- I understand that my name and other information will not be released and that I will be assigned a number to protect my confidentiality.
- I am willing to sign a release to allow Michael Lucido to contact my current or past providers (school or clinic) who diagnosed me with autism.

- I was not told to go into this study by a referral from a clinic for treatment.
- I am not a family member of Michael Lucido.
- I am not in therapy with Michael Lucido.
- I understand if I drop out of the study I will not be prevented from ongoing treatment at [redacted] for services.
- I understand that I may stop the research study at any time without any penalty or problems for future services.
- I understand that testing and assessments are not intended to diagnose disorders.
- I am consenting to participate in interviews, testing, and assessments.
- I understand that there may be no effect at all from participating in the research.
- I understand that there may be discomfort in completing the interviews, testing, and assessments including the time involved.
- I understand that involvement in this research will include 5 assessment procedures over the course of 4-6 weeks for a total of 1-2 hours per session.
- If I am in crisis, I will be offered an immediate referral for services at the local emergency room or community mental health. Also, I will be provided the crisis phone number: [redacted].
- I understand that my testing and assessment data may be reviewed with a mentor, therapist, and/or doctor on a consulting basis.
- I understand that my research records are private to the fullest extent of the law, except in cases of state and federal laws that mandate mental health providers to report risks such as harm to self or others, or civil/criminal proceedings.

- If I have concerns about how my participation might impact my wellbeing, I will consult my doctor prior to my participation in the research study.
- I will disclose a list and changes of my medications or vitamins/supplements throughout the course of this study and talk with my doctor regarding any concerns.
- I understand that I will be able to continue neurofeedback treatment through [redacted] following the study and that it will not impact my treatment.
- I accept that I have been offered time to ask questions regarding all the information above and that these questions have been answered to my liking.

Canceling Appointments and Stopping the Study:

Please call 24 hours to reschedule any appointment.

You may stop the research study at any time for any reason. As a courtesy, please feel free to call or write a note about what led you to stop.

Contact information:

You may ask any questions you have now. Or if you have questions later, you may contact the researcher via researcher's phone at ([redacted] or email address at [redacted]. If you want to talk privately about your rights as a participant, you can call Dr. Leilani Endicott. She is the Walden University representative who can discuss this with you. Her phone number is 1-800-925-3368, extension 1210. Walden University's approval number for this study is **09-08-11-0072997** and it expires on **August 8, 2012.**

Consent to Participate in Study:

Signature participant code	DATE
Advocate witness (e.g., family member, therapist, doctor)	DATE

Participant should keep a copy of the consent form.

Comprehension Check for Informed consent

Do you understand that the purpose of the research sessions is not to help you, but rather to learn if there are any changes testing and assessments?

What will you are doing in this study?

Tell me what you are agreeing to do for the study?

Can you stop at any time?

How long will this study be and the length of each session?

Do you know how the research report will be able to protect your identity?

Do you understand that we might learn that there will be no changes during the study?

Am I consenting to neurofeedback as a part of the study?

Appendix E

Confidentiality Agreement

Name of Signer:

During the course of my activity in collecting data for this research: "The Effect of Neurofeedback Live Z Score Training on Neuropsychological Functioning in Adults with Autism Spectrum Disorder: A Single-Case Research Design" I will have access to information, which is confidential and should not be disclosed. I acknowledge that the information must remain confidential, and that improper disclosure of confidential information can be damaging to the participant.

By signing this Confidentiality Agreement I acknowledge and agree that:

1. I will not disclose or discuss any confidential information with others, including friends or family.
2. I will not in any way divulge, copy, release, sell, loan, alter or destroy any confidential information except as properly authorized.
3. I will not discuss confidential information where others can overhear the conversation. I understand that it is not acceptable to discuss confidential information even if the participant's name is not used.
4. I will not make any unauthorized transmissions, inquiries, modification or purging of confidential information.
5. I agree that my obligations under this agreement will continue after termination of the job that I will perform.
6. I understand that violation of this agreement will have legal implications.

7. I will only access or use systems or devices I'm officially authorized to access and I will not demonstrate the operation or function of systems or devices to unauthorized individuals.

Signing this document, I acknowledge that I have read the agreement and I agree to comply with all the terms and conditions stated above.

Signature: **Date:**

Appendix F

Research Volunteer Registration and Screening Form

Last Name_____First Name_____Middle Initial_____

Address:_____

City: _____State:_____ZipCode:_____

Phone:_____Work Phone:_____Cell Phone:_____

Birth Date:_____Gender (M/F):_____Marital Status:_____Pregnant:____

Race/Ethnicity:

___American Indian/Alaskan Native ___Asian ___Black/African American

___Native Hawaiian/Pacific Islander ___White ___Other

Current Living Arrangement (Check all that apply)

___Alone ___Mother ___Father ___Sibling(s) ___Relatives/Kin

___Guardian ___Spouse ___Partner/Significant Other ___Child(ren)

___Foster Children ___Unrelated persons

Years of Education_____ Occupation_____

Previous Mental Health Services (Y-Yes N-No)

___Inpatient Care ___Partial Care ___Other 24-Hour Care ___Outpatient

May we contact you (Y-Yes N-No)

___Call at Home? ___Call at Work?___Message at Home?___Message at work?___Mail Information?

Emergency Contact: _____

Home phone:_____Work phone:_____Cell phone:_____

Have you or a family member been diagnosed with a developmental disability, autism spectrum disorder, speech or language delay, obsessive-compulsive disorder, or learning disability?_____

Are you diagnosed with Rett's Disorder: YES NO

If you were diagnosed, please indicate what age you were first diagnosed with autism or a developmental disability: _____

Are you currently taking any medications? If yes, please specify: _____

_____

_____

Do you have a guardian? YES NO

Can you read a newspaper or magazine? YES NO

Did you graduate with a high school diploma or have a GED? YES NO

Appendix G

Authorization to Disclose PHI for Research Purposes

A copy of the form will be given to the research participant for his/her personal records.

Research Participant Name: _____

Phone:_____Address:_____

Discloser of Information: _____

Recipient of Information: Michael Lucido, Principal Investigator

Means of disclosing information (i.e., verbal, written, etc.): Verbal or written.

Information to be disclosed: Confirmation that this individual has an autism spectrum condition-pervasive developmental disorder, Asperger's syndrome, or autistic disorder.

Reason for the Release: Released/obtained for the purpose of research.

Authorization Provided by Research Participant:

I understand that this authorization permits the release of information between the two parties named above.

I understand that I have the right to refuse to sign this release form.

I understand that upon release, this information will be kept confidential; my identity will be concealed and data will not be disclosed outside of the specified individuals/agencies.

I understand a photocopy of this release will be as effective as the original.

I understand this authorization will be in effect for 1 month from the date signed unless cancelled by me in writing.

_____

Signature DATE

_____

Advocate Signature DATE

Appendix H

Neuropsych Questionnaire-Long Form Subscales

Asperger/Autism Questions	**0**	**1**	**2**	**3**
1 Avoiding eye contact				
2 Difficulty developing friendships				
3 Difficulty understanding sarcasm, metaphors or jokes				
4 Hard to relate to other people				
5 I can't relate to other people, socially or emotionally				
6 I don't attend to social signals				
7 I don't respond to other people's expressions or body language				
8 Not able to begin or to sustain a conversation with other people				
9 Not responsive to other people's feelings				
10 Odd preoccupations or interests				
11 Preoccupied by a particular interest to the exclusion of other things				
12 Rigid, inflexible, resistant to change				
13 Strongly attached to routines or sameness in the environment				
14 I can't feel close to another person				
15 Withdrawn, isolated				
Depression Questions	**0**	**1**	**2**	**3**
1 Crying spells				
2 Feeling depressed				
3 Feeling discouraged about the future				
4 Feeling empty inside				
5 Feeling hopeless				
6 Feeling irritable				
7 Feeling little or no interest in things				
8 Feeling lonely				
9 Feeling sad				
10 Feeling that doing anything is a real effort				
11 Feelings of guilt or remorse				
12 Having nightmares or bad dreams				
13 I feel like a failure				
14 I feel like I'm being punished				
15 Loss of interest in sex				
16 Not enjoying things as much as before				
17 Withdrawn, isolated				

Anxiety Questions	**0**	**1**	**2**	**3**
1 Feeling anxious				
2 Feeling keyed up or on edge				
3 Feeling nervous				
4 Feeling restless				
5 Feeling tense				
6 Fidgety, I can't sit still				
7 Having nightmares or bad dreams				
8 High-strung or keyed up				
9 I find it hard to relax				
10 Worrying too much				
Mood Stability Questions	**0**	**1**	**2**	**3**
1 Anger				
2 Angry outbursts				
3 Crying spells				
4 Easily agitated				
5 Easily annoyed				
6 Easily frustrated				
7 Elevated mood, euphoria				
8 Excitable				
9 Explosive				
10 Feeling irritable				
11 Feeling negative				
12 My moods change quickly				
13 Temper tantrums				
ADHD Questions	**0**	**1**	**2**	**3**
1 Difficulty concentrating				
2 Difficulty paying attention				
3 Easily distracted				
4 Feeling restless				
5 Feeling scattered, disorganized				
6 Fidgety, I can't sit still				
7 Forgetful, I need constant reminding				
8 Impatient				
9 Impulsive, act without thinking				
10 Leaving things behind and having to go back to get them.				
11 Losing things				
12 Making careless mistakes				
13 Not finishing chores, homework or projects				
14 Overly active				
15 Short attention span				

Appendix I

MOSES Checklist

Neurological Signs/Symptoms	**0**	**1**	**2**	**3**	**4**
1. Arm swing: Decreased					
2. Contortions/neck -arching back					
3. Gait: Imbalance/unsteady					
4. Gait: Shuffling					
5. Limb jerking/writhing					
6. Movement: Slowed					
7. Restlessness/pacing/can't sit still					
8. Rigidity/muscle pain or aches					
9. Tremor/shakiness					
10. jitteriness/jumpiness/nervousness					
11. fainting/dizziness/Upon standing					
12. seizures: increased					
13. tingling/numbness					
14. weakness/fatigue					
Psychological Signs/Symptoms	**0**	**1**	**2**	**3**	**4**
1. Agitation					
2. Confusion					
3. Crying/feelings of sadness					
4. Drowsiness/Lethargy/Sedation					
5. Irritability					
6. Withdrawn					
7. attention/concentration difficulty					
8. morning "hangover"					
9. nightmares/vivid dreams					
10. perceptual: hallucinations/delusions					
11. sleep: excessive					
12. sleep: insomnia					

Appendix J

Letters of Permission

10-20 system for EEG

I, the copyright holder of this work, release this work into the public domain. This applies worldwide. In some countries this may not be legally possible; if so:

I grant anyone the right to use this work for any purpose, without any conditions, unless such conditions are required by law.

Wikipedia

Monitoring of Side Effects Scale

The MOSES (Monitoring of Side Effects Scale) is in the public domain so no official permission is required for its use.

Chris Coleman, Ph.D.

Department of Social and Health Services

Clinical Director

Division of Developmental Disabilities

Neuropsych Questionnaire/CNSVS Neurocognitive Tests

We are happy to help support academic research.

To export the data, open up the application and click on Menu>Export and then select the files to be exported. The data exports to a tab delimited file. To get in excel, simply cut and paste from the notepad file.

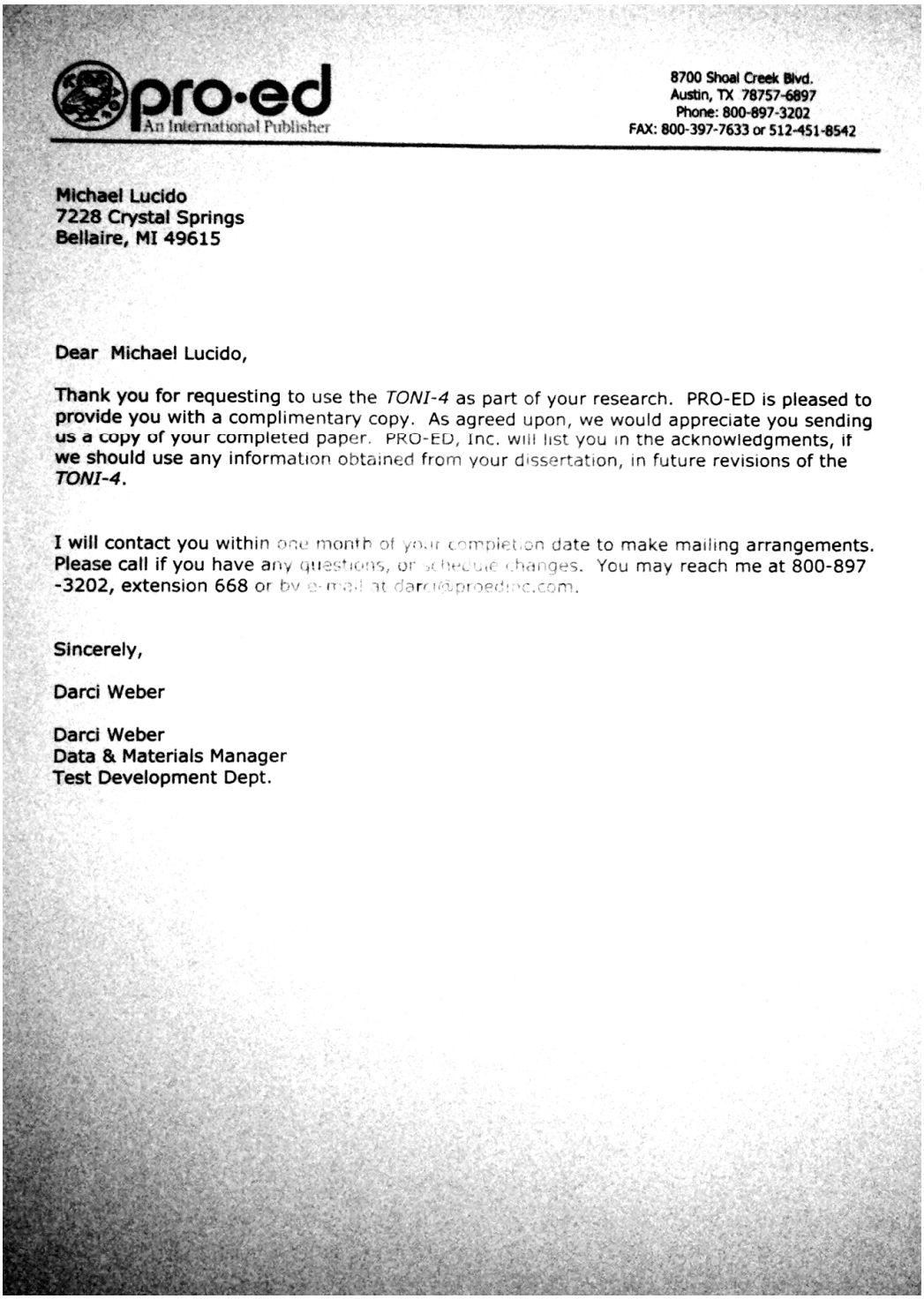
Kind regards,

Meghan Nolan

CNS Vital Signs

EEG Frequency Pictorials

Wikipedia



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Michael Lucido
7228 Crystal Springs
Bellaire, MI 49615

Dear Michael Lucido,

Thank you for requesting to use the *TONI-4* as part of your research. PRO-ED is pleased to provide you with a complimentary copy. As agreed upon, we would appreciate you sending us a copy of your completed paper. PRO-ED, Inc. will list you in the acknowledgments, if we should use any information obtained from your dissertation, in future revisions of the *TONI-4*.

I will contact you within one month of your completion date to make mailing arrangements. Please call if you have any questions, or schedule changes. You may reach me at 800-897-3202, extension 668 or by e-mail at darci@proedinc.com.

Sincerely,

Darci Weber

Darci Weber
Data & Materials Manager
Test Development Dept.

Curriculum Vitae

Michael J. Lucido

CAREER OBJECTIVE:

Acquire a doctoral position within a comprehensive mental health care center.

ACADEMIC PREPARATION:

2007-2012	Walden University, PhD in Clinical Psychology, GPA 3.9
2008-2010	Behavioral Medicine Research and Training Foundation Board Certified in Neurofeedback (BCN)
2001-2003	University of Detroit Mercy, MA in Clinical Psychology, GPA 3.9 Summa Cum Laude graduate Limited Licensed Psychologist, ID# 6301012513
1997-2001	University of Detroit Mercy, BA in General Psychology, Major GPA 3.9 Summa Cum Laude graduate Leadership Medallion Award

CAREER EXPERIENCE:

(6/10-6/11) Internship Student
40 hours per week
Community Consultation and Treatment,
North Country Community Mental Health
Duties include intake assessments, treatment planning, individual and group therapy, consultation with psychiatrists and medical director, and providing crisis intervention and assessments. Intern supervisor at one of the CMH clinics. Facilitated an 8 session intervention for parents with children who have Autism/Asperger's syndrome. Group therapy for 6 adults with dual diagnoses: bipolar and substance abuse disorders.

(1/09-6/10) Outpatient Therapist
40 hours per week
Community Consultation and Treatment,
North Country Community Mental Health
Duties include intake assessments, treatment planning, individual and group therapy, consultation with psychiatrists and medical director, and providing crisis intervention and assessments. Co-facilitated two 6 week interventions for a group of individuals with Autism/Asperger's syndrome teaching psychosocial skills and conflict resolution.

(10/05-1/09) Supports Coordinator/Psychologist
40 hours per week
Julie Moran, MSW, QMRP, Supervisor
Developmental Disability Program,

North Country Community Mental Health
Duties include psychological evaluations for guardianship and treatment recommendations, individual therapy, behavioral approaches, consultation with psychiatrists and medical director, providing crisis intervention, coordinating and linking community and NCCMH services, establishing and monitoring treatment plans, advocating for the optimal level of multimodal services, and intake/annual assessments. Linked with multiple IEP meetings and coordinated with TBAISD Autism Specialist, School Psychologists, and School Social Workers. Co-facilitated three 6 week interventions for a group of individuals with Autism/Asperger's syndrome teaching psychosocial skills and conflict resolution.

(4/04-10/05) Contractual Psychologist
32 hours per week
Anne Kennedy, PhD, Supervisor
Psychological Services Program,
Detroit East, Inc. Community Mental Health Center
Duties include facilitating a psychosocial skills/solution-focused group, individual therapy for adolescents and adults, conducting comprehensive psychological evaluations, and presenting continuing education (CE) workshops. Left position to relocate in northern Michigan.

(6/03-10/03) Practicum Student
Steven Genden, PhD, Supervisor
Adult Outpatient Program,
Downriver Guidance Center
Duties included administering intake assessments, conducting psychological evaluations, maintaining a small caseload, and managing case files. Completed 400 hours and finished practicum requirements.

(2/03-5/03) Practicum Student
F. Edward Rice, PhD, Supervisor
Children and Family Services,
Northeast Guidance Center
Duties included administering intake assessments, conducting psychological evaluations, and assisting in home based services. Completed 200 hours and continued practicum at Downriver Guidance Center.

(1/03-4/03) Student Therapist
Susan Birndorf, PhD, Supervisor
Outpatient Therapy Course,
University of Detroit Mercy Psychology Clinic
Duties included conducting short-term cognitive-behavioral therapy, providing session summaries, and participating in weekly case conferences. Completed semester course work.

(10/01-5/03) Art Therapist Volunteer
20 hours per week
Sr. Nancyann Turner, Director
Art Therapy Services,
Capuchin Soup Kitchen
Duties included utilizing art therapy techniques to help disadvantaged youth cope with stress through creating art. Supervised by a trained art therapist.

PSYCHOLOGICAL ASSESSMENT/TESTING EXPERIENCE:
(9/01-present) Completed many comprehensive psychological evaluations utilizing:
Intelligence Tests:
- Wechsler Nonverbal Scale of Abilities
- Wechsler Adult Intelligence Scale 3rd Edition
- Wechsler Abbreviated Scale of Intelligence
- Wechsler Intelligence Scale for Children 4th Edition
- Test of Nonverbal Intelligence 3rd Edition

Neuropsychological Tests:
- CNSVS Neurocognitive Test
- Comprehensive Trail Making Test
- Visual Motor Integration Test
- Bender Visual-Motor Gestalt Test
- Benton Visual Retention Test, Revised
- Mini-Mental Status Examination
- Verbal Fluency Test and Sentence Repetition/Memory
- Repeatable Battery for Assessment of Neuropsychological Status
- Quick Neurological Screening Test, 2nd Edition

Achievement Tests:
- Wechsler Individual Achievement Test, 2nd Edition
- Wide Range Achievement Test, 3rd Edition
- Test of Language Development, 2nd Edition
- Peabody Picture Vocabulary Test, Revised
- Slosson Oral Reading Test, 2nd Edition

Checklists and Self-Reports:
- Minnesota Multiphasic Personality Inventory 2nd Edition and Restructured Format
- Achenbach Child Behavior Checklist and Teacher Report Form
- Beck Youth Inventories, Self Report
- Child Symptom Inventories, Teacher and Parent Checklists
- Kovacs' Children's Depression Inventory
- Conners Teacher and Parent Reports/Conners Adult ADHD Self and Observer Reports
- Child and Adolescent Functional Assessment Scale
- Adaptive Behavior Assessment System and Vineland Adaptive Behavioral Scales
- Neuropsych Questionnaire Long and Short Form
- Adult Asperger Assessment, Asperger Syndrome Diagnostic Scale, Asperger Diagnostic Interview

WORKSHOPS PRESENTED:

(3/12) Suicide Prevention Workshop with local community organizations
(6/11) Autism Spectrum Disorder: Putting the Pieces Together
(1/11) Child and Adolescent Functional Assessment Scales for the DD program
(11/10) Suicide Prevention Network Presentation to Charlevoix-Emmet ISD
(10/10) Michigan Association of Community Mental Health: Destigmatizing Autism
(9/10) CMH Board Presentation: Evidenced-Based Practices for Autism
(8/09) NCCMH Board Presentation: Social Skills Groups for Autism
(8/08) Diagnostic and treatment interventions for autism at DD Conference
(6/08) "Normal People Scare Me"-discussion on autism at Charlevoix Library
(8/07) "Normal People Scare Me"-discussion on autism at DD conference
(6/07) "Normal People Scare Me"-discussion on autism at Alden Library
(3/05) Overview of a Comprehensive Psychological Evaluation: Case Study
(2/05) Behavior and Self-Report Checklists at Detroit East, Inc.
(2/05) Achievement Tests and Learning Disabilities, at Detroit East, Inc.
(2/05) Neuropsychological Tests and Brain Functioning, at Detroit East, Inc.
(2/05) Overview of Wechsler Intelligence Scale for Children, Fourth Edition
(9/00-9/02) 20 Service-Learning presentations each semester on Servant Leadership

NEWS ARTICLES:

(11/1/02) Detroit News, Metro Section C, by Margarita Bauza, "UDM's 125th honors core values"
(10/25/02) Michigan Catholic, Local News, by Audrey Sommers, "U of D Mercy celebrates 125 years"
(1/24/01) Varsity News, by Michael Lucido, "Racism on UDM campus: Breaking the Boundaries"