EFFECTS OF CHILDHOOD SEXUAL ABUSE ON BRAIN FUNCTION AS MEASURED BY QUANTITATIVE EEG, NEUROPSYCHOLOGICAL, AND PSYCHOLOGICAL TESTS

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Childhood sexual abuse (CSA) has been the subject of much recent controversy as a result of Rind, Tromovitch and Bauserman's (1998) meta-analytic examination of CSA, which found a weak relationship between CSA and self-reported psychopathology in college samples. There have been few studies of CSA which look beyond self-report. The present study is an exploration of the relationships between CSA, quantitative electroencephalographic (QEEG), neuropsychological, and psychological measurements in 24 high-functioning, unmedicated CSA adults who were matched for age, gender, and handedness with a group of adults without CSA (NCSA). The objectives of this study were to: 1) examine EEG abnormalities associated with CSA, 2) investigate QEEG cortical coherence in the groups using neuroelectric eigenimage (NEI) connectivity indices (Hudspeth, 1999), 3) integrate personality differences associated with CSA with EEG differences, and 4) better understand left versus right hemisphere functioning in CSA using intelligence testing. An examination of QEEG cortical coherence revealed moderate to large effect sizes indicating patterns of decreased connectivity between brain regions on the right frontally in the delta band, and frontally and centro-temporally on the right in the alpha band, and posteriorly in the alpha and beta bands, as well as in the cross-correlation; increased connectivity between brain regions was evidenced centrally across the motor strip and on the left temporally in the delta band, which differentiated the groups. Large effect sizes obtained

on measures of personality were related to poorer adjustment for CSA adults in comparison to NCSA adults. In contrast to prior findings with clinical groups (Black, Hudspeth, Townsend, & Bodenhamer-Davis, 2002; Ito et al., 1993), hypotheses related to QEEG cortical coherence (left hemisphere alpha hypercoherence and right hemisphere theta hypocoherence), EEG abnormalities, and IQ (Verbal less than Performance) were not supported. Walker's (2003) theoretical modular coherence model was utilized to integrate coherence and personality variables and provide treatment options.

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TABLE OF CONTENTS

Page

ACKNOWLEDGMENTS ii

LIST OF TABLES v

LIST OF ILLUSTRATIONS vi

Chapter

1. BACKGROUND AND REVIEW OF THE LITERATURE 1

    CSA and Psychological Functioning 2
    Trauma and Neuropsychological Functioning in Animals 5
    CSA and Neuropsychological Functioning - Brain Structure 6
    CSA and Neuropsychological Functioning - Brain Function 7
    Research with Humans Experiencing PTSD 14
    A Possible Mechanism for the Psychological and Neuropsychological Correlates of CSA 16

2. PURPOSES OF THE STUDY 19

    Hypotheses 20
    Exploratory Analyses 22

3. METHOD 23

    Participants 23
    Measures 25
    Procedures 27

4. DATA ANALYSIS 30

5. STATISTICAL ANALYSIS 35

6. RESULTS 39

    Preliminary Analyses 39
    Primary Analyses 40
    Exploratory Analyses 44
    Post Hoc Analyses 50

7. DISCUSSION 52

Comparison of Findings to Earlier Research 52
Theoretical and Clinical Implications 56
Statistical Limitations 60

APPENDICES

Appendix A. Statistical Tables from NEI Analyses
Appendix B. Statistical Code for NEI Analyses

REFERENCES

LIST OF TABLES

Table Page

1. Sociodemographic Characteristics for CSA and NCSA Groups 24
2. Sociodemographic Characteristics for CSA and NCSA Groups 25
3. Chi Square Analysis of Descriptive Variables 39, 40
4. Chi Square Analysis for Qualitative Ratings of Raw EEG Data 41
5. Chi Square Analyses of MMPI-2 Pattern Variables 43
6. Student's T Analyses of MMPI-2 Scales 50

A1. Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep.. NEI's for Sites F1, F2, F7, and F8 64

A2. Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep.. NEI's for Sites F3, F4, T3, and T4 65

A3. Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep.. NEI's for Sites C3, C4, T5, and T6 66

A4. Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep.. NEI's for Sites P3, P4, O1, and O2 67

A5. Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep.. NEI's for Sites FZ, CZ, and PZ 68

LIST OF ILLUSTRATIONS

Figure Page

1. Neuroelectric Images based on means for the CSA and NCSA groups 33
2. Left and right hemisphere effect sizes based on mean distance from the origin for the alpha and theta bands 42
3. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together for sites F1, F2, F7, and F8..... 45
4. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together for sites F3, F4, T3, and T4..... 45
5. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together for sites C3, C4, T5, and T6..... 46
6. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together for sites P3, P4, O1, and O2 47
7. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together for sites FZ, CZ, and PZ..... 47
8. Classical and robust effect size topographic maps; Dependent measure – distance from the origin for NeuroRep (Hudspeth, 1999) neuroelectric images; Based on means and standard deviation..... 48
9. Mean MMPI-2 T scores for the CSA group and the NCSA group 49

CHAPTER 1

BACKGROUND AND REVIEW OF THE LITERATURE

Childhood sexual abuse (CSA) has become epidemic in the United States. U.S. government statistics for 1994 report the substantiated sexual abuse incidence rate to be 2 in 1,000 (U.S. Department of Health and Human Services, 1996). General population surveys, which include cases not reported to authorities, suggest annual rates as high as 11 in 1,000 (Finkelhor and Dziuba-Leatherman, 1994). Estimates from grouping studies of prevalence indicate 25-30% of females and 8-10% of males have experiences of childhood sexual abuse that meet legal definitions of sexual abuse (Bagley, 1990; Finkelhor, 1986; Finkelhor, Hotaling, Lewis, & Smith, 1990). It has been associated with the development of a host of maladies including depression, anxiety, substance abuse, sexual dysfunction, suicidal ideation and attempts, as well as other self-destructive behaviors (Beitchman, Zucker, Hood, DaCosta, Akman, & Cassavia, 1992; Briere & Elliott, 1997; Browne & Finkelhor, 1986). CSA has been found in groups experiencing somatoform, dissociative, affective, personality, posttraumatic (PTSD), eating, and anxiety disorders as well as chemical dependency, psychogenic amnesia, and headaches (Bowman & Markand, 1996; Coons, 1992; Rastam, 1994; Bowman, 1993; Clay, Olsheski, & Clay, 2000; Coons & Milstein, 1992; Domino & Haber, 1987). More specifically, research indicates six categories of impairment are associated with CSA: 1) emotional reactions which are aversive, 2) perceptions of the self which are distorted and negative, 3) physical complaints, 4) somatic complaints, 4) difficulties with development of positive sexuality, 5) interpersonal relationship difficulties, and 6) difficulties in social functioning.

In addition to the negative psychological associations with CSA, researchers have begun to look at the neuropsychological associations, examining its impact on brain structures and functions (Bremner et al., 1997; Teicher, Glod, Surrey, and Swett, 1993; Ito et al., 1993; Schiffer, Teicher, and Papanicolaou, 1995). The present study is an exploration of the relationships between CSA and neuropsychological and psychological functioning.

CSA and Psychological Functioning

Early identification of individuals who have experienced CSA is of clinical concern since early detection has been shown to be related to positive therapeutic outcomes (Courtois, 1988; Herman, 1992) and documentation of negative sequelae are extensive, as cited above. The Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989), has been a part of early detection research in an effort to discover common psychological profiles of adults with a history of CSA. Follette, Naugle, and Follette (1997) looked at females presenting for group counseling to deal with issues related to CSA and found a five-cluster solution for their MMPI-2 profiles. The first cluster solution included elevations on scales F, 2, 6, and 7. The second cluster solution included a higher elevation on F than with the first cluster solution as well as elevations on scales 2, 4, 5, 6, 7, 8, and 0. The third cluster solution only included a scale 4 spike. The fourth cluster solution included elevations on scales F, 1, 2, 3, 4, 6, 7, 8, and 0. And, the fifth cluster solution did not include significant elevations.

Knisely, Barker, Ingersoll, and Dawson (2000) compared pregnant and recently postpartum outpatients with a DSM-IV diagnosis of substance abuse according to whether or not they reported experiencing sexual abuse. Significantly higher T scores for scales F, 1, 2, 3, 4, 6, 7, and 8 were found for the group reporting sexual abuse. Additionally, Keane's post traumatic stress disorder scale (PK) was found to correctly classify 75% of those in the group reporting sexual abuse and 46% of those who did not report sexual abuse.

Griffith, Myers, Cusick, and Tankersley (1997) compared 115 women with and without CSA and according to different sexual orientations (heterosexual versus lesbian). Women with histories of CSA showed significantly higher elevations on scales 1, 2, 4, 6, 7, 8 and 9 than did those without histories of CSA. Main effects were found for CSA and the 4-5-6 configuration ("Scarlet O'Hara V") as well as for the 8-4 codetype. These findings were consistent for both heterosexual and lesbian females.

A pilot study from the University of North Texas looked at QEEG and MMPI-2 patterns of 12 adult outpatients reporting CSA and compared them with a matched sample of outpatients who reported they had not experienced CSA (Townsend, Black & Bodenhamer-Davis, 2001). The researchers found the CSA group had significantly higher MMPI-2 T score elevations on scales F, 1, 2, 3, 4, 7, 8, and PS (symptoms of posttraumatic stress).

MMPI-2 profiles of female outpatients reporting CSA were compared according to the coercion strategies used by their perpetrators by Lucenko, Gold, Elhai, Russo, and Swingle (2000). They found that the use of promised or actual rewards was associated with increased elevations on scales 6, 7, 8 and PK. No significant main

effects were found for the use of threatened or actual force in CSA, and no interaction was found between promised or actual rewards and threatened or actual force. Interestingly, promised or actual rewards by an authority figure were found to be more important than whether or not the perpetrator was a father figure, as no main or interaction effects were found for father figure relationship.

Comparing outpatients diagnosed with PTSD, Elhai, Frueh, Gold, Gold, and Hamner (2000) compared CSA trauma with Vietnam War trauma. The CSA group was found to score significantly higher on scales 4, 7, 8, and 9, but lower on scale 0. However, when age was controlled for, most group differences disappeared. Validity scale profiles for both groups showed the inverted V pattern with scale F mean 35 points higher than the means for scales L and K. Basic scales evidenced floating profiles and the 4-5-6 configuration. This study lends support to the idea that the sequelae of CSA may be due in part to posttraumatic stress similar to what is experienced by war veterans.

Overall, findings indicate adults with a history of CSA show significantly increased elevations when compared with NCSA adults. Three basic psychological profiles for CSA have emerged with the MMPI-2: 1) high F scale in comparison to L and K, 2) floating profiles (six or more basic scale elevations), and 3) a 4-5-6 configuration ("Scarlet O'Hara V"), in which scales 4 and 6 are elevated above a T score of 65 and scale 5 is at or below a T score of 35. The consistency of these findings is interesting in light of Bremner's (2002) assertion after many years of brain and trauma research that these types of psychological patterns are the result of neurological damage. In order to better understand the role that the brain may play in the increased pathology evidenced,

it is necessary to look at the neuropsychological research beginning with more controlled animal studies and moving to less controlled human studies. This next section looks at the neuropsychological implications of trauma on the animal brain.

Trauma and Neuropsychological Functioning in Animals

Long ago, it was hypothesized by Charcot (1887), Janet (1889), Breuer and Freud (1893) that psychological symptoms related to childhood trauma have their basis in the brain (as summarized in van der Hart & Friedman, 1989; van der Kolk, Brown, and van der Hart, 1989). However, because techniques were not available for studying the human brain non-invasively, strong conclusions were not possible. Animal studies have been useful in illuminating negative neuropsychological effects of trauma in the animal brain. Researchers over 30 years ago began a series of studies which manipulated the environments of rat pups and then measured brain differences in the groups (Krech, Rosenzweig, & Bennett, 1962: Rosenzweig, 1971; Rosenzwig, Bennett, & Diamond, 1972). Specifically, rat pups were placed into one of two environments, one that was enriching and communal with substantial stimulation or one that was impoverished and isolated with little stimulation. The rat pups from the enriched environment were later found to have developed thicker, denser cortices with brain chemistry that was different than those placed in the impoverished environment. Denenberg (1988) found more specific lateralized deficits in the brains of rat pups as a result of exposure to stressful versus enriched early environments. Rat pups who had been stressed through early human handling exhibited a left-brain bias spatially. More recently, Plotsky and Meaney (1993) found that by separating newborn rats from their mothers repeatedly led to an increase in the production of stress hormones which

resulted in decreased hippocampal size. While animal models are important in generating important ideas about how the human brain may respond to trauma. It is not known how well they generalize. However, some interesting similarities are apparent as will be seen in this next section that looks at Bremner et al.'s (1997) research measuring the human hippocampus.

CSA and Neuropsychological Functioning – Brain Structure

New techniques for examining brain structure non-invasively in humans have made brain-behavior relationships in human trauma more available to examination. Magnetic Resonance Imaging (MRI) is a technique that can distinguish different body tissues based on their differing chemical compositions (Martin, Brust, & Hilal, 1990). It provides spatial resolution comparable to fixed, sectioned anatomical slides. Bremner et al. (1997) used MRI to examine differences in cortical and subcortical structures in 17 adults with a history of childhood physical and/or sexual abuse who currently met criteria for PTSD and controls matched for gender, race, education level, and handedness without such a history or diagnosis. Measurements were obtained for the mid-hippocampal segment, temporal lobe, caudate, and the amygdala. Neuropsychological test data was gathered as well. Results revealed significantly smaller (12 percent) left hippocampal volume in the group experiencing childhood abuse, which was strongly correlated with duration of abuse. Relatively smaller (5 percent) right hippocampal volume and greater left temporal lobe volume was also found in the abuse group. Significant deficits were seen on verbal memory tasks (Immediate Recall, Delayed Recall, and Percent Retention) in the abuse group as compared to the non-CSA group, but this did not show a correlation with hippocampal

volume. This study serves to provide more evidence for limbic system involvement in CSA as well as potential left hemisphere deficits. More specifically, it provides a picture of the possible structural implications of experiencing CSA.

CSA and Neuropsychological Functioning – Brain Function

Another way to examine possible brain correlates of CSA is to look at functional relationships or neural circuitry between brain regions. CSA has been hypothesized to change neural circuitry (Teicher et al., 1993). Functional relationships between such circuitry are often inferred from neuropsychological testing. Moore (2001) looked at the impact of childhood maltreatment on adult left-hemisphere functioning. Specifically, she utilized a dual-task paradigm, pairing right index finger tapping with a left-hemisphere cognitive task, in order to assess left-hemisphere functioning and examine differences in inferred hemispheric organization. Much research has demonstrated that competition for cerebral resources results in greater cognitive interference when two simultaneously performed tasks engage the same hemisphere (Ashton & McFarland, 1991; Hiscock, Kinsbourne, Samules, & Krause, 1985; Kinsbourne & Cook, 1971; Seth-Smith, Ashton, & McFarland, 1989, for example). Thus, a left-hemisphere language processing task interferes with right finger tapping more than left finger tapping. Additionally, if there are deficits in the left hemisphere, as is suggested in the CSA literature (Bremner et al.,1997; Ito, Teicher, Glod, and Ackerman,1998; Ito, Teicher, Glod, Harper, Magnus, and Gelbard,1993), the interference should be greater for those with higher levels of abuse. Though Moore (2001) hypothesized that higher levels of abuse would be associated with greater lateralized interference, she found that lower levels of abuse were associated with greater lateralized interference for females. The females who

experienced high levels of abuse did not exhibit lateralized interference, but the females who experienced low levels of abuse did. Level of abuse did not predict level of lateralized interference for males. Her findings were explained in terms of possibly greater compensation by the right hemisphere in females with higher levels of abuse and crudeness of measurement for capturing the finer details. Because no normal control group was included in this study, it is not known if the high abuse group would have shown lateralized interference in comparison. This study lends support to changes in lateralized neural circuitry in females experiencing low levels of abuse, building on Denenberg's (1988) findings of lateralized deficits in the brains of rat pups as a result of exposure to stressful versus enriched early environments. Additionally, it points to the need for more direct measures of hemispheric organization, while underscoring the importance of distinguishing between levels of abuse in order to better interpret findings.

Changes in neural circuitry are also supported by reports connecting CSA with seizure activity similar to that experienced by individuals with temporal lobe epilepsy but without evidence for surface electroencephalographic (EEG) epileptiform activity (Bowman & Markand, 1996; Bowman, 1993; LaBarbera, & Dozier, 1980; Goodwin, Simms, & Bergman, 1979; Gross, 1979). These types of seizures are found in 10 to 40 percent of people who present to epilepsy centers for comprehensive evaluation of seizures (Gates, Luciano, and Devinsky, 1991). They are called nonepileptic seizures (NES), and they involve sudden changes in behavior with such symptoms as somatic disturbances, brief hallucinatory events, visual disturbances, automatisms, dissociative experiences, and other disturbances of consciousness. These symptoms are consistent with the functional characteristics of subcortical regions highly interconnected to the

temporal lobes. For example, the ` mygdale is associated with processing of emotional, olfactory, and visceral events and with arousal. Differential diagnosis of NES from temporal lobe epilepsy is difficult and expensive requiring video documentation of the individual's seizure-like event, synchronized with EEG data to conclude that there is no surface epileptiform EEG activity (Fisch, 1999). However, Shorvon (1991) has stated that types of complex partial seizures, particularly those of frontal lobe origin, may not be observable with surface EEG measurements for the following reasons: 1) the focus may originate in subcortical structures, 2) the focus may be small, or 3) the focus may have an unusual spatial orientation with respect to the recording electrodes (Fisch, 1999). The temporal lobes are rich in connectivity to deep structures of the limbic system and basal ganglia (Kolb and Whishaw, 1996). It is not uncommon for an individual with severe temporal lobe epilepsy to undergo removal of the affected temporal lobe along with that lobe's deeper limbic structures as a way to resolve epileptic foci (Kolb and Whishaw, 1996).

Building on this knowledge of EEG, brain physiology, and concomitant symptomatology, researchers at McLean Hospital in Belmont, Massachusettes wondered if NES could involve a dysfunction in the deeper structures of the temporal lobes which are not visible to surface EEG (Teicher et al., 1989). Specifically, Teicher et al. (1993) have pursued a line of study that has suggested the limbic system is particularly vulnerable to dysfunction when exposed to early trauma or stress. These researchers developed the Limbic System Checklist-33 (LSCL-33) to measure somatic, sensory, behavioral, and memory symptoms suggestive of temporal lobe epilepsy phenomenon in a study to ascertain its association with CSA. Ratings of lifetime

frequency for experiencing these phenomena were made by 253 outpatients. A group of individuals without psychiatric or neurological abnormalities and no history of abuse had total LSCL-33 scores < 10, whereas patients with documented temporal lobe epilepsy had scores > 23. Abuse was found to have a significant effect on total LSCL-33 scores. Specifically, CSA was associated with a 49 percent increase in LSCL-33 scores compared to normal controls, and patients sexually abused before the age of 18 had scores 66 percent higher than patients whom were never abused. The results suggest limbic system dysfunction in persons experiencing CSA.

Electroencephalograms (EEGs) have been used to record fluctuations of the electrical activity of large ensembles of pyramidal neurons in the brain. Specifically, this technique provides a measure of extracellular current flow associated with summed electrical potentials of neurons beneath the skull. The EEG provides excellent information regarding when activity is occurring in the brain (temporal resolution), although information regarding where activity is occurring in the brain (spatial resolution) is compromised due to volume conduction. Quantitative EEG (QEEG) is a digitized record of the EEG that is subjected to quantitative spectral analyses. Results of spectral analysis from 0.5-30 Hz can be displayed as computed color-graduated topographic maps and can provide measures of frequency, magnitude, absolute power, percent power, coherence, phase, and amplitude asymmetry. Another derivative measure of EEG is the evoked potential. Evoked potentials provide a measure of brainwave activity that is time-locked to a specific stimulus. Measures are obtained during specific sensory stimulation such as an auditory click (auditory evoked potential – AEP) or a visual flash of light (visual evoked potential – VEP).

Ito et al. (1993) used EEG, QEEG, AEP, VEP, and other neurological and neuropsychological measures to look at the association between early abuse and neurological abnormalities. Their subjects were children approximately 13 years old, 27 without a history of verified abuse, 22 with a history of verified psychological abuse, and 55 with a history of verified physical and/or sexual abuse. Patients were evaluated with neurological exams to detect age inappropriate soft neurological signs, and neuropsychological testing to detect poor performance on specific test areas or significant verbal-performance test splits. EEG was used to detect the presence of paroxysmal events, QEEG, AEP (clicks), and VEP (flashes) to detect asymmetries and/or regions of focal slowing. CT and MRI were used to detect mild Chiari I malformations (failure of proper development resulting in malformed cerebellomedullary regions). This comprehensive study found that patients with a history of verified physical and/or sexual abuse were twice as likely as non-abused to have an abnormal EEG, and these abnormalities were found to affect the left hemisphere (frontal, temporal, or anterior regions). Although neurological exams, neuropsychological tests, and imaging studies in this report did not significantly differentiate the abused from the non-abused group, neuropsychological testing in the abuse group revealed substantially better performance than verbal scores, implying left hemisphere “underdevelopment.” This study highlights the importance of looking at lateralized effects in brain function, undergirding Denenberg’s (1988) animal research. It is hampered by its failure to control for drug effects and its failure to assess for PTSD level.

Continuing with this line of research, Ito et al.(1998) looked at the relationship between EEG coherence and early abuse. Coherence is the morphological similarity

between two sites in the brain irrespective of time synchronization, with a correction for absolute magnitude. Mathematically it is the cross-correlation of the power/amplitude of activity at two sites. Thus, sites that covary highly are presumed to possess high connectivity or are processing related cortical/subcortical information. The value of coherence is its ability to infer activity of local arcuate, fronto-temporal (uncinate) and interhemispheric ` mygdale tracts, which connect sites and are invisible to the EEG. Coherence adds substantial clinical value to surface EEG data because it enables inferences about deeper structures, like the limbic system. A limitation of EEG coherence is that it is not precise in its specification of location in anatomical space. Talking about hypercoherence at an electrode site is analogous to describing a person as “big” without specifying height and width. Ito et al. (1998) found higher levels of left hemisphere alpha coherence and significant left greater than right asymmetries in the alpha band in individuals experiencing childhood sexual and/or physical abuse. Additionally, it was found that left hemisphere coherence decayed more rapidly across electrode distance in normal subjects as compared to abused subjects, implicating deficits in left-sided brain functional differentiation among abused subjects. Unfortunately, this study is limited by its focus on alpha band (8-12 Hz) coherence only, its failure to control for drug effects, and its failure to assess PTSD status.

In an EEG auditory evoked potential study (AEP), Schiffer, Teicher, and Papanicolaou (1995) compared functional activity between the two hemispheres during recall of a recent neutral memory and then a painful childhood memory in 10 subjects with a reported history of childhood trauma and 10 subjects without. The trauma group showed a significant shift in N1-P2 amplitude over the left and right auditory cortex

between the two conditions, while the non-trauma group did not. Specifically, the trauma group showed significant left-dominant asymmetry during the neutral task and relative right dominance during the painful memory. The findings suggest a lack of integration of the two hemispheres in those experiencing early trauma, as well as preferential storage of traumatic memories in the right hemisphere.

Another pilot study from the University of North Texas looked at QEEG and MMPI-2 patterns of 12 adult outpatients reporting CSA and compared them with a matched sample of outpatients who reported they had not experienced CSA (Townsend, Black & Bodenhamer-Davis, 2001). The researchers found the CSA group had significantly lower alpha relative power in most all leads of the International 10-20 electrode placement sites. Alpha is a brain rhythm associated with suspended processing, and relative percent power provides an index for the allocation of energy in the brain to the various frequency bands. Exploratory post hoc analyses found differences between the groups in the relationship between variability of alpha relative power in the posterior regions and frontal-posterior coherence. Specifically, focal versus diffuse alpha relative power in posterior regions was only related to frontal-posterior connectivity in the non-CSA group. These preliminary findings implicate a lack of regulation of the posterior sites via the frontal cortex in individuals with a history of CSA.

A related study by Black et al.(2002) looked at EEG abnormalities and the QEEG coherence patterns of 15 adult outpatients reporting CSA and compared them with a matched sample of outpatients who reported they had not experienced CSA. Increased EEG abnormalities were found to be associated with the CSA group. Decreased connectivity (functional differentiation) was found to characterize left frontal regions in

the theta and beta bands, while increased connectivity (functional redundancy) characterized posterior central regions across all bands in the CSA group as compared to the NCSA group. Important caveats of this study were that it did not control for drug effects nor did it assess PTSD status.

The above self-report and EEG studies provide evidence for neuropsychological consequences associated with CSA, more specifically, left hemisphere deficits with possible limbic system involvement and inadequate frontal lobe regulation of posterior sites in CSA. Unfortunately, it is not known how PTSD may or may not be involved in the neuropsychological correlates found. While some of the findings are consistent with PTSD research, there are some differences. Screening for PTSD status would be useful for reducing confounding variables. The next study described explored the relationship between PTSD status and brain function.

Research with Humans Experiencing PTSD

Positron Emission Tomography (PET) combines the principles of computerized tomography and radioisotope imaging to provide a picture of the distribution in brain tissue of an injected or inhaled isotope that emits radiation. This technique is valuable for examining biochemical processes such as brain metabolism and distribution and density of transmitter receptors. PET provides a picture of both structure and function in the brain. Shin, et. Al (1999) used PET to compare CSA groups with and without DSM-III-R PTSD diagnosis (N=8/group) for differential activation in anterior limbic and paralimbic regions of the brain during recall and imagery of a neutral personal experience and a stressful sexually abusive personal experience. Heart rate and blood pressure was measured concomitantly, and emotional intensity and vividness of

imagery ratings were collected immediately following the PET procedure. Results indicated that both groups exhibited regional cerebral blood flow increases in orbitofrontal cortex and anterior temporal poles, and decreases in visual cortex and related visual areas during recall of sexual trauma. However, increases were greater in the PTSD group. The non-PTSD group exhibited regional cerebral blood flow increases in anterior ` mygdale` d gyrus that were greater than the PTSD group. The non-PTSD group exhibited regional cerebral blood flow increase in insular cortex during recall of sexual trauma, while the PTSD group did not. Regional cerebral blood flow increases in the ` mygdale were expected but not seen in either group during any of the conditions. Significantly greater heart rate responses were revealed for the PTSD group during the recall of sexual trauma, and ratings showed this group experienced greater increases in emotional arousal and vividness of imagery across conditions. Findings of orbitofrontal and anterior temporal pole activation are consistent with studies indicating this region's association with normal emotion. The greater intensity of emotion and vividness experienced by the PTSD group likely explains why the activations were greater in this group than the non-PTSD group. That the PTSD group exhibited less activation of the anterior ` mygdale` d gyrus, a region associated with activation during recall of sad events in normal controls, may suggest dysfunction consistent with the above evidence for limbic system dysfunction in these subjects. It also illuminates important differences between groups who have resolved early trauma and those who have not, indicating the importance of screening study subjects accordingly.

Thus far, it is apparent that there are differences exhibited both psychologically and neuropsychologically between individuals who have and have not experienced

CSA. This next section will briefly explore a possible mechanism for the differences evidenced.

A Possible Mechanism for the Psychological and Neuropsychological Correlates of CSA

The above research suggests that CSA affects the brain adversely structurally and functionally. Negative associations are seen in both children and adults. Experimental research demonstrating the adverse impact of trauma on the developing brain serves to make the negative neuropsychological associations seen in humans stronger by controlling for possible confounding variables. How trauma might produce negative neuropsychological consequences will be addressed below.

A well-researched hypothesis for how trauma might change brain structure and function is through the release of stress hormones. Cannon (1914) introduced the concept of “fight-or-flight” reactions to stress, illuminating the body’s response to emergency situations. Selye (1956) proposed the theory of a general adaptation syndrome (GAS) in the face of prolonged stress. The brain’s response to threat begins with sensory input from various organ systems to the thalamus, which is then routed to the appropriate cortical regions and the limbic system. The right ` mygdale (part of the limbic system) assesses the emotional meaning and content of the sensory input, attaching emotional valence, and sends it on to the right hippocampus and orbitofrontal cortex for further information and memory processing. This is done via norepinephrine input from the locus ceruleus (part of the reticular activating system) so that the body can respond motorically and autonomically to the emergency (LeDoux, 1996).

Input from the orbitofrontal cortex to the hypothalamus leads to sympathetic arousal via the release of epinephrine by the adrenal medulla. Further input from the

locus ceruleus activates the hypothalamic/pituitary/adrenal (HPA) axis. In response, the hypothalamus releases corticotrophic releasing hormone (CRH) which triggers release of adrenal corticotrophic hormone (ACTH) by the pituitary, which then triggers release of cortisol by the adrenal cortex. The effect of cortisol on the body is to turn some body systems on while at the same time turning off other body systems in order to mobilize the body for either a fight response or a flight response by sacrificing energy storage and diffuse tissue building in favor of energy release and tissue catabolization (LeDoux, 1996). Cortisol is positive and facilitative to deal with all kinds of arousal situations. However, its effects can become negative when prolonged or in excess. For example, hippocampal cell death is seen in primates exposed to prolonged and elevated levels of cortisol (Sapolsky, Uno, Rebert, & Finch, 1990). In a study of human infants, Gunnar and Nelson (1994) found cortisol levels to be negatively related to hippocampal activity.

That this state of on-going arousal with its concomitant negative results is a part of the effects of CSA has been researched. An important longitudinal study which looked at the psychobiological effects of CSA in girls age 6-15 found evidence for changes in the regulatory dynamics of trauma-associated biological responses (Putnam & Trickett, 1993; Trickett, & McBride-Chang, 1995; Trickett, Reiffman, Horowitz, & Putnam, 1997). Specifically, they found evidence for HPA axis dysregulation, elevated catecholamine levels (implicated in stress and trauma responses), and decreased immune function in sexually abused girls in comparison to controls. It is important to note that only one of the girls included in the cortisol portion of the study met DSM-IIIR criteria for PTSD. Though PTSD research with combat-related trauma also implicates changes in the regulatory dynamics of neuroendocrine pathways (Yehuda & McFarlane,

1995), there are differences in CSA trauma, which implicate the interaction of developmental variables (Trickett & McBride-Chang, 1995). This interaction with development offers us clues as to why previous research suggests greater left-hemisphere deficits in CSA (Ito et al., 1993; Ito et al., 1998).

Ito et al., 1998 offered an explanation for how development may interact with CSA to affect brain-related changes in functioning. Though there appears to be a bias toward right-hemisphere development in the first few months of life, by the sixth month, rate of development for the left-hemisphere increases over the right-hemisphere. This increased rate of differentiation continues for 3 to 6 years. If CSA with its concomitant physiological changes interrupts this process, deficits may result which are different than those seen when trauma occurs in adulthood.

CHAPTER 2

PURPOSES OF THE STUDY

To summarize, research to date exploring psychological and neuropsychological correlates of childhood sexual abuse (CSA) suggests psychological profiles with increased levels of pathology and neuropsychological effects in the left hemisphere with possible limbic system involvement and inadequate frontal lobe regulation of posterior brain sites. With this in mind, the first aim of the present study was to extend to a non-clinical group the findings of Black et al. (2002) and to an adult group the findings of Ito et al. (1993) showing childhood sexual trauma to be differentially associated with neurological abnormalities. Two adult groups (CSA and NCSA) were compared on prevalence of regions of focal slowing, epileptiform activity, and/or asymmetries in the EEG. The second aim of this study was to extend Ito et al. (1998) findings of higher levels of left hemisphere alpha coherence and reversed asymmetry in children experiencing physical and/or sexual abuse to an adult sexual trauma group, as well as explore activity in other EEG frequency bandwidths. This objective also extended to an adult non-clinical group the findings of Black et al. (2002). Specifically, the CSA group was compared with the non-CSA group for overall left versus right hemisphere coherence as well as focal coherence differences. Third, the Townsend et al. (2001) findings of a lack of anterior-posterior regulation in childhood sexual trauma as well as Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989), findings of

elevated F, floating profiles, and/or 4-5-6 configuration was extended as a way to infer pattern and degree of cortical development through coherence measures as it combines with personality. Lastly, this study extended the Black et al. (2002) examination of EEG connectivity through the addition of instruments to screen for confounding factors (e.g. Post Traumatic Stress Disorder) and to confirm asymmetries behaviorally through examination of intelligence.

Hypotheses

Nine hypotheses were tested:

Hypothesis 1: There will be significantly more total raw records including epileptiform events (defined as a spike followed by slowing which occurs in the same location more than one time in the same record and cannot be attributed to artifact) in the EEG records of the CSA group subjects than in those of the NCSA group.

Hypothesis 2: There will be significantly more temporal alpha rhythm patterns (defined as alpha rhythm in regions other than occipital or parietal areas which is present 75 percent of the time that alpha is present in other posterior regions and attains amplitude equal to or higher than posterior regions at least once in the record) in the raw EEG records of subjects in the CSA group than in those of the NCSA group.

Based on these hypotheses, it was expected that consistent with Black et al.'s (2002) findings the CSA group would show a greater statistical prevalence of paroxysmal events and mixed dominance in the EEG than the NCSA group.

Hypothesis 3: There will be significantly fewer records showing a 25 percent or greater decrease in the amplitude of the alpha rhythm in the occipital or parietal regions

in the eyes open as compared to eyes closed raw EEG records of subjects in the CSA group compared to those in the NCSA group.

Based on this hypothesis, it was expected that the CSA group would fail to demonstrate the normal EEG pattern of alpha blocking upon eye opening. Though eyes open data have not been studied in conjunction with CSA, there is some clinical evidence (Brownback & Mason, personal communication, September 14, 2002) to suggest a lack of attenuation in this group.

Hypothesis 4: There will be significantly smaller average vector length distances from the origin in the theta band on the right for the CSA group when compared to the NCSA group.

Hypothesis 5: There will be significantly smaller average vector length distances from the origin in the alpha band on the left for the CSA group when compared to the NCSA group.

According to these hypotheses, it was expected that the two groups would exhibit differences of right and left hemisphere coherence in at least one of the bandwidths. Specifically, it was expected that Black et al.'s (2002) findings of theta hypercoherence on the right and alpha hypercoherence on the left in CSA adults as compared to NCSA adults and Ito et al.'s (1998) findings of greater average alpha left hemisphere coherence in abused children as compared to normals would be replicated.

Hypothesis 6: There will be significantly more records exhibiting Scale F as 10 points or greater than Scales L and K for the CSA group than for the NCSA group.

Hypothesis 7: There will be significantly more records showing the 4-5-6 configuration (defined as Scale 4 and 6 above a T score of 65, and scale 5 below a T score of 35) in the CSA group than in the NCSA group.

Hypothesis 8: There will be significantly more records showing the floating profile pattern (defined as 6 or more Scales elevated above a T score of 65) in the CSA group than in the NCSA group.

Therefore, it was expected that the CSA group would exhibit elevated levels of pathology on the MMPI-2 (high F, floating profiles, and/or 4-5-6 configuration).

Hypothesis 9: The CSA group will exhibit significantly greater Performance – Verbal IQ differences than the NCSA group.

According to this last hypothesis, the two groups were expected to differ with regard to verbal-performance patterns of intelligence, with the CSA group exhibiting greater deficits in verbal abilities.

Exploratory Analyses

The two groups were expected to exhibit a different degree and pattern of coherence in at least one of the bandwidths at the level of the individual electrode as was demonstrated in the Black et al. (2002) study. However, because an unmedicated, nonclinical sample was recruited, it was not known prior to the study what frequency or localization patterns might be exhibited. An exploration of this difference in QEEG coherence patterns between groups was undertaken.

Additionally, the two groups were expected to exhibit a different item endorsement pattern on the MMPI-2. In order to describe more specifically differences

between the CSA and NCSA groups, an exploration of individual MMPI-2 scale differences was undertaken.

CHAPTER 3

METHOD

Participants

Childhood Sexual Abuse (CSA) Participants. Thirty-three participants who reported they had experienced childhood sexual abuse were recruited from the Dallas/Fort Worth area by fliers posted in the University of North Texas (UNT) Psychology Clinic, the UNT Counseling and Testing Center, the UNT Student Health Center, Denton Friends of the Family (a local woman's shelter), and areas designated for public postings across campus. Incoming new clients of the UNT DRSWA Neurotherapy Lab were also recruited as participants. The individuals were interviewed to assess exclusionary criteria and childhood sexual abuse.

NCSA Participants. Thirty-two matched control participants who reported they had not experienced CSA were recruited from the Dallas/Fort Worth area by fliers posted in the University of North Texas (UNT) Psychology Clinic, the UNT Counseling and Testing Center, the UNT Student Health Center, and areas designated for public postings across campus. Incoming new practicum students of the UNT Neurotherapy Lab were also recruited as participants. The individuals were interviewed to assess exclusionary criteria and childhood sexual abuse.

Potential subjects were included without regard to gender or race. Requirements for subject inclusion were sexual trauma experienced before age 14 and denial of traumatic brain injury, loss of consciousness greater than five minutes, current alcohol or substance abuse, or current use of medication suspected to affect the EEG. Participants were asked to meet medication wash-out times (5 to 7 times the half-life)

for all substances except caffeine and nicotine, for which one to four times the half-life was considered sufficient to avoid withdrawal effects on the EEG. Requirements for control inclusion were denial of any type of childhood trauma (e.g., physical abuse) plus no major head injuries, no current depression, no sleep disorders, no current psychiatric diagnoses, no current overuse of drugs or alcohol, no neurological disease, and matched age (within 7 years), gender, and handedness with a subject. In addition, all participants were 18 years of age or older. All participants meeting selection criteria were given a free topographic map of their eyes closed QEEG results and a 20-minute explanation upon completion.

There were 24 participants included in each group for this analysis based on meeting match criteria, having an EEG meeting acceptance criteria, and being right-handed. Tables 1 and 2 provide sociodemographic characteristics of the sample. Their ages ranged from 18 to 59 (mean $\pm$ SD = 32.31 $\pm$ 11.42). Females comprised 92% of the sample, and a European/white ethnic background comprised 83% of the sample. Participants with some undergraduate college education comprised 50% of the sample, while participants with a graduate level education comprised 46% of the sample.

Table 1

Sociodemographic Characteristics for CSA and NCSA Groups

	CSA n=24		NCSA n=24	
	M	SD	M	SD
Age	31.6	10.4	33.0	12.4
Verbal IQ	115.0	11.2	115.5	11.5
Performance IQ	115.1	8.1	112.6	10.8
P-V Difference	.1	11.8	-2.8	9.0

Table 2

Sociodemographic Characteristics for CSA and NCSA Groups

	CSA n=24		NCSA n=24	
Variable	n	%	n	%
Gender				
Male	2	8	2	8
Female	22	92	22	92
Ethnicity				
Caucasian	21	87	19	79
African American	3	13	2	8
Asian	0	0	3	13
English as 2nd Language	0	0	4	17
Education				
12 years	0	0	2	8
13-14 years	9	38	4	17
15-16 years	7	29	4	17
16 + years	8	33	14	58
PTSD	4	17	0	0

Measures

All participants gave written informed consent for participation after receiving a full explanation of the procedures. All testing was performed by an advanced level doctoral candidate in the field of psychology, who was under the supervision of a

licensed psychologist. All procedures were approved by the Committee on the Use of Human Subjects at the University of North Texas.

QEEG Clinical Interview. Medical and developmental history as well as childhood sexual abuse was gathered by clinical interview providing information on the perpetrator, participant's age at the time of the first abuse, duration, frequency, nature, and severity of the abuse. Sexual abuse was defined as sexual intrusion, genital or digital penetration, molestation with genital contact, or fondling at or before the age of 14 by others exhibiting some sort of power over the child by virtue of being older or more powerful physically or mentally than the child.

Clinician-Administered PTSD Scale (CAPS; Blake, Weathers, Nagy, Kaloupek, Klauminzer, Charney, Keane, & Buckley, 2000). The CAPS is a semi-structured interview developed by the National Center for Posttraumatic Stress Disorder which provides information related to DSM-IV criteria for PTSD. The semi-structured format allows the clinician to inquire symptoms from the past week, month or lifetime depending on the desired diagnosis. For the purposes of this study, symptoms over the last month were inquired in order to gain information related to current PTSD symptoms. Empirical research has demonstrated the CAPS to be reliable psychometrically based on inter-rater reliability, test-retest reliability, and internal consistency (Weathers, Ruscio, and Keane 1999; Nagy et al., 1999).

Wechsler Abbreviated Scale of Intelligence™ (WASI™) owned by The Psychological Corporation (Harcourt Brace & Company, San Antonio, TX) . Psychological testing of IQ was performed using the WASI. Scores were evaluated for the presence or absence of abnormality based on significantly poor performance on

specific test areas or significantly high verbal-performance splits in comparison to norms.

Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989). The MMPI-2 is a 567-item inventory which uses a true/false self-report format to objectively assess personality. It has 3 scales which assess validity and ten which assess basic personality characteristics. The MMPI-2 was used to assess long-term effects of CSA on personality. The MMPI-2 has two supplementary scales which assess PTSD, the PK (Keane, Malloy, & Fairbank, 1984)and PS (Schlenger & Kulka, 1989) scales, which also were utilized in this study.

Procedures

All procedures for this study were approved by the Committee on the Use of Human Subjects at the University of North Texas. All data collection procedures for the study were performed by the researcher and a QEEG-qualified colleague.

Prior to the EEG collection appointment, potential participants completed an initial phone screening where the study was explained and eligibility on some of the inclusion/exclusion criteria was assessed, most importantly that related to drug usage. If initial criteria were met, potential participants were given an appointment time and a list of instructions for complying with standard procedures. Those included: Double washing the hair with a stripping shampoo, obtaining a good night's rest for two nights prior to the appointment, eating a substantial meal 1 ½ - 2 hours prior to appointment time,

abstaining from caffeinated beverages the morning of the appointment, and abstaining from alcohol and over-the-counter drugs according to wash out times.

Upon arrival a face-to-face interview was conducted to explain the study, obtain written informed consent, and to determine eligibility for the remaining inclusion/exclusion criteria. Those who met criteria were prepared for EEG collection.

During EEG collection, participants were seated comfortably in a sound-attenuated room with windows allowing for natural light. Brain electrical activity was digitally recorded on a Lexicor digital EEG system from 19 scalp electrodes on a Lycra cap (ElectroCap), according to the International 10-20 System of electrode placement (Jasper, 1958), and referenced to linked ear electrodes and to a forehead ground contained in the cap. Eye movements were detected using a bipolar vertical electro-oculogram (EOG) lead. A single forehead electrode served as ground. Electrode resistances were kept below 5 Kohms and equal to within $\pm$ 1 Kohm between leads. Bandpass filters were set at 0.5-30 Hz, and the sampling rate was set at 128 samples per second. Recordings were obtained in the awake state with eyes closed (2 collections of 5-8 minutes) and in the awake state with eyes open (2 collections of 5-8 minutes). To meet the objectives of the study, recordings were digitized, visually edited to reduce artifacts (contamination resulting from muscle tension, eye and body movements) according to field standards (Hammond & Gunkelman, 2001), and subjected to quantitative spectral analysis. A total of 30 to 100 seconds of artifact-free recording were selected from each collection in the eyes closed condition for analysis. EEG was reformatted in a variety of referential and bipolar montages for visual evaluation of the presence or absence of regions of focal slowing, epileptiform activity,

and/or asymmetries, by two trained QEEG technicians. Interrater reliability of ratings were 80 percent or greater for all visual evaluations of abnormality. Discrepancies were resolved for 100 percent agreement. NeuroRep: The QEEG Analysis and Report System developed by Hudspeth, 1999 and owned by Grey Matter, Inc., Reno, NV (NeuroRep) was used to analyze absolute magnitude and EEG coherence in each of the bands (delta, theta, alpha, and beta), and did not contain paroxysmal discharges. Scalp electrodes were sterilized after collection from each participant.

After EEG collection, participants completed the clinical interview. They were then given a break to walk around, get something to eat and/or drink. Psychological testing was then conducted. It was possible to schedule this segment of participation for a separate time if necessary. Participants filled out the MMPI-2. They were then administered the WASI and the CAPS (Blake, Weathers, Nagy, Kaloupek, Klauminzer, Charney, Keane, & Buckley, 2000). Upon completion of the study, participants were given the opportunity to schedule an appointment with the researcher to obtain a color printout of their brain map along with a free 20-minute explanation of their results with the principal investigator.

CHAPTER 4

DATA ANALYSIS

Visual inspection of the EEG data included ratings for presence or absence of abnormal activity. The researcher and a QEEG-qualified colleague rating the data were blind to group inclusion. Abnormal activity was defined as: 1) focal 0 to 8 Hz activity which is seen in a region encompassing no more than 5 of the 10-20 System sites, 2) spike activity followed by slowing (epileptiform) which is seen more than once in a record with the same focus of origin, and 3) alpha amplitude asymmetry greater than 75 percent between homologous locations and which does not exhibit shifting power between homologous sites. Data was also rated for the presence or absence of spindling beta activity, low voltage ($\leq$ 20 uV) 20 to 24 Hz pattern of rising and falling amplitude activity, and temporal alpha rhythm in which the alpha rhythm is present in temporal regions at least 75 percent of the time that posterior alpha is present and reaches an amplitude that is equal to or greater than posterior alpha rhythm amplitude at least one time. The posterior alpha rhythm was further rated for its presence in the eyes open recording according to a 3-point scale: 0 representing a lack of attenuation in which the alpha rhythm is present at an amplitude within 20 percent of that exhibited in the eyes closed record, 1 representing attenuation of the alpha rhythm in which amplitude is decreased by at least 25 percent and is impersistent in comparison, and 2 representing a lack of alpha rhythm in either record.

Editing was performed on the data by the researcher according to field standards as outlined in Hammond and Gunkelman (2001). All data was coded so that the researcher was blind to group inclusion. Movement artifacts, muscle tension creating

amplitudes greater than 5 microvolts, artifacts associated with drowsiness, electrode artifacts, and transients 50 percent greater than background activity which also represented a change in kind from ongoing activity were removed from the eyes closed records. Reliability between the records was reviewed, and the best record or a combination of the two recordings was analyzed according to the following criteria: If reliability measures for coherence bandwidths as well as individual channels were greater than or equal to 80 percent, the records were added together and analyzed. If the reliability between the records did not meet these standards, the record that included the participant's best performance was chosen for analysis according to the researcher's clinical judgment. The eyes open records were used only to assess for alpha blocking and were not edited or analyzed.

Connectivity indices were created by means of a complex demodulation program that computes auto- and cross-spectral power density for all 19 channels, using a second order recursive digital filter developed by John, expanded by Hrybyk and then extended to 19 channels by Hudspeth (Hudspeth, 1988; John et al., 1980; Thatcher, Krause & Hrybyk, 1987). Coherence, phase, and asymmetry relationships between all pairwise combinations of 19 electrodes (n=171) for four frequency bands (delta, theta, alpha, and beta) and the unfiltered cross correlation are computed in the program. The results produced by the program are essentially identical to a fast Fourier transformation, though it is more efficient as results are only computed for the frequency bands (Otnes & Enochson, 1972).

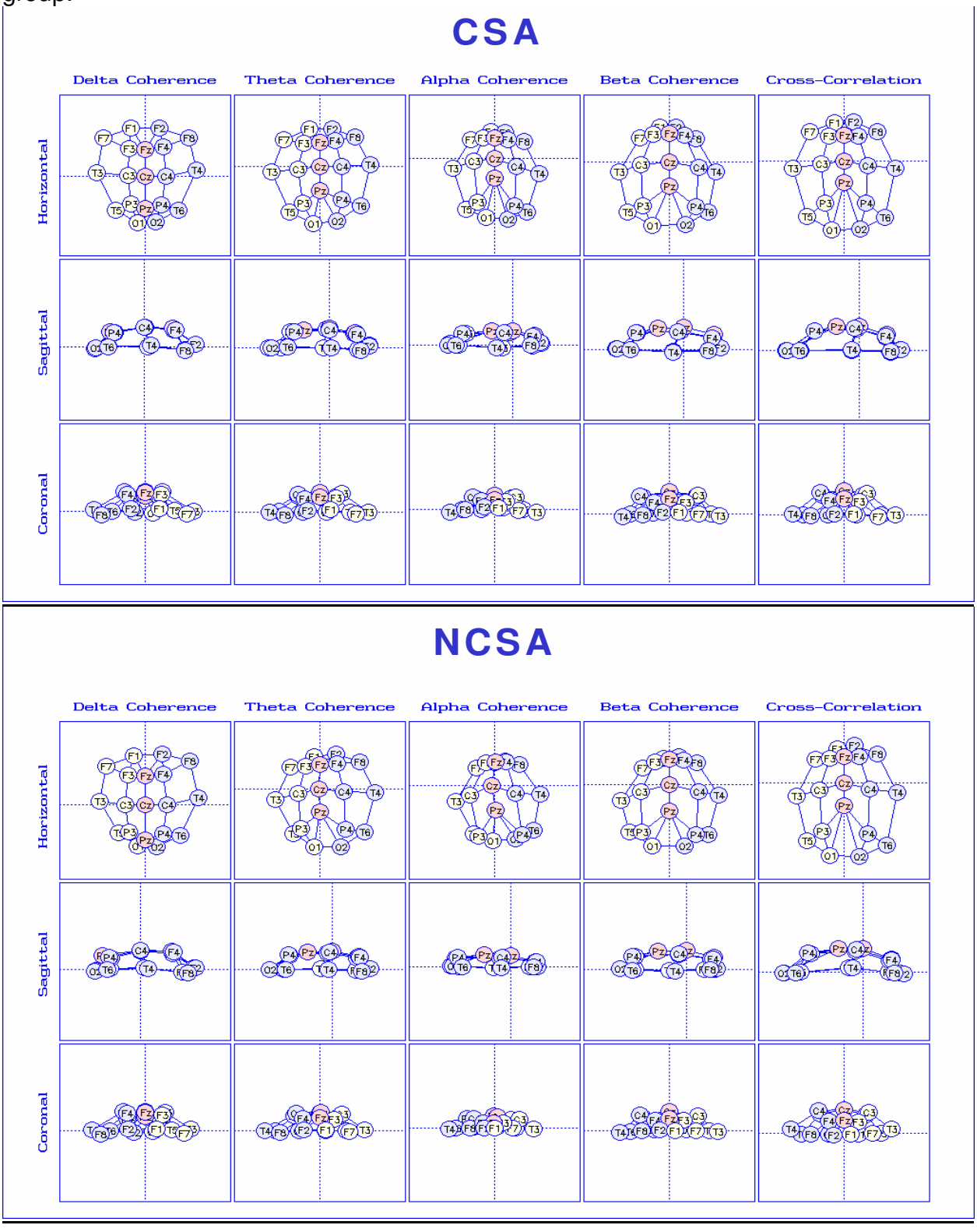
In order to reduce statistical problems associated with the large number of statistical comparisons (171 X 19 matrixes) inherent in analysis of coherence, data was

subjected to Principal Components Analysis (PCA) (Pearson, 1901; Hotelling, 1933) as adapted for waveform data (John et al, 1964). The component loadings were then used to determine the three dimensions (basis waveforms) inherent in each set of 19 waveforms which reflect anatomical location (Hudspeth, 1993). The resulting 3-dimensional eigenimage or “neuroelectric image” (NEI) reflects a reduction of the data into a 19 X 3 matrix (Hudspeth, 1999).

NEI’s were generated in the NeuroRep (Hudspeth, 1999) software and based on the mean for each group. An example of an NEI can be seen in Figure 1. The NEI provides three different views of the coherence relationships for four frequency bands plus a combination of all frequency bands. The horizontal view provides left-right and anterior-posterior location information. The sagittal view provides anterior-posterior and dorsal-ventral location information. The coronal view provides left-right and dorsal-ventral location information. The horizontal and vertical dotted lines represent functional brain divisions. The intersection of the horizontal and vertical dotted line represents the point of origin, which is a point in brain space directly below CZ.

NEI’s uniquely map brain anatomy into functional space so that the functional connectivities between sites are represented by the distance between brain locations (Hudspeth, 1999). Large distances are associated with hypocoherence or increased differentiation among sites, and small distances are associated with hypercoherence or redundancy among sites.

Figure 1. Neuroelectric Images based on means for the CSA group (top) and NCSA group (bottom). Shows three views of coherence for each band (delta, theta, alpha, beta) as well as the bands collapsed together (cross-correlation) for each group.



NEI's provide an immense reduction of coherence information while preserving a parsimonious view of functional brain space (19 X 3 matrix). Despite this reduction, it is necessary to further reduce the data for statistical analysis due to the small sample size. Therefore, all three dimensions (component loadings) of the NEI were used to compute a vector length for each electrode:

$$\sqrt{x^2 + y^2 + z^2}$$

The resulting value represents the distance from the origin, which is a point directly below CZ.

The above represents a reduction of the 3 X 19 matrix to 19 vector lengths with a functional anatomic grounding. Each vector length represents functional distances, where larger distances from the origin are associated with less coherence or connectivity in that region and smaller distances from the origin are associated with more coherence or more connectivity in that region.

In order to compare these findings with those of Ito et al. (1998), the data were further reduced to reflect differences between left and right hemisphere coherence. To do this, vector lengths representing distance from the origin for each of eight electrodes per hemisphere (excludes vertex sites) were averaged to obtain composite hemisphere measures.

Data was also analyzed for each site (electrode). Thus, vector lengths representing distance from the origin for each of 19 electrodes were compared. Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University

of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989), Wechsler Abbreviated Scale of Intelligence™ (WASI™) owned by The Psychological Corporation (Harcourt Brace & Company, San Antonio, TX), and the Clinician-Administered PTSD Scale (CAPS; Blake, Weathers, Nagy, Kaloupek, Klauminzer, Charney, Keane, & Buckley, 2000). CAPS data was scored. MMPI-2 and WASI test scores were converted into T scores. CAPS scores were used to determine presence or absence of PTSD.

CHAPTER 5

STATISTICAL ANALYSIS

Descriptive statistics, graphs, and correlation matrices representative of the data were generated and examined in order to identify means, standard deviations, ranges, univariate outliers, as well as highly correlated variables. Robust analyses of data were performed along with standard analyses to test the specific hypotheses.

Lateralized as well as individual vector length differences between groups were compared using Student's t-tests to accept or reject the null hypothesis that the groups' mean vector lengths were equal. Pearson's chi square tests with Yates' continuity correction were used to analyze differences between groups for incidence of neurological assessment abnormalities.

Due to small sample size and potential for outliers, there was an increased likelihood the two groups would not meet the assumptions of normality. It has been demonstrated (Wilcox, 1998) that small departures from normality decrease power substantially, which decreases chances for detecting real differences between groups. Robust analyses were therefore implemented to prevent small distribution changes from making large changes in power or probability coverage. Classical analyses were provided as well for comparison purposes; however, only robust results were interpreted. The goal was to avoid missing important discriminations in the data due to low power (Type II error) or unequal variances between groups (Type I error). Specifically, robust mean and robust variance estimates were used to calculate robust effect sizes. See Wilcox (1997) for a detailed discussion of robust analysis.

Cohen's d which assumes equal variance was used in the calculation of standard effect sizes for group differences (Cohen, 1988):

$$d_{Cohen} = \left| \frac{\hat{u}_1 - \hat{u}_2}{\hat{\delta}_{pooled}} \right|$$

Where $\hat{u}$ is the estimated population mean, and $\hat{\delta}_{pooled}$ is the pooled estimated population standard deviation. Tentative interpretive benchmarks for d_{Cohen} are: .10 to .30 = a small effect size; .40 to .60 = a medium effect size; and .70 and beyond = a large effect size when there is no prior research to rely on for effect size estimates (Cohen, 1992). For Cohen's d, the amount of variance in the dependent variable by membership in the childhood sexual abuse (CSA) group for a small effect size is 2 to 2.2 percent, for a medium effect size is 3.8 percent to 8.3 percent, and for a large effect size is 10.9 percent and beyond.

M-estimators which do not assume equal variances among groups was used in the analysis of robust effect sizes to control for inflated sample variance, long confidence intervals, and poor power with the following formula:

$$d_{robust} = \frac{P_{Mest1} - P_{Mest2}}{\hat{\xi}_{bi1}}$$

Where P_{Mest} is the robust M-estimator, and $\hat{\xi}_{bi1}$ is the square root of the biweight midvariance for group 1. M-estimators remove extreme values when estimating a measure of location and trim to achieve evenly tailed distributions (making possible asymmetric trimming or no trimming). For a more detailed discussion of M-estimators see Wilcox (1997).

Cohen's r was used to calculate effect sizes for the descriptive variables and qualitative EEG ratings with the formula:

$$r_{Cohen} = \sqrt{\frac{X^2(1)}{N}}$$

described by Rosenthal and DiMatteo (2001). Cohen (1992) reports that using r, a value of .05 to .148 can be interpreted as a small effect size, a value of .196 to .287 can be interpreted as a medium effect size, and a value of .330 and higher can be interpreted as a large effect size. For Cohen's r, the amount of variance in the dependent variable by membership in the CSA group for a small effect size is 2 to 2.2 percent, for a medium effect size is 3.8 percent to 8.3 percent, and for a large effect size is 10.9 percent and beyond.

Bootstrapping techniques were used to generate robust p-values, power, and confidence intervals (Herrington, 2001; Wilcox, 1997; Beran, 1986). Bootstrapping techniques treat the sample as if it were the population, resampling from the mean centered scores with replacement to produce new groups of scores to compare. The critical value of the bootstrap test will be obtained from the sample distribution using 500 bootstrap samples. This technique does not assume normality, but does assume symmetry in order to obtain narrow confidence intervals.

In order to investigate the differences between the two groups for Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989), and intelligence data, means and standard deviations were computed for each group. MMPI-2 scores were graphed

to detect the inverted-V (scale F at least 10 T score points higher than L and K) validity patterns, floating profiles, and 4-5-6 configurations (scale 5 at least 30 points lower than scales 4 and 6). MMPI-2 scores were subjected to Student's t-tests with Benjamini False Discovery Rate p-value corrections for dependent variables, while patterns were subjected to Pearson's chi square analysis to determine significant differences between the groups. A one-tailed Student's t-test was performed on the IQ differences.

CHAPTER 6

RESULTS

Preliminary Analyses

Prior to analyzing the data for the separate hypotheses, a Pearson's chi square analysis was performed on the descriptive variables to assess for differences between the groups. Responses yielded scores of 0 (no) or 1 (yes) for all but two variables (number of words remembered and reported level of depression) for which Pearson's point biserial correlation was used. Table 3 provides chi square, degrees of freedom, p-value, and effect size values for the descriptive variables. The groups could be differentiated by large effect sizes on several variables. Childhood sexual abuse (CSA) group membership was associated with having experienced seizures or seizure-like symptoms, past drug abuse, memory difficulties (as confirmed by remembering less words they were asked to remember at the beginning of the interview), experiencing sleep difficulties, experiencing frequent headaches or migraines, and having participated in counseling.

Table 3

Chi Square Analysis of Descriptive Variables

DESCRIPTIVE STATS	chi- square	d.f.	p-value	Cohen's r
Birth complications	2.509	2	0.285	0.229
High fevers/Ear infections	2.101	2	0.35	0.209
Met milestones on time	1.021	2	0.6	0.146
Repeated a grade in school	2.087	2	0.352	0.208
Seizure or symptoms of	5.581	2	0.061	0.341
Prior EEG	1.021	2	0.6	0.146

DESCRIPTIVE STATS for Table 3 (cont.)	chi square	d.f.	p-value	Cohen's r
Past drug abuse	10.157	2	0.006	0.46
Past alcohol abuse	2.944	2	0.229	0.248
Family hx of alcoholism	2.978	2	0.226	0.249
Memory difficulties	19.664	2	0.0001	0.64
Words remembered	pt. biser. -0.4548	6		0.207
Episodes of confusion	4.364	2	0.113	0.3
Level of reported depression	pt. biser. 0.087	12		0.008
Family hx of depression	3.136	2	0.208	0.256
Sleep difficulties	6.047	2	0.049	0.355
Daytime drowsiness	3.998	2	0.136	0.288
Bizarre mentation	3.2	2	0.202	0.258
Headaches/migraines	7.005	2	0.03	0.382
Past counseling	15.736	2	0.0004	0.573
Ever arrested	4.639	2	0.098	0.311
Daily Caffeine	1.861	2	0.394	0.197
Smoker	1.627	2	0.443	0.184
Use meditation	0.9	2	0.638	0.137
Exposure to toxic agents	1.282	2	0.527	0.163
PTSD	4.364	2	0.113	0.302
High Reliability b/w ec records	4.321	2	0.115	0.3
Suspected EMG < 5 uV incl	2.126	2	0.345	0.21

Primary Analyses

The first three hypotheses addressed differences in the raw EEG records of the groups. Hypothesis 1 predicted there would be significantly more total raw EEG records

that included epileptiform events in the CSA group as compared to the NCSA group. The second hypothesis predicted more temporal alpha rhythm pattern in the raw EEG records of subjects in the CSA group compared to those of the NCSA group. And the third hypothesis predicted that compared to the NCSA group EEG records there would be significantly fewer CSA records showing a 25 percent or greater decrease in the amplitude of the alpha rhythm in the occipital or parietal regions in the raw eyes open as compared to eyes closed conditions.

Pearson's chi square tests with Yates' continuity correction were performed on the qualitative ratings of the raw EEG data. Referring to Table 4, it can be seen that there were no epileptiform events in any of the records of either group. Small effect sizes (>.2 for r_{Cohen}) were obtained for the temporal alpha pattern and attenuation of the alpha rhythm upon eye opening. These specific EEG patterns did not reveal differences between the two groups.

Table 4

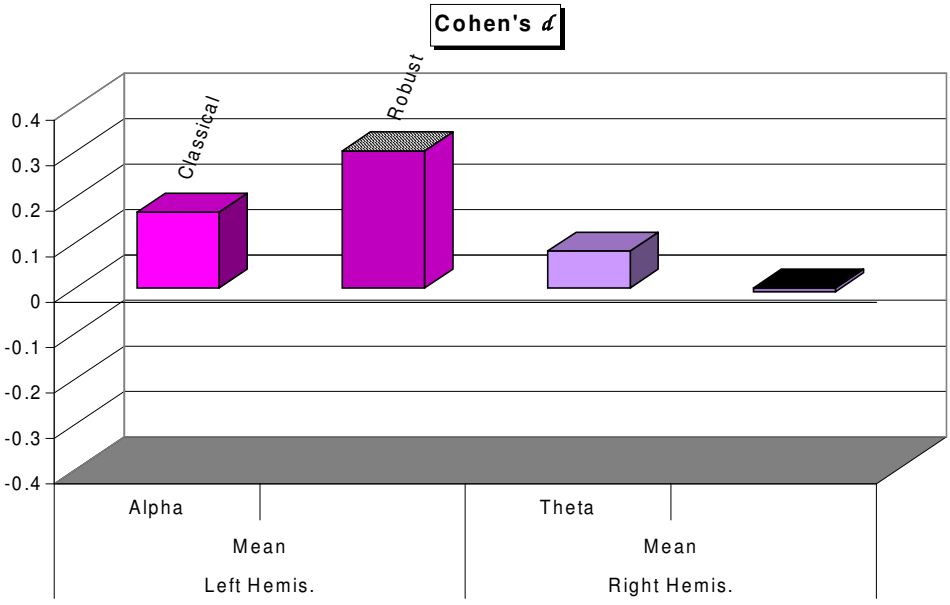
Chi Square Analysis for Qualitative Ratings of Raw EEG Data

Type of Activity	Chi-square	d.f.	p-value	Cohen's r
Epileptiform	0	1	1	0
Temporal Alpha	0.339	1	0.561	0.080
Alpha Attenuation	1.546	2	.462	0.180

The fourth and fifth hypotheses addressed differences in right and left hemisphere coherence as defined by average vector length. Specifically, the fourth hypothesis predicted smaller average vector length distances from the origin in the theta band on the right for the CSA group when compared to the NCSA group. The fifth

hypothesis predicted smaller average vector length distances from the origin in the alpha band on the left for the CSA group when compared to the NCSA group. To test these hypotheses, one-tailed Student's t-tests were performed and failed to reveal significantly greater differences in the CSA group (left hemisphere alpha: M = .53, SD = .09), t (46) = .58, p > .05; (right hemisphere theta: M = .57, SD = .04), t (46) = .28, p > .05. As shown in Figure 2, Cohen's d_{robust}, the measure of robust effect size, revealed small effect sizes in both hemispheres (d_{robust} alpha in left hemisphere = .30, d_{robust} theta in right hemisphere = -.008). The CSA group did not exhibit increased connectivity in the right hemisphere in the theta band or in the left hemisphere in the alpha band when compared to the NCSA group.

Figure 2. Left and right hemisphere effect sizes based on mean distance from the origin for the alpha and theta bands. The plain bars represent classical effect sizes and the hatched bars represent robust effect sizes.



Hypotheses 6, 7, and 8 examined differences in the Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989), records of the groups. Hypothesis 6 predicted there would be significantly more records exhibiting Scale F as 10 points or greater than Scales L and K for the CSA group than for the NCSA group. Hypothesis 7 predicted there would be significantly more records showing the 4-5-6 configuration (defined as Scale 4 and 6 above a T score of 65, and scale 5 below a T score of 35) in the CSA group than in the NCSA group. And, hypothesis 8 predicted there would be significantly more records showing the floating profile pattern (defined as 6 or more Scales elevated above a T score of 65) in the CSA group than in the NCSA group.

To test these hypotheses, chi-square analyses were performed to compare the groups on the MMPI-2 patterns. As shown in Table 5, these analyses revealed moderate effect size for the differences between the two groups for two patterns, Scale F at least 10 points higher than Scales L and K, and the floating profile (6 or more Scale elevations above a T score of 65). The CSA group exhibited more patterns with Scale F 10 points higher than Scales L and K as well as floating profiles. No participants exhibited the 4-5-6 configuration according to its definition of Scales 4 and 6 being 30 points higher than Scale 5.

Table 5

Chi Square Analyses of MMPI-2 Pattern Variables

Pattern	Chi-square	d.f.	p-value	Cohen' s r
F higher than L & K	3.798	1	0.051	0.281
4 & 6 30 pts > 5	0	1	1	0
Floating Profile	3.572	1	0.059	0.273
4 & 6 above 5	9.108	1	0.002	0.436

Lastly, hypothesis 9 predicted that the CSA group would exhibit greater Performance - Verbal IQ differences. A Student's t-test (one-tailed) was performed and failed to reveal significantly greater differences in the CSA group (M = -1.35, SD = 0.47), t (46) = -.978, p > .05. Cohen's d (d_{Cohen} = -.288), the measure of effect size, revealed a small effect size. The CSA group did not reveal larger Performance – Verbal IQ score differences.

Exploratory Analyses

To further explore possible differences between the two groups, the pattern of coherence for each of the bands at the level of the individual electrode was examined. Student's t-tests were used to accept or reject the null hypothesis that the groups mean vector lengths were equal.

Effect sizes in both bands were positive, indicating increased left hemisphere alpha and beta vector length was associated with CSA. Large distances from the origin are indicative of hypocoherence (differentiation).

Figures 3-7 provide summaries of the information found in Tables A1-A5 of Appendix A. Positive effect sizes indicate increased vector length (hypocoherence) for

the CSA group in comparison to the NCSA group. Negative effect sizes indicate decreased vector length (hypercoherence) for the CSA group in comparison to the NCSA group. Beginning with the frontal sites shown on Figure 3 (Table A1), it is apparent that a medium positive effect size is obtained for delta at F8, indicating large distances from the origin (hypocoherence) for the CSA group as compared to the NCSA group. Continuing across the head from front to back, Figures 4 and 5 (Tables A2 and A3) indicate moderate effect sizes in the positive direction (hypocoherence) in the alpha band at F3, F4, T4 and C4. Figure 5, Table A3 shows moderate effect sizes are also obtained for delta in the negative direction (hypercoherence) at C3, C4, and T5.

Figure 3. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together (cross-correlation) for sites F1, F2, F7, and F8. The plain bars represent classical effect sizes and the hatched bars represent robust effect sizes.

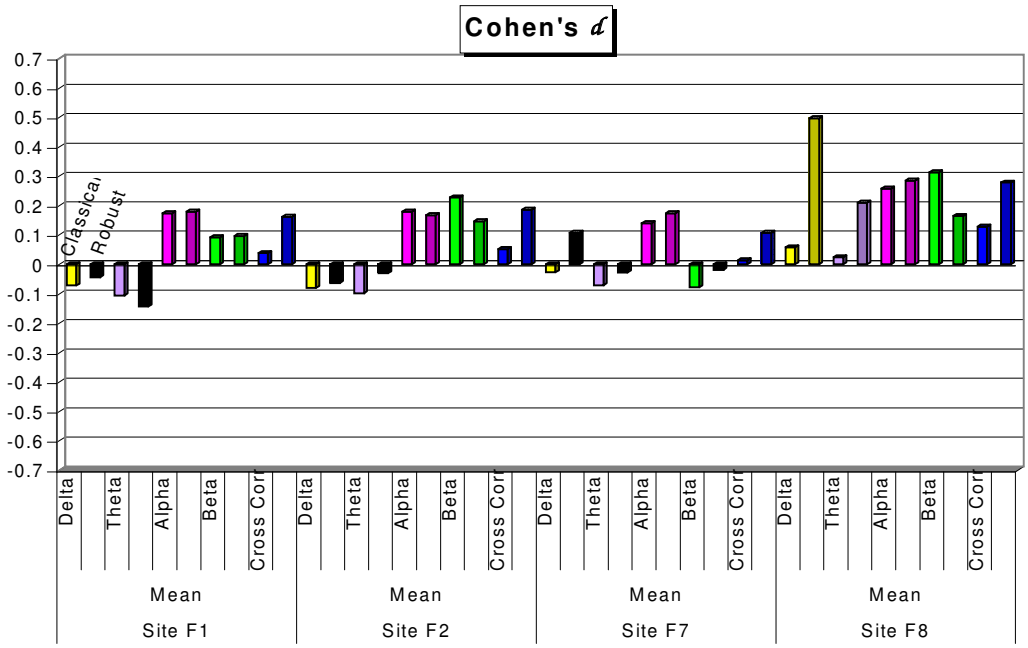


Figure 4. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together (cross-correlation) for sites F3, F4, T3, and T4. The plain bars represent classical effect sizes and the hatched bars represent robust effect sizes.

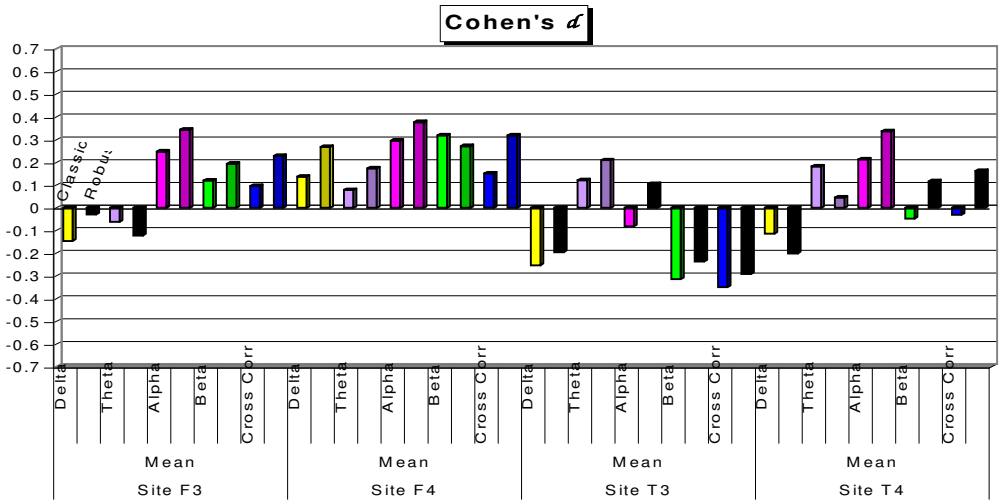
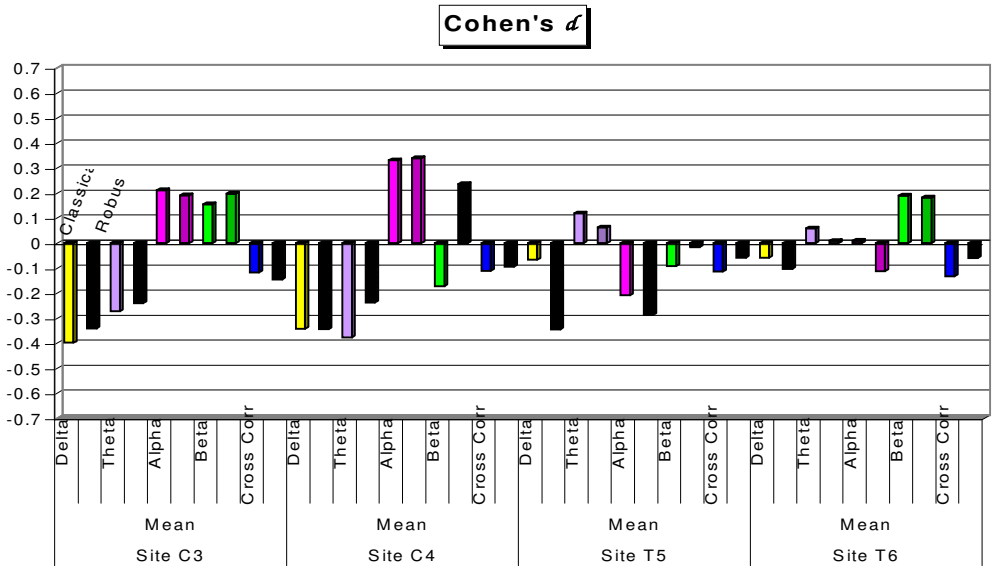


Figure 5. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together (cross-correlation) for sites C3, C4, T5, and T6. The plain bars represent classical effect sizes and the hatched bars represent robust effect sizes.



Continuing toward the posterior sites (Figure 6, Table A4) indicates small effect sizes until approaching posterior vertex sites (Figure 7, Table A5). At PZ, a medium to large effect size in the positive direction for alpha is obtained, indicating large distances from the origin (hypocoherence) for the CSA group as compared to the NCSA group. A medium effect in the positive direction is also seen at PZ for beta and for the cross correlation (all bands collapsed), indicating large distances from the origin (hypocoherence) for the CSA group as compared to the NCSA group.

Figure 6. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together (cross-correlation) for sites P3, P4, O1, and O2. The plain bars represent classical effect sizes and the hatched bars represent robust effect sizes.

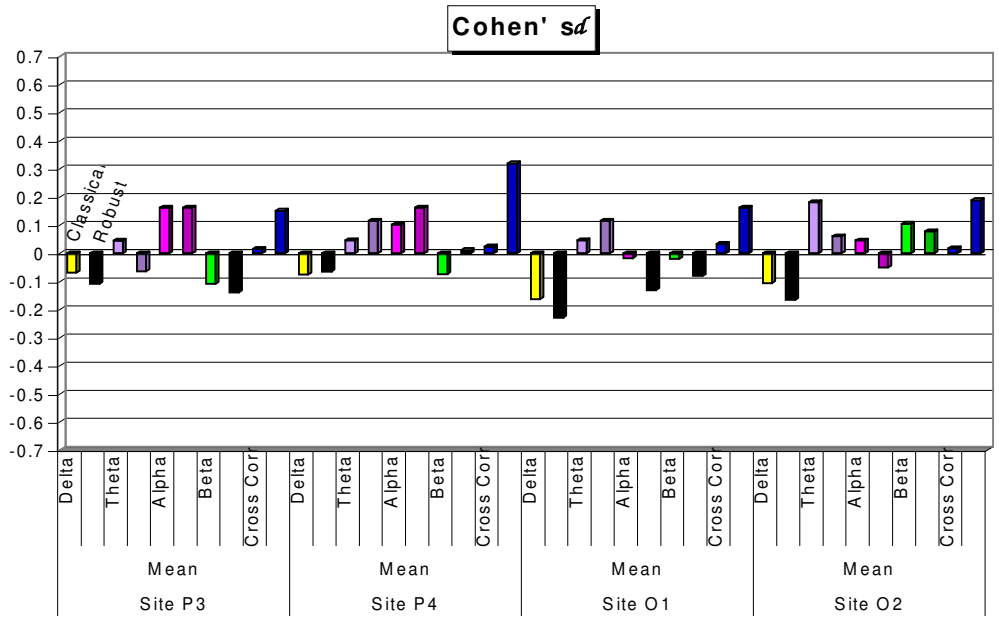
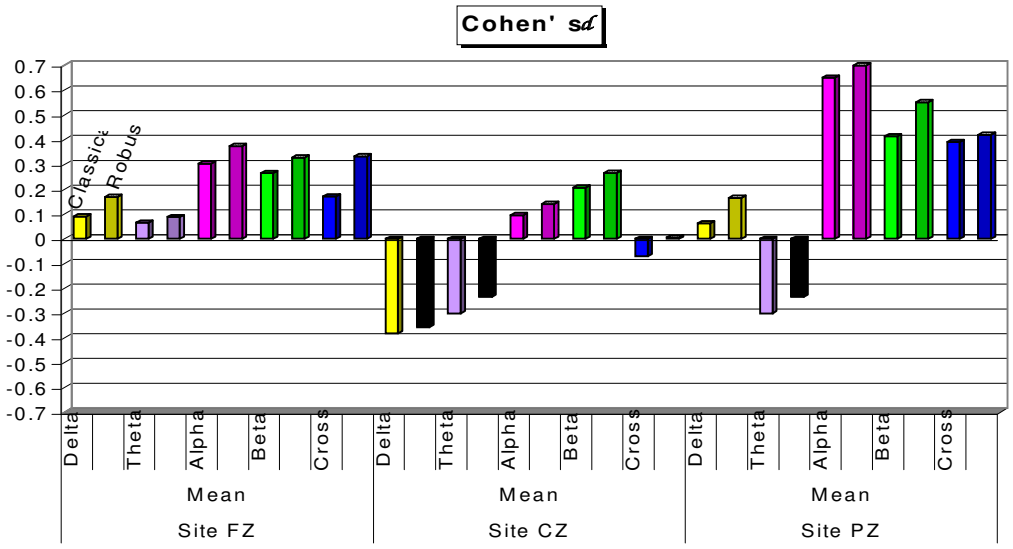
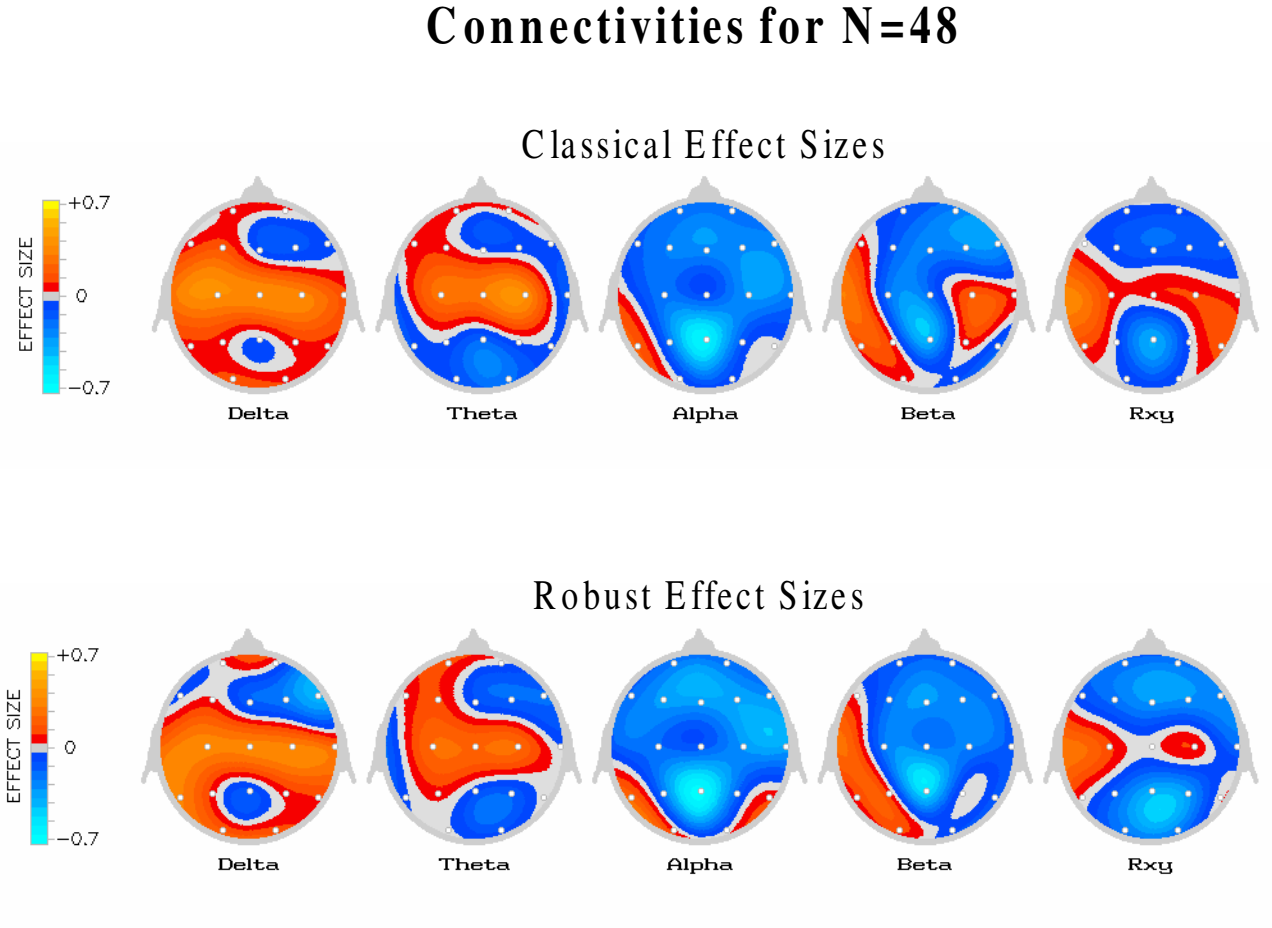


Figure 7. Effect sizes based on mean distance from the origin for each band as well as the bands collapsed together (cross-correlation) for sites FZ, CZ, and PZ. The plain bars represent classical effect sizes and the hatched bars represent robust effect sizes.



The above results are summarized in Figure 8, where yellow and light orange indicate medium to large effect sizes in the negative direction, and light blue indicates medium to large effect sizes in the positive direction. Thus, hypocoherence or decreased connectivity (differentiation) in the CSA group as compared to the NCSA group is apparent on the right frontally in delta, and posteriorly (PZ) in alpha and beta, as well as the cross correlation. Hypercoherence or increased connectivity (redundancy) in the CSA group as compared to the NCSA group is apparent centrally across the motor strip and on the left temporally in delta.

Figure 8. Classical and robust effect size topographic maps; Dependent measure – distance from the origin for NeuroRep (Hudspeth, 1999) neuroelectric images; Based on means and standard deviation.



The interested reader is referred to the statistical tables containing t-statistics, confidence intervals, p-values, power, means, and effect sizes in Appendix A.

To further describe MMPI-2 scale differences between the two groups, Student's t-tests with Benjamini false discovery rate corrections for dependent variables were performed to control family wise error rate. These values along with means, confidence intervals, and effect sizes for MMPI-2 scales are shown in Table 6. Figure 3 provides a graph of the means for the two groups. Large effect sizes were obtained for the

differences between the two groups for Scales F, 1, 2, 3, 4, 6, 7, 8, PK, and PS (Benjamini False Discovery Rate corrected p-values < .05).

Figure 9. Mean MMPI-2 T scores for the CSA group (red) and the NCSA group (blue).

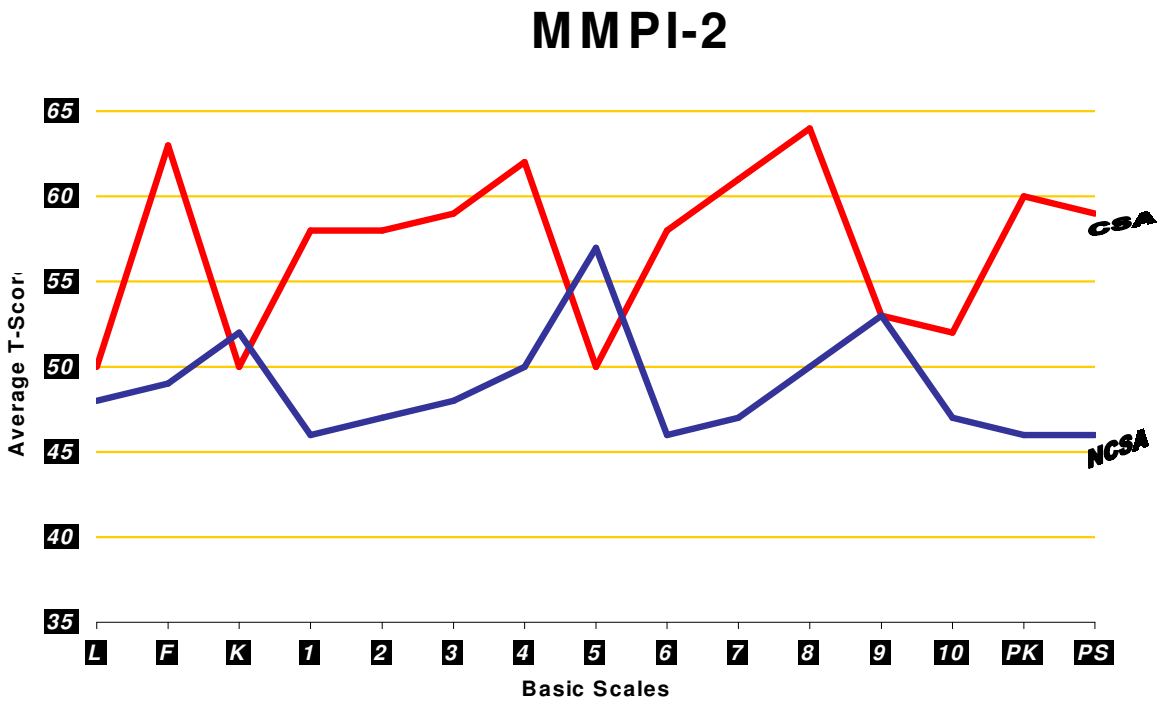


Table 6

Student's T Analyses of MMPI-2 Scales

Scale	t-statistic	d.f.	p-value	FDR	Mean CSA	Mean NCSA	Lower Conf	Upper Conf	Cohen's d
L	-0.809	46	0.422	1	47.5	49.54	-7.12	3.04	-0.239
F	-2.924	46	0.005	0.03	49.21	63.25	-23.71	-4.38	-0.862
K	0.6411	46	0.525	1	52.04	50.12	-4.1	7.93	0.189
1	-4.101	46	0.0001	0	45.88	58.29	-18.42	-6.41	-1.209
2	-3.418	46	0.001	0.01	47.35	58.33	-17.61	-4.56	-1.008
3	-4.322	46	0.0001	0	47.75	59.33	-16.98	-6.19	-1.274
4	-5.05	46	0	0	50.33	62.33	-16.78	7.22	-1.489
5	2.091	46	0.042	0.19	56.63	50.29	0.24	12.43	0.617
6	-4.271	46	0.0001	0	46.17	57.92	-17.29	-6.21	-1.259
7	-4.728	46	0	0	46.79	61.42	-20.85	-8.4	-1.394
8	-3.914	46	0.0003	0	50.04	64.25	-21.52	-6.9	-1.154
9	0	46	1	1	52.79	52.79	-6.76	6.76	0
0	-1.894	46	0.065	0.27	46.75	51.75	-10.32	0.32	-0.558
PK	-3.894	46	0.0003	0	46.38	59.92	-20.54	-6.54	-1.148
PS	-3.719	46	0.0005	0	45.83	59.29	-20.74	-6.17	-1.097

Post Hoc Analyses

Due to the high functioning nature of the sample, very few MMPI-2 records contained elevations beyond 65 T score points. Yet, a 4-5-6 pattern (Scales 4 and 6 higher than Scale 5) as seen in Figure 9, was evident in the graphed MMPI-2 mean

scores. A movement within personality assessment has suggested there is much to be gained by understanding patterns evidenced in MMPI-2 profiles which do not show elevations beyond 65 T score points (Caldwell, 2003; Finn, 1995; Lewak, Marks, & Nelson, 1990; Nichols, 2001). Therefore, Pearson's chi square analysis was performed to assess for differences in the presence or absence of this 4-5-6 pattern between the groups. Referring to Table 5, it can be seen that the CSA group exhibited significantly more of these patterns than did the NCSA group, evidencing a large effect size.

CHAPTER 7

DISCUSSION

Comparison of Findings to Earlier Research

EEG data was used to examine brainwave patterns associated with childhood sexual abuse (CSA). Information related to abnormalities in the raw EEG were used to extend and replicate prior research by Black et al. (2002) which found increased EEG abnormalities (e.g., epileptiform events, low voltage fast patterns, and temporal alpha rhythm) were associated with childhood sexual abuse (CSA) adults, and by Ito et al. (1993) which found increased EEG abnormalities (e.g., paroxysmal events, asymmetries, and/or regions of focal slowing) in children who had experienced sexual abuse. In this research, non-clinical, unmedicated CSA and NCSA groups were compared qualitatively for prevalence of epileptiform events, temporal alpha rhythm, and alpha attenuation upon eye opening in the raw EEG. Qualitative analysis of the EEG provided important amplitude versus time relationships, which are different than the relationships illustrated after quantification (amplitude or phase versus frequency relationships). However, in contrast to the above noted studies, small effect sizes resulted, and CSA was not found to differentiate the 2 groups on raw EEG measures. This finding is not surprising when the high functioning level of the mostly college student sample is considered. It would be expected that both groups in this study would exhibit fewer abnormalities, decreasing chances of finding differences. Comparisons in the Black et al. (2002) study were made between clinical groups and comparisons in the Ito et al. (1993) study were made with clinical as compared to normal groups.

While earlier research has uncovered evidence of damage to subcortical structures of the brain affecting cortical function (Bremner et al., 1997; Teicher et al., 1993), none has had available Hudspeth's neuroelectric image methodology to closely identify cortical-cortical intercommunication dysfunction. Neuroelectric image data based on EEG coherence information was used to extend to non-clinical, unmedicated adults prior research by Black et al. (2002) with clinical samples suggesting CSA impacts cortical function resulting in lateralized differences (left hemisphere alpha hypercoherence and right hemisphere theta hypercoherence). It was also used to extend to adults those of Ito, Teicher, Glod, and Ackerman (1998) who similarly found greater average left hemisphere alpha coherence in abused children compared to normal children. In contrast to these study findings, small effect sizes were obtained for high functioning, unmedicated CSA adults. This sample was not found to exhibit lateralized differences in either alpha or theta. This may relate to important differences in hemispheric balance in non-clinical verses clinical samples. The balance exhibited between Performance and Verbal IQ may be the outgrowth of this more balanced interhemispheric communication which may then facilitate resilience in response to trauma.

Personality information obtained from the Minnesota Multiphasic Personality Inventory – 2™ (MMPI-2™), a psychological instrument owned by The Regents of The University of Minnesota, Minneapolis, MN (Hathaway & McKinley and the MMPI™ Restandardization Committee of The University of Minnesota Press: Butcher, Dahlstrom, Graham & Tellegen, 1989), indicated important differences between the groups, though neither group exhibited means in excess of 65 T score points. This

finding of low pathology in CSA college samples is consistent with the findings of Rind et al.'s (1998) metaanalysis surveying college samples. In contrast to Rind et al.'s findings that poorer adjustment in CSA groups did not account for a large portion of the variance, our findings indicated the opposite. Up to 36 percent of the variance in this study's adjustment measure (MMPI-2) could be attributed to CSA group membership. Stepping beyond MMPI-2 self-report, up to 11 percent of the variability in findings of decreased and increased connectivity seen in this study could be accounted for by CSA group membership. Our findings seem to give strong support to the conclusion that important differences in both psychological and neurological function are evident in CSA adults when compared to NCSA adults.

Though both group MMPI-2 T score means were under 65, Caldwell (2003) and others (Lewak, 2003; Nichols, 2001) have argued that scores below the 65 T score cut-off are interpretable, revealing signs and symptoms of particular problems. Caldwell (2003) has stated that the height of the MMPI-2 scale elevation can be understood in terms of breadth and width of the endorsed problem. He went further to emphasize that personality styles are often revealed within the normal range (50 to 65 T score points) and can provide valuable and accurate information about individuals, especially when overall patterns are assessed. However, interpretation of these normal range profiles requires a move away from a pathological perspective. The scale interpretations provided are tempered in terms of normal range functioning (below 65 T score points) as explicated by Lewak (2003).

Important features seen in the CSA sample which exhibited large effect sizes in comparison to the NCSA sample can be interpreted in terms of normal range

functioning. According to Lewak (2003) these differences in comparison to the NCSA group relate clinically to feelings of pain, fear, and anxiety which may be overstated (Scale F $\geq$ 10 points higher than Scales L and K), anxiety and fear related to health (Scale 1), feelings of being inefficient and less happy than desired (Scale 2), conflict avoidance to avoid emotional pain (Scale 3), fear of emotional involvement or letting guard down (Scale 4), sensitivity to criticism (Scale 6), feelings of being on edge, worried, and tense that something bad is about to happen (Scale 7), feelings of being unlovable/inefficient and vulnerable to other's hostility (Scale 8), symptoms of general maladjustment and emotional distress (Scales PK and PS), and a tendency to manage others or situations out of fear of mistreatment (Scale 4 and 6 higher than Scale 5). Perhaps these MMPI-2 findings make sense when a history of CSA is considered.

Although right versus left hemisphere differences were hypothesized in accordance with the Ito et al. (1993) findings of better Performance than Verbal IQ, they were not exhibited in our results. In fact, the means for Verbal IQ and Performance IQ were identical in the CSA group (see Table 1). These findings in combination with our failure to replicate previous findings of left hemisphere alpha hypercoherence (Black et al., 2002; Ito et al., 1993) and right hemisphere theta hypercoherence (Black et al., 2002) might be interpreted as increased hemispheric balance in high functioning samples. Perhaps increased hemispheric balance and high IQ interact to produce the means for resilience when confronted with trauma.

Important in this research were the findings related to pattern and degree of functional differences from the NCSA group across all bands. Despite statistical error ramifications for making more comparisons rather than averaging cortical regions, the

researcher determined that it was of greater clinical import to gain information related to integration across brain regions since high functioning samples have not before been considered in the CSA EEG literature. Priority was therefore given to examining whether important differences were missed by limiting the evaluation to only one band per hemisphere. This required lenience with regard to Type I – Type II error balance. With this in mind, a fuller evaluation made it possible to see decreased connectivity on the right frontally (F8) in the delta band, and frontally (F3,FZ, F4) and centro-temporally on the right in the alpha band (T4, C4),and posteriorly (PZ) in the alpha and beta bands, as well as in the cross-correlation. Increased connectivity was also evidenced centrally across the motor strip (C3, C4, and CZ) and on the left temporally (T5) in the delta band.

These findings are in contrast to prior findings by Black et al. (2002) of decreased connectivity in left frontal regions in the theta and beta bands, and increased connectivity in posterior central regions across all bands in the CSA group as compared to the NCSA group. Thus, a very different picture arises for our non-clinical groups than was obtained for our clinical groups. It may be the obtained differences in results relate to medication effects and lower functionality in the Black et al. (2002) findings. Perhaps these findings were derived by chance because of multiple comparisons. Or, it is possible that the current findings describe the functional brain characteristics of resilience in the face of CSA.

Theoretical and Clinical Implications

It is difficult to form conclusions about the clinical significance of the findings from this study because of the limited knowledge neuropsychological science has about

cortical structures and their interactions. However, an attempt will be made to tie these findings to a theoretical model and its therapeutic implications.

An interesting clinical application of the connectivity findings from this study can be made by using Walker's (2003) theoretical model of modular coherence. According to this model, classical localization of cortical function (Penfield & Jasper, 1954) combines with coherence to predict functional interaction between functional cortical modules. For example, cooperation between brain regions is required to perform complex functions such as seeing an image while also attending to it. This requires that left prefrontal modules interact with occipital visual modules. Temporal memory modules must then cooperate with occipital visual modules in order to retain the image so that it can be remembered. When connectivity between these modules is impaired, visual learning impairments result.

Applying Walker's modular coherence model to the CSA group's decreased connectivity results produces an emerging theoretical picture of neurological functional inefficiency for adults with CSA. According to this model, decreased connectivity at F8 is associated with less efficient emotional expression. As this F8 cortical module affects other cortical modules, affective inefficiency is exhibited in interaction with, for example, emotional memory (T4) resulting in memory processing and storage inefficiency in emotionally evocative situations. As less efficient emotional expression interacts with modules important in motor preparation (F3, FZ, F4), inefficient responding to emotionally evocative situations results. As inefficient emotional expression interacts with areas important in sensorimotor interpretation of the left upper extremity (C4), misinterpretations of sensations during emotionally charged situations results. And

lastly, as inefficient emotional expression interacts with regions important in perception, spatial relations, midline vision, touch, proprioception, and left extremities (PZ), misperceptions and misunderstandings of space and sensation result.

As Walker's modular coherence model is applied to the CSA group's increased connectivity results, a picture of inflexibility is evidenced. The model predicts that increased connectivity on the left temporally (T5) results in rigid logical understanding, auditory processing, and word recognition. As this rigidity combines with motor strip regions (C3, C4, CZ) important in sensorimotor integration, ability for spontaneous physical and sensory processing, understanding, and responding is decreased. Disturbances of perceptual processing are seen with temporal damage (Milner, 1958). Speculation leads to questions about the lasting impact of CSA on stress coping systems and the possible adaptive aspects of misperceiving on coping with CSA. Could it be CSA reorganizes the way in which brain regions work together to process information so that the individual is preserved emotionally when reminders of the abuse intrude into daily living?

Cortical connectivity and MMPI-2 findings of the present study combined to provide a possible explanation of the effects of CSA that have been described to date in the literature (Allen, 2001; Levine, 1997; Ross, 2000; Scaer, 2001) and to provide some confirmation for Walker's coherence module model. For example, it is known that deeper structures of the temporal lobes add affective components to sensory and kinesthetic information coming from central regions. These regions (e.g.,T4) are also important in sensory and perceptual integration (Kolb & Whishaw, 1996). One might hypothesize that at the time of abuse, the child was unable to garner reasoning and

expressive ability from the frontal lobes (e.g., F8) that were still at a lower stage of brain development than formal operations (Ginsburg & Opper, 1988). As a result, decreased connectivity between sites may have resulted when brain regions responsible for emotional integration with reasoning, expression, perceptual and memory processing (right frontal, temporal and parietal) were presented with incongruent emotional information (the one who loves and protects me also hurts me). The behavioral manifestation of this decreased connectivity may be related to the highest mean MMPI-2 Scale elevation for the CSA group, Scale 8. It could be hypothesized that feelings of being unlovable/inefficient and vulnerable to other's hostility is a pattern of misperception that continues as a result of this decreased connectivity in these regions that was originally meant to preserve the child during the traumatic event.

To summarize, both emotional inefficiency and lack of spontaneity would be predicted from Walker's model in the CSA group as compared to the NCSA group. Specifically, functional inefficiency when emotional expression interacts with other modules important in memory processing, perception, interpretation, and responding (right hemisphere) combined with functional rigidity and decreased spontaneity when temporal modules important in processing and understanding interact with central modules important in physical and sensory responding (left hemisphere).

Clinical implications are warranted for findings of anterior, temporal, and posterior hypocoherence and central and left temporal hypercoherence that may indicate these regions are not working together efficiently or effectively for integrated processing in CSA groups. Cognitive-behavioral approaches would likely be helpful in attempting to bring frontal lobe reasoning capacity to bear in the brain's emotional regions as well as

improving processing of available response options in sensorimotor regions for enhanced coping. Psychodynamic approaches which confront transference phenomena (misperceptions) would likely serve to concomitantly address both frontal and temporal abnormalities providing for integration of thinking and feeling within relationships where misperceiving occurs. EEG biofeedback protocols that target coherence imbalances may serve to improve perceptual and auditory processing and reduce misperceptive behaviors related to self and others (Walker, 2003), as well as enhance effectiveness of previously mentioned psychotherapeutic intervention.

Statistical Limitations

A concern in this data analysis is that of p-value significance. In reaction to much debate over the past decade about over reliance on statistical significance (Borenstein, 1997; Kirk, 1996) as well as recommendations issued by the American Psychological Association Task Force on Statistical Inference (see Thompson, 2002 for review), statistical significance was not relied on as the determining factor for the interpretations made in this study. Instead, effect sizes were used to characterize the degree to which the CSA group diverged from the NCSA group in order to highlight clinical significance. Since effect sizes have not been reported in prior coherence research with CSA, it was not known how best to interpret these effects; so Cohen's benchmarks for effect size interpretation were used (e.g. for d_{Cohen} .4 - .6 = a medium effect size, for r_{Cohen} .196 - .287 = medium effect size). It is hoped the effect sizes reported here will serve as a starting point for future research to provide meaningful understandings of these effect sizes as they relate to CSA.

Beyond effect sizes, power was taken into account in interpretation of results. A perusal of the power column located in the tables in Appendix A show that low power served to prevent statistically significant p-values despite moderate effect sizes. But this was a time intensive study (6 to 8 hours of testing with each participant) with a small number of participants due to rigid exclusion criteria. Though power was compromised due to low N, the value of gaining a purer look at physiological and psychological underpinnings of CSA without the mask of medications was gained. The rigid exclusion criteria used may have biased the sample toward CSA participants who have developed resilience. A methodological study by Clark-Carter (1997) found researchers typically do not consider power in their analyses and run a high risk of Type II error, resulting in decreased recognition of interesting effects. However, the compatibility of the coherence and personality results from this study with Walker's (2003) theoretical model of modular coherence may provide a greater measure of credibility and clinical significance to the findings.

Relatedly, given the low power evident partly as a result of the small sample size, it did not make sense to adjust for multiple contrasts. Therefore, effect sizes are considered a summarization of the data providing information about the degree to which CSA impacted the variable.

Overall, this study presents some interesting results which are in many ways undermined by a small N. Results may also be masked by heterogeneity within the two samples. The CSA group was not differentiated according to severity of CSA, though presence of current PTSD diagnosis was considered. Replication with larger samples

that allow for group divisions according to severity level will be required for confidence in interpretation.

Additionally, a model has been presented for working with coherence data in a statistically modern and parsimonious way. As this study is one of the first attempts to examine the impact of CSA on cortical integration in high functioning adults, it provides a starting point for future research, suggesting important differences exist. A representative sample of the statistical code required to run these coherence analyses has been provided in Appendix B for the purpose of future replication.

APPENDIX A

STATISTICAL TABLES FROM NEI ANALYSES

Table A1

Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep NEI's for Sites F1, F2, F7, and F8

LOCATION	CONTRAST	BAND	TYPE	t-statistic	LowerConf	UpperConf	p-value	Power	Mean CSA	Mean NCSA	Cohen's d
Site F1	Mean	Delta	**Classical**	-0.249	-0.084	0.065	0.804	0.057	0.65	0.659	-0.072
			Robust		-0.075	0.073	0.87	0.038	0.655	0.661	-0.044
		Theta	**Classical**	-0.364	-0.09	0.063	0.728	0.065	0.519	0.533	-0.106
			Robust		-0.085	0.053	0.572	0.104	0.531	0.548	-0.143
		Alpha	**Classical**	0.596	-0.078	0.144	0.554	0.092	0.405	0.372	0.174
			Robust		-0.088	0.149	0.566	0.082	0.399	0.364	0.179
		Beta	**Classical**	0.318	-0.067	0.092	0.752	0.062	0.414	0.402	0.092
			Robust		-0.076	0.108	0.728	0.088	0.411	0.398	0.097
		Cross Corr	**Classical**	0.132	-0.071	0.081	0.896	0.052	0.538	0.533	0.038
			Robust		-0.058	0.092	0.63	0.088	0.538	0.517	0.162
Site F2	Mean	Delta	**Classical**	-0.278	-0.084	0.063	0.782	0.059	0.657	0.668	-0.08
			Robust		-0.081	0.072	0.868	0.088	0.659	0.667	-0.064
		Theta	**Classical**	-0.334	-0.085	0.061	0.74	0.063	0.524	0.536	-0.099
			Robust		-0.074	0.065	0.928	0.054	0.539	0.543	-0.03
		Alpha	**Classical**	0.616	-0.073	0.138	0.541	0.095	0.41	0.378	0.18
			Robust		-0.09	0.143	0.566	0.122	0.406	0.375	0.168
		Beta	**Classical**	0.786	-0.05	0.113	0.436	0.123	0.434	0.402	0.227
			Robust		-0.057	0.107	0.622	0.082	0.421	0.4	0.146
		Cross Corr	**Classical**	0.181	-0.067	0.08	0.857	0.054	0.54	0.533	0.052
			Robust		-0.045	0.089	0.536	0.086	0.542	0.519	0.186
Site F7	Mean	Delta	**Classical**	-0.093	-0.076	0.069	0.927	0.051	0.683	0.687	-0.027
			Robust		-0.068	0.07	0.71	0.03	0.706	0.693	0.108
		Theta	**Classical**	-0.241	-0.087	0.068	0.81	0.057	0.558	0.567	-0.071
			Robust		-0.097	0.078	0.928	0.046	0.573	0.576	-0.028
		Alpha	**Classical**	0.48	-0.087	0.141	0.633	0.077	0.412	0.385	0.14
			Robust		-0.081	0.145	0.592	0.092	0.412	0.378	0.173
		Beta	**Classical**	-0.268	-0.084	0.064	0.79	0.583	0.42	0.43	-0.078
			Robust		-0.075	0.078	0.956	0.05	0.427	0.43	-0.019
		Cross Corr	**Classical**	0.049	-0.066	0.069	0.961	0.05	0.56	0.558	0.014
			Robust		-0.056	0.073	0.688	0.054	0.563	0.551	0.108
Site F8	Mean	Delta	**Classical**	0.2	-0.067	0.081	0.842	0.055	0.71	0.702	0.058
			Robust		-0.05	0.112	0.098	0.234	0.753	0.697	0.497
		Theta	**Classical**	0.082	-0.07	0.076	0.935	0.051	0.565	0.562	0.024
			Robust		-0.053	0.099	0.472	0.182	0.585	0.56	0.21
		Alpha	**Classical**	0.892	-0.053	0.137	0.377	0.145	0.443	0.401	0.258
			Robust		-0.066	0.172	0.398	0.168	0.447	0.4	0.285
		Beta	**Classical**	1.083	-0.034	0.114	0.284	0.192	0.467	0.427	0.313
			Robust		-0.043	0.096	0.59	0.112	0.462	0.442	0.164
		Cross Corr	**Classical**	0.445	-0.052	0.082	0.659	0.073	0.566	0.551	0.129
			Robust		-0.037	0.09	0.316	0.202	0.578	0.546	0.278

Table A2

Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep NEI's for Sites F3, F4, T3, and T4

LOCATION	CONTRAST	BAND	TYPE	t-statistic	LowerConf	UpperConf	p-value	Power	Mean CSA	Mean NCSA	Cohen's d
Site F3	Mean	Delta	**Classical**	-0.501	-0.063	0.038	0.619	0.079	0.496	0.508	-0.145
			Robust		-0.053	0.045	0.904	0.08	0.503	0.505	-0.029
		Theta	**Classical**	-0.214	-0.059	0.047	0.831	0.055	0.447	0.453	-0.062
			Robust		-0.057	0.042	0.644	0.06	0.448	0.459	-0.122
		Alpha	**Classical**	0.857	-0.058	0.143	0.396	0.137	0.381	0.339	0.248
			Robust		-0.049	0.154	0.288	0.21	0.386	0.328	0.345
		Beta	**Classical**	0.416	-0.048	0.073	0.679	0.07	0.41	0.398	0.12
			Robust		-0.042	0.077	0.518	0.092	0.415	0.395	0.195
		Cross Corr	**Classical**	0.334	-0.047	0.066	0.74	0.063	0.448	0.439	0.096
			Robust		-0.032	0.075	0.43	0.134	0.431	0.452	0.229
Site F4	Mean	Delta	**Classical**	0.474	-0.039	0.063	0.638	0.076	0.521	0.509	0.138
			Robust		-0.028	0.074	0.296	0.158	0.528	0.505	0.269
		Theta	**Classical**	0.267	-0.045	0.059	0.79	0.058	0.449	0.442	0.079
			Robust		-0.042	0.064	0.566	0.062	0.46	0.445	0.174
		Alpha	**Classical**	1.025	-0.044	0.136	0.311	0.176	0.387	0.341	0.296
			Robust		-0.041	0.15	0.22	0.234	0.391	0.332	0.378
		Beta	**Classical**	1.109	-0.028	0.095	0.273	0.198	0.431	0.397	0.32
			Robust		-0.031	0.09	0.386	0.146	0.433	0.434	0.272
		Cross Corr	**Classical**	0.522	-0.04	0.068	0.604	0.082	0.452	0.438	0.151
			Robust		-0.033	0.08	0.308	0.22	0.458	0.429	0.32
Site T3	Mean	Delta	**Classical**	-0.868	-0.072	0.029	0.39	0.14	0.574	0.596	-0.253
			Robust		-0.065	0.041	0.56	0.102	0.571	0.587	-0.194
		Theta	**Classical**	0.423	-0.032	0.049	0.674	0.071	0.616	0.608	0.122
			Robust		-0.027	0.045	0.446	0.114	0.626	0.612	0.21
		Alpha	**Classical**	-0.278	-0.102	0.077	0.782	0.059	0.559	0.571	-0.081
			Robust		-0.096	0.099	0.734	0.086	0.577	0.562	0.105
		Beta	**Classical**	-1.08	-0.061	0.018	0.286	0.191	0.616	0.638	-0.313
			Robust		-0.065	0.028	0.426	0.136	0.616	0.632	-0.236
		Cross Corr	**Classical**	-1.207	-0.083	0.021	0.234	0.227	0.611	0.642	-0.349
			Robust		-0.073	0.022	0.34	0.16	0.618	0.642	-0.291
Site T4	Mean	Delta	**Classical**	-0.395	0.064	0.043	0.695	0.068	0.607	0.617	-0.114
			Robust		-0.066	0.04	0.444	0.134	0.608	0.626	-0.201
		Theta	**Classical**	0.635	-0.028	0.054	0.528	0.097	0.625	0.612	0.183
			Robust		-0.039	0.045	0.91	0.056	0.629	0.626	0.045
		Alpha	**Classical**	0.727	-0.059	0.125	0.471	0.112	0.614	0.581	0.214
			Robust		-0.051	0.135	0.246	0.248	0.613	0.563	0.337
		Beta	**Classical**	-0.16	-0.055	0.048	0.873	0.053	0.653	0.657	-0.047
			Robust		-0.039	0.049	0.668	0.05	0.656	0.646	0.117
		Cross Corr	**Classical**	-0.1	-0.054	0.049	0.921	0.051	0.641	0.643	-0.03
			Robust		-0.029	0.047	0.576	0.064	0.629	0.64	0.163

Table A3

Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep NEI's for Sites C3, C4, T5, and T6

LOCATION	CONTRAST	BAND	TYPE	t-statistic	LowerConf	UpperConf	p-value	Power	Mean CSA	Mean NCSA	Cohen's d
Site C3	Mean	Delta	**Classical**	-1.372	-0.051	0.01	0.177	0.279	0.332	0.353	-0.398
			Robust		-0.05	0.011	0.274	0.194	0.332	0.349	-0.34
		Theta	**Classical**	-0.938	-0.044	0.016	0.353	0.155	0.364	0.378	-0.272
			Robust		-0.044	0.017	0.39	0.102	0.364	0.376	-0.24
		Alpha	**Classical**	0.73	-0.04	0.085	0.469	0.113	0.346	0.323	0.212
			Robust		-0.046	0.085	0.522	0.112	0.345	0.324	0.192
		Beta	**Classical**	0.531	-0.025	0.043	0.598	0.083	0.425	0.416	0.155
			Robust		-0.023	0.04	0.462	0.072	0.427	0.416	0.198
		Cross Corr	**Classical**	-0.404	-0.05	0.033	0.688	0.069	0.41	0.419	-0.117
			Robust		-0.05	0.033	0.66	0.082	0.409	0.419	-0.145
Site C4	Mean	Delta	**Classical**	-1.182	-0.043	0.011	0.243	0.219	0.346	0.362	-0.342
			Robust		-0.049	0.014	0.294	0.148	0.346	0.362	-0.344
		Theta	**Classical**	-1.302	-0.051	0.011	0.199	0.256	0.367	0.387	-0.376
			Robust		-0.04	0.014	0.422	0.116	0.375	0.386	-0.236
		Alpha	**Classical**	1.14	-0.03	0.189	0.26	0.207	0.36	0.321	0.332
			Robust		-0.03	0.114	0.25	0.234	0.359	0.318	0.34
		Beta	**Classical**	-0.593	-0.052	0.028	0.556	0.091	0.434	0.446	-0.172
			Robust		-0.03	0.038	0.432	0.15	0.44	0.428	0.236
		Cross Corr	**Classical**	-0.376	-0.046	0.032	0.709	0.066	0.424	0.431	-0.11
			Robust		-0.04	0.026	0.742	0.086	0.42	0.426	-0.094
Site T5	Mean	Delta	**Classical**	-0.228	-0.082	0.065	0.82	0.056	0.554	0.562	-0.066
			Robust		-0.107	0.068	0.248	0.174	0.514	0.552	-0.345
		Theta	**Classical**	0.413	-0.062	0.094	0.682	0.07	0.722	0.706	0.12
			Robust		-0.083	0.101	0.832	0.102	0.708	0.7	0.063
		Alpha	**Classical**	-0.695	-0.132	0.064	0.49	0.107	0.717	0.75	-0.208
			Robust		-0.143	0.069	0.358	0.14	0.717	0.764	-0.286
		Beta	**Classical**	-0.316	-0.071	0.052	0.753	0.062	0.797	0.806	-0.091
			Robust		-0.066	0.063	0.948	0.042	0.799	0.801	-0.016
		Cross Corr	**Classical**	-0.393	-0.135	0.091	0.696	0.068	0.849	0.871	-0.113
			Robust		-0.136	0.095	0.852	0.024	0.831	0.841	-0.058
Site T6	Mean	Delta	**Classical**	-0.202	-0.097	0.079	0.841	0.055	0.573	0.582	-0.058
			Robust		-0.104	0.069	0.734	0.078	0.549	0.563	-0.103
		Theta	**Classical**	0.202	-0.072	0.087	0.841	0.055	0.719	0.711	0.059
			Robust		-0.079	0.083	0.976	0.072	0.708	0.707	0.009
		Alpha	**Classical**	0.034	-0.093	0.096	0.972	0.05	0.751	0.749	0.01
			Robust		-0.125	0.078	0.714	0.09	0.745	0.763	-0.112
		Beta	**Classical**	0.653	-0.04	0.078	0.517	0.1	0.81	0.791	0.19
			Robust		-0.046	0.079	0.528	0.116	0.81	0.792	0.183
		Cross Corr	**Classical**	-0.456	-0.144	0.091	0.65	0.074	0.881	0.908	-0.132
			Robust		-0.126	0.098	0.844	0.05	0.863	0.874	-0.059

Table A4

Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep NEI's for Sites P3, P4, O1, and O2

LOCATION	CONTRAST	BAND	TYPE	t-statistic	LowerConf	UpperConf	p-value	Power	Mean CSA	Mean NCSA	Cohen's d
Site P3	Mean	Delta	**Classical**	-0.237	-0.066	0.052	0.814	0.056	0.451	0.458	-0.069
			Robust		-0.079	0.041	0.734	0.076	0.445	0.456	-0.109
		Theta	**Classical**	0.154	-0.067	0.078	0.879	0.053	0.576	0.57	0.044
			Robust		-0.079	0.07	0.828	0.062	0.564	0.572	-0.064
		Alpha	**Classical**	0.558	-0.077	0.136	0.58	0.086	0.678	0.648	0.161
			Robust		-0.088	0.156	0.626	0.136	0.675	0.645	0.161
		Beta	**Classical**	-0.379	-0.09	0.062	0.707	0.067	0.716	0.73	-0.109
			Robust		-0.098	0.072	0.674	0.068	0.709	0.728	-0.139
		Cross Corr	**Classical**	0.051	-0.091	0.096	0.96	0.05	0.671	0.669	0.015
			Robust		-0.087	0.107	0.61	0.094	0.661	0.639	0.151
Site P4	Mean	Delta	**Classical**	-0.263	-0.079	0.061	0.794	0.058	0.469	0.478	-0.076
			Robust		-0.086	0.059	0.84	0.062	0.457	0.464	-0.065
		Theta	**Classical**	0.158	-0.07	0.081	0.875	0.053	0.589	0.583	0.046
			Robust		-0.085	0.093	0.728	0.076	0.584	0.569	0.115
		Alpha	**Classical**	0.352	-0.081	0.115	0.727	0.064	0.706	0.689	0.102
			Robust		-0.085	0.172	0.648	0.134	0.706	0.679	0.161
		Beta	**Classical**	-0.257	-0.093	0.072	0.798	0.058	0.742	0.753	-0.074
			Robust		-0.078	0.085	0.966	0.05	0.739	0.737	0.011
		Cross Corr	**Classical**	0.078	-0.096	0.104	0.938	0.051	0.698	0.694	0.023
			Robust		-0.046	0.122	0.37	0.066	0.695	0.653	0.319
Site O1	Mean	Delta	**Classical**	-0.565	-0.103	0.058	0.575	0.087	0.568	0.59	-0.163
			Robust		-0.113	0.058	0.438	0.184	0.552	0.583	-0.229
		Theta	**Classical**	0.158	-0.07	0.081	0.875	0.053	0.589	0.583	0.046
			Robust		-0.085	0.093	0.728	0.076	0.584	0.569	0.115
		Alpha	**Classical**	-0.059	-0.111	0.105	0.953	0.05	0.756	0.759	-0.017
			Robust		-0.138	0.088	0.696	0.054	0.763	0.785	-0.131
		Beta	**Classical**	-0.069	-0.073	0.078	0.945	0.05	0.823	0.826	-0.02
			Robust		-0.084	0.059	0.758	0.058	0.821	0.831	-0.081
		Cross Corr	**Classical**	0.111	-0.113	0.126	0.912	0.051	0.882	0.876	0.032
			Robust		-0.102	0.149	0.628	0.066	0.864	0.835	0.162
Site O2	Mean	Delta	**Classical**	-0.366	-0.102	0.071	0.716	0.065	0.58	0.596	-0.106
			Robust		-0.106	0.067	0.584	0.066	0.56	0.584	-0.166
		Theta	**Classical**	0.622	-0.053	0.1	0.537	0.095	0.742	0.718	0.181
			Robust		-0.07	0.093	0.824	0.086	0.731	0.723	0.06
		Alpha	**Classical**	0.156	-0.098	0.115	0.876	0.053	0.779	0.771	0.045
			Robust		-0.133	0.129	0.848	0.078	0.778	0.787	-0.051
		Beta	**Classical**	0.359	-0.058	0.083	0.721	0.065	0.833	0.82	0.104
			Robust		-0.084	0.09	0.826	0.078	0.833	0.823	0.078
		Cross Corr	**Classical**	0.058	-0.116	0.123	0.954	0.05	0.894	0.89	0.017
			Robust		-0.092	0.149	0.61	0.052	0.88	0.848	0.189

Table A5

Classical and Robust Analyses of Mean Distance from the Origin for NeuroRep NEI's for Sites FZ, CZ, and PZ

LOCATION	CONTRAST	BAND	TYPE	t-statistic	LowerConf	UpperConf	p-value	Power	Mean CSA	Mean NCSA	Cohen' sd
Site FZ	Mean	Delta	**Classical**	0.309	-0.037	0.05	0.759	0.061	0.453	0.446	0.09
			Robust		-0.034	0.05	0.574	0.102	0.457	0.447	0.169
		Theta	**Classical**	0.222	-0.043	0.054	0.825	0.056	0.41	0.405	0.065
			Robust		-0.04	0.057	0.754	0.07	0.417	0.409	0.088
		Alpha	**Classical**	1.05	-0.044	0.141	0.299	0.183	0.367	0.319	0.304
			Robust		-0.035	0.14	0.232	0.198	0.37	0.312	0.374
		Beta	**Classical**	0.917	-0.031	0.083	0.364	0.15	0.403	0.377	0.265
			Robust		-0.027	0.092	0.236	0.276	0.409	0.376	0.329
		Cross Corr	**Classical**	0.592	-0.037	0.068	0.556	0.091	0.424	0.409	0.171
			Robust		-0.021	0.078	0.264	0.114	0.428	0.399	0.333
Site CZ	Mean	Delta	**Classical**	-1.314	-0.038	0.008	0.195	0.26	0.237	0.251	-0.382
			Robust		-0.04	0.007	0.262	0.31	0.235	0.25	-0.356
		Theta	**Classical**	-1.041	-0.036	0.011	0.303	0.18	0.252	0.264	-0.301
			Robust		-0.037	0.014	0.458	0.134	0.254	0.264	-0.234
		Alpha	**Classical**	0.328	-0.033	0.046	0.744	0.062	0.189	0.183	0.096
			Robust		-0.027	0.048	0.616	0.118	0.193	0.184	0.141
		Beta	**Classical**	0.716	-0.02	0.042	0.478	0.11	0.281	0.27	0.207
			Robust		-0.018	0.04	0.354	0.118	0.289	0.276	0.266
		Cross Corr	**Classical**	-0.242	-0.036	0.028	0.81	0.057	0.316	0.32	-0.07
			Robust		-0.033	0.035	0.982	0.062	0.314	0.314	0.006
Site PZ	Mean	Delta	**Classical**	0.215	-0.038	0.048	0.831	0.055	0.469	0.464	0.063
			Robust		-0.037	0.055	0.52	0.134	0.477	0.465	0.165
		Theta	**Classical**	-1.041	-0.036	0.011	0.303	0.18	0.252	0.264	-0.301
			Robust		-0.037	0.014	0.458	0.134	0.254	0.264	-0.234
		Alpha	**Classical**	2.199	0.007	0.156	0.033	0.594	0.432	0.35	0.65
			Robust		0.009	0.164	0.01	0.596	0.444	0.348	0.732
		Beta	**Classical**	1.422	-0.011	0.063	0.162	0.296	0.447	0.421	0.414
			Robust		-0.006	0.07	0.052	0.498	0.454	0.42	0.551
		Cross Corr	**Classical**	1.356	-0.015	0.077	0.182	0.273	0.47	0.439	0.391
			Robust		-0.012	0.078	0.122	0.342	0.474	0.44	0.419

APPENDIX B

STATISTICAL CODE FOR NEI ANALYSES

Representative Statistical Code for Analyzing NeuroRep NEI's Mean Distance from the Origin for the Alpha Band in the Left or Right Hemisphere Using the S-PLUS® 6.1 Statistical Package from Insightful (Robust Code from Wilcox, 1997)

```
###Script for Alpha Band Distance from the Origin/NEI for Left & Right Hemisphere ##
########Coefficient of Variation and Mean measurements #####
####Change output accordingly####

############ Robust Functions

bivar<-function(x){
# compute biweight midvariance of x
m<-median(x)
u<-abs((x-m)/(9*qnorm(.75)*mad(x)))
top<-length(x)*sum((x[u<=1]-m)^2*(1-u[u<=1]^2)^4)
bot<-sum((1-u[u<=1]^2)*(1-5*u[u<=1]^2))
bi<-top/bot^2
bi
}

robust.effect.size<-function(x, y){
    d<-(mest(x)-mest(y))/((bivar(x)^.5 + bivar(y)^.5)/2)
   d
}

effect.size<-function(x, y){
    d<-(mean(x)-mean(y))/((var(x)^.5 + var(y)^.5)/2)
   d
}

winvar<-function(x,tr=.2){
#
#  Compute the gamma Winsorized variance for the data in the vector x.
#  tr is the amount of Winsorization which defaults to .2.
#
y<-sort(x)
n<-length(x)
ibot<-floor(tr*n)+1
itop<-length(x)-ibot+1
xbot<-y[ibot]
xtop<-y[itop]
y<-ifelse(y<=xbot,xbot,y)
y<-ifelse(y>=xtop,xtop,y)
winvar<-var(y)
winvar
}
```

```
yuen<-function(x,y,tr=.2,alpha=.05){
#
#  Perform Yuen'  s test for trimmed means on the data in x and y.
#  The default amount of trimming is 20%
#  Missing values (values stored as NA) are automatically removed.
#
#  A confidence interval for the trimmed mean of x minus the
#  the trimmed mean of y is computed and returned in yuen$ci.
#  The significance level is returned in yuen$siglevel
#
#  For an omnibus test with more than two independent groups,
#  use t1way.
#  This function uses winvar from chapter 2.
#
x<-x[!is.na(x)]  # Remove any missing values in x
y<-y[!is.na(y)]  # Remove any missing values in y
h1<-length(x)-2*floor(tr*length(x))
h2<-length(y)-2*floor(tr*length(y))
q1<-(length(x)-1)*winvar(x,tr)/(h1*(h1-1))
q2<-(length(y)-1)*winvar(y,tr)/(h2*(h2-1))
df<-(q1+q2)^2/((q1^2/(h1-1))+(q2^2/(h2-1)))
crit<-qt(1-alpha/2,df)
dif<-mean(x,tr)-mean(y,tr)
low<-dif-crit*sqrt(q1+q2)
up<-dif+crit*sqrt(q1+q2)
test<-abs(dif/sqrt(q1+q2))
yuen<-2*(1-pt(test,df))
list(ci=c(low,up),siglevel=yuen,dif=dif,se=sqrt(q1+q2),teststat=test,crit=crit,df=df)
}

mest<-function(x,bend=1.28){
#
#  Compute M-estimator of location using Huber'  sPsi.
#  The default bending constant is 1.28
#
if(mad(x)==0)stop("MAD=0. The M-estimator cannot be computed.")
y<-(x-median(x))/mad(x)  #mad in splus is madn in the book.
A<-sum(hpsi(y,bend))
B<-length(x[abs(y)<=bend])
mest<-median(x)+mad(x)*A/B
repeat{
y<-(x-mest)/mad(x)
A<-sum(hpsi(y,bend))
B<-length(x[abs(y)<=bend])
newmest<-mest+mad(x)*A/B
```

```
if(abs(newmest-mest) <.0001)break
mest<-newmest
}
mest
}

hpsi<-function(x,bend=1.28){
#
#   Evaluate Huber`s Psi function for each value in the vector x
#   The bending constant defaults to 1.28.
#
hpsi<-ifelse(abs(x)<=bend,x,bend*sign(x))
hpsi
}

############  End Robust Functions

########## Begin Data Analysis

##### Right Hemisphere

alpha.right.F2<-((nei.data.frame$F2.alpha.1)^2+
                 (nei.data.frame$F2.alpha.2)^2+
                 (nei.data.frame$F2.alpha.3)^2)^.5

alpha.right.F8<-((nei.data.frame$F8.alpha.1)^2+
                 (nei.data.frame$F8.alpha.2)^2+
                 (nei.data.frame$F8.alpha.3)^2)^.5

alpha.right.F4<-((nei.data.frame$F4.alpha.1)^2+
                 (nei.data.frame$F4.alpha.2)^2+
                 (nei.data.frame$F4.alpha.3)^2)^.5

alpha.right.C4<-((nei.data.frame$C4.alpha.1)^2+
                 (nei.data.frame$C4.alpha.2)^2+
                 (nei.data.frame$C4.alpha.3)^2)^.5

alpha.right.T4<-((nei.data.frame$T4.alpha.1)^2+
                          (nei.data.frame$T4.alpha.2)^2+
                          (nei.data.frame$T4.alpha.3)^2)^.5

alpha.right.O2<-((nei.data.frame$O2.alpha.1)^2+
                 (nei.data.frame$O2.alpha.2)^2+
                 (nei.data.frame$O2.alpha.3)^2)^.5
```

```
alpha.right.T6<-((nei.data.frame$T6.alpha.1)^2+
                 (nei.data.frame$T6.alpha.2)^2+
                 (nei.data.frame$T6.alpha.3)^2)^.5

alpha.right.P4<-((nei.data.frame$P4.alpha.1)^2+
                 (nei.data.frame$P4.alpha.2)^2+
                 (nei.data.frame$P4.alpha.3)^2)^.5

alpha.right.all<-cbind(alpha.right.F2,alpha.right.F8,alpha.right.F4,
                       alpha.right.C4,alpha.right.T4,alpha.right.O2,
                         alpha.right.T6,alpha.right.P4)

alpha.right.std<-apply(alpha.right.all, 1, var)
alpha.right.std<-alpha.right.std^.5
alpha.right.mean<-apply(alpha.right.all, 1, mean)
alpha.right.cv<-alpha.right.std/alpha.right.mean
alpha.right.cv

###### Left Hemisphere

alpha.left.F1<-((nei.data.frame$F1.alpha.1)^2+
                (nei.data.frame$F1.alpha.2)^2+
                (nei.data.frame$F1.alpha.3)^2)^.5

alpha.left.F7<-((nei.data.frame$F7.alpha.1)^2+
                (nei.data.frame$F7.alpha.2)^2+
                (nei.data.frame$F7.alpha.3)^2)^.5

alpha.left.F3<-((nei.data.frame$F3.alpha.1)^2+
                (nei.data.frame$F3.alpha.2)^2+
                (nei.data.frame$F3.alpha.3)^2)^.5

alpha.left.C3<-((nei.data.frame$C3.alpha.1)^2+
                (nei.data.frame$C3.alpha.2)^2+
                (nei.data.frame$C3.alpha.3)^2)^.5

alpha.left.T3<-((nei.data.frame$T3.alpha.1)^2+
                    (nei.data.frame$T3.alpha.2)^2+
                    (nei.data.frame$C3.alpha.3)^2)^.5

alpha.left.O1<-((nei.data.frame$O1.alpha.1)^2+
                (nei.data.frame$O1.alpha.2)^2+
                (nei.data.frame$O1.alpha.3)^2)^.5
```

```
alpha.left.T5<-((nei.data.frame$T5.alpha.1)^2+
                (nei.data.frame$T5.alpha.2)^2+
                (nei.data.frame$T5.alpha.3)^2)^.5

alpha.left.P3<-((nei.data.frame$P3.alpha.1)^2+
                (nei.data.frame$P3.alpha.2)^2+
                (nei.data.frame$P3.alpha.3)^2)^.5

alpha.left.all<-cbind(alpha.left.F1,alpha.left.F7,alpha.left.F3,
                      alpha.left.C3,alpha.left.T3,alpha.left.O1,
                        alpha.left.T5,alpha.left.P3)

alpha.left.std<-apply(alpha.left.all, 1, var)
alpha.left.std<-alpha.left.std^.5
alpha.left.mean<-apply(alpha.left.all, 1, mean)
alpha.left.cv<-alpha.left.std/alpha.left.mean
alpha.left.cv

alpha.hemisphere.cv<-data.frame(cbind(alpha.right.cv, alpha.left.cv))
alpha.hemisphere.cv

alpha.hemisphere.mean<-data.frame(cbind(alpha.right.mean, alpha.left.mean))
alpha.hemisphere.mean

##########

csa<-alpha.hemisphere.mean$alpha.left.mean[nei.data.frame$CSA==1]
csa
ncsa<-alpha.hemisphere.mean$alpha.left.mean[nei.data.frame$CSA==0]
ncsa

x<-csa
y<-ncsa

normal.sample.size(mean=mean(csa), mean2=mean(ncsa), sd1=var(csa)^.5,
                   sd2=var(ncsa)^.5, n1=length(csa), n2=length(ncsa))

####### Begin Robust Mean Difference Test with Bootstrapped
#       Observed P value, and Bootstrapped Empirical Power

#### Calculate the size of the two groups

N1<-length(x)
N2<-length(y)
```

```
##### Number of bootstrap samples

nboot<-500

##### Alpha criterion

alpha<-.05

#### Non-directional test=2, directional test=1

empTAIL<-1

#### Assign type of estimator (mean, median, mest.MASS) and Tuning Constant

est<-mest
k<-1.28

######## Re-center data so that H0
#       holds in original sample

z.x<-x
z.y<-y

c.x<-(z.x-est(z.x, k)) # Center x vector
                # according to estimation method

c.y<-(z.y-est(z.y, k)) # Center y vector according
                # to estimation method

cx.cy<-c(c.x,c.y)      #Stack x and y vector

#############################################################
#
# Sampling from H0:  Population is stacked x and y vector;
#                 Sample with replacement

h0datax <- matrix(sample(cx.cy, size = N1 *
        nboot, replace = T), nrow = nboot)

h0datay <- matrix(sample(cx.cy, size = N2 *
        nboot, replace = T), nrow = nboot)

h0bvecx <- apply(h0datax, 1, est, k)
h0bvecy <- apply(h0datay, 1, est, k)
h0bvec <- sort(h0bvecx - h0bvecy)
```

```
###### Calculate critcal cutoffs

critup<-quantile(h0bvec,.975)
critlow<-quantile(h0bvec,.025)

#### Calculate two-sided mean difference test probability for
#    observed difference

diff.empirical<-est(z.x, k)-est(z.y, k)

count<-length(h0bvec[abs(h0bvec)>=abs(diff.empirical)])
pvalue.empirical<-count/nboot

#####################################################
#
# Sampling from H1: Sample wih replacement from
# x and y vectors separtely

h1datax <- matrix(sample(x, size = N1 *
        nboot, replace = T), nrow = nboot)

h1datay <- matrix(sample(y, size = N2 *
        nboot, replace = T), nrow = nboot)

#### Apply M-estimator to each data vector and
#    accumulate in new data vectors

h1bvecx <- apply(h1datax, 1, est, k)
h1bvecy <- apply(h1datay, 1, est, k)

#### Sort and subtract Bootstrap M-estimate data vectors
#    and accumulate into a M-estimator difference vector

h1bvec <- sort(h1bvecx - h1bvecy)

##### Calculate the upper and lower cuttoff percentiles
#     for the lower and upper alpha criterion

effectlow <- round((alpha/empTAIL) * nboot)
effectup <- round((1 - alpha/empTAIL) * nboot)

#### Calculate Empirical Power

countup<-length(h1bvec[h1bvec>=critup])
countlow<-length(h1bvec[h1bvec<=critlow])
power.twotail<-(countup+countlow)/nboot
```

```
#####Calculate M-estimate differences that correspond to the
#    upper and lower alpha criterion cuttoffs
#

h1.ci<-list(ci = c(h1bvec[effectlow], h1bvec[effectup]))

t.test(x,y)
yuen(x,y)

mean(x)
mean(y)
effect.size(x,y)

mest(x)
mest(y)
robust.effect.size(x,y)

### Bootstrapped p-values, power and confidence intervals

pvalue.empirical
power.twotail
h1.ci

par(mfrow=c(1,2))
qqnorm(csa)
qqline(csa)
qqnorm(ncsa)
qqline(ncsa)
```

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