Running head: QUANTITATIVE ELECTROENCEPHALOGRAPHIC AMPLITUDE

Quantitative Electroencephalographic Amplitude, Memory, and Cognitive Ability in Canadian First Nation Adult Offenders With and Without Solvent Abuse Histories

Michael R. Moland

Supervisor:	Dr. Charles Netley
Readers Internal to the Department:	Dr. Dwight Mazmanian Dr. Gordon Hayman
Reader Internal to the University:	Dr. Ian Newhouse

Your file Votre référence
ISBN: 0-494-10687-5
Our file Notre référence
ISBN: 0-494-10687-5

Acknowledgements

Many have played a vital role in this process including, but not limited to:
my supervisor Dr. Netley for his patience and wisdom,
Cameron Dokis, my indefatigable research assistant,
Dan Blair, social worker, and staff at the Thunder Bay Correctional Centre,
Drs. Michael Wesner and Josephine Tan for their friendship and encouragement,
Mary Lysenchuk who was always there to help,
Sheila Delin– without your continual support and compassion I couldn't have made it,
my cat Zach for his unwavering companionship,
my dad for his support and continual pushing to get things done,
and last but clearly not least, my wife Tanya for her patience, love, organizational ability, and direction that helped make the completion of my dissertation a reality.

Dedication

In loving memory of Lorraine Marie Moland
November 27, 1939 – March 27, 2003

I owe this dissertation and my success to my mom. Through my younger years, she held herself and her children to the highest standard. She always expected that we would go to university, despite her having to leave school to work after Grade Eight. As a testament to her perseverance and work ethic, she completed grade 9 when she was in her late 40s while running the household and completing hairdressing school. Her expectations have been realized, and because of her encouragement my brother Mark is a civil engineer, my sister Tina is a physical therapist and recent MBA graduate, and my youngest sister Jody is a pharmacist.

She tragically passed away from cancer during the completion of this dissertation and she will not witness yet another fruit of her labour on my graduation day. I will be thinking of you when I walk across the stage, mom. My sister Tina wrote this in her memory:

One of my earliest memories of mom is sitting with her on our couch. She was reading to me, and when she finished she pulled me in close and said, "when mom and dad are finished working on the house, I'll have a lot of time to read with you." Of course, they were still working on our house when I entered first-year university. But I didn't mind. Yep, without giving a second thought to gender roles or stereotypes, my mom rolled up her sleeves and, alongside my dad, built an addition onto our home for me and my brothers and sister.

They say that everything you do is a self-portrait. If this were the case, then hard work would definitely be reflected in my mom's image. The lines of her portrait would be clearly defined, the attention to detail would be meticulous, and the brush strokes would be strong and deliberate. Her picture would most definitely capture her determined and dedicated way. The feature that would be the most striking about my mom, however, would be her uncompromising selflessness. Everyone who knew her can attest to the exceptional and limitless nature of her ability to give of herself to others.

It is these qualities that inspired and motivated me growing up, and continue to do so to this very day. It is also these same qualities that served both of us well throughout, and especially toward the end of my mom's illness. I'd like to think that these things--the 'best of my mom'--have been passed down to me. Everyday, in everything that I do, the best of mom will be reflected in my own image. And for this incredible gift that she has given me, I both love and thank her for.

Table of Contents

Abstract

Most electroencephalographic (EEG) studies of solvent exposure have involved factory workers chronically exposed to industrial chemicals. Few studies have examined the neurological effects of recreationally abused solvents, and none have explored EEG and solvent abuse in Canadian First Nations people. Measures of EEG amplitude, working memory, cognitive ability, depression, and anxiety were used to compare adult First Nations male offenders (n = 28) with First Nations male university students (n=14). Offenders showed lower absolute theta and alpha amplitudes bilaterally. Compared to university students, they also showed significantly reduced scores on digit-span backward, letter-number sequencing, and a measure of nonverbal cognitive ability. No group differences were found for depression and anxiety. Offenders reporting a history of solvent abuse (n = 10) were also compared to those with no history of solvent abuse (n = 18) and controls (n = 14). Solvent-abusing offenders scored significantly lower than controls on digits forward, digits backward, letter number sequencing, and cognitive ability, while offenders without solvent abuse histories scored lower than controls on letter-number sequencing and cognitive ability. Only digits-forward scores significantly differentiated solving-abusing from non-solving-abusing offenders. On EEG measures, significant group differences were found on absolute alpha amplitudes in eyes-open and letter-number sequencing tasks, with solvent abuse history offenders showing decreased alpha EEG amplitudes compared to non-solvent abusing offenders and controls. The findings suggest that First Nations offenders reporting a history of solvent abuse show evidence of reduced memory performance and reduced cortical EEG functioning compared to non-solvent abusing First Nations offenders and First Nations controls.

Quantitative Electroencephalographic Amplitude, Memory, and Cognition in Native Canadian Adult Offenders With and Without Solvent Abuse Histories

The problems and consequences of solvent abuse are the focus of increasing concern in northwestern Ontario, particularly in isolated communities, where the prevalence of substance abuse in general and solvent abuse in particular is high, especially among First Nations populations (Byrne, Kirby, Zilbin, & Ensminger, 1991; Edminster & Bayer, 1985). Youth appear at greatest risk because of community isolation, few organized social activities, and limited access to alcohol and other drugs (Remington & Hoffman, 1984). Research into the etiology of solvent abuse suggests that family problems, parental alcohol abuse, physical or sexual abuse, and poverty contribute to a high incidence of abuse (Smart, 1986) and these themes figure prominently in demographic profiles of many northern native communities (Brant, 1993). Recent research indicates that First Nations youth from reservations and rural communities use inhalants at high rates (Howard et al., 1999). For instance, Gfellner & Hundleby (1995) reported that Canadian First Nations students from grades seven through twelve school were significantly more likely to have sniffed glue or solvents in the past year than their Caucasian peers. Similarly, high solvent rates were reported among First Nations high school students compared to rural Francophone students within the same geographic area (Lalinec-Michaud et al., 1991). Others have found variations in solvent abuse of between 16 and 31% in rural and reservation seventh to twelfth graders (Beauvais et al., 1989) and rates of 44% in a boarding school sample (Okwumabua & Duryea, 1987) which has confirmed suggestions that rates of inhalant use increase in geographically remote regions (Howard et al., 1999) including isolated First Nations reservations (Edwards &

Oetting, 1995). Many of the observations from studies that have explored patterns of inhalant abuse and classification of inhalant users in First Nation communities support three conclusions: (1) recreational use of solvents is widespread among preteens and teens between 15 and 18 years of age, (2) polydrug use and antisocial traits are typical in studied samples, and (3) geographically and socially disadvantaged adults make up a significant proportion of inhalant abusers (May & Del Vecchio, 1997; Oetting, Edwards & Beauvais, 1988).

The health consequences of inhalant abuse are often life threatening, either directly through toxic exposure or indirectly through asphyxiation and accidental injury (Anderson, MacNair, & Ramsey, 1985; Espeland, 1993). Recent understanding of the clinical features and abnormal electrophysiological profiles associated with chronic inhalation and sniffing have led to a growing concern for the welfare of individuals in at-risk populations. Symptoms such as acute and chronic encephalopathy, cerebellar ataxia, seizure, abnormal movement, and anorexia have been related to solvent abuse (Brady & Torzillo, 1994). There have been a number of attempts to document more specific effects of chronic exposure to solvents on the central nervous system (CNS) (Dinwiddie, 1994) and to determine to what extent these alterations are irreversible in abstinence (Brady & Torzillo, 1994). Little is known, however, about the basic neurophysiological effects of chronic solvent abuse or the residual effects that may be seen during abstinence in humans (Roemer, Cornwell, Dewart, Jackson, & Ercegovac, 1995).

Research also suggests that the sequelae of substance abuse may alter treatment efficacy and substance abuse relapse (Roemer, et al., 1995). Such investigations have been undertaken with substances like cocaine and support the

view that drugs can produce changes in neurotransmission that persist in drug abstinence (Noldy, Santos, Politzer, Blair, & Carlen, 1994; Prichep et al., 1996; Roemer et al., 1995). Therefore, increased understanding of brain dysfunction in solvent abuse and dependence is needed, and the development of an objective profile of CNS recovery from solvent exposure would be a useful contribution (Roemer et al., 1995). This has prompted a recent drive toward designing research strategies to study youths prospectively who are at risk for inhalant and other drug abuse, and to examine rates of co-morbid psychopathology among inhalant users in different subcultures (Dinwiddie, 1994). Systematic investigations of the effects of solvent abuse on the brain may serve to increase our understanding of brain dysfunction associated with solvents (Dinwiddie, 1994) and a clearer understanding of the relationship between specific chemicals and risk of neurological damage will have obvious relevance to the treatment of solvent abusers, as well as other forms of chemical dependency. Because of the serious long-term effects of solvent ingestion (e.g., gasoline "sniffing") and its potential lethality, any level of success at reducing solvent abuse would be highly beneficial (Remington & Hoffman, 1984).

The Effects of Solvent Abuse

The abuse of volatile substances has received increasing attention since the 1960s, and more recently because of the rash of deaths involving aboriginal youth in Davis Inlet, Newfoundland (Clayton-Olfield & Keefe, 1999). Aerosols, gasoline, cleaning fluids, and paint thinners are commonly abused solvents (Dinwiddie, Zorumski, & Rubin, 1987). Usually, fumes from a solvent-soaked rag are "sniffed" or the solvent is placed in a

plastic bag and inhaled by mouth (Barnes, 1979; Dinwiddie, 1994). Drug inhalation provides immediate access to the capillary bed lining of the lungs and systemic circulation, with onset and intensity of effects approaching that of intravenous injection (Grilly, 2002). A notable pharmacokinetic characteristic of organic solvents is their volatility and their affinity for lipid tissue, which allows solvents to readily bind to fat tissue (Morrow, 2002) and to be gradually released from these fat stores into systemic circulation to produce their prolonged toxic effects (Baker, Smith, & Landrigan, 1985; White & Proctor, 1997).

Clinically, solvent intoxication resembles alcohol intoxication with behavioural disinhibition and euphoria, followed by depression at higher doses (Dinwiddie, 1994). Physiological correlates of chronic use include tinnitus, cerebellar ataxia, and, less often, visual and auditory hallucinations (Dinwiddie et al., 1987). The behavioural and physiological effects of solvents typically subside after thirty to forty-five minutes, at which time the user usually becomes sleepy and may have poor recollection of preceding events (Ron, 1986). Peripheral nervous system complications arising from solvent exposure include renal dysfunction, abdominal pain, generalized weakness, ventricular fibrillation, and in rare cases, sudden death due to heart failure (Morton, 1987; Ron, 1986). CNS manifestations of long-term exposure involve cerebellar toxicity, atrophy, and dysfunction, as well as irreversible encephalopathy, and dementia (Dittmer, Jhamandas, & Johnson, 1993; Filley, Heaton, & Rosenberg, 1990; Fornazarri, Wilkinson, Kapur, & Carlen, 1983) consistent with reductions in brain mass identified through computerized axial tomography (CAT) (Fornazarri, Wilkinson, Kapur, & Carlen, 1983). *Acute*

encephalopathy arising from gas sniffing can cause visual hallucinations and ataxia, though these effects appear to be reversible (Remington & Hoffman, 1984). However, irreversible encephalopathy can occur in *chronic* gasoline inhalers and is characterized by dementia and ataxia that remain following acute symptom remission (Valpey, 1978). Clinical examination, magnetic resonance imaging (MRI), and cortical evoked potentials showed evidence of neurological damage in 13 of 20 (65%, Holmes, Filley, & Rosenberg, 1986) and 6 of 11 (55%, Rosenberg, Spitz, Filley, Davis, & Schaumburg, 1988) of chronically exposed individuals.

Most of the available research on the effects of solvents is confined to chronically-exposed factory workers. Several EEG abnormalities not seen in unexposed controls have been documented. In one study, a group of workers exposed to jet fuel over 17 years revealed significant EEG changes marked by lower amplitude, lower quantity rhythmic activity, and higher alpha activity compared to unexposed matched controls (Knave et al., 1978). Industrial spray painters showed subtle EEG abnormalities in the form of increased alpha activity (Elofsson et al. 1980). In a similar study, EEG abnormalities persisted for 3 to 9 years in 42% of a group of patients diagnosed as having chronic occupational solvent intoxication (Seppalainen & Antti-Poika, 1983).

However, serious methodological problems exist in the current literature. For instance, poor selection protocols (e.g., participants are often referred because of suspected neurological dysfunction) have compromised the generalizability of research findings. Furthermore, since most studies of solvent abuse conducted to date consist of case reports, the true prevalence and type of neurological damage associated with solvent abuse is virtually unknown (Dinwiddie, 1994). Moreover,

because most research into the pathophysiology of solvent abuse has included workers exposed to industrial chemicals rather than those individuals from specific populations that intentionally ingest solvents for recreational use, little can be concluded regarding the risk of neurological damage or at what level it occurs in other at risk populations. Furthermore, polysubstance use and other confounds (e.g., acquired brain injury, disease) make it exceedingly difficult to ascertain the specific neurological damage contributed by solvents in the absence of controlled studies (Roemer, Shagass, Dubin, Joffe, & Katz, 1991).

Better-controlled studies examining EEG and evoked potentials have verified the existence of abnormalities in chronic solvent abusers (Metrick & Brenner, 1982; Poklis & Burkett, 1977). To what degree these findings are reversible with abstinence is unknown, though Bruhn et al. (1981) report poor or no recovery from intellectual impairment and cortical degeneration.

Nevertheless, despite methodological inadequacies in previous research, there are clear indications that solvent abuse is associated with neuropsychological impairment (Fornazzari et al., 1983; Grabaski, 1961; Kelly, 1975; Lolin, 1989) and that chronic use in particular may progress to atrophy and dysfunction (Lazar, 1983). However, without systematic, well-controlled studies, particularly involving groups who engage in chronic recreational abuse of solvents, a clear picture of the nature and pathophysiological complexities of solvent abuse remains undetermined.

Treatment and Relapse Prevention of Solvent Abuse

Treatment of chronic solvent abusers is very difficult, expensive, and not highly effective (Committee on Substance Abuse and Committee on Native

American Child Health, 1996). Co-morbidity between psychopathology and polysubstance use by many solvent users are among the array of complicating factors preventing the development of specific treatment protocols for this population. For instance, many solvent abusers enter rehabilitation programmes to treat addiction and dependence stemming from a variety of other drugs (e.g., alcohol, cocaine). Consequently, little is known about solvent abusers or their long-term outcomes (Dinwiddie, 1994), despite the prevailing view that prevention of inhalant abuse and relapse should be a primary goal of treatment (Espeland, 1993). Although residential management is widely believed to be important to successful treatment outcome, few systematic studies concerning efficacy or comparisons with alternative treatment approaches exist to support this contention (Dinwiddie, 1994).

Available studies suggest that treatment often has an unfavourable prognosis. Skuse and Burrell (1984) found that of 14 individuals offered treatment, 10 participated and only five were reported to have improved. Long-term follow-up was not available, but there is reason to be sceptical of enduring treatment benefit, as the extent of problems in solvent abusers is great and current treatment strategies have not been very successful (Dinwiddie, Zorumski, & Rubin, 1987). One study of 10 patients in an inpatient drug rehabilitation program found that only 5 completed the program and all participants relapsed at six-month follow-up (Dinwiddie, Zorumski, & Rubin, 1987).

Solvent Abuse in Canada

Although some research has been devoted to the study of solvent abuse in different ethnic groups, comparatively little has focused specifically on Native Canadian populations (Beauvais, Wayman, Jumper-Thurman, Plested, & Helm, 2002). In Canada, an estimated three to six percent of young people have tried inhalants, with comparatively higher rates of use among aboriginal youth (Smart, 1986). Most solvent abuse begins during childhood and adolescence (Dinwiddie et al., 1987) and the use of solvents in this age group has been associated with concurrent behavioural and mental problems, including academic difficulties, conduct disorder, and shoplifting (Lockhart & Lennox, 1983). Boeckx, Posh, and Cohen (1977) and Chalmers (1991) report that Canadian First Nations and Inuit communities are at high risk for solvent abuse, as are other impoverished ethnic minorities.

Antisocial Characteristics and Solvent Abuse

Although important aspects of inhalant use disorders remain unresolved, such as the degree to which dependence is associated with a characteristic withdrawal syndrome, it is generally agreed that inhalant use is most prevalent among preteens and early adolescents and often occurs in conjunction with other antisocial conduct (Howard et al., 1999). The family backgrounds of inhalant users have consistently been found to be chaotic and disruptive, with emotionally distant parents, frequent physical abuse, and antisocial behaviour by other family members (Morton, 1987). There are clear associations in the literature between adolescent inhalant abuse and antisocial characteristics, social deviance and criminal backgrounds (Compton et al., 1994; Jacobs

& Ghodse, 1988; McGarvey, Canterbury, & Waite, 1996). However, such negative behavioural backgrounds tend to disappear when poverty diminishes (Costello, Compton, Keeler, & Angold, 2003). Furthermore, adolescents who abuse solvents are at increased risk for violence related to accidents, and suicide (Gompton et al., 1994).

The incidence of antisocial personality disorder among solvent abusers is consistently high (Dinwiddie et al., 1997) with one study reporting antisocial personality disorder in 63% of a solvent abuse sample compared to 12% in non-solvent abusing controls (Dinwiddie et al., 1990). Non-solvent-abusing siblings also seem at greater risk for the diagnosis of antisocial personality disorder compared to non-solvent-abusing, drug-using controls, suggesting that this diagnosis may be an important risk factor in the abuse of solvents above and beyond recreational use of other drugs (Crites & Schuckit, 1979; Dinwiddie et al., 1987). Lifetime conduct and alcohol dependence disorders were 3.3 and 2.6 times more prevalent among inhalant users than non-users in one sample (Howard et al., 1999). In solvent abusers, antisocial characteristics are often seen to co-exist with other disorders such as depression. In a study of 130 inhalant users by Dinwiddie, Reich, and Cloninger (1990) inhalant use was associated with significantly elevated levels of depression, alcoholism, and antisocial personality disorder. However, this relationship disappeared after controlling for the mediating effects of the antisocial personality.

In one study examining the empirical correlates of the MMPI-2 in two American Indian tribes, researchers found significant correlates between the Schedule for Affective Disorders – Lifetime (SADS-L) semi-structured interview information and antisocial and

anger symptoms (Green et al., 2003). Howard & Jenson (1998) reported that inhalant abuse was a "red flag" distinguishing offenders with greater suicidality, criminality, family problems and heavy substance abuse from less disordered offenders. Likewise, McGarvey, Canterbury & Waite (1996) noted that inhalant abusing offenders were significantly more likely to report threatening to hurt people, have relatives that attempted suicide, and to have committed crimes while under the influence than their non-abusing counterparts. Others have confirmed the general finding that inhalant abuse among adolescents frequently occurs in conjunction with other antisocial and aggressive behaviour (Compton et al., 1994; Howard et al., 1999; May & Del Vecchio, 1997; Oetting & Webb, 1992; Reed & May, 1984). Friedman and Friedman (1973) found that inhalant abusers reported the highest levels of violence and aggression when compared to other-drug and non-drug users. A review of previous research concluded that in inhalant abusers, conduct disorder was more prevalent than any other disorder (50% to 90%) (Otto et al., 1992). These findings are consistent with those of other investigators who have identified high rates of antisocial conduct, alcohol dependence, deviant peer associations, low SES, male gender and low self-esteem among inhalant users relative to nonusers (Oetting & Webb, 1992; May & Del Vecchio, 1997). Antisocial behavioural patterns are particularly evident in rural Indian inhalant abusers compared to their non-abusing peers (Howard et al, 1999). Moreover, solvent abusers also report increased *expression* of aggressive and delinquent behaviour than non-abusers, and this aggressive behavior has been found to be an important predictor of inhalant abuse (Howard et al., 1999). Indian youth are also over-represented throughout the criminal justice system (Armstrong et al., 1996). Krisberg et al. (1987) found that with the exception of African-Americans, Indian

males had the greatest incarceration rates in all public juvenile correctional facilities. In a more recent study in rural Wisconsin, Poupart (1995) found that Indian youths were twice as likely to be detained than their white counterparts.

Depression

While case studies have found high rates of psychological disorder in inhalant abusers (e.g., Lewis, Moritz, & Merlis, 1981), it is difficult to distinguish symptoms that indicate psychological distress (e.g., motivational problems, psychomotor slowing) from symptoms that are commonly associated with gross neurological damage (Dunwiddie, 1994). Compounding this problem is evidence suggesting a high rate of depression in adult offender populations (Fazel & Danesh, 2002) as well as in substance abusers (Kelly, McKellar, & Moos, 2003; Raimo & Schuckit, 1998) and in First Nations populations in general (Brant, 1993). A number of research investigations suggest Indian adolescents may be troubled by more serious mental health problems than adolescents in the general population (Beals et al., 1997; Jones et al., 1997). Findings from larger scale studies indicate a clear relationship between depression and First Nations peoples. In the revised MMPI-2 normative study, the 77 American Indian individuals sampled scored higher than European Americans on most of the clinical scales (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) including depression. Depression is often identified among the difficulties experienced by American Indian youth (Beiser & Attneave, 1982; Yates, 1987). Depression is among the most frequently acknowledged psychopathologies in American Indian Communities, second only to suicide and alcoholism (Shore & Manson, 1983). Perceived discrimination has also been associated with stress and depression in First Nations adults and adolescents (Rumbaut, 1994)

particularly in those who identify more strongly with their culture (Whitbeck, et al., 2001).

Unfortunately, there remain few attempts to study depression in First Nations populations on a larger scale. One reason for a lack of progress in this area stems from a resistance by service providers in providing labels like depression to American Indian adolescents due to concerns over cultural appropriateness and possible stigma (Neligh, 1990). Another reason is that studies exploring depression in First Nations populations either do not thoroughly screen or properly follow up equivocal responses to depression inventory items. Previous studies showing high rates of depressive disorders in American Indian communities have utilized methods involving additional probing and discussion of the meaning of questions rather than conventional question-response style interviewing techniques (Kinzie et al., 1992; Robin et al., 1998). Yet another explanation that is achieving some support in the literature is that First Nations groups may differ from each other and from Caucasian groups in they way in which they conceptualize, understand, and express depressive symptoms. For instance, among the Hopi, symptoms that comprise major depression are common to at least 5 other illness types, while expressions such as "loneliness" are common markers of depression in other tribes (Beals et al., 2005).

Furthermore, Manson (1994) found consistencies within and variation between groups comprising 160 Pacific Northwest tribe and 150 middle class European American adult outpatients on depression and anxiety symptoms, demonstrating the importance of attending to cultural factors in assessing and interpreting depressive symptomatology in

and across First Nations groups. It is important to consider that traditional measures of depression were developed for use in primarily white populations, and constructs have been assumed to apply to ethnic minorities (Thrane, Whitbeck, Hoyt, & Shelley, 2004). However, given the evidence that First Nations peoples conceptualise depressive symptoms differently, these instruments must be used cautiously or modified so they are more suitable for the intended population (Ackerson, Dick, Manson, & Baron, 1990; Manson et al., 1990). Another explanation for the lack of progress in understanding depression in First Nations populations may stem from cultural factors or the lack of effectiveness of health services that discourage First Nation individuals from seeking treatment, or it could be that depression may be expressed through antisocial behaviour and eventual incarceration in some instances where the depression is not addressed and treated. In a study of 130 inhalant users by Dinwiddie, Reich, and Cloninger (1990) inhalant use was associated with significantly elevated levels of depression, alcoholism, and antisocial personality disorder. It is evident that the lives of many Indian adolescents are rife with stressors that can lead to serious emotional and behavioral problems and resources to treat such problems are typically inadequate, particularly in reservation settings (Duclos et al., 1998). Given the poor living conditions in Northern reserves, and lack of meaningful activities and alternatives, as well as the under-funded and poorly managed nature of health care in general and substance abuse programming in particular, it is tenable that many detention centers are serving as mental health treatment facilities for First Nations peoples (Duclos et al., 1998; Neligh, 1990; Soler, 1992). It is likely Indian youths who enter the criminal justice system will have an even higher prevalence of mental health problems than other youths (Duclos et al., 1998).

Occupational exposure to organic solvents may also be associated with increased psychiatric symptomatology, particularly depression (Hakkola, 1994; Linz et al., 1986; Morrow, Kamis, & Hodgson, 1993). This may be particularly true for those with a history of chronic exposure, as findings have shown low rates of past psychiatric disorder in acutely exposed and non-exposed individuals, but a significantly greater prevalence of current *DSM–IV* Axis I disorders (50% met criteria for a mood disorder) in those with a history of solvent exposure (Morrow et al., 2000).

Whether this is due to changes in the brain produced as a consequence of solvent abuse or from a result of a predispositional tendency to seek out solvents in depressed individuals remains to be determined (Morrow, Stein, Bagovich, Condray, & Scott, 2001). Depression may also be inferred from the high rates of suicide among Native adolescents (Duclos et al., 1994; May & Van Winkle, 1994). Researchers have suggested that among American Indians depression is thought to be disproportionately greater than in non-American Indian populations (Wardman & Khan, 2004). Consistent with this view, researchers have found a high prevalence of antidepressant medication use in a west coast sample of First Nations individuals (9.8%) and suggest that this might reflect higher prevalence of depression or that depression is over-diagnosed in First Nations people (Wardman & Khan, 2004). Of note, a 3:1 ratio of female to male depression identified in the above study has been found by others (Barron, Oge, & Markovich, 1999) and is similar to the 2:1 ratios found in Western cultures (Weissman et al., 1993) and might indicate a similar pathophysiological manifestation of depression in First Nations and Western cultures. Despite the above findings, considerable variation in the expression of depression has been reported among different American Indian tribal

groups (Somervell et al., 1993; Shore & Manson, 1981) so caution should be taken before generalizing findings across American Indian populations.

In summary, studies of depressive symptoms among First Nation populations using a wide range of methodological approaches suggest that depression is common in samples of solvent abusers. Nevertheless, primary issues concerning the generalizability of Western conceptualizations of depression to the assessment of depression in First Nations populations suggest the potential for drawing misleading conclusions from standardized tests with this group.

Anxiety

There is a paucity of literature examining anxiety in First Nations populations. Based on evidence indicating increased frequency of depression in First Nations solvent abusers, one might expect to see elevated rates of other disorders that share co-morbid features with depression. In one study of organic solvent exposure, the most prevalent diagnoses included anxiety (58%) and mood (50%), and there was considerable co-morbidity, with 36% of solvent exposed persons showing both disorders (Morrow et al., 2000). Similar findings were reported in a study of juvenile First Nation offenders with a history of substance abuse revealing that 75% of the sample had at least one other diagnosis with anxiety as high as 41% (Milin et al., 1991). Elevations in anxiety have also been reported on the MMPI-II in a First Nation sample (Green et al., 2003).

Memory, Neuropsychology, and Solvent Abuse

When examining neuropsychological symptoms in solvent exposed populations, it is difficult to determine whether decrements in neuropsychological test performance are due to underlying depressed mood or true organic impairment. Thus, depressed, non-

exposed individuals often show impairments in memory (Basso & Bornstein, 1999) and attention (Beats, Sahakian, & Levy, 1996). To further complicate this issue, researchers have also noted that heavy inhalant use can induce cortical changes that lead to depression in abusers (Berry, Heaton, & Kirby, 1976). Efforts to control for the effect of depression in solvent exposed participants undergoing neuropsychological testing have been absent from the literature. Nevertheless, one recent study examining the relationship between neuropsychological test performance and depression (50% of the test sample met DSM-IV criteria for a current mood disorder) found that depression was only related to impaired performance on visuospatial (Block Design) and non-verbal memory tasks (WMS-R Visual Reproduction) (Morrow, Stein, Bagovich, & Condray, 2001). While other investigations have found depression to be associated with decrements in learning, attention, and psychomotor speed (Abas, Sahakian, & Levy, 1990; Beats et al., 1996) reductions in visuospatial ability and non-verbal memory appear to be the most consistent findings in the literature (Cassens, Wolfe, & Zola, 1990).

In studies that have failed to control for depression, repeated inhalant abuse appears to produce diffuse cerebral damage and dementia as possible long-term consequences (AoAlousi, 1989; King, Day, Oliver, Lush, & Watson, 1981; Lazar, Ho, Melvin, & Dagrasrani, 1983). Decrements in neuropsychological test performance have also been identified among solvent inhalers (Allison & Jerrom, 1984; Tsushima & Towne, 1977) in the areas of motor speed, memory, auditory discrimination, visuomotor functioning, attention, concentration, and non-verbal intelligence. In addition, formal neuropsychological testing has revealed that solvent exposed patients show significant decrements on measures of learning and memory, attention, visuospatial ability, mental

flexibility, perceptuomotor speed, and information processing compared to non-exposed control participants (Edling et al., 1990; Morrow et al., 2000; Morrow, Robin, Hodgson, & Kamis, 1992; Morrow et al., 1990). Indeed, much of the literature examining neurobehavioural effects in populations exposed to solvents found decrements mainly in psychomotor functions and short-term memory when compared to unexposed controls (Boeckelmann et al., 2002).

Reductions in memory performance have often been seen as a hallmark for solvent abuse with studies reporting poor concentration ability and reduced performance in short-term memory in the forward digit span test (Boeckelmann et al., 2002). Similarly, others have found differences between solvent and control groups on attention and mental flexibility (i.e., digit span), learning and memory, visuospatial ability, and psychomotor speed (Morrow et al., 2000).

Cognitive Ability and Solvent Abuse

Relatively little research examining cognitive ability has been carried out in First Nations populations. A consistent finding in the current literature is that American Indian students as a group score significantly lower on verbal than performance indexes of the Wechsler Intelligence Scale for Children-Revised (WISC-R), with overall scores usually falling in the low average range, and an 8 to 19 point separation between verbal and performance IQs (McShane & Plas, 1984). One reason for this finding is that intelligence test results can be influenced by a variety of factors including cultural background and motivation (Devers, Bradley-Johnson, & Johnson, 1994). This finding suggests that performance indices of cognitive capacity may be a more accurate reflection of cognitive ability than verbal indices based on norm-referenced means. Nevertheless, it has been

suggested that careful attention should be given to motivation during testing to lessen the likelihood than non-cognitive differences are interpreted as differences on cognitive ability measures (Scarr, 1981).

Brandt (1984) cautioned that cultural differences in norms that govern question-answer behaviour may also impede test performance in First Nation samples, and contends that in most Indian cultures, speakers self-designate turns to speak and typically wait at least 10 seconds to speak compared to 2 seconds for Anglo speakers. Furthermore, question-answer approaches are often considered rude depending on tribal affiliation and background, and questions deemed inappropriate or dumb by American Indians, will often go unanswered (Allen, 1998).

The Specific Advantages of Quantitative Electroencephalography

Quantitative electroencephalographic (QEEG) methods have been used increasingly in psychiatric and psychopathological research (Barcelo & Gale, 1997), supplanting traditional electroencephalographic (EEG) methods that rely on subjective interpretation and time-consuming evaluation (Noldy & Carlen, 1990). The development of commercially available computerized QEEG permits quantification of many channels of data, allowing topographical and statistical analyses of the effects of drugs on human EEG activity (Noldy & Carlen, 1990). In particular, developments in computerized analysis of electrophysiographic recordings have enabled researchers to create colourful "maps" of the brain that clearly and dramatically show the damage caused by substance abuse. It is possible for these images to be used as psychoeducational tools as part of a treatment strategy designed to deter future abuse (Prichep & John, 1992). It is a relatively easy, quick, and practical way for researchers to examine patterns of brain

disturbance and has been shown to be a sensitive indicator of cortical electrophysiological dysfunction in individuals with neurological disorders (Prichep & John, 1992). As well, it has also been shown to be more sensitive than conventional EEG, MRI, and measures of cerebral blood flow in identifying functional CNS disturbance (John, Prichep, Fridman, & Easton, 1988; Jonkman, Poortvliet, Veering, deWeerd, & John, 1985). Compared to neuroimaging using positron emission tomography (PET), CAT, or MRI, QEEG is typically less costly, uses no radiation, does not induce claustrophobic feelings, and can be done portably or continuously in a convenient, comfortable setting (Neuwer, 1996).

The availability of computerized EEG technology and its sensitivity to drug effects has inspired renewed interest in "pharmaco-EEG" (Noldy & Carlen, 1990). Recently, QEEG has proven useful in measuring the magnitude of drug effects (Rosenthal, 1996). The potential of QEEG assessment lies in its utility as a method for identification of subtype membership and the selection of treatment in patient populations (John & Prichep, 1992). Considerable literature is now building about drug effects on EEG patterns in hopes of identifying and predicting which patients will respond well to a particular pharmacotherapeutic strategy (Frost, Kellaway, Hrachovy, Glaze, & Mizrahi, 1986). This research effort is exemplified by studies that have identified neurometric patterns capable of delineating subtypes of depressed patients who will respond to electroconvulsive therapy (ECT) (Roemer, Shagass, Dubin, Joffe, & Katz, 1991), cognitively impaired older patients who will exhibit significant cognitive decline within five years (John & Prichep, 1990), and head trauma patients who will regain cognitive competence (Thatcher, Walker, Gerson, & Geisler, 1989).

QEEG has most recently been used to identify brain anomalies in substance abusers and to direct rehabilitation strategies. Despite evidence of brain damage associated with solvent exposure, controversy continues regarding the specific sequelae of chronic solvent inhalation (Byrne et al., 1991). Thus, QEEG brain mapping might serve to more clearly isolate the effects of solvent use on the brain and, perhaps, identify a possible pathophysiological substrate in this population of substance abusers. Furthermore, QEEG has been particularly well suited to detecting significant signs associated with epileptic spikes that have failed to be detected by modern neuroimaging techniques (e.g., PET, MRI, CT) (Neuwer, 1996). Allowing solvent abusers to view the damage to their own brains from a QEEG topographic map during treatment could serve as a potentially powerful communications tool used in conjunction with traditional aboriginal teachings.

Another recent trend is QEEG's promise in identifying specific brain disturbances, enabling researchers to predict length of treatment as well as relapse in populations of substance abusers (Prichep, Alper, Kowalik, & Rosenthal, 1996). Such research suggests a potential avenue to investigate markers for relapse (Remington and Hoffman, 1984), such that early prediction of relapse potential may be used to help prevent further brain trauma. Its use in predicting relapse in chronic alcoholics has provided superior relapse prediction compared to clinical methods (Noldy & Carlen, 1990). Seventy-eight inpatients with alcoholism underwent EEG recordings (eyes closed) and additional clinical evaluations seven days after the beginning of detoxification. After discharge from hospital, patients were regularly re-evaluated for three months in order to determine relapse rates during this time. For classification of the

two diagnostic subgroups (relapsers vs. abstainers), multivariate discriminant analysis as well as artificial neural network technology was applied. Correct classification using patients' EEGs was achieved in 83-85% of cases. In comparison to abstainers, relapsers evidenced EEGs that were more desynchronized over frontal areas, suggesting functional disturbance of the prefrontal cortex.

The Present Study

The purpose of the present study was to: 1) investigate whether male Native offenders could be distinguished from male Native university controls on EEG amplitude within delta, theta, alpha, and beta bands, and on measures of working memory, cognitive ability, depression, and anxiety, 2) determine whether Native offenders who report a history of solvent abuse differ from Native offenders without solvent abuse histories and Native controls on the same measures, and 3) to have participants complete EEG testing under conventional eyes-opened and eyes-closed conditions, as well as under concentration "challenge" tasks involving working memory and cognitive ability to enhance EEG resolution.

This investigation specifically examined EEG amplitude effects because of their clinical utility (John et al., 1977; John, 1977), as well as simplicity and ease of interpretation (Pollock, Schneider, & Lyness, 1991).

Hypotheses. Given that there is little research involving solvent abusers, Natives within correctional settings, and virtually no known research examining Native EEG, working memory, and cognitive ability, the following hypotheses take into account the exploratory nature of the present study.

1) Correctional group depression and anxiety scores will be greater than controls.

2) Control participants will score significant higher on measures of working memory (i.e., digit span forward, digit span backward, letter-number sequencing) and cognitive ability (i.e., TONI) than correctional participants.
3) Correctional inmates will evidence differences in EEG amplitude measures compared to controls.
4) QEEG findings will reveal decreased alpha EEG amplitude in offenders with a history of solvent abuse (CSA) compared to offenders without a history of solvent abuse (CNSA) and control participants, consistent with decreased alpha amplitude EEG findings with industrial solvents (Knave et al., 1978)
5) Statistically significant decreases in alpha amplitude in CSA will occur most readily among challenge task conditions versus eyes open and eyes closed conditions, as challenge tasks are expected to focus attention, reduce variability, and increase the reliability of EEG measures (Gevins et al., 1997; Sterman, Kaiser, & Veigel, 1996).
6) CSAs will demonstrate higher scores on the Beck Depression Inventory and Beck Anxiety Inventory consistent with Native solvent demographic patterns and show decreased performance on the Test of Non-verbal Intelligence (TONI), the measures of working memory (i.e., Digit-Span forward and backward), and the Letter-Number Sequencing task compared to CNSA and control participants consistent with impairment in cortical functioning in solvent abuse.

Method

Participants

Twenty-eight First Nations correctional offenders from the Thunder Bay Correctional Centre (mean age = 27.3, S.D. = 8.0) and 14 First Nations controls, comprising 12 Lakehead University students, and 2 community participants (mean age = 28.9, S.D. = 7.5) volunteered and received a $25 gift certificate to a local music store as remuneration. Correctional offenders were serving sentences of less than two years in duration. Of the offenders, 79% were right handed, 18% were left handed and 3% were mixed, while among the controls 79% indicated they were right handed, 7% reported being left handed, and 14% indicated mixed handedness. Native status in the study was determined by First Nation affiliation (e.g., Mohawk, Cree, Ojibwe), and reporting at least one biological parent with full First Nation status. Correctional participants comprised approximately 60% of the total inmate population and, thus, are likely representative of the inmate population at the Thunder Bay Correctional Centre. Correctional offenders as a group reported a wide variety of past drug use involving such agents as gasoline, paint, glue, hairspray, plane fuel, nail polish, naptha, marijuana, cocaine, LSD, mushrooms, ecstasy, barbiturates, opiates, and phencyclidine. Almost all identified marijuana as the most frequently used and preferred drug. A full 90% of CSAs, 100% of CNSAs, and 50% of controls reported recent marijuana use and of these participants 60% of CSAs, 61% of CNSAs, and 21% of controls reported heavy marijuana use. (See Table 1 for a listing of drug use frequency reported across groups).

Exclusion criteria for experimental and control groups included: 1) age <18, 2) history of seizure, and 3) history of brain injury. Although 46% of the corrections sample

and 21% of the control sample reported a history of head injury, there was no report of serious injury resulting in a loss of consciousness or a diagnosis of brain damage, so it was decided to retain these participants in the study. Treatment resources were made available for participants endorsing significant depressive symptoms. No participant endorsed current suicidal ideation.

Materials

1. 16 Channel Topographic Neuromapping Instrument. The Mindset system includes a 16-channel, 1024 sample-per-second, 16 bit digital EEG acquisition amplifier in conjunction with Mindmeld Live Data Capture topographic neuromapping and electroencephalographic software for off-line analysis (Nolan Computer Systems, 2001). The amplifier includes 2 fourth-order Sallen-key active filters, 48dB roll-off per octave and 1.8Hz – 36Hz frequency pass band and was connected to a Dell Inspiron 5100 Pentium 4 notebook computer via an Adaptec 1480 SCSI card connection. The headgear was the Physiometrix e-net elastic head cap with 16 Physiometrix HydroDot Ag/AgCl biosensors.

2. Beck Depression Inventory (BDI-II). The BDI-II is a self-report measure of clinical and non-clinical depression with 21 items describing themes related to mood, sense of failure, guilt, hopelessness, pessimism, and loss of appetite (Beck, Brown, & Steer, 1996). Within each item are four statements of increasing severity that are arranged on a four-point scale (eg. "0" indicating no problem, to "3" that implies an increased problem with the item). Higher scores indicate an increase in severity of depression. A commonly used measure of First Nation depression, the CES-D (Center for Epidemiologic Studies Depression Scale) has been found to correlate moderately

(.70) with the BDI in a sample of high school students (Roberts, Lewinsohn, & Seeley, 1991), indicating that they are comparable psychometrically, yet measure slightly different facets of depression. Even though the factor structure of the CES-D has differed from that obtained with European Americans in various studies, the CES-D has displayed good overall internal consistency when used with American Indian people (Allen, 1998). However, because the BDI emphasizes somatic symptoms to a greater extent than does the CES-D (Campbell & Cohn, 1991), and factors associated with depressive affect in First Nations individuals have been shown to correlate highly with their reports of somatic complaints (Beals, Manson, Keane, & Dick, 1991) the BDI was selected as a preferred measure of depression in this study. Furthermore, unlike the BDI, the CES-D does not have an item specifically measuring suicidality (Wilcox, Field, Prodromidis, & Scafidi, 1998) and there is an elevated risk for suicide in First Nations populations (Brant, 1993).

3. Beck Anxiety Inventory (BAI). The BAI is a self-report measure assessing the severity of anxiety in individuals through responses to 21 items rated on a scale of 0 to 3 (Beck, Epstein, Brown, & Steer, 1988). Each item is descriptive of subjective, somatic, or panic-related symptoms of anxiety. It was specifically designed to reduce the overlap between depression and anxiety scales by measuring anxiety symptoms shared minimally with those of depression. Both physiological and cognitive components of anxiety are addressed. The BAI differentiates well between anxious and non-anxious groups in a variety of clinical settings and is appropriate for all adult mental health populations. (Beck, Epstein, Brown, & Steer, 1988). As anxiety is frequently seen to co-occur with depression, it was decided that a measure of anxiety

would be suitable for this study. A recent study of anxiety sensitivity found greater overall anxiety related scores in Native college students compared to matched Caucasian college students (Zvolensky, McNeil, Chebon, Porter, & Stewart (2001).

4. Letter-Number Sequencing, Digits Forward, and Digits Backward subtests of Wechsler Memory Scale – III (WMS-III). The Letter-Number Sequencing subtest of the WMS-III measures sequential processing by requiring an individual to correctly order letters and numbers presented orally. Digits Forward and Digits Backward sample short-term memory by requiring the individual to recall increasingly longer strings of numbers in the same and reverse order, respectively. Studies have shown reduced attentional ability and lower performance in short-term memory in solvent abusers (Boeckelmann et al., 2002 Morrow et al., 2000). Electroencephalographic (EEG) peak alpha frequency (PAF) has been shown to correlate with short-term memory performance (e.g., Klimesch, Schimke, Ladurner, & Pfurtscheller, 1990).

5. Test of Nonverbal Intelligence (TONI). The TONI is a language-free, 45-item non-timed measure of abstract and figural problem solving requiring participants to identify relationships among abstract figures and solve problems of increasing complexity created by the manipulation of these relationships (Brown, Serbenou, & Johnsen, 1990). Each item presents a sequence of stimulus patterns ending with a missing part. The participant completes the pattern by selecting the correct response from among either four or six alternatives. The test requires approximately 15 minutes to administer and provides a measure of cognitive functioning that is not heavily loaded with linguistic, motoric, or cultural factors (Brown, Serbenou, & Johnsen, 1990). The TONI provides a relatively culture-free measure of cognitive

functioning because it relies less on exposure to specific language material and thus minimizes bias against participants from racial, ethnic, and socioeconomic minorities (Sattler, 1988). Thus, the TONI can be used to evaluate the cognitive capacity of participants who are traditionally difficult to assess, including people with learning disabilities, mental retardation, stroke, and brain injuries or other neurological impairment. Few studies have examined cognitive ability in adult First Nations samples. Evidence suggests that First Nations students perform in the low-average range on non-First Nation standardized and norm-referenced measures of cognitive functioning, and show a preference for performance type tests over verbal oriented measures (McShane & Plas, 1984). As the TONI involves visuopatial performance type items in assessing cognitive ability, its use was deemed appropriate for this study.

Procedure

Although the amplifier arrived from the manufacturer pre-calibrated, it was re-calibrated before commencement of the study using a MINDSET calibrator that injects a precise 16 Hz, 50 microvolt signal simultaneously into all 16 channels of the amplifier. Quiet rooms at the Thunder Bay Correctional Centre and Lakehead University were tested to ensure 60Hz interference from electrical wiring would not intrude upon the EEG channels. Participants' caffeine intake was restricted to at least 2 hours before EEG testing. Ethics approval was obtained by the Lakehead University Research Ethics Board and informed consent was received and all participants were explained the nature of the study, what we intended to find out from it, and how the information would be used. A First Nations research assistant with knowledge of First Nations culture and values and

interest in the study adopted the role of interviewer, and was observed to develop a good rapport with each participant (e.g., finding common interests) before moving into background demographic questions of a more personal nature (Allen, 1998). Each participant then answered the background questions, including age, recent medication use, time of last meal, history of seizure activity, brain injury, family history of disease, recent caffeine consumption, handedness, solvent abuse history, and history of other recreational drugs (see Appendix A). These questions were selected in accordance with American Society of Electroneurodiagnostic Technologists' standards (National Competencies for Performing an Electroecephalogram, www.aset.org). The BAI and BDI-II were read aloud to each participant to ensure comprehension of inventory items and this permitted specific questions by participants to be addressed in a more natural and culturally appropriate manner. Information gathering of solvent abuse history was elaborated by asking about the inhalation of glue, gasoline, paints, and aerosols, as well as the frequency of past use and most recent exposure to gain a clearer understanding of solvent demographics and to ensure that participants understood the meaning of solvent abuse, and the range of agents classed as solvents.

EEG derivations (electrode placement) were established according to a standard 10-20 montage (Fisch, 1999). The head cap was placed using a linked ear (mastoid) reference. Abrasive paste (lemon prep) on a cotton swab was used to prepare each electrode site. A frontal adhesive pad secured the head cap to the forehead and markers ensured consistency of fit and standardized electrode placement across all participants. A UFI 1089ES impedance tester measured electrode impedance and values were kept below

5 kΩ. EEG data were sampled at 1024 samples per second and analogue-filtered with a high-pass filter.

Before commencing each condition, participants were instructed to remain as still as possible, and to refrain from blinking their eyes whenever possible. When blinking did occur, they were instructed to blink in rapid succession to facilitate removal of eye-blink artifacts in later offline analysis. Participants were encouraged to stay alert and were informed that the examiner would verbally countdown the time left in the eyes open and eyes-closed conditions in one-minute intervals to ensure that each participant remained alert and oriented. Clients were monitored for drowsiness and verbally alerted by the examiner when such states were observed behaviourally or through visual inspection of the real-time EEG record.

QEEG recording occurred under the following conditions: 1) eyes-open (5 minutes) relaxation trial as a baseline (these data were excluded from analysis), 2) a second eyes-open trial (5 minutes), 3) eyes-closed (5 minutes), 4) digits forward, 5) digits backward, 6) letter-number sequencing, and 7) the TONI.

Results

Sample

One person was eliminated from the analysis due to a history of seizure activity. Twelve percent of participants reported psychotropic medication use (2 Control and 3 Correctional). EEG investigations typically exclude participants currently on medication as a matter of course (Nuwer, 2003). However, in this sample the number of participants on medication was modest and evenly distributed between the groups, so to preserve statistical power it was decided that inclusion of these participants was warranted.

Diurnal phases throughout the day can produce variability in EEG measurements and such effects have not been controlled for in EEG investigations until recently (Aeschbach, Matthews, Postolache, Jackson, Giesen, & Wehr, 1999; Hayashi, Sato, & Hori, 1994; Sterman, 2001). For this reason, all EEG data collection sessions were controlled with respect to time of day by ensuring that control participant's EEG testing times were matched to EEG testing times from correctional participants. However, as there were twice as many corrections participants than controls, full matching of participants and controls was not possible. A chi-square analysis was conducted between experimental condition and time of day (i.e., 8am-12pm = morning; 12:01pm – 4:00pm = afternoon; and 4:01pm to 8:00pm = evening) to determine whether any statistical differences existed between the groups with respect to the time of day that EEG testing occurred. The results indicate that time of day effects were non-significant (χ^2 (2) = 2.92, p = .23) between the groups.

In light of evidence suggesting that adult offenders are at increased risk for brain injury during their lifetime (Slaughter, Fann, & Ehde, 2003) it was important to determine whether inmates showed a significantly greater probability of reported brain injury compared to controls, to rule out brain injury as a cause of possible group differences in EEG, cognition, and memory findings and because brain injury could produce spurious effects on QEEG analyses. Based on participant's self-reported recollection of past head injury, the results indicate that the incidence of brain injury between inmates and controls was not statistically significant (χ^2 (1) = 2.47, p = .12).

Pre-analysis Issues

Before data analysis, Mindmeld Analysis Functions software (Nolan Computer Systems, 2001) was used to screen all EEG data visually off-line for artifacts from eye-blinks, movement, transients, drowsiness, and cardiac pulse rate (Fisch, 1999). Data from each condition were then subjected to Fournier analysis using a Blackman window filter yielding the square root of power in ranges corresponding to delta (0.5 to 3.0 Hz), theta (3.5 to 7.5 Hz), alpha1 (8 to 14 Hz), alpha2 (14.5 to 18 Hz), and beta (18.5 to 40 Hz) frequency bands. Each participant's amplitudes were averaged over the artefact-free data within each frequency band, creating absolute amplitude measures in each of the five frequency ranges. To help increase statistical power in the presence of a large number of dependent variables, all the individual EEG scalp sites were collapsed into four quadrants, left anterior, right anterior, left posterior, and right posterior for all EEG bandwidths (Fisch & Pedley, 1989; Pollock, Schneider, Zemansky, Gleason, & Pawluczyk, 1992).

Outliers. The data were examined for missing values, univariate outliers, and multivariate outliers. No cases were found to have missing data. A total of 53 univariate outliers were identified among the EEG variables by cases with z-scores of greater than ± 3.29, and were adjusted closer to the mean until their z-score value no longer exceeded ± 3.29 (Tabachnick & Fidell, 1996). No univariate outliers were found on any of the remaining variables (age, depression, anxiety, digit span, letter-number sequencing, and TONI). Multivariate outliers were investigated using linear regression to calculate Mahalanobis distance with a chi-square criterion $p < .001$. No multivariate outliers were identified.

Assumptions. Assumptions of multivariate normality, linearity, homoscedasticity, homogeneity of variance-covariance matrices, multicollinearity, and singularity were investigated to ensure no violations. Univariate and multivariate outliers were adjusted closer to the group mean until they obtained a z-score of less than ± 3.29. This improved the normality of the data. Detrended expected normal probability plots were evaluated to ensure normality of the distributions of the variables (Tabachnick & Fidell, 1996). Bivariate scatterplots were reviewed to ensure that variables obtained an elliptical shape. Residual plots indicated that assumptions of linearity and homoscedasticity were within acceptable limits. Nevertheless, linearity, skewness, and kurtosis were modestly improved by a natural logarithmic transformation of the EEG data (Lawson, et al., 1998; Pollock et al., 1991) and this also served to ensure statistical power was optimized.

Correlations between independent and dependent variables were examined for multicollinearity and singularity. Multicollinearity occurs when a correlation between variables exceeds .90 (Tabachnick & Fidell, 1996). Multicollinearity was found within 11 of 125 EEG variables, with the majority of pairings falling within the .91 to .93 range. Given that EEG variables are highly correlated and multicollinearity is common in EEG data, this finding was deemed acceptable (Fisch, 1999). Singularity occurs when two variables are highly related to each other (e.g., $\geq .99$) and viewed as essentially identical. No singularity was found between the measured variables. (See Table 2 for pooled correlations between all paper and pencil measures; Table 3 through 22 contains pooled correlations between EEG variables and pencil and paper measures).

Analysis

To investigate whether measures of anxiety, depression, memory, and cognitive ability would serve as suitable covariates for the QEEG analysis, a correlational analysis was conducted. Results indicated that only age exerted any consistently significant influence on the cognitive ability, memory, mood, anxiety, and EEG data. For this reason, only age was used as a covariate. Analyses using age as a covariate are noted below.

A MANCOVA was conducted comparing the effect of condition (Correctional vs. Control groups) on the dependent variables of anxiety, depression, digits forward, digits backward, letter-number sequencing, and TONI. Using Wilk's criterion with age as a covariate the combined dependent variables were significantly related to the combined independent variables ($F(7,34) = 5.02$, $p < .001$). Follow-up inspection using Tukey post-hoc tests indicated that controls scored significantly higher on digits backward, letter-number sequencing, and TONI. (See Table 23 for cell means and standard deviations comparing correctional and control groups for paper-and-pencil measures). To examine whether differences existed between inmates and controls on EEG variables, multiple between-subjects MANCOVAs were performed on the four EEG locations by bandwidth (delta, theta, alpha1, alpha2, beta) by condition (eyes open, eyes closed, letter-number sequencing challenge task, test of non-verbal intelligence challenge task), using age as a covariate as described above. To reduce the chance of a spurious result and to increase power, Keppel's modified Bonferroni was used to reduce the alpha level to .0125 (Cohen, 1977; Keppel, 1992). Both theta amplitude with eyes open ($F(4,36) = 4.94$, $p=.003$) and eyes closed ($F(4,36) = 3.78$, $p = .012$), alpha1

amplitude with eyes open ($F(4,36) = 4.50$, $p = .005$), and letter-number sequencing conditions ($F(4,36) = 4.10$, $p = .008$) showed lower EEG amplitudes in the correctional group compared to controls (See Table 24 for cell means).

In light of a multiplicity of reported drug use by all inmates, it was not possible to separate users into discrete drug groups (i.e., most reporting recreational drug use, including solvent abusers, had used a number of different drugs, predominantly marijuana). While it would be ideal for substance abuse groups to include specific drug categories (e.g., marijuana vs. cocaine vs. solvents), such an undertaking would be impractical as polysubstance use is ubiquitous in substance abusing populations including corrections groups and eliminating multiple-substance users from the study would severely compromise statistical power. Similarly, all who reported more than one instance of solvent abuse also used other drugs recreationally. As a result, there was no attempt to remove polysubstance abusers from the study.

Corrections participants reporting more than one experience with solvents (CSA) were compared with all other drug-using inmates (CNSA) and controls. To explore the effect that a history of solvent abuse might have on cognition and working memory, a MANCOVA on the following dependent variables (digits forward, digits backward, letter-number sequencing, and TONI IQ) using Wilk's criteria and age as a covariate showed a significant effect of group ($F(8,72) = 4.52$, $p < .001$. Planned contrasts revealed that the control group performed better than CSA on digits forward $t(35) = 1.70$, $p = .021$, digits backward $t(35) = 1.57$, $p = .048$, letter-number sequencing $t(35) = 3.03$, $p = .005$, and TONI IQ $t(35) = 17.85$, $p = .003$. Controls performed better than CNSA on letter-number sequencing $t(35) = 2.48$, $p = .008$, and TONI IQ $t(35) = 17.67$, $p < .001$.

CNSA differed from CSA only on digits forward $t(35) = 1.49$, $p = .032$. Of note, the mean values on the above variables for the offenders with a solvent history were in the expected direction (i.e., solvent history offenders showed reduced performance compared to offenders without a solvent history and controls). However, they failed to reach statistical significance (See Table 25 for cell means).

Four separate MANCOVAs were performed to examine group (CSA, CNSA, control) differences on the four tasks (eyes open, eyes closed, letter-number sequencing, test of non-verbal intelligence) on alpha1 amplitude (see hypothesis 4) using age as a covariate. Eyes open ($F(8,70) = 2.44$, $p = .02$) and letter-number sequencing ($F(8,70) = 2.53$, $p = .02$) showed significant group differences. Planned contrasts revealed that the control group yielded higher alpha1 EEG amplitude on the eyes-open task compared to the CSA and CNSA groups in anterior and posterior brain regions. There were no differences between the CSA and CNSA groups in alpha1 EEG amplitudes in anterior or posterior sites. Planned contrasts also revealed that the control group showed higher alpha1 EEG amplitude on the letter-number sequencing task in anterior and posterior brain regions compared to the CSA group, and higher alpha1 EEG amplitude on the letter-number sequencing task in posterior brain regions compared to the CNSA group. The CSA group showed decreased alpha1 EEG amplitudes on the letter-number sequencing task in anterior brain regions compared to the CNSA group. Of note, the mean EEG amplitude values for the offenders with a solvent history were lower across all tasks and brain regions compared to offenders without a solvent history and controls, although as indicated, not all of these trends were significant (See Table 26 for cell means).

Discussion

Correctional Versus Control Group

Corrections participants showed lowered amplitudes in bilateral anterior and posterior scalp regions on theta (eyes open, eyes closed) and alpha1 (eyes open, letter number sequencing) EEG activity compared to controls. The group differences tended to be greatest in the posterior scalp regions with left posterior regions showing the lowest amplitude figures. Lower left alpha rhythm is a common finding in EEG data, and is independent of handedness (Fisch, 1999). However, local differences of amplitude (asymmetries) marked by locally decreased EEG production may be indicative of disorders of cortical functioning (Fisch, 1999). Research suggests alpha EEG amplitude decreases as more neurons become involved in task-related processing (McEvoy, Pellouchoud, Smith, & Gevins, 2001). Alpha amplitude also decreases with age from its maximum in childhood (Fisch, 1999). However, there were no statistical differences in age between the groups and including age as a covariate controlled for its influence on this data (Dustman, Shearer, & Emmerson, 1999; Fisch, 1999; Niedermeyer, 1993;). Thus, it appears unlikely that variability in age across groups explains group differences in lower alpha amplitude findings found in the present study.

The regional distribution of theta and alpha1 amplitudes that distinguished the groups in this study appear robust in that they emerged despite the heterogeneous make up of the corrections sample. Statistical power was enhanced in the current study through exclusion of demographic factors that might otherwise produce spurious results such as a history of seizures, and controlling for unforeseen factors such as time of day effects, age, and depression and anxiety. Similarly, the probability of Type I error was

also reduced through the inclusion of the modified Bonferroni correction and by collapsing and averaging the 16 EEG scalp sites into four regional brain areas to reduce the number of dependent variables before statistical analyses. Keppel's adjustment (Cohen, 1977; Keppel, 1992) was used instead of the traditional Bonferroni (Dunn's test) correction, as it is widely regarded as too conservative (McDonald, Seifert, Lorenzet, Givens & Jaccard, 2002).

The functional significance of the theta and alpha amplitude distribution that characterized the corrections sample in this study is unclear, but evidence suggests that theta rhythms are important in learning and memory (Berry & Thompson, 1978) and appear to originate in the hippocampus (Bland, Andersen & Ganes, 1975) a site where degeneration of neurons is associated with increased theta rhythms (Rae-Grant, Blume, Lau, Hachinski, Fisman, & Merskey, 1987). Recent studies have found that the amplitude of theta EEG signals over the frontal regions are enhanced in response to greater working memory demands (Gevins, Smith, McEvoy, & Yu, 1997; Gevins et al., 1998) and tend to increase in a variety of tasks that require sustained attention (Miyata, Tanaka, & Hono, 1990; Yamamoto & Matsuoka, 1990; Gundel & Wilson, 1992; Gevins et al., 1997; Gevins et al., 1998). Such effects on memory demands have also been found for alpha amplitude signals (Herrmann, Sensowski, & Rottger, 2004).

Interestingly, alpha amplitude differences emerged during eyes open and eyes closed conditions, yet the only challenge task to produce significant changes in EEG activity was the letter-number sequencing condition. One explanation for this finding is that the challenge conditions in this study might have obscured group differences rather than delineating them. For instance, it is possible that the challenge tasks may have been

too challenging, eliciting maximal cortical effort across both groups and reducing variability between them.

Nevertheless, the finding of alpha1 amplitude differences between groups on the letter-number sequencing challenge task is consistent with research demonstrating that general memory performance is predicted by alpha activity (Clark et al., 2004). Researcher reports from clinical EEG studies suggest that white matter lesions are associated with increased EEG delta amplitude, while grey matter lesions are related to decreased EEG alpha amplitude (Gloor, Kalabay, & Giard, 1968; Gloor, Ball, & Schaul, 1977) marked by MRI and neuropsychological findings indicative of reduced cognitive performance (Thatcher, Biver, McAlaster, Camacho, & Salazar, 1998). Klimesch, Vogt, and Doppelmayr (2000) and Hummel, Andres, Altenmuller, Dichgans, and Gerloff (2002) suggest that large alpha amplitudes reflect cortical deactivation, while small alpha amplitudes denote cortical activation. Memory performance appears to improve if the cortex is deactivated before a task is performed by decreasing interference with and increasing the attentional resources required for optimally accessing a memory trace (Hanslmayr et al., 2005). Perhaps in our correctional sample, participants lower EEG amplitudes compared to controls might be indicative of gray matter lesions and frontal dysfunction that inhibits cortical deactivation just prior to engaging in memory tasks in this population.

Research has found that sedating drugs, such as nitrous oxide, cause a decrease in amplitude and frequency of alpha rhythm (Ghoneim, 2001;Yamamura, Fukuda, Takeya, Goto, & Furukawa, 1981) and jet fuel sniffers have been shown to produce EEG patterns characterized by low EEG amplitudes compared to controls (Knave et al., 1978). Similar

findings have been reported with EEG amplitude, suggesting that under conditions of sustained performance and high working memory load, high-ability subjects show enhanced frontal theta amplitude (Gevins & Smith, 2000). These findings may partially explain the low amplitude alpha and theta bands found in the correctional participants in the current study. The results are consistent with other findings indicating that low-level exposure to the solvent toluene is associated with decrements in memory test performance, as seen in digit span forward (Chouaniere et al., 2002; Echeverria, Fine, Langolf, Schork, & Sampaio, 1991).

Digits Forward provides an estimate of attention, concentration, and short-term memory capacity and is sensitive to left-hemisphere damage (Lezak, 1995). Digits Backward is sensitive to left-hemisphere frontal lobe damage by nature of the ordering and sequencing aspects of the task (Lezak, 1995). The forward and backward digit span tasks have been used extensively to measure short-term memory in neuropsychological and clinical research (Risberg & Ingvar, 1973).

It is now generally thought that digit span tests involve verbal working memory (Baddeley, 1992) and that such measures decline with age (Parkin & Walter, 1991; Yakota et al., 2000) and the rate of age-related decline in performance is equivalent for digit span forward and backward (Hester, Kinsella, & Ong, 2004). However the functional relationship between digits forward and digits backward remains equivocal and contentious (Hoshi et al., 1999). Researchers have argued that forward and backward digit-span are conceptually distinct tasks utilizing different cognitive abilities and discrete brain areas (Banken, 1985; Griffin & Heffernan, 1983).

Some researchers believe that different neurological processes underlie these two measures, with digits forward involving the preservation of serial orders in time through the use of verbal encoding, while digits backward requires retention and re-positioning of a given serial order into left-right spatial orientations (Rudel & Denckla, 1974). Forward digit span employs attention and short-term memory ability that appears largely independent of the medial temporal lobe hippocampal memory system compared to other verbal memory measures, as lesions in this region do not produce significant deficits on this task (Kolb & Wishaw, 1990). However, left parietal lobe damage can lead to impairments in short-term memory, particularly on forward digit span (Kolb & Wishaw, 1990; Warrington and Weizkrantz, 1973). Backward digit span, which engages working memory, short-term memory and attention appears to involve more hippocampal function, since amnesic patients with hippocampal damage show impairments on tasks that require working memory (Carlson & Sherwin, 1998). The current findings of lower digits forward performance in solvent history corrections (CSA) participants may stem from reduced functioning in areas of the brain other than the hippocampus such as the frontal or parietal lobes.

Others contend that digits forward and digits backward engage a common verbal component (Richardson, 1977). Larrabee and Kane (1986) assert that the digits backward task employs both verbal and visual cognitive, memory and attentional processes. These studies were comprised largely of subjects with brain lesions and central nervous system dysfunction. Newer research suggests that both forward and backward digit span tasks implicate central executive processing for successful task performance (Hester et al., 2004). However, newer research with intact subjects suggests

that the forward digit span task produces significant activation in the right mid-ventrolateral cortex (Brodmann area 47) consistent with previous studies of spatial span (Owen, Evans, & Petrides, 1996; Owen et al., 1999; Owen, 2000; Jonides et al., 1997). Similarly, the backward digit span (DB) task activates the dorsolateral prefrontal cortex of each hemisphere more than the forward digit span (DF) in healthy adult volunteers, and increased scores on the DB task are closely linked to activation of the right dorsolateral prefrontal cortex (Hoshi et al., 1999). The authors suggest that visuospatial imagery is a useful strategy for the DB task (Hoshi et al., 1999; Cabeza & Nyberg, 2000; Cohen, Perlstein, Braver, & Nystrom, 1997; Klingberg, O'Sullivan, & Roland, 1997; Rypma, Prabhakaran, Desmond, Glover, & Gabrieli, 1999) and this is consistent with other research implicating the dorsolateral prefrontal cortex in working memory (Berman, et al., 1995), and findings that verbally mediated tasks tap primarily left-hemisphere functions, while spatial tasks invoke call upon right hemisphere regions (Smith, Jonides, Koeppe, Awh, Schumacher, & Minoshima, 1995; Smith, Jonides, & Koeppe, 1996).

Depression

The correctional participants assessed in the present study were a heterogeneous group that included subjects with depression. Researchers have identified a number of mental health risk factors in Native communities in Canada, including increased risk for family violence, depression, hopelessness, and suicide (Elias & Greyeyes, 1999; Jodoin, 1997; Brant, 1993). The present findings suggest that depression levels reported by Native corrections participants falls within the mild to moderate range, and that overall scores between the corrections and control groups were statistically indistinguishable.

This finding appears consistent with recent research examining mental illness among Caucasian offenders (Hagedorn & Willenbring, 2003). Furthermore, assuming that our measure of depression was valid in this sample of First Nations participants, it is unlikely that the current findings could be better explained by cognitive impairment associated with depression, as there were no differences in reported depression between the groups and levels of depression were not significantly related to the other variables in the analysis. Likewise, if performance differences in our study were related to depression, one would also expect to see differences in TONI performance between solvent history and non-solvent history offenders, as this measure relies heavily on visuospatial processing (Morrow et al., 2002). However, this was not supported in our study.

Anxiety

The findings of the current study suggest that self-reported anxiety levels among Native corrections participants and controls falls within the mild range. This finding is contrasted with the available literature that suggests a high prevalence and negative effect of anxiety problems in aboriginal peoples (Zvolensky et al., 2001), and that anxiety and stress-related problems are commonly recognized as most prominent in treatment settings (Rhodes, Marshal, Attneave, Echohawk, Bjork, & Beiser, 1980). However, because of significant intragroup diversity among First Nation peoples, it may be that our sample of First Nations offenders was culturally distinct from First Nations cultures sampled in other studies (Allen, 1998). Another tenable explanation for the observed findings may be that Native offenders evidence a different level of anxiety compared to Native individuals in the general population due to increased rates of antisocial personality disorder in this population. Consistent with this view, research has found that adult Caucasian offenders

endorse levels of self-reported anxiety on the BAI (Hagedorn & Willenbring, 2003) that is similar to levels found in our sample of Native offenders. Another explanation is that the factor structure of the Beck Anxiety Inventory might load differently on Native people, as it was not designed to measure anxiety in this population. There is a longstanding controversy about the cross-cultural applicability of such concepts as major depression (Kleinman and Good, 1985). There have been few attempts to develop assessment instruments or to investigate the validity of existing ones for use in Indian communities (Somervell, Beals, Kinzie, Boehnlein, Leung, & Manson, 1993). While it is unclear whether the factor structure of the BAI reflected a valid measure of depression in our First Nations sample, developing and validating measures of depression and anxiety specific to First Nations peoples would improve our understanding of these constructs in this population and reduce the equivocal nature of future research findings. Nevertheless, there is little empirical research examining the clinical aspects of anxiety disorders in aboriginal populations (McNeil, Zvolensky, Porter, Rabaliais, McPherson, & Kee, 1997; Trimble, 1990; Zvolensky, McNeil, Porter, & Stewart, 2001).

Recreational Drug Use

Almost all the corrections participants reported a history of multiple drug use that included mostly marijuana, but also cocaine, amphetamine, and heavy alcohol use, and it was unusual to find a participant reporting minimal drug experiences. Even among those who reported a history of solvent abuse there were inter-individual differences in the type of solvent abused. It is unclear what effect if any, drugs like marijuana might have exerted on the present findings. For instance, differences between solvent history inmates vs. non-solvent history inmates might be obscured by extensive marijuana or

cocaine use. For instance, marijuana use has been linked to working memory difficulties (Croft, Mackay, Mills, & Gruzelier, 2001; Ehrenreich et al., 1999; Pope, Gruber, Hudson, Huestis, & Yurgelun-Todd, 2001; Solowij, 1995) and cocaine has been associated with attention and memory impairment as well (Beatty, Katzun, Moreland, & Nixon, 1995; Easton & Bauer, 1996; Gillan et al., 1998; Horner, 1997; Robinson, Heaton, & O'Malley, 1999; Selby & Azrin, 1998).

The current study of the effects of solvent history provide a naturalistic investigation of memory and cognitive performance in corrections users rather than an experimental study where controlled doses of a drug are administered to volunteers with a history of use (Verdejo-Garcia,, Lopez-Torrecillas, Orozco-Gimenez, & Perez-Garcia, 2004). In one naturalistic-like study, Page, Fletcher, and True (1988) found deficits in information processing, attention, and memory in Costa Rican participants with more than 25 years of cannabis use compared to controls matched for socio-demographic characteristics, and was followed-up by a larger prospective study that detected significant impairment in attention and free recall memory that were suggested to be long lasting (Fletcher et al., 1996). Nevertheless, more recent investigations in the neuropsychological performance of cannabis users suggest impairments are likely related to residual effects of the drug as a result of slow excretion rates, rather than long-term effects. For instance, Pope et al. (2001) found only mild urinary THC metabolite related impairments in neuropsychological functioning in cannabis users following one week of abstinence, and no impairments compared to controls after twenty-eight days. Similarly, Grant, Gonzalez, Carey, Natarajan, & Wolfson (2003) conducted a meta-analytic review of recent studies and found that cannabis use did not demonstrate a significant

detrimental effect in several domains of cognitive functioning, with the exception of long-term heavy cannabis use which appeared to be related to mild and selective memory impairment. Considering the findings of the present study that showed THC use (90%) among those with a solvent history is similar to those inmates without a solvent history (100%), if THC use was responsible for the current finding one would expect no difference in cognitive performance between the groups.

It may be argued that the present findings may reflect differences in offender characteristics rather than cognitive differences between the groups. However, there are at least two lines of evidence which suggest that the differences in forward digit-span results obtained in this investigation are not likely to be attributable to factors related to offender characteristics. First, results indicate that corrections participants with solvent use histories show significantly poorer performance on a measure of short-term memory (digit-span forward) compared to corrections participants without solvent histories, thus providing a natural control for offender characteristics. Second, it has already been mentioned that as a group the correctional participants with solvent histories showed trends toward lower mean scores on cognitive ability, memory, and EEG measures compared to non-solvent reporting corrections participants. This trend suggests that the reduced performance of the solvent history group was not likely to be merely a chance finding. Alternatively, one may argue that the reduced performance of the solvent history group might be related to another factor. For instance, those with solvent histories may be slow to learn strategies to enhance their success on the digit-span tests and because digit-span forward preceded digit-span backward in the protocol (the order of the tests was not counterbalanced) this might explain why digit-span backward performance was

not significantly different between corrections participants with and without solvent histories. This concern appears without merit however, as research controlling for order of presentation in large scale multiple trial studies incorporating digit-span forward and backward have not found any influence of counterbalancing on task performance (Hoshi et al., 1999). Furthermore, if the solvent abusing group did in fact exhibit a reduced ability to adopt flexible memory strategies, this would still be a real effect and warrant further investigation.

It is important to consider that good control strategies tend to reduce error variance and lend more credibility to findings and conclusions. The current study controlled for the effects of age, incidence of head injury, and time of day effects - variables that have often been ignored or neglected in previous investigations.

It is important to consider that a number of EEG comparisons in the present study achieved significance at alpha <.05 but were not deemed statistically significant based on our decision to modify the alpha level to control for multiple comparisons. In light of the relatively modest sample sizes involved in this study, and the heterogeneity among the corrections samples the current EEG findings likely reflect a tendency toward Type II errors (i.e., failing to detect EEG differences across spectral values that did, in fact, differentiate the groups). Thus, the inability to identify differences between groups across other conditions (e.g., challenge tasks) and across other EEG bandwidths (e.g., delta and beta) should be regarded as preliminary at this time until similar studies are available.

Limitations

This study is not without some limitations. The correctional participants reporting a solvent history comprised a small sample, and less than half of them reported any recent

solvent abuse activity. Furthermore, as it has previously been stated, First Nations groups may share cultural similarities, but are not culturally uniform (Allen, 1998). Thus, we should be cautious in generalizing results from individuals in this sample to all First Nations people.

Another limitation involves the accuracy of reporting solvent abuse history. Fredlund (1992) reported that 24% of adolescents indicated that they had in the past inhaled a variety of solvents including paints, correction fluid, gasoline, freon, shoe shine, glue, and aerosols. However, when the same adolescents were subsequently interviewed about their inhalant use, half denied any solvent use (Howard et al., 1999). These findings suggest that general queries about unfamiliar or stigmatized drugs such as inhalants and their abuse may lead to under-reporting of actual use (Howard et al., 1999; Pedersen, 1990).

Also, time limitations in this study did not allow for an extended testing period at the correctional facility. If time and resources permitted, the testing period at the correctional facility could have been extended to assist in recruiting a greater number of recent solvent abusing offenders as they were admitted to the facility. A larger sample would also serve to increase statistical power and perhaps help clarify the interpretation of research findings. In addition, the Native control group was comprised mainly of university students, and could have been better matched to the experimental group of adult offenders. The backgrounds of Natives attending university are likely to be different from the backgrounds of Native correctional offenders. To control for health and socioeconomic differences between groups, one could conduct the testing of controls in regions where solvent abuse is problematic, such as remote Native communities.

Furthermore, it would have been useful to incorporate a more thorough battery of cognitive and performance measures in the current study that extend beyond measures of working memory and cognitive ability such as visual spatial processing, visual and auditory memory, executive functioning, attention, and comprehension. However, the length of the testing sessions might make participating in such studies less appealing, particularly from a traditional First Nations perspective and might overextend resources from the cooperating facility. Further investigations would also benefit from careful control of type, amounts, and duration of inhalant use and incorporate participants whose inhalant histories are limited to only one type of solvent. Future directions for research could include examinations of the neuropsychological manifestations of solvent abuse in Native individuals and would benefit from purer samples of abusers like those attending treatment in solvent abuse treatment centers.

One potential problem with averaging scalp sites is that it may mask topographic differences that distinguish subject samples (Pollock et al., 1991). However, including all of the scalp sites into the analysis would have grossly inflated Type I error. Furthermore, no hypotheses were suggested, and no rationale was provided to warrant examination of discrete EEG scalp sites in the study. As an exploratory analysis, the intent was to investigate more regional differences in EEG activity. It appears that the findings reflect weak but consistent theta and alpha1 differences between groups that was probably enhanced by averaging clusters of scalp sites.

Furthermore, the correctional sample consisted of offenders – many of whom it can be assumed meet diagnostic criteria for psychopathy and antisocial personality disorder (Crites & Schuckit, 1979; Dinwiddie et al., 1987) attributes that are much less

likely to characterize a university sample of native students. This makes interpretation of the current findings more challenging, as some of the variance within and between groups is likely to be attributable to the effect of specific solvents, unidentified offender characteristics, socio-economic differences, organic effects due to solvent history, or factors related to frontal dysfunction and antisocial characteristics. Nevertheless, as little is known about the neuropsychological effects of solvents in First Nations peoples, studying such populations within correctional facilities may increase access to this often neglected and rarely studied group (Beauvais, Wayman, Jumper-Thurman, Plested, & Helm, 2002). Further etiological and ethnographic studies of First Nations inhalant abuse are needed to determine whether certain factors might predict use of specific inhalants, extent of inhalant use, relapse rates, protective personality traits, motivations for use, physical and psychological effects of abuse, and factors leading to the cessation and escalation of inhalant use.

Potential Practical Implications

In light of the present findings, it would be important to find ways of using the current results to improve the clinical treatment of solvent abuse. One important strategy, and a major inspiration for this research, was the claim among youth attending a local First Nations solvent abuse treatment facility that they did not believe that solvents produced any damage to their brains (Cameron Dokis, personal communication). As stated previously, our research allows the creation of colourful QEEG topographic "maps" of the brain that can dramatically show changes to the brain that are likely related to damage associated with solvent abuse.

Furthermore, our findings suggest that First Nations corrections participants with a history of solvent abuse show decreased short-term working memory performance and otherwise lower mean values across all memory and cognitive tasks compared to non-solvent abusing corrections and university participants. These findings can be used as part of psychoeducational tools to enhance treatment and better enable solvent abusers to "personalize" these findings with the aim of deterring further abuse (Prichep & John, 1992). Also, documentation of memory and cognitive difficulties could be incorporated into school or vocational planning, such as Individualized Education Plans, to facilitate academic or work success through appropriate teaching and study strategies.

Recent studies have used QEEG to identify specific brain disturbances in cocaine users that predict length of treatment and relapse (e.g., Prichep, Alper, Kowalik, & Rosenthal, 1996), in a manner superior to the usual clinical methods (Noldy & Carlen, 1990). Future research might use QEEG to reveal markers identifying solvent abusers at greatest risk for relapse as well as those who are more relapse-resistant. Such findings could prevent additional brain trauma.

Lastly, many feel that solutions to community problems are best generated from within. To this end, data collected from a particular community showing brain damage from solvents would be more compelling to someone in treatment or to administrators looking to possibly fund local projects or treatment initiatives. For instance, the current work might serve as a foundation for future testing at the regional solvent abuse treatment centre in Thunder Bay.

References

Abas, M A., Sahakian, B. J., & Levy, R. (1990). Neuropsychological deficits and CT scan changes in elderly depressives. Psychological Medicine, 20, 507-520.

Ackerson, L., Dick, R., Manson, S., & Baron, A. (1990). Properties of the inventory to depression and American Indian adolescents. Journal of the American Academy of Child and Adolescent Psychiatry, 29, 601-607.

Aeschbach D, Matthews J. R., Postolache, T.T., Jackson, M.A., Giesen, H.A., & Wehr, T.A. (1999). Two circadian rhythms in the human electroencephalogram during wakefulness. American Journal of Physiology, 277, 1-9

Allen, J. (1998). Personality assessment with American Indians and Alaska Natives: Instrument considerations and service delivery style. Journal of Personality Assessment, 70, 17-42.

Allison, W. M., & Jerrom, D. W. A. (1984). Glue sniffing: a pilot study of the cognitive effects of long-term use. International Journal of the Addictions, 19, 453-458.

Anderson, H. R., MacNair, R. S. & Ramsey, J. D. (1985). Deaths from abuse of volatile substances: A national epidemiological study. British Medical Journal, 290, 403-307.

AoAlousi, L. M. (1989). Pathology of volatile substance abuse: A case report and a literature review. Medicine Science and the Law, 29, 189-202.

Azayem, A., Abdeen, A, Gunaidy, A., & Amin, M. (1991). Deterioration of brain functioning in relapsing heroin addicts. Integrative Psychiatry, 7, 113-119.

Baddeley, A. (1992). Working memory. Science, 255, 556-559.

Baker, E. L., Smith, T. J., & Landrigan, P. J. (1985). The neurotoxicity of industrial solvents. A review of the literature. American Journal of Industrial Medicine, 8, 207-217.

Banken, J. A. (1985). Clinical utility of considering digits forward and digits backward as separate components of the Wechsler adult intelligence scale-revised. Journal of Clinical Psychology, 41, 686-691.

Barcelo, F., & Gale, A. (1997). Electrophysiological measures of cognition in biological psychiatry: Some cautionary notes. International Journal of Neuroscience, 92, 219-240.

Barnes, G. E. (1979). Solvent abuse: A review. International Journal of Addiction, 14, 1-26.

Basso, M. R., & Bornstein, R. A. (1999). Relative memory deficits in recurrent versus first episode major depression on a word list learning task. Neuropsychology, 34, 557-563.

Beals, J., Manson, S. M., Keane, E. M., & Dick, R. W. (1991). Factorial structure of the center for epidemiologic studies – depression scale. Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3, 623-627.

Beals, J., Manson, S. M., Whitesell, N. R., Spicer, P., Novins, D. K., & Mitchell, C. M. (2005). Prevalence of DSM-IV disorders and attendent help-seeking in 2 American Indian reservation populations. Archives of General Psychiatry, 62, 99-108.

Beals, J., Piasecki, J., Nelson, S., Jones, M., Keane, E., Dauphinais, P., Shirt, R. R., Sack, W. H., & Manson, S. M. (1997). Psychiatric disorder among

American Indian adolescents: Prevalence in Northern Plains youth. Journal of the American Academy of Child Adolescent Psychiatry, 36, 1252-1259.

Beats, B. C., Sahakian, B. J., & Levy, R. (1996). Cognitive performance in tests sensitive to frontal lobe dysfunction in the elderly depressed. Psychological Medicine, 26, 591-604.

Beatty, W. W., Katzun, V. M., Moreland, V. J., & Nixon, S. J. (1995). Neuropsychological performance of recently abstinent alcoholics and cocaine abusers. Drug and Alcohol Dependence, 37, 247-253.

Beauvais, F., Wayman, J. C., Jumper-Thurman, P., Plested, B., Helm, H. (2002). Inhalant abuse among American Indian, Mexican American, and non-Latino white adolescents. American Journal of Drug and Alcohol Abuse, 28, 171-187.

Beck, A. T., Brown, G., & Steer, R. A. (1996). Beck depression inventory II manual. San Antonio, TX: The Psychological Corporation.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988) An inventory for measuring clinical anxiety: Psychometric properties. Journal of Consultation and Clinical Psychology 56,893-897

Beiser, M. & Attneave, C. L. (1982). Mental disorders among Native American children: Rates and risk periods for entering treatment. American Journal of Psychiatry, 139, 193-198.

Berman, K. F., Ostrem, J. L., Randolph, C., Gold, J., Goldberg, T. E., Coppola, R., Carson, R. E., Herscovitch, P., & Weinberger, D.R. (1995). Physiological

activation of a cortical network during performance of the Wisconsin card sorting test: a positron emission tomography study. Neuropsychologia, 33, 1027 – 1046.

Berry, G. J., Heaton, R., & Kirby, M. W. (1976). Neuropsychological assessment of chronic inhalant abusers: A preliminary report. University of Colorado Medical Center: Denver. In Smith, D. B., Gatchel, R. J., Korman, M., & Saffer, S. Eeg and autonomic responding to verbal, spatial and emotionally arousing tasks: Differences among adults, adolescents and inhalant abusers. Biological Psychology, 9, 189-200.

Berry, S. D., & Thompson, R. F. Prediction of learning rate from the hippocampal electroencephalogram. Science, 200, 1298-1300.

Bland, B. H., Andersen, P., & Ganes, T. (1975). Two generators of hippocampal theta in rabbits. Brain Research, 94, 199-218.

Boeckelmann, I., Darius, S., McGauran, N., Robra, B. P., Peter, B., & Pfister, E. A. (2002). The psychological effects of exposure to mixed organic solvents on car painters. Disability and Rehabilitation, 24, 455-461.

Boeckx, R. I., Posh, B., & Cohen, F. J. (1977). Gasoline sniffing and tetraethyl lead poisoning in children. Pediatrics, 60, 140-145.

Brady, M., & Torzillo, P. (1994). Petrol sniffing down the track. The Medical Journal of Australia, 160, 176-177.

Brandt, E. (1984). The cognitive functioning of American Indian children: A critique of McShane and Plas. School Psychology Review, 13, 74-82.

Brant, C. (1993). Suicide in Canadian Aboriginal peoples: Causes and prevention. In: The Path to Healing: Report of the National Round Table on Aboriginal Health and

Social Issues – Royal Commission on Aboriginal Peoples. 55-71. Ottawa: Ministry of Supply and Services Canada.

Brown, L., Sherbenou, R. J., & Johnsen, S. K. (1990). Test of Nonverbal Intelligence-Second Edition (TONI-2). Austin, TX: Pro-Ed.

Bruhn, P., Arlien-Soborg, P., Gyldensted, C., & Christiansen, B. L. (1981). Prognosis in chronic toxic encephalopathy. Acta Neurologica Scandinavica, 64, 259-272.

Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A., & Kaemmer, B. (1989). Manual for the restandardized Minnesota multiphasic personality inventory: MMPI-2. Minneapolis: University of Minnesota Press.

Byrne, A., Kirby, B., Zibin, T., & Enswinger, S. (1991). Psychiatric and neurological effects of chronic solvent abuse. Canadian Journal of Psychiatry, 36, 735-738.

Cabeza, R., & Nyberg, L. (2000). Imaging cognition II: An empirical review of 275 PET and fMRI studies. Journal of Cognitive Neuroscience, 12, 1-47.

Campbell, S. B., & Cohn, J. F. (1991). Prevalence and correlates of postpartum depression in first-time mothers. Journal of Abnormal Psychology, 100, 594-599.

Carlson, L. E., & Sherwin, B. (1998). Steroid hormones, memory and mood in a healthy elderly population. Psychoneuroendocrinology, 23, 583-603.

Cassens, G. Wolfe, L., & Zola, M. (1990). The neuropsychology of depressions. Journal of Neuropsychiatry and Clinical Neurosciences, 2, 202-213.

Chalmers, E. M. (1991). Volatile substance abuse. Medical Journal of Australia, 154, 269-274.

Chouaniere, D., Wild, P., Fontana, J. M., Hery, M., Fournier, M., Baudin, V., Subra, I., Rousselle, D., Toamain, J. P., Saurin, S, & Ardiot, M. R. (2002). Neurobehavioral disturbances arising from occupational toluene exposure. American Journal of Industrial Medicine, 41, 77-88.

Clark, R. C., Veltmeyer, M. D., Hamilton, R. J., Simms, E., Paul, R., Hermens, D., & Gordon, E. (2004). Spontaneous alpha peak frequency predicts working memory performance across the age span. International Journal of Psychophysiology, 53, 1-9.

Clayton-Olfield, C. & Keefe, S. (1999). Assessing stereotypes about the Innu of Davis Inlet, Labrador. Canadian Journal of Behavioural Science, 31, 86.

Cohen, J. (1977). Statistical power analysis for the behavioral sciences. New York: Academic Press

Cohen, J. D., Perlstein, W. M., Braver, T. S., & Nystrom, L. E. (1997). Temporal dynamics of brain activation during a working memory task. Nature, 386, 604-608.

Compton, W. M, Cottler, L. B., Dinwiddie, S. H., Sptiznagel, E. L., Mager, D. E., and Asmus, G. (1994). Inhalant use: Characteristics and predictors. The American Journal on Addiction, 3, 263-272.

Convit, A., Czobor, P., Volavka, J. (1985). Lateralized abnormality in the juvenile delinquents and adolescent psychiatric inpatients. Journal of the American Academy of Child Psychiatry, 24, 310-315.

Costello, J. E., Compton, S. N., Keeler, G., & Angold, A. (2003). Relationships between poverty and psychopathology. Journal of the American Medical Association, 15, 2023-2029.

Crites, J., & Schuckit, M. A. (1979). Solvent misuse in adolescents at a community alcohol center. Journal of Clinical Psychiatry, 40, 39-43.

Croft, R. J., Mackay, A. J., Mills, A. T. D., & Gruzelier, J. G. D. (2001). The relative contributions of ecstasy and cannabis to cognitive impairment. Psychopharmacology, 153, 373-379.

Devers, R., Bradley-Johnson, S., & Johson, C. M. (1994). The effect of token reinforcement on WISC-R performance for fifth through ninth grade American Indians. Psychological Record, 44, 441-449.

Dinwiddie, S. H. (1994). Abuse of inhalants: a review. Addiction, 89, 925-939.

Dinwiddie, S. H., Reich, T. & Cloninger, C. R. (1990). Solvent abuse and psychiatric co-morbidity. British Journal of Addiction, 85, 1647-1656.

Dinwiddie, S. H., Zorumski, C. F., & Rubin, E. H. (1987). Psychiatric correlates of chronic solvent abuse. Journal of Clinical Psychiatry, 48, 334-337.

Dittmer, D. K., Jhamandas, J. H., & Johnson, E. S. (1993). Glue-sniffing neuropathies. Canadian Family Physician, 39, 1965-1971.

Duclos, C. W., LeBeau, W., & Elias, G. L. (1994). American Indian adolescent suicidal behavior in detention environments: Cause for continued basic and applied research. American Indian and Alaska Native Mental Health Research, 4, 189-214.

Dustman, R. E., Shearer, D. E., & Emmerson, R. Y. (1999). Life-span changes in EEG spectral amplitude, amplitude variability and mean frequency. Clinical Neurophysiology, 110, 1399-1409.

Easton, C., & Bauer, L. O. (1996). Neuropsychological correlates of urine toxicology results. Progress in Neuropsychopharmacology and Biological Psychiatry, 20, 969-982.

Echeverria, D., Fine, L., Lnagolf, G., Schork, T., & Sampaio, C. (1991). Acute behavioural comparisons of toluene and ethanol in human subjects. British Journal of Industrial Medicine, 48, 750-761.

Edling, C., Ekberg, K., Ahlborg, G., Alexandersson, R., Barregard, L., Ekenvall, L., Nilsson, L., & Svensson, B. G. (1990). Long-term follow up of workers exposed to solvents. British Journal of Industrial Medicine, 47, 75-82.

Edminster, S. C., & Bayer, M. J. (1985). Recreational gasoline sniffing: Acute gasoline intoxication and latent organolead poisoning: Case reports and literature review. Journal of Emergency Medicine, 3, 365-370.

Ehrenreich, H., Rinn, T., Kunert, H. J., Moeller, M. R., Poser, W., Schilling, L., Gigerenzer, G., & Hoehe, M. R. (1999). Specific attentional dysfunction in adults following early start of cannabis use. Psychopharmacology, 142, 295-301.

Elias, J.W., & Greyeyes, D. (1999). Report on an environmental scan of mental health services in First Nations communities in Canada. Unpublished manuscript.

Elofsson S., Gamberale F., Hindmarsh T., Iregren A., Isaksson A., Johnsson I., Knave B., Lydahl E., Mindus P., Persson H., Philipson B., Steby M., Struwe

G., Soderman E., Wennberg A., & Widen L. (1980). Exposure to organic solvents. A cross-sectional epidemiologic investigation on occupationally exposed car and industrial spray painters with special reference to the nervous system. Scandinavian Journal of Work and Environmental Health, 6, 239-273.

Espeland, K. (1993). Inhalant abuse: assessment guidelines. Journal of Psychosocial Nursing, 31, 11-14.

Fazel, S., & Danesh, J. (2002). Serious mental disorder in 23000 prisoners: a systematic review of 62 surveys. Lancet, 359, 545-550.

Filley, C. M., Heaton, R. K., & Rosenberg, N. L. (1990). White matter dementia in chronic toluene abuse. Neurology, 40, 532-534.

Fisch, B. J. (1999). Fisch and Spehlmann's EEG primer. (3rd Ed.). New York, NY: Elsevier.

Fisch, B. J., & Pedley, T. A. (1989). The role of quantitative topographic mapping or 'neurometrics' in the diagnosis of psychiatric and neurological disorders: The cons. Electroencephalography and Clinical Neurophysiology, 73, 5-9.

Fletcher, J. M., Page, B., Francis, D. J., Copeland, K., Naus, M. J., Davis, C. M., Morris, R., Krauskopf, D., & Satz, P. (1996). Cognitive correlates of long-term cannabis use in Costa Rican men. Archives of General Psychiatry, 53, 1051-1057.

Fornazzari, L., Wilkinson, D. A., Kapur, B. M., & Carlson, P. L. (1983). Cerebellar cortical and functional impairment in toluene abusers. Acta Neurologica Scandinavica, 67, 319-329.

Frost, J. D., Kellaway, P., Hrachovy, R.A., Glaze, D.G., & Mizrahi, E.M. (1986). Changes in epileptic spike configuration associated with attainment of seizure control. Annals of Neurology, 20, 732-726.

Gevins,A., & Smith, M. E. (2000). Neurophysiological measures of working memory and individual differences in cognitive ability and cognitive style. Cerebral Cortex, 10, 829-839.

Gevins, A. S., Smith, M. E., McEvoy, L., & Yu., D. (1997). High resolution EEG mapping of cortical activation related to working memory: Effects of task difficulty, type of processing, and practice. Cerebral Cortex, 7, 374-385.

Gevins, A., Smith, M. E., Leong, H., McEvoy, L., Whitfield, S., Du, R., & Rush, G. (1998). Monitoring working memory load during computer-based tasks with EEG pattern recognition methods. Human Factors, 40, 79-91.

Ghoneim, M. M. (2001). Nitrous oxide effects on EEG and awareness. Best Practice & Research Clinical Anesthesiology, 15, 397-407.

Gillen, R. W., Kranzler, H. R., Bauer, L. B., Burleson, J. A., Samarel, D., & Morrison, D. J. (1998). Neuropsychological findings in cocaine-dependent outpatients. Progress in Neuropsychopharmacology and Biological Psychiatry, 22, 1061-1076.

Grabaski, D. A. (1961). Toluene sniffing producing cerebellar degeneration. American Journal of Psychiatry, 118, 461-462.

Grant, I., Gonzalez, R., Carey, C. L., Natarajan, L., & Wolfson, T. (2003). Non-acute (residual) neurocognitive effects of cannabis use: A meta analytic

review. Journal of the International Neuropsychological Society, 9, 679-689.

Green, R. L., Robin, R. W., Albaugh, B., Caldwell, A., & Goldman, D. (2003). Use of the MMPI-2 in American Indians: II. Empirical correlates. Psychological Assessment, 15, 360-369.

Griffin, P. T., & Heffernan, A. (1983). Digit span, forward and backward: Separate and unequal components of the WAIS digit span. Perceptual and Motor Skills, 56, 335-338.

Grilly, D. M. (2002). Drugs and human behavior. (4th ed.). Boston, MA: Allyn and Bacon.

Gundel, A., & Wilson, G. F. (1992). Topographical changes in the ongoing EEG related to the difficulty of mental tasks. Brain Topography, 5, 17-25.

Hakkola, M. (1994). Neuropsychological symptoms among tanker drivers with exposure to solvents. Occupational Medicine, 44, 243-246.

Hanslmayr, S., Klimesch, W., Sauseng, P., Gruber, W., Doppelmayr, M., Freundberger, R., & Pecherstorfer, T. (2005). Visual discrimination performance is related to decreased alpha amplitude but increased phase locking. Neuroscience Letters, 375, 64-68.

Hayashi, M., Sato, K., & Hori, T. (1994). Ultradian rhythms in task performance, self-evaluation, and EEG activity. Perceptual & Motor Skills, 29, 791-800.

Herrmann, C. S., Sensowski, D., & Rottger, S. (2004). Phase-locking and amplitude modulations of EEG alpha: Two measures reflect different

cognitive processes in a working memory task. Experimental Psychology, 51, 311-318.

Hester, R., Kinsella, G. J., & Ong, B. (2004). Effect of age on forward and backward span tasks. Journal of International Neuropsychological Society, 10, 475-481.

Holmes, J. T., Filley, C. M., & Rosenberg, N. L. (1986). Neurologic sequelae of chronic substance abuse. Neurology, 36, 698-702.

Horner, M. D. (1997). Cognitive functioning in alcoholic patients with and without cocaine dependence. Archives of Clinical Neuropsychology, 12, 667-676.

Hoshi, Y., Oda, I, Wada, Y., Ito, Y., Yamashita, Y., Oda, M., Ohta, K., Yamada, Y., & Tamura, M. (1999). Visuospatial imagery is a fruitful strategy for the digit span bacward task: A study with near-infrared optical tomography. Cognitive Brain Research, 9, 339-342.

Howard, M. O., Walker, R. D., Walker, P. S., Cottler, L. B., & Compton, W. M. (1999). Inhalant use among urban American Indian youth. Addiction, 94, 83-95.

Hummel, F., Andres, F., Altenmuller, E., Dichgans, J.,&Gerloff, C. (2002). Inhibitory control of acquired motor programmes in the human brain. Brain, 125, 4040-420.

Jacobs, A., & Ghodse, A. (1988). Delinquency and regular solvent abuse: An unfavourable combination? British Journal of Addiction, 83, 965-968.

Jensen, A. (1980). Bias in mental testing. New York: Free Press.

John, R. E. (1990). Principles of neurometrics. American Journal of EEG technology, 30, 251-266.

John, E. R., Prichep, L. S., & Almas, M. (1992). Subtyping of psychiatric patients by cluster analysis of QEEG. Brain Topography, 4, 321-326.

John, E.R., Prichep, L.S., Friedman, J., & Easton, P. (1989) Neuro-metric topographic mapping of EEG and evoked potential features: Application to clinical diagnosis and cognitive function. In Maurer, K. (Eds.) Topo-graphic brain mapping of EEG and evoked potentials. Berlin: Springer-Verlag.

John, R. E., Prichep, L. S., Fridman, J., & Easton, P. (1988). Neurometrics: Computer assisted differential diagnosis of brain dysfunctions. Science, 293, 162-169.

Jones, M. C., Dauphinais, P., Sack. W. H., Somervell, P. (1997). Trauma related symptomatology among American Indian adolescents. Journal of Traumatic Stress, 10, 163-173.

Jonides, J., Smith, E.E., Koeppe, R. A., Awh, E., Minoshima, S., & Mintun, M. A. (1993). Spatial working memory in humans as revealed by PET. Nature, 363, 623-625.

Jonkman, E. J., Poortvliet, D.C., Veering, M. M., de Weerd, A. W., & John, E. R. (1985). The use of neurometrics in the study of patients with cerebral ischemia. Electroencephalograpy and Clinical Neurophysiology, 61, 333-341.

Kelly, T. W. (1975). Prolonged cerebellar dysfunction associated with paint-sniffing. Pediatrics, 56, 605-606.

Kelly, J. F.,McKellar, J. D., & Moos, R. (2003). Major depression in patients with substance use disorders: Relationship to 12-step self-help involvement and substance use outcomes. Addiction, 98, 499–508.

Keppel, G. (1991). Design and analysis: A researchers handbook. New Jersey: Prentice Hall.

King, M. D., Day, R. E., Oliver, J. S., Lush, M. & Watson, J. M. (1981). Solvent encephalopathy. British Medical Journal, 283, 663-665.

Kinzie, J. D., Leung, P. K., Boehnlein, J., Matsunaga, D., Johnston, R., Manson, S. M., Shore, J. H., Heinz, J., Williams, M. (1992). Psychiatry epidemiology of an Indian village: A 19-year replication study. Journal of Nervous and Mental Disorders, 180, 33-39.

Klimesch, W., Schimke, H., Ladurner, G. & Pfurtscheller, G. (1990). Alpha frequency and memory performance. Journal of Psychophysiology, 4, 381-390.

Klimesch, W., Vogt, F., & Doppelmayr, M. (2000). Interindividual differences in alpha and theta power reflect memory performance. Intelligence, 27, 347-362.

Klingberg, T., O'Sullivan, B. T., & Roland, P. E. (1997). Bilateral activation of fronto-parietal networks by incrementing demand in a working memory task. Cerebral Cortex, 7, 465-471.

Knave, B., Anshelm-Olsen, B., Elofsson, S., Gamberale, F., Isaksson, A., & Mindus, P. (1978). Long-term exposure to jet fuel II. A cross-sectional epidemiological investigation on occupationally exposed industry workers

with special reference to the nervous system. Scandinavian Journal of Work and Environment Health, 4, 19-45.

Larrabee, G. J., & Kane, R. L. (1986). Reversed digit repetition involves visual and verbal processes. International Journal of Neuroscience, 30, 11-15.

Lawson, J. S., Adams, S. J., Brunet, D. G., Criollo, M., Galin, H., & MacCrimmon, D. J. (1998). Do QEEG topographic map data have a multivariate normal distribution? Implications for test of individual maps. Brain Topography, 10, 211-219.

Lazar, R. B., Ho, S. U., Melvin, O. & Dagrasrani, A. N. (1983). Multifocal central nervous system damage caused by toluene abuse. Neurology, 33, 1337-1340.

Lewis, J. D., Moritz, D., & Merlis, J. P. (1981). Long-term toluene abuse. American Journal of Psychiatry, 138, 368-370.

Lewis, D. O., Pincus, J. H., Lovely, R., Spitzer, E., & Moy, E. (1987). Biopsychosocial characteristics of matched samples of delinquents and nondelinquents. Journa of the Academy of Child Adolescent Psychiatry, 26, 744-752.

Lezak, M. D. (1995). Neurospsychological assessment. (3rd Ed.). New York: Oxford University Press.

Lindgren, M., Osterberg, K., Orbaek, P., & Rosen, I. (1997). Solvent-induced toxic encephalopathy: Electrophysiological data in relation to neuropsychological findings. Journal of Clinical and Experimental Neuropsychology, 19, 772-783.

Linz, D. H., de Garmo, P. L., Morton, W. E., Wiens, A. N., Coull, B. M., & Maricle, R. A (1986). Organic solvent-induced encephalopathy in industrial painters. Journal of Occupational Medicine, 28, 119-125.

Lockhart, W. H., & Lennox, M. (1983). The extent of solvent abuse in a regional secure unit sample. Journal of Adolescence, 6, 43-55.

Lolin, Y. (1989). Chronic neurological toxicity associated with exposure to volatile substances. Human Toxicity, 8, 293-300.

Manson, S., Ackerson, L., Dick, R., Baron, A., & Fleming, C. (1990). Depressive symptoms among American Indian adolescents: Psychometric characteristics of the Center for Epidemiologic Studies Depression Scale (CES-D). Psychological Assessment, 2, 231-237.

May, P. A., & Van Winkle, N. (1994). Indian adolescent suicide: The epidemiological picture in New Mexico. American Indian Alaska Native Mental Health Research, 4, 2-23.

McDonald, R. A., Seifert, C. F., Lorenzet, S. J., Givens, S., & Jaccard, J. (2002). The effectiveness of methods for analysing multivariate factorial data. Organizational Research Methods, 5, 255-274.

McEvoy, L. K., Pellouchoud, E., Smith, M.E., & Gevins, A. (2001). Neurophysiological signals of working memory in normal aging. Cognitive Brain Research, 11, 363-376.

McGarvey, E. L., Canterbury, R. J., & Waite, D. (1996). Delinquency and family problems in incarcerated adolescents with and without a history of inhalant use. Addictive Behaviors, 21, 537-542.

McNeil, D. W., Zvolensky, M. J., Porter, C., Rabaliais, A., McPherson, T., & Kee, M. (1997). Anxiety in American Indian and Alaska Natives: identification and treatment. Indian Health Service Primary Care Provider, 22, 181–185.

McShane, D. A. (1988). An analysis of mental health research with American Indian youth. Journal of Adolescents, 11, 87-116.

McShane, D. A., & Plas, J. M. (1984). The cognitive functioning of American Indian children: Moving from the WISC to the WISC-R. School Psychology Review, 13, 61-73.

Metrick, S. A., & Brenner, R. P. (1982). Abnormal brainstem auditory evoked potentials in chronic paint sniffers. Annals of Neurology, 12, 553-556.

Milin, R. Halika, J. A., Meller, J. E., & Morse, C. (1991). Psychopathology among substance abusing juvenile offenders. Journal of the American Academy of Child and Adolescent Psychiatry, 30, 569-574.

Miyata, Y., Tanaka, Y., & Hono, T. (1990). Long term observation on Fm-theta during mental effort. Neuroscience, 16, 145-148.

Morrow, L. A. (2002). Comparison of neuropsychological test scores between men and women with prior exposure to organic solvents. Applied Neuropsychology, 9, 240-243.

Morrow, L., Gibson, C. Bagovich, G., Stein, L., Condray, R. & Scott, A. (2000). Increased anxiety and depression disorders in persons with organic solvent exposure. Psychosomatic Medicine, 62, 746-750.

Morrow, L. A., Kamis, H., & Hodgson, M. J. (1993). Psychiatric symptomatology in persons with organic solvent exposure. Journal of Consulting and Clinical Psychology, 61, 171-174.

Morrow, L. A., Robin, N., Hodgson, M. J., Kamis, H. (1992). Assessment of attention and memory efficiency in persons with solvent exposure. Neuropsychologia, 30, 911-922.

Morrow, L. A., Stein, L., Bagovich, G. R., Condray, R., Scott, A. (2001). Neuropsychological assessment, depression, and past exposure to organic solvents. Applied Neuropsychology, 8, 65-73.

Morton, H. G. (1987). Occurrence and treatment of solvent abuse in children and adolescents. Pharmacology and Therapeutics, 33, 449-469.

Neligh, G. (1990). Mental health programs for American Indians: Their logic, structure, and functions. American Indian and Alaska Native Mental Health Research, 3, 24-32.

Neuwer, M. C. (1996). Quantitative EEG analysis in clinical settings. Brain Topography, 8, 201-208.

Newer, M. R. (2002). Clinical use of EEG. Clinical Neurophysiology, 114, 2225.

Noldy, N. E., & Carlen, P.L. (1990). Acute, withdrawal, and chronic alcohol effects in man: Event-related potential and quantitative EEG techniques. Annals of Medicine, 22, 333-339.

Noldy, N. E., Santos, C.V., Politzer, N., Blair, R.D.G., & Carlen, P. L. (1994). Quantitative EEG changes in cocaine withdrawal: Evidence for long-term CNS effects. Neuropsychobiology, 30, 189-196.

Owen, A. M. (2000). The role of the lateral frontal cortex in mnemonic processing: the contribution of functional neuroimaging. Experimental Brain Research, 133, 33-43.

Owen, A. M., Evans, A. C., & Petrides, M. (1996). Evidence for a two-stage model of spatial working memory processing within the lateral frontal cortex: a positron emission tomography study. Cerebral Cortex, 6, 31-38.

Owen, A. M., Herrod, N. J., Menon, D. K., Clark, J. C., Downey, S. P., Carpenter, T. A., Minhas, P. S., Turkheimer, F. E., Williams, E. J., Robbins, T. W., Sahakian, B. J., Petrides, M., & Pickard, J. D. (1999). Redefining the functional organisation of working memory processes within human lateral prefrontal cortex. European Journal of Neurosciences, 11, 567-574.

Parkin, A. J. & Walter, B.M. (1991). Aging, short-term memory and frontal dysfunction. Psychobiology, 19, 175-179.

Pederson, W. (1990). Reliability of drug use responses in a longitudinal study. Scandinavian Journal of Psychology, 31, 28-33.

Pillmann, F., Rohde, A., Ullrich, S., Draba, S., Sannemuller, U., Marneros, A. (1999). Violence, criminal behavior, and the EEG: Significance of left hemisphere focal abnormalities. Journal of Neuropsychiatry and Clinical Neurosciences, 11, 454-457.

Poklis, A., & Burkett, C. D. (1977). Gasoline sniffing: A review. Clinical Toxicology, 11, 35-41.

Pollock, V., E., Schneider, L. S., & Lyness, S. A. (1991). Reliability of topographic quantitative EEG amplitude in healthy late-middle aged and elderly subjects. Electroencephalography, and Clinical Neurophysiology, 79, 20-26.

Pollock, V. E., Schneider, L. S., Zemansky, M. F., Gleason, R. P., & Pawluczyk, S. (1992). Topographic quantitiative EEG amplitude in recovered alcoholics. Psychiatry Research: Neuroimaging, 45, 25-32.

Pope, H. G., Gruber, A. J., Hudson, J. L., Huestis, M. A., & Yurgelun-Todd, D. (2001). Neuropsychological performance in long-term cannabis users. Archives of General Psychiatry, 58, 909-915.

Prichep, L., Alper, K., Kowalik, S., Merkin, H., MeeLee, T., John, E. R., & Rosenthal, M. S. (1996). Quantitative electroencephalographic characteristics of crack cocaine dependence. Biological Psychiatry, 40, 986-993.

Prichep, L., Alper, K., Kowalik, S., & Rosenthal, M. (1996). Neurometric QEEG studies of crack cocaine dependence and treatment outcome. Journal of Addictive Diseases, 15, 39-54.

Rae-Grant, A., Blume, W., Lau, C., Hachinski, V. C., Fisman, M., & Merskey, H. (1987). The electroencephalogram in Alzheimer-Type dementia. Archives of Neurology, 44, 50-54.

Raimo, E. B., and Schuckit, M. A. (1998). Alcohol dependence and mood disorders. Addictive Behaviors, 23, 933–946.

Remington, G., & Hoffman, B. F. (1984). Gas sniffing as a form of substance abuse. Canadian Journal of Psychiatry, 29, 31-35.

Richardson, J. T. E. (1977). Functional relationship between forward and backward digit repetition and a non-verbal analogue. Cortex, 13, 317-320.

Risberg, J., & Ingvar, D. H (1973). Patterns of activation in the grey matter of the dominant hemisphere during memorizing and reasoning. A study of regional cerebral blood flow changes during psychological testing in a group of neurologically normal patients. Brain, 96, 737-756.

Roberts, R. E., Lewinsohn, P. M., & Seeley, J. R. (1991). Screening for adolescent depression: a comparison of depression scales. Journal of the American Academy of Child and Adolescent Psychiatry, 30, 58-66.

Robin, R. W., Long, J. C., Rasmussen, J. K., Albaugh, B., Goldman, D. (1998). Relationship of binge drinking to alcohol dependence, other psychiatric disorders, and behavioural problems in an American Indian tribe. Alcoholism Clinical and Experimental Research, 22, 518-523.

Robinson, J. E., Heaton, R. K., & O'Malley, S. (1999). Neuropsychological functioning in cocaine abusers with and without alcohol dependence. Journal of the International Neuropsychological Society, 5, 10-19.

Roemer, R. A., Cornwell, A., Dewart, D., Jackson, P., & Ercegovac, D. V. (1995). Quantitative electroencephalographic analyses in cocaine-preferring polysubstance abusers during abstinence. Psychiatry Research: Neuroimaging, 58, 247-257.

Roemer, R., Shagass, C., Dubin, W., Joffe, R., & Katz, R. (1991). Relationship between pretreatment electroencephalographic coherence measures and

subsequent response to electroconvulsive therapy: A preliminary study. Neuropsychobiology, 24, 121-124.

Ron, M. A. (1986). Volatile substance abuse: A review of possible long-term neurological, intellectual, and psychiatric sequelae. British Journal of Psychiatry, 148, 235-246.

Rosenberg, N. L., Spitz, M. C., Fillry, C. M., Davis, K. A., & Shaumberg, H. H. (1988). Central nervous system effects of chronic toluene abuse: Clinical, brainstem evoked response and magnetic imaging studies. Neurotoxicology and Teratology, 10, 489-495.

Rudel, R. G., & Denckla, M. B. (1974). Relation of forward and backward digit repetition to neurological impairment in children with learning disabilities. Neuropsychologia, 12, 109-118.

Rumbaut, R. (1994). The crucible within: Ethnic identity, self-esteem, and segmented assimilation among children of immigrants. International Migration Review, 28,748-794.

Rypma, B., Prabhakaran, V., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. E. (1999). Load-dependent roles of frontal brain regions in the maintenance of working memory. Neuroimage, 9, 216-226.

Sattler, J. M. (1988). Assessment of children. (3rd ed.). San Diego, CA.

Scarr, S. (1981). Testing for children: Assessment and the many determinants of intellectual competence. American Psychologist, 36, 1159-1166.

Schatzberg, A., Elliot, G., Lerbinger, J., Marcel, B., & Duffy, F. (1986). Topographic mapping in depressed patients. In Duffy, F. (Ed.) Topographic mapping of brain electrical activity. Boston: Butterworth.

Schultz, C. G., Chilcoat, H. D., & Anthony, J. C. (1994). The association between sniffing inhalants and injecting drugs. Comprehensive Psychiatry, 35, 99-105.

Selby, M. J. & Azrin, R. L. (1998). Neuropsychological functioning in drug abusers. Drug and Alcohol Dependence, 50, 39-45.

Seppalainen, M. & Antti-Poika, M (1983). The course of electrophysiological findings in patients with solvent poisoning. Scandanavian Journal of Work and Environmental Health, 9, 15-24.

Shore, J. H., & Manson, S. M. (1981). Cross-cultural studies of depression among American Indians and Alaska Natives. White Cloud Journal, 2, 5-12.

Skuse, D., & Burrell, S. (1982). A review of solvent abusers and their management by a child psychiatric out-patient service. Human Toxicology, 1, 321-329.

Slaughter, B., Fann, J. R., & Ehde, D. (2003). Traumatic brain injury in a county jail population: prevalence, neuropsychological functioning and psychiatric disorders. Brain Injury, 17, 731-741.

Smart, R. G. (1986). Solvent use in North America: aspects of epidemiology, prevention and treatment. Journal of Psychoactive Drugs, 18, 87-96.

Smith, E. E., Jonides, J., Koeppe, R. A., Awh, E., Schumacher, E. H., & Minoshima, S. (1995). Spatial versus object working memory: PET investigations. Journal of Cognitive Neuroscience, 7 337-356.

Smith, E. E., Jonides, J., & Koeppe, R. A. (1996). Dissociating verbal and spatial working memory using PET. Cerebral Cortex, 6, 11-20.

Solowij, N. (1995). Do cognitive impairments recover following cessation of cannabis use? Life Sciences, 56, 2119-2126.

Somervell, P. D., Beals, J., Kinzie, J. D., Boehnlein, J., Leung, P., & Manson, S. M. (1993). Criterion validity of the center for epidemiologic studies depression scale in a population sample from an American Indian village. Psychiatry Research, 47, 255-266.

Sterman, M.B., & Kaiser, D.A. (2001). Comodulation: A new QEEG analysis metric for assessment of structural and functional disorders of the CNS. Journal of Neurotherapy, 4, 73-83.

Sterman, M.B., Kaiser, D.A. & Veigel, B. (1996) Spectral analysis of event-related EEG responses during short-term memory performance. Brain Topography, 9, 21-30.

Thatcher, R., Walker, R., Gerson, I., & Geisler, F. (1989). EEG discriminant analyses of mild head trauma. Electroencephalograpy and Clinical Neurophysiology, 73, 94-106.

Thatcher, R. W., Biver, C., McAlaster, R., Camacho, M., & Salazar, A. (1998). Biophysical linkage between MRI and EEG amplitude in closed head injury. Neuroimage, 7, 352-367.

Thrane, L. E., Whitbeck, L. B., Hoyt, D. R., & Shelley, M. C. (2004). Comparing three measures of depressive symptoms among American Indian adolescents. American Indian and Alaska Native Mental Health Research, 11, 20-42.

Tsushima, W. T. & Towne, W. S. (1977). Effects of paint sniffing on neuropsychological test performance. Journal of Abnormal Psychology, 86, 402-407.

Trimble, J. E. (1990). Application of psychological knowledge for American Indians and Alaska Natives. The Journal of Training and Practice in Professional Psychology, 4, 45–63.

Valpey, R., Sumi, S. M., Copass, M. K., & Goble, G. J. (1978). Acute and chronic progressive encephalopathy due to gasoline sniffing. Neurology, 28, 507-510.

Verdejo-Garcia, A., Lopez-Torrecillas, F., Orozco-Gimenez, C., & Perez-Garcia, M. (2004). Clinical implications and methodological challenges in the study of the neuropsychological correlates of cannabis, stimulant, and opioid abuse. Neuropsychological Review, 14, 1-41.

Wardman, D. & Khan, N. (2004). Antidepressant medication use among First Nations peoples residing within British Columbia. American Indian and Alaskan Native Mental Health Research, 11, 20-42.

Warrington, E. K., & Weizkrantz, L. (1973). An analysis of short-term and long-term memory defects in man. In: Deutsch, J. A. (Ed.) The physiological basis of memory. Academic Press: New York.

Wechsler, D. (1997) Wechsler adult intelligence scale – III. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (1997) Wechsler Memory Scale-Third Edition. San Antonio: The Psychological Corp.

Weissman, M. M., Bland, R., Joyce, P. R., Newman, S., Wells, J. E., & Wittchen, H. U. (1993). Sex differences in rates of depression: Cross cultural perspectives. Journal of Affective Disorders, 29, 77-84.

Whitbeck, L., B., Hoyt, D., McMorris, B., Chen, X., & Stubben, J. (2001). Perceived discrimination and early substance abuse among American Indian children. Journal of Health and Social Behavior, 42, 405-424.

White, R. F., Proctor, S. P. (1997). Solvents and neurotoxicity. The Lancet, 349, 1239-1243.

Wilcox, H., Field, T., Prodromidis, M., & Scafidi, F. (1998). Correlations between BDI and CES-D in a sample of adolescent mothers. Adolescence, 33, 565-574.

Wong, M. T., Lumsden, J., Fenton, G. W., & Fenwick, P. B. (1994). Electroencephalography, computed tomography and violence ratings of male patients in a maximum-security mental hospital. Acta Psychiatrica Scandinavica, 90, 97-101.

Yamamoto, S., & Matsuoka, S. (1990). Topographic EEG study of visual display terminal VDT performance with special reference to frontal midline theta waves. Brain Topography, 2, 257-267.

Yamamura, T., Fukuda, M., Takeya, H., Goto, Y., & Furukawa, K. (1981). Fast oscillatory EEG activity induced by analgesic concentrations of nitrous oxide in man. Anesthesia and Analgesia, 60, 283-288.

Yates, A. (1987). Current status and future directions of research on the American Indian child. American Journal of Psychiatry, 144, 1135-1142.

Yokota, M., Miyanaga, K., Yonemura, K., Watanabe, H., Nagashima, K., Naito, K., Yamada, S., Arai, S., & Newfeld, R. W. (2000). Declining of memory functioning of normal elderly persons. Psychiatry and Clinical Neurosciences, 54, 217-225.

Zvolensky, M. J., McNeil, D. W., Porter, C. A., & Stewart, S. H. (2001). Assessment of anxiety sensitivity in young American Indians and Alaska natives. Behaviour Research and Therapy, 39, 477-493.

Table 1

Frequency of Type of Drug Used Among Experimental and Control Groups

Type of Drug	CSA	CNSA	Controls
Plane Fuel	1	0	0
Gasoline	7	2	2
Hairspray	1	0	0
Naptha	2	0	0
Paint	1	0	0
Marijuana	9	17	7
Cocaine	4	8	1
Alcohol	1	6	3
LSD	1	5	2
Mushrooms	0	5	2
Opiates	1	2	0
Ecstacy	0	1	0

Note. N = 42. CSA = Corrections participants with significant solvent histories, CNSA = Corrections participants without significant solvent histories.

Table 2

Pooled Correlations Among All Paper and Pencil Measures

	1	2	3	4	5	6	7
1. Age (years)	--	-.16	-.53**	.03	.02	.24	.02
2. BAI	--	--	.43**	.06	-.06	.12	-.17
3. BDI-II	--	--	--	-.11	-.02	-.16	-.23
4. Digit Span (Forward)	--	--	--	--	.35*	.41**	.39**
5. Digit Span (Backward)	--	--	--	--	--	.52**	.44**
6. Letter-Number Sequencing	--	--	--	--	--	--	.59**
7. TONI IQ	--	--	--	--	--	--	--

Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 3

Pooled Correlations Among Delta Bandwidth in Anterior and Posterior Quadrants for Eyes Open Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.85**	.66**	.60**
Right	.85**	--	.55**	.59**
Posterior				
Left	.66**	.55**	--	
Right	.60**	.59**	.64**	--
Age (years)	.43**	.43**	.48**	.63**
BAI	.23	.17	.09	.08
BDI-II	.15	.22	.14	.24
Digit Span (Forward)	.01	-.05	.22	-.18
Digit Span (Backward)	.01	.00	.24	.10
Letter-Number Sequencing	-.09	-.10	-.03	-.21
TONI IQ	-.07	-.04	.18	.12

Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 4

Pooled Correlations Among Delta Bandwidth in Anterior and Posterior Quadrants for Eyes Closed Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.94**	.65**	.65**
Right	.94**	--	.57**	.67**
Posterior				
Left	.65**	.57**	--	
Right	.65**	.67**	.76**	--
Age (years)	-.47**	-.45**	-.44**	-.59**
BAI	-.02	-.01	-.05	.01
BDI-II	.15	.15	.08	.22
Digit Span (Forward)	.09	.00	.22	-.07
Digit Span (Backward)	-.07	-.05	.00	.01
Letter-Number Sequencing	-.10	-.12	-.16	-.24
TONI IQ	.12	.12	.16	.14

Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 5

Pooled Correlations Among Delta Bandwidth in Anterior and Posterior Quadrants for Letter-Number Sequencing Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.86**	.75**	.63**
Right	.86**	--	.60**	.68**
Posterior				
Left	.75**	.60**	--	
Right	.63**	.68**	.73**	--
Age (years)	-.02	-.05	-.06	-.11
BAI	-.09	-.10	-.24	-.21
BDI-II	-.15	-.12	-.14	-.13
Digit Span (Forward)	.35*	.23	.34*	.11
Digit Span (Backward)	.17	.17	.15	.10
Letter-Number Sequencing	.07	.01	-.09	-.14
TONI IQ	-.06	-.10	.03	.05

Note. N = 42. *Significant at $p < .05$, **at $p < .01$.

Table 6

Pooled Correlations Among Delta Bandwidth in Anterior and Posterior Quadrants for TONI Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.79**	.68**	.52**
Right	.79**	--	.55**	.59**
Posterior				
Left	.68**	.55**	--	
Right	.52**	.59**	.68**	--
Age (years)	-.22	-.31*	-.32*	-.44**
BAI	.07	.08	-.04	-.04
BDI-II	.02	.12	.07	.08
Digit Span (Forward)	.23	.10	.33*	.09
Digit Span (Backward)	.22	.18	.24	.29
Letter-Number Sequencing	.08	-.01	-.06	-.08
TONI IQ	.10	.11	.10	.21

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 7

Pooled Correlations Among Theta Bandwidth in Anterior and Posterior Quadrants for Eyes Open Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.92**	.77**	.80**
Right	.92**	--	.63**	.79**
Posterior				
Left	.77**	.63**	--	
Right	.80**	.79**	.74**	--
Age (years)	-.44**	-.44**	-.42**	-.57**
BAI	.09	.06	.02	.05
BDI-II	.18	.22	.18	.23
Digit Span (Forward)	.03	-.03	.26	-.05
Digit Span (Backward)	.04	-.00	.22	.17
Letter-Number Sequencing	.01	-.05	.11	-.04
TONI IQ	.06	.06	.31*	.26

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 8

Pooled Correlations Among Theta Bandwidth in Anterior and Posterior Quadrants for Eyes Closed Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.97**	.86**	.88**
Right	.97**	--	.78**	.87**
Posterior				
Left	.86**	.78**	--	
Right	.88**	.87**	.86**	--
Age (years)	-.43**	-.46**	-.41**	-.55**
BAI	.01	.02	-.05	.02
BDI-II	.20	.24	.18	.24
Digit Span (Forward)	.03	-.00	.21	.04
Digit Span (Backward)	-.07	-.07	.05	.08
Letter-Number Sequencing	.02	-.02	.07	.03
TONI IQ	.08	.09	.27	.26

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 9

Pooled Correlations Among Theta Bandwidth in Anterior and Posterior Quadrants for Letter-Number Sequencing Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.88**	.76**	.68**
Right	.88**	--	.53**	.68**
Posterior				
Left	.76**	.53**	--	
Right	.68**	.68**	.70**	--
Age (years)	-.14	-.17	-.14	-.19
BAI	-.18	-.15	-.24	-.23
BDI-II	-.06	-.04	-.06	.00
Digit Span (Forward)	.33*	.17	.39*	.19
Digit Span (Backward)	.24	.25	.21	.19
Letter-Number Sequencing	.16	.09	.12	.05
TONI IQ	.01	-.04	.22	.21

Note. N = 42. *Significant at $p < .05$, **at $p < .01$.

Table 10

Pooled Correlations Among Theta Bandwidth in Anterior and Posterior Quadrants for TONI Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.90**	.77**	.69**
Right	.90**	--	.62**	.72**
Posterior				
Left	.77**	.62**	--	
Right	.69**	.72**	.70**	--
Age (years)	-.31*	-.33*	-.33*	-.39*
BAI	-.02	-.01	-.09	-.09
BDI-II	.15	.19	.17	.15
Digit Span (Forward)	.16	.04	.34*	.15
Digit Span (Backward)	.18	.23	.24	.26
Letter-Number Sequencing	.08	.05	.02	.00
TONI IQ	.15	.18	.26	.34*

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 11

Pooled Correlations Among Alpha1 Bandwidth in Anterior and Posterior Quadrants for Eyes Open Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.94**	.90**	.88**
Right	.94**	--	.82**	.90**
Posterior				
Left	.90**	.82**	--	
Right	.88**	.90**	.86**	--
Age (years)	-.21	-.35*	-.25	-.36*
BAI	.06	.07	.11	.10
BDI-II	.06	.13	.18	.17
Digit Span (Forward)	.05	.03	.15	.03
Digit Span (Backward)	.06	.08	.12	.19
Letter-Number Sequencing	.06	.06	.06	.05
TONI IQ	.05	.11	.14	.21

Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 12

Pooled Correlations Among Alpha1 Bandwidth in Anterior and Posterior Quadrants for Eyes Closed Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.97**	.91**	.88**
Right	.97**	--	.84**	.89**
Posterior				
Left	.91**	.84**	--	
Right	.88**	.89**	.89**	--
Age (years)	-.18	-.25	-.27	-.41**
BAI	.07	.10	.00	.08
BDI-II	.09	.14	.13	.22
Digit Span (Forward)	.08	.03	.16	.08
Digit Span (Backward)	.13	.09	.22	.26
Letter-Number Sequencing	.14	.08	.13	.09
TONI IQ	.18	.19	.30	.31*

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 13

Pooled Correlations Among Alpha1 Bandwidth in Anterior and Posterior Quadrants for Letter-Number Sequencing Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.91**	.81**	.77**
Right	.91**	--	.75**	.82**
Posterior				
Left	.81**	.75**	--	
Right	.77**	.82**	.87**	--
Age (years)	.06	.05	.01	-.08
BAI	-.11	.00	-.09	-.08
BDI-II	-.19	-.15	-.06	-.02
Digit Span (Forward)	.28	.16	.32*	.23
Digit Span (Backward)	.21	.15	.26	.27
Letter-Number Sequencing	.17	.12	.17	.19
TONI IQ	.02	-.01	.20	.24

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 14

Pooled Correlations Among Alpha1 Bandwidth in Anterior and Posterior Quadrants for TONI Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.89**	.73**	.69**
Right	.89**	--	.58**	.77**
Posterior				
Left	.73**	.58**	--	
Right	.69**	.77**	.71**	--
Age (years)	-.23	-.32*	-.30	-.33*
BAI	-.07	-.03	.06	.01
BDI-II	.06	.10	.27	.22
Digit Span (Forward)	.20	.10	.28	.20
Digit Span (Backward)	.19	.21	.22	.31*
Letter-Number Sequencing	.09	.08	.02	.07
TONI IQ	.05	.21	.14	.32*

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 15

Pooled Correlations Among Alpha2 Bandwidth in Anterior and Posterior Quadrants for Eyes Open Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.82**	.68**	.65**
Right	.82**	--	.52**	.70**
Posterior				
Left	.68**	.52**	--	
Right	.65**	.70**	.73**	--
Age (years)	-.03	-.18	-.22	-.31*
BAI	-.14	.01	.07	.04
BDI-II	-.02	.10	.21	.20
Digit Span (Forward)	-.08	-.14	.15	-.02
Digit Span (Backward)	-.10	-.09	.12	.16
Letter-Number Sequencing	-.12	-.21	.00	-.04
TONI IQ	-.12	-.09	.02	.19

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 16

Pooled Correlations Among Alpha2 Bandwidth in Anterior and Posterior Quadrants for Eyes Closed Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.91**	.79**	.77**
Right	.91**	--	.60**	.78**
Posterior				
Left	.79**	.60**	--	
Right	.77**	.78**	.75**	--
Age (years)	-.07	-.12	-.15	-32*
BAI	-.09	-.03	-.06	-.01
BDI-II	.09	.12	.11	.20
Digit Span (Forward)	.09	-.01	.29	.16
Digit Span (Backward)	.12	.13	.27	.32*
Letter-Number Sequencing	-.06	-.11	.13	.07
TONI IQ	.00	.03	.22	.29

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 17

Pooled Correlations Among Alpha2 Bandwidth in Anterior and Posterior Quadrants for Letter-Number Sequencing Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.78**	.67**	.56**
Right	.78**	--	.52**	.69**
Posterior				
Left	.67**	.52**	--	
Right	.56**	.69**	.74**	--
Age (years)	.19	.01	.08	.09
BAI	-.21	-.07	-.13	-.12
BDI-II	-.24	-.13	-.03	-.02
Digit Span (Forward)	.16	.05	.33*	.32*
Digit Span (Backward)	.08	.10	.17	.24
Letter-Number Sequencing	-.02	-.07	.06	.13
TONI IQ	-.13	-.06	.06	.17

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 18

Pooled Correlations Among Alpha2 Bandwidth in Anterior and Posterior Quadrants for TONI Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.85**	.65**	.60**
Right	.85**	--	.53**	.69**
Posterior				
Left	.65**	.53**	--	
Right	.60**	.69**	.76**	--
Age (years)	.05	-.10	-.21	-.22
BAI	-.18	-.16	.00	.02
BDI-II	-.11	-.10	.23	.24
Digit Span (Forward)	.10	.07	.28	.26
Digit Span (Backward)	.14	.23	.23	.31*
Letter-Number Sequencing	.06	.05	.04	.12
TONI IQ	.00	.16	.06	.23

Note. N = 42. *Significant at $p < .05$, **at $p < .01$.

Table 19

Pooled Correlations Among Beta Bandwidth in Anterior and Posterior Quadrants for Eyes Open Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.71**	.62**	.48**
Right	.71**	--	.45**	.39**
Posterior				
Left	.62**	.45**	--	
Right	.48**	.39**	.75**	--
Age (years)	.16	-.05	-.08	-.06
BAI	-.23	-.12	.03	-.04
BDI-II	-.20	.01	.14	.04
Digit Span (Forward)	-.04	-.06	.14	.04
Digit Span (Backward)	-.04	-.02	.13	.19
Letter-Number Sequencing	-.03	-.16	.00	.04
TONI IQ	-.11	-.07	-.10	.15

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 20

Pooled Correlations Among Beta Bandwidth in Anterior and Posterior Quadrants for Eyes Closed Condition and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.92**	.78**	.75**
Right	.92**	--	.63**	.76**
Posterior				
Left	.78**	.63**	--	
Right	.75**	.76**	.81**	--
Age (years)	.14	.05	.05	-.11
BAI	-.14	-.11	-.16	-.11
BDI-II	-.13	-.07	-.06	-.03
Digit Span (Forward)	.07	-.01	.24	.13
Digit Span (Backward)	.17	.18	.30	.30
Letter-Number Sequencing	-.05	-.11	.05	.01
TONI IQ	.02	.06	.16	.22

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 21

Pooled Correlations Among Beta Bandwidth in Anterior and Posterior Quadrants for Letter-Number Sequencing Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.82**	.73**	.65**
Right	.82**	--	.62**	.61**
Posterior				
Left	.73**	.62**	--	
Right	.65**	.61**	.87**	--
Age (years)	.19	-.06	.18	.24
BAI	-.25	-.12	-.08	-.07
BDI-II	-.28	-.14	-.07	-.09
Digit Span (Forward)	.04	-.01	.20	.23
Digit Span (Backward)	.04	.12	.14	.13
Letter-Number Sequencing	-.10	-.13	.02	.08
TONI IQ	-.16	-.05	-.06	.02

Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 22

Pooled Correlations Among Beta Bandwidth in Anterior and Posterior Quadrants for TONI Task and Paper and Pencil Measures

	Anterior		Posterior	
	Left	Right	Left	Right
Anterior				
Left	--	.87**	.61**	.58**
Right	.87**	--	.55**	.62**
Posterior				
Left	.61**	.55**	--	
Right	.58**	.62**	.82**	--
Age (years)	.12	-.06	-.10	-.06
BAI	-.18	-.18	.03	.04
BDI-II	-.22	-.14	.16	.14
Digit Span (Forward)	.02	.00	.22	.19
Digit Span (Backward)	.13	.24	.21	.27
Letter-Number Sequencing	.05	.05	.06	.11
TONI IQ	-.02	.13	.00	.14

Note. N = 42. *Significant at p < .05, **at p < .01.

Table 23

Cell Means (and Standard Deviations) Comparing Correctional and Control Groups for Paper-and-Pencil Measures

Characteristic	Correctional	Control
N	28	14
Age (years)	27.25 (8.01)	28.86 (7.46)
BAI	8.71 (9.66)	8.21 (9.23)
BDI-II	16.54 (11.31)	12.14 (7.48)
Digit Span (Forward)	9.25 (1.62)	10.00 (2.00)
Digit Span (Backward)*	5.04 (1.82)	6.36 (1.78)
Letter-Number Sequencing**	8.57 (2.27)	11.36 (2.79)
TONI IQ**	79.61 (7.66)	97.21 (11.28)

Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 24

Cell Means (and Standard Deviations) for EEG Results Comparing Correctional and Control Group

	Left		Right	
	Anterior	Posterior	Anterior	Posterior
Theta Eyes Open				
Correctional**	1.48 (1.13)	1.26 (1.13)	1.48 (1.12)	1.27 (1.14)
Controls**	1.57 (1.12)	1.38 (1.13)	1.55 (1.13)	1.42 (1.11)
Theta Eyes Closed				
Correctional*	1.58 (1.15)	1.36 (1.17)	1.57 (1.15)	1.39 (1.17)
Controls*	1.68 (1.16)	1.51 (1.19)	1.68 (1.17)	1.57 (1.16)
Alpha1 Eyes Open				
Correctional**	1.35 (1.13)	1.23 (1.16)	1.34 (1.13)	1.25 (1.17)
Controls**	1.52 (1.19)	1.43 (1.17)	1.48 (1.14)	1.51 (1.17)
Alpha1 Letter-Number Sequencing				
Correctional**	1.45 (1.13)	1.34 (1.14)	1.45 (1.12)	1.38 (1.14)
Controls**	1.57 (1.15)	1.55 (1.17)	1.57 (1.16)	1.62 (1.15)

Note. Regional means and standard deviations are based on natural log-transformed amplitudes of averaged individual scalp sites. Note. $N = 42$. *Significant at $p < .05$, **at $p < .01$.

Table 25

Cell Means (and Standard Deviations) Comparing Correctional Groups with Solvent Histories (CSA) and No Solvent History (NCSA) and Control Group on Paper and Pencil Measures

Characteristic	Correctional		Controls
	Solvent History	No Solvent History	
N	10	18	14
Age (years)	28.40 (7.85)	26.61 (8.25)	28.86 (7.46)
BAI	8.70 (9.08)	8.72 (10.22)	8.21 (9.23)
BDI-II	13.80 (12.40)	18.06 (10.72)	12.14 (7.48)
Digit Span (Forward) *	8.30 (1.49)	9.78 (1.48)	10.00 (2.00)
Digit Span (Backward)*	4.80 (1.55)	5.17 (1.98)	6.36 (1.78)
Letter-Number Sequencing*	8.30 (1.89)	8.72 (2.49)	11.36 (2.79)
TONI IQ*	79.40 (8.17)	79.72 (7.60)	97.21 (12.23)

Note. N = 42. *Significant at $p < .05$, **at $p < .01$.

Table 26

Cell means (and Standard Deviations) for EEG Results Comparing Correctional Groups with Solvent Histories (CSA) and No Solvent History (NCSA) and Control Group

	Left		Right	
	Anterior	Posterior	Anterior	Posterior
Alpha1 Eyes Open				
Solvent History*	1.27 (1.07)	1.20 (1.05)	1.26 (1.07)	1.20 (1.11)
Non-Solvent History*	1.39 (1.15)	1.27 (1.21)	1.38 (1.14)	1.28 (1.20)
Controls*	1.52 (1.19)	1.43 (1.17)	1.48 (1.14)	1.51 (1.17)
Alpha1 Letter-Number Sequencing				
Solvent History*	1.36 (1.09)	1.26 (1.08)	1.35 (1.08)	1.28 (1.13)
Non-Solvent History*	1.51 (1.12)	1.39 (1.16)	1.49 (1.12)	1.43 (1.14)
Controls*	1.57 (1.15)	1.55 (1.17)	1.57 (1.16)	1.62 (1.15)

Note. Regional means and standard deviations are based on natural log-transformed amplitudes of averaged individual scalp sites. N = 42. *Significant at $p < .05$, **at $p < .01$.

Appendix A

Ethics Approval Letter

Lakehead
UNIVERSITY

Office of Research
Tel. (807) 343-8283
Fax (807) 346-7749

2 October 2003

Mr. Michael Moland
Department of Psychology
Lakehead University
THUNDER BAY, ON

Dear Mr. Moland:

Based on the recommendation of the Research Ethics Board, I am pleased to grant ethical approval to your research project entitled, "QUANTITATIVE ELECTROENCEPHALOGRAPHIC AND DEMOGRAPHIC MARKERS OF SOLVENT ABUSE IN NATIVE CANADIAN SOLVENT ABUSERS: A NORTHWESTERN ONTARIO SAMPLE."

The Research Ethics Board requests an annual progress report and a final report for your study in order to be in compliance with Tri-Council Guidelines. This annual review will help ensure that the highest ethical and scientific standards are applied to studies being undertaken at Lakehead University.

Completed reports may be forwarded to:

Lynn Howe
Office of Research
Lakehead University
955 Oliver Road
Thunder Bay, ON P7B 5E1
FAX: 807-346-7749

Best wishes for a successful research project.

Sincerely,

Dr. Lori Chambers
Chair, Research Ethics Board

/lmh
Encl.
cc: Dr. C. Netley, Supervisor

Appendix B

Consent Form

BRAIN-WAVE EXAMINATION PROJECT
CONSENT FORM - PARTICIPANT

1. I, _____, consent to take part in this study that will examine brain wave (EEG) patterns.

2. If I choose to participate, I will be asked to provide three types of information: basic demographic information, a screen for depression, anxiety, thinking ability, memory and the presence of medical conditions that might affect EEG results.

3. I understand that all of my responses will be anonymous and confidential. After the study, my information will be coded to preserve anonymity. The research information will be stored for seven years by Dr. Netley at Lakehead University.

4. I understand that I am free to discontinue my participation at any time and for any reason, without explanation or penalty.

5. In keeping with university policy, the new data will be stored securely for seven years by Dr. Charles Netley at Lakehead University.

6. I understand that there are no known risks associated with participating in this research.

7. I will receive a gift certificate for $25 for my participation.

My signature on this form indicates that I may participate in a study by Michael Moland and Dr. Charles Netley of Lakehead University on examining brain-wave activity in drug abusing individuals compared to non-drug abusing individuals.

I have received an explanation about the nature of the study and its purpose. I understand the following:

1. As a volunteer, I can withdraw from the study at any time.

2. There is no apparent danger of physical or psychological harm.

3. The data I provide will remain confidential.

4. I will receive a summary of the project, upon request, following the completion of the project.

_____ _____

(Signed) (Date)

_____

(Witness)

Appendix C

Cover Letter

BRAIN-WAVE EXAMINATION PROJECT

Cover Letter

Dear potential participant:

I want to look for differences in brain-wave activity between drug using individuals and non-drug abusing individuals as part of a research project to complete my Ph.D. in clinical psychology at Lakehead University.

You would be asked to answer some questions about depression, anxiety, and drug use history, and background information that might affect the EEG testing. You will also be asked some questions about your memory and thinking ability while we measure your brain activity. The procedure is safe and painless, and involves no risk. The whole procedure will take about an hour and will be conducted at _____ (location).

In return for your participation, you will receive a summary of your test results when the study has been completed.

All information will remain confidential and securely stored at Lakehead University for seven years, as required by university policy.

As the principal investigator, it will be my role to collect all the data and review it on a computer. The testing will be carried out by me and my research assistant Cameron Dokis, a 4th year psychology student at Lakehead University.

Dr. Chuck Netley, a professor of Psychology at Lakehead University is supervising this project. He can be reached at 343-8486 if you have any questions or concerns.

Sincerely,

Michael Moland

Principal Investigator

Appendix D

Demographic Information Questionnaire

BRAIN-WAVE EXAMINATION PROJECT

BACKGROUND INFORMATION

1. Age: _____
2. ID Number _____ (year/number/group) (e.g., 0401control)
3. Recording Time: _____
4. Date: _____
5. Recent Medication or Drug Use? Time: _____
6. When was your last meal? Time: _____
7. Have you ever had a seizure? Time and Date: _____ Brain Injury? _____
8. Any family history of disease? _____
9. Which Solvent(s) have you taken? _____
10. Have you abused any other drugs? _____
11. Do you smoke cigarettes or drink coffee? _____
12. Handedness? Right handed _____ Left handed _____
13. EEG conducted by _____