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MEMORY PATTERNS: DIFFERENTIATED BETWEEN ENVIRONMENTAL SENSITIVE PATIENTS AND PSYCHIATRIC PATIENTS

DISSERTATION

Presented to the Graduate Council of the University of North Texas in Partial Fulfillment of the Requirements

For the Degree of

DOCTOR OF PHILOSOPHY

By

Esther Lockart, M.A.

Denton, Texas

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The purpose of the present study was to ascertain if environmentally sensitive patients would demonstrate different memory deficit patterns than psychiatric patients on objectively measurable memory tasks. One-hundred sixteen patients were surveyed; 56 environmentally sensitive patients were compared to 60 psychiatric patients. All subjects were administered a Wechsler Adult Intelligence Scale-Revised screen, the Wechsler Memory Scale-Revised and the Harrell-Butler Comprehensive Neurocognitive Screen after history of head injury was ruled out.

Results indicate a significantly different pattern of memory dysfunction between the environmental patients and the psychiatric patients, indicating two different etiologies. A screening device derived from the coefficients from a Canonical Analysis is proposed to distinguish between the two populations in the absence of blood serum levels of environmental toxins or poisons. The detrimental effects of misdiagnosis and the beneficial effects of accurate diagnosis of environmental illness are discussed.

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CHAPTER I

INTRODUCTION

While in many diseases a diagnosis can be made based on clear-cut symptoms, the symptoms of pesticide and organic solvent poisonings have taken many forms. As a result, there has been no exclusive set of symptoms emerge to make the diagnosis of these toxic reactions straightforward. All of the major pesticides and organic solvents work on the central nervous system, and individual effects of the poisoning have been reported to vary (Baker, White & Murawski, 1985). Whether the pesticides or solvents are more likely to initially effect the liver or the kidneys has been found to vary individually, depending upon whether the exposed person had pre-existing liver or kidney problems, or genetic predisposition to one or the other. Headaches, dizziness, fatigue, malaise, vision impairment, stomach pains, and memory deficits have been the most frequent symptoms reported (Weiss, 1985). It is thought that these vague symptoms have been caused by a number of different pesticides and/or by a variety of solvents or other toxic chemicals.

The pesticides and solvents which are Central Nervous System (CNS) inhibitors have also been found to cause

symptoms in people which have traditionally been labeled psychological. Some researchers have concluded that "monitoring adverse effects by psychological measures may yield more information than monitoring them by blood chemistry," (Weiss, 1985, p. 21). Fein, Schwartz, Jacobson and Jacobson (1983) have reported that a multiple-effect model emphasized subtle behavioral alteration as an early sign of toxicity. They also found evidence that a particular chemical agent may have produced long-term impairment in susceptible individuals.

A majority of pesticides and solvents have been classified neurotoxins. Weiss (1985, p. 22) suggested that the field of Behavioral Toxicology should gauge "the adverse impact of environmental chemicals... by how people feel and function, not solely by death or overt damage." How people function has become an important clinical consideration because many poisonings begin with vague, unspecific complaints followed by overt clinical signs. As stated earlier, these chemicals act primarily on the CNS, and therefore, have produced distinct behavioral as well as psychological reactions. It was likely that by the time the symptomatology had progressed to clinically recognizable symptoms, there was already significant damage to the nervous system (Weiss, 1985). For instance, Weiss pointed out that while a rat may have functioned without noticeable impairment after so much neurological impairment, it may not

have been the case with the skills required by crop-duster pilots whose blood levels of cholinesterase may have been reduced to one fourth of normal.

A survey of 80 Swiss workers who had been employed an average of 3.5 years at an agricultural plant were exposed to air concentration levels of trichloroethylene (TCE) between 1 and 335 parts per million (ppm) showed a number of psychological changes: 19% complained of reduced memory capability and 20% of reduced intellectual processes (Bang, 1984). Another report on 25 adults who sniffed toluene revealed three different clinical symptoms: muscle weakness, gastrointestinal complaints, and neuropsychiatric disorders, including altered mental status and peripheral neuropathy (Streicher, Gabow & Moss, 1981).

Methylene chloride (MC) has been demonstrated to be a mild CNS depressant (Repko, 1980). MC exposure may be related to the impairment of biologic oxidation. MC has been found to rapidly metabolize to carbon monoxide. The amount of carbon monoxide formed in the body has been directly related to the amount of MC absorbed (Repko, 1981). In the research literature, MC has received little attention as an occupational hazard. However, chronic exposure to MC may have resulted in neuropsychiatric effects, such as depression, change of personality, irritability, insomnia, and disturbance of intellectual functioning (Winneke, 1974). Rodepierre (1955) illustrated the symptomatology of a man

who had been exposed to low doses of MC for 10 years. Symptoms included mental confusion, amnesia, slowness of mental reaction, and insomnia. In psychiatric evaluations of painters exposed to toluene, xylene, TCE, and "white spirits" the exposed subjects exhibited more neurasthenic symptoms, including poor memory, slowed perceptions and reaction time than did the controls (Bang, 1984).

Savage (1980) studied 100 agricultural workers who had been exposed to a toxic dose of organophosphates at least once between 1950 and 1976. There was no significant difference between poisoned workers and controls on physiological measures such as hearing and eye tests, brain waves, and blood tests. However, the poisoned workers scored markedly worse than the control group on measures of depression, intellectual functioning, academic skills, abstraction, flexibility of thinking, and simple motor skills. They also performed at lower levels on tests of reading recognition, comprehension, written verbal fluency, problem solving, and spelling. These findings were inconsistent with the poisoned workers previous levels of functioning. Furthermore, people in the exposed group reported more difficulty with memory (remembering names of objects), language (recognizing printed or written words), and thinking (understanding the speech of others) problems.

Workers exposed to Keopane reported deficits of recent memory. Cannon, Veazey, Jackson, Burse, Hayes, Straub,

Landrigan and Liddle (1978) found mean blood Kepone levels of 2.58 parts per million in employees of the Allied Chemical Corporation. The most frequently reported symptoms were nervousness and tremor. However, workers complained of other symptoms such as incoordination, weight loss, skin rashes, sterility, abnormal liver function, ocular flutter, joint pain, and pleuritic pain. Personality changes also occurred, marked by irritability, problems with recent memory and depression, but not every worker showed the same collections of symptoms. Neither management nor plant workers raised the issue of toxicity until an alert internist forwarded a blood sample to the Center for Disease Control for analysis of toxicity. There was convincing evidence that exposure to even low levels of Kepone could induce toxic responses.

Sandulescu (1982) studied the clinical manifestations of intoxication with mercury-ethyl chloride (Cryptody), a pesticide, in 20 patients hospitalized for chronic and subacute reactions. There were in order of predominance neurological changes consisting of headache, vertigo, asthenia, and sleep disturbances. Behavioral changes consisted of agitation, increased impulsiveness, memory disturbances, and/or confusional states.

A variety of experiments have been conducted on animal subjects. Results of these experiments confirmed that pesticides can cause symptom multiplicity. Van Gelder

(1975) reported that mild doses of dieldrin interfered with sheep's ability to relearn a visual discrimination task. Bloom, Staatz, and Dieringer (1983) found that subconvulsive doses of pyrethroid insecticides can have significant effects on learning behavior in male rats. Gause, Hartmann, Leal and Geller (1985) administered a variety of sublethal doses of the organophosphate nerve gas, Soman (SO), to six juvenile baboons. Attentional deficits and intermittent generalized seizures were persistent effects. The authors suggested that the occurrence of attentional deficits may have been associated with generalized and/or focal seizures and that these effects may have reflected irreversible lesions that would become more threatening over time. The persistent attentional deficits of humans exposed to organophosphorus insecticides may have represented an operant behavioral analog of the mental confusion defined as attention lapses. Finally, Mactutus and Tison (1984) administered 1 mg of chlordecone to 80 four-day-old rats. The test rats needed, on the average, two days longer to learn a one-way or two-way active avoidance task than rats in a control group.

Much research has been conducted on the effects of organic solvents on behavioral and neurological assays. Lendstrom's (1982) literature review indicated that epidemiological studies have detected an increased risk of neuropsychiatric diseases among groups occupationally

exposed to organic solvents. Psychological studies of solvent-exposed workers had investigated both cognitive and sensorimotor functions. Most of these studies have dealt with trichloroethylene, toluene, styrene, and mixtures of organic solvents. A decline in sensorimotor functions has been observed in many studies. Of the cognitive functions, short-term memory in particular has been proven to be sensitive to solvent exposure. Arlien-Soborg (1984) evaluated behavior and cognitive functioning in 70 house painters (24-70 years of age) referred to a hospital's neurological department. The subjects' most common complaints were impaired learning and memory. Data from tests measuring immediate verbal memory span, verbal learning and memory, visual-spatial learning, vigilance, and psychomotor speed indicated that the symptoms were caused, at least partly, as a result of exposure to the solvents. Baker, White and Murawski (1985) noted that the most characteristic findings in solvent-induced toxic encephalopathy were deficits in memory function and impaired psychomotor ability. Memory loss in terms of immediate memory span, short-term memory and learning ability was observed. Bergholtz and Odkvist (1984) illustrated that subjects exposed to jet fuel experienced fatigue, unsteadiness, memory, and concentration problems. Crossen and Wiens (1988) administered the Wechsler Memory Scale-Revised (WMR-R) to 20 industrial painters who had a history

of solvent exposure. Test results indicated there was memory impairment. Although the more educated subjects had better WMS-R performance than the others, all educational groups performed at the impaired level relative to WMS-R education-adjusted norms.

Toluene, an organic solvent most commonly used in paints, is highly toxic. In recent years, it has become a substance of abuse by young adults because of it's intoxicating effects (Juntunen, 1985). Iregren (1982) evaluated 34 painters, who had been exposed to a single solvent, with a psychological test battery that included measures of dexterity, memory, and mental arithmetic. Their performances were compared to those of a matched group of spray painters who had been exposed to a mixture of solvents. Both groups scored within the impaired range, however, the scores of the group exposed to a mixture of solvents were worse than the scores of the group exposed to the single solvent.

Ryan, Morrow and Hodgson (1988) examined the interrelationship between occupational exposure to mixtures of organic solvents and neurobehavioral functioning. The authors administered a battery of cognitive tests, including the Wechsler Memory Scale and subtests of the Wechsler Adult Intelligence Scale-Revised, to 17 men with a history of solvent exposure. When compared to 17 controls, the exposed men were found to be more impaired across a wide range of

cognitive domains such as learning and memory, visuospatial skills, attention, mental flexibility, and psychomotor speed, than those in the control group.

None of the previously mentioned studties focused on identifying a pattern in these memory deficits, although, there could possibly be a common thread. Immediate, short-term, or what is often referred to as fluid memory, were frequently cited. One might suspect that short-term memory impairment could be one of the diagnostic signs for environmental sensitivities. Some have referred to these sensitivities as environmental illness (EI), indicating that one has become ill through exposure to his or her surroundings (Hemming, 1978, Moyer, 1976, Philpott, 1977a). Others have considered these sensitivities to be allergies in that the patient's immune system has been found to react or over react to substances in the environment (Randolph, 1976b). Doctors and clinicians specializing in chemical exposure therapy have been called clinical ecologists. The media coined this phenomenon as the "20th Century Disease" (KXAS-TV, 1988, American Broadcasting Company, 20/20, 1990). However, the medical community has been largely skeptical of environmental illness. It has produced position papers critical of clinical ecology and various testing procedures (Black, Rathe & Goldstein 1990). According to these representatives of the American medical establishment, there

are currently neither acceptable case definitions nor established methods to verify the existence of EI.

Five case series (Brodsky, 1983; Pearson, Rix & Bently, 1983; Stewart & Raskin, 1985; Terr, 1986; Terr, 1989) that have been presented in the literature are relevant to EI. The authors, almost uniformly, have observed that many symptoms of EI overlap with recognizable psychiatric syndromes, such as affective and anxiety disorders. These researchers have contended that psychiatric diagnoses offer a more parsimonious explanation for the symptoms. In each series, the patients were middle-aged, predominantly female, and polysymptomatic. In four studies, the frequency of subjects reported to have psychiatric diagnoses or symptoms ranged from 42% to 100% (Brodsky, 1983; Pearson et al., 1983; Stewart & Raskin, 1985; Terr, 1989).

Brodsky (1983) reported on eight cases referred for evaluation for a pending disability claim. Most of these patients had a history of "doctor shopping" for evaluation or recurrent physical complaints finally sought evaluation from the same network of clinical ecologists. Most led a life-style organized around the illness; they had stopped working and spent a great amount of time reading about allergies, taking tests for sensitivities, planning diets, and attending to their compensation claims. Stewart and Raskin (1985) reported on 18 cases referred to a university occupational medicine clinic. All 18 subjects in their

study met the DSM-III criteria for a psychiatric diagnosis. Seven subjects met the criteria for somatization disorder, which includes a history of physical symptoms of several years' duration beginning before the age of 30, complaints of at least 14 symptoms for women and 12 for men and the belief that one is sick much of the time. Four subjects were diagnosed with an anxiety disorder, three with an affective disorder, and one with a personality disorder.

Pearson et al. (1983) studied 23 patients who had been referred to an allergy clinic for suspected food allergy. Four had confirmable food allergies and displayed typical symptoms, such as asthma or urticaria. A psychiatric diagnosis was assigned to all but one of the remaining patients. The most common diagnosis was neurotic depression, neurasthenia and hysterical neurosis. Terr (1989), extending an earlier report by Brodsky (1983), reviewed the medical records of 90 workers who had filed disability claims on the basis of EI diagnoses. Psychiatric diagnoses, including depression, anxiety, somatization disorder, functional gastrointestinal illness, and stress, were reported in 38 subjects.

Sparks, Simon, Katon, Altman, Ayars and Johnson (1990) evaluated a case series of 53 aerospace workers who had filed compensation claims for work-related illnesses characterized by multiple somatic and neuropsychiatric complaints, although EI had not been diagnosed. These

investigators found that 39 of the workers had met the criteria for major depression, panic disorder or both. This accounts for 79% of the workers diagnosed with a psychiatric illness. No physical causes for the symptoms were found and the authors concluded that the physical and emotional disturbances were psychiatric in nature.

The possibility that all of these authors failed to consider, was that the psychiatric symptoms could have been secondary to physical illnesses. In the past, symptoms which have had their beginnings as a result of exposure to chemicals had initially been assigned to "mental diseases" or psychiatric disorders. For example, it was found that in Minnimota, Japan, a manufacturing plant had released mercury into the fishing waters for years causing undue damage to the residents of Minnimota (Weiss, 1983). The "dancing cat disease" was attributed to people being "mad," until it was discovered that the mercury being poured into the ocean from a nearby factory was destroying the nervous systems of the villagers. Another example given was the "madhatters" of Europe (Weiss, 1983). It was assumed that the hat makers were just "going crazy" until it was discovered that the lead used in the manufacturing of the hats was causing the neuropsychiatric symptoms. While these are famous and extreme cases and it can be argued that scientists and doctors didn't have the means to test for such obscure etiologies, all too often, difficult to diagnose or poorly

understood ailments are considered to be "made up by the patient," and labeled somatoform, hysteria, or hypochondriasis.

In some of the aforementioned studies, (Brodsky, 1983; Pearson et al., 1983; Stewart & Raskin, 1985; Terr, 1989) psychiatric diagnoses had been assigned to the subjects after they had been diagnosed with EI. Before diagnosis, no attempt was made to ascertain prior psychological functioning of the individuals mentioned in these studies.

Riecken and Butler (1991) analyzed the mental status exams of 89 patients whose environmental exposure was verified by the presence of toxins in the blood serum. Psychological functioning, before and after acquiring the illness, was investigated. A substantial number of patients (84%) classified themselves as having had good psychological functioning before symptoms arose. However, after becoming symptomatic, their psychological functioning became increasingly poor. Therefore, it was not until they became symptomatic did these patient begin to have troubles in daily living (i.e., depression, anxiety etc.).

The historical background of the patients did not indicate psychopathology. Most of the patients in the Riecken and Butler (1991) study had stable work histories, unlike those with schizophrenia or bipolar disorders, and expressed satisfaction with their jobs. Finally, many of the patients reported being healthy throughout most of their

lives prior to a toxic single large dose exposure or to multiple low dose exposures. This finding was inconsistent with the pattern for hypochondriasis or malingering. Also, the age of onset was inconsistent with that of schizophrenia.

The development of such psychological symptoms as depression and anxiety in EI patients has been found to be understandable considering that EI has not been considered an "accepted" illness (Black et al., 1990). EI patients typically have seen many doctors in their efforts to find relief from persistent physical and psychological symptoms. It is not surprising to find that many of these patients have been told that their illness is "in their head." Further psychological symptoms resulted when these patients understood that further exposure to chemicals, to which they are sensitive have been related to further deterioration or relapse. It has been argued that the depression and anxiety, along with the emotional and social withdrawal, described in the EI population, have been the byproducts of the illness and its situational consequences.

Therefore, in order to help resolve the controversy over environmental illness, it would make sense to find objective data from which conclusions might be drawn. Many of the studies mentioned relied heavily on observation instruments. Black et al. (1990) used the Diagnostic Interview Schedule and the Structured Interview for the DSM-

III Personality Disorders. Other instruments which were used were self-rating scales completed by the subjects.

In Riecken and Butler (1991) one easily measured symptom was memory deficits. Memory has been measured utilizing a variety of objective instruments. A pilot to the present study (Lockart & Butler, 1992) assessed memory function in 56 patients whose blood serum analysis indicated toxic chemical exposure. These patients' memory functions were assessed using the Wechsler Memory Scale-Revised (WMS-R), the Wechsler Adult Intelligence Scale-Revised (WAIS-R) and the items from the Harrell-Butler Comprehensive Neurocognitive Screen (H-B CNS) (1988). Prior memory and general intellectual level was assessed by the level of education prior to exposure. All patients had finished high school and 65% of the patients has received a Bachelors degree or higher. Therefore, the Lockart and Butler estimated I.Q. levels were based on the subjects' demonstrated educational and professional achievements.

The results of the Lockart and Butler (1992) study indicated a lowering of general intellectual functioning, relative to previous functioning, as measured by the WAIS-R. While long-term memory assays were in the normal range, short-term memory tasks (including verbal, visual and delayed memories) were greatly diminished on all assays. This study indicated that patients with a history of chemical exposure have memory dysfunction. It did not

determine whether these dysfunctions were caused by chemical effects on cerebral functions or if their psychological symptoms were interfering with their ability to perform the tasks required of them. To explore the possibility that psychological symptoms were interfering with cognitive memory functions, one would need to assess the memory functions of patients diagnosed with psychological disorders, who do not have a history of chemical exposure or head injury.

Memory dysfunction is not a criterion for any of the functional psychogenic disorders. According to the Diagnostic and Statistical Manual of Mental Disorders Third Edition (American Psychiatric Association, 1980), major depressive episodes are characterized by difficulty in concentrating, slowed thinking and indecisiveness. Patients with depressed moods may complain of memory difficulty and appear easily distracted. However, a review of the literature may prove to highlight cognitive characteristics of the functional psychogenic disorders.

Depression

A large number of investigators who have studied cognitive functioning in affective disorders have looked at memory and verbal learning function. Cronholm and Ottosson (1961) demonstrated impaired learning ability in a group of endogenous depressives using a twenty figure test, a thirty item word pair test, and a thirty item personal data test,

compared with normal controls. They found that the group of depressives was not significantly different in its performance on any of the aforementioned tests. Zung, Rogers, and Krugman (1968) investigated the effects of ECT on memory. They administered several cognitive tests to 23 moderately and markedly depressed psychiatric inpatients prior to treatment. Three of these tests have been clinically employed as measure of cortical dysfunction: the Bender-Gestalt test, the Benton Visual Retention test, and the Wechsler Memory Scale. They noted that before ECT, all of the patients scored at levels which, according to the tests' normative criteria, indicated the presence of CNS dysfunction.

Henry, Weingartner, and Murphy (1973) tested hospitalized unipolar depressive patients as well as bipolar patients in both the manic and depressive phases on two learning tasks. Clinical state was measured by the Bunney Humburg 15-point word rating scales. Depressive patients showed a significant decrease in serial learning in comparison with their own performance on test days when they were less depressed. They also demonstrated an impairment in the free recall learning task. Henry et al. (1973) concluded that depression interferes with the transfer of information from short-term to long-term memory storage.

Whitehead (1973) compared performance on 10 tasks measuring verbal learning and memory function (for example,

digits forward, serial learning tasks, logical memory-immediate, and delayed) in patients suffering from an acute depressive episode. A group of well preserved demented patients and a group of depressed patients were given the same tests after recovery. The depressives were all 60 years of age or older and were tested before any treatment commenced. The ill depressives did not vary significantly from the well depressives. For those tests measuring logical memory, the ill depressives were found to be superior to the demented patients and again, did not differ from the well depressives. The type of errors made by each group were classified in three categories: (1) omission errors (i.e., the subject did not appear to know the answer); (2) transposition errors (for example, responses that would be correct in another position); and (3) random errors (for example, words or answers that had nothing to do with the question). The demented patients made more random errors, and produced more false positives than the ill depressives, and made significantly less transposition errors. The ill depressives made significantly more omission errors than the well depressives and tended to produce more false negative answers. These findings were thought to provide support for the theory that the verbal learning impairment in dementia has a different etiology than that in depressive illness.

Sternberg and Jarvic published a series of articles in 1976 attempting to further elucidate the memory deficit in depression. They tested a group of 26 hospitalized depressed patients who had never received antidepressant medication and 26 nonhospitalized controls, who exhibited no evidence of current mental or physical illness. These groups were matched with respect to sex, level of education, and age. Memory function was evaluated with a fifteen item word-pair test, a fifteen figure test, and a personal data inventory. Sternberg and Jarvic defined three subsections of memory function as registration (immediate reproduction of previously untested material), retention (long-term memory, i.e., reproduction of material presented 3 hours earlier), and retrieval. Both groups were tested before any treatment was instituted for the depressive group. The depressive patients were retested after taking antidepressant medication (either amitriptyline or imipramine) for 26 days. Thirteen members of the control group were retested to make sure that no practice effect was confounding the results. The depressed patients were globally rated as either recovered, markedly improved, or moderately improved. The results demonstrated a significantly lower score for immediate reproduction in the depressed patients before treatment when compared with controls. There were no significant differences with regard to the scores for forgetting (the difference between the scores for immediate

reproduction and the scores for delayed reproduction of the same test). Retesting of 13 of the original controls after 26 days, showed no significant change in any of the test scores, arguing against any practice effect.

Ferrell, Cluver and Whybrow (1971) studied 10 hospitalized, depressed patients. The patients were given a WAIS and the Halstead-Reitan Battery when admitted to the hospital and again after recovery. Results showed no change in psychometric intelligence as judged by the WAIS, however, performance of five patients on the Halstead-Reitan Battery was consistent with Halstead's original criteria for cerebral dysfunction. These deficits resolved with resolution of the depressive episode. Donnelly, Dent and Murphy (1972) also used the WAIS and the Halstead-Reitan Battery to compare a group of 13 hospitalized depressives and 13 temporal lobe epileptics with unequivocal EEG abnormalities. They found that 69% of the depressed subjects scored in a range consistent with mild to severe brain damage.

Miller, Seligman, and Kurlander (1974) tested depressed-anxious, nondepressed-anxious, and nondepressed-nonanxious college students on a multidimensional discrimination learning problem. Depressed-anxious subjects required significantly more trials to reach criterion for learning then nondepressed-anxious subjects, but this difference appeared only when the discrimination-learning

task was presented last in a series of tasks. Nondepressed-anxious and nondepressed-nonanxious subjects did not differ in discrimination learning.

Silverstein, McDonald and Meltzer (1988) investigated neuropsychological dysfunction in schizophrenia, schizoaffective disorder, and major depression using the Luria-Nebraska Neuropsychological Battery (LNNB). The study examined whether important clinical components of neuropsychological dysfunction yielded specific patterns of differential impairment in these diagnoses. Although all scales of the LNNB revealed differences as a function of the customary decision rules for discriminating impaired and nonimpaired neuropsychological performance, the majority of the measures did not show any diagnosis-specific differences among schizophrenia, depression, and schizoaffective disorder. A factor analysis of the memory scale revealed that the verbal memory function showed greater impairment for schizoaffective disorder patients than for the schizophrenic or depression patients. There were no group differences on the visual/complex memory factor.

One implication of these data challenged the belief that schizophrenics characteristically show the most impaired functioning among psychiatric patients. Silverstein et al. (1988) indicated that schizoaffective disorder patients and schizophrenics showed comparable impairment on select neuropsychological variables. This

finding, which was based on Research Diagnostic Criteria (RDC) (Spitzer, Endicott, & Robins, 1978), was consistent with previous findings based on DSM III criteria by Spitzer et al. Schizophrenia and depression not only showed comparable performance levels overall, combining neuropsychologically impaired and nonimpaired subgroups within each diagnosis, but, in addition, there were no clearly discernible neuropsychological patterns of performance or specific areas of dysfunction that discriminated schizophrenics from depressives when schizoaffective disorder patients were included for comparison.

Anxiety

Interest in the manner in which individuals perceive, selectively attend to, and retrieve personally relevant information from memory (collectively regarded as information processing) has been posited as an explanation for memory patterns in anxiety disorders. Lang (1977, 1979, 1985) proposed an information processing theory of fear and anxiety which maintains that fear-relevant stimuli are encoded in a propositional form of memory. These highly organized, semantic, "fear networks" have been posited to contain three related types of information: (a) information about the stimulus cues that elicited fear; (b) information about cognitive, motor and psychophysiological responses (e.g., heart rate increases, scripts for behavioral

avoidance); and (c) information which defined the meaning of the stimulus cues and responses for the individual (e.g., "I will faint or go crazy unless I escape this situation"). Lang (1985) proposed that fear information was stored in memory in a way that facilitated cognitive, motor and psychophysiological responding to these cues.

Chemtob, Roitblatt, Himada, Carlson, and Twentyman (1990) proposed that patients with Post-Traumatic Stress Disorder (PTSD) typically responded to a perceived threat by utilizing a survival mode of functioning. This formerly adaptive pattern of behavior (hypervigilance, psychophysiological hyper-reactivity and a perceptual "readiness" to attend and react to threat or danger cues) was suggested to be represented in memory in the form of a highly organized hierarchical structure. It was assumed that the fear structure included information about emotions (e.g., anger, dread, or panic), plans for action or sequences of behavior (e.g., fight or flee), and was associated with images and memories of past threatening experiences. Because information about danger and threat has been thought to be stored in memory through rich multidimensional semantic frameworks, a broad variety of retrieval cues have been found that activate memories of threat (Tulving & Watson, 1975).

Chemtob et al. (1990) suggested that threat-related arousal might have been at least partially potentiated in

PTSD at all times. This arousal was also thought to set the stage for the expectation of threat whereby the patient with PTSD had begun to look for validating evidence of threat and arousal. Chemtob et al. (1990) also predicted that patients with PTSD would be likely to interpret ambiguous stimuli as being potentially harmful, dangerous, or threatening. These patients also would be more likely to focus their attention on threat-salient cues in the environment. Thus, it was proposed that weak evidence of threat, both in the environment and in the internal milieu, would be used by the information processing system to further potentiate, bias, and select threat-relevant stimuli (at the same time inhibiting alternative, more adaptive networks) in a manner which would create the expressed symptom picture in PTSD.

Burgess, Jones, Robertson, Radcliffe, and Emerson (1981) found that agoraphobic and social phobic patients were more likely than normal controls to detect words that were idiographically related to their phobias in the attended channel during a dichotic listening (shadowing) task. Phobic subjects, when compared to normals, were able to detect a greater proportion of the targets presented to the unattended channel. These threat-stimuli were said to draw on attentional resources because patients with anxiety disorders are pre-potently ready to attend to fear-related cues.

In a subsequent study utilizing a variation of the shadowing paradigm, Mathews and MacLeod (1986) had patients with generalized anxiety disorder perform a standard dichotic listening task and a simultaneously presented visual detection task during presentation of threat or nonthreat target words in the unattended channel. This secondary task was designed to determine if the presence of threat cues in the unattended channel captured processing resources. If so, it should have fostered longer reaction time to detect the presence of a probe stimulus. These researchers found that anxious patients exhibited greater reaction time latencies when to-be-detected visual probes coincided with threat as opposed to nonthreat stimuli, whereas the performance of normal controls did not reveal any such reaction time changes. This finding clearly showed that anxious patients would be more likely to divert their attentional resources to threat cues and away from on-going task demands.

Based on the theories discussed previously, it has been predicted that there should be a memory bias in anxiety disorders. Threat-cues seemed well organized in memory and drew attentional resources, which suggested that such information would get preferential encoding and would thus bias and enhance recall and recognition performance. Mathews and MacLeod (1985) failed to find any recognition memory differences for threat words presented during a

Stroop color-naming paradigm between generalized anxiety patients and normal controls. Thus, even though threat words had been preferentially processed during Stroop trials this had not lead to biased retrieval of such stimuli. Similarly, Mogg, Mathews, and Weinman (1987) failed to find evidence for a threat-related memory retrieval bias. Although the data to date has been suggestive, it still remains to be seen whether anxiety disorders in general are indeed associated with a retrieval deficit for fear-network related material.

The articles reviewed here, have suggested equivocal patterns of memory deficits for depression or anxiety. The authors have come to different conclusions as to the type and cause of memory dysfunctions in their respective subject populations. Lockart and Butler (1992) have shown a definite decrease in memory functioning in environmental patients. The purpose of the present study was to explore whether or not there were distinguishable differences in memory functioning between environmental patients and patients with functional psychogenic disorders.

It was hypothesized that: (1) A specific memory pattern would emerge for environmentally sensitive patients; and (2) These memory deficits would differ significantly from those of patients with psychogenic disorders.

CHAPTER II

METHOD

Subjects

The total number of patients surveyed was 116; 56 of the subjects were environmentally ill and referred for psychological evaluation at the Environmental Health Center in Dallas, Texas. Their diagnosis was determined by the presence of pesticides or organic solvents in blood serum tests. The mean and standard deviation for age and level of education were 45; $\pm$8.7 and 14.7; $\pm$2.6 years respectively. The environmentally ill patients were compared to 60 patients, diagnosed with depression or anxiety disorders, referred to the Utah Valley Regional Medical Center for psychological treatment. Participation by all patients was voluntary. The mean and standard deviation for their ages and level of education were 40.65; $\pm$9.45 and 14.8; $\pm$2.4 years respectively. The patients were screened for a history and were eliminated if such a history was present.

Materials

The neurocognitive and psychological batteries included the Wechsler Adult Intelligence Scale-Revised (WAIS-R) (1980), the Wechsler Memory Scale-Revised (WMS-R) (1988) and

the Harrell-Butler Comprehensive Neuropsychological Screen (HB-CNS) (1988).

The Wechsler Adult Intelligence Scale-Revised (1980) provided a base level of cognitive functioning and separated scores for verbal and non-verbal abilities. This test also was used to provide an overall Intelligence Quotient (IQ). Reliability studies of the three categories were very high across all age groups tested. Average coefficients were .97 for full-scale IQ, .97 for the verbal IQ, and .93 for the performance or non-verbal IQ. Comparison of means for groups of various levels of educational attainment consistently showed the average WAIS-R scores for individuals with lower levels of education to be lower than scores for individuals with higher levels of education. Matarazzo (1972) concluded from a number of studies of various measures of intelligence that the correlation between IQ and performance in school is about .50. The present study utilized the subtests of information, digit span, vocabulary, comprehension, block design and digit symbol. Digit span and digit symbol were used because these subtests assess short-term memory. Information and vocabulary assess long-term memory. Block design and comprehension were used to help determine basic intellectual functioning.

The Wechsler Memory Scale-Revised (WMS-R) (1988) was comprised of nine subtests, with a second presentation of

four of them. The subtests included information and orientation, mental control, figural memory, logical memory, visual paired associates, verbal paired associates, digit span, visual reproduction, and a readministration of logical memory, visual pair memory, verbal paired memory and visual reproduction. Five measures derived from the subtests were verbal memory, visual memory, general memory, attention/ concentration, and delayed recall. Retest reliability was .97. The present study utilized all five measures.

The Harrell-Butler Comprehensive Neuropsychological Screen (H-B CNS) (1988) is a 43 item research tool which measures a variety of neurocognitive and psychological abilities of which memory is a major factor. This test measures verbal, visual, logical, attention/concentration, working and delayed recall memory.

Procedure

All subjects were administered the subtest mentioned previously from the WAIS-R, the WMS-R and the Harrell-Butler Comprehensive Neuropsychological Screen. The patients were divided according to the diagnosis given to them following evaluation of symptoms (i.e., EI patients, depressive patients or anxious patients). The battery was administered by a qualified psychological intern who was experienced in administration and scoring of all items in the test battery.

CHAPTER III

RESULTS

Multivariate (MANOVA) techniques were used to analyze the results because the test battery contained a number of intercorrelated variables. These techniques reduced the probability of making a type I or type II error. MANOVA test criteria and exact F statistics were used to test the hypothesis of no overall difference between the environmental group and the psychiatric group for age, sex and education. The results of these statistics are found in Table 1. The comparison of relevant variables indicated equivalency of groups, such that the assumption of MANOVA statistics was not violated.

Using MANOVA techniques, it was discovered that the differences in performance on the tests administered were not due to the variables of age, sex, or educational level. Educational level did effect performance on the tests in general, in that the higher the educational level, the better the performance on the test. This factor did not contaminate the results as level of education did not distinguish the two groups. In fact, both groups represented a wide range of educational levels, 10 to 20 years. Group assignment did explain the difference between the groups.

Table 1

Multivariate Analysis of Variance, Environmental Patients and Psychiatric Patients on Sex, Age, and Education

Variables	Mean	STD	Wilks' Lambda	Hotellings Lawley	F	Pr > F
Sex	1.64	.47	.79	.26	.92	.59
Age	42.73	9.36	.71	.39	1.37	.13
Education	14.72	2.55	.67	.47	1.64	.04
Group effects			.17	4.82	16.78	.26

Degrees of Freedom = 1, 115

A Canonical Discriminant Analysis was used to verify the results of the MANOVA. These results are found in Table 2.

Table 2

Class Level Canonical Discriminant Analysis

Statistic	Value	F	Pr > F
Wilks' Lambda	.1479	19.93	.001
Hotelling-Lawley	5.757	19.93	.001

Degrees of Freedom = 1, 115

The data were submitted for step-wise discriminant analysis as well. A subset of the test data explained more clearly the variables which best decreased the difference

between the groups. The subtests which best distinguished between the two groups were Information, Digit Span and Digit Symbol from the WAIS-R; Verbal Memory and Delayed Recall from the WMS-R; and the total score, digit span forward and backward, immediate recall for a short story and a memory for design task from the HB-CNS. Table 3 illustrates the raw Canonical coefficients for the subset variables.

Table 3

Raw Canonical Coefficients for the Subset from the WAIS-R, WMS-R, and HB-CNS

Test	Coefficient
WAIS-R	
Information	.0727360863
Digit Span	.1707477892
Digit Symbol	.1559073704
WMS-R	
Verbal Memory	.0199185551
Delayed Recall	.0131413914
HB-CNS	
HB-CNS Total	-.064368241
HB-CNS Q4	-.517431191
HB-CNS Q5	-.671011049
HB-CNS Q32	-.452050344
HB-CNS Q34	-.307278267

The Canonical Discriminant Analysis assigned each subject to one of the groups based on his or her scores on the subset of tests. The coefficients for the environmentally sensitive group ranged from -1.45 to 3.195. The coefficients for the psychiatric patients ranged from 3.174 to 6.88. An illustration of coefficients for each patient can be found in Appendix B. The subset of tests can be applied to classify subjects using discriminant statistics. Using the mean scores for both groups, Table 4 demonstrates how the coefficients, derived from this research, may be used to decrease the likelihood of misdiagnosing environmental patients. If a subject scored between -1.4 and 3.19, he or she would be classified as an environmental patient.

A class level discriminant analysis was attempted to test the ability to classify members of the two groups correctly. The "hold out" method of classification was used. The hold out method takes out one observation, computes the classification function without taking the hold out observation into account, and then uses the results of the classification function to classify the hold out observation (see Table 5). This method was used for every observation in the data pool. There were 116 observations, 10 variables and two classes, and this method classified

Table 4

Classifications for Patients Using Coefficients from Discriminant Analysis

Variable	ESX	Pi X	Coef	Coeff * ES	Coef * PI
Inf	10	11	.072	0.72	0.79
Dit Span	7	10	.17	1.19	1.70
Dit Sym	7	9	.155	1.09	1.40
Verb Mem	86	103	.019	1.63	1.95
Del Rec	86	105	.013	1.12	1.36
HB-CNS Tot	40	26	-.064	-2.58	-1.66
HB-CNS Q4	1.53	.38	-.517	-0.79	-0.19
HB-CNS Q5	1.51	.35	-.671	-1.01	-0.23
HB-CNS 32	1.44	.41	-.452	-0.66	-0.19
HB-CNS Q34	1.58	.93	-.307	-0.65	-0.30
			Total Scores:	0.2303	4.636

only three subjects incorrectly. Specifically, two subjects from the environmentally sensitive group and one subject from the psychiatric group were misclassified. This results in a 96 percent accuracy rate of classifying persons who have environmental sensitivities using the subset of test questions.

Table 5

Class Level Discriminant Analysis Between Environmental and Psychiatric Patients

Actual group	No. of cases	Predicted group membership 1	2
Group 1:			
Environmental	56	54	2
Patients		96.49%	3.51%
Group 2:			
Psychiatric	60	1	59
Patients		1.67%	98.33%

CHAPTER IV

DISCUSSION

The results of the current study strongly support the hypotheses that memory functioning is impaired in the environmentally sensitive patients and significantly different from the measurably normal memories of the psychiatric patients. Environmentally sensitive patients are able to be correctly classified 96% ($p > .001$) of the time by objective measures of memory functioning. Many of the symptoms of environmental sensitivities and psychiatric syndromes are similar, but memory is not one of the overlapping variables. None of the functional psychogenic disorders, such as anxiety, somatoform, or neurotic depression, have memory impairment as part of their diagnostic criteria, while memory impairment is a primary symptom in environmental patients.

Although the environmentally sensitive patients and psychiatric patients in this study were of comparable age and education levels, those individuals with a documented history of exposure to pesticides or organic solvents showed impairment across a wide range of neurocognitive dimensions as indicated by the significant differences ($p > .001$) of the total scores on the Harrell-Butler Comprehensive

Neurocognitive Screen. Moreover, memory dysfunction was significantly more pronounced in the environmentally sensitive group. The data suggest that exposed individuals who present with subjective cognitive complaints, particularly complaints of problems with memory and concentration, are likely to manifest significant impairment when objectively evaluated with standardized psychometrically-sound neuropsychological tests.

The data collected support the investigator's hypothesis that environmentally sensitive patients would perform differently than psychiatric patients on measures of memory functioning. Hence, if these two populations perform differently on memory functioning, they are likely being affected by two different processes. The present study was able to show that there were significant differences ($p > .001$) between the two groups on measures of short and long-term memory. The environmentally sensitive group demonstrated significantly poorer performances on measures of abilities which access retrieval of information from experience and education, immediate auditory recall, shifts in sets, psychomotor speed and dexterity, logical and verbal memory, ability to recall information after a five to thirty minute delay, and on a variety of tasks which assess general brain functioning. The psychiatric group performed within normal limits on these tasks. With such a significant difference between the two groups, it seems safe to surmise

that there are two different etiologies for these two groups.

Black et.al., (1990) concluded that psychiatric, not organic diagnoses, should be considered when dealing with patients who have previously been diagnosed with environmental illness and who have complaints of multiple ill defined symptoms. These authors suggested that physical complaints were caused by a psychiatric illness or distress. These so called ill defined symptoms are characteristic of environmentally sensitive individuals, (Bell, 1982; Riecken & Butler, 1991), however, there are also clear markers and symptoms of environmental illness. Memory dysfunction is one of the markers that differentiates the environmentally ill patient from the psychiatric patient. Additionally, the symptom pattern, as well as the history and blood serum analysis, provide a much better defined basis for diagnosis, particularly for cause and effect, than does any psychiatric diagnosis, not based on organicity. Further, symptoms for environmental illness may vary in frequency and intensity, depending on exposure and total stress load, and as these changes occur symptoms may also change accordingly, thus appearing to be ill defined.

Performance on the test battery presented in the present study is objective. The environmentally sensitive patients scored significantly poorer than the psychiatric patients on measures of objective memory. The results

indicate that utilization of the same standardized tests would enable clinical researchers to correctly classify environmental patients by their performance on the tests 96 percent of the time.

There can be great value in using such a screening device because it would decrease the chances of misdiagnosing and mistreating patients. Patients with memory deficits who have been diagnosed with psychiatric disorders and who do not respond effectively to medications or psychotherapy may well have been misdiagnosed. Environmental illness should be considered as a possible factor. If treatment is not effective in providing a high measure of relief or a cure, it is no wonder that patients seek other means of treatment or other doctors.

Riecken and Butler (1991) surveyed of 89 environmentally sensitive individuals and found that most had at least one previous form of unsuccessful treatment. Brodsky (1983) studied eight previously diagnosed environmentally ill patients and reported that all had a history of "doctor shopping." To consider such patients as "doctor shopping" is unfair and unhelpful labelling which will bias most examining physicians and increase the chances for continuing incorrect interventions. Using the coefficients obtained as a screening device in conjunction with other diagnostic indicators has the potential for saving much time and money for the patient, physician, and

society. Timely and accurate diagnosis also may decrease the development of secondary psychiatric problems which often occur in the environmentally ill as their sensitivities go untreated.

Many environmental patients also demonstrate symptoms of depression and anxiety because they have to contend with the insidious changes and deficits in functioning caused by environmental toxins. Such changes, however, are insufficient for psychiatric labelling. Diagnoses of anxiety, depression, or somatoform disorders are secondary to recognized physiological insults (toxins and chemicals), which are known to generate biopsychological disorders, and therefore, would exclude a primary diagnosis of a psychiatric disorder. As indicated by the significant differences on the Harrell-Butler Comprehensive Neurocognitive Screen between the psychiatric and environmental patients, the environmental patients show deficits in a variety of areas besides memory. Their scores may well be indicative of diffuse brain dysfunction so that CNS dysfunction occurs. Many psychological-neurocognitive symptoms are likely to occur. These symptoms can confuse the psychiatrist and psychologist not knowledgeable about the adverse effects of exposure to environmental toxins/poisons and who might too quickly label such symptoms as a psychiatric syndrome.

Many pesticides and organic solvents have an affinity for fatty tissues such as those insulating nerve tissue. Given that pesticide or organic solvent poisoning is so rarely considered in a regular evaluation of symptoms, there is a possibility that many people diagnosed in the early stages of syndromes such as Alzheimer or Parkinsons could be showing symptoms of pesticide or organic solvent poisoning.

There are several famous cases in the literature concerning this possibility. For example, Parkinsonism has been produced by injecting the designer drugs dehydro-4-phenyl-N-methylpiperidine (DPMP) (Davis, Williams, Markey, Ebert, Caine, Reickert & Kopin, 1979) and 1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine (MPTP) (Langston & Ballard 1983). MPTP is actively taken up by the dopamine uptake system. Langston (1985) has suggested that most cases of Parkinsons result from an environmental insult, such as exposure to MPTP, that causes the death of some fraction of the nigral neurons during midlife. Langston also points out that exposure does not have to be by injection. Five chemists, who had been working with MPTP, developed early symptoms of Parkinsons. They did not inject the substance but either through inhalation or cutaneous contact had developed Parkinsons. MPTP may be found in the pesticide cyperquat.

Alzheimers may be associated with exposure to certain organic solvents. Edling et al. (1990) investigated the relationship between organic solvent exposure and

Alzheimer's disease. They found that the longer one is exposed to organic solvents (i.e., occupation using organic solvents) the more deviant one performs on neuropsychological evaluations. Kandel (1989) described a 69-year-old man who had been diagnosed with Alzheimer's disease but who actually had long-term exposure to solvents used in dry cleaning.

The present study demonstrates clear support for the hypothesis that it is possible to differentiation between environmental illness and psychiatric disorders using memory impairment as a marker of EI. It also proposes the use of a helpful screening device, which could be used in conjunction with blood serum and history of exposure to environmental toxins/poisoning, to make these determinations.

APPENDIX A

INFORMED CONSENT FORM

SUBJECT INFORMED CONSENT FORM

The purpose of this research project is to ascertain to what extent memory functioning may be used to determine criteria for the diagnosis of environmentally sensitive individuals.

Subjects will be asked to complete a regime of psychological tests which evaluate memory functioning. These tests include the Wechsler Adult Intelligence Scale-Revised, the Wechsler Memory Scale-Revised, and the Harrell-Butler Comprehensive Neuropsychological Screen.

The risks for your involvement are negligible.

The benefits of this research project to the subject is objective evaluation of one's memory. This may help soothe worry about adverse side effects of current medication or may help subject to decide to change to a medication which may reduce memory side effects. Also by participating in the research project the subject may be screened for early signs of organic dysfunction.

"I agree to participate in this research project as it has been described to me. I know that I am free to discontinue my participation at any time during the session and to not answer any questions put to me. I understand that all my responses will be confidential."

_____ _____

Subject's Signature Date

Phone: ( ) -

APPENDIX B

RAW CANONICAL COEFFICIENTS

FOR EACH SUBJECT

Raw Canonical Coefficients for Each subject

Group 1

Subject #	Coefficient	Subject #	Coefficient
1	.01906	31	-.47861
2	.08439	32	1.71271
3	.70561	33	-.04295
4	0.49787	34	.13123
5	-.66314	35	.79491
6	.36979	36	1.18187
7	-.33379	37	.67620
8	-1.45552	38	-.28643
9	2.90710	39	2.34120
10	-.31946	40	-.25769
11	-.04828	41	1.27024
12	2.19018	42	2.45968
13	2.21973	43	.74772
14	2.68402	44	-.16984
15	.99162	45	.90297
16	.31170	46	.94637
17	-.08245	47	2.11587
18	3.08437	48	1.09471
19	.01285	49	.45679
20	1.41894	50	.53929

Appendix B--Continued

Subject #	Coefficient	Subject #	Coefficient
21	-.68641	51	1.38513
22	-1.91496	52	.43204
23	1.37916	53	1.30009
24	1.27323	54	.56531
25	1.66574	55	.90853
26	.94302	56	.42416
27	-.20524	57	-.00096
28	.59627		
29	2.21019		
30	.61713		

Group 2

Subject #	Coefficient	Subject #	Coefficient
1	4.19004	31	5.05121
2	4.96591	32	3.82273
3	3.94911	33	5.94487
4	5.61133	34	3.82256
5	3.76289	35	5.06861
6	6.04664	36	5.10456
7	5.36488	37	6.68527
8	5.72387	38	5.85877
9	5.89619	39	4.01949
10	4.06123	40	4.39610

Appendix B--Continued

Subject #	Coefficient	Subject #	Coefficient
11	5.50693	41	5.88044
12	6.06642	42	5.37762
13	6.80695	43	5.04056
14	6.88573	44	5.74682
15	7.90802	45	5.80517
16	5.68188	46	5.50760
17	4.68402	47	4.61724
18	3.26214	48	6.06053
19	3.83111	49	3.90862
20	3.59780	50	4.88320
21	5.58095	51	4.70098
22	4.37195	52	5.32956
23	4.52428	53	4.53992
24	5.73270	54	4.27315
25	5.98337	55	5.27692
26	5.29956	56	3.75692
27	3.17520	57	5.88478
28	5.90450	58	5.24530
29	5.50077	59	4.99709
30	4.49308	60	5.24849

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