ECOLOGICAL TREATMENT AND FASTING EFFECTS ON
PSYCHOLOGICAL MEASURES

DISSERTATION

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The purpose of the present study was to objectively verify psychological and behavioral changes in a group of identifiably susceptible patients who were treated for environmental sensitivities in a highly controlled environment. The subjects were 71 Environmental Control Unit (ECU) patient volunteers (55 females, 16 males, age range 17-71) and 16 nonpatient volunteers (12 females, 4 males, age range 16-52) for a total of 87. The patients were divided into a short-treatment (fasting 3-4 days) group ($n = 35$) and a long-treatment (fasting 5-7 days) group ($n = 36$). A third group, consisting of the 16 nonpatients, formed a nonpatient/no-treatment control group. All subjects were administered tests for assessment of intellectual/cognitive functioning, neurological/cerebral functioning, perceptual-motor skills, mood states, and general psychopathology. Results indicated that before and after fasting in an environmentally controlled condition there were significant differences among the groups in neurological/cerebral functioning, general psychopathology, depression, and anxiety.
There were significant improvements in several areas upon posttreatment evaluation. Although both the short- and long-treatment groups improved in perceptual-motor skills, only the long-treatment group improved in depression, anxiety, and intellectual/cognitive abilities. Also, the short-treatment group alone improved in neurological functioning. It was concluded that fasting had a positive effect which has implications for both physical and psychological treatment of environmentally sensitive patients and which perhaps could be generalized to a broader range of psychological and medical patient groups.
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ECOLOGICAL TREATMENT AND FASTING EFFECTS ON
PSYCHOLOGICAL MEASURES

Clinical ecologists have been concerned with the application of ecological principles to the diagnosis and treatment of health problems resulting from the effects of environmental incitants (Dickey, 1976). Any environmental substance which disturbed an individual's physiological homeostasis constituted an incitant. Individual susceptibility (governed by genetic background), preexisting state of physical and mental health, and individual adaptive capacity, determined sensitivities to potential incitants as well as the nature of the subsequent reactions.

Although clinical ecologists have attributed a variety of somatic and psychological problems to environmental incitants, much of the research intended to confirm this relationship has been criticized for failing to control for possible multiple sensitivities (Mayron, 1979). Rea (1979) believed that multiple sensitivities and constant exposure to incitants masked diagnostically significant changes in susceptible individuals. Masking occurred when physiological adaptation to one or more incitants obscured symptoms from another incitant. This resulted in misdiagnoses and confounded research results. In order to avoid masking as well as other problems with diagnosis and
treatment of environmental sensitivities, Randolph (1962) developed a method of extraordinary environmental control through which to study ecological disorders. Until now, few studies have been done in such a controlled environment, especially studies utilizing objective measures of somatic and psychological change.

Health and Environmental Incitants

Beaconsfield, Krebs, Borlaug, and Rainsbury (1975) discussed the role of "internal pollution" in illness. Their list of pollutants included inhaled environmental pollutants, chemical food additives, impurities in prepacked foods, and medications. Feingold (1975a) believed certain foods and food additives contributed to a variety of health problems. LaVerne (1970) attributed slowed healing processes to air pollution. Randolph (1962) linked many physical and psychological difficulties to individual susceptibility to the chemical environment.

Numerous diseases have been attributed to environmental incitants. Some examples were urticaria (Michaelsson & Juhlin, 1973), asthma (Chaffee & Settipane, 1967; Juhlin, Michaelsson & Zetterstrom, 1972), emphysema and bronchitis (LaVerne, 1970), headache (McGovern & Haywood, 1970), celiac disease (McCuffin, Gardiner, & Swinburne, 1981), thrombophlebitis, parotiditis sicca (Rea, 1976, 1979b) and vasculitis (Rea, Bell, & Smiley, 1980).
Mayron (1979) explained that the circulatory system may carry incitants to any organ in the body. Rea (1979), stated that any part of the smooth muscle, mucosa, and collagen systems can be affected by environmental incitants. 

Psychological and Behavioral Disorders

In 1922, Shannon reported allergic reactions in the nervous system. He believed behavior problems could be primary allergic reactions in some instances.

Subsequent to Shannon's report, several authors have attributed psychological and behavioral disorders to foods and chemicals encountered in the environment. Schneider (1945) thought more attention should be given to the role of allergy in either causing or accentuating emotional disturbances. Davison (1949) reported cerebral allergy may result in sleep disturbance, inability to concentrate, childish compulsions, feelings of unreality, general unhappiness, morbid depression, loss of pride, and loss of sexual interest. Clarke (1950) described character problems due to allergy. Speer (1954, 1970) identified an allergic tension/fatigue syndrome in which motor and sensory overactivity alternated with motor and memory fatigue. Leonard (1966) found a group of allergic children had reduced dexterity, more symptoms of anxiety, increased sensitivity to cold, more nervous symptoms, and more withdrawing tendencies than nonallergic children. Feingold (1975) attributed hyperactivity in many children to foods
and food additives. In one of their patients, Fand and Hubbard (1976) found anxiety attacks, followed by depression, resulted from food allergy.

Campbell (1970) reviewed behavior disorders resulting from allergy of the nervous system, including reports of emotional immaturity reactions, antisocial behavior, depressive symptoms, anxiety, organic brain syndrome reactions, schizophreniform reactions, and habit reactions. **Mechanisms**

Proposed mechanisms, through which environmental incitants affect health, appear to fall into the two broad categories of toxic and immunologic reactions. Taylor (1979) distinguished between toxic and immunologic effects on the body. A toxin damaged the body directly and upon first contact. Alternatively, an allergen triggered the body's immune system only after sensitivity had been established on a previous exposure. It was the allergens' subsequent interaction with the immune system which produced symptoms. **Toxic Mechanisms.** LaVerne (1970) proposed that air pollutants have a toxic effect on the body's healing processes. He stated that toxins may pass from the blood through cell membranes into any tissue in the body. Brain tissues were especially susceptible. Inhalants (such as carbon monoxide, sulphur dioxide, oxides of nitrogen, and hydrocarbons) were believed to enter protoplasmic as well as cellular solution. There they ionized, then exerted
noxious effects on chemical, enzymatic, bioelectric, and neuronal processes. LaVerne reported that this mechanism was by no means validly proven, but he believed it should not be considered entirely speculative.

Feingold (1975b) believed that toxic effects from certain foods and food additives produced hyperkinesis and learning disabilities in children. He equated food additives with drugs and proposed that they may have similar psychopharmacological effects in predisposed individuals. Genetic variations in metabolic processes, enzymatic variations, and receptor site anomalies provided possible mechanisms for individualized psychopharmacological reactions. Taylor (1979) criticized Feingold's proposed mechanisms for lack of supporting research data.

Immunologic Mechanisms. McGovern and Haywood (1970) described a four-part immunologic mechanism in allergic reactions: (a) an antigen–antibody reaction, (b) release or formation of chemical mediators, (c) a physiopharmacologic action of mediators within tissues, and (d) a target tissue response resulting in observable physical symptoms. Suggested mediators were histamine, kinins, slow reaction substance, serotonin, catecholamines, complement, and anaphylotoxin.

Mayron (1979) defined allergy as an abnormal response to a food, chemical, or inhalant to which most other people
do not respond. This abnormal response involved antibody or immunoglobulin mediation. Five types of immunoglobulins (designated IgA, IgD, IgE, IgG, and IgM) were believed to be involved. Most people with inhalant allergies had IgE mediated responses.

IgE mediated allergies appeared to be more widely accepted as a clinical entity (May, 1975), while food and chemical sensitivities were controversial and less understood (Golbert, 1975; Mayron, 1979; Rapp, 1978). Mayron (1979) believed the controversy derived from a failure of many clinicians to recognize the fact that other immunoglobulins besides IgE mediated sensitivity reactions. He preferred the term "immunologic sensitivity," rather than allergy, to refer to the more broadly defined reactions.

In an attempt to confirm the concept of food allergy, Mayron (1978) reviewed research supporting the position that foods may enter the circulatory system, thus becoming allergens. He concluded that allergen absorption may occur through almost any surface of the body with the lungs and gastrointestinal tract provided the greatest surface area for absorption.

Edema was a frequently mentioned allergic reaction in the brain (Clarke, 1944), which was thought to affect psychological functioning. Although Taylor (1979) believed the role of brain edema in allergic reactions was speculative, Campbell (1970) indicated that both edema and
hypoxia resulting from vascular injury were implicated to nervous system allergic reactions. Rea (1976, 1977, 1980) provided support for Campbell's beliefs with reports of inflammatory reactions resulting in vascular injury throughout the body.

Randolph (1962) took an ecological approach to the mechanisms of environmental sensitivities. Randolph believed human ecology embodied the concept of adaptation to natural and man-made environmental substances. He described a specific adaptation syndrome through which the body adapted to its environment. This was a clinical counterpart to Selye's general adaptation syndrome.

Randolph's specific adaptation syndrome consisted of three stages. The nonadapted developmental stage was an acute reaction in an individual who either previously had not been exposed or had not recently been exposed to the offending incitant. Repeated frequent exposures to an incitant resulted in a reduction of acute symptoms as the body adapted to the incitant, the adapted stage. Then, at a critical level of exposure, the individual's capacity to adapt became exhausted. At this point, the individual was no longer adapted. Disease processes, referred to as maladaptive responses, occurred next. This was the maladjusted stage.
Diagnosis

The diagnostic process for environmental sensitivities has typically involved identification of offending incitants. In the course of clinical experience, Feingold (1968) came to believe that certain patterns of clinical history were diagnostically significant in patients with toxic reactions to foods and food additives. The most important clue to sensitivity for Feingold was variation in the patient's dietary pattern and particularly the addition of dietetic beverages and dietetic foods. In addition, Feingold looked for a history of aspirin sensitivity and nasal polyps. He also suspected toxin sensitivity in patients who either did not respond to allergy skin tests when their history indicated they would, or who did react to allergy skin tests but did not respond to routine allergy management at some point in their treatment. Rather than attempting to identify a specific incitant, Feingold placed his patients on a diet free of certain classes of chemicals. He assumed that if the patient responded favorably, then one or more of the chemicals was the offending incitant.

Immunological sensitivities have been diagnosed in a similar manner. When Rowe (1931) observed a syndrome typical of food allergy, she first ruled out other possible causes of the symptoms, then attempted to identify the offending food. She took a dietary history of the patient's
likes and dislikes to find potential allergens. Next, she used diet trials to verify her diagnosis. In symptom-free patients, she attempted to reproduce symptoms by having them ingest suspected foods. Drug allergies were similarly diagnosed. Inhalant allergens were identified with skin tests.

Randolph (1962) stated environmental sensitivities were difficult diagnoses to establish because of individual variations in manifestations and potential scope. He used a history, as did Rowe, for clues to offending incitants.

Randolph developed a method of comprehensive environmental control to aid in diagnosis and treatment of his patients. He believed that diagnosis depended principally on observing patients while their environment was manipulated. In practice, this meant the patient was observed while simultaneously avoiding all potential foods, drugs, and environmental incitants (including dust, pollens, and other inhalants). Through this method, the patient was believed to return to a symptom-free, nonadapted state. Next, suspected incitants were returned to the patient's environment one at a time. Incitants to which the patient was sensitive were identified by the reactions they caused in patients.

The reactions Randolph reported using for diagnosis were not always clearly defined. He grouped them into two
multiple-leveled phases, the stimulatory or pick-up phase and the withdrawal or hangover phase. The pick-up phase immediately followed a given exposure, while the hangover phase occurred after the incitant was withdrawn. A given reaction was potentially manifested at any level of the two phases.

Pick-up reactions were predominantly motor in type. Although otherwise symptom-free, activity, alertness, and relative stimulation characterized the first-level pick-up phase. Energetic bursts of physical activity and hyperactivity (in which the individual remained nervous, keyed-up, and irritable for several hours) characterized the second level. At the third level, the individual became flushed in the face, clumsy, ataxic, argumentative, and aggressive. The fourth-level pick-up phase was characterized by a state of uncontrollable agitation and excitement with rhythmical muscle contractures ranging from muscle twitching to flailing of extremities and epileptiform seizures. Both transitory and more prolonged loss of consciousness also occurred.

Initially, hangover reactions were localized principally to major points of body contact. Thus, typical first-level hangover reactions were irritation, rhinitis, burning of the lips and skin, pruritis, bronchitis, and mild gastrointestinal or other relatively minor symptoms.
At the next level, localized hangover symptoms consisted of more severe chronic respiratory symptoms (such as nasal obstruction, sinus involvement, severe coughing, and bronchial asthma), various dermatoses, a wide range of more troublesome gastrointestinal manifestations, and sometimes urgent and frequent urination. Mild constitutional symptoms such as physical and mental fatigue frequently accompanied the chronic localized symptoms. These constitutional symptoms usually took the form of tiredness, reduced energy level, loss of initiative, loss of enthusiasm for work, forgetfulness, difficulty in thinking, poor concentration, reduced reading comprehension, and sometimes a relative impairment in the sense of humor. Additional frequently occurring symptoms at this level were headaches, various musculoskeletal pain syndromes (including myalgia, fibrositis, bursitis, arthralgia, and arthritis), neuritis and certain other neurological symptoms, as well as more generalized effects such as edema, palpitation, excessive perspiration, pallor, and weakness.

The third-level hangover symptoms were characterized by chronic constitutional syndromes such as more advanced mental confusion and mild depression. Individuals varied considerably, but symptoms included a tendency to fixed ideas, one-track thoughts, and asocial attitudes. Some became either morose, sullen, seclusive, hostile, or paranoid.
Others were described as negative to suggestion, dopey, groggy, or so indifferent to their surroundings as to approach lethargy.

The fourth and deepest level of hangover reaction was characterized by severe depression, disorientation, and regression. Sometimes patients also hallucinated, became delusional, or became amnesic.

Some clinical tests have been developed to assist in identifying environmental sensitivities. Many of these tests were controversial (Golbert, 1975), but some support for their usefulness existed (Dickey, 1976; Green, 1974). Some of these clinical tests were the skin sensitivity test, the intracutaneous provocative test, the sublingual provocative test, deliberate feeding, the pulse test, and the leukocytotoxic test (Bryan & Bryan, 1960).

Mayron (1979) said that much of the controversy over these tests arose from a failure to consider reactions other than those involving IgE mediation. The IgE mediated skin sensitivity test appeared to be the most widely and (for some) the only accepted allergy test (Golbert, 1975).

Rea (1979) believed most environmental sensitivities did not involve IgE mediation. Following Randolph's (1962) suggestion, Rea recommended challenge testing, in a contamination-free environment, supported by laboratory data, as the most effective diagnostic procedure.
Challenge testing is the administration of a small amount of a suspected incitant to a patient with the intent of eliciting symptoms and signs which confirm a sensitivity to the substance administered. Rea believed challenge tests administered in a contamination-free environment avoided masking.

Research

Numerous case reports (Akerley, 1976; Chaffee & Settipane, 1967; Crook, Harrison, Crawford & Emerson, 1961; Crowe, 1941; Feingold, 1975; Shannon, 1922; Speer, 1954) have attributed various symptoms to the somatic effects of environmental incitants. Until recently, few empirical studies were reported.

Most investigations have evaluated the effects of small amounts of suspected incitants on susceptible individuals. Among these studies were some in which the challenge tests were made while the participants remained on their normal diets. Green (1974) evaluated the technique of sublingual provocation testing and found it effective in eliciting reactions to numerous foods, dyes in foods, drugs, and cosmetics. Douglas (1975) and Juhlin, Michaelsson, and Zetterstrom (1972) elicited allergic reactions, such as urticaria, during clinical challenge tests with aspirin and food additives.
Elimination diets have been studied in several types of investigations. Reports from uncontrolled clinical trials (Baldwin, Kittler & Ramsay, 1968; Cook & Woodhill, 1976; Crook, 1980; Salzman, 1976) supported the use of elimination diets to alleviate allergy and hyperactivity resulting from food and food additive sensitivities.

Elimination diets combined with single-blind challenge tests were used to diagnose sensitivities to foods in hyperactive (Rapp, 1978a), schizophrenic (Dohen & Graesberger, 1973) and urticaric (Michaelsson & Juhlin, 1973) patients.

The most frequent topic of recent investigations appeared to be that of the role of foods and food additives in causing hyperactivity (Conners, Goyette, Southwick, Lees & Androulonis, 1976; Goyette, Conners, Petti & Curtis, 1978; Harley, Ray, Tomasi, Eichman, Mathews, Chun, Cleeland & Traisman, 1978; Levy, Dubrell, Hobbes, Ryan, Wilton & Woodhill, 1978; O'Shea & Porter, 1981; Rapp, 1978; Williams, Cram, Tausig, Webster, 1978). Such studies found conflicting evidence for the effectiveness of elimination diets in alleviating hyperactivity. Taylor (1979) reviewed this topic and concluded that further work in the area was justified.

King (1981) tested the hypothesis that sublingual exposure to allergens would produce cognitive/emotional symptoms in allergy patients. He conducted double-blind
provocative tests with allergy patients complaining of at least one psychological symptom. Dependent measures (including self-report, heart rate, psychological performance, and mood techniques) were obtained within 10 minutes after exposure to the suspected allergens. Patients reported significantly greater cognitive/emotional symptoms following exposure to allergens. Placebo effects were not significant, and mood and performance measures were not affected by allergen exposure.

King believed that self-report may have been the most sensitive measure of reaction because patients included in their reports any momentary allergic symptoms occurring subsequent to exposure. Other techniques assessed only effects occurring at the time they were administered during the 10 minutes following exposure. Also, Randolph (1962) suggested that masking effects confound results in procedures such as Kings.

Research which involved fasting fell into two categories. The first involved simple avoidance of food without particular attention to other sources of environmental incitants. The second category involved an attempt at total environmental control through extraordinary means of eliminating all potential incitants, as recommended by Randolph (1962).
Strict fasting has been used to treat a variety of somatic diseases in the Soviet Union (Boehme, 1977). On the list of disorders so treated were functional psychosis, manic depressive states, involutional psychosis, epilepsy, posttraumatic encephalopathy, encephalitis, certain endocrine disorders, and hypertension. Although the Soviets had investigated numerous physiological changes resulting from their fasting treatment, they apparently had not linked observed symptoms or changes to immunological or toxic effects of ingestants.

Klotz (1976) gave 11 residential school students with various diagnoses nothing but distilled water. The students remained on this fast until their symptoms had cleared for 12 hours. Although the students were said to be in chemical-free environments, it was not clear what measures were taken to eliminate potential incitants. Of the 11 students, 8 became distinctly improved or symptom-free following the fast, then exhibited return of symptoms following single food meals.

O'Banion, Armstrong, Cummings, and Stange (1978) studied behavior changes in an 8-year-old autistic boy. They confined the boy to an uncarpeted room equipped with a bed and toys. A 6-day medically supervised fast on spring water was followed by single food challenge tests. All foods were obtained from "organic" sources to prevent
possible confounding effects of chemical additives, preservatives, pesticide residues, and other contaminants.

O'Banion et al. observed rates of hyperactivity, uncontrolled laughter, and disruptive behaviors (e.g., screaming, biting, scratching, and object throwing). The rates of these behaviors increased following consecutive ingestion of single food meals of wheat products, corn, mushrooms, tomatoes, milk, and raw cane sugar. No reactions occurred following ingestion of several additional foods or meals combining nonreactive foods. O'Banion et al. cautioned future investigators to allow for the variations in reaction times they observed during their study.

The most thorough control of potential incitants appeared to have been achieved by Rea (1979) and his associates in their Environmental Control Unit (ECU) in Dallas. This unit was constructed following the recommendations of Randolph (1962). Edgar, Fenyves, and Rea (1979) evaluated this unit and concluded it was adequately free of potential inhalant and contact incitants. In this unit, patients fasted on water empirically proven to be incitant free, attempting to achieve maximum possible avoidance of environmental incitants. Challenge tests conducted in this environment were believed to be essentially free of masking from unrecognized incitants.
Rea, Bell, Suits, and Smiley (1978) studied 12 patients who had developed signs and symptoms of inflammatory diseases following overexposure to commonly used environmental chemicals. The majority of symptoms and signs cleared in the ECU. Double-blind exposures to ambient dose levels of synthetic chemicals reproduced most of the symptomatology. Laboratory findings included abnormalities in complement, T-lymphocytes, eosinophils, and IgG.

Rea, Bell, and Smiley (1980) studied 12 selected patients with large-vessel vasculitis-related symptoms. Following a period of fasting in the ECU, 10 of the 12 patients had their symptoms alleviated. The symptoms were reproduced in 10 patients with double-blind challenges of individual foods and chemicals to which the patients were susceptible. Laboratory tests revealed changes in IgG, eosinophils, complement, and T-lymphocytes.

Treatment

Treatment of environmental sensitivities took several forms. Symptoms were treated with drugs such as antihistamines (Fishbein, 1969). Desensitization by administering small amounts of the incitant through injection or sublingual absorption was a common practice (Golbert, 1975). Avoidance measures were widely applied also (Randolph, 1962; Rea, 1979). Feingold (1975) and Rowe (1931) advocated elimination diets to avoid or reduce ingested incitants.
Randolph (1962) was an early advocate of avoidance practices covering many substances. His patients were frequently required to avoid indoor and outdoor air pollutants, chemical food additives, contaminants of food and water, and many synthetics such as drugs, cosmetics, and textiles.

Criticisms and Recommendations

Various criticisms and recommendations have been made with respect to research on the effects of environmental incitants on susceptible individuals. Dietary infractions were frequently suspected of confounding results (Cook & Woodhill, 1976; Rapp, 1978; Williams et al., 1978). May (1975) and Mayron (1979) objected to reliance on subjective measures of symptoms for criteria purposes. Goyette et al. (1978) and O'Banion et al. (1978) recommended attention be given to variability in reaction times following challenge tests. Williams et al. (1978) suggested using populations of identifiably susceptible individuals. Taylor (1979) requested replication of existing studies, inclusion of objective measures of change, more independent evidence of allergy, and more attention to the possibility of predicting change.

Mayron (1979) examined food-dye-related immunological sensitivity studies that obtained negative results. He found one primary difference from studies with positive results.
The negative studies ignored potential masking by allergies to commonly eaten foods. To avoid the problem of masking effects, Mayron recommended the identification and elimination of any dietary incitants previous to challenge tests with food dyes.

Rea (1979) reported several clinical principles which must be considered in studies of environmental sensitivities. One was that clinicians were not usually aware of the chemical environment's role in disease and were likely to overlook such effects in diagnosis. Another was the concept of total body load. This was the total of all environmental incitants which the body's homeostatic mechanisms handle at one time. Symptoms were believed to occur when the total body load exceeded the body's capacity to handle the cumulative effects of the total load. A third principle was that of masking. Frequently repeated exposure to any given incitant placed the body in an adapted state which appeared to be relatively symptom free. A clearly definable reaction only occurred after that incitant and any other incitants to which the individual may have become adapted had been avoided long enough for the adaptation to clear. Lastly, a bipolar reaction was often observed. Some individuals had a stimulatory reaction until that point in time when the body's defenses broke down and more debilitating symptoms appeared.
In summary, numerous case reports described physiological, psychological, and behavioral disorders resulting from exposure to environmental incitants (Akerly, 1976; Chaffee & Settipane, 1976; Crook et al., 1961; Crowe, 1941; Feingold, 1975; Kittler, 1970; Randolph, 1967; Rea, 1979; Rowe, 1931; Shannon, 1922; Speer, 1954). Studies involving various designs and degrees of control have found some support for attributing these problems to various environmental incitants (Mayron, 1979; Taylor, 1979). However, several problems existed in past studies. They frequently did not use objective measures of change; they did not eliminate masking by uncontrolled and unrecognized incitants; they did not allow for individual differences in reactions; and they did not use populations of identifiably susceptible individuals.

The purpose of the present study was to objectively verify psychological and behavioral changes in a group of identifiably susceptible patients who were treated for environmental sensitivities in a highly controlled environment. Hypotheses tested were that susceptible individuals in an adapted state (when administered appropriate objective measures) would

1. improve as to their intellectual/cognitive and neurological/cerebral functioning, and ability to learn a new task.
2. decrease their level of psychopathology (depression/anxiety), and

3. improve until such time as they were free of the effects of incitants (symptom free) or were again exposed to an incitant and their functioning deteriorated.

**Method**

**Subjects**

The subjects were 71 Environmental Control Unit (ECU) patient volunteers (55 females, 16 males, age range 17-71) and 16 nonpatient volunteers (12 females, 4 males, age range 16-52) for a total of 87. All the ECU patients had been referred for diagnosis and treatment of suspected hypersensitivities to environmental incitants. Referrals typically had been made after treatment failures in various outpatient settings. The nonpatients were solicited from among the employees of another area hospital.

The subjects were divided into three groups. The 71 patients were divided into a short-treatment (fasting 3-4 days) group \( (n = 35) \) and a long-treatment (fasting 5-7 days) group \( (n = 36) \). The third group, consisting of the 16 nonpatient volunteers, formed a nonpatient/no-treatment control group.

**Assessment Techniques**

The revised WAIS was standardized on a representative stratified sample of the population of the United States. The average reliabilities across age groups were .97 for Verbal IQ, .93 for Performance IQ, .97 for Full Scale IQ, and .82 for the Digit Symbol subtest. Wechsler believed that, since the WAIS-R measured the same abilities as the 1955 WAIS and overlapped considerably in test content of the earlier scales, validities of these scales were essentially the same.

The State-Trait Anxiety Inventory (STAI, Spielberger, Gorsuch & Lushene, 1970) assessed two theoretically distinct anxiety conditions. State anxiety was a transitory condition of perceived tension. Trait anxiety was a relatively stable condition of anxiety proneness. This 40-item inventory had 20 items assessing current feelings and 20 items assessing general feelings. Test-retest reliabilities for the trait scale ranged from .73 to .86. Test-retest reliabilities for the state anxiety scale were low, having a median \( r \) of .32 across six samples. Appropriately, these low reliabilities reflected the influence of planned differing situational factors existing at the time of evaluation.

Both concurrent and construct validities were reported for the STAI. Concurrent validity was determined by correlations with the IPAT Anxiety Scale (\( N = 112, r = .77 \),
Cattell & Sheier, 1963) and the Taylor (1953) Manifest Anxiety Scale ($N = 66, r = .83$). Construct validity was demonstrated by two studies in which the state anxiety scale significantly discriminated between normal and anxiety-producing situations (Spielberger, Gorsuch & Lushene, 1970).

The Self-Rating Depression Scale (SDS, Zung, 1966) quantitatively assessed depression. This scale has been shown to differentiate depressive patients from those with other psychiatric disorders, and to reflect clinical changes following treatment for depression (Zung, Richards & Short, 1965). The SDS scores correlated significantly with those on the MMPI depression scale.

The Trail Making Test A & B (TMT, Reitan, 1966) assessed frontal lobe functioning. It was recommended by Walsh (1978) as a single-test screening instrument for brain damage. This test required the patient to connect a series of numbered circles in ascending numerical order (Part A), then to connect a series of numbered and lettered circles in alternating ascending numerical and alphabetical order (Part B). This task required planning, regulating, and checking a program of action.

Walsh (1978) recommended Reitan's (1966) Finger Oscillation Test as a neurological test. This test consisted of determining the maximum frequency at which a lever could
be depressed during 5 consecutive 10-second trials. A rest break was given after every third trial. A Veeder Root model 727245-001 counter was used for this task.

Additional data were provided by two other instruments. The Minnesota Multiphasic Personality Inventory (MMPI, Hathaway & McKinley, 1967) assessed psychopathology. The Bender-Gestalt Test (Bender, 1938) assessed visual-motor coordination, and scored utilizing the Embree/Butler scoring sheet (see Appendix A).

Procedure

Immediately upon admission, all patients at the ECU began fasting as the initial phase of their treatment. No later than the evening of their second day in the ECU, all patients admitted were asked to participate in a study intended to evaluate the treatment they received. Each patient was assured that participation in the present study was not required. They completed their initial assessment on the first or second day in the unit. For some patients, during their fast, the physician in charge decided it was necessary to begin intracutaneous challenge tests of some nonfood potential incitants. Beginning with the fourth day of their treatment, the participating patients were requested to begin their second and final assessment.
Both initial and final assessments were administered to the patients in two parts. The first part was administered by the psychology staff of the ECU. It consisted of the WAIS-R, the MMPI, and the Bender. These tests were part of the battery routinely administered at the ECU for differential diagnosis of psychological and ecological disorders. The second part of the assessment was administered by the author. It was referred to as the partial battery and consisted of the State-Trait Anxiety Inventory, the Self-Rating Depression Scale, the Finger Oscillation Test, and the Trail Making Test A and B.

For the patients, the two parts began within 24 hours of each other for both the initial and the final assessments. Completion of the first part of the battery, especially the MMPI, was delayed if a patient was unwilling to spend the time required to finish that part of the assessment in one session. Completion of both administrations of the partial battery were always within 45 minutes of the times they began.

While fasting, the patients consumed only water which had been empirically proven to be incitant free. Typically, the water consumed was glass-bottled spring water.

By the sixth day of treatment, most patients ended their fasts and began eating single food meals of unprocessed "organic" foods. The exact times the patients
ended their fasts were determined by the physician in charge according to the patient's needs. Typically, the physician ended a patient's fast when the patient's symptoms cleared or when an extreme physical condition, such as very low weight, made it imprudent to continue.

The foods consumed after the patients' fasts ended were produced without synthetic chemicals such as fertilizers, insecticides, herbicides, ripening agents, or preservatives. Any foods resulting in a reaction were eliminated from the patient's diet. No more than 4 foods were tried within a 24-hour period. Testing of the next food began after any reaction to the previous food had subsided.

The presence of a reaction was determined by two types of criteria. Patients used a self-report checklist prepared from clinical experience (see Appendices B & C) to record their subjective experiences. Also, clinical observation of such signs as increased pulse rate, skin changes, and respiratory difficulties were recorded by the medical staff (see Appendix D).

The control group was administered both parts of the assessment battery by the author on two occasions, which were similarly spaced to the patient's two assessments. During the time between their two assessments, the control group conducted their lives routinely.
Results

The eight variables are analyzed in four ways. Analyses of variance determine if the three groups differ at initial testing. Then, t-tests determine if there are differences between initial scores and those at retest. Next, additional analyses of variance determine if the groups differ in their retest scores. Finally, analyses of covariance, on the posttreatment scores of each variable (with the pretreatment scores for that variable as covariate), determine if there are differences among the groups with respect to the degree of change between their initial scores and their retest scores. Tables of relevant results are reported in Appendix E through Appendix Q.

All statistical analyses are performed with the Statistical Package for the Social Sciences (SPSS, Nie, Hull, Jenkins, Steinbrenner & Bent, 1975) and its updates (Hull & Nie, 1981). The analyses of variance are performed with the SPSS Oneway subprogram, which uses the Scheffe procedure for post hoc comparisons. The T-test subprogram is used for the t-tests. The Manova subprogram is used for the analyses of covariance.

Zung Depression Scale. The analysis of variance on the pretreatment Zung Depression Scale scores of the three groups is significant, $F(2,84) = 20.73, p \leq .0001$ (see Table 1, Appendix E). The comparison among the groups
indicates that the two patient groups are significantly different from the control group, $p < .05$. The $t$-test comparing the long-treatment group's pre- and posttreatment depression scores is significant, $t(35) = 2.67$, $p = .011$ (see Table 2, Appendix 2). The $t$-tests for the other two groups are not significant. The analysis of variance on the posttreatment Zung Depression Scale scores of the three groups is significant, $F(2,84) = 15.12$, $p < .0001$ (see Table 3, Appendix G). The post hoc comparison reveals that the two patient groups remain significantly different from the control, $p < .05$. The analysis of covariance for the three group's depression scores is not significant.

State Anxiety. The analysis of variance on the pretreatment state-anxiety scores from the State-Trait Anxiety Inventory is significant, $F(2,84) = 8.76$, $p = .0004$ (see Table 4, Appendix H). The comparison among the groups indicates that the two patient groups are significantly different from the control group, $p < .05$. The $t$-test comparing the long-treatment group's pre- and posttreatment state-anxiety scores is significant, $t(35) = 2.33$, $p = .026$. The $t$-tests for the other two groups are not significant. The analysis of variance on the posttreatment state-anxiety scores of the three groups is significant, $F(2,84) = 6.42$, $p = .0025$ (see Table 5, Appendix I). The post hoc comparison reveals that the two patient groups
remain significantly different from the control group. The analysis of covariance on the three groups' state-anxiety scores is not significant.

**Finger Oscillation.** The analysis of variance on the pretreatment dominant-hand finger-tapping scores from the Finger Oscillation Test is significant, $F (2, 84) = 6.44$, $p = .0025$ (see Table 6, Appendix J). The comparison among the three groups' pretreatment scores indicates that the short-treatment group is significantly different from the control group. The $t$-test comparing the short-treatment group's pre- and posttreatment dominant-hand finger-tapping scores is significant, $t (34) = 3.15$, $p = .003$. The $t$-tests for the two other groups are not significant. The analysis of variance on the posttreatment dominant-hand finger-tapping scores is significant, $F (2, 84) = 6.26$, $p = .0029$ (see Table 7, Appendix K). The post hoc comparison among the three groups' posttreatment scores reveals that the two patient groups are significantly different from the control group, $p < .05$. The analysis of covariance on the posttreatment finger-tapping scores with the pretreatment finger-tapping scores as a covariate is significant, $F (2, 83) = 3.33$, $p = .041$ (see Table 8, Appendix L). A post hoc comparison reveals that the long-treatment group is significantly different from the control group, $p < .05$. 
Embree/Butler Bender Score. The analysis of variance on the pretreatment Embree/Butler Bender scores is not significant. The mean pretreatment Bender scores are 13.9 for the control group, 14.9 for the short-treatment group, and 16.0 for the long-treatment group. The t-test comparing the pre- and posttreatment Bender scores of the short-treatment group is significant, \( t (27) = 3.11, p = .004 \). Also, the t-test comparing the pre- and posttreatment Bender scores of long-treatment group is significant, \( t (25) = 2.73, p = .011 \). The t-test is not significant for the control group. The mean posttreatment Bender scores are 14.5 for the control group, 12.4 for the short-treatment group, and 13.6 for the long-treatment group. Neither the analysis of variance nor the analysis of covariance on the posttreatment Bender scores are significant.

Digit Symbol. The analysis of variance on the pretreatment Digit Symbol raw scores is significant, \( F(2,67) = 8.17, p = .0007 \) (see Table 9, Appendix M). The post hoc comparison reveals that the two patient groups are significantly different from the control group, \( p < .05 \). The t-test comparing the pre- and posttreatment Digit Symbol scores of the control group is significant, \( t (15) = 3.43, p = .004 \). The t-tests for the two patient groups are not significant. The analysis of variance on the posttreatment Digit Symbol raw scores is significant.
$F_{(2,67)} = 6.86$, $p = .0019$ (see Table 10, Appendix N). The post hoc comparison reveals that the two patient groups are significantly different from the control group, $p < .05$. The analysis of covariance on the posttreatment Digit Symbol raw scores is not significant.

**MMPI.** On the MMPI, the number of $T$-scores equal to or greater than 70 is the pathology score for each person. The analysis of variance on the pretreatment MMPI pathology scores is significant, $F_{(2,45)} = 8.52$, $p = .0007$ (see Table 11, Appendix O). The post hoc comparison reveals that the two patient groups are significantly different from the control group, $p < .05$. None of the $t$-tests comparing pre- and posttreatment MMPI pathology scores are significant. The analysis of variance on the posttreatment MMPI pathology scores is significant, $F_{(2,45)} = 8.52$, $p = .0007$ (see Table 12, Appendix P). The post hoc comparison reveals that the two patient groups are significantly different from the control group, $p < .05$. The analysis of covariance on the posttreatment pathology scores is not significant.

**WAIS-R Full Scale IQ.** The analysis of variance on the pretreatment WAIS-R full scale IQ's is not significant. The only $t$-test that is significant is the one comparing the pre- and posttreatment IQ's of the long-treatment group, $t_{(26)} = 3.39$, $p = .002$. Neither the analysis of variance
nor the analysis of covariance on the posttreatment IQ's are significant.

**Trail Making Test Part B.** The analysis of variance on the pretreatment Trail Making Test Part B scores is not significant. The $t$-tests comparing the pre- and posttreatment Trail Making B scores are all significant. Those results are $t_{(34)} = 2.95, p = .006$ for the short-treatment group; $t_{(35)} = 3.47, p = .001$ for the long-treatment group; and $t_{(15)} = 3.75, p = .002$ for the control group. Neither the analysis of variance nor the analysis of covariance for the posttreatment Trail Making B scores are significant.

**Discussion**

In general, the results of the present study support the contention that objectively verifiable psychological and behavioral changes occur in a group of identifiably susceptible patients when those patients fast in an environment relatively free of contaminants. In support of the first hypothesis, the data indicate that fasting environmentally sensitive patients improve in their intellectual/cognitive and neurological/cerebral functioning. As hypothesized, the data indicate that fasting environmentally sensitive patients measurably decrease their level of psychopathology (depression and anxiety). Further, there is some indication for the support of the
third hypothesis that when these patients are exposed to
cicitants to which they are sensitive (after being
relatively incitant free for a period of time) their
functioning deteriorates.

In the present study, three variables assess psycho-
pathology. The state-anxiety scale of the State-Trait
Anxiety Inventory determines the degree of anxiety present,
and the Zung Depression Scale evaluates depression. The
number of MMPI T-scores equal to or greater than 70 (the
pathology score) determines the overall level of pathology
for each person.

Interestingly, the Zung depression score and the STAI
state-anxiety score behave statistically in a similar
manner. At initial testing, both treatment groups score
higher than the control group on both the Zung Depression
Scale and the state-anxiety scale of the State-Trait Anxiety
Inventory. Although at retest, both patient groups continue
to score higher than the control group on both measures,
the long-treatment group's levels of depression and
anxiety are significantly lower at retest than at admission.
The short-treatment group's levels of depression and anxiety
are not significantly lower at retest.

Thus, in comparison to a nonpatient/no-treatment
control group, the patients appear to be more emotionally
distressed upon admission. This is not surprising since
many of the patients come to the ECU only after failures in extensive efforts to diagnose and treat a variety of their disorders. These chronic health problems often result in interpersonal conflicts, vocational problems, and financial difficulties. Such circumstances alone may produce depression and anxiety. In addition, ECU patients may have a physiological component contributing to their emotional distress.

Based on the significant improvement the long-treatment group demonstrates on the depression and anxiety measures, it appears that the ECU treatment helps alleviate some of the patient's emotional distress. However, despite the patients' posttreatment decrease in emotional distress, when the three groups are retested, both patient groups are still significantly more emotionally distressed than the control group. Since most patients spend from 3 to 6 weeks in the ECU, it seems reasonable to speculate that further alleviation takes place as the patients' treatment progresses and they gain insight into the role that physiological factors have in what they experience.

The short-treatment group does not show this significant depression and anxiety reduction at retest. This difference between the two groups may be attributable to the fact that many of the long-treatment group patients are experiencing reactions to incitants for which they are being tested. These reactions, although frequently
uncomfortable, may act as empirical confirmation for the patients that what they have been told by the ECU staff about their condition is true and that their condition is likely to improve. It seems highly probable that this reduces the patient's depression and anxiety. On the other hand, the short-treatment group has not yet experienced this empirical confirmation and they remain unconvinced that they are being helped. Being thus unconvinced, their level of emotional distress remains relatively unchanged.

An alternative explanation for the differing levels of depression and anxiety between the short- and long-treatment groups is that the emotional distress has a physiological basis which is altered during the ECU treatment. If this is so, then the difference in emotional distress may be due to different levels of physiological change associated with different lengths of treatment.

A third and more probable explanation is that both psychological and physiological stressors contribute to the patients' emotional distress. What is not discernable from the results of the present study is the extent to which each type of stress contributes to each patient's condition. Neither is it possible to determine what portion of the emotional changes are attributable to the various elements of the patients' ECU experience. For example, it may be physiological stress reduction from the relatively low total body load of incitants found in the ECU which lowers
the emotional distress. It may be psychological stress reduction from the supportive attitude of other patients or from newly acquired knowledge about ecological disorders. Or it may be any combination of reductions in these stressors which results in the detected emotional changes.

Closely related to the degree of emotional distress reported by the ECU patients is the degree of general psychopathology exhibited in the MMPI pathology score. As expected, the patients initially have significantly higher MMPI pathology scores than the control group. There is no significant MMPI pathology reduction in either patient group at retest. Thus, after 1 week of treatment, the patients continue to have a higher level of pathology than the no-treatment control group. It is not determinable from the present study whether or not this level of pathology is maintained until discharge.

There were three instruments utilized to assess neurological/cerebral functioning. The Finger Oscillation Test assessed neurological functioning as manifested in a motor skill (the finger-tapping score). The Embree/Butler Bender score determined the degree of organic brain dysfunction as manifested in a visual-motor coordination task. The WAIS-R Digit Symbol subtest raw score indicated general cerebral functioning (Walsh, 1978).

One of the more meaningful findings occurred in the results of the Finger Oscillation Test. At admission, only
the short-treatment group's finger-tapping scores are significantly lower than the control group's scores. Also, this is the only group to improve significantly at retest. However, at retest, both patient groups perform poorer than the control group, and the change in the long-treatment group is significantly less than the change in the control group. One possible explanation for the apparent worsening of the long-treatment group's finger-tapping performance can be found in the two group's different stages of treatment. First, the short-treatment group was retested on the fourth or fifth day of treatment, and the long-treatment group was retested on the sixth, seventh, or eighth day of treatment. Therefore, more of the long-treatment group had been exposed to potential incitants, and more of the short-treatment group had not entered that phase of their treatment. Thus, it may be that the long-treatment group's poorer performance on the Finger Oscillation Test is due to acute incitant sensitivity reactions at the time of their retest. This interpretation is consistent with the fact that, on five measures (the Finger Oscillation Test, Zung, STAI, MMPI, and Digit Symbol), the long-treatment group continues to function poorer than the control group, even though the long-treatment group shows significant improvement over their admission scores on the Zung and STAI, and three additional measures (Trail Making Test, WAIS-R IQ, and Bender).
This result also suggests that the Finger Oscillation Test is a more sensitive measure of the acute reaction than the Trail Making Test or the Bender. Although the Trail Making Test scores may be affected by an acute reaction, in the present study, the improved retest Trail B scores appear to reflect familiarity with the task (since the control group improves along with the patient groups). The Bender scores may not be as sensitive to an acute reaction as those of the Finger Oscillation Test since this is more purely a measure of motor functioning—specifically, a measure of the ability to sustain a rapid repetitive finger-tapping motion. On the other hand, the Bender task requires perceptual ability coordinated with a seemingly different kind of fine motor control which may not be as easily affected by an acute reaction.

On the Bender, neither patient group differs significantly at admission or retest from the control group, but the initial mean Bender scores for both patient groups are in the pathological range for the Embree/Butler scoring system (i.e., greater than 14). However, both patient groups improve their Bender scores significantly during treatment so that at retest, their mean Bender scores are in the normal range. Thus, it appears that visual-motor coordination improves significantly during ECU treatment, and organic brain dysfunction as measured by the Embree/Butler Bender scoring system is reduced.
Also on the Bender, the initial mean control group scores are not in the pathological range; however, the mean retest scores are slightly above in pathology cutoff. Although the control group changed from normal to pathological in the Embree/Butler scoring system, the degree of change was not significant. In comparison to the control group's initial scores, the patient's initial scores are higher, although not significantly so. Although the change in the patients' scores is not significantly greater than the change in the control group's scores, both patient group's retest scores are not only in the normal range but also are significantly lower than at admission.

One possible explanation for why the control group's Embree/Butler Bender scores have risen nonsignificantly into the pathological range at retest is that the control group, compared to the patients, may have been minimally motivated to do well on this test. Thus, they may have given minimal effort to the task and elevated their scores through carelessness. This would account for their initial scores being nearly into the pathological range and also for the slightly higher retest scores. Another, although seemingly less likely explanation, is that the control group may have by chance performed in the pathological range.

The WAIS-R Digit Symbol subtest can be interpreted in two meaningful ways. It is a general measure of cerebral
functioning (Walsh, 1978), and it is a measure of the ability to learn a new task (Matarazzo, 1972). In both respects, the patients initially perform poorer than the controls. The control group's superior ability to learn a new task is particularly evident in the control group's ability to significantly improve their retest scores, while the patient's retest scores do not improve.

The Wechsler Adult Intelligence Scale-Revised and the Trail Making Test Part B assessed intellectual/cognitive functioning. The WAIS-R full-scale IQ indicated general intellectual functioning. The Trails B reflected cognitive functioning through the testee's ability to plan, regulate, and check an activity program.

Although there are no significant WAIS-R IQ differences among the groups at admission or at retest, after approximately 1 week of treatment, the long-treatment group obtains a significantly higher IQ score than at admission. This suggests that general intellectual functioning is improved during treatment at the ECU and that the improvement is a function of time in treatment. The longer the stay in the ECU, the more IQ improvement the patients exhibit.

The Trail Making Test Part B does not appear to discriminate between the patients and the controls. There are no significant differences in performance on
this measure at any point. Both the patients and the controls perform this task significantly faster during the second administration. It appears that some improvement occurs simply due to familiarity with the task. If there is any change in the abilities assessed by this instrument, it is confounded with this apparent familiarity effect.

Several aspects of the present study were not under the control of the researcher and may have confounded the results. First, selection of patients at the ECU was dependent upon their willingness to participate. Some did not participate because they were too ill. Other patients were simply not interested. There were other patients who participated in the initial assessment but were unwilling to retest. Also, some patients completed only part of the second assessment. The most common reason for not retesting was that the patient was reacting to an incitant and was too ill. The main effect of this selection factor seems to have been to eliminate those patients with the most severe reactions.

Another aspect of the study which was not researcher controlled was the variability in the length of each patient's fast. Closely related to the length of fast was the time at which incitant testing began. Both of these factors were controlled by the treating physician according to the needs of the patient and both were quite variable. This problem was dealt with by dividing the two
patient groups according to the most frequent time for the patients to end their fasts and begin eating single food meals. Thus, it is assumed that (to the extent possible) the short-treatment group is clear of symptoms, has a minimal total body load of incitants, and is not reacting acutely to an incitant. Alternatively, the long-treatment group is more likely to be reacting acutely to an incitant to which they have been exposed and to be experiencing the relevant symptoms of their reactions.

An important question that is not answered by the results of this study is how much of the improvement shown on the various behavioral tasks is attributable to the emotional improvement and how much is attributable to physiological changes. It is well-established that depression lessens effort expenditure, and that anxiety affects performance. Thus, it seems plausible that performance on the tasks in the present study is to some extent affected also. The magnitude of this effect remains to be assessed at a future time.

The results of this study have implications relevant to the practice of clinical ecology. In general the results of this study argue for an holistic approach to the treatment of ecology patients. It appears that for the chronically ill environmentally sensitive patients seen at the ECU both physiological and psychological dysfunctions occur simultaneously.
Attention should be given to both aspects of a patient's health. Along with determining which environmental incitants are contributing to the patients' condition, it appears to be relevant to determine what psychological stressors are present which might be contributing to the kinds of general psychopathology and emotional distress detected in this study. Then, treatment should be directed to the psychological as well as the physiological problems. A wide range of reactions can be expected in the environmentally sensitive patient. It should be kept in mind that the more severely debilitated patients were frequently excluded from this study. Several patients were admitted to the ECU in such a weakened condition that their physicians decided not to allow them to fast. On numerous occasions posttreatment tests were not administered due to the severity of incitant reactions following a challenge test. A few patients had such severe reactions to the test materials that they could not tolerate the test procedure. The clinician should be prepared to cope with such extreme reactions.

In summary, when patients are admitted to the ECU, they appear to have more anxiety, more depression, more general psychopathology, poorer visual-motor coordination, poorer motor functioning, and diminished learning capacity in comparison to the control group. After 1 week of treatment, the patients are less depressed and less anxious than when
they are admitted. Also, their intellectual functioning and visual-motor coordination are improved. Although motor functioning improves initially, it appears to deteriorate during acute reactions.
Appendix A

Embree/Butler's Bender Scoring Sheet

Name

Dates tested:

<table>
<thead>
<tr>
<th>Error Scores</th>
<th>1st</th>
<th>2nd</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>5 points</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial rotation (A, 4, 5, 6, 7, 8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Omission of angles (A, 4, 7, 8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>4 points</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Added angles (A, 4, 7, 8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overlap difficulty #7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distortion (all)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tremor (all)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>3 points</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Embellishments (A, 4, 6, 7, 8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lack of closure (A, 4, 7, 8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angles flattening #3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total
Appendix B

Sample Self-Report Checklist for Food Symptoms

Indicate if symptoms are present or if smell of food produces symptoms.

Name ___________________________ Date __________

Food _______________________ Begin _____ Finish _____

Feeling before eating ______________________________

REST 15 MINUTES BEFORE TAKING PULSE.

Resting pulse 5 minutes before eating __________________

Symptoms noted while eating ______________________________

5 minutes after ______________________________

10 minutes after ______________________________

20 minutes after ______________________________

40 minutes after ______________________________

Please note any symptoms beyond 40 minutes pulse. Record time from finish of meal to start or reaction. __________________
### Appendix C

**Sample Self-Report Checklist for Chemical Booth Testing**

<table>
<thead>
<tr>
<th>Name</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical</td>
<td>Start</td>
</tr>
<tr>
<td>Feeling before test</td>
<td></td>
</tr>
</tbody>
</table>

- In room pulse __________ prior to testing
- In booth pulse __________

#### 5 minute pulse

**Symptoms noted after 5 minute exposure** __________

#### 10 minute pulse

**Symptoms noted after 10 minute exposure** __________

#### 15 minute pulse

**Symptoms noted after 15 minute exposure** __________

Be sure to record any symptoms you have after leaving testing booth and time each symptom as it occurs.
Appendix D

Sample Clinical Observation Report Form

<table>
<thead>
<tr>
<th>Time &amp; Date</th>
<th>Chemical</th>
<th>Reactions</th>
<th>12 hours later</th>
<th>24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Booth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Room 20</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Booth 25</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Natural Gas</td>
<td>Room 30</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Booth 45</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>&quot;A&quot;</td>
<td>Room</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Booth 5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix E

Table 1
Analysis of Variance: Pretreatment Zung Depression Scale Raw Scores

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>4685.08</td>
<td>2</td>
<td>2342.54</td>
<td>20.732*</td>
</tr>
<tr>
<td>Within groups</td>
<td>9491.36</td>
<td>84</td>
<td>112.99</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>14176.43</td>
<td>86</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P < .0001
Table 2

Comparison of t-Test for Correlated Means: Comparing Mean Pre- to Posttreatment Scores of the Groups for Eight Variables

<table>
<thead>
<tr>
<th>Scores</th>
<th>Short-treatment</th>
<th>Long-treatment</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zung Depression Scale</td>
<td>t (34) = 1.66</td>
<td>t (35) = 2.67*</td>
<td>t (15) = .94</td>
</tr>
<tr>
<td>State-Anxiety Scale</td>
<td>t (34) = 1.24</td>
<td>t (35) = 2.33*</td>
<td>t (15) = 1.26</td>
</tr>
<tr>
<td>Dominant-Hand Finger-Tapping</td>
<td>t (34) = 3.15**</td>
<td>t (35) = .19</td>
<td>t (15) = 1.52</td>
</tr>
<tr>
<td>Embry-Butler Bender</td>
<td>t (27) = 3.11**</td>
<td>t (25) = 2.73*</td>
<td>t (15) = .45</td>
</tr>
<tr>
<td>WAIS-R Digit Symbol Subtest</td>
<td>t (27) = 1.06</td>
<td>t (25) = 1.74</td>
<td>t (15) = 3.43**</td>
</tr>
<tr>
<td>MMPI Pathology</td>
<td>t (13) = .81</td>
<td>t (17) = 1.83</td>
<td>t (15) = 1.58</td>
</tr>
<tr>
<td>WAIS-R Full Scale IQ</td>
<td>t (27) = .54</td>
<td>t (25) = 3.39**</td>
<td>t (15) = .47</td>
</tr>
<tr>
<td>Trail Making Test Part B</td>
<td>t (34) = 2.95**</td>
<td>t (35) = 3.47**</td>
<td>t (15) = 3.75**</td>
</tr>
</tbody>
</table>

*P < .05  
**P < .01  
***P < .001
### Table 3

**Analysis of Variance: Posttreatment Zung Depression Scale Raw Scores**

<table>
<thead>
<tr>
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<th>df</th>
<th>MS</th>
<th>F</th>
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</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>1928.03</td>
<td>2</td>
<td>964.02</td>
<td>6.421*</td>
</tr>
<tr>
<td>Within groups</td>
<td>12612.21</td>
<td>84</td>
<td>150.15</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>14540.24</td>
<td>86</td>
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</tr>
</tbody>
</table>

*P < .0001
Appendix H

Table 4
Analysis of Variance: Pretreatment STAI State-Anxiety Scores

<table>
<thead>
<tr>
<th>Source</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>2796.47</td>
<td>2</td>
<td>1398.24</td>
<td>8.756*</td>
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<td>Within groups</td>
<td>13414.53</td>
<td>84</td>
<td>159.70</td>
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<td>Total</td>
<td>16211.00</td>
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</table>

*p = .0004
Appendix I

Table 5
Analysis of Variance: Posttreatment STAI State-Anxiety Scores

<table>
<thead>
<tr>
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<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>1928.03</td>
<td>2</td>
<td>964.02</td>
<td>6.421*</td>
</tr>
<tr>
<td>Within groups</td>
<td>12612.21</td>
<td>84</td>
<td>150.15</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>14540.24</td>
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*P = .0025
## Appendix J

**Table 6**

*Analysis of Variance: Pretreatment Dominant Hand Finger Oscillation Scores*

<table>
<thead>
<tr>
<th>Source</th>
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</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>968.06</td>
<td>2</td>
<td>484.03</td>
<td>6.436*</td>
</tr>
<tr>
<td>Within groups</td>
<td>6317.53</td>
<td>84</td>
<td>75.21</td>
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<td>Total</td>
<td>7285.59</td>
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*R = .0025

*P = .0025
## Analysis of Variance: Posttreatment Dominant Hand Finger Oscillation Scores

<table>
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<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>666.28</td>
<td>2</td>
<td>333.14</td>
<td>6.258*</td>
</tr>
<tr>
<td>Within groups</td>
<td>4471.70</td>
<td>84</td>
<td>53.23</td>
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<td>Total</td>
<td>5137.98</td>
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*p = .0029
Appendix II

Table 8
Analysis of Covariance: Posttreatment Dominant-Hand Finger-Tapping Scores with the Pretreatment Dominant-Hand Finger-Tapping Scores as Covariate

<table>
<thead>
<tr>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>158.83</td>
<td>2</td>
<td>79.42</td>
<td>3.333*</td>
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<tr>
<td>Within groups</td>
<td>1977.51</td>
<td>83</td>
<td>23.83</td>
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</tr>
<tr>
<td>Total</td>
<td>2136.34</td>
<td>85</td>
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<td></td>
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<table>
<thead>
<tr>
<th>Group</th>
<th>Observed Means</th>
<th>Adjusted Means</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short-treatment</td>
<td>49.99</td>
<td>52.75</td>
</tr>
<tr>
<td>Long-treatment</td>
<td>50.34</td>
<td>50.67</td>
</tr>
<tr>
<td>Control</td>
<td>57.30</td>
<td>54.22</td>
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</table>

*p < .05
Appendix M

Table 9

Analysis of Variance: Pretreatment WAIS-R Digit Symbol Subtest Raw Scores

<table>
<thead>
<tr>
<th>Source</th>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>2370.82</td>
<td>2</td>
<td>1185.41</td>
<td>8.168*</td>
</tr>
<tr>
<td>Within groups</td>
<td>9723.06</td>
<td>67</td>
<td>145.12</td>
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<td>Total</td>
<td>12093.87</td>
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*P = .0007
Appendix M

Table 10

Analysis of Variance: Posttreatment WAIS-R Digit Symbol Subtest Raw Scores

<table>
<thead>
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<th>Source</th>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>3035.32</td>
<td>2</td>
<td>1517.66</td>
<td>6.860*</td>
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<tr>
<td>Within groups</td>
<td>14823.56</td>
<td>67</td>
<td>221.25</td>
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<td>Total</td>
<td>17858.88</td>
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*p = .0019
Appendix Q

Table 11

Analysis of Variance: Pretreatment MMPI Pathology Scores

<table>
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</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>72.61</td>
<td>2</td>
<td>36.31</td>
<td>8.515*</td>
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<td>Within groups</td>
<td>191.87</td>
<td>45</td>
<td>4.26</td>
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<td>Total</td>
<td>264.48</td>
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*p = .0007
Appendix P

Table 12
Analysis of Variance: Posttreatment Pathology Scores

<table>
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<th>Source</th>
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<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>72.82</td>
<td>2</td>
<td>36.41</td>
<td>8.515*</td>
</tr>
<tr>
<td>Within groups</td>
<td>192.43</td>
<td>45</td>
<td>4.28</td>
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<td>Total</td>
<td>265.25</td>
<td>47</td>
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*p = .0007
Table 13

Pre- and Posttreatment and Adjusted Mean Scores of the Groups for Eight Variables

<table>
<thead>
<tr>
<th>Groups</th>
<th>Zung Depression Scale</th>
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<th>State-Anxiety Scale</th>
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<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Adjusted</td>
<td>Pre</td>
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<tr>
<td>Short-treatment</td>
<td>N = 35</td>
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<td></td>
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<tr>
<td></td>
<td>47.60</td>
<td>45.20</td>
<td>42.73</td>
<td>46.43</td>
</tr>
<tr>
<td>Long-treatment</td>
<td>N = 36</td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>52.47</td>
<td>48.31</td>
<td>42.46</td>
<td>50.33</td>
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<tr>
<td>Control</td>
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<td></td>
<td>32.00</td>
<td>31.06</td>
<td>39.38</td>
<td>34.50</td>
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References


Clarke, T. W. Allergy of the central nervous system. *Annals of Allergy*, 1944, 2, 189-196, 279.


Douglas, H. M. G. Reactions to aspirin and food additives in patients with chronic urticaria, including the physical urticarias. British Journal of Dermatology, 1975, 92, 135-144.


Feingold, B. F. Adverse reactions to food additives with special reference to hyperkinesis and learning difficulty (H-LD). In F. Steele & A. Bourne (Eds.), The man–food equation. London: Academic Press, 1975. (a)

Feingold, B. F. Hyperkinesis and learning disabilities linked to artificial food flavors and colors. American Journal of Nursing, 1975, 75, 797-803. (b)


Golbert, T. A. A review of controversial diagnostic and therapeutic techniques employed in allergy. Journal of Allergy and Clinical Immunology, 1975, 56, 170-190.


Juhlin, L., Michaelson, G., & Setterstrom, O. Urticaria and asthma induced by food and drug additives in patients with aspirin hypersensitivity. *Journal of Allergy and Clinical Immunology*, 1972, 50, 92-98.


Matarazzo, J. D. Wechsler's measurement and appraisal of adult intelligence (5th ed.). Baltimore, Md.: Williams & Wilkins, 1972.


Rea, W. J. Diagnosing food and chemical susceptibility. *Journal of Continuing Education, in ORL and Allergy*, 1979, 47-59. (a)


Rowe, A. H. *Food allergy, its manifestations, diagnosis, and treatment, with a general discussion of bronchial asthma*. Philadelphia: Lea & Febiger, 1931.


Taylor, E. Food additives, allergy, and hyperkinesis.  

Taylor, J. A. A personality scale of manifest anxiety.  

Walsh, K. W. Neuropsychology: A clinical approach.  


Williams, J. I., Cram, D. M., Tausig, F. T., & Webster, E.  