PERSONALITY FACTORS IN OBESITY AND RESPONSE TO NAVY RESIDENTIAL TREATMENT PROGRAMS

By

DAVID SCOTT ADKINS

Bachelor of Science
Oklahoma State University
Stillwater, Oklahoma
1978

Master of Science
Oklahoma State University
Stillwater, Oklahoma
1981

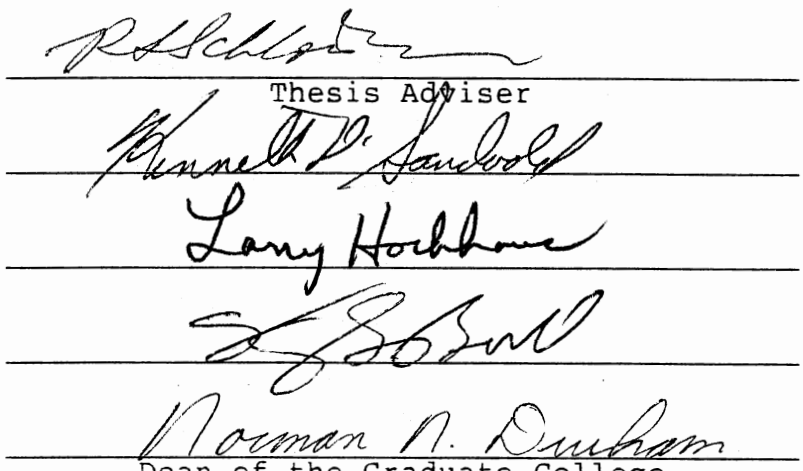
Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
DOCTOR OF PHILOSOPHY
July, 1988

PERSONALITY FACTORS IN OBESITY AND

RESPONSE TO NAVY RESIDENTIAL

TREATMENT PROGRAMS

Thesis Approved:



Thesis Adviser

Dean of the Graduate College

ACKNOWLEDGEMENTS

Many thanks are due to my staff and colleagues at the U.S. Naval Hospital, Yokosuka for their tolerance of my distractability and unavailability during the final phase of this project. I am very grateful to Dr. Bob Schlottman for his large time contributions and flexibility in guiding the completion of the study, on a highly compressed schedule. A great debt is owed to my extended family for vast amounts of encouragement and logistical support that enabled me to meet deadlines. Major credit for this and every other success that I have enjoyed in the past 12 years belongs to my wife, and best friend, Marcie. Without her unique combination of nurturance, toughness, and organizational ability, this accomplishment would never have been realized.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

A recent government report estimated that 19% of adult males and 28% of adult females in the United States are obese (National Center for Health Statistics (NCHS), 1983). Since obesity is a significant health problem these statistics should be alarming to health care professionals. In a culture obsessed with physical fitness and thinness, obesity would be a serious problem for its victims, even if there were no associated health risks. Obese persons face social and psychological hazards beginning at an early age and suffer not only from the stigma of obesity, but also from being blamed for their condition (Brownell, 1982).

Despite the various dangers associated with obesity, it continues to rank among the most intractable of medical problems. A superficial evaluation of the problem might indicate that the solution should be simple. To lose weight, one needs only to unbalance the energy equation in a negative direction. All that is required is to absorb fewer calories each day than are expended in physical activity and metabolic functions. This may be done by reducing caloric intake, increasing energy expenditure, or both. When an

acceptable body weight is achieved, simply rebalance the equation and maintain that balance.

There are literally thousands of weight reduction programs available in this country. These range from the "miracle cures," whose safety and legality are often questionable, to the experimental programs offered by prestigious teaching hospitals. The common element among all of these programs is their long-term success ineffectiveness in controlling obesity. The short-term success rates for various reducing programs are highly variable and the attrition rates are often very high. Those who do manage to lose weight, initially, will most likely regain it within two to three years, if not sooner (Johnson & Drenick, 1977; Stunkard & Penick, 1979). Past epidemiological studies have implied that the probability of indefinite remission was higher for most forms of cancer than it was for obesity. While there have been some advances in recent years, the long-term prognostic picture is still grim for most obese people (Brownell, 1982).

The understanding of the physiology of adipose tissue and its relationship to chronic obesity has increased greatly in the past 15 years. Raw number of adipocytes (fat cells) vary tremendously between individuals and the differences in these numbers are the primary determinants of variations in weight among persons of similar heights and skeletal structures. Sjostrom (1980) has found numbers of adipocytes in adult individuals ranging from 20 billion to

160 billion. That is, some adults have eight times as many fat cells as other adults. The same study estimated adipose tissue mass in these subjects to range from one kg to 200 kg. He concluded that, "the contribution of fat cells to the determination of body weight is fundamental" (p. 151).

Bjorntorp and Sjostrom (1971) hypothesized that there are two types of obesity in humans. A hypertrophic form of obesity is explained by increased adipocyte size, while a hyperplastic form results primarily from increases in numbers of adipocytes. This hypothesis was later confirmed by Salans, Cushman, and Weissman (1973). Further investigations have shown that hypertrophic and hyperplastic obesity differ in relation to age of onset and prognosis for long-term weight loss. Exaggerated numbers of fat cells (hyperplastic obesity) appear to be associated with early onset of obesity (Sjostrom, 1980). While development of hypercellularity has been known to occur in subjects over the age of 20, this happens primarily in cases of extreme (morbid) obesity and obesity developed during pregnancy (Hirsch & Batchelor, 1976). Persons who become moderately obese later in life tend to suffer from the hypertrophic form. The late-onset obese group has a much higher probability of maintaining long-term weight loss than the early-onset group, whose condition appears to be quite intractable (Krotkiewski, Sjostrom, & Bjorntorp, 1977).

In light of these findings, it would be logical, and perhaps clinically useful, to view the early- and late-onset

groups as distinct subpopulations within the population of moderately obese humans. However, most research and all treatment programs reviewed tend to treat obese persons as if they were members of a physiologically and psychologically homogeneous population. Past research has successfully established the existence of physiological subgroups, but there were no studies found that attempted to isolate psychological subgroups. Since most treatment programs have behavior modification or other psychological components, it seems that an understanding of the psychology of obesity would be a critical element in assessment and treatment.

Attempts to define a set of psychological descriptors for the obese population have met with limited success. Various studies have reported finding no differences between obese subjects and non-obese groups (Johnson, Stern, & Gruen, 1976; Pomerantz, Greenberg, & Blackburn, 1977). Other studies have identified differences, but are criticized for methodological problems (Bruch, 1980; Coates & Thorensen, 1980; Collipp, 1980; Klesges, 1984; Stunkard & Mendelson, 1967). The literature review revealed that the majority of the relatively few controlled studies of personality factors in obesity concentrate upon females and/or the morbidly obese (Hutzler, Keen, Molinari, & Carey, 1981; Kolotkin, Revis, Kirkley, & Janick, 1987; Ruderman, 1985; Scott & Barrofio, 1986). No studies were found that addressed the psychology of the moderately obese male.

Further research designed to clarify the characteristics of subpopulations within the population of obese persons could significantly impact the clinical management of the disorder. Behaviorally-oriented systems tend to deal exclusively with altering the energy intake and expenditure balance, with little attention being given to the emotional factors in eating behavior. The insight-oriented and social support systems generally attempt to deal with the social and emotional precursors of eating behavior. However, they do not address the physiological drive states that precipitate hunger in hypercellular (early-onset) obese persons.

The current study has two major purposes. The primary purpose is to demonstrate the existence of psychological differences between the early- and late-onset groups of obese persons. The secondary purpose is to test the feasibility of a weight control program that is being implemented in the military. The U.S. Navy is currently developing a system of obesity treatment programs that uses the Overeaters Anonymous philosophy as the core of the treatment. This philosophy views all obese persons as compulsive overeaters whose use of food is an addiction that is psychologically similar to alcoholism. The treatment programs are practically identical to the Navy programs for treatment of alcoholism and are being conducted in the same facilities. If the study succeeds in its purposes, it could have a significant impact on the Navy's approach to treatment of obese service members.

CHAPTER II

LITERATURE REVIEW

The Epidemiology of Obesity

The fact that obesity is a serious health problem is well established in the medical and psychological literature. Various studies have linked obesity to a host of medical problems including hypertension, diabetes mellitus, surgical and anesthesia risks, and renal problems. This does not mean that being a few pounds above the "ideal" weight necessarily reduces a person's life expectancy. However, in an extensive study of variations in mortality across weight index categories, Lew and Garfinkel (1979) found the lowest mortality rates among persons who were close to average weight or 10 to 20% below average weight. Men and women in the 30 to 40% above average weight group had a mortality rate nearly 50% higher than the average weight group. Among those more than 40% heavier than average, the mortality rate was 90% higher. This study and others clearly show that gross obesity is dangerous, but the amount of health risk incurred by persons less than 30% overweight is not clear (Brownell, 1982).

The emotional costs of obesity are more difficult to quantify than the medical ones and the research literature shows mixed results in efforts to establish their existence. Some studies indicate that obese persons tend to be more depressed, have lower self-esteem, are more self-conscious, and are less assertive than the general population (Collipp, 1980; Pomerantz et al., 1977). Whether these problems are causes or effects of obesity has yet to be determined. Stunkard and Mendelson (1967) found that, because obese persons are viewed negatively by much of the rest of the world, many of them detest their bodies and are preoccupied with their weight. There is also evidence that the social stigma of obesity translates into more tangible problems. Legal proceedings have established the fact of discrimination against obese persons in selection for employment and promotion (Brownell, 1982).

Despite the various dangers associated with obesity, it continues to rank among the most prevalent and intractable of medical problems. A recent government report estimated that 19% of adult males and 28% of adult females in the United States are obese (National Center for Health Statistics (NCHS), 1983).

To date, the best predictor of obesity is socioeconomic status (Overfield, 1980). In the United States, individuals in the lower classes are more likely to be obese than those in the upper classes. The reasons for this difference are quite complex and involve variations in prenatal care, early

nutrition, social norms, and ethnic background, to name a few. Across the economic levels, gender and family history have predictive value for obesity. These facts suggest the possible existence of a genetic component in the etiology of the disorder, although the nature and magnitude of the component are still unclear. Most of the family patterns of obesity can be explained by socioeconomic factors (Garn, 1976). However, the "spot fat" phenomenon, or the tendency of individuals to have concentrations of adipose tissue at different locations on the body, appears to be genetically determined. Animal studies show a definite genetic influence in obesity, but conclusive proof is lacking in research with human subjects (Brook, Huntley, & Slack, 1975).

The Physiology of Obesity

The understanding of the physiology of adipose tissue and its relationship to chronic obesity has increased greatly in the past 15 years. Raw numbers of adipocytes (fat cells) vary tremendously between individuals and the differences in these numbers are the primary determinants of variations in weight among persons of similar heights and builds. Sjostrom (1980) has found numbers of adipocytes in adult individuals ranging from 20 billion to 160 billion. That is, some adults have eight times as many fat cells as other adults. The same study estimated adipose tissue mass in these subjects to range from one kg to 200 kg. He

concluded that, "the contribution of fat cells to the determination of body weight is fundamental" (p. 151).

While adipose tissue mass and body weight vary greatly between individuals, the stability of body weight within individuals surpasses that of most other physiologic variables (Keesey, 1980). During relatively short periods of observation, average variation in body weight was found to be less than 0.6% of the individual means. White American males increase only one to five pounds in average weight between the ages of 30 and 60 (NCHS, 1982). Observations such as these have led some investigators to theorize about the existence of a biologically dictated "set point" for body weight. This would mean that individuals who have accumulated large stores of body fat are biologically programmed to maintain their obesity (Nisbett, 1972). Animal studies demonstrate a tendency to regulate body weight around a stable level or set point. This occurs in both normal weight specimens and in animals who are congenitally obese. When these animals gain or lose weight as a result of laboratory manipulations, they tend to return to their original weights when the manipulations end. However, there are important differences in the nature of the adipose tissue accumulated by these groups. The normal animals have a normal number of adipocytes that increase in size as the animal gains weight. Obesity in the other group results primarily from exaggerated numbers of fat cells (Zucker & Zucker, 1961).

Early- and late-onset obese humans may be distinct subpopulations within the population of the moderately obese. Differences in the nature of the adipose tissue mass must be accompanied by differences in the endocrinological feedback systems that control the physiological components of hunger. Eating behavior occurs in response to the subjective experience of hunger, which results from a complex interaction of physiological and psychological inputs. It logically follows that, if the early- and late-onset groups are physiologically different, then they may also differ in the psychological components of the eating behavior that maintains their obesity. If two persons are equally obese, but they are physiologically different, then it would be reasonable to hypothesize that the psychological factors in the etiology of their obesity might be different (K. D. Brownell, personal communication, August 30, 1983).

Consider the hypothetical case of two adult males who are 40% above average weight. Subject A suffers from early-onset, hyperplastic obesity. Because adipocytes do not go away when he loses weight (they only decrease in volume), if Subject A reduced to an average weight, his adipose tissue mass would be significantly atrophied. This results in a homeostatic imbalance that the organism would be physiologically driven to correct. The above contention is supported by studies showing that obese humans who lose large amounts of weight experience physiological states that mimic starvation (Nisbett, 1972). Subject B, however, suffers

from the adult-onset hypertrophic form of obesity; meaning that he has a normal or near normal number of adipocytes that are significantly hypertrophied. For Subject B, being obese represents a state of homeostatic imbalance (at least for his adipose tissue mass). If he does not have a physiological imbalance that drives him to maintain hypertrophied adipocytes, it can be hypothesized that behavioral and emotional factors are primary in the etiology of his obesity.

The above example, while hypothetical, is quite plausible in light of the research cited earlier. The possible existence of fundamental differences between subjects in the etiology of obesity forms the basis for some hypotheses of the current study.

The Psychology of Obesity

The success of studies designed to define a set of personality factors that distinguish obese individuals from the normal weight population has been marginal, at best. Studies that yielded positive results (Bruch, 1980; Collipp, 1980; Stunkard & Mendelson, 1967) were criticized because the research was based on clinical impressions, inappropriate psychometric methods, or research designs that failed to include control groups (Coates & Thoresen, 1980). Other studies found no significant differences between obese and non-obese groups (Johnson et al., 1976; Pomerantz et al., 1976). Perhaps, the common flaw in all of these studies is

their tendency to view obese subjects as members of a physiologically and psychologically homogeneous population. In light of the research cited, it would be reasonable to hypothesize that, within the population of obese people, there are at least three identifiable subpopulations. The first, and most easily distinguished group, are those individuals who can be medically diagnosed as endogenously obese. These are persons who suffer from a detectable glandular or neurophysiological disorder that accounts for their inability to metabolize calories at a normal rate. The second group would include those persons with early-onset obesity, to whom the set point theory would apply. The third group consists of those whose conditions are attributable primarily to psychological and environmental factors. Previous studies have failed to separate the second and third subpopulations, resulting in a possible dilution of important data. The negative or ambiguous results can also, in part, be attributed to the use of dependent variables that fail to measure personality factors logically associated with stress related eating (Klesges, 1984).

A similar line of reasoning is put forth by Herman and Polivy (1975, 1980) in their study of restrained eating. They divided the obese population into three groups, based on etiology. Their "childhood/genetic" form of obesity is caused by an overendowment of adipocytes and is protected by the physiological set point phenomenon described by Nisbett

(1972). The "postpubescent/psychodynamic" form serves a psychological purpose (e.g. avoidance of sexuality) and is often accompanied by atypical eating behaviors such as binges and night eating. The "adult/sedentary" obesity occurs when, in adulthood, individuals become less active, but fail to compensate with lower caloric consumption. This study does not address the issue of obesity induced by glandular dysfunction, but it is assumed that this type of obesity was simply beyond the scope of the research.

While the above study discussed the issue of etiology, this was not linked to the major focus of the research, which was the "restrained eating" phenomenon. Their restraint scale is a measure of the degree to which a person exercises conscious control in the regulation of caloric consumption. The authors found that restrained eaters consumed considerably more food in an ad libitum eating situation if they were subjected to a high calorie preload (eating a high calorie snack at the beginning of the experiment). The unrestrained eaters decreased consumption in a linear fashion, relative to the calorie content of the preload. The interpretation of these results and the follow-up reliability study indicated that some subjects regulate weight naturally, while others must constantly exert effort to suppress weight. However, they did not measure the correlation between levels of restraint and weight classes.

A replication of the above study (Hibscher & Herman, 1977) addressed the issue of restraint versus obesity. As

was expected, most obese subjects were restrained eaters, as measured by the Revised Restraint Scale, which constitutes partial confirmation of set point theory. However, when considered independently, the weight classification variables were unrelated to the response to preloading. This indicates that obese subjects respond differently to hunger or disinhibition cues that precipitate eating. Still, this study made no attempt to measure the concommitance between restraint levels and obesity etiology/onset classifications.

Of particular interest in the current study are previous studies utilizing the Minnesota Multiphasic Personality Inventory (MMPI) in examining personality factors in obesity. Ayoob (1984) attempted to find specific scales of the MMPI that predicted successful weight loss in obese subjects. He concluded that the MMPI does not predict success in a behavioral weight loss program. However, this study did not examine differences between obese and non-obese subjects, nor did the treatment program address psychological characteristics peculiar to obese subjects. Willcockson (1986) utilized obese patients as a quasi-control group in a study of the MMPI's capacity for differentiating between brain-damaged and other psychiatric patients. As in the previous study, no comparisons were made between obese subjects and non-obese controls.

Scott and Barrofio (1986) compared the MMPI profiles of morbidly obese outpatients with those of anorexic and bulimic inpatients and non-obese controls. They found that

obese subjects differed from controls on nine of 13 MMPI scales. However, they did not find all of the expected differences between obese subjects and the anorexic or bulimic patients. This study concluded that obese subjects showed lower levels of identity confusion and reality distortion than the inpatient groups, but were higher than controls on measures of dependency, immaturity, anxiety, somatic concerns, passive-aggressiveness, and others. There is a limitation on the generalizability of these findings to the current study, because all subjects were female.

Obesity Treatment

The techniques for reducing caloric absorption range widely in terms of expense, complexity, and safety. Limiting intake by counting calories is safe, simple, and inexpensive. At the other end of the spectrum are the surgical techniques of intestinal bypass and gastric stapling, which are complex, expensive, and dangerous. The methods of increasing caloric expenditure are more limited. In the past, many physicians prescribed stimulants for this purpose, but this method is quite hazardous and is currently considered to be unethical. The only practical alternatives for increasing physical activity and metabolic rate are the various aerobic exercise regimens. These can be safe and effective, if implemented in a prudent manner and if long-term compliance can be obtained. However, the obese individuals, who need this type of exercise the most,

are generally the ones who find it most distasteful (Stunkard, 1980).

The common element among all the methods described above is their long-term ineffectiveness in controlling obesity. The short-term success rates for various reducing programs are highly variable and the attrition rates are often very high. Those who do manage to lose weight, initially, will most likely regain it within two to three years, if not sooner (Johnson & Drenick, 1977; Stunkard & Penick, 1979). Past epidemiological studies have implied that the probability of indefinite remission was higher for most forms of cancer than it was for obesity. While there have been some advances in recent years, the long-term prognostic picture is still grim for most obese people (K. D. Brownell, personal communication, August 30, 1983).

In a review of existing studies with follow-ups of one year or more, Stunkard and Penick (1979) found the average loss after one year to be approximately ten pounds. This represents only a slight decrease from loss at post-treatment. The variance around this average was extreme and increased as the posttreatment follow-up period increased. The conclusion drawn from these results was that the long-term weight loss achieved in the various programs was not clinically significant. However, others feel that these types of results demonstrate progress in the field over the past ten years (Brownell, 1980).

The study cited above, combined with results showing better short-term weight loss, indicates that existing programs fail to make a lasting impact upon the factors that precipitate or perpetuate obesity. There are indications that extended peer pressure/support after completion of treatment greatly improves the chances for long-term success (Stuart & Mitchell, 1980). The same phenomenon has been consistently demonstrated in the treatment of chemical dependencies. Patients who have been treated for alcoholism or drug addiction have a much better chance of maintaining abstinence if they maintain affiliation with self-help groups such as Alcoholics Anonymous or Narcotics Anonymous (Hoffman & Belille, 1982).

It is this line of reasoning that is used to justify the U.S. Navy's current approach to the treatment of obese service members (Chief of Naval Operations, 1984). Their inpatient obesity treatment program includes diet, health education, and physical training, as do most other programs. However, the core of the program is the Overeaters Anonymous philosophy (Griffith, Owen, & Marcinik, 1981). This philosophy views all obese persons as compulsive overeaters whose use of food is an addiction with psychological features that are essentially the same as alcoholism (Obrien & Bankston, 1984). In fact, the inpatient treatment programs for obesity are conducted in the same facilities as the alcohol and drug treatment programs, utilizing the same staff and most of the same treatment interventions. The

standard operating procedure for Navy residential treatment facilities (Naval Military Personnel Command, 1986) states, "Overeaters experience the normal rehabilitation process used for drug and alcohol abuse" (pp. 5-16). While this type of treatment may be appropriate for obese persons whose overeating is primarily stress related (the adult- onset and/or restrained eater groups), it might be ineffective or psychologically detrimental for persons with the hyper-plastic form of obesity.

There is some support in the literature for use of common components in treatment systems for alcoholism and obesity. Sternberg (1985) examined situations that were high risk for relapse in dieters, alcoholics, smokers, and heroin addicts. She found a high degree of similarity in the situations and cognitions that precipitated relapse and concluded that relapse was a common response to painful or uncomfortable feelings in all four groups. Marlatt's (1985) theoretical article discusses attribution processes in maintenance of abstinence in alcoholics, smokers, gamblers, and dieters. He draws numerous parallels among the groups in the attribution cognitions regarding their ability to control the unwanted behaviors. However, neither of the above authors utilized standard personality measures in the formulation of their assumptions. No studies were found that compared MMPI profiles of obese and alcoholic subjects.

The existing obesity treatment programs (even the more successful ones) treat moderately obese patients as if they

are members of a psychologically homogeneous population. Behaviorally-oriented systems tend to deal exclusively with altering the energy intake and expenditure balance, with little attention being given to the origins of hunger. The insight-oriented and social support groups, such as Over-eaters Anonymous, Weight Watchers, and TOPS, generally stress the social and emotional precursors of hunger. However, they do not address the physiological drive states that precipitate hunger in early-onset (high set point) obese persons.

The medical and psychological communities are beginning to realize that at least part of the obese population are victims of their own social and biological histories. While there is still much to be learned, the accumulating scientific evidence indicates that the etiology of obesity lies in a complex combination of genetic, physiological, psychological, and sociocultural factors. In order to arrive at widely applicable solutions, scientists must first develop methods for isolating each factor and then begin to explore the interactions among factors. At this point in time, the understanding of the individual factors is far from complete. Further research designed to clarify the characteristics of subpopulations within the population of obese persons could significantly impact the clinical conceptualization and treatment of the disorder.

CHAPTER III

STATEMENT OF THE PROBLEM

Research into the physiology of obesity has contributed to the understanding of the neurophysiological mechanisms that tend to protect existing body mass in some obese persons (Hirsch & Batchelor, 1976). These mechanisms exacerbate the problem of sustaining weight loss for these persons (Krotkiewski et al., 1977). However, the fundamental behavioral requirements for reducing (diet and exercise) remain unchanged. The failure of existing treatment systems results from the inability to obtain long-term compliance with these requirements. It is reasonable to assume that a better understanding of the psychology of obesity is a prerequisite to improvements in the treatment systems.

Research into the psychology of obesity is a relatively new paradigm. Previous studies (Johnson et al., 1976; Klesges, 1984) have fallen short of developing a set of psychological descriptors that distinguish between obese and non-obese populations. Among the possible reasons for this shortfall is the tendency to view the obese population as one that is psychologically homogeneous (Garn, 1976). While there is evidence in the literature of the existence of

physiological subpopulations within the obese population (Sjostrom, 1980), there were no studies found that sought to determine whether or not there are psychological differences between these groups. Another possible reason lies in methodological problems that include small group size (Bruch, 1980), lack of age and gender matching (Hibscher & Herman, 1977), and use of nonstandard measures (Klesges, 1984).

The current study attempted to avoid the problems described above through the use of larger group sizes, the use of an exclusively male subject pool, tests for age differences of subjects, and division of the obese group according to age of onset of obesity. In addition, a large set of Minnesota Multiphasic Personality Inventory (MMPI) scales (20) was examined, to increase the chances of developing a list of distinguishing characteristics in the obese group. A newer measure, the Revised Restraint Scale (RRS) (Herman & Polivy, 1982), was added for the same reason and due to its previous success in differentiating between obese and non-obese subjects. It was expected that obese subjects would differ from controls on some of the 21 measures as a result of methodological improvements and that the early-onset obese would differ from the late-onset obese on some measures because of the differences in etiology and course of these types of obesity.

At the same time, this study examined the logic of the U.S. Navy's policy of treating obese service members in the

same residential programs with alcoholic service members. This was accomplished by comparing the pretreatment measures of the obese groups and the alcoholic group. Because of the differences in etiology and impact of the two diseases, it was expected that obese patients would differ from alcoholic patients on some personality test measures.

Posttreatment measures for these groups were compared to determine whether or not the treatment had a differential impact upon these groups. It was expected that the patient groups would differ in their response to treatment, due to the differences in psychological needs among the groups prior to the application of treatments. All of expectations stated above are nondirectional, because there is little previous research that clearly indicates directionality.

Previous studies indicated that the early-onset (hyperplastic) form of obesity is more resistant to change than the late-onset (hypertrophic) form (Krotkiewski et al., 1977). In this study, measures of short-term weight loss for these two groups were compared. It was expected that the late-onset group would lose more weight than the early-onset group.

CHAPTER IV

METHOD

Subjects

The total subject pool consisted of 240 male, active duty Navy personnel between the ages of 20 and 47. One-hundred and twenty of the subjects had been medically diagnosed as chronically obese and were admitted to residential treatment programs for that condition. The Navy's definition of obesity was based upon a table of height/weight ratios contained in a service-wide set of health and physical readiness standards (Chief of Naval Operations, 1984). All obese subjects exceeded the maximum weight allowed for their height by at least 10%. Sixty additional subjects had been medically diagnosed as alcohol dependent and were admitted to residential treatment programs for that condition.

The 180 patient subjects described above were drawn from Navy treatment programs in Jacksonville, Florida, San Diego, California, and Yokosuka, Japan. One-third of the obese subjects and one-third of the alcoholic subjects were drawn from each location. Obese subjects were equally divided into early- and late-onset groups based upon data

from the weight history questionnaire (described in the Instruments section).

It is standard procedure in the obesity treatment programs to screen medical records for signs or symptoms of alcohol abuse. Obese subjects who received a secondary diagnosis of alcohol abuse or dependency based upon this screening (or based upon disclosures made in treatment) were excluded from use in the study. Alcoholic patients who were currently or previously obese were not used as subjects.

A total of 60 control subjects were drawn from a combination of fleet and shore activities. One-third of the controls were drawn from each of the geographic locations in which the treatment programs were conducted. The medical records of these subjects were screened for histories of alcohol abuse or obesity. Control subjects completed all of the instruments completed by patient subjects. Controls who disclosed a history of obesity or alcohol abuse on the weight history questionnaire were excluded from the study.

Instruments

All instruments were paper-and-pencil tests and questionnaires completed independently by each subject. Personality variable measurements were obtained from individual scales of the Minnesota Multiphasic Personality Inventory (MMPI) Group Form (University of Minnesota, 1970). Depending upon the location of the subjects, these were either scored by hand or by computer. The computer

scoring was performed on a Radio Shack TRS-80 Model II microcomputer using a published software package for the group form (Williams, 1981). In addition to the 14 basic clinical and validity scales, several newer scales were utilized. These include: Anxiety (A) and Repression (R), developed by Welsh (1956) using factor-analytic techniques; the rationally constructed Manifest Anxiety Scale (MAS) authored by Taylor (1953); the Ego Strength (Es) scale which was empirically developed by Barron (1953); the Dominance (Do) scale developed by Gough, McClosky, and Meehl (1951); the Control (Cn) scale that was empirically developed by Cuadra (1953); and the MacAndrew Alcoholism Scale (MAC) (MacAndrew, 1965) which was empirically developed and discriminates well between alcoholics and non-alcoholics. Appendix A has descriptions of MMPI scales.

The Restrained Eating Scale was first developed by Herman and Polivy (1975) in a study examining the phenomenon of anxiety-induced eating. They found that subjects scoring high on their measure of conscious dietary control (restrained eaters) ate somewhat more when they became anxious. For unrestrained eaters, the relationship between anxiety and eating was reversed. They hypothesized that anxiety in restrained eaters served to disrupt conscious self-control processes, including dietary restraint. Follow-up studies (Herman & Mack, 1975; Hibscher & Herman, 1977) developed and refined the measure of conscious control into the Revised Restraint Scale (RRS) (Herman & Polivy,

1982). Responses to the questionnaire were found to be quite stable, with a test-retest reliability coefficient of 0.93 over the course of a week.

The weight history questionnaire (Appendix B) was intuitively developed by this author in an attempt to gain more accurate estimates of age of onset for obese subjects. The questionnaire keys on significant events in, and phases of, the subjects' lives and asks them to recall whether or not they were overweight at these times. There is a certain amount of redundancy built into the questionnaire that is designed to induce subjects to think more carefully about their weight histories. The objective is to separate the subjects into two groups, early-onset (prior to completion of high school) and adult-onset, based on the clinical judgements of health care providers. While no normative data exist for this questionnaire, it should be an improvement over the procedure of simply asking subjects, "At what age did you become obese?" Two questions have been added to the questionnaire to screen for current or historical alcohol abuse in obese subjects, which could be a confounding factor.

Procedure

The three experimental groups (early- and late-onset obese and alcoholic) were tested at the beginning, and again at the end of the treatment programs for their respective conditions. The alcoholism treatment program is six weeks

in length and consists of substance abuse education, problem-solving group counseling (conducted by paraprofessionals under the supervision of Navy psychologists), extensive participation in Alcoholics Anonymous, and physical fitness training. The obesity treatment program is the same length as the alcoholism treatment and is very similar in content. The major differences are the replacement of Alcoholics Anonymous with Overeaters Anonymous and the addition of extensive diet and nutrition education, along with a specific calorie per day dietary limit (the individual limit varies among patients, as prescribed by dieticians). Only data from patients completing the full six-week programs were used. Fewer than 5% of obese patients failed to complete the full six weeks. However, approximately 20% were excluded from the study due to the assignment of secondary diagnoses of alcohol abuse or dependency. Alcoholic and control subjects were selected based upon approximate age matching with obese patients.

All subjects received a verbal briefing, that was very general in nature, regarding the nature and purpose of the study. They then signed and dated a consent form (Appendix C) that contained a synopsis of the briefing. As is stated on the consent form, subjects had the option of refusing to participate or to cease participation at any time.

The pretreatment testing for obese subjects consisted of the weight history questionnaire, the Revised Restraint Scale, and the MMPI. The alcoholic and control group

subjects were also required to complete all three instruments, since this provided a level of standardization between groups and gave additional screening information. Subjects completed all instruments independently and there was no time limit. Obese and alcoholic patients completed the RRS and the MMPI again at the end of the treatment period. Control subjects completed the same instruments approximately six weeks after the initial testing.

Each weight history questionnaire was evaluated independently by three persons. These persons included various combinations of a psychiatrist, three psychologists, and an internal medicine specialist. Each made a subjective determination as to the approximate age of onset of obesity using the options, early-onset or adult-onset. All of the clinicians involved were briefed on the nature and purpose of the study. They also listened to a verbal review of two studies that addressed the issues of cellularity and age of onset in obesity (Hirsch & Batchelor, 1976; Keesey, 1980). They were instructed to use the background information and their clinical judgement in deciding to which group each subject should be assigned. If all of the raters agreed in this determination, their decision was applied to the subject. If the raters did not agree on the age of onset, the subject's data were not used. Data from the RRS for all subjects were analyzed as the raw point total, which has a possible range of 0 - 35. All MMPI scale scores were converted to T scores that were K corrected

where appropriate (University of Minnesota, 1970). The success measure for obese subjects was weight loss from time of admission to time of discharge from the treatment program.

Design

Independent Variables

The between groups variable used in analyses of pretreatment personality factors and dietary restraint was treatment group. All patient groups (early-onset obese, late-onset obese, and alcoholic) were compared to one another and to the control group. An additional between-groups variable for pretreatment measures was location. This was used to screen for preexisting geographic differences within the groups.

For posttreatment measures, the only independent variable was treatment group. The three patient groups were compared to controls to test for presence or absence of response to treatment. Patient groups were compared to one another to test for differential responses of personality factors and dietary restraint.

For measures of short-term weight loss, the between groups variable was treatment group. The early- and late-onset obese were compared on this measure.

Dependent Variable

Measures of pretreatment personality factors were obtained by converting raw scores to T scores on the various MMPI scales. The pretreatment dietary restraint measure was the raw score (0 - 35) on the RRS for each subject. The posttreatment test scores were analyzed, and were adjusted for pretreatment scores through an analysis of covariance (ANCOVA).

A measure of short-term weight loss for early- and late-obese subjects was obtained by recording the subjects' weight at the time of admission and discharge from the treatment programs. Differences in posttreatment weights for the two groups were examined using an ANCOVA, with pretreatment weight as the covariate.

CHAPTER V

RESULTS

In order to clearly describe the very large volume of data, the analyses will be presented in two sections. The first section will examine differences in pretreatment measures among the Early-Onset Obese (OBE), Late-Onset Obese (OBL), Alcoholic (ETOH), and Control groups (n=60 per group). The second section describes changes in test scores as a result of treatments. This section also contains a description of the short-term weight loss results for the early- and late-onset obese groups.

The significance level for all analyses of test scores was initially set at $p=.05$. However, due to the large number of dependent variables, using $p=.05$ for each individual analysis would not provide adequate protection against Type I errors. Dunn (1961) advocates calculation, in advance, of an allocation of the total error rate evenly across a group of related experiments. While Dunn's article actually discusses multiple comparisons among means, the same principle is used by Wilcox (1987) when he advocates the use of a Bonferroni Inequality to determine the safe level across a family of experiments. Wilcox' error rate adjustment is given by the formula, $\alpha = 1-Pr(a_1)+...+Pr(A_k)$,

where Pr is the probability of Type I error and A is an individual experiment. In order to maintain an overall Type I error probability of .05 for 21 experiments, the significance level of each must be set at p=.00023809. This level was used to determine whether or not there were significant overall differences on each of the ANOVA's and ANCOVA's for pretreatment and posttreatment measures.

In order to screen for possible age differences, a preliminary group (4) x location (3) analysis of variance was performed on the subject's ages. The four levels of the group variable were OBE, OBL, ETOH, and Control. The three levels for location were Jacksonville (JAX), San Diego (SD), and Yokosuka (YOKO). There were no significant main effects for group or location. The overall analysis did show a significant group by location interaction, $F(6,228)=2.33$, p=.0334. However, comparisons of group by location means, using Tukey's HSD procedure, revealed no differences at the p=.05 level. (Details of the F statistics are found in Appendix D, Table I.) Based upon the results of this analysis, age was not considered as a factor in any subsequent analyses.

Pretreatment Measures of Patient Groups and Controls

Group (4) x location (3) ANOVA's were performed on the Revised Restraint Scale scores (RRS) and 20 Minnesota Multiphasic Personality Inventory (MMPI) scale T scores.

The four groups involved were the OBE, OBL, ETOH, and Control subjects. The three locations were JAX, SD, and YOKO. There were no significant differences found on any pretreatment measures as a function of location. There was a significant group by location interaction found for the MMPI R scale, $F(6,228)=3.99$, $p=.001$, indicating some differences in the levels of repression among groups in different locations. Within the OBE group, all locations differed at the $p < .01$ level. The SD subjects had the highest mean, followed by JAX and YOKO, in that order. ETOH subjects in JAX had a significantly lower mean than ETOH subjects in the other two locations ($p < .01$). Control subjects in JAX had a higher mean than controls in the other locations ($p < .01$). There were no location differences within the OBL group. Locations means for the groups are found in Appendix D, Table II. Possible reasons for these differences and implications for interpretation of group differences on this measure will be discussed in Chapter VI.

There were 15 significant main effects for treatment groups identified by this analysis. The RRS, L, HS, D, HY, PD, MF, PA, PT, SC, MA, SI, MAS, DO, and MAC scales showed differences at or beyond the .0002 level (Appendix D, Table III). A listing of group means and contrast results for measures showing differences on the ANOVA's is found in Appendix D, Table IV. The group means are graphically presented in Figures 1 and 2.

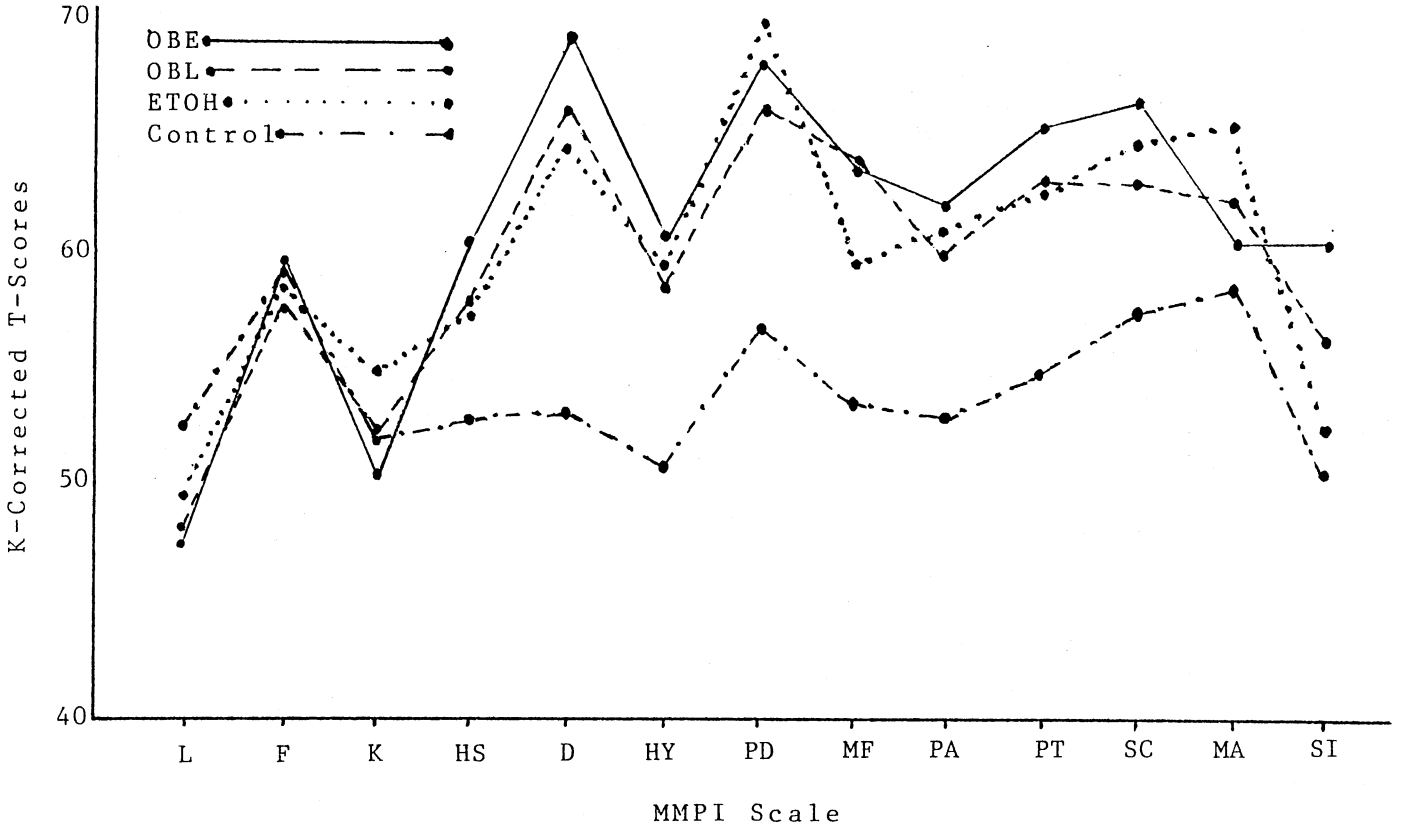


Figure 1. Pretreatment Group Means (n=60) for MMPI Validity and Clinical Scales

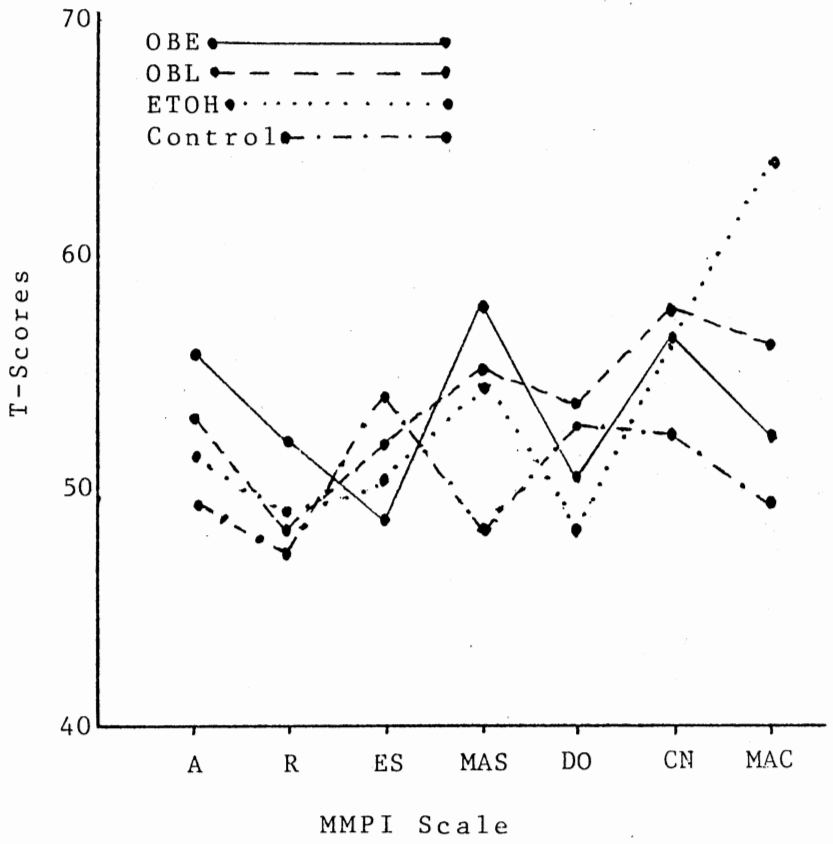


Figure 2. Pretreatment Group Means (n=60) for MMPI Supplemental Scales

The most consistent finding in comparisons of pretreatment groups means was that the control group scores were significantly lower than all three patient groups and that there were no significant differences among the patient groups. This was the case for the MMPI HS, D, HY, PD, MF, PA, PT, SC, and MAS scales. This would indicate that control subjects showed less pathology on the MMPI than patient subjects, although that issue is debatable for some scales. This will be discussed, in detail, in the next chapter.

On RRS scores, the ETOH group mean (9.43) was lower than all other groups. The OBE and OBL group means (21.3 and 20.1, respectively) were significantly higher than the control group mean (13.95). This clearly showed obese subjects to be different from non-obese subjects in their levels of concern about eating and weight fluctuations. The expected differences between early- and late-onset obese did not occur.

On the L scale, the control group scored significantly higher than the ETOH and OBL groups, indicating slightly more defensiveness in the controls' approach to the MMPI. The OBE group did not differ from any other groups and the ETOH and OBL groups did not differ.

The ETOH group scored significantly higher than OBE and control on the MA scale, indicating that alcoholic patients reported higher activity levels than early-onset obese and

non-patients. There were no other significant contrasts on this measure.

Comparisons of the SI scale group means showed that the OBE group was significantly more socially isolated than all other groups. The OBL group was also significantly higher than controls on this measure, indicating that obese persons, in general, experience more problems with social isolation than non-obese persons. There was no difference between alcoholics and controls on this measure.

The only significant contrast for the DO scale was between OBL and ETOH indicating that the late-onset obese tend to be more dominating in interpersonal situations. On the MAC scale, the ETOH group endorsed significantly more items correlated with alcohol abuse than all other groups, as would be predicted by the stated purpose of the scale. The OBL group also scored significantly higher than controls.

Posttreatment Measures

The statistical procedures performed on posttreatment RRS and MMPI measures were designed to test for differential effects of the treatment programs upon the three patient groups, as compared to changes in the control group. The initial analysis was a one-way, four-group ANCOVA of the 21 test scores, using pretreatment scores as the covariate. This procedure indicated the presence of differences in group means only for the MMPI MF, $F(3,235)=11.53$, $p < .001$,

and MA, $F(3,235)=7.23$, $p < .001$ scales. Details of the F statistics for this set of analyses are found in Appendix D, Table V.

Contrasts among means for both the MF and MA scales showed that each patient group was significantly different from the controls. However, none of the expected differences among the patient groups were found. This would indicate that the treatments had an approximately equal impact upon the patient groups' attitudes about sex roles and reported activity levels. All of the group means, adjusted for pretreatment scores, are graphically presented in Figures 3 and 4. Adjusted group means and contrast results for the MF and MA scales are presented in Appendix D, Table VI.

To test for differences in short-term weight loss between the OBE and OBL groups, a one-way, two-group ANCOVA was performed on posttreatment weight, using pretreatment weight as the covariate. The analysis yielded $F(1,118)=1.124$, $p=.291$. Based upon the result, it was concluded that there was no difference in short-term weight loss between the groups. The ANCOVA results for this measure are contained in Appendix D, Table VII.

The question of whether or not the obese subjects were successful in losing weight during treatment was not formally addressed in the design of this study. However, this is an issue of interest to those involved in conducting the treatment programs. To answer this question, separate

ANOVA's were conducted comparing the pretreatment and post-treatment weights of the OBE and OBL subjects. For the OBE group, the pretreatment mean was 237.3 pounds and posttreatment mean of 218.8 pounds. The ANOVA showed a significant difference ($F(1,118)=14.23$, $p=.0003$). The pretreatment and posttreatment means for the OBL group were 236.32 and 216.65 respectively. The ANOVA for this was also significant ($F(1,118)=21.41$, $p=.0001$). These analyses showed that both groups lost significant amounts of weight during treatment, but the previously reported ANCOVA on weight measures detected no difference between the groups. Details of the ANOVA's are presented in Appendix D, Tables VIII and IX.

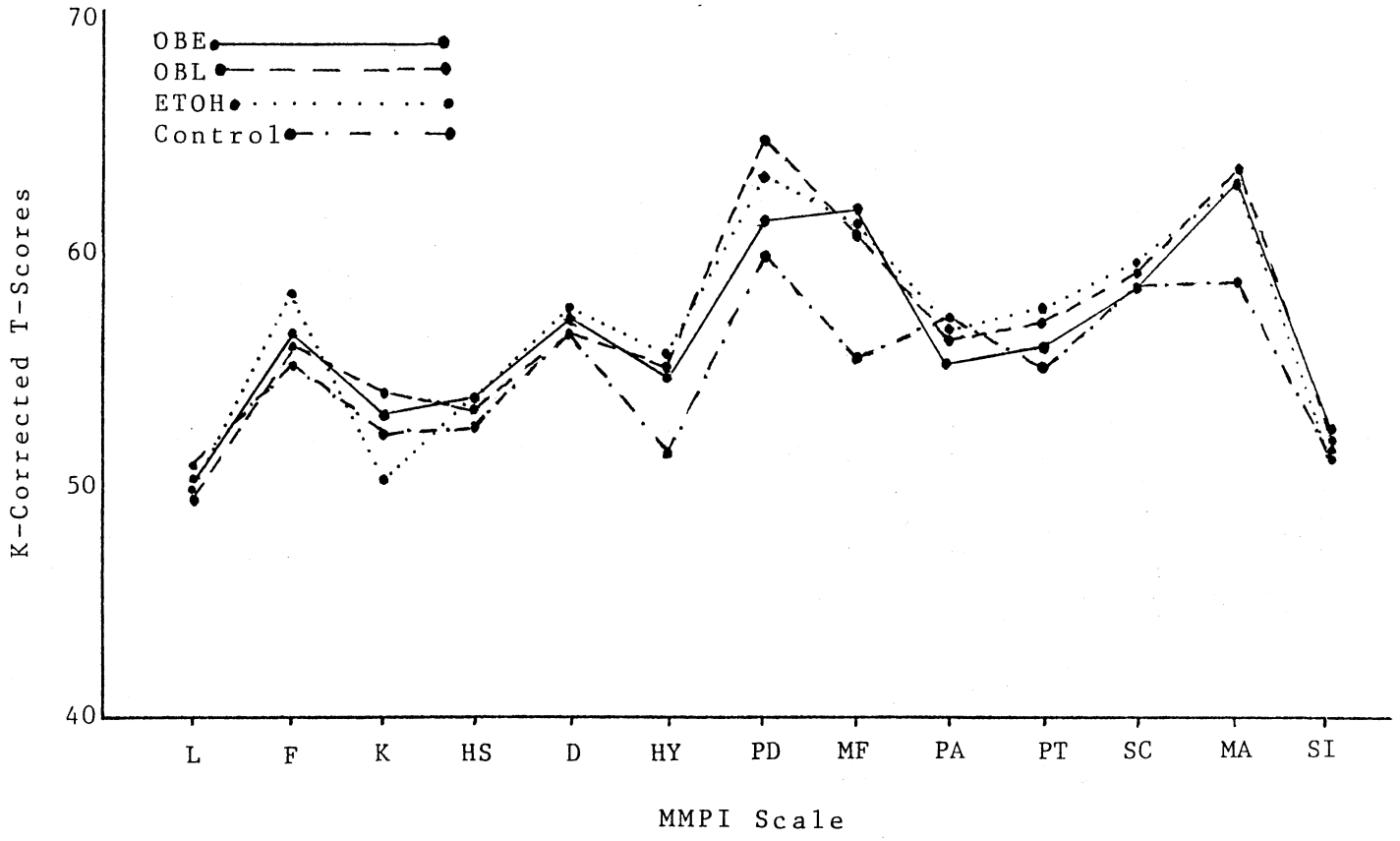


Figure 3. Posttreatment Group Means (n=60) Adjusted for Pretreatment Scores on MMPI Validity and Clinical Scales

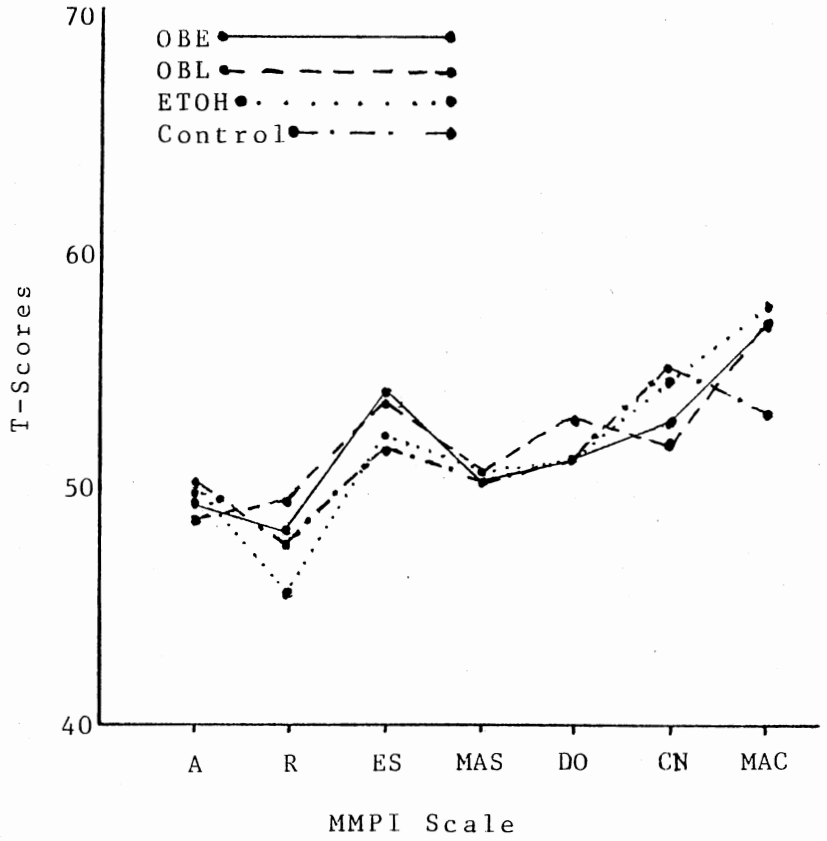


Figure 4. Posttreatment Group Means (n=60) Adjusted for Pretreatment Scores on MMPI Supplemental Scales

CHAPTER VI

DISCUSSION

This study had two major purposes. The first was to identify a complex of psychological factors that differentiated among the various groups prior to the application of treatments. The second was to determine which of these factors were impacted by the treatments and whether or not there was a differential impact among the treatment groups.

Because of the primary utility of the dependent variables lies in the clinical information they provide, the first section of this chapter will present standard clinical interpretations of group mean profiles on the pretreatment Minnesota Multiphasic Personality Inventories (MMPI). Possible meanings of the Revised Restraint Scale (RRS) group means will also be discussed. The second section will examine significant findings of differences among the groups on pretreatment measures and discuss the research and clinical implications of these. The significance of location effects will be considered in this discussion. A third section will discuss the presence and absence of treatment impact, as measured by the posttreatment MMPI and RRS scores. The last section will present ideas for further

research in the paradigm generated by this study and propose modifications to existing treatment systems.

Interpretations of Pretreatment Personality Factors

Visual examination of the pretreatment group mean MMPI profile for the control group would not result in an assessment of any psychopathology. The validity scales indicate that these subjects were within the average range in their willingness to disclose worries and unusual experiences and that the average profile was safely interpretable. There were no scale elevations in excess of 60 T. The highest clinical scale was the MA score, which was in the upper end of the normal range, indicating that these subjects had a slightly higher than average activity level. The SC and PD scales were also in the upper half of the normal range. This suggests a possibility that these subjects may be somewhat higher than the average male in their tendency to engage in unconventional thought patterns and to complain about authority and boredom.

While none of the scale elevations would be considered clinically significant, the means that were above average could be explained by the occupational status of these subjects. Being on active duty in the Navy requires members to be physically vigorous and to tolerate frequent changes in locations and jobs. These factors could contribute to the elevations on the MA and SC scales. The rigid authority

structure in the military could contribute to the slight elevation on the PD scale. Clinically, the control group appeared to be well within the expected range of personality factors for Navy men.

The OBE and OBL groups had mean profiles that showed very few statistical differences and almost no clinical differences from one another. The validity scales indicate that the average profile for both groups was valid and that the average subject was somewhat more willing than average to disclose minor personal shortcomings and unusual experiences. For both groups, there were seven scales that exceeded 60 T (D, PD, MF, PA, PT, SC, and MA), with D and PD being the two high scales. The two-point code interpretation of the mean profile for these groups would speculate these persons were experiencing some sort of acute difficulty in their lives and that they may find themselves recurrently at odds with societal demands and values.

Greene's (1980) interpretive guidelines state that the obese patients' elevations on the D scale indicate that they are, "dissatisfied with something or with themselves, but they may not recognize this state as depression . . . or they may have learned to adjust to a chronic depressed existence" (p. 77). Greene's interpretive statements regarding the PD scale include, "they may be responding to situational conflict or they may have adjusted to a habitual level of interpersonal and social conflict" (p. 89). He

further contends that, if the conflict is situational, the PD elevation should decline as the conflict resolves.

The standard interpretation of both of the high MMPI scales for obese subjects allows for the possibility of situational and trait components in the elevations. Because all of these subjects completed the MMPI within two days of their admission to residential treatment, it could be reasonably postulated that this situation could account for some portion of the elevations. If so, then it could be reasonably expected that these elevations would moderate by the time of the posttest, since these subjects were being released from treatment at that time and should be experiencing lower stress levels.

There is also an intermediate term stressor that could influence the clinical profiles of the obese subjects. The military services are quite intolerant of obesity in its members. All of these subjects were in jeopardy of being administratively separated from the Navy, if they did not meet body composition standards within a reasonable period of time. (The definition of "reasonable" varies widely from case to case, dependent upon a number of location, occupation, and interpersonal factors.) The stress of this uncertainty could certainly precipitate varying levels of depression and anger in obese Navy men.

There is evidence, from previous research, (Scott & Barrofio, 1986) that the psychological profiles of the obese subjects may be more indicative of enduring traits than of

situational stressors. This research will be discussed in the section on group differences.

The group mean pretreatment MMPI profile for alcoholic subjects was within expected limits for adult male alcoholics as indicated by Lanyon (1968). The validity scales indicated that the average profile was safely interpretable and that these subjects were reasonably self-disclosing in their response to the test. There was one scale elevation of 70 T (PD) and six elevations between 60 T and 65 T (D, PA, PT, SC, MA, and MAC). The two-point code interpretation (as was the case with the obese subjects) suggests that these subjects are dealing with an acute conflict in their lives and that this may be a recurrent pattern.

Utilizing the same single scale interpretations (Greene, 1980) cited for the obese group, there would be a slightly different evaluation for the ETOH group, due to a higher PD elevation. It would be expected that these subjects experience chronic difficulty with authority figures precipitated by egocentric and/or irresponsible behavior. These subjects are likely to be superficially charming, but tend to lapse into sociopathic behavior in longer relationships or when stressed. The D scale elevation is more likely to be a response to external pressure, rather than itrapsychic conflict and will probably moderate when the pressure eases.

Due to the recent inpatient status of the alcoholic subjects, the same postulations regarding state versus trait components of these elevations should be considered. However, the added elevation on the PD scale would indicate that these subjects are more likely to have chronic conflicts with societal limits.

Because of the relative newness of the scale, there is little in the way of normative data available for the RRS. The obvious components of the scale are concerned with dieting and weight fluctuations. The original research on the scale (Herman & Polivy, 1975) showed that it discriminated well between obese and non-obese subjects and that obese persons scored higher. While they identified some non-obese subjects who were restrained eaters and some obese subjects who were unrestrained, overall, the obese group invested much more cognitive effort in controlling their consumption. This is consistent with the present findings, in which the OBE and OBL groups' mean RRS scores (21.3 and 20.1 respectively) exceeded the control groups' average of 13.9. However, it does not explain the significantly lower mean score for alcoholic subjects (9.48). It is possible that the alcoholics' apparently low level of concern with body weight and dieting is secondary to a higher level of concerns with other problems. However, it is also possible that some personality factors related to eating behavior are negatively correlated to factors related to alcoholism.

In a more recent study comparing the RRS and other cognitive measures, Ruderman (1985) concluded that restrained eaters are more prone to rigid, absolute beliefs than are unrestrained eaters. If her findings were applied to these data, it would be assumed that the alcoholic group would be the most tolerant and relativistic in their thinking. Examination of the scales that are likely to be correlated with these attributes (high MF, low PA), does not support this assumption. The MMPI data suggest that the relationship between RRS scores and intellectual rigidity is much more complex than concluded by Ruderman.

Pretreatment Differences Among Groups

Contrary to the findings of various studies cited in the literature review, the results of this study show a large group of MMPI scales that differentiate between obese subjects and controls. For the majority of the dependent measures, the obese groups differed from controls in the upward or pathological direction. However, the term pathological must be used with some caution for two reasons. First, some of the scales do not purport to measure psychopathology. Two widely used manuals for interpretation of the MMPI (Graham, 1987, Greene, 1980) describe elevations on the MF, ES, DO, and CN scales primarily in terms of personality style differences that are not necessarily associated with psychopathology. Second, in the interpretation of the MMPI, what is statistically significant is not necessarily

clinically significant. On some of the MMPI scales, a difference between group means of less than five T score points resulted in a significant statistical finding. However, descriptive statements generated by most scale elevations would not differ based upon a five-point difference. Throughout this section, descriptions of differences among groups will emphasize the relative strength of traits, rather than the presence or absence of traits among the groups.

On ten (L, HS, D, HY, PD, MF, PA, PT, SC, and MAS) of the 20 MMPI scales studied, both obese groups differed in the same direction from the controls. A very superficial interpretation of these differences might conclude that obese persons have lower self-esteem, are more hypochondriacal, more depressed, more histrionic, more angry, more aesthetic, more interpersonally sensitive, more worrisome, more unconventional, and more anxious than non-obese persons. However, the factors that influence differences on the MMPI are far too complex to justify such a simplistic set of assumptions.

Among the three validity scales of the MMPI, the obese groups differed from controls only on the L scale. The obese groups scored 4.7 to 5.3 lower, which corresponds to a difference of two to three raw score points. This indicates that obese subjects may be more willing than the non-obese

to acknowledge personal flaws. Standard clinical interpretations would generally not differ based upon this T score difference.

Among the ten basic clinical scales, the only one on which the obese groups did not score higher was the MA scale. The obese groups did not report activity levels that differed from non-obese subjects. The obese groups did report a higher level of somatic concerns on the HS scale. This could probably be explained by the increased incidence of physical disorders associated with obesity and/or by the negative body image found in many obese persons (Stunkard & Mendelson, 1967). The D scale is generally positively correlated with the HS scale and obese subjects were much higher than controls (13 to 16 T). This scale is fairly sensitive to situational stressors and the obese subjects may have been responding, to some extent, to their current career jeopardy. However, a study of obese civilian outpatients (Scott & Barrofio, 1986) showed a similar gap between obese subjects and controls. Those authors reported a number of findings that paralleled those of the current study. These will be discussed, in detail, later in this section.

The relative elevations of obese groups on the HY scale (7.5 to 9.3 T) is statistically significant and could indicate a greater tendency in obese subjects to avoid unpleasant emotional issues. The traditional name for the PD

scale (Psychopathic Deviate) is somewhat misleading in consideration of moderate elevations. The relative elevation in obese groups of 9.5 to 10.5 T does not indicate that these subjects are more prone to antisocial behavior than controls. It does suggest that they are more distressed by social and organizational demands made upon them and that they may respond in a passive-aggressive manner. As with the D scale, there may be a significant situational factor in this elevation.

The MF scale is generally considered to be less sensitive to situational factors, but is related to intelligence, education, and socioeconomic levels. The differences of 10.3 to 10.8 T in obese groups indicate that they are more aesthetic and tend to approach problems in a more indirect or intellectual manner than controls. Obese subjects were higher than controls on the PA scale by differences of 8.1 and 10.2 T. Clinically, this indicates a somewhat higher level of interpersonal sensitivity, rigidity of beliefs, or suspiciousness. The OBE and OBL groups differed from controls by 11.1 and 8.8 T on the PT scale. These show clinically significant increments in levels of worry and/or personal dissatisfaction.

The OBE and OBL differences of 9.5 and 7.5 on the SC scale are somewhat more difficult to interpret. In light of other elevations, it is likely to indicate a somewhat higher level of creativity and unconventional thinking than

controls, but may also be an indicator of some social alienation experienced by obese persons in our culture. The latter possibility appears to be supported by the obese groups' higher scores on the SI scale indicating less social contact. The obese groups' relative elevations of 7.1 to 9.3 T on the MAS scale may be confounded somewhat by their physical conditions, since many of the MAS items involve physical manifestations of anxiety such as sweating, blushing, and gastrointestinal complaints.

The overall assessment of this set of contrasts is that the MMPI successfully measured a number of personality factors that distinguished obese and alcoholic subjects from controls. Of equal importance for the purposes of this study is the fact that the OBE and OBL groups did not show differences on any of these scales, except for SI. The early-onset group was significantly higher than the late-onset group (4.6 T). While this would not result in a different clinical interpretation, it suggests that childhood obesity impacts adult socialization to a greater degree than adult-onset obesity.

It should be emphasized, though, that the similarities between the OBE and OBL groups far outweighed the differences. This was contrary to stated expectations and has implications for future research that will be discussed in the final section of this chapter.

It is acknowledged that the obese groups' inpatient status and military situations may have influenced some of

the differences from controls. However, there is one study that appears to counter these arguments. Scott and Barrofio (1986) used the MMPI to investigate similarities and differences among female hospitalized anorexics and bulimics, morbidly obese outpatients, and normal weight controls (n=30 per group). For this discussion, the focus of interest is upon the obese and control groups. They found that the obese subjects had significant elevations, relative to controls, on eight of the ten basic clinical scales. The exceptions were the MF and MA scales. This parallels the findings of the current study on all scales except MF, which has somewhat different elevation meanings for males and females. The other scales use the same interpretive statements for the same elevations in men and women (Graham, 1987; Greene, 1980). That study also found a profile configuration that was very similar to this study, with D and PD as the two high scales. The obese groups in these two studies were quite different demographically (female versus male; civilian versus military; outpatient versus inpatient), yet they showed remarkably similar clinical profiles. In the absence of some undetected common denominator for these groups, it must be assumed that the similarities in their profiles are related to the condition of obesity.

Another very important finding on these sets of analyses was the lack of differences between the obese and alcoholic groups. This was true for eight of the ten basic clinical scales and one of the additional MMPI scales.

Those scales that did show differences among these groups can be explained in terms of the premorbid differences between the disorders and differences in the physical and social impact of alcoholism and obesity.

The similarities between the two-point code interpretations and most of the individual scales for obese and alcoholic subjects was striking. This appears to have significant practical implications for clinicians dealing with obese and alcoholic patients, in that the two groups present similar psychological needs, as measured by the MMPI. These findings provide a degree of support for the Navy's policy of treating alcoholism and obesity with parallel systems. However, this support must be evaluated in light of observations of treatment impacts upon these needs, which will be discussed in the next section.

The single dependent measure that showed a significant group by location interaction was the DO scale. The pairwise comparisons resulted in seven significant contrasts, but no meaningful pattern was apparent among these. It was concluded that this may be a chance occurrence of the type that can be expected when conducting large numbers of analyses. In any case, these findings do not appear to have any value with regard to the purposes of this study.

Measures of Response to Treatments

The logic underlying the use of the ANCOVA to test for differences in response to treatments is widely accepted.

Pretreatment to posttreatment differences would yield the same results as the ANCOVA only if the regression of the pretreatment measures on the posttreatment measures are linear. This assumption could not be met with the MMPI, since clinical experience indicates that these scores tend to be more variable at higher elevations. There were no data available that addressed this issue for the RRS and body weight measurements. Therefore, it was considered prudent to use the ANCOVA to test for response to treatment on all measures.

These procedures yielded significant findings on only two of the 21 test scores. All three patient groups showed significant changes, as compared to controls, on the MF and MA scales. There were no differences among the patient groups in response to treatment, contrary to the stated expectation. The changes among patient groups on the MA scale are possibly residual effects of the general structure of the treatment programs. The pace of operations in all Navy residential treatment centers is quite rapid. This is a function of the "stress innoculation" philosophy of the programs and the large number of treatment interventions attempted in six weeks. The patients have little or no idle time and are constantly in interaction with others. It is understandable that they would report higher activity levels after six weeks of maintaining this schedule. Another possible contributor to this effect is the daily exercise component of the programs. Patients leave the programs in

better physical condition than they were in when they entered. An increase in physical vigor could contribute to an elevation on the MA scale.

The changes among patient groups on the MF scale are more difficult to link to the treatments. An upward change in this scale for males could indicate various combinations of decreases in gender role stereotyping, increases in aesthetic interests, or increased intellectual/introspective approaches to problem-solving. Through group counseling and various educational programs, the treatment systems attempt to foster formation of emotionally intimate, therapeutic relationships among patients. There are also a few of the didactic sessions that address sexuality and sex role stereotypes. These features of the system could account for relative elevations on the MF scale.

It can be argued that the changes described above are beneficial, but it is unclear whether or not they promote the long-term lifestyle modification necessary for successful treatment of alcoholism and obesity. Perhaps more noteworthy than the characteristics that changed, are the ones that did not change. The two-point code characteristics that were common to all three patient groups did not appear to respond to the treatments. This observation is significant to the clinical interpretation of pretreatment measures, in that it indicates that these characteristics are not significantly influenced by the acute stress of being admitted to treatment.

Throughout treatment, the patients are imbued with the idea that the skills they acquire in the program, properly applied, will improve their lives and restore their good standing with the Navy. If this idea is, in fact, assimilated by the patients, then this should serve to reduce the intermediate term stressors that may contribute to elevations on the LD and PD scales. If the situational explanations of the D and PD elevations are eliminated, then it must be assumed that this profile describes an enduring set of traits in obese and alcoholic Navy men.

One possible alternative explanation should be considered. It may be that patients become institutionalized to a significant degree during six weeks in treatment. If so, then the fact of being discharged from the program would create a new set of situational stressors that could serve to elevate the scales in question. However, the nature of this conflict would be qualitatively different from the type that is generally assumed for elevations on these scales.

The measures of short-term weight loss for the early- and late-onset obese groups did not support the findings of Krotiewski et al. (1977). This lack of concordance may have been the result of various methodological differences. The earlier study used actual histological examinations to differentiate between hyperplastic and hypertrophic obese subjects and measured weight loss over a longer period. The possibility must be considered that this study's use of questionnaire data to determine age of onset for obesity is

not effective in determining cellularity differences. However, a follow-up study at six months posttreatment (the same period used in the previous study) might result in the expected differential between OBE and OBL groups.

Research and Clinical Implications

The current study succeeded in its goal of demonstrating differences on personality measures between obese subjects and non-obese controls. However, it did not find the expected differences between the early- and late-onset obese groups. This does not necessarily mean that differences do not exist. An underlying assumption in the postulation of differences was the concomitance of age of onset and the cellularity of obesity. In order to clearly establish this relationship, it would be necessary to assess cellularity through the use of adipose tissue biopsies and correlate these to the weight histories of the subjects. Since this procedure was not possible in this study, it is possible that the cellularity assumption was invalid. With the appropriate facilities and professional assistance (from a surgeon and a pathologist), the suggested procedure could be accomplished. There could be a problem, though, in obtaining sufficient numbers of subjects who would submit to such a procedure.

It is also possible that the failure to find differences resulted from use of wrong measures. Use of a similar design with different measures (perhaps the 16PF)

that may be more sensitive to subtle differences could solve the problem. Using the same raw data, subscales available for some of the MMPI basic clinical scales may reveal differences. However, the small number of items and lack of validity data for many of these subscales could be a source of confounding in this technique.

The failure to find treatment effects, much less differential effects, may be a result of inadequate measurements or it may indicate that the treatments do not significantly impact personality factors. The solution to this theoretical dilemma will require two separate research approaches. First, different measures should be analyzed in an attempt to demonstrate changes and differentials that this study could not detect. Second, long-term follow-up studies on these subjects could determine whether or not personality factors have any predictive value for long-term treatment success. If they do not, then major paradigmatic changes are indicated in order to develop a scientific basis for treatment systems.

In the interim, the treatment programs must examine their intervention systems to determine why they apparently do not impact the major clinical scale elevations. This will require establishment of a new program evaluation system that utilizes the most current clinical knowledge regarding the psychological needs of the patients. The profile types described in this study are assumed, by most

clinicians, to be quite resistant to change. This assumption is supported by the observed recidivism rates in obesity and alcoholism. However, with improved interventions and staff training, it may be possible to improve the short-term outcomes, as assessed with standard psychological measures. This will be a prerequisite for longitudinal studies to determine what short-term changes are correlated with long-term success.

BIBLIOGRAPHY

Ayoob, K. T. (1984). Predicting success in a behavior modification weight loss program using the Minnesota Multiphasic Personality Inventory. Dissertation Abstracts International, 44(12-A).

Barron, F. (1953). Ego-strength and the management of aggression. In G. S. Welsh and W. D. Dahlstrom (Eds.), Basic reading on the MMPI in psychology and medicine. Minneapolis: University of Minnesota Press.

Bjorntorp, P. & Sjostrom, L. (1971). Number and size of adipose tissue fat cells in relation to metabolism in human obesity. Metabolism, 20, 703-713.

Brook, C. G. D., Huntley, R. M. C., & Slack, J. (1975). Influence of heredity and environment in determination of skinfold thickness in children. British Medical Journal, 2, 719-721.

Brownell, K. D. (1982). Obesity: Understanding and treating a serious, prevalent, and refractory disorder. Journal of Consulting and Clinical Psychology, 50, 820-840.

Bruch, H. (1980). The importance of overweight. In P. J. Collipp (Ed.), Childhood obesity (2nd ed.). MA: PSG Publishing Co.

Chief of Naval Operations. (1984). Instruction 6110.1B (Enclosure 1). Philadelphia: Naval Publications Center.

Coates, T. J. & Thorensen, C. E. (1980). Obesity among children and adolescents. In B. B. Lahey & A. E. Kazdin (Eds.), Advances in child clinical psychology. New York: Plenum Press.

Collipp, P. J. (1980). Obesity in childhood. In A. J. Stunkard (Ed.), Obesity (pp. 405-414). Philadelphia: Saunders.

Cuadra, C. A. (1953). A scale for control in psychological adjustment (Cn). In G. S. Welsh and W. D. Dahlstrom (Eds.), Basic readings on the MMPI in psychology and medicine. Minneapolis: University of Minnesota Press.

Dunn, O. J. (1961). Multiple comparisons among means. American Statistical Association Journal, 56, 52-64.

Ellsworth, G. A., Strain, G. W., Strain, J. J., & Vaillant, G. E. (1986). Defensive maturity ratings and sustained weight loss in obesity. Psychosomatics, 27, 772-781.

Garn, S. (1976). The origins of obesity. American Journal of Diseases of Children, 130, 465-467.

Gough, H. G., McClosky, H., & Meehl, P. E. (1951). A personality scale for dominance. Journal of Abnormal and Social Psychology, 46, 360-366.

Graham, J. R. (1987). The MMPI: A practical guide (2nd ed.). New York: Oxford University Press.

Greene, R. L. (1980). An MMPI interpretive manual. New York: Grune & Stratton.

Griffith, W. L., Owen, E., & Marcinik, E. J. (1981). Pilot inpatient obesity treatment program. U.S. Navy Medicine, 72, 1-5.

Herman, C. P. & Mack, D. (1975). Restrained and unrestrained eating. Journal of Personality, 43, 647-660.

Herman, C. P. & Polivy, J. (1980). Restrained eating. In A. J. Stunkard (Ed.), Obesity. Philadelphia: Saunders.

Herman, C. P. & Polivy, J. (1975). Anxiety, restraint, and eating behavior. Journal of Abnormal Psychology, 91, 374-380.

Hibscher, J. A. & Herman, C. P. (1977). Obesity, dieting, and the expression of 'obese' characteristics. Journal of Comparative and Physiological Psychology, 91, 374-380.

Hirsch, J. & Batchelor, B. (1976). Adipose tissue cellularity in human obesity. Clinics in Endocrinological Metabolism, 5, 299-311.

Hoffman, N. G. & Belille, C. A. (1982). Chemical abuse/addiction treatment outcome registry. St. Paul, MN: Ramsey Medical Education and Research Foundation.

Hutzler, J. C., Keen, J., Molinari, V., & Carey, L. (1981). Superobesity: A psychiatric profile of patients electing gastric stapling for the treatment of morbid obesity. Journal of Clinical Psychiatry, 42, 458-462.

Johnson, D. & Drenick, E. J. (1977). Therapeutic fasting in morbid obesity: Long-term follow-up. Archives of Internal Medicine, 137, 1381-1382.

Johnson, P. R., Stern, J., & Gruen, R. (1976). Development of adipose depot cellularity, plasma insulin, and insulin resistance in the Zucker obese female rat. Federation Proceedings, 35, 657.

Keesey, R. E. (1980). A set-point analysis of the regulation of body weight. In A. J. Stunkard (Ed.), Obesity. Philadelphia: Saunders.

Klesges, R. C. (1984). Personality and obesity: Global versus specific measures? Behavioral Assessment, 6, 347-356.

Kolotkin, R. L., Revis, E. S., Kirkley, B. G., & Janick, L. (1987). Binge eating in obesity: Associated MMPI characteristics. Journal of Consulting and Clinical Psychology, 55, 872-876.

Krotiewski, M., Sjostrom, L., & Bjorntorp, P. (1977). Adipose tissue cellularity in relation to prognosis for weight reduction. International Journal of Obesity, 1, 395-416.

Lanyon, R. I. (1968). A handbook of MMPI profiles. Minneapolis: Lund Press, Inc.

Lew, E. A. & Garfinkel, L. (1979). Variations in mortality by weight among 750,000 men and women. Journal of Chronic Diseases, 32, 563-576.

MacAndrew, C. (1965). The differentiation of male alcoholic out-patients from nonalcoholic psychiatric patients by means of the MMPI. Quarterly Journal of Studies on Alcohol, 26, 238-246.

Marlatt, G. A. (1985). Cognitive factors in the relapse process. In G. A. Marlatt & J. R. Gordon (Eds.), Relapse prevention (pp. 128-200). New York: Guilford Press.

McKinley, J. C. & Hathaway, S. R. (1980). Scale 1 (Hypochondriasis). In W. G. Dahlstrom & L. Dahlstrom (Eds.), Basic readings on the MMPI. Minneapolis: University of Minnesota Press.

National Center for Health Statistics (NCHS). (1982). Obese and overweight adults in the United States (DHHS Publication No. 83-1680). Washington, DC: U. S. Government Printing Office.

Naval Military Personnel Command. (1986). Drug and alcohol treatment manual (For residential facilities) (NAVPERS Instruction 15561). Philadelphia: Naval Publications and Forms Center.

Nisbett, R. E. (1972). Hunger, obesity, and the ventromedial hypothalamus. Psychological Review, 79, 433-454.

Obrien, M. S. & Bankston, W. B. (1984). The moral career of the reformed compulsive eater: A study of conversion to charismatic conformity. Deviant Behavior, 5, 141-150.

Overfield, T. (1980, September). Obesity: Prevention is easier than cure. Nurse Practitioner Magazine, 48-50.

Pomerantz, A. S., Greenberg, I., & Blackburn, G. L. (1977). MMPI profiles of obese men and women. Psychological Reports, 41, 731-734.

Ruderman, A. J. (1985). Restraint and irrational cognitions. Behavior Research and Therapy, 23, 557-561.

Salans, L. B., Cushman, S. W., & Weissman, R. E. (1973). Adipose cell size and number in non-obese and obese patients. Journal of Clinical Investigations, 52, 929-941.

Schwartz, G. F. (1977). An investigation of the stability of single scale and two-point MMPI code types for psychiatric patients. Dissertation Abstracts International, 43.

Scott, R. L. & Barrofio, J. R. (1986). An MMPI analysis of similarities and differences in three classifications of eating disorders: Anorexia nervosa, bulimia, and morbid obesity. Journal of Clinical Psychology, 42, 708-713.

Sjostrom, L. (1980). Fat cells and body weight. In A. J. Stunkard (Eds.), Obesity. Philadelphia: Saunders.

Sternberg, B. (1985). Relapse in weight control: Definition, processes, and prevention strategies. In G. A. Marlatt & J. R. Gordon (Eds.), Relapse prevention (pp. 521-545). New York: Guilford Press.

Stevenson, R. (1984). Experimental statistics. Alburg, VT: Lionheart Press.

Stuart, R. B. & Mitchell, C. (1980). Self-help for self-management. In R. B. Stuart (Ed.), Behavioral self-management. New York: Brunner Mazel.

Stunkard, A. J. (1975). From explanation to action in psychosomatic medicine: The case of obesity. Psychosomatic Medicine, 37, 195-236.

Stunkard, A. J. (1980). Psychoanalysis and psychotherapy. In A. J. Stunkard (Ed.), Obesity. Philadelphia: Saunders.

Stunkard, A. J. & Mendelson, M. (1967). Obesity and body image: 1. Characteristics of disturbances in the body image of some obese persons. American Journal of Psychiatry, 36, 801-806.

Taylor, J. A. (1953). A personality scale of manifest anxiety. Journal of Abnormal and Social Psychology, 48, 285-290.

University of Minnesota. (1970). MMPI group form. Minneapolis: University of Minnesota Press.

Welsh, G. S. (1956). Factor dimensions A and R. In G. S. Welsh and W. D. Dahlstrom (Eds.), Basic readings on the MMPI in psychology and medicine. Minneapolis: University of Minnesota Press.

Wilcox, R. R. (1987). New statistical procedures for the social sciences. Hillsdale, NJ: Lawrence Erlbaum Associates.

Willcockson, N. K. (1986). A comparison of the effectiveness of 21 MMPI indices in the discrimination of brain-damaged from functional psychiatric and medical patient groups. Dissertation Abstracts International, 46(11-B), 4034.

Williams, J. (1981). MMPI group form scoring program. Odessa, FL: Psychological Resources, Inc.

Zucker, L. M. & Zucker, T. M. (1961). Fatty, a new mutation in the rat. Journal of Heredity, 52, 275-278.

APPENDIXES

APPENDIX A

DESCRIPTIONS OF MMPI SCALES

DESCRIPTIONS OF MMPI SCALES

Abbreviation	Common Name	Interpretations of Extremes High Score (H) - Low Score (L)
L	Lie	H-Unsophisticated attempt to present self in a favorable light. L-Willingness to admit to minor flaws.
F	Frequency? (Exact meaning F has been lost in history)	H-Tendency to respond in atypical or deviant manner/"Fake bad" L-Socially conforming/"Fake good"
K	Defensiveness	H-Highly conventional/Emotionally inhibited/ "Fake good" L-Critical of self/socially inept, blunt/ "Fake bad"
HS	Hypochondriasis	H-Excessive bodily concerns, somatic symptoms/Unhappiness L-Healthy/Optimistic/Lives effectively
D	Depression	H-Dysphoric/Pessimistic/Self-derogating/ Irritable L-Cheerful/Relaxed/Socially Adept
HY	Hysteria	H-Low insight/Self-centered/Exhibitionistic/ Somatizes stress L-Conventional/Suspicious/Unadventurous
PD	Psychopathic Deviate	H-Antisocial tendencies/Poor judgement/ Insensitive L-Conforming/Passive/Moralistic
MF	Masculinity-Femininity (Males)	H-Effeminate/Aesthetic interests/Androgynous L-Traditional sex role attitudes/Unemotional
PA	Paranoia	H-Hypersensitive/Hostile/Moralistic L-Trusting/Cheerful/Extroverted
PT	Psychasthenia	H-Anxious/Introspective/Self-critical L-Self-confident/Adventurous/Success oriented
SC	Schizophrenia	H-Unconventional/Confused/Aloof L-Friendly/Flexible/Conventional

DESCRIPTIONS OF MMPI SCALES (Continued)

Abbreviation	Common Name	Interpretations of Extremes High Score (H) - Low Score (L)
MA	Hypomania	H-Excessive activity/Grandiose/Easily bored L-Low energy/Apathetic/Humble
SI	Social Introversion	H-Retiring/Overcontrolled/Self-effacing L-Gregarious/Competitive/Impulsive
A	Anxiety	H-Uncomfortable/Pessimistic/Defensive L-Vigorous/Expressive/Competitive
R	Repression	H-Conventional/Internalizing/Cautious L-Excitable/Informal/Aggressive
ES	Ego Strength	H-Stable/Confident/Realistic L-Brooding/Rigid/Anxious
MAS	Manifest Anxiety	H-Excited/Restless/Somatic Complaints L-Controlled/Comfortable/Healthy
DO	Dominance	H-Poised/Frank/Realistic L-Pessimistic/Inefficient/Self-absorbed
CN	Control	H-Realistic/Flexible/Nontraditional L-Moralistic/Rigid/Lacks insight
MAC	MacAndrew's Alcoholism Scale	H-Impulsive/Socially nonconforming L-Conventional/Quiet lifestyle

APPENDIX B

WEIGHT HISTORY QUESTIONNAIRE

We realize that it may be difficult to give exact ages and weights in response to the questions below. Please estimate as accurately as possilbe.

1. How old were you when you first entered the military service?

2. Approximately how much did you weigh at that time?

3. Were you required to lose weight in order to be accepted for military service?

4. At what age did you first feel that you were overweight?

5. At what age did others (family, friends, teachers, etc.) first perceive you as being overweight?

6. How old were you the first time you were placed on or chose to go on a diet in order to lose weight?

7. Was this first decision to diet the result of any special need or situation (sports eligibility, medical problems, dating, etc.)? If yes, please explain briefly.

8. Does it seem, for you, that being overweight is a natural condition and that keeping your weight down is a life-long struggle? If yes, at what age did you first feel this way?

9. How many pounds would you need to lose in order to be at what you consider to be an acceptable weight? How old were you the last time you were at an acceptable weight?

10. Thinking back about the age periods listed below, were you overweight during all, most, some, or none of each period? Circle one for each period.

Birth to age 5--------	all	most	some	none
Age 6 to age 10-------	all	most	some	none
Age 11 to age 14------	all	most	some	none
Age 15 to age 18------	all	most	some	none
Age 18 to present-----	all	most	some	none

11. Have you dieted successfully in the past? If yes, at what ages, how much did you lose, and how long did you keep the weight off?

Age	Amount Lost	Kept off how long

12. Did you volunteer for this program? How do you feel about being here?

13. Have you ever been evaluated or treated for any alcohol related problems (medical, occupational or family).

14. Please estimate your average weekly consumption of alcoholic beverages during the past year.

APPENDIX C

WEIGHT CONTROL STUDY PARTICIPATION CONSENT FORM

WEIGHT CONTROL STUDY PARTICIPATION
CONSENT FORM

Your participation is requested in a clinical study being conducted (with command approval) by LT Adkins, Department Head of Alcohol Rehabilitation Service, U.S. Naval Hospital, Yokosuka, Japan. This research is designed to investigate the personality and emotional factors involved in weight control and alcohol problems. We need testing data from a large group of non-obese, non-alcoholic Navy personnel to compare with our patient data. Participation in the study is voluntary and will not effect your life or career in any way. Since only pooled, group data will be analyzed, the confidentiality of your responses will not be jeopardized. While you may not benefit directly from your participation, the findings of this research could result in future improvements in the understanding and treatment of weight control and alcohol problems. If you agree to participate, you will be asked to complete two paper and pencil questionaires now, and again, in approximately six weeks.

I have read the above paragraph and volunteer to participate. I understand that I may cease participation at any time, with no repercussions.

Name (Printed):_____

Signature: _____

Date: _____

When this study is completed, interested participants will receive a summary of the results. If you would like to have a copy, please complete the block below. Because the results may not be available for up to one year, this must be a permanent address.

Rank/Name: _____

Street/Box No: _____

City/State: _____

Zip Code: _____

APPENDIX D

TABLES

TABLE I

ANALYSIS OF VARIANCE SUMMARY TABLE
FOR EFFECTS OF GROUPS (G) AND
LOCATION (L) ON SUBJECT AGE

Source	SS	df	MS	F	p
G	97.539	3	32 513	.39	NS
L	84.116	2	42.058	.76	NS
GL	2317.05	6	386.175	2.33	p=.0334
Error	37788.797	228	165.74		

TABLE II

GROUP BY LOCATION MEANS FOR
PRETREATMENT R SCALE

Group	Location	Mean
OBE	JAX	51.95
	SD	56.4
	YOKO	47.7
OBL	JAX	49.35
	SD	48.15
	YOKO	47.8
ETOH	JAX	44.3
	SD	51.0
	YOKO	52.1
Control	JAX	50.15
	SD	44.55
	YOKO	46.6

TABLE III

ANALYSIS OF VARIANCE SUMMARY TABLE FOR EFFECTS OF TREATMENT GROUPS (G) AND LOCATION (L) ON PRETREATMENT TEST SCORES

Source	SS	df	MS	F	p
Scale: RRS					
G	5524.212	3	1841.404	51.76	p <.001
L	78.758	2	39.379	1.11	NS
GL	155.175	6	25.863	.73	NS
Error	8111.65	228	35.577		
Scale: L					
G	1002.279	3	334.093	6.53	p <.001
L	7.058	2	3.529	.07	NS
GL	466.408	6	77.734	1.48	NS
Error	11993.55	228	52.603		
Scale: F					
G	225.15	3	75.05	.74	NS
L	172.9	2	86.45	.85	NS
GL	1679.5	6	279.917	2.75	NS
Error	23170.3	228	101.624		
Scale: K					
G	679.846	3	226.615	3.43	NS
L	126.4	2	63.2	.96	NS
GL	181.066	6	30.178	.46	NS
Error	15064.35	228	66.072		
Scale: HS					
G	1833.946	3	611.315	7.14	p <.001
L	431.385	2	215.679	2.52	NS
GL	512.541	6	85.424	1.00	NS
Error	19515.15	228	85.593		
Scale: D					
G	8979.616	3	2993.205	21.52	p <.001
L	4.275	2	2.138	.02	NS
GL	632.659	6	105.443	.76	NS
Error	31713.3	228	139.093		
Scale: HY					
G	3566.646	3	1188.882	20.66	p <.001
L	135.658	2	67.829	1.18	NS
GL	366.342	6	61.057	1.06	NS
Error	13119.25	228	57.541		

TABLE III (Continued)

Source	SS	df	MS	F	p
Scale: PD					
G	6368.313	3	2122.771	20.32	p < .001
L	507.775	2	253.888	2.43	NS
GL	185.625	6	30.937	.30	NS
Error	23813.45	228	104.445		
Scale: MF					
G	4480.413	3	1493.471	13.04	p < .001
L	252.3	2	126.15	1.10	NS
GL	1239.9	6	206.65	1.80	NS
Error	26122.55	228	114.573		
Scale: PA					
G	3325.283	3	1108.428	12.59	p < .001
L	284.158	2	142.079	1.61	NS
GL	503.642	6	83.940	.95	NS
Error	20072.5	228	88.037		
Scale: PT					
G	4227.579	3	1409.193	11.38	p < .001
L	160.558	2	80.279	.65	NS
GL	318.008	6	53.001	.43	NS
Error	28222.75	228	123.784		
Scale: SC					
G	3021.35	3	1007.117	7.71	p < .001
L	45.758	2	22.879	.18	NS
GL	563.975	6	93.996	.72	NS
Error	29778.1	228	130.606		
Scale: MA					
G	1531.412	3	510.471	5.90	p=.001
L	203.308	2	101.654	1.17	NS
GL	637.325	6	106.221	1.23	NS
Error	19734.95	228	86.557		
Scale: SI					
G	3831.7	3	1277.233	13.91	p < .001
L	302.633	2	151.317	1.65	NS
GL	606.4	6	101.067	1.10	NS
Error	20935.6	228	91.823		
Scale: A					
G	1274.746	3	424.915	3.70	NS
L	99.975	2	49.988	.43	NS
GL	710.981	6	118.482	1.03	NS
Error	26200.35	228	114.914		

TABLE III (Continued)

Source	SS	df	MS	F	p
Scale: R					
G	775.946	3	258.689	3.59	NS
L	93.558	2	46.779	.65	NS
GL	1723.942	6	287.324	3.99	p=.001
Error	16436.55	228	72.09		
Scale: ES					
G	1024.333	3	341.444	3.13	NS
L	179.108	2	89.554	.82	NS
GL	879.592	6	146.598	1.35	NS
Error	24833.7	228	108.919		
Scale: MAS					
G	2920.316	3	973.439	8.08	p < .001
L	263.508	2	131.754	1.09	NS
GL	1278.858	6	213.143	1.77	NS
Error	27477.3	228	120.515		
Scale: DO					
G	1183.146	3	394.382	4.94	p=.002
L	88.958	2	44.479	.56	NS
GL	397.342	6	66.224	.83	NS
Error	18204.55	228	79.845		
Scale: CN					
G	961.683	3	320.561	3.43	NS
L	43.658	2	21.829	.23	NS
GL	445.241	6	74.207	.79	NS
Error	21287.4	228	93.366		
Scale: MAC					
G	7062.15	3	2354.05	23.36	p < .001
L	334.558	2	167.279	1.66	NS
GL	698.775	6	116.462	1.16	NS
Error	22890.805	228	100.398		

TABLE IV

GROUP MEANS AND CONTRAST RESULTS FOR PRETREATMENT SCORES SHOWING DIFFERENCES ON ANOVA

Group	Mean	Contrast	p
Measure: RRS			
OBE.....	21.3	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	$p < .01$
OBL.....	20.1	OBL vs. CONTROL.....	$p < .01$
		OBL vs. ETOH.....	$p < .01$
ETOH.....	9.43	OBL vs. CONTROL.....	$p < .01$
		ETOH vs. CONTROL.....	$p < .01$
CONTROL.....	13.95		
Measure: L			
OBE.....	47.5	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	48.08	OBL vs. CONTROL.....	$p < .01$
		OBL vs. ETOH.....	NS
ETOH.....	49.88	OBL vs. CONTROL.....	$p < .01$
		ETOH vs. CONTROL.....	NS
CONTROL.....	52.75		
Measure: HS			
OBE.....	60.52	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	57.43	OBL vs. CONTROL.....	$p < .01$
		OBL vs. ETOH.....	NS
ETOH.....	57.3	OBL vs. CONTROL.....	$p < .05$
		ETOH vs. CONTROL.....	$p < .05$
CONTROL.....	52.77		
Measure: D			
OBE.....	69.33	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	66.07	OBL vs. CONTROL.....	$p < .01$
		OBL vs. ETOH.....	NS
ETOH.....	64.42	OBL vs. CONTROL.....	$p < .01$
		ETOH vs. CONTROL.....	$p < .01$
CONTROL.....	53.08		

TABLE IV (Continued)

Group	Mean	Contrast	p
Measure: HY			
OBE.....	60.72	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	58.57	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	59.7	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	50.93		
Measure: PD			
OBE.....	68.23	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	66.13	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	70.08	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	56.7		
Measure: MF			
OBE.....	63.88	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	64.18	OBL vs. CONTROL.....	p < .05
		OBL vs. ETOH.....	NS
ETOH.....	59.58	OBL vs. CONTROL.....	p < .05
		ETOH vs. CONTROL.....	p < .05
CONTROL.....	53.53		
Measure: PA			
OBE.....	62.17	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	60.06	OBL vs. CONTROL.....	p < .05
		OBL vs. ETOH.....	NS
ETOH.....	61.48	OBL vs. CONTROL.....	p < .05
		ETOH vs. CONTROL.....	p < .05
CONTROL.....	52.92		

TABLE IV (Continued)

Group	Mean	Contrast	p
Measure: PT			
OBE.....	65.67	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	63.35	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	62.97	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	54.6		
Measure: SC			
OBE.....	67.03	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	63.18	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	65.03	OBL vs. CONTROL.....	p < .05
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	57.52		
Measure: MA			
OBE.....	60.77	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	p < .05
OBL.....	62.53	OBL vs. CONTROL.....	NS
		OBL vs. ETOH.....	NS
ETOH.....	65.78	OBL vs. CONTROL.....	NS
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	58.93		
Measure: SI			
OBE.....	60.9	OBE vs. OBL.....	p < .05
		OBE vs. ETOH.....	p < .01
OBL.....	56.35	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	52.83	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	NS
CONTROL.....	50.25		
Measure: MAS			
OBE.....	57.75	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	p < .01
OBL.....	55.42	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	54.55	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	p < .05
CONTROL.....	48.32		

TABLE IV (Continued)

Group	Mean	Contrast	p
Measure: DO			
OBE.....	50.33	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	53.97	OBL vs. CONTROL.....	NS
		OBL vs. ETOH.....	$p < .01$
ETOH.....	48.18	OBL vs. CONTROL.....	NS
		ETOH vs. CONTROL.....	NS
CONTROL.....	52.7		
Measure: MAC			
OBE.....	52.5	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	$p < .01$
OBL.....	56.3	OBL vs. CONTROL.....	NS
		OBL vs. ETOH.....	$p < .01$
ETOH.....	64.05	OBL vs. CONTROL.....	$p < .01$
		ETOH vs. CONTROL.....	$p < .01$
CONTROL.....	49.58		

TABLE V

ANALYSIS OF COVARIANCE OF POSTTREATMENT TEST SCORES BY GROUP WITH PRETREATMENT SCORE AS COVARIATE

Source	SS	df	MS	F	p
Scale: RRS					
REGR.	7998.756	1	7998.756	500.37	p < .001
GROUPS	158.596	3	52.865	3.307	p < .05
ERROR	3756.632	235			
Scale: L					
REGR.	6502.592	1	6502.592	230.107	p < .001
GROUPS	78.573	3	26.191	.926	NS
ERROR	6640.835	235			
Scale: F					
REGR.	10964.62	1	10964.62	269.413	p < .001
GROUPS	232.898	3	77.633	1.907	NS
ERROR	9564.041	235			
Scale: K					
REGR.	5318.846	1	5318.846	136.319	p < .001
GROUPS	332.064	3	110.688	2.836	p < .05
ERROR	9169.091	235			
Scale: HS					
REGR.	4065.74	1	4065.74	62.378	p < .001
GROUPS	69.066	3	23.022	.353	NS
ERROR	15316.94	235			
Scale: D					
REGR.	11258.0	1	11258.0	243.243	p < .001
GROUPS	41.57	3	13.857	.299	NS
ERROR	10876.49	235			
Scale: HY					
REGR.	5983.775	1	5983.775	152.545	p < .001
GROUPS	316.105	3	105.368	2.686	p < .05
ERROR	9218.12	235			
Scale: PD					
REGR.	12453.89	1	12453.89	258.981	p < .001
GROUPS	759.376	3	253.125	5.263	p < .01
ERROR	11300.67	235			
Scale: MF					
REGR.	18018.51	1	18018.51	479.13	p < .001
GROUPS	1301.045	3	433.682	11.532	p < .001
ERROR	8837.572	235			

TABLE V (Continued)

Source	SS	df	MS	F	p
Scale: PA					
REGR.	8393.774	1	8393.774	192.315	p < .001
GROUPS	86.658	3	28.886	.661	NS
ERROR	10256.75	235			
Scale: PT					
REGR.	6195.359	1	6195.359	111.377	p < .001
GROUPS	193.748	3	64.583	1.161	NS
ERROR	13071.89	235			
Scale: SC					
REGR.	10267.65	1	10267.65	160.215	p < .001
GROUPS	57.366	3	19.122	.298	NS
ERROR	15060.36	235			
Scale: MA					
REGR.	8535.736	1	8535.736	194.816	p < .001
GROUPS	949.723	3	316.574	7.225	p < .01
ERROR	10296.35	235			
Scale: SI					
REGR.	11691.59	1	11691.59	318.193	p < .001
GROUPS	60.599	3	20.199	.549	NS
ERROR	8634.747	235			
Scale: A					
REGR.	10064.18	1	10064.18	238.842	p < .001
GROUPS	95.345	3	31.782	.754	NS
ERROR	9902.283	235			
Scale: R					
REGR.	7492.067	1	7492.067	185.938	p < .001
GROUPS	357.355	3	119.118	2.956	p < .05
ERROR	9468.891	235			
Scale: ES					
REGR.	8879.269	1	8879.269	206.062	p < .001
GROUPS	338.114	3	112.705	2.615	NS
ERROR	10126.18	235			
Scale: MAS					
REGR.	13542.13	1	13542.13	335.216	p < .001
GROUPS	16.296	3	5.432	.134	NS
ERROR	9493.571	235			

TABLE V (Continued)

Source	SS	df	MS	F	p
Scale: DO					
REGR.	9443.372	1	9443.372	283.858	$p < .001$
GROUPS	87.049	3	29.016	.872	NS
ERROR	7817.954	235			
Scale: CN					
REGR.	8035.346	1	8035.346	166.202	$p < .001$
GROUPS	301.203	3	100.401	2.076	NS
ERROR	11361.45	235			
Scale: MAC					
REGR.	17000.32	1	17000.32	423.826	$p < .001$
GROUPS	589.474	3	196.491	4.898	$p < .01$
ERROR	9426.208	235			

TABLE VI

GROUP MEANS (ADJUSTED FOR PRETREATMENT SCORES) AND CONTRAST RESULTS FOR POSTTREATMENT SCORES SHOWING DIFFERENCES ON ANCOVA

Group	Mean	Contrast	p
Measure: MF			
OBE.....	62.07	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	61.18	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	61.26	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	55.84	OBE+OBL vs. ETOH.....	NS
		OBE+OBL vs. CONTROL.....	p < .01
		OBE+OBL+ETOH vs. CONTROL...	p < .01
Measure: MA			
OBE.....	63.38	OBE vs. OBL.....	NS
		OBE vs. ETOH.....	NS
OBL.....	63.94	OBL vs. CONTROL.....	p < .01
		OBL vs. ETOH.....	NS
ETOH.....	63.37	OBL vs. CONTROL.....	p < .01
		ETOH vs. CONTROL.....	p < .01
CONTROL.....	58.92	OBE+OBL vs. ETOH.....	NS
		OBE+OBL vs. CONTROL.....	p < .01
		OBE+OBL+ETOH vs. CONTROL...	p < .01

TABLE VII

ANALYSIS OF COVARIANCE FOR EFFECTS OF OBE AND OBL GROUPS ON POSTTREATMENT WEIGHT WITH PRETREATMENT WEIGHT AS COVARIATE

Source	SS	df	MS	F	p
Regression	62179.31	1	62179.31	1412.427	p<.001
Groups	49.49609	1	49.496	1.124	NS
Error	5150.691	117			

TABLE VIII

ANALYSIS OF VARIANCE ON PRETREATMENT AND POSTTREATMENT WEIGHT FOR OBE GROUP

Source	SS	df	MS	F	p
Pre-Post	10267.5	1	10267.5	14.23	p .0003
Error	85168.2	118	721.764		

TABLE IX

ANALYSIS OF VARIANCE ON PRETREATMENT AND POSTTREATMENT WEIGHT FOR OBL GROUP

Source	SS	df	MS	F	p
Per-Post	11603.33	1	11603.33	21.41	p .0001
Error	63942.63	118	541.89		

VITA

DAVID SCOTT ADKINS

Candidate for the Degree of

Doctor of Philosophy

Thesis: PERSONALITY FACTORS IN OBESITY AND RESPONSE TO NAVY RESIDENTIAL TREATMENT PROGRAMS

Major Field: Psychology

Biographical:

Personal Data: Born in Ponca City, Oklahoma, September 15, 1951, the son of Walter and Mary Jane Adkins; married in Coronado, California, December 18, 1976 to Marcia Anne Jones; son, Brandon Jacob, born on June 13, 1985 in Kitakyushu, Japan; daughter, Kathryn Elizabeth, born on June 13, 1987 in Iwakuni, Japan.

Education: Graduated from Ponca City High School in May, 1969; attended Northern Oklahoma College and Oklahoma State University until entering the U.S. Navy in December, 1972; served in Pacific Fleet until reentering Oklahoma State University in January, 1977; received Bachelor of Science degree from Oklahoma State University in May, 1978; enrolled in clinical psychology program at Oklahoma State University in June, 1978; completed requirements for the Master of Science Degree at Oklahoma State University in July, 1981; Internship in Clinical Psychology at National Naval Medical Center, Bethesda, Maryland from October, 1981 to October, 1982; completed requirements for the Doctor of Philosophy Degree at Oklahoma State University in July, 1988.

Professional Experience: Research assistant in biofeedback 1978-79; Psychology trainee at Central Oklahoma Juvenile Treatment Center, 1979; Psychological Associate at The Psychological Services Center, 1979-81; Practicum therapist at OSU branch of Bi-State Mental Health Clinic, 1980-81; Psychological examiner at Carl Albert Community Mental Health Center, 1981; teaching assistant for Psychology of Human Problems

and Individual Mental Testing at Oklahoma State University, 1979-81; Staff Psychologist at Naval Alcohol Rehabilitation Center, Jacksonville, Florida from October, 1982 to September, 1984; Staff Psychologist and Head of Alcohol Rehabilitation/ Substance Abuse Department, U.S. Naval Hospital, Yokosuka, Japan from November, 1984 to October, 1988.