

AN EVALUATION OF THE OCCURRENCE AND FLUCTUATION OF DEPRESSION IN OBESE PATIENTS IN TWO TYPES OF WEIGHT REDUCTION PROGRAMMES

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ABSTRACT

This study investigated the occurrence and fluctuation of depression in obese patients in two types of weight-reduction programmes at VAALMED, Vanderbijlpark.

It was hypothesized that there would be a difference between the results yielded by the different depression inventories used; that depression would occur over time; that the level of depression which occurred, would differ in the different treatment programmes; that there would be a fluctuation in the level of depression which occurred over time and that there would be a difference in the depression which occurred and the outcome to treatment (weight-loss) in the different treatment programmes.

In order to evaluate the hypotheses, patients who were referred to the obesity clinic at VAALMED, Vanderbijlpark, by their general practitioners were medically evaluated for contra-indications for the treatment of obesity. The patients were then randomly assigned from stratified blocks (based on percentage overweight and sex) to one of the two treatment programmes, which comprised a) both a standard behaviour therapy and a very low-calorie diet and b) a cognitive behaviour therapy, plus a very low-calorie diet. All patients attended weekly treatment sessions of 90 minutes which were conducted by two clinical psychologists and a medical practitioner. Pre-therapy measurements of depression were conducted on both experimental groups on a bi-weekly basis during the three months programme. Follow-up measurements were conducted for the next four months.

The measuring instruments used, were self rating scales of depression i.e. the Beck Depression Inventory and the Carroll Rating Scale in order to monitor the occurrence of depression throughout the treatment programmes. A biographical questionnaire composed by the clinical

psychologists and the general practitioner was used. The purpose of this questionnaire, was to obtain specific information not covered by the other measuring instruments. The observed data were analysed by applying the Statistical Analysis System (SAS) computer programme. Any results reported as significant had a probability value of $p \leq 0,1$.

No significant differences between the groups emerged in respect of the level of depression which occurred in the different treatment programmes or in the depression which occurred during treatment and the outcome of treatment (weight-loss) in the different treatment programmes.

However, significant differences were found between the results yielded by the different depression inventories. It was also evident that depression occurred throughout the different treatment programmes and that there was a fluctuation in the level of depression which occurred over time.

The implications of findings in this study for the planning and design of a weight-reduction programme are: measuring instruments must be carefully screened before use; pre- and post measurement of depression are inadequate to monitor for any adverse effects of the treatment programme; a behaviour therapy plus a very low-calorie diet programme does not cause adverse effects; programmes must cater for the occurrence and fluctuation of depression throughout the entire period of the programme.

Problems experienced in this study are discussed and recommendations made for further research.

OPSOMMING

DIE VOORKOMS EN FLUKTUASIE VAN DEPRESSIE BY VETSUGPASIËNTE IN TWEË TÍPES GEWIGSVERLIESPROGRAMME

Die primêre oogmerk van die studie was om die voorkoms en fluktuering van depressie by vetsugpatiënte, gedurende twee tipes gewigsverliesprogramme, te VAALMED Vanderbijlpark, te ondersoek.

Die hipotese is gestel dat:

- daar 'n verskil sal wees tussen die resultate verkry uit die verskillende depressievraelyste wat gebruik is
- depressie tydens die behandelingsprogram sal voorkom by vetsugpatiënte
- die vlak van depressie wat voorkom, sal verskil na gelang van die besondere behandelingsprogram
- die vlak van depressie sal fluktueer
- die vlak van depressie en resultaat van behandeling (gewigsverlies) na gelang van die besondere behandelingsprogram sal verskil

Ten einde die hipoteses te evalueer, is die volgende metode van ondersoek gevolg. Pasiënte wat deur 'n algemene praktisyn na die Vetsugklinik te VAALMED, Vanderbijlpark verwys is, is medies ondersoek vir newe-effekte met betrekking tot die behandeling van vetsug. Die pasiënte is vervolgens na willekeur vanuit gestratifiseerde groeperings, gebaseer op persentasie wat hul oorgewig was en geslag, toegewys aan een van twee behandelingsprogramme, wat

onderskeidelik die volgende behels het: a) standaard gedragsterapie en 'n laekaloriedieet en b) kognitiewe gedragsterapie en 'n laekaloriedieet. Al die pasiënte het aan 'n weeklikse behandelingsessie van negentig minute deelgeneem, onder leiding van twee kliniese sielkundiges en 'n mediese dokter. Die voorkoms van depressie by albei eksperimentele groepe is op 'n tweeweeklikse basis gemeet, voor die aanvang van die behandelingsessie, oor 'n tydperk van drie maande vir die duur van die behandeling. Opvolgevaluering is gedurende die volgende vier maande uitgevoer.

Die Beck depressie- en die Carroll selfbeoordelingskale is gebruik om die voorkoms en fluktuasie van die depressievlak tydens die behandelingstydperk te monitor. 'n Biografiese vraelys, deur die kliniese sielkundiges en die algemene praktisyn saamgestel, is ook gebruik. Die doel met die vraelys was om spesifieke inligting te bekom wat nie in die ander vraelyste vervat is nie.

'n Analise van die inligting verkry uit die vraelyste is gemaak deur middel van die "Statistical Analysis System" of SAS-rekenaarprogram. Enige beduidende resultate wat verkry is, het 'n 10% waarskynlikheidswaarde gehad.

Geen beduidende verskille is verkry tussen:

- die vlak van depressie by die verskillende eksperimentele groepe nie.
- en
- die vlak van depressie en die resultaat van behandeling (gewigsverlies) in die verskillende behandelingsprogramme nie.

Die onderskeie depressiebeoordelingskale het nogtans resultate gelewer wat beduidend van mekaar verskil het. Depressie het deurlopend tydens die behandelingsprogramme voorgekom, en die vlak daarvan het gedurende die ondersoektydperk gefluktueer.

Die studie het die volgende implikasies met betrekking tot die beplanning en ontwerp van gewigsverlies programme aan die lig gebring:

- meetinstrumente moet versigtig en oordeelkundig gekeur word.
- om depressie voor en na die behandelingsprogram te meet is ontoereikend vir die bepaling van die negatiewe uitwerking wat so 'n program mag hê.
- geen negatiewe uitwerking word ondervind wanneer gedragsterapie in 'n program gekombineer word met 'n laekalorie dieet nie.
- programme moet deurlopend voorsiening maak vir die voorkoms en fluktuering van depressie.

Probleme wat met die studie ondervind is en aanbevelings vir verdere navorsing is vervolgens bespreek.

CHAPTER 1 - INTRODUCTION

1.1 Statement of the Problem

There is a strong indication that depression is an important factor to be considered in the treatment of obesity (Stunkard, 1987; Wadden, 1984; Weighill & Buglass, 1984; Wing, Epstein, Marcus, & Kupfer, 1984).

Controversy exists in the literature with regards to the role depression plays in obesity (Stunkard, 1987; Wadden & Stunkard, 1986; Wing et al., 1983, 1984). The problem appears to be that no clear differentiation is made between:

- a) depression as an etiological factor in obesity, and
- b) depression which first occurs during treatment of obesity.

With regards to the former category it has already been clearly proved that depression is etiologically related to obesity (Stunkard, 1987; Wadden & Stunkard, 1986; Wing et al., 1984).

On reviewing the role of depression during treatment for obesity, Craighead, Stunkard, and O'Brien (1981) reported that patients on reducing diets, frequently had increased incidence of depression and other emotional disturbances. Having included behaviour therapy in their treatment programme, these researchers found that there was a noteworthy decrease in the intensity of depression and an increase in positive cognitions. In a study conducted by Wing et al., (1983) it was found that changes in mood during the behaviour therapy treatment of obesity were strongly related to initial mood, i.e. the presence of depression before commencement of treatment. Wing et al., (1983) concluded that change in mood was clearly related to initial mood, as patients with the most negative initial mood, showed the greatest improvement, whereas those with the most positive initial mood, showed the least improvement.

In contrast to the above findings, the findings of Weighill and Buglass (1984) revealed that, at the end of two months of dieting, patients were significantly more depressed, although the depression usually remitted when they were about to come off the diet. They concluded that the diet treatment for obesity contributed to the occurrence of depression. These findings were in line with the findings of Stunkard and Rush (1974) who reviewed literature on emotional responses to weight reduction and concluded that treatment can and does result in adverse psychological (depression and anxiety) and metabolic reactions in obese patients. Weighill and Buglass (1984) concluded that there is a statistically significant correlation between change in mood and change in weight. Loss of weight accompanied an improved mood state but when weight increased, so did that patient's distress. In studies of outpatients, it was found that weight reduction was beneficial to the patients' affective state. Therefore, it is theoretically possible that either depressed patients lose weight as their mood improves and they feel able to restrain their appetite, or alternatively, that mood is reactive to weight change and that overweight people become happier as their weight goes down (Weighill & Buglass, 1984).

Many existing studies have found that a strict medical programme consisting of only a very low-calorie diet, could contribute to the occurrence of depression and that the coping skills taught in behavioural programmes, appear to help subjects deal with the untoward emotions that so frequently afflict patients using both very low-calorie diets and conventional reducing diets (Stunkard & Rush, 1974; Wadden & Stunkard, 1986; Wing et al., 1984). Other studies indicate that depression also develops during psychotherapeutic and combined treatment programmes (Stunkard & Rush, 1974; Wadden & Stunkard, 1986; Wing et al., 1984).

The general conclusion which can be derived from the above studies, is that depression which was already present prior to commencement of treatment, eases with weight-loss during a behaviour therapy weight reduction programme (Weighill &

Buglass, 1984; Wing et al., 1984).

The study of the role of depression during the treatment of obesity is, however, complicated by the following variables:

Firstly, the various studies in which different treatment programmes were used can not be directly compared because of differences with regards to the definition of obesity, dissimilar measuring instruments used, differences in the duration of the programmes, etc. (Wadden & Stunkard, 1986). It is, therefore, necessary to directly compare the occurrence of depression in different treatment programmes (Stunkard, 1987).

Secondly, Wadden, Stunkard, and Smoller (1986), as well as Stunkard and Rush (1974), also found that the level of depression fluctuated during the treatment of obesity and that pre- and post-measurements alone are not adequate to monitor this fluctuation of depression. Accurate, specific information with regards to this fluctuation of depression during the different stages of a treatment programme, has important implications for the planning of the treatment programme (Wadden, 1984; Wing et al., 1984).

Thirdly, the instrument used to measure depression can influence the results obtained; Wing et al., (1984) recommend the use of different measures of mood, each with its appropriate psychometric properties, since some instruments may be more sensitive to changes in mood induced by dieting. Wadden et al., (1986) support this view and state that the results of their study show that the method of mood assessment determines the answer to the question of whether dieting is associated with adverse psychological consequences. When assessment by objective inventories was limited to before-and-after treatment, significant decreases were observed in depression and anxiety. The results of weekly assessment using the same inventories, painted a different picture, since more than half of the patients experienced increases in depression of 25% or more, while half of the patients also experienced comparable increases in anxiety. The results of an open-ended interview, suggest

that dieters may experience numerous physical and psychological symptoms not assessed by questionnaires such as the Beck Depression Inventory (Wadden et al., 1986).

In conclusion, it can be stated that further research is needed on the following:

- a direct comparison of the occurrence of depression in different treatment programmes for obesity.
- monitoring to ascertain whether the level of depression fluctuated during the treatment of obesity
- the use of bi-weekly measurements during the treatment programmes (instead of simply pre- and post-treatment measurements of depression) to monitor the fluctuation of depression at different stages during the obesity treatment programmes
- the use of different measuring instruments to measure depression, in order to compare their sensitivity to changes in mood induced by dieting.

1.2 Purpose of this Study

General Aim

The general aim of the present study is to assess the occurrence and fluctuation of depression in obese patients in two different weight-reduction treatment programmes, i.e. a standard behaviour therapy programme, plus a very low-calorie diet and a cognitive behaviour therapy programme, plus a very low-calorie diet.

Research Aims

The following research aims are specified, in order to:

- assess whether the different depression inventories used, yielded different results
- to assess whether depression occurred over time in the patients participating in the different treatment programmes
- to compare the level of depression which occurred over time in the patients participating in the different treatment programmes

- to compare whether there was a fluctuation in the level of depression which occurred over time in the treatment programmes
- to assess whether there was a difference in the depression which occurred during treatment and the outcome of treatment (weight-loss) in the different treatment programmes.

In order to achieve these specific aims, the following procedure was followed:

The subjects were randomly assigned from stratified blocks (based on percentage overweight and sex) to one of the two treatment programmes.

Stunkard's (1987) classification of obesity was used, i.e.:

- mild obesity (20 - 40% overweight)
- moderate obesity (40 - 100% overweight)
- severe obesity (>100% overweight)

This study focused only on the Mild and Moderate divisions of obesity, as the available subjects all fell into these categories. Stunkard (1987) stated that severe obesity should receive surgical treatment.

The duration of the treatment programmes was three months. The first month was a period of preparation, in which all the subjects in the experimental groups participated, after which they were allocated to one of the two treatment groups, namely the standard behaviour therapy programme, plus a very low-calorie diet (Group 0) or the cognitive behaviour therapy programme, plus a very low-calorie diet (Group 1).

Group 0 were treated by using a standard behaviour therapy programme plus a very low-calorie diet (as prescribed by a dietitian), weekly weigh-in sessions, calorie education, medical tests (blood pressure, urine and blood analysis), plus a programme which consisted of self-monitoring, stimulus control, the acquiring of new eating behaviour and a new reinforcement schedule, as well as cognitive

restructuring, centered on the negative cognitions about the treatment itself.

Group 1 were treated by using a cognitive behaviour therapy programme, plus a very low-calorie diet (as prescribed by a dietitian), weekly weigh-in sessions, calorie education and medical tests (blood pressure, urine and blood analysis). The therapy programme contained all the components of the programme of Group 0, as well as a more comprehensive cognitive therapy component which focused upon the different aspects in the acquiring of improved self-control.

Treatment was undertaken by a medical doctor and two clinical psychologists. These three professionals were present at all the sessions of both the standard behaviour therapy Group 0 and the cognitive behaviour therapy Group 1.

Patients who were referred to the obesity clinic by their doctors during the period 15 March to 13 April 1988, were included in the control group. The patients in the control group did not receive any therapy or dietary treatment for their obesity. The control group were assessed for the occurrence and fluctuation of depression by using the same measuring instruments as for Groups 0 and 1; this was done on the same bi-weekly dates during the treatment programmes, in order to control for variables which could have influenced the depression scores of the patients.

Pre-therapy measurements of depression were obtained bi-weekly for both Group 0 and Group 1, before weighing. Wing et al., (1983) state that all measurements should be applied prior to weighing the subjects, in order to control for the effect of weight-loss or increase on the level of depression reported.

A three-month follow-up programme, after termination of the initial treatment programmes, included measurement of depression by using the same measuring instruments as in the initial programme. Gormally and Rardin (1981), as well as Graham, Taylor, Hovell, and Siegel (1983) stress the importance of a follow-up. Gormally and Rardin (1981)

emphasize that the mechanisms needed to maintain weight-loss, differ markedly from those needed to lose weight.

Subjects completed questionnaires designed to monitor the occurrence of depression. This battery of measuring instruments provided the required data relating to the occurrence and fluctuation of depression, to which this study addressed itself. Pre-treatment, pre-therapy and post-treatment measurements of depression were conducted and compared.

Information regarding age, sex, educational level, family background, marital status, medical history and history of obesity was obtained by using a biographical questionnaire.

1.3 Definitions and Usage

The purpose of this section is to list, for convenient reference, the manner in which terms and conventions have been used in this study. No special merit is claimed for these definitions over alternative definitions: they are chosen for clarity and convenience for the purpose of the argument presented in this study. Many of the definitions differ according to the theoretical framework of the various authors and they are presented here to highlight the complexity of the disorders being studied.

Adipose Tissue

The white fat which represents 15 to 30 % of total body weight (Stock & Rothwell, 1982).

Body Fat

The quantity of triglyceride and other fats which the body contains (Bray, 1976).

Depression

Depression is a feeling of despair, accompanied by pessimistic, melancholic and self-depreciating thoughts,

accompanied by a slowness of speech, movement and all activity (Stuart, 1978). The onset of depression is sometimes a consequence of past failures (e.g. unsuccessful attempts to lose weight), which make the prospects of future successes seem all but non-existent. It is an affect or feeling tone and is a ubiquitous and universal human condition which extends on a continuum ranging from normal mood swings, to a pathological state. Thus, depression as a concept, could be used to describe a full spectrum of emotional states of varying degrees of intensity, to a set of identifiable clinical disorders, which involve distinct patterns of symptomatology.

Major depression is classed as a mood disorder in which the essential feature is either a depressed mood, or a loss of interest or pleasure in all or in almost all activities and associated symptoms for a period of at least two weeks (American Psychiatric Association, [APA], 1987).

In this study, depression does not refer to clinical depression as diagnosed according to the APA (1987) in the DSM-III-R, but rather indicates mood changes in mild to moderate obese patients, excluding morbid obese patients. This depression is measured by using the Beck Depression Inventory.

Eating Behaviour

The eating behaviour as described by psychologists in respect of subjects who are bored, apprehensive or depressed, seems to be unrelated to hunger, appetite or satiety sensations.

Obese patients may report that they have overeaten, but at the same time deny that they had been hungry (Garrow, 1978). The possibility that excessive eating behaviour is a defence mechanism utilized by obese patients in an attempt to handle emotional and environmental demands or pressures is proposed by numerous researchers (Simon, 1963; Stunkard & Rush, 1974).

Energy Balance

This term refers to a positive energy balance as a contributing factor in the development of obesity. Garrow (1978), states that if the energy intake of a subject equalled his energy output (or expenditure) then he would be in a negative energy balance, and energy stores would remain constant. However, if the energy intake exceeded output, he would be in a positive balance and this contributed to obesity. In the latter case, the treatment approach would be to get the energy output to exceed the energy input, thus creating a negative balance which would result in weight-loss (Bray, 1987).

Free Fatty Acid (FFA)

The Free Fatty Acid concentration in the blood, (denoting circulating fat to be transported for energy purposes) is constantly elevated in most obese subjects (Gordon, 1969).

Hyperplastic Obesity

Hyperplastic obesity is associated with over-eating during early life, may have a genetic component and is probably of life-long duration. Because of its early onset, it may be referred to as a juvenile-onset type obesity. Prognosis for weight reduction is often poor (Curtis-Prior, 1983)

Hypertrophic Obesity

Hypertrophic obesity is marked by an increase in fat cell size, unaccompanied by changes in fat cell number. This is usually a mature-onset type of obesity and invariably leads to a variety of metabolic and other complications. It is the more prevalent form of human obesity (Curtis-Prior, 1983).

Metabolism and Basal Metabolism

Metabolism is a term which refers to all the chemical changes which body tissue undergoes (Bray, 1976). Basal

metabolism is defined as the sum total of the minimal activity of all tissues of the body under steady-state condition. Careful studies of basal energy expenditure have indicated that it is influenced by a number of factors including body weight, age, sex, climate, hormones and drugs (Bray, 1976). Basal Metabolic Rating (BMR) is the minimum amount of energy the body needs to maintain its functioning, during a state of inactivity. This energy, used by the body as fuel, can be measured in kilojoules.

Obesity

In 1978 Bray had stated that obesity exists when fat makes up a greater than normal fraction of the total body weight. In males aged 18, approximately 15 - 18 per cent of body weight is made up of fat, while for females the percentage is 20 - 25. Bray (1987) states that obesity is an excess of body fat, and results from a positive energy balance; this occurs when more energy has been ingested than used by the body, and this extra energy is stored as fat. Bray (1987) views obesity is a symptom of a disease, not a disease in itself. It represents the consequence of progressive storage of calories in fatty tissue. All obesity develops because of an imbalance in caloric intake, since without the ingestion of more calories than are needed, obesity will not develop.

According to the First Law of Thermodynamics, it is concluded that for adipose stores of a human being to increase, more calories than are required to meet metabolic demands must be consumed (Curtis-Prior, 1983). This provides a common definition of obesity as being a situation in which the body contains an excess of calories as storage triglyceride in adipose tissue. It therefore follows, that obesity occurs if the fat percentage in the adipose tissue is larger than the normal relationship with regards to the total body mass. In women it is 30% in excess and in men 25% (Oberholzer, 1984).

According to Plug, Meyer, Louw, and Gouws (1987), obesity is the accumulation of excessive body fat. In the present

study, obesity refers to the presence of an abnormally high proportion of body fat, whereas overweight refers to a body weight which is above an arbitrary standard. These terms are often interchangeable. Obesity is associated with many disorders which increase morbidity and mortality. Its prevalence is increasing in developed countries. Since the majority of obese people eat no more than their normal weight peers obese people as a group are deficient in their utilization of energy (Stunkard, 1980).

In psychiatry, obesity is a psychogenic condition characterized by an excessive drive to eat (bulimia) and excessive weight (at least 20% above age and sex norms, taking individual constitution into consideration) (Goldenson, 1984). Psychological factors frequently associated with compulsive overeating, are persistent emotional tension, use of food as a substitute satisfaction in cases of sexual frustration or disappointment, an unbearable life situation, parental overemphasis on food, or the use of food as a reward, emotional deprivation during the oral phase of psychosexual development, and a need to escape the risks and anxiety of maintaining social relationships (Goldenson, 1984).

In this study obesity refers to Stunkard's (1987) classification of mild obesity (20 - 40% overweight) and moderate obesity (40 - 100% overweight).

Obesity is a condition characterized by an excessive build-up of fat in the body, and according to Oberholzer (1984), approximately a third of the population in South Africa are overweight.

Some of the most commonly used definitions of obesity follow:

- Obesity occurs if the fat percentage in the adipose tissue is higher than the normal relationship to the total percentage body mass. In women it is 30% in excess and in men 25% (Oberholzer, 1984).
- Obesity is a condition characterized by excessive accumulation of fat in the body. By convention, obesity

is said to be present when body weight exceeds by 20% the standard weight listed in the Metropolitan Life Insurance (1959) ideal weight norms (Gormally, 1977).

- Obesity refers to the presence of an abnormally high proportion of body fat, whereas overweight refers to a body weight which is above an arbitrary standard (Stunkard, 1984).
- The psychiatric definition of obesity is a psychogenic condition characterized by an excessive drive to eat (bulimia) and excessive weight (at least 20% above age and sex norms, taking individual constitution into consideration) (Goldenson, 1984).

Most experienced clinicians claim that the diagnosis of obesity is best understood, not by the use of tables of ideal weight ranges, but rather by subjective consideration of each individual (Stock & Rothwell, 1982).

In the present study obesity refers to the accumulation of excessive fat in the body contributing to a body weight which falls within Stunkard's (1987) division of 20 to 40% overweight (Mild obesity) or 40 to 100% overweight (Moderate obesity).

Overweight

Overweight, as distinct from obesity, is defined in relation to tables of desirable weight which is usually prepared from insurance company information and denotes a body weight which is higher than the standard weight (Bray, 1976). Obese people are usually overweight, but not that all overweight people are obese.

Patients

The patients in this study were white adult, male and female members of Vaalmed in Vanderbijlpark. The patients were referred to the Obesity Clinic by their doctors. These patients took part in the treatment programmes on a voluntary basis and patients diagnosed as suffering from - or being treated for - clinical depression, were excluded

from this study. Patients referred by their doctors during the period September 1987 to 15 March 1988, were included in the two experimental groups. Patients referred from 15 March 1988 to 13 April 1988, made up the control group and were included in the next treatment programme scheduled to begin in August 1988.

Satiety

According to Garrow (1978) this term is impossible to define with any precision, since it is a sensation intermediate between the relief of hunger and the nausea which ultimately limits overeating.

1.4 Summary

This study concentrated on specific aspects of depression, i.e. its occurrence and fluctuation in obese patients in two weight-reduction programmes. Attention was also paid to monitoring the depression of obese patients who formed the control group, to determine whether there were differences in their scores. The focus of the literature study is on studies related to humans and does not include studies undertaken on laboratory animals.

It is important to note that this study has a bi-dimensional approach (medical and psychological), and that the treatment programmes made use of a very low-calorie diet, coupled to a standard behaviour therapy programme for Group 0 and a very low-calorie diet, coupled to a cognitive behaviour therapy programme for Group 1.

CHAPTER 2 - LITERATURE STUDY

2.1 Orientation

This chapter consists of three sections. The first section contains a discussion of obesity and the second a discussion of depression, while the third section covers the association between the treatment of obesity and depression. Although the focus of this study is on depression which occurs during the treatment of obesity, a holistic approach is followed in the review of available literature on obesity. Hence both psychological and physiological aspects are discussed.

2.2 OBESITY

In this section of the chapter obesity is discussed by referring to recent literature. Attention will be paid to theories concerning obesity, etiological factors and the treatment of obesity. It must be emphasized that this study does not support any specific theoretical model of obesity.

2.3 THEORIES ON OBESITY

Historical overview

For many years overweight has been viewed as being an exclusive medical problem (Van Strien, 1986). However, psychoanalysts reached the conclusion that the overwhelming majority of obesity cases, simply resulted from overeating and not from any inorganic disorder of metabolism. The result, was that the focus of interest moved from biochemical regulatory mechanisms to the psychological factors contributing to an increase in the amount of food eaten. However, perceptions of the somatic determinants of obesity were not completely discarded. Psychoanalysts with medical training first made use of psychoanalysis in the treatment of obesity. The result was that a number of viewpoints were developed under the heading of psychosomatic theory. The increased interest in carefully conducted research resulted in an attempt to empirically test the

psychosomatic theory; however, the initial results were negative. In an attempt to explain the observed results, the externality theory was formulated, which was later replaced by the theory of restrained eating (Van Strien, 1986).

In order to emphasize the multifactorial etiology of obesity physiological theories of obesity (Set Point Theory, Boundary Model, Glucostatic Theory and ATPase Theory) will also receive attention.

2.3.1 Psychological Theories

The Psychoanalytic Theory

Traditionally psychoanalysts have stressed the obese person's fixation on, or regression to, the oral stage of development as an explanation of obesity. According to this view, the predominant form of obesity (that of juvenile onset) is developmental obesity a condition associated with severe emotional and personality disturbances resembling pre-schizophrenic development (Leon & Roth, 1977). Conclusions reached from studies of the parents of 250 obese youngsters conducted by Bruch and Touraine in 1940, were that the mothers felt a fundamental rejection towards their children and compensated for this by overprotective measures and excessive feeding (Leon & Roth, 1977).

According to Van Strien (1986) classical psychoanalysts such as Bruch in 1974, Freud in 1917 and Orbach in 1979 had explained emotional eating in terms of fixation at the oral stage. The explanation of this is, that food intake is the principal source of pleasure during the first year of life, and therefore, the mouth functions as the predominant erogenous zone. It is here that the child learns to associate food consumption with maternal care, as the mother is the central love object of the child, and is the source of many instinctual gratifications. If a traumatic event occurs at this stage, the child may become fixated at the oral stage and continue to recognize food as a symbol of maternal care. The possibility exists, that when the

individual experienced emotional stress, he would most likely to turn to food in an attempt to recapture the security and comfort experienced in infancy (Van Strien, 1986). According to the psychoanalytic approach obesity is viewed as a symptom of an underlying state of depression. This approach views overeating as a defence mechanism derived from the unconscious effects of helplessness and hopelessness arising through object loss situations (Bruch, 1974; Leon & Roth, 1977). The psychoanalytic view supports the association of obesity with depression, anxiety, or phobic states and obesity as being a symptom of a more complex emotional disorder (Bruch, 1974; Leon & Roth, 1977).

When the above is considered, it could be that obese persons could be classified on a scale characterized by increasing degrees of emotional instability. The suggestion is, that patients range from those who are emotionally stable to those who eat as a defence against emotional tensions, to patients whose eating disturbances are a central concern in their lives. In contrast to numerous other studies, which have failed to find a consistent personality constellation or pattern of underlying pathology in obese persons (Leon & Roth, 1977; Mendelson, 1982), this classification scheme covers the wide variety of people who are obese.

Although psychoanalytic theories agree that overeating results from internal emotional arousal, psychoanalytic theories disagree on the significance of overeating for the particular individual (Van Strien, 1986). The results of studies of emotional reactions have indicated that obese individuals experience depression or anxiety more often and with greater intensity than is the case with normal-weight individuals. Support for this theory was found in clinical settings using interviews and questionnaires as it was found that the majority of obese individuals overeat when emotionally aroused (Van Strien, 1986). When experimental tests to examine the effects of arousal manipulations on eating behaviour of obese and non-obese subjects were used no or relatively weak indications were found that obese individuals overeat in response to emotional arousal. The results of an attempt by Slochower (1983) to explain the

discrepancy between clinical and experimental observations, was the introduction of the concepts of diffuse - as opposed to clearly labeled - emotions, and controllability - as opposed to the uncontrollability - of emotional states. It was found that overweight subjects overeat only in response to emotional states which are experienced as being diffuse and out of control. The findings indicated that obese individuals overeat in response to certain types of stressors and that the overeating has an anxiety-reducing function (Van Strien, 1986).

Summary

There is agreement amongst psychoanalytic theorists with regards to the fact that unconscious conflicts are central to the emotional distress that leads to overeating. Disagreement exists about the significance of overeating for the individual. Nevertheless, overeating is regarded as a response to internal emotional arousal (Van Strien, 1986). The psychoanalytic theory of obesity stresses the obese person's fixation at - or regression to - the oral stage of development: it views obesity as being a symptom of an underlying state of depression and regards overeating as a defence mechanism (Leon & Roth, 1977).

The Psychosomatic Theory

In the fields of psychology, psychiatry and psychosomatic medicine, the belief exists that psychological factors exert an important influence with regards to the development and maintenance of obesity (McReynolds, 1982). Obesity is, therefore, regarded as being a psychosomatic disorder.

McReynolds (1982), studied the reports on the mental state of obese patients referred to psychiatrists and other helping professionals in mental health settings, and concluded that psychological factors played a role in contributing to obesity. However, McReynolds (1982) warns against generalization. He concludes that possible cause-effect relationships could be indicated in the kinds of disturbances in the reports relating to case studies of

obese psychiatric patients. It would, therefore, appear that many obese persons seen by mental health professionals in the course of their clinical practice, suffer from a personality or neurotic disturbance. The possibility exists that parental relationships and issues of nurturance, growth and dependence are involved. McReynolds (1982) reached the conclusion that the evidence indicates that obesity sometimes appears in conjunction with psychological disturbance (neurotic or psychosomatic), as well as in the absence of psychological disturbance.

The psychosomatic theory also states that overeating is caused by emotional arousal and stress (Rodin, 1980; Van Strien, 1986). This "emotional eating" is found in individuals who eat when anxious or depressed i.e. in response to emotional states and not in response to internal cues of hunger. Accordingly, the psychosomatic theory assumes that overeating is caused by the confusion of physiological symptoms of hunger and satiety, accompanied by emotional stress (Van Strien, 1986).

According to McReynolds (1982) and Rodin (1980) obesity is viewed as the somatic expression of a specific pattern of personality traits: passive dependence, emotional frustration, a strong desire to be loved, and poor coping abilities or the physiological expression of emotional turmoil, namely anxiety and depression.

Summary

The psychosomatic theory places emphasis on internal emotional factors (Rodin, 1980) and states that the eating behaviour of overweight individuals is comparatively unresponsive to internal physiological signals, such as gastric motility. Rodin (1980) concludes that the psychosomatic theory of obesity attributes overeating as a reaction to the effects of emotional distress i.e. an attempt to cope with anxiety, fear or emotional disturbance.

The Externality Theory

Finding no support for the psychosomatic theory (Polivy, Herman, & Warsh, 1978), Schachter in 1968 formulated the externality theory. Schachter suggests that the eating behaviour of the obese may be triggered by external food-relevant cues, such as the smell, sight and availability of food. In agreement with the psychosomatic theory, the externality theory also suggests that the eating behaviour of overweight individuals is relatively unresponsive to internal physiological signals. The externality theory focuses on the external food environment as stimuli for overeating. The obese individuals are said to be hyperresponsive to external food-related cues (Polivy et al., 1978). This theory was extended to cover behaviours not related to eating (cognitive behaviours) i.e. the obese were considered to exhibit an external cognitive style (Polivy, et al., 1978). Rodin (1980) reports that the idea that an overweight person is tempted beyond his self-restraint by potent cues in the environment - bakery smells, ice cream trucks etc. - have an intuitive appeal and strong face validity. The result was that the externality theory generated substantial research. A few of the relevant studies will be discussed.

On reviewing the origins of Schachter's externality theory, Rodin (1980) commented on the failure to find that overweight people are any more responsive to external cues than are persons of normal-weight. She provided persuasive evidence that most people are responsive to external food cues. However, externality is a valid psychological characteristic found not only in obese individuals. Rodin (1980), failed to confirm the hypothesis that obese people are less responsive than non-obese people to internal, psychological cues and concluded that externality could cause overeating and subsequent weight-gain, even though the relationship of the externality theory to obesity is not well-established. She proposed the possibility that early detection and control of responsiveness to environmental food cues, could contribute towards the prevention of obesity.

According to Leon and Roth (1977), the most controversial aspect of Schachter's theory, is his discounting of the psychosomatic hypothesis of obesity, as eating in response to emotional arousal, has been considered an important factor in obesity and reported by investigators such as Leckie and Withers (1967). Leon and Roth (1977) concluded that the proposition that obese persons are generally external in orientation, irrespective of the particular stimulus or modality, and are especially sensitive to potent external cues, receives mixed support from the data at hand.

Results of a study conducted by Bray (1976) indicated that obese patients who were identified as being more responsive to external stimuli and therefore, more susceptible to external cues in their home or social environments, had more difficulty in a weight-reduction programme (indicated by smaller weight-losses).

However, the externality theory has been challenged by investigators who contend that the internal/external distinction is possibly too simple to explain the differences in the eating behaviour of obese and normal-weight individuals as they found individuals in every weight category who were responsive to external stimuli or unresponsive to internal stimuli (Van Strien, 1986).

Summary

The externality theory attributes overeating to a hypersensitivity to external food cues - the outcome of an external cognitive style (Van Strien, 1986). Schachter's research indicated that as the obese individuals' eating behaviour is largely controlled by external, environmental cues unrelated to the physiological state of hunger, there appears to be almost no relationship between their internal state and their eating behaviour (Leon & Roth, 1977). Rodin Bray, Atkinson, Dahms, Greenway, Hamilton, and Molitch (1977a) concluded that externality is a response to a widely generalized set of environmental stimulus conditions, which could lead to overeating and weight-gain.

However, the results obtained by researchers failed to prove conclusively that externality was a psychological characteristic found only in obese individuals. As a result a theory which posited that physiological variables could possibly influence the final level of an individual's body weight, called the "restrained eating theory", replaced the externality theory.

The Restrained Eating Theory

In 1975 Herman, a student of Schachter, developed the restrained eating theory in an attempt to explain the differences (Schachter: 1971) between the eating patterns of obese individuals and those of normal-weight people (Rodin, 1980). This theory proposed that the regulation of food depends not only on external responsiveness, but also on the interaction of physiological, sensory, cognitive-motivational and socio-cultural variables (Van Strien, 1986).

The theory of restrained eating attributes overeating to dieting (Herman & Polivy, 1980). Dieting attempts to lower the body weight of the individual which, according to this theory, is homeostatically regulated. The conscious restriction of food intake may result in persistent hunger, which causes stress. Anxiety, depression, the intake of alcohol or eating energy dense food could undermine the individual's self-control and could result in excessive food intake or counter-regulation (Herman & Polivy, 1980). The resultant stress, could cause an increase in emotionality. The assumption is made, that the majority of obese individuals are chronic dieters and that normal-weight individuals are not. According to Herman and Polivy (1980) and Hibscher and Herman (1977), this fact explains obese/normal differences in eating behaviour.

Herman and Polivy (1980) developed a restrained eating questionnaire to determine whether people were trying to limit the amount they ate. Their reasoning was, that the person would be aware of the fact that he maintained a body weight below set-point by means of dieting. The

questionnaire enabled them to select people whose behaviour would change when experimentally manipulated. To test their theory, Herman and Polivy (1980) used an experiment which involved pre-loads of food. The subjects were given pre-loads of either two glasses of milkshake, one glass, or no pre-load. The amount the subjects ate, was measured. The results confirmed their expectations, as the unrestrained eaters, who managed their food intake without any particular effort ate progressively less with the increase in pre-load. On the other hand the restrained eaters ate more, demonstrating what Herman and Polivy (1980) called counter-regulation. This indicated that normally restrained eaters experience a state of chronic caloric deprivation when they suspend their self-imposed restraint. They then give up hope of staying within their self-imposed caloric limits and experience a motivational collapse. The result is, that they give in to the demands of hunger which they have been suppressing.

Herman and Polivy (1980) also researched the effect of anxiety and depression as possible disinhibitors of self-control processes on the eating behaviour of restrained and unrestrained eaters. Their findings showed that the restrained eaters eat more when anxious and depressed, while unrestrained eaters eat less. They concluded that it was the obese individuals' restraint rather than their obesity, that leads to counter-regulation. The final conclusion Herman and Polivy (1980) reached, was that dieting and not obesity per se, is the critical correlate - and the primary cause of obesity. The explanation they offer is that dieting is a stressor, and therefore, a source of frustration. This stress drains the individual's resources, which enables him to cope with emotional demands. Herman and Polivy (1980) suggest that this chronic state of stress restrained eaters find themselves in, has implications for their behavioural, emotional and medical well-being.

Ruderman (1985) states that even though the relationship between restraint and loss of control over eating is becoming more clear, the relationship between restraint and obesity is not yet fully understood. According to Ruderman

(1985), Herman's 1975 hypothesis that the degree of overweight was likely to be correlated with restraint and that differences in restraint might underlie differences in the eating patterns of obese and normal-weight individuals, is not corroborated by recent evidence. However, findings suggest that restrained normal-weight eaters are likely to become disinhibited when faced with diet-disrupting events (an initial dietary violation or a stressful experience). This disinhibition phenomenon, which characterizes restrained eaters, bears a strong similarity to binge-eating.

Summary

The theory of restrained eating views overeating as one of the side-effects of dieting. Herman and Polivy (1980) state that the obese are chronic dieters and that they demonstrate counter-regulation. In other words, when restrained eaters suspend their self-imposed restraint, they are faced with a chronic state of caloric deprivation. The result is, that they give up hope of staying within their self-imposed caloric limits and suffer from a motivational collapse. They then give in to the demands of hunger that they have continually been suppressing. Herman and Polivy (1980) conclude that dieting is a stressor, a source of frustration and a drain on the person's resources to cope with emotional demands. They suggest that restrained eaters are chronically in a state of stress, which could have implications for their behavioural, emotional and medical well-being. The implication, is that dieting places stress on the restrained eater. The resulting strong emotions interfere with self-control and produce binge-eating and weight-gain (Herman & Polivy, 1980).

2.3.2 Physiological Theories

The theories which follow, propose a more physiological than psychological explanation of obesity and are included to provide a holistic view.

The Set Point Theory

In 1972 Nisbett proposed another explanation of obese/normal differences in eating behaviour. He suggested that the heightened external sensitivity of the obese was caused by a state of relative physiological deprivation, rather than by externality as a personality trait. According to Van Strien (1986), Nisbett posited that some individuals are physiologically programmed to be obese because of an excess number of fat cells related to genetic factors, or as a result of overfeeding during pre-adult development. This formed the basis of the "Set Point Theory". Nisbett maintained that to feed these cells, these individuals are physiologically predisposed by their hypothalamic feeding center to overeat. At the same time, however, social and cultural constraints discourage the individual from fully satisfying his biological requirements. The result could be an overweight person who is physiologically far below the set-point at which the fat cells are adequately nourished. This compromise between biological and social demands, results in an individual who is both overweight and chronically hungry.

The implication of Nisbett's Set Point Theory that body weight is homeostatically regulated, is that a person whose weight is below set-point, should show obese eating behaviour patterns, regardless of whether or not this person is of normal-weight. The result of Nisbett's belief that an individual was able to use a diet to lower his body weight below set-point, was the introduction of a new element to the complex range of factors which govern eating behaviour that of self-control (a mental process). This idea of self-control was expanded by Herman and co-workers in 1980 when they made cognitive restraint (i.e. the cognitive resolve not to eat) the focal point of their theory of restrained eating.

The set-point theory is, therefore, based on the belief that there is a set-point around which body weight is regulated. This regulation is not just to maintain a constant body weight, but also to defend this body weight, when challenged

e.g. as in the case of dieting. According to Keesey (1980), regulation is not just the result of changes in caloric intake, but also because of changes in energy expenditure. He adds that weight-loss triggers the dual pressure of increased food intake and decreased caloric expenditure, while weight-gain triggers the opposite pressures of decreased food intake and increased caloric expenditure.

Various researchers (Keesey, 1980; Keesey & Corbell, 1984; Stunkard & Penick, 1979), obtained evidence that individuals vigorously and effectively defend a particular body weight! Experiments in which the weight of the subject's had been displaced from the subjects normal-weight (by 25 per cent through dietary restriction) were conducted. These individuals' body weight rapidly returned to normal when the individuals were again allowed to eat ad libitum. The implication is, that when individuals are permitted to eat normally, their weights quickly return to their former level.

The above indicates that an individual has the ability to maintain his body weight at a relatively constant level under widely differing external and internal conditions. It suggests an affirmative answer to the question of whether body weight could be viewed as being regulated by a set-point controller. However, according to Brownell (1982), it must be kept in mind that the set-points for body weight, unlike those of most other physiological factors, vary widely among members of the same species. Central to a better understanding of the numerous problems involved in human overweight and obesity, are the answers to the question of which mechanisms are involved in determining the body weight set-point, and an appreciation of the physiological conditions associated with regulation of body weight at different levels (Brownell, 1982).

In 1987 Sims and Danforth evaluated Daniel's 1984 set-point hypothesis, which assumes that similar to temperature, the body tries to maintain its body weight at a constant level. Sims and Danforth (1987) encouraged human volunteers to increase their body weight by 20 to 30 percent. Results of

their study indicated that many of the volunteers did not gain weight despite a very high calorie intake and that those who did, quickly returned to their original weight when allowed to follow their normal eating patterns. However, Daniels (1984) reported that the opposite is also true, as obese individuals who lose weight when following a calorie restricted diet, quickly regain it as soon as they return to their normal eating patterns. The prediction made by Daniels (1984), is that there is no weight-loss programme that is effective in achieving permanent weight-loss and that the reason for this, is to be found in the set-point hypothesis.

However, there is a serious flaw in this theory: in that it cannot be tested directly, since it would entail a knowledge of the body weight set-point of individual people (Keeseey, 1980).

Summary

The most important corollary of the set-point theory is that the organism will defend its body weight against pressure to change (Brownell, 1982). Daniels (1984) compares the set-point to a thermostat which regulates the temperature around an ideal. In support of the set-point theory, Keeseey (1980), reports numerous studies which show a strong defense of the body set-point. It appears that the body effects metabolic adaptations to mediate its defense of this set-point. It would, therefore, seem that some obese persons may be fighting a battle against a biology that never relents. Keeseey (1980) concludes, that if the set-point theory applies to humans, dieting may be far more difficult than most people believe.

The Boundary Model

The Boundary Model was proposed by Herman and Polivy in 1980 in their attempt to explain the counter-regulatory eating behaviour of dieters (Herman, Polivy, Lank, & Heatherton, 1987). According to this model, the consumption of food is regulated by two boundaries corresponding to hunger and

satiety. The belief is, that consumption is regulated by boundaries (rather than a point), which give rise to a zone of indifference, this being where food intake is not constrained by physiological factors of hunger and satiety, but where non-physiological agents such as cognitive, social and other psychological factors, are in force. According to Herman et al., (1987) it is assumed that dieters have a lower hunger boundary and a higher satiety boundary than non-dieters; this is based on the observation that on normal occasions they eat less than non-dieters, yet on other occasions eat considerably more. The assumption is also, that dieters have a diet boundary - entirely cognitive in character - which is said to fall well short of the satiety boundary. The difference is, that in non-dieters the satiety boundary provides the upper boundary for food intake; while in dieters, the diet boundary provides the upper boundary for the regulation of food intake, but only if it has not been breached. However, if the diet boundary has been surpassed, i.e., when the dieter thinks that it has been transgressed, it no longer has a regulatory function, and the satiety boundary becomes the boundary of reference. On evidence of this model, it is assumed that the counter-regulatory eating behaviour of dieters following a dense energy pre-load, is not unregulated; or - it is regulated on a different basis by the satiety boundary, rather than the diet boundary. Herman and Polivy (1980) note that in this respect, dieters differ from patients with eating disorders such as anorexia and bulimia, since these individuals regularly transgress the hunger or satiety boundary.

In sum, it appears that the boundary model of eating proposes that normal eating proceeds in order to keep the person within a zone defined by two boundaries corresponding to hunger and satiety. A person may be in a state of hunger, satiety or indifference (neither hungry nor sated). When hungry (deprived), the normal-weight person will, if possible, increase food intake in order to escape the hunger zone and achieve indifference. The experience of satiety likewise operates through negative reinforcement to terminate intake and allow the person to subside into a

state of comfortable indifference. When indifferent, the person is not subjected to the aversive physiological pressures that occur in the hunger and satiety zones (Herman et al., 1987).

With respect to dieters (obese patients), the boundary model proposes that their behaviour is largely under the control of a third and somewhat unnatural boundary, i.e. the diet boundary which is located within the zone of indifference. Dieters attempt to restrict their intake in order not to exceed this boundary, which is dictated by diet calculations. Once the diet boundary has been breached, however, eating may proceed in a disinhibited way, often in defiance of normal considerations of hunger and satiety. In respect to the obese, the boundary model proposes that they be considered as dieters (Herman et al., 1987).

Results of a study by Herman et al., (1987) provide substantial support for the hypothesis derived from Herman and Polivy's (1980) boundary model of eating: this hypothesis suggests that food consumption will vary as an interactive function of dieting status, level of anxiety and degree of repletion.

Summary

The boundary model assumes that the consumption of food is regulated by two boundaries corresponding to hunger and satiety. Once the upper boundary in dieters (i.e. the hunger boundary which regulates food intake and which is lower than that of non-dieters) has been breached, it loses its regulatory function, and the satiety boundary (which is higher than that of non-dieters) becomes the boundary of reference, resulting in increased food intake.

The Glucostatic Theory

In 1953 Mayer proposed that appetite was determined by the level of blood glucose or carbohydrate reserves in the body. This glucostatic hypothesis was later modified, to suggest that it is the rate of entry of glucose into cells and its

oxidation which influence the appetite. Thus, even though patients with diabetes mellitus (because of a lack of insulin) have very high blood glucose concentrations, glucose cannot enter the cells. This results in the patients' experiencing voracious appetites and food intake is, therefore, elevated. However, according to Stock and Rothwell (1982), it seems unlikely that the level of carbohydrate reserves or the rate of glucose utilization, are the primary factors involved in the long-term control of food intake, because of the large meal-to-meal and day-to-day fluctuations in blood glucose and glycogen stores. Nevertheless, glucostatic control could be very important in determining the amount of food consumed.

The Glucostatic Theory was reviewed by Stunkard (1976) and he suggested it to be a mechanism which controls food intake. The argument and thinking behind this theory, had its origin in the discovery that the brain has areas which control hunger and satiety. According to Stunkard (1976), the brain is unique among body organs as it is totally dependent on body sugar for its nourishment which it obtains from an individual's consumed carbohydrates.

On considering the question of what goes wrong in obesity, Stunkard (1976) suggested that there are two possible answers within this theory:

- (a) The glucostatic mechanism itself might break down, the result being that the presence of available sugar would be inadequately signaled to the brain. The obese person would continue to experience hunger and consequently eat, despite the availability of adequate amounts of blood sugar.
- (b) The glucostatic mechanism might be intact but for some reason the obese person might not have enough blood sugar available. Possible causes of this, could be diabetes and hypoglycemia, two disorders both of which are associated with obesity.

Stunkard (1976) conducted various experiments in an attempt to determine whether the glucostatic mechanism worked for both obese and non-obese people. The results of these experiments clearly indicated that the obese responded to the sugar injections in exactly the same way as the non-obese did. Analysis of data revealed that the sugar tolerance tests of obese patients did not vary more than those of non-obese patients over a period of time. The variations were, in fact, so small, that they could hardly have played a part in altering the food intake of the obese patients.

Stunkard (1976) reached the conclusion, that the glucostatic theory could still account for many aspects of the regulation of body weight, although it could not account for all situations; he also states, that there must be other messages to the brain upon which the decision to stop eating, is based. Just as the glucostatic theory cannot completely explain the feeling of satiety at the end of a meal, it also encounters problems concerning the long-range regulation of food intake.

Summary

According to Stock and Rothwell (1982), the glucostatic theory proposes that appetite is determined by the level of blood glucose or carbohydrate reserves in the body, which are important in determining short-term eating patterns. Support for this theory emerged from the discovery that the brain has areas which control hunger and satiety and that the brain depends upon body sugar for its nourishment (Stunkard, 1976). The level of blood glucose is seen as the biochemical trigger of hunger, and if the glucostatic mechanism breaks down or malfunctions, the result is increased food intake, which encourages obesity.

The Adenosine Triphosphatase (ATPase) Enzyme Theory

This theory of obesity proposes that a deficiency of an enzyme, adenosine triphosphatase (ATPase), may predispose certain people to weight-gain by lowering their resting

energy expenditure by as much as 25 percent (Haskew & Adams, 1984). It is stated that obese people typically have 20 to 25 percent less ATPase than people of normal-weight. The assumption is, that the more obese the person is, the lower the patient's ATPase concentration is likely to be. The result of these lower levels of ATPase, would be to alter caloric efficiency in favour of obese people, who burn fewer calories than normal-weight people when they perform the same degree of activity. An important metabolic process known as the sodium pump, is seen to be the reason for this. Haskew and Adams (1984) state that it has been shown that obese people have lower pressure differentials across cell membranes than do normal-weight people. The sodium pump, which maintains different concentrations of sodium and potassium ions across certain cell membranes, is adversely affected by the lower pressure, with the result that the sodium pump consumes less energy in obese people, who therefore, survive on fewer calories.

Summary

According to Haskew and Adams (1984) a deficiency of the ATPase enzyme may predispose certain people to weight-gain by lowering their resting energy expenditure by as much as 25%. This implies that obese people, who have 20 to 25 percent less ATPase than do normal-weight people and burn fewer calories than normal-weight people when they perform the same amount of activity; this contributes to their weight problem.

2.3.3 A review of the theories

Psychological theories involving overeating and weight-gain are comprised of the following:

- (a) The psychoanalytic theory. This theory stresses the obese person's fixation at, or regression to, the oral stage of development as an explanation of obesity. It views obesity as a symptom of an underlying state of depression, where overeating functions as a defence mechanism and a response to internal emotional arousal (Leon & Roth, 1977; Van Strien, 1986).

- (b) The psychosomatic theory focuses on internal emotional factors: it attributes overeating to a confusion due to the incapability to distinguish between the internal arousal states accompanying hunger and satiety, and the physiological symptoms of hunger and satiety (Rodin, 1980), accompanied by emotional stress (Van Strien, 1986).
- (c) The externality theory. This theory focuses on external food cues, and attributes overeating to a hyperresponsiveness to food-related cues in the environment, combined with an unresponsiveness to internal cues of hunger and satiety (Rodin, 1980). This behaviour is seen to be the outcome of an external cognitive style (Van Strien, 1986).
- (d) The theory of restrained eating. This theory attributes overeating to the conscious restriction of food intake, i.e. dieting, which causes counter-regulation (Herman & Polivy, 1980). Overeating is viewed as one of the side-effects of dieting, which places stress on the restrained eater.

Physiological theories involving overeating and weight-gain are comprised of the following:

- (a) The set-point theory supports the view that there is a set-point around which body weight is regulated; this theory involves not only the maintenance of a constant body weight, but also the defence of that body weight when the latter is challenged - person attempts to lose weight) (Keeseey, 1980). The most important aspect of this theory, is that the organism will defend its body weight against pressure to change and implies that dieting may be far more difficult than most people believe (Brownell, 1982).

- (b) The boundary model focuses on the regulation of food consumption between two boundaries, i.e. hunger and satiety, and contends that when the hunger boundary is breached by the obese person, the satiety boundary, which is higher, becomes the regulator of food consumption and results in increased food intake (Herman & Polivy, 1980).

- (c) The glucostatic theory proposes that appetite is determined by the level of blood glucose or carbohydrate reserves in the body and includes the role of the brain in the controlling of hunger and satiety, as well as the brain's dependence on body sugar for nourishment (Stock & Rothwell, 1982; Stunkard, 1976). The level of blood glucose is seen as the biochemical trigger of hunger and it is assumed that if the glucostatic mechanism broke down, the result could be increased food intake, which encourages obesity.

- (d) The ATPase theory asserts that obese people have 20 to 25% less ATPase enzyme than do normal-weight people and that it is this deficiency which results in the phenomenon that obese people burn fewer calories than normal-weight people during the performance of the same degree of activity, which in turn, contributes to their weight problem (Haskew & Adams, 1984).

2.3.4 Summary

Evidence strongly suggests that there are very few personality characteristics shared by obese persons, which could be considered causative in the development of obesity (Leon & Roth, 1977). Neither orality or externality nor depression universally characterize the obese individual. While some obese persons have been shown to be sensitive to external and environmental cues, they also demonstrate a greater degree of emotionality. Evidence obtained, strongly suggests that obesity is not a unitary syndrome and that there is an interaction between psychological and physiological factors.

2.4 ETIOLOGY AND DEVELOPMENT OF OBESITY

The incidence of obesity has been studied by various disciplines, including those of psychology and medicine. Each of these disciplines focus on different areas of numerous etiological factors. In the present study attention will be paid to psychological and physiological factors which contribute to the development of obesity, in an attempt by the researcher to provide a holistic view.

A wide spectrum of causes of obesity has been proposed: for example, a provoking disturbance in caloric intake and subsequent development of obesity, including insult to the supposed hypothalamic satiety center; emotional disturbance; socially established eating habits; reduced exercise; endocrine disorders; defective enzyme regulation; reduced activity of the sodium pump; aberrant production of polyunsaturated fatty acid metabolites. The existence of these various theories suggests, that obesity has a multifactorial origin.

Psychological factors frequently associated with compulsive overeating are: a stressful life situation; emotional deprivation during the oral phase of psychosexual development; parental preoccupation with food, or the use of food as a reward; persistent emotional tension; use of food as a substitute satisfaction in cases of sexual disappointment or frustration (Goldenson, 1984). The need to escape the risks and anxiety of social relationships, could also give rise to a pattern of overeating, as obese individuals are not expected to be socially active.

The following possible etiological factors are reviewed:

2.4.1 Psychological Factors

In this section attention is paid to psychological factors which play a role in the etiology and development of obesity.

Stunkard (1980), states that psychological factors in the

development of obesity are widely recognized, but attempts to define a specific personality type in association with obesity, have proven difficult. Characteristics like the need for autonomy and wariness of entangling relationships, defiance, stubbornness and conflicts in respect of exhibitionism, are prominent features in the personality structures of obese patients and contribute to the traditional reputation of the obese as being difficult patients. Obese girls show considerably more anxiety and some degree of immaturity and depression than do obese boys. According to Stunkard (1980), the most consistent and important differentiation between juvenile and adult onset types of obesity, may lie in the perception of body image, with distortion almost entirely limited to individuals whose obesity began in childhood or adolescence.

Stunkard's (1980) findings that a compulsive eating pattern occurs in 5% of obese individuals were supported by Bronkhorst (1984). This compulsive eating is characterized by a sudden compulsive intake of large quantities of food in a very short period which leads to guilt feelings and self-rejection. These researchers found that this eating pattern is coupled to stress and certain life situations.

Numerous authors, Bray (1978, 1987), Bronkhorst (1984) and Leon and Roth (1977) have reported that overeating could be seen as a form of compensation for a lack of affection and that this is often a causal factor in many individuals' obesity. It would appear that these individuals fall into three groups: (a) children who receive too little love from their parents; (b) single individuals or married couples without children and (c) individuals who experience a lot of tension, unhappiness and too little love. As reported under the psychosomatic theory, overeating could be a defence mechanism evolved to cope with unpleasant feelings such as anxiety and depression, as well as a means to avoid undesirable persons or situations - for example, sexual interactions.

Because of the natural human tendency to seek simple comforts like food, when troubled, research has been

directed toward finding a link between emotional distress and obesity (Haskeew & Adams, 1984). Research by Stunkard (1980), revived speculation that anxiety, depression and stress are emotional states that are critical to the onset of obesity in some people. Therapists have reported the use of food as a compensation for life's upsets. It would appear that food replaces that which seems to be missing in life, soothes and calms and helps in handling the daily stresses. According to Haskeew and Adams (1984), people under stress have a tendency to do the opposite of what is good for them - fat people go on binges, while thin people stop eating. Studies by Bray (1978) and Simon (1963) suggested that obese people - especially obese women - use fat as a shield, i.e. the fat person makes a statement to the world about what he can and cannot do. The individual feels that obesity protects him from certain social and cultural obligations, because few people expect a fat person to date or have an active sex life. It would appear that many people still believe that being fat totally limits social opportunity.

Reactive Obesity

Reactive obesity is thought to result from ingestion of excess food as an emotional reaction to situations in the environment. Stunkard (1980) states that reactive obesity could also be an abnormality which is a reflection of inappropriate responses to the feeding situation during the child's growth and development. Individuals with reactive obesity, have been classified as persons who:

- (a) overeat in response to emotional tensions,
- (b) overeat as a substitute gratification for other emotional needs,
- (c) overeat as a symptom of underlying emotional illness,
- (d) have an addiction to food.

Numerous studies (Haskeew & Adams, 1984; Klesges, 1984; Wadden & Stunkard, 1986) have shown that depression, although not severe, is common in obese patients. Stunkard (1980) found that ingestion of food has frequently been used

to reduce the feelings of emotional deprivation which had been present since early childhood. This eating pattern has been historically associated with unstable marriages in the family of many obese patients (Stunkard, 1980).

Stunkard (1980) reported that the obese individual is aware of the fact that he eats more when tense or anxious, that he has a compulsive inclination to eating, as well as a neurotic need for food. According to Bronkhorst (1984), these adults are characterized by the fact that they normally hide all negative emotions: for example, they do not overtly become angry. This behaviour leads to feelings of depression and anxiety and as a defence mechanism against these emotions, they overeat.

Developmental Obesity

As a result of parental rejection, or as a result of a serious disturbance in the parent-child relationship developmental obesity occurs during childhood (Bronkhorst, 1984). The parents are aware of this rejection and compensate by over-feeding the child. The child, therefore, never learns to distinguish between emotional and physical needs, because the mother reacts to all the child's needs singly with food. This leads to a disturbed perception of internal needs and signals (Bronkhorst, 1984).

Stunkard (1980) found that in comparison to individuals with reactive obesity, individuals with developmental obesity, have minimal emotional problems.

Cultural Attitudes

Bruch (1974) states that whatever the psychological problems of obese patients, these problems are compounded and reinforced by a hostile cultural attitude, which regards even a mild degree of overweight as being ugly and abnormal and condemns it as a sign of greed and self-indulgence. She states that there is no doubt that the campaign against overweight is damaging in its effect on mental health. It seems that the medical profession has all along shared the

condemning cultural attitude. Bruch (1974) reports that increasing knowledge about various underlying physiological and psychological mechanisms has encouraged a more tolerant and respectful approach to the problem of obesity and advocates social acceptance of human diversity and the fostering of freedom and initiative in the individual as a means of preventing obesity. She repudiates manufactured, stereotyped perceptions and demands instead respect for human individuality.

Personality

McReynolds (1982) on reviewing reports on the mental status of obese individuals who were referred to psychiatrists in mental health settings, concludes that psychological factors are implicated in the etiology and course of obesity. McReynolds (1982) further concludes that obesity results from a developmentally related disturbance of personality and emotional adjustment. McReynolds (1982) however, cautions against the generalization of clinical case findings and suggests instead that a sensible conclusion would be that obese individuals suffer from a personality or neurotic disorder. He suggests that the origin of this personality or neurotic disorder could be traced back to parental relationships, problems of nurturance, growth or dependence.

Klesges (1984) reports that data obtained from behaviour specific personality measures indicate that individuals who are overweight perceive themselves as less assertive, but more self conscious and depressed than individuals of normal weight in situations related to eating, dieting and weight. Klesges (1984) cautions against the generalization of this finding to other populations as the study was carried out on college students who were only slightly overweight. However, Klesges (1984) states that the finding that only weight related assertiveness, self consciousness and depression discriminated weight classes concurs with theories that hypothesize a high degree of behavioural specificity in individuals.

On evaluating the hypothesis that obesity is a depressive equivalent Simon (1963) concludes that there is a selective connection between an individuals personality and his overweight.

Anxiety

Leon and Roth (1977) reported an association between anxiety (emotional arousal) and food intake based on findings from clinical literature. They state that it appears that overeating is used as a defense mechanism during periods of stress.

Stuart (1978) states that most researchers believe that overweight individuals tend to be more emotional than individuals of normal weight. The implication is that the overweight are more likely to look for comfort in food during times of stress. Findings by Stuart (1978) indicate that many overweight individuals cite depression and anxiety as emotional triggers to overeating. It is possible that the anxiety and depression experienced, are the result of previous unsuccessful attempts to lose weight and trigger a vicious cycle of tension/eating/more tension/more eating. Stuart (1978) suggests that the daily confrontation with a tangible reminder (his overweight) that he has failed to meet his own expectations possibly contribute to psychological stress resulting in anxiety and depression.

Pitta, Alpert, and Perelle (1980) refer to McKenna who in 1973 found that obese individuals in his study increased their food consumption when anxious. This concurs with Shipman and Plesset's 1963 findings that anxiety scores of obese individuals were much higher than those of normal weight individuals. Pitta et al., (1980) carried out a study of the relationship of anxiety and depression to weight-loss during behavioural treatment of obesity plus the effect that successful weight-loss has on anxiety and depression. Results of their study indicate that anxiety and depression decrease with weight-loss suggesting a relationship between anxiety, depression and obesity. Hibscher and Herman (1977) also report that obese

individuals appear to overreact significantly to anxiety which leads to overindulgence in food. Their study categorized the obese as dieters who exhibit obese behavioural attributes in that they overeat in reaction to anxiety. Hibscher and Herman (1977) state that it appears that the stress the obese individuals experience as a chronic dieter results in anxiety. The obese individual then attempts to handle the emotions of anxiety and depression by overindulging in food. Hibscher and Herman (1977) conclude that this overeating contributes to the obese individuals weight problem.

Body Image

Leon and Roth (1977) on reviewing studies by researchers on individuals who had an intense preoccupation with obesity, report that it appears that these individuals viewed their bodies as grotesque and loathsome and that their view was not affected by weight-reduction. Leon and Roth (1977) state that in 1967 Stunkard and Burt carried out a study on the age of onset of body image disturbance. These researchers found that when the body image disturbance began in adolescents it persisted for at least 20 years even after successful weight-loss. It appears that the obese overestimate their body size even after losing weight. In concluding, Leon and Roth (1977) state that research on body image and obesity indicates that individuals whose obesity began in adolescence were inclined to report the greatest affective distortions of body image.

Findings in a study conducted by Wolf and DeBlassie (1983) supports the view that obese individuals have poor body images and low self esteem. The obese individuals who participated in their study scored below the twenty fifth percentile on the self esteem score of the Tennessee Self Concept Scale (TSCS) before treatment. There were only slight differences in the scores of moderately and severely overweight individuals at pre-testing in this study. The conclusion reached by Wolf and DeBlassie (1983) is that weight problems can have a negative influence on the obese individuals perception of his body image and on his self

esteem.

Oelofse (1984) states that in 1980, Wineman concluded that wrong eating habits result when a child is rewarded with food and that this leads to the forming of a negative body image reinforced by the critical reaction and feedback received from others during childhood. As a result of this feedback, which causes the child to withdraw socially, the obese individual does not acquire the skills needed to handle the demands of adult life. Oelofse (1984) further reports that in 1974, Gerhardt et al., concluded that these negative environmental factors strengthen the child's distorted body image and result in a low self image. Oelofse (1984) on reviewing literature on obesity concluded that psychological factors play a crucial role in the etiology of obesity of which a distorted body image is one.

Williamson, Kelley, Ruggiero and Blouin (1985) on conducting a comparison of bulimic, obese and normal weight subjects report that disordered eating behaviour as observed in obese individuals possibly worsens body image distortion as the obese individuals perceived themselves as larger than they were. Results of their study indicated that the obese individuals participating on their study were characterized by preoccupation with body image, obsessive thinking, alienation and guilt. Williamson et al., (1985) suggests that these characteristics could possibly play a role in the etiology of obesity and bulimia.

Problem-solving skills

Gormally and Rardin (1981) found that behavioural treatment of obesity led to obese individuals learning more effective coping and problem-solving skills. These skills made it possible for the obese individuals to lose weight, as they coped in a problem-solving way when confronted with emotional cues or dietary slips which previously led to overeating. Results of their study indicated that obese individuals who did not lose weight reported eating in response to emotional cues. It would therefore appear that obese individuals have ineffective emotional coping and

problem-solving skills which contribute to their overeating in response to negative emotions. The result being that these emotions are unresolved and weight-gain occurs.

2.4.2 Physiological Factors

Adipose Cells

According to Bray (1978), obesity is a disease symptom, not a disease in itself and it represents the consequence of progressive storage of calories in fat tissue or adipose cells. The number of adipose cells normally increase in an individual until adolescence and thereafter remain relatively fixed (Bray & Teague, 1980). However, these adipose cells could increase as much as three to five times above normal. The onset of this type of obesity is usually in early or late childhood, but may occur in adults as well. A diagnosis of this type of hypercellular obesity, could be made on clinical grounds; the distribution of fat is widespread when the onset is in childhood. However, when the onset of obesity occurs during adult years or during pregnancy, the condition often mainly involves enlargement of adipose cells and tends to be more central in distribution (Bray & Teague, 1980).

Stock and Rothwell (1982) reviewed the relationship between fat cell size and number in human obesity. It was proposed, that obesity which developed during adult life, resulted entirely from the enlargement of existing fat cells (hypertrophic obesity), while excess fat deposition in childhood, led to a synthesis of new adiposities and hypercellularity of adipose tissue. Both these conditions predispose individuals to obesity later in life. It was claimed that overfeeding young babies often led to obesity in later life, in a form (hyperplastic) that is reputedly more difficult to treat than hypertrophic obesity. Biopsies of human tissue have revealed that the total fat cell numbers increase in some grossly obese patients. However, they found that there was still considerable debate about the validity of the experimental techniques and the interpretation of results in this area. Stock and Rothwell

(1982), concluded that it was possible that the apparent increase in fat cell number in obese subjects simply represented a filling up of empty cells, and that this increase was not necessarily related to overfeeding in early life, particularly since many of the subjects did not report childhood obesity.

Haskew and Adams (1984) report that research has shown that fat cells are developed at certain critical periods of human growth i.e. in infancy, adolescence and in pregnancy. The degree of growth depends upon nutritional and environmental factors, which could cause the individuals genetic potential to be unfulfilled or overdeveloped. In the female, fat cells may develop during pregnancy and more may appear when existing ones grow to their maximum size, resulting in a permanent addition to the body's tissue. In addition, the physical and mental stress of pregnancy and coping with postpartum depression may create the kind of arousal state that appears to accompany overeating. Finally, it would appear that the work of caring for a new family member and the stress of adjusting to the change, may also generate the need for extra stamina, a need accommodated by the storage of extra fat (Haskew & Adams, 1984).

Smith and Fremouw (1987) concluded that it appears that fat cells are almost invariably enlarged in the obese patient, and that the total number of fat cells may increase in patients whose obesity began in childhood. Weight loss and weight-gain in adult life are, therefore, accompanied by the growth or shrinking of these adipose cells.

Energy Balance

According to Garrow (1978) the problem of obesity is essentially a problem of energy balance i.e. excess caloric intake, relative to caloric expenditure, results in storage of extra fat. Garrow (1978) states that a reduction in basal metabolic expenditure may provide the imbalance between energy intake and energy expenditure, which produces obesity. The central factor affecting the development of obesity, is the presence of a positive caloric balance.

Although the biological and physiological mechanisms leading to the positive caloric balance are often vague, the fact remains, that for a patient to add fat to his body stores, he must ingest more calories than he is expending in his daily activities (Daniels, 1984). This may occur under the following conditions:

- (a) excessive food intake
- (b) lower than normal caloric expenditure
- (c) reduction of basal caloric needs
- (d) any combination of these reasons.

Daniels (1984) states that a complicating factor, both in the development and in the continued presence of obesity, is that obesity itself limits both spontaneous physical activity and the maximal amount of work that could be performed, which results in a disturbed energy balance.

This disturbed energy balance served as a basis for the thermodynamic view of obesity. The thermodynamic view holds, that body metabolism usually speeds up during a drop in temperature, since the individual must expend more energy in order to maintain body temperature. It is suggested that obesity is caused by a dysfunction in the thermodynamic regulation of the body, since the metabolic rate of obese individuals does not increase with a drop in body temperature, as it does in normal-weight individuals (Bronkhorst, 1984). Haskeew and Adams (1984) join Bronkhorst (1984), in the view of the role of thermodynamics as a cause of obesity, and state that it has long been speculated that mammals may control their weight by raising body temperature and burning off excess food in a process known as thermogenesis. The laws of thermodynamics, therefore, state that obesity results from a positive energy balance, in which energy intake exceeds expenditure (Stock & Rothwell, 1982).

Physical Inactivity

According to Brownell and Stunkard (1980), the age-old bias that obesity results solely from excessive eating, still persists, even though it is commonly known that body weight is determined by energy expenditure, as well as by energy intake. The view that physical inactivity is the major contributor to obesity, and that obese persons eat no more (and perhaps less) than normal-weight persons, but that the former exercise less, is supported by various researchers: (Bray, 1978; Thompson, Jarvie, Lahey, and Cureton 1982). However, Brownell and Stunkard (1980) state that the issue is not simple, since physical activity and energy expenditure are often related in terms of fat used for the generation of energy. They conclude that physical inactivity is associated with obesity and that it plays a role in the development of obesity, in that obese adults are less active than their normal-weight counterparts.

Stunkard (1976) reviewed the widespread decrease in physical activity in society, caused by the many mechanical and automatic devices which have taken over tasks formerly carried out by human muscles: he states that it is clear that obesity is associated with a decrease in physical activity. Evidence exists that physical activity has a function in the regulation of body weight, not only by means of the caloric expenditure but by decreasing, or at least discouraging, an increase in food intake. However, according to Brownell and Stunkard (1980), studies suggest that inactivity is a result rather than a cause of obesity, and that it plays a more important role in the maintenance of excess weight than in the development of this excess.

In support of the above, Bray (1987) stated, that with sedentary occupations, food intake and body weight tend to be higher than with moderate degrees of activity, and reiterated that physical inactivity plays an important role in the development of obesity. Bronkhorst (1984), also reports that it is apparent that the reduction in physical activity in Western society has led to an increase in the occurrence of obesity.

Body fat distribution

Various factors such as sex, frame size, muscle mass, age and physical activity influence body fat distribution in individuals. VanItallie and Woteki (1987) report that it could be conjectured that persons who are at the upper end of the scale regarding frame size and/or muscle mass, are also at increased risk of becoming obese.

Body fat content differs significantly between men and women, with the most striking differences being in the after-puberty cycle. Stock and Rothwell (1982) state, that at puberty, females have almost twice as much body fat as males. It is at this stage that differences in distribution are most obvious, with fat accumulating predominantly in the upper part of the body in males and in the lower part in females (Stock & Rothwell, 1982).

Other factors which could also modify body composition, are the frequency with which food is ingested, diabetes in the mother influencing the body composition of newborn infants, and age of onset. Childhood obesity leads to an even distribution of body fat, whereas late onset obesity tends to induce fat deposition in the central part of the body. Stock and Rothwell (1982) state that there is a strong genetic influence on body fat distribution, and that familial trends in size and shape are commonly observed. At present, however, there is no single answer as to why fat is specifically deposited in certain areas of the body.

Endocrine Disorders

According to Frawley (1984) the major endocrine disorders that could cause obesity, are hypothalamic disorders, pituitary dysfunctions, thyroid hypofunction or adrenocortical disease; these, however, are rare (Bray, 1987; Daniels, 1984). Even though obesity influences most endocrine systems, the increase in fat observed with human endocrine diseases, is usually small. Hyperinsulinism produced by islet cell tumours or by injection of excess quantities of insulin, produces increased food intake and

increased fat storage, but the magnitude of this effect is small. A somewhat more substantial obesity is observed with the increased cortisol secretion which occurs in Cushing's syndrome. Obesity may also occur with hypothyroidism; aberrations in the distribution of body fat are observed with hypogonadism (Bray, 1978 ,1987; Daniels, 1984; Frawley, 1984), while in the adult-onset form of diabetes mellitus, weight-gain frequently precedes or is associated with the onset of the disease.

According to Bray (1987), Cushing's disease is probably the form of endocrine disease most often associated with obesity. This illness is associated with weight-gain, hypertension, glucose intolerance, hirsutism, amenorrhea, plethora and fullness of face. The pattern of weight-gain in Cushing's Syndrome is characteristic. Fat is accumulated around the trunk in the supraclavicular fossa and over the dorsal posterior cervical region. The development of obesity as a manifestation of Cushing's disease, is most striking in children, but fortunately it is a surgically curable form of obesity.

A syndrome of obesity which occurs as a result of injury to the ventromedial hypothalamus is hypothalamic obesity (Daniels, 1984; Frawley, 1984), tumors being the most frequent cause of this injury. Hypothalamic obesity in man is a hypertrophic form of obesity associated with increased food intake, weight-gain and hyperinsulinemia, but is a rare syndrome in man. Bray (1987) states that hypothalamic obesity has been reported in human subjects with hypothalamic damage caused by trauma, malignancy and inflammatory diseases.

The conclusion reached, is that obesity infrequently has an endocrine basis and is most often due to an imbalance between caloric intake and energy expenditure (Frawley, 1984).

Genetic Factors

Bray (1987), states that genetic factors in human obesity manifest themselves in two ways. First, there is a group of rare diseases in which the evidence suggests that genetic factors are of major importance. In most of these syndromes obesity is only moderate, but in a few it may be pronounced. These forms of obesity are transmitted both by recessive and dominant genes.

Some syndromes which are genetically transmitted and could lead to the development of obesity, include:

The Laurance-Moon-Bardet-Biedl (LMBB) syndrome

The Alström-Hallgren syndrome

The Prader-Willi syndrome of hyperostosis frontalis interna (Morgagni-Stewart-Morel)

The Triglyceride storage disease

Cohen's syndrome

Carpenter's syndrome

Studies undertaken with twins, provide evidence that genetic factors play a role in the etiology of obesity (Price, 1987). The most acceptable hypothesis seems to be, that there is a complex interaction between environmental and genetic factors. This suggests that a predisposition exists, which implies a vulnerability to obesity. Environmental factors will, however, determine whether or not a condition of obesity will develop (Price, Cadoret, Stunkard, & Troughton, 1987).

The most definitive studies of genetic versus environmental factors in obesity, emerged from the examination of the relationship between body weight in twins (Price, 1987; Smith & Fremouw, 1987). These studies indicate that the inheritability of obesity is between 0,6 and 0,8. This was supported by studies conducted by Stunkard (1987), who found that the inheritability for obesity ranges from 0,51 to 0,88 for twins. This suggests that nearly two-thirds of the variability in body weight is attributable to genetic factors.

On reviewing genetic causes of obesity, Bronkhorst (1984) concluded that the genetic potential for the development of obesity is determined by conception, but how this potential develops, depends on the interaction with the environment. According to this observation, it appears that obesity is the result of environmental factors, rather than emanating from genetic factors. However, according to Price (1987), human obesity is a familial disorder. Family members share diet, cultural background, various aspects of lifestyle and genes. Because of this confounding of the genetic and cultural factors in families, the question of the genetic inheritance of obesity has remained controversial.

In summary, it is clear that single and polygenic inheritance are involved in the transmission of obesity in man. However, it is also clear that in susceptible individuals (those who are genetically predisposed) environmental factors may play a role of overriding importance (Bray, 1987; Price et al., 1987)

2.4.3 Biographical and Demographic Factors

Social Environment and Socio-economic Factors

According to Wooley and Wooley (1984), a possible cause of obesity, could be the cultural weight obsession. They propose that this obsession is responsible for the current epidemic of eating disorders. These disorders are a reaction to the social demand to maintain a body weight at which extreme hunger is to be expected. Wooley and Wooley (1984) state that this moralistic social pressure aggravates inner psychological problems and conflicts of youngsters whose weights deviate from the stereotypic picture of a desirable figure. They express concern that peer pressure on youngsters to be thin, seems to be increasing and report that there appears to be an increase in parents - and even physicians - condoning or encouraging excessive thinness.

With regards to socio-economic factors, VanItallie and Woteki (1987) report that studies have provided evidence that overweight is more common among lower-class women than

among upper-class women. However, men at or above the poverty line, exhibit a somewhat higher occurrence of overweight. Among women, the situation is reversed; between the ages of 25 and 75, women in the poverty category show a much higher prevalence of overweight than do women above the poverty line. VanItallie and Woteki (1987) found a strong association between overweight women and low-economic status; this encourages speculation that environmental factors play an important role in obesity. Daniels (1984) and Stunkard (1980) stated that the relationship between social factors and obesity have repeatedly been established and that they regard social factors as being among the most important influences on the occurrence of obesity. Stunkard (1980) reported that among the three variables of age, social class and sex, social class showed the most significant correlation with obesity.

According to Stunkard (1980), obesity is in large part a function of our life-styles and ultimately, of those powerful social and economic forces which have given rise to and which sustain those life-styles. These include food advertising and labour-saving devices (Daniels, 1984). However, Shaw (1977) states that the community of which man is part, also plays a role in man's social environment and that it is the geographical community in which he lives and that he calls his home, that impersonal social forces and intimate personal forces have a major impact. Stunkard (1980) stressed that the influence of calorie-dense packaged foods, promoted by an industry that spends millions of rands a year on advertising, have an immense impact on the eating habits of individuals. The implication is that obesity is a result of the way man lives; indeed of his whole life-style. Stunkard (1980) suggested that the most effective means of controlling obesity appeared to lie in the changing of that life-style. The implication is, that the social and economic forces which support the life-style, would have to be altered.

Further support of the above findings are provided by Stunkard (1976, 1980) who, on analyzing the data used in the Midtown Study (comprising of 1660 subjects), conducted by

Srole et al., in 1962, deduced that the nearer a group of people approached the standards of the upper-class, the less the occurrence of obesity would be. Results obtained, also showed that the socio-economic status of the parents of the subjects, influenced the occurrence of obesity and vice versa and that the relationship of the parents' social class to the subjects' obesity, was almost as strong as that of the subjects' own social class. In 1976, Stunkard stated that the fact that obesity is six times more frequent in lower-class than in upper-class women, had profound implications for theory and for therapy, as it implies that whatever its genetic and biochemical determinants, obesity in man is susceptible to an extraordinary degree of control by social factors.

Support for the above, is to be found in the systems theory which views obesity as a psychosomatic disorder with a multifactorial etiology and complexity. According to Stock and Rothwell (1982), this theory states that when the individual's interaction with his family were ignored, the treatment of obesity could be less successful. The implication is, that disorders should not be studied in isolation but as a interdependent component of the total system. An individual's behavioural and psychic conditions exist in interaction with his social environment. The continual influence of the family members on one other is taken into account and both the influence of the past on the development of the psychosomatic symptoms and to what extent the family members are maintaining the symptoms, are investigated. The theoretical view is, that the individual forms part of his social context and that change in one demands change in the other. Therefore, instead of a simplistic, one-way causal connection, this theory calls for a process of mutual accommodation between the individual's obesity and the system's rules. The possibility that the interaction patterns of the family could be contributory towards the development of obesity, is also considered.

Stock and Rothwell (1982) state that the factors which affect food intake, are complex and numerous. However, it is certain that human feeding patterns are strongly

influenced by psychological, economic and social factors. Observations with regards to the phenomena of sensory specific satiety, suggest that when the various social and psychological incentives to eat are removed, man probably can control food intake quite precisely (Stock & Rothwell, 1982).

In summary, obesity is a significant problem, both in affluent nations and in many developing countries as well. Both overweight and obesity are more prevalent in females than in males and its frequency is influenced by ethnic, social and economic factors.

Age

With reference to age, VanItallie and Woteki (1987) report that studies have shown that among adults, the occurrence of overweight increases for at least three decades after the age of 25 years. Several life events for women may be associated with weight-gain, i.e. marriage, pregnancy and menopause, as well as the use of oral contraceptives (VanItallie, 1984). It would appear, that for men, the greatest change in weight occurs from the age of 20 to 30 years, possibly because of changes in life-style.

Bray (1987) provides a classification of etiological factors in obesity as related to age of onset (see Table 1).

Table 1

Classification of etiological factors in obesity by age of onset

Age at onset	Etiological factors
Childhood (0 - 18 years)	Genetic Infant feeding Endocrine Hypothalamic Unknown Progressive Infantile Drugs
Adulthood (over 18 years)	Feeding patterns Inactivity Endocrine Pregnancy Hypothalamic Drugs

According to Brownell and Stunkard (1980), obese children become obese adults and the psychological consequences are more troublesome for persons experiencing the onset of obesity in childhood, because body image disparagement, binge-eating (bulimia) and night eating occur.

Oberholzer (1984) states that progressive obesity which begins in childhood and continues throughout life, is a particularly ominous form of weight-gain. It has been noted that 75 to 80% of obese children become obese adults. This has led to the conclusion, that the prognosis of childhood obesity is generally poor. A reason for an increasing interest in obesity in children, is the strong research evidence that the so-called baby fat will not be outgrown.

Race

Regarding the role of race as a determinant of obesity and its prevalence in women, it is noteworthy that obesity is common among black females in Africa as well as in the USA (VanItallie & Woteki, 1987). VanItallie and Woteki (1987) report on a study conducted by Walker and Segal in 1980 at the Baragwanath Hospital in Johannesburg, Republic of South Africa, which found that among new black employees and those having being in employment for at least a year, more than 50 percent of the females were obese, while obesity in the males was uncommon. In 1980, Walker and Segal stated that obesity in black African females develops even while they are on a diet high in fiber and low in animal fat and protein (VanItallie & Woteki, 1987).

2.4.4 Summary

Garrow (1978) reached the conclusion that neither is there a single cause for the positive energy balance which causes obesity nor is there any single genetic or environmental factor which renders an individual immune to obesity, or which makes obesity inevitable.

That a strong relationship exists between the prevalence of obesity and social class, ethnicity and religious background was demonstrated by epidemiological research (Stunkard, 1980). Daniels (1984) stated that it is an accepted fact that social attitudes, fads and fashions also affect eating behaviour and weight; therefore obesity cannot be viewed merely as an individual predisposition. The conclusion is, therefore, that social norms and pressures, or elements of the individual's environment, influence the body weight that a person maintains.

Obesity has multiple determinants; biological, metabolic, genetic, psychological, cultural and environmental factors may contribute to the development and/or maintenance of excess weight (Brownell & Stunkard, 1980).

From the above discussion, it is clear that obesity is an extremely complex condition and that psychological factors play a significant role in the etiology of obesity. It is also quite clear that no single etiological factor causing obesity could be found and that numerous factors interact, all of which differ from individual to individual.

Should obesity be treated at all?

Garrow (1978) and Wooley and Wooley (1984) suggest that it is difficult to construct a rationale for treating any but massive, life-endangering obesity. It is, however, clear that health care specialists must vigorously treat weight obsession and its manifestations such as:

- (a) poor self image and body image disparagement,
- (b) disordered eating patterns created by dieting,
- (c) metabolic depression produced by dieting,
- (d) inadequate nutrition due to constricted eating behaviour,
- (e) unhealthy lifestyles which are often marked by inadequate exercise.

Stunkard (1987) states that there are circumstances in which obesity is not a major concern, for example when the patient has other disabilities and the loss of excess weight would confer little net benefit. He recommends that care must be taken against indiscriminately including every obese patient in a weight-reduction programme, and suggests careful selection of patients for whom there is some likelihood of success. It appears that the successful patients could be identified by their stable personality, high motivation, late onset of obesity and absence of any previous unsuccessful attempt at weight-loss (Stunkard, 1987).

In spite of the enormous social pressure to be thin, most obese people do not become thin (Brownell, 1982). Obesity is one of the most difficult medical and psychological problems, because of its resistance to treatment and its tendency to constitute one of the most important medical and public health problems in contemporary society.

Smith and Fremouw (1987) suggest that a more worthwhile effort, especially for those obese individuals unsuccessful in losing weight, might be to help these individuals develop

a healthier life-style and to develop a better self-concept independent of weight-loss. If overweight people learnt to feel happy, attractive and self-confident, less emphasis would be given to the importance of being thin, and the prejudicial attitudes towards obesity in our society might diminish.

2.5.1 Psychological Treatment

Psychotherapy in the treatment of obesity

Garrow (1978) believes that by simply providing psychotherapy to obese patients to relieve their psychological stresses - with no attempt to alter their food intake patterns - would have no effect on their weight and could therefore, not be seen as a treatment for obesity. However, Bruch (1974) states that, although there is a widespread assumption that psychiatric treatment is a costly and self-indulgent alternative to reducing, this is not necessarily true. She sees psychological support as being a necessary part of every reducing regimen. Although psychotherapy could help some obese patients approach life in a more mature and realistic way, its influence on weight control is possibly indirect. However, opening up alternative ways of experiencing satisfaction as a solution to underlying emotional problems, may remove the basis for excessive eating. Bruch (1974) stresses that psychotherapy is a process of inner growth, which cannot be hastened beyond the individual's innate rhythm in treatment and should, therefore, be approached with care.

As no evidence has to date been produced concerning the usefulness of psychotherapy and psychoanalysis when compared to other less expensive aids in the treatment of obesity, there is serious questioning concerning its usefulness (Stunkard, 1980). However, Fawzy, Wellisch, Pasnau, Dornfeld, Maxwell, and Schroth (1984), agree with Bruch (1974), that psychological intervention should be an essential component of any weight-loss programme, since some obese persons with a number of psychological and emotional problems begin dieting. It has also been found, that

weight-loss programmes can and do cause adverse psychological and metabolic complications, as well as disturbances of the body image, with the result that it seems apparent that only people who have already failed to lose weight on their own, go to a doctor's consulting room, and only the failures of medical treatment are referred to the psychotherapist.

Fawzy et al., (1984) found that three indications for psychotherapy or psychoanalysis in the treatment of obesity are disparagement of body image, bulimia (binge-eating) and emotional disturbance (anxiety or depression). These findings provide support for Stunkard's (1980) recommendation for an assessment of the suitability for psychotherapy (including realistic goals on the part of the patient), and at least a measure of psychological-mindedness, including the ability to relate symptoms and behaviour to specific life problems before proceeding with treatment. Stunkard (1980) further cautions that care must be taken to prevent the obese patient's overdependence on the therapist. He concludes that a therapeutic approach which encourages assertiveness and which corrects cognitive distortions, is useful in the treatment of obesity.

Bruch (1974) sees psychotherapy as a means of helping individuals who have an eating disorder to learn a more competent, less painful way of handling their problems. Bruch (1974) states that the intrinsic task of psychotherapy effects a meaningful change in the personality, so that a constructive life becomes possible, without misusing the eating function. Bruch (1974) concludes that conventional psychotherapy, with its focus on intrapsychic conflicts, fails to correct these conceptual distortions and inadequacies in self-perception; she adds, that the therapeutic goal is to make it possible for a patient to uncover his own abilities, his resources and inner conflicts in thinking, judging and feeling. Once the capacity of self-recognition has been experienced, there usually is a change in the whole involvement atmosphere. Stunkard (1980) supports Bruch (1974) in voicing the belief that psychotherapy cannot change the basic pattern of over-eating

in response to stress that is so common among obese patients. His findings indicate, that years after apparently successful psychotherapy and successful weight-reduction, people who have over-eaten under stress, would continue to do so. However, Stunkard (1980) emphasizes that psychotherapy could help obese people live less stressful and more gratifying lives, resulting in a lower inclination to over-eat.

Orbach (1979) states that a psychoanalytic approach has much to offer toward a solution to eating disorders resulting in overweight. She states, that it provides ways for exploring the roots of such problems in early experiences, and added that psychoanalytic insight helps the patient to understand what getting fat and overeating means to him, merely by explaining his conscious and unconscious acts. However, with regards to female patients, she cautions that an approach based exclusively on classical psychoanalysis devoid of a feminist perspective, is inadequate. The reason for this, is that she sees the central issues of overeating as being rooted in the social inequality of women.

In contrast to the above, Adams and Chadbourne (1982) suggest the use of therapeutic metaphor as a psychotherapy in the treatment of obesity. This method aids patients both in the loss of weight and in maintaining the new lower-weight level. Basically, metaphors are employed to:

- (a) uncover underlying feelings,
- (b) reveal hidden solutions,
- (c) create an aversion to undesirable intakes,
- (d) provide the patient with a "thin" self-concept,
- (e) increase compliance.

The results of a study conducted by Fawzy et al., (1984), show that individual crisis-oriented psychotherapy does enhance the patient's ability to complete a weight-reduction programme. Positive factors in their study were: the increased ability to discuss the emotional crises that arise during weight-loss programmes and the additional support patients gained through contact with professional staff.

Fawzy et al., (1984) recommend that serious consideration should be given to individual crisis-oriented therapy, in an attempt to facilitate successful programme completion and compliance for patients undergoing a programme for weight-loss that includes fasting.

Group Therapy a Psychotherapy

Group psychotherapy, which has the goal of obtaining relief of symptoms, attitude and personality change, is a form of treatment which entails the regular meeting of a small group of patients with a trained conductor over a long period of time (Jones, McPherson, Whitaker, Sutherland, Walton, & Wolff, 1971). According to Vander Kolk (1985), who quotes Gazda (1978), group work refers to the dynamic interaction between collections of individuals, for personal growth and enrichment or in an attempt to find solutions to their problems. The results of a study conducted by Basler, Brinkmeier, Buser, Haehn, and Mölders-Kober (1985) demonstrate that group therapy produces specific intervention effects: an improvement in health knowledge and health behaviour, a reduction in salt intake and body-weight, a decrease in blood pressure and a long-term reduction in medication.

Orbach (1979) describes six important steps in group psychotherapy for eating disorders. They aim to -

- (a) demonstrate that the patient has an interest in being fat,
- (b) show that this interest is largely subconscious,
- (c) conduct specific exercises to bring this theme to the patient's consciousness,
- (d) explore the meaning that being fat holds for each individual,
- (e) ask the question whether the fat does what it is supposed to do, and
- (f) help the patient to reclaim aspects of the self, previously attributed solely to fat.

Orbach (1979) found that people in groups begin to develop different self-images because they are enabled to incorporate positive aspects and benefits into their views on fatness. She states that what is important, is that the conflict is brought to light, though not necessarily resolved; that the patient should understand that it exists and that overeating will not make it vanish. This acknowledgement then becomes a powerful weapon in the fight against disordered eating. Orbach (1979) found that the group process allows other members of the group to identify with what the individual is experiencing. It provides an opportunity to mutually share difficulties, which reduces their isolation and feelings of impotence which could have led to weight-gain.

In group psychotherapy the goal is for the patient to break his addictive relationship with food. While weight-loss is generally an important sign that the addiction is broken, a primary concern, is that the individual should begin to feel more comfortable about food. Orbach (1979) states that in a group psychotherapy situation, individuals have the opportunity to openly discuss their eating habits, and to explore the complicated feelings they may have about their bodies. The benefits of belonging to the group, include help in getting through a seemingly insoluble problem and opportunities in this way to try out more assertive behavioural patterns. Members could provide feedback and encouragement; they could help each other to challenge unreal expectations; and they could also provide a direct way of dealing with the phenomena of disordered eating. Although members expect that joining the group would produce dramatic and instant weight-loss, the immediate goal remains to break the addictive relationship with food. This includes the exploration of the symbolic meanings of fatness and thinness for the individuals in the group and creating new ways to approach food and hunger (Orbach, 1979).

Bess and Marlin (1985) conclude that the formation of a group reinforces motivation and promotes dietary and behavioural modification. Group discussions made the members of the group (in this case physicians) consciously

aware of their own negative attitudes which derive from cultural stereotypes of obesity.

Lacey (1983) conducted a study based on the implementation of group therapy for chaotic eating patterns. Findings indicated, that a psychodynamically orientated group therapy (aimed at handling and controlling the pronounced tension, anger and depression which follows giving up the symptoms of bulimia nervosa), provided very good treatment results. It is extrapolated, that obese patients would also benefit from this type of treatment.

However, a study conducted by Fawzy et al., (1984) found that there was a high dropout rate in the group therapy area part of a weight-reduction programme. A number of patients in this study reported, that assignment to a psychotherapy group elicited intense negative feelings. Some of these patients experienced problems with their perception of the group. They state that the group sessions create considerable anxiety, based on the implication that they would be compelled to reveal themselves in a group setting. To overcome this problem, Fawzy et al., (1984) suggest that consideration be given to the critical variables in the patient's history, such as those suggested by Stunkard (1980) in his description of indicators for individual psychotherapy.

Behaviour Therapy in the treatment of obesity

Findings from a study conducted by Stunkard (1987), suggest that behaviour therapy could help obese patients to lose a moderate amount of weight and to maintain the new lower-weight level for at least one year. Behaviour therapy appears superior to the other major treatment modality for obesity - pharmacotherapy - because of its more effective maintenance of the lower-weight level. Stunkard (1987) states that behaviour therapy is based upon a set of assumptions, which derives directly from the radical behaviourism of Watson and Skinner; the characteristics are -

- (a) the assumption that all behaviour - normal and abnormal - is acquired and maintained according to definable principles,
- (b) that people are best described by their behaviour i.e. what they think, feel and do in specific situations and not by dispositional tendencies such as hostility and insecurity,
- (c) the attempt to specify treatment measures as precisely as possible and to evaluate outcomes as objectively as possible,
- (d) the individualism of treatment,
- (e) an effort to provide a continual and critical assessment of the treatment while it lasts.

Jeffrey (1987) states that, considering the above basic assumptions, it is evident that behaviour therapy concentrates on the teaching of self-management. Behaviour therapy attempts to teach patients to identify inappropriate behaviours and the environmental stimuli that support them, at the same time using methods which include self-monitoring and functional analysis. Using problem-solving, self-reinforcement, role playing, incremental goal setting and social contracting, alternative behavioural patterns and support systems are developed. Jeffrey (1987) emphasizes that skills that are thought to play an important role in successful weight management, receive special attention. These include skills which enable the patient to reduce his eating rate and to improve management of his personal environment, aimed at limiting exposure to undesirable stimuli. Gormally and Rardin (1981) describe behaviour therapy as an attempt to train patients to abandon unhealthy eating habits. They state that change in behaviour is brought about by using a system of rewards or punishments, as well as by effecting changes in the patient's environment. Haskew and Adams (1984) sum up behaviour therapy as being an attempt to wean patients away from junk foods and snacks, to train them to eat reasonable meals at regular mealtimes and to teach them to develop activities which could serve as substitutes for eating.

However, Wilson (1980) states that no treatment strategy would be successful in the long run, unless clients adhered to therapeutic instructions. He stresses that more than any other form of psychological intervention, behaviour therapy involves requiring the person to do something; for example, placing the fork down between bites, monitoring daily calorie intake, replacing identified maladaptive cognitions with more constructive self-statements and acting assertively. Wilson (1980) stresses the importance of the patient's being able to comply with the therapeutic assignments. He suggests that the patient could be unable rather than unwilling to comply; he concludes, that this would indicate the need for direct training and rehearsal of the assignment during treatment. However, Wilson (1980) cautions, that in spite of the consistent finding that compliance decreases as the complexity of the treatment increases, most behavioural treatments have involved multifaceted programmes that require far-ranging changes in lifestyle over the course of a relatively short time-span. Unfortunately, however, patients infer that compliance to at least one or two is sufficient, even though the choice might not be the most desirable. Wilson (1980) found that patients are very likely to forego time-consuming and effortful, but effective methods.

Bronkhorst (1984) refers to Miller (1980) and provides a summary of the approach adopted by behaviour therapy, i.e. the belief that -

- (a) a learning defect causes obesity and that it is curable by using conditioning,
- (b) obesity is a simple deviation (caused by eating too many calories),
- (c) obese persons are over-eaters,
- (d) obese persons are more sensitive to food stimuli than are non-obese persons,
- (e) the eating patterns of obese and non-obese persons differ in important ways, and
- (f) an obese persons weight will reduce if he could be taught to act like a non-obese person.

Brownell (1984) states that the strength of behaviour therapy, as a treatment of obesity, lies in long-term changes, and not in producing initial weight-losses. He adds, that changes produced in behavioural programmes, are well-maintained, at least as far as the average patient is concerned. Brownell (1984) concludes, that behaviour therapy contributes positively towards the long-term maintenance of a lower weight-level. He states that the time is ripe for teaming behaviour therapy, with more aggressive approaches to weight-loss.

Wilson and Brownell (1980) reviewed available literature and found that the only significant variable that emerged from the study, was that behaviour therapy is most appropriate for the mild to the moderately obese. They found that morbidly obese individuals (those above 300 lbs (136,36 kg), or 100 lbs (45,45 kg) or more above ideal body-weight), are not good candidates for behavioural treatment.

In his comparison of the attrition rate from treatments, Wilson (1983) reports that behaviour therapy lowers attrition among dieters. He adds that attrition in behaviour therapy studies is far lower than in traditional treatments, or in other widely used programmes, e.g. self-help groups.

According to Stunkard (1984) traditional behavioural treatment of obesity is based on classic applied behavioural analysis. This considers in great detail the behaviour to be changed, its antecedents and its consequences. As the primary behaviour to be changed, is eating, a number of exercises are designated to slow the rate of eating and allow physiological mechanisms of satiety to exert their effect. Stunkard (1984) states, that focus is fixed on the antecedents of eating behaviour, which among others include the control of relatively remote antecedents such as shopping for food, to more proximate ones, such as the availability of high-calorie food. The treatment entails assisting patients to remove stimuli that cause eating from their environment, helping them to plan strategies to control eating when stimuli are present and to avoid television and reading materials that might distract their

attention from their efforts at stimulus control. Stunkard (1984) introduces another element in applied behavioural analysis: that of the consequence of behaviour. This entails rewards for carrying out the various prescribed behaviours, primarily those involving stimulus control.

In addition to this basic behaviour analysis, Stunkard (1987) states that behavioural treatment of obesity includes four other elements, namely -

- (a) recording the behaviours to be modified in detail, or self-monitoring,
- (b) nutrition education,
- (c) increased physical activity, and
- (d) cognitive restructuring.

Stunkard (1987) reports that behaviour modification is easy to learn, because it is easy to specify. He reports that it is administered by persons who do not have professional training, because many of the key elements are available in easy-to-use treatment manuals.

However, Wadden, Stunkard, Brownell, and Day (1983) report, that although behaviour modification has greatly improved the treatment of obesity in the brief period since its introduction in 1967, it suffers from a serious problem, namely the small size of the weight-losses it produces. They propose the incorporation of very low-calorie diets, limiting the duration to a period of three months, under careful medical supervision. The results of a study conducted by Wadden et al., (1983), suggest that the combination of a very low-calorie diet and behaviour modification, offers promise for the treatment of moderately overweight persons. They obtained evidence that group behavioural treatment protects dieters from the adverse emotional consequences of dieting.

Cognitive Behaviour Therapy

According to Stunkard (1987), cognitive behaviour therapy stems from the recognition that the internal monologues that occupy so much of one's time are readily accessible. He

states that they could be quantified and treated very much as any traditional operant, in terms of reinforcement and extinction. In cognitive behaviour therapy, attention is directed to the critical role that cognitions and private monologues might play in the maintenance and control of obesity (Stunkard, 1987). He proposed that the first step in applying cognitive strategies to weight-control is to help patients discover their most common negative monologues, or self-statements and to estimate their frequency. Arguments are then used to counteract these negative monologues, which are stereotyped and limited in number. The patient is guided towards the learning and use of more appropriate self-statements as automatic responses to negative statements. Stunkard (1987) reports that this type of training i.e. arguing with oneself, has proved to be effective in weight-reducing behaviours. In sum then, cognitive behaviour therapy is based on teaching the patient to identify the irrational basis of his thoughts, to enable him to dismiss them and to improve his behaviour (Stunkard, 1987). Mayer (1983) states, that there has been a renewed interest in a cognitive-based behavioural viewpoint, which has become a substantial influence within the behavioural therapy movement. The notion that changing cognitions are central to changing overt behaviour, has led to the development of a variety of new techniques focusing on changing thought processes (e.g. cognitive restructuring, thought-stopping and covert assertion). According to Mayer (1983), the cognitive view held, is that behaviour is merely the manifestation or outcome of thought. Stunkard (1987) provides the following examples of negative self-statements, together with the counter-arguments which could be used by patients on a weight-reduction programme:

Weight loss:

It's taking so long to lose weight.

Counter-argument: But I am losing it. And this time I am going to learn how to keep it off.

Ability to lose weight:

I have never done it before. Why should I succeed this time?

Counter-argument: There always has to be a first time. And this time I have a new programme going for me.

Goals:

I have got to stop snacking.

Counter-argument: That is an unrealistic goal. Just keep on trying to cut down on the number of snacks.

Food thoughts:

I keep finding myself thinking how good chocolate tastes.

Counter-argument: Stop that! It is just frustrating you. Think of lying on the beach in the sun (or whatever activity the patient finds enjoyable).

Excuses:

Everyone in my family has a weight problem. It is in my genes.

Counter-argument: That just makes it harder, not impossible. If I stick with this programme, I will succeed.

With regards to self-regulation as part of a cognitive-behaviour therapy weight-reduction programme, in 1976 Bellack worked with overweight people, to see whether self-monitoring (keeping record of what one eats) or self-monitoring, combined with self-reinforcement, would result in weight-loss. One group did not report back after it received instructions in self-regulation, while the other groups periodically reported back to the therapist. Findings showed that the self-monitoring groups did not do as well as the group that also used self-reinforcement, which continued to lose weight during a follow-up study (Vander Kolk, 1985). In a study by Sperduto, Thompson, and O'Brien (1986) an examination of weight-reduction groups utilizing behaviour modification was undertaken. Their aim was to

estimate the effect of monitoring and charting eating and exercise behaviours on drop-out rate and weight lost. The results of this study support the hypothesis that the completion rate in groups utilizing the monitoring forms, was significantly higher. The conclusion reached, is that the use of self-monitoring and charting techniques makes it possible for the patient to use self-reinforcement and to set realistic goals. A further advantage is, that the therapist is presented with the opportunity to directly reinforce behaviour change without having to wait for weight-loss.

Flanery and Kirschenbaum (1986) report that the self-reinforcement style is currently the best predictor of weight-control success among adults. Their findings indicate that obese adults who frequently positively reinforce themselves and who infrequently inflict self-punishment, lose more weight.

Fairburn (1981) adopted a cognitive behavioural approach to the treatment of eating disorders. Patients were trained in problem-solving and identifying thoughts which prevented behaviour change. He found that these were irrational concerns about body-weight and shape. Fairburn (1981) made use of cognitive change methods, plus conventional behavioural strategies. The result of this treatment, was a reduction in the intensity of abnormal attitudes towards food, eating, body-weight and shape. He stresses the importance of the patient's retaining responsibility for change and learning how to regulate food intake, which leads to improved self-control. This is obtained by examining attitudes, learning problem-solving skills, challenging avoidance and using cue exposure to strengthen control. The results of this study, indicate that a cognitive behavioural approach to the treatment of obesity is very useful. Fairburn's (1981) findings are supported by a study conducted by Black (1987), who found that clinically significant weight-losses result when a combined minimal intervention and a problem-solving programme are presented. The results of this study, suggest that problem-solving may be an important cognitive component in

behavioural weight-loss programmes, as well as a means of continuing weight-loss after treatment. Fairburn's (1981) findings are also supported by Woods and Heretick (1984), who in their study of self-schemata in anorexia and obesity, found that the obese showed a low level of self-control. These researchers investigated cognitive biases which underlie and encourage obesity. The findings of their study showed that the obese clearly demonstrated low self-control. Buxton, Williamson, Absher, and Warner (1985) in support of the above researchers, recommend that patients be trained to use self-control procedures to modify their diets and improve compliance with nutritional counseling.

Jordan, Canavan, and Steer (1986) evaluated the success of a cognitive behaviour therapy treatment programme in the maintenance of weight-loss, 6 to 10 years after treatment. The goals of the treatment programme were to provide management skills needed for weight-reduction, plus long-term maintenance of weight-loss. Cognitive behaviour therapy strategies for weight-loss and management were discussed. The topics stressed, were -

- (a) calorie and nutritional education,
- (b) changes in -
 - basic eating style,
 - daily activity levels
- (c) increased assertiveness in food related situations,
- (d) moods and eating,
- (e) control of food and eating environments,
- (f) impact of lifestyle changes upon weight change,
- (g) development of individualized daily eating patterns,
- (h) variations in motivation,
- (i) cognitive restructuring of -
 - attitudes and thoughts about food, hunger, eating
 - and body-weight.

Findings by Jordan and Canavan (1985) are that the weight of the 111 patients involved in the treatment programme, indicated that a cognitive behavioural programme led to short-term weight-loss, as well as to the maintenance and further weight-loss 6 to 10 years after treatment. In a follow-up study Jordan et al., (1986) determined the use of

cognitive and behavioural activities during periods of weight maintenance, as well as loss and gain in the same 111 respondents 6 to 10 years after treatment. The conclusion drawn, was that the respondents were able to differentiate between the types of attitudes and behaviours that they experienced during times of gain, maintenance and loss.

A study conducted by Collins, Rothblum, and Wilson (1986) aims at evaluating the therapeutic efficacy of a cognitive, a behavioural and a combined cognitive behaviour therapy approach to weight-reduction and maintenance. Collins et al., (1986) suggest the use of behavioural techniques, followed by cognitive techniques, to serve as maintenance strategies. They state that this could be a more powerful intervention than the weekly alternation of the two approaches, i.e. behavioural and cognitive; they also conclude, that the addition of cognitive techniques to existing behavioural treatment programmes, shows promise as a means of enhancing maintenance of the new lower-weight level.

According to Fairburn (1984), cognitive behavioural treatment of obesity is a modification of the standard behavioural programme for obesity - the differences being, that the treatment is more time-consuming and more intensive during the initial stage, with greater emphasis on cognitive factors and the maintenance of change. In addition, the cognitive components could benefit obese patients suffering from body image disparagement.

In conclusion, the above studies indicate that the inclusion of cognitive interventions (cognitive restructuring, problem-solving, improving self-control and self-regulation) into a behavioural weight-reduction programme, facilitates the maintenance of initial behaviour change needed for weight-loss and also for the maintenance of the new lower-weight level.

Self-help groups

Group treatment of weight-reduction ranges from informal get-togethers among friends and neighbours to complex organizations. These groups are formed by overweight people who are looking for support, as well as by researchers collecting information, health professionals offering help, profit oriented corporations marketing programmes, promises and various combinations (Eyton, 1987; Haskew & Adams, 1984). These programmes offer diet, exercise, and behaviour modification within a social context of mutual support and competition. The group leader attempts to create a highly motivating atmosphere, in which dispirited fat people find acceptance, challenge and renewed self-esteem (Eyton, 1987; Haskew & Adams, 1984).

Stuart and Mitchell (1980) views self-help groups as the outcome of attempts to meet physical and mental health problems, using peer and personal resources, with little if any professional help. Individuals with similar problems such as obesity or alcoholism, form groups and use a mixture of self-developed or professionally devised methods, in an attempt to obtain coping skills. Organizations staffed by paid employers who adhere to a programme developed and monitored by professionals for effectiveness, do exist. At each weekly meeting the members attempt to deal with specific challenges. The group accepts fellow-members problems as normal reactions to stressful situations, and during the weekly meetings members encourage and reinforce active participation. Members benefit from the exchange of ideas, involvement in the group social process as well as from practical and applicable recommendations obtained from the group (Stuart & Mitchell, 1980). According to Stuart and Mitchell (1980), self-help programmes have a major role to play in the management of obesity. They state that a gradual (indirect) approach is required in order to change habitual health behaviours. Because obesity is viewed as a disorder with multiple contributory causes, it requires adjustments which need supportive conditions extending through months of gradual weight-reduction, along with exposure to successful models which could accurately portray the

experience of changing core aspects of daily living.

It would appear that, in spite of the high drop-out rate experienced by self-help groups, the commercial weight-loss groups achieve weight-losses for their members, which are comparable with that of other groups treated either in general practice or at hospital out-patient clinics (Ashwell, 1979). However, the results concerning the maintenance of weight-loss, are as poor for self-help groups as they are for the medical profession. Eyton (1987), as well as Volkmar, Stunkard, Woolston, and Bailey (1981) report a very high attrition rate in the self-help groups, as well a serious lack of follow-up studies calculated to determine the success of the members in maintaining their lower level of weight.

According to Stuart and Mitchell (1980), follow-up on maintenance of the lower-weight level, suggests that the self-help group programmes into which behavioural self-management techniques are incorporated, may constitute the current treatment of choice for the mildly to moderately overweight individual. However, no concrete data on the success achieved in combating obesity exist, since no figures have been published (Bray, 1976). Bray (1987) states that results were very good when behavioural techniques were added to the positive group environment and nutritionally-balanced food programme.

Eyton (1987) states that with regards to their success or lack of success in maintaining the lower-weight level, commercial self-help groups are certainly open to criticism. However, maintaining the lower-weight level, is a universal problem and apparently an unavoidable one in the treatment of obesity.

Hypnosis

It appears, that for some individuals, hypnosis could be valuable in achieving weight-loss (Bray, 1976). Treatment to promote weight-loss, is a stock-in-trade technique of virtually all hypnotherapists, and short-term changes in

eating behaviours are not difficult to achieve by using post-hypnotic suggestions. However, Haskew and Adams (1984) caution that the effect is rarely lasting, unless it is accompanied by therapy which brings insight, understanding and emotional growth.

In contrast to the above the results of a study conducted by Bolocofsky, Spinler, and Coulthard-Morris (1985) strongly support the use of hypnosis as an adjunct to the behavioural treatment of obesity. Cochrane and Friesen's (1986) study provides empirical support for the view that hypnotherapy is a useful treatment for weight-loss, but add that further research is needed to identify the most effective hypnosis programmes, as well as the client-characteristics that are responsive to these programmes.

2.5.2 Physiological Treatment

Dietary treatment of obesity

Because such a deep conviction exists in our culture that unusual fatness is linked to overindulgence in food, it is natural to believe that changing consumption is the correct way to achieve weight-loss. However, Haskew and Adams (1984) state that virtually all diets work, but only for a while (i.e. the dieter experiences short-term weight-loss), the reason being, that all diets lower calorie intake, which in turn results in a degree of starvation, and which imposes a nutritional shock to the system (because the patient alters his dietary habits). Haskew and Adams (1984) recommend careful dietary and emotional guidance, which is individualized and which involves a long-term programme.

VanItallie (1980) suggests that following a diet, constitutes an attempt to gain a measure of control over one's eating behaviour. The idea of prescribing a diet for weight-reduction, results from the belief that lack of knowledge about the calorie value of foods was the limiting factor in weight-control. As time passed, the belief was that knowledge and sufficient motivation to adhere to an appropriate diet, was the essential ingredient in

successfully reducing weight. However, the problem with emphasizing the key role of motivation is, that the patients who are unsuccessful, are judged as having been insufficiently motivated. Sadly, by this criterion it appears that a majority of obese patients are insufficiently motivated to lose weight. VanItallie (1980) challenges this and states that the ability to control one's eating behaviour, does not depend merely on motivation and information about calories; it calls for considerable self-knowledge and the learning of skills and techniques that could help the individual achieve a particular weight level.

In conclusion Garrow (1987) states that the use of a diet in weight-control is but one approach to a very complex problem and that it would continue to be used, even in programmes whose major emphasis is on other issues such as behaviour control or social support.

Total starvation

According to Garrow (1987), this method is used to completely sever the energy intake from food. However, total starvation causes complex metabolic and endocrine reactions, not all of which are favourable. A major responsibility lies with the doctor, because of the dangers involved, of which liver damage, vitamin deficiencies, electrolyte depletion and disturbance of acid-base regulation, increase in uric acid excretion, which could cause gout, and a major loss of lean tissue, are but a few. There is also a serious risk of sudden demise. Garrow (1987) states, that follow up studies indicated that the long-term results of prolonged fasting are also poor, since weight is usually rapidly regained by the patients.

This treatment method is not often used, because of its negative side-effects (Bronkhorst, 1984). However, according to Garrow (1987), the greatest advantage of this method is that it brings about rapid weight-loss accompanied by minor discomfort and is a relatively simple procedure. The desire for food decreases after two or three days of fasting, and

if the environment does not place pressure on the patient, he is able to stick to his new eating pattern. Bray (1976) and Bronkhorst (1984), as well as Garrow (1987) emphasize that it is vital that this procedure be carried out only under medical supervision in a hospital.

Very low-calorie diets

Stunkard (1980) states that the use of very low-calorie diets (about 300 kcal per day - high in protein), with supplemented fasting as a means of rapid weight-loss, has focused attention on the issue of the safety of such diets, related to the risk of serious complications or even of death. The problem with these diets, is that a fat-loss of 0,66 pounds requires an energy deficit of about 2600 calories per day in the absence of any degree of energy intake. It would seem that, in order to achieve this loss, subjects lose significant quantities of body protein. The recommendation of VanItallie (1980) is, that supplemented fasts providing fewer than 800 kcal per day, should be employed with great caution and prescribed only for the very obese under careful supervision by physicians in a research setting.

However, these very low-calorie diets are popular, as these constitute a good way to produce rapid and significant weight-losses. Brownell (1984) states, that from clinical experience, patients find the weight-losses gratifying and adhere more strictly to diets of 400 to 800 calories per day than to diets of 1000 to 1500 calories per day. Patients attribute this to the fact that no food decisions need to be made and that the absence of a palatable diet suppresses their hunger. However, the major drawback of these diets, is the high probability of regaining the weight lost (Garrow, 1978; Wadden et al., 1983); these researchers also suggest, that a comprehensive programme combining a very low-calorie diet (to achieve a large initial weight-loss) with nutrition education, exercise training, and behaviour modification would possibly ensure a better maintenance of weight-loss.

Cook, Howard, and Mills (1981) state that the development of the very low-calorie formula diet provides the clinician with a highly effective tool for the treatment of obesity. Considerable experience via this method shows that it may be safely administered to hospital out-patients and that it does not significantly interfere with normal activities and employment. The results of their study, show that although some patients are mildly depressed at the onset, a very low-calorie diet has mood-elevating rather than mood-lowering properties, which are most prominent in the early phase of treatment (i.e. during the period of maximal weight-loss). The conclusion reached, is that very low-calorie formula diets are highly effective means of achieving significant weight-loss in selected obese patients, and that it may be safely employed (Cook et al., 1981).

Wadden et al., (1983) refer to the very important aspect of patient selection in the use of the very low-calorie diet in the treatment of obesity. The use of the very low-calorie diet, is usually limited to adults (ages 18 to 70) who are moderately (40% to 100%) or morbidly (100% or more) overweight. Wadden et al., (1983), emphasize that patients must be screened by using history and physical examination, electrocardiogram, complete blood count, SMA-20 (including electrolytes), fasting glucose and blood urea nitrogen, as well as complete urine analysis. Contra-indications to treatment via very low-calorie diets, include a recent myocardial infraction, cerebro-vascular disease, cancer, type I diabetes, hepatic disease, renal failure and overt psychosis. They furthermore caution that, in the event of pregnancy, the diet must be terminated immediately.

Conventional Reducing Diets

Garrow (1978) states that the conventional dietary management of obesity is, to advise the patient to eat normal food, but to reduce the quantities of certain foods, especially those of high-energy density. The objective is to reduce the energy intake below the normal level of energy expenditure, so that the deficit is made up by burning the

energy stores of the body (chiefly fat). According to Garrow (1978) and Munves (1980) the advantages are, that this method is both relatively safe with regards to any possible dietary deficiencies and inexpensive, as foods used, consist of those the patient would have in the home. However, it must be emphasized, that the effectiveness of conventional low-carbohydrate diets depend on the total energy intake being reduced - i.e. one could eat fat and grow slim, but only when in the process one also absorbs fewer calories. The conclusion is, therefore, that the diet should create an energy deficiency (but not a deficiency of any essential nutrient) and that it should be well-tolerated (Garrow, 1987).

Calorie Counting

The first requirement of a reducing diet, is that it should be low in energy (calories or joules). Garrow (1987) states that the logical way to design such a diet is, by summing up the energy provided by each food item to the desired limit of, say, 1 000 kcal. However, calorie-counting requires careful monitoring of portion sizes and could be difficult and tedious if one consumes a variety of foods (Garrow, 1987). In contrast to the assumption that obese persons exhibit an orderly pattern of eating and simply eat in excess at certain meals, there is growing evidence that many obese individuals have a poorly-structured eating pattern, with some meals skipped, many snacks consumed and occasional food-binges. This kind of eating pattern does not lend itself positively to calorie-counting.

However, VanItallie (1980) states that the availability of homogeneous liquid diets that are nutritionally complete, and packaged in units, could make calorie-counting very simple. The exclusive use of such diets does not expose the obese patient to the temptations associated with the preparation and ingestion of normal foods.

Other diets

The carbohydrate-restricted diet in which the intake of bread, cereals, sugar and preserves is restricted, was reviewed by Garrow (1987). He found no evidence that this diet has any fat-mobilizing properties in excess of that which would be expected from its calorie content.

With regards to the fat-restricted diet, Garrow (1987) states that it is not prudent to advise people to avoid fat, as if this would be the sole strategy for dieting, since much fat comes from meat and dairy produce; therefore, a total ban on fat would involve losing other important nutrients like protein and calcium. Garrow (1987) suggests that an alternative method would be to advise people to consume large quantities of high-fibre foods.

According to Garrow (1987), the high-protein diet actually incorporates the same operative principles as low-carbohydrate and low-fat diets, since both carbohydrates and fats are restricted. The only other source of energy, therefore, is protein. Unfortunately, the high-protein designation may mislead people into believing that the more protein they eat, the more weight they will lose.

Manipulation of dietary constituents

This method includes diets that do not specify calorie control but manipulates dietary constituents in such a way, that energy intake may be reduced spontaneously. An attempt is made towards facilitating the controlling of energy intake for the dieter, by simply prohibiting certain dietary items or classes of food (Garrow, 1978). VanItallie (1980) reports that this method is particularly successful when all alcoholic drinks are prohibited for persons who obtain as much as 25 per cent of their daily calorie needs from ethanol. The removal of this energy source from the diet (if not replaced by other foods), could result in weight-loss or the more effective maintenance of weight.

According to Sclafani (1980), diets which limit the obese

person to just a few foods that may be palatable in the context of a normal diet, but which soon lose their capacity to arouse the appetite when they are eaten repeatedly, also inhibit the energy-intake of obese persons. Examples of this type of diet are grapefruit diets, the milk-and-banana diets and ice-cream diets.

Impairment of intestinal absorption

With the intention of increasing weight-loss or preventing weight-gain, diets which prescribe foods or food-like products that are poorly absorbed from the gut lumen and, therefore, impair intestinal absorption, are used (VanItallie, 1980). The notion exists, that it might be possible to prevent or treat obesity by ingesting palatable foods or food-like products that are poorly digested or absorbed. However, VanItallie (1980) states that to date no successful developments have been reported as reliable methods for weight-control; the price to be paid, is intestinal distress, loss of essential nutrients and inconvenient changes in bowel habits. The use of fibre-rich foods or the addition of fibre to various food products is another way of reducing the digestibility coefficient of what is being consumed (Garrow, 1987). However, more research is needed concerning the amount and type of fibre that should be present in the diet.

Excretion of calories

This is brought about by diets that promote the excretion of incompletely metabolized substrates such as ketone bodies in the urine (Garrow, 1978; Garrow & Warwick, 1978). VanItallie (1980) states that, although ketogenic regimens increase the excretion in the urine of incompletely oxidized substrates (principally ketone bodies), the amount of extra energy that could be lost via this route during any 24-hour period, is very limited and there does not appear to be any diet, however drastic, that could appreciably increase the rate of fat-loss in this fashion.

In conclusion

The excess weight of moderately obese persons, sets the primary goal of dietary treatment as the establishment of the largest calorie deficits, but a large calorie deficit is limited in terms of the patient's safety and comfort. Conventional reducing diets of 1 200 to 1 500 calories are safe and comfortable but produce too slow a weight-loss for these to be practical for many moderately obese persons. According to Stunkard (1980), a very low-calorie diet of 400 to 700 calories, consisting largely or exclusively of protein, appears to be safe when administered under careful medical supervision for periods of up to 3 months and this diet achieves striking weight-losses. Stunkard (1980) states, that the problem remains in the maintenance of the lower-weight level. He concludes that the treatment of moderate obesity is the most problematic and that one must either use diets that produce good weight-loss and poor maintenance of the lower-weight level, or elicit behaviour modification that produces good maintenance of the lower-weight level, but relatively poor weight-loss. A possibility would be the combination of both.

According to Munves (1980) a diet for weight-loss could be effective if it were adapted to the individual life style of each patient. This implies, that the diet should be planned so as to create a calorie deficit, at the same time providing adequate nutrition to meet the short-term and long-term weight-loss goals of the patient. Munves (1980) stresses that the individual needs to recognize that his diet for weight-loss is as important as any other medical prescription he may have. A long-term commitment on the part of both the therapist and the patient, could result in successful dietary management.

Garrow (1987) found that the poor results of dietary treatment of obesity, apply especially to outpatient regimes. He found that, given time and complete control of the diet in a metabolic ward, virtually any amount of weight could be lost by dietary methods, but that success depends on the priority which the patient gives to adhering

to a reducing diet; and that this depends on the balance established between the inconvenience of the diet and the benefit which the patient believes weight-loss will bring.

The pharmaceutical treatment of obesity

Haskew and Adams (1984) report that the pharmaceutical treatment of obesity is by far the most appealing route to thinness, because it offers an effortless way to weight-loss. Its association with medicine implies that a cure is being accomplished. Unfortunately, most potent drugs in use, are available by prescription or via pharmacy counters. Amphetamines promote modest temporary weight-loss. However, Haskew and Adams (1984) caution that the continued use of this medication fails to produce further losses and that the long-term use of these drugs is dangerous. In addition, termination of treatment is followed by weight-gain. Although the initial use of amphetamines in diets reduces appetite and may increase energy expenditure, years of research on animals and humans have not fully explained the action of amphetamines. Haskew and Adams (1984) report that, as amphetamines induce conditions in the body that mimic a state of alarm or arousal, it is possible that they may inhibit the digestive functions, causing the body to use fat rather than food for energy. It is also possible that some of the anorexic effect of these drugs, is a consequence of their inhibition of the salivary glands, which creates a dry mouth, makes food less palatable and results in a loss of appetite. This loss of appetite is only temporary as the body draws on its immense recuperative powers, adapts to the chemical, and restores digestion, salivation and appetite back to normal. Stunkard (1987) supports the findings of Haskew and Adams (1984) and states that it is possible that appetite reduction is a side-effect of the use of amphetamines and not the principal action which would explain why the effect is short-lived. He suggests that amphetamines may work by resetting the body's preferred weight, or set-point, to a slightly lower level. However, once the new lower-weight is achieved, the appetite is restored to normal. If this is the way they work, further weight-loss could be achieved only by increasing dosage, and

this raises the hazard of an ever-greater dependence on chemicals.

Findings by Garrow (1978) indicate that patients who have used an anorectic drug, do worse in maintaining weight-loss than those who have never used it. It seems that the best an anorectic drug could do is to remove hunger, which is only one of many reasons why patients default on reducing diets.

In comparison to anorectic drugs, thermogenic drugs are used to increase energy expenditure, but Garrow (1978) reports that they are ineffective in producing weight-loss in non-toxic doses, unless the diet should also be strictly controlled. The danger existing here, is that the use of high doses is associated with large losses of lean tissue.

Garrow (1978) comments on the common use of diuretics to produce weight-loss in obese patients and states it is ineffective and that it deflects the patient's attention from the fundamental problems of obesity and its relationship to energy balance. According to Lasagna (1980), the use of diuretics is rarely indicated on medical grounds and he warns that their use could be dangerous to patients on low-calorie diets, since the diuretics might aggravate the loss of electrolytes.

Blundell (1980) states that if it were possible to positively identify the cause of human obesity within the biological system, drugs could be specifically designed to counteract the malfunction and to rectify it by acting directly on the causal agent. However, it is evident that human obesity does not arise from a single causal factor, and certain conditions important for the development of obesity, may lie beyond the generally recognized boundaries of a physiological system, for example external constraints, long-term buying habits, preparation and consumption of specific foods. Blundell (1980) advises obese patients to use will-power, rather than drugs. This advice is based on the apparently small advantage to weight-loss resulting from the use of a drug. He suggests that drugs could be administered in conjunction with techniques of

behaviour therapy designed to deal with the demands of the psychological and social environment and to provide control over internal factors and the external milieu. Blundell (1980) concludes, that drugs could be used as tools to enable patients to achieve an increased awareness of the consequences of their eating behaviour, as well as of assisting them to better understand the forces - biological and psychological - which control their lives.

Bronkhorst (1984) and Oelofse (1984) both regard chemotherapy as not being a very successful method of treating obesity, and state that the majority of appetite suppressors contain amphetamine or fenfluramine, both of which are habit-forming; and even when they are combined with a low-calorie diet, they are only effective for a short period. They add, that even this slight positive effect is overshadowed by the danger of addiction.

In support of the above, Silverstone (1986) reports that an extensive review of outpatient management of obesity reveals that drug therapy fails to produce weight-losses in excess of those achieved by other means. The results support the contention that none of the currently available drugs produce a lasting effect on eating habits, and that the weight-loss that they induce, is only temporary. In conclusion, he states that it is clear that appetite suppressant drugs do help obese patients to adhere to a calorie-restricted diet and that drugs could play a therapeutically useful role in the clinical management of obesity. However, with regards to the rebound effect of using drugs in the treatment of obesity, Simpson, Abernethy, and Munro (1987) state that there is absolutely no evidence that the administration of anorectic agents would help to retrain previous faulty eating habits. Unless some method could be devised to prevent weight regain, drug therapy could be justified only if there were a clearly defined, short-term need for weight-loss. It is important to note that anorectic drugs form only a part of the overall management of the obese, and their widespread use cannot be justified (Simpson et al., 1987).

Exercise in the treatment of obesity

Wirth (1987) states that it is general practice to advise obese patients to take more exercise, in addition to reducing food intake. In principle, body-weight could be reduced by increasing energy expenditure, thereby inducing a negative energy balance. However, Wirth (1987) supports findings by Brownell and Stunkard (1980) that exercise cannot bring about effective weight-reduction on its own and should primarily be seen as an important part of the maintenance of weight-loss.

Thompson et al's., (1982) extensive study on exercise and obesity sets forth the following conclusions -

- (a) because of methodological limitations of past research, the role of activity-level versus calorie-intake as an etiological factor in the development of obesity is not clear,
- (b) tentative evidence suggests that exercise in conjunction with other treatments, produces greater weight-loss than do single intervention procedures,
- (c) the effects of exercise on calorie intake, metabolic rate and body compositions, have a significant effect on energy expenditure,
- (d) a combination of routine and programmed activities may be optimal for weight-control.

It has been reported that an increase or decrease in exercise has no effect on food intake behaviour. According to Stern (1984) and Wirth (1987), this is because of differences in the sex and age of the individuals, as well as the duration and intensity of the exercise training and the type of exercise. However, caution is advised, as findings of studies cited to encourage sedentary obese individuals to increase their activity, in the hope of decreasing their food intake, are not universally recognized.

Stern (1984) states, that the psychological benefits associated with exercise, are feelings of well-being and even of euphoria. He finds that in some distressed

individuals or in those who are anxious or physically unfit, exercise-training is associated with improvements of mood. When combined with a weight-reduction programme, exercise is associated with a shift toward a more internal locus of control or feelings of self-control regarding one's life.

Haskew and Adams (1984), as well as Brownell (1982), state that exercise helps reverse the effects of poor diet. It appears that obese people with slow metabolisms and overeaters with disordered appetites, could experience an easier relationship with food if they maintained suitable activity. According to Lampman, Schteingart, and Foss (1986) physical exercise may also be advantageous in facilitating adherence to a weight-reduction programme and for aiding in the long-term maintenance of weight-reduction.

Lampman et al., (1986), as well as Wirth (1987), conclude that, together with positive changes in psychological and social behaviour, the adaptations on a physiological level, brought about by intensive exercise, considerably increase the well-being of obese patients.

Surgical treatment of obesity

Haskew and Adams (1984) state that there are three forms of surgical treatment for the purpose of weight-loss: cosmetic, gastric and oral. In cosmetic surgery, unwanted adipose tissue is cut from the body or removed via liposuction. Gastric intervention (stomach and intestinal surgery) is limited to grossly obese patients and aims at reducing digestive efficiency by shortening the small intestine or by reducing the effective size of the stomach, creating a state of semi-starvation and corresponding weight-loss. Jaw-wiring, a dental procedure, causes weight-losses, as the semi-liquid diet that must be consumed, is unlikely to contain the calories that will maintain weight.

According to Garrow (1978), surgical treatment of obesity is never justified, until at least a rudimentary study of energy balance in the patient has been carried out. Numerous researchers, (Gries, 1987; Kral & Kissileff, 1987;

Pasulka, Bistran, Benotti, & Blackburn, 1986), warn that surgical treatment for obesity is a serious matter, exacerbated by the fact that obese patients are often poor surgical risks.

Both Oelofse (1984) and Bronkhorst (1984) state that surgical intervention is regarded as radical therapy, and as a result of the possibility of long term health risks, it should be carried out only in extreme circumstances.

In conclusion, Stunkard, Stinnett, and Smoller (1986) emphasizes that surgery for obesity should not be entered into lightly and should never be done for cosmetic reasons or for obesity of less than great severity. They stress that informed consent is essential and that patients should be informed about the potential risks, as well as of the benefits of surgery. Stunkard et al., (1986) also recommend that, whenever possible, patients should meet with other patients who have undergone both successful and unsuccessful operations. Finally, patients should realize that a drastic change in eating habits would be necessary and that they should be committed to losing weight.

Acupuncture

Acupuncture is the Chinese art of inserting needles at special points on the body for pain relief. The belief is, that there is a feeding center, located in the region of the ear, which can be manipulated by acupuncture. However, there is no evidence that this method of treatment of obesity has any value (Bray and Teague, 1980).

2.5.3 Summary of the treatment of obesity

Stunkard (1984) concludes, that the most effective treatment for severe obesity (more than 100% overweight) is surgical measures, particularly ones that reduce the size of the stomach and that of its opening into the lower gastrointestinal tract. The reason is that this surgery may produce very large weight-losses, with relatively few untoward consequences.

However, moderate obesity (40 to 100% overweight) is treated medically by using diets that often result in satisfactory weight-loss, but a poor maintenance of this loss, or by behaviour modification that achieves good maintenance, but only modest weight-loss. Stunkard (1987) states, that pharmaceutical treatment of obesity appears to lower the body-weight set-point, but that cessation of the use of drugs is followed by a rapid regain of lost weight and for this reason he suggests that drugs should probably be used either for a definite period or not at all.

With regards to mild obesity (20 to 40% overweight), its treatment is mainly covered by both commercial and non-profit organizations (Stunkard, 1987). The basis of treatment is behaviour modification in groups, a liberal, balanced diet and exercise. Stunkard (1987) reports, that in spite of extremely high drop-out rates from these programmes, their low costs result in favourable cost-effective ratios and they are continuing to increase the number of obese people they treat.

Bronkhorst (1984) advises caution when prescribing treatment for obesity. She refers to Arieti's (1975) statement, that it must always be remembered that obesity is a chronic condition in which resistance to the changes required is common and in which attrition is a general occurrence.

In summary, Bronkhorst (1984) states that -

- (a) attention should also be paid to the psychological well-being of the patient and not only to reducing food intake and increasing activity,
- (b) during therapy, the individual's total functioning must be considered. This implies that the individual's physical, psychological and environmental or family functioning are considered. She stresses that this is the only way of ensuring that a treatment programme would be successful.

2.6 DEPRESSION

In this section literature on theories, as well as the etiology and symptoms of depression are reviewed. However, in order to provide a holistic view, both psychological and physiological factors are reviewed.

Introduction

The word depression is used in everyday language and is understood to refer to a feeling, a reaction to a situation or an individual's characteristic behaviour pattern (Sarason & Sarason, 1984). Illness, personal frustration or failure, bad weather, holidays, moving to a new home or having a baby, are all factors which could cause depression. However, this depression is only a temporary, non-incapacitating feeling that usually fades when the situation changes or the individual adjusts to the new circumstances (Boyd & Weissman, 1982; Sarason & Sarason, 1984).

Carson, Butcher, and Coleman (1988) discuss the depression which follows the death of a family member or the end of a love affair. They state that it is normal to experience grief and that the physical (sighing, tightness of the throat, an empty feeling in the abdomen and a feeling of muscular weakness) and psychological (feelings of loss, guilt and hostility) symptoms gradually disappear as time passes. However, Sarason and Sarason (1984) caution, that feelings of depression have been experienced by the majority of individuals and that they do not necessarily indicate that the individual is suffering from a mood disorder. They emphasize, that clinical depression is a more serious and long-lasting problem.

Both Carson et al., (1988) and Paykel (1982) stress that depression is classed as a mood disorder when the mood disturbance becomes central and possibly also includes anxiety. The American Psychiatric Association [APA] (1987), describes a mood disorder as a disturbance of mood, which is accompanied by a partial/full manic or depressive syndrome. The American Psychiatric Association [APA] (1987), defines

mood as a prolonged emotion which affects the individual's total psychic life and involves both states of depression or elation. Mood disorders are divided into bipolar (one or more Manic/Hypomanic episodes, plus a history of major depressive episodes) and depressive (one or more periods of depression, but with no history of manic or hypomanic episodes).

Carson et al., (1988) state that psychologists and mental health specialists only conclude that a patient is suffering from a mood disorder when the mood change is so intense, that it seriously endangers the patient's welfare.

2.6.1 THEORIES ON DEPRESSION

Sarason and Sarason (1984) report, that the cause of depression has already been examined from many of the major theoretical viewpoints. At present the popular view is, that there are distinct groups of depressives, whose symptoms have different origins; for example, the bipolar type is thought to be caused by genetic factors, while major depression is thought to have two causal subgroupings. These subgroupings are: (a) inability to cope with difficult life events or (b) an organic malfunction (neural transmission problems, because of the unavailability of enough catecholamines or chemical transmitters).

The present study reviews the following five schools of thought as proposed by various researchers:

- (a) The Psychoanalytic Theory, which attributes depression to the loss of an ambivalently-loved object or the reaction to separation from an important loved one, which causes the interjection of hostility resulting in depression (Sarason & Sarason, 1984).
- (b) The Learning Theory which conceptualizes depression as being the consequence of learned helplessness and/or a reduction in positive reinforcement from the environment (Lewinsohn, Munoz, Youngren, & Zeiss, 1978; Seligman, 1975).

- (c) The Cognitive Theory, which states that the individual's negative and distorted cognitions of his world, of the future and of himself, cause depression (Beck, 1979; Beck, Rush, Shaw, & Emery, 1979).
- (d) The Humanistic-existential Theory, which holds that depression is caused by a loss of self-esteem, which in its turn results in too great a difference between the ideal self and the real self that is to be tolerated (Sarason & Sarason, 1984).
- (e) The Biological Theory, which proposes, that depression is caused by a physiological malfunction (deficiency of catecholamines) and/or inherited genes (Sarason & Sarason, 1984).

Psychoanalytic Theory

Sarason and Sarason (1984) report that the psychological study of depression was initiated respectively by Abrahamson in 1911 and Freud in 1917. Freud's major work "Mourning and Melancholia" describes both normal mourning and melancholia or depression as being a response to a loss (i.e. of someone or something). However, Freud in 1917 states, that the melancholic experiences a decrease in self-regard and of the ego. It is interesting to note that depression was the first disorder Freud ascribed the cause to emotions and not to sexual desires. Freud views depression (or melancholy) as constituting disproportionate grief (drawn-out, unrelated to the environment and unwarranted). According to Mendelson (1982), Freud considered depressives as having strong punitive consciences or superegos, which causes guilt. Freud proposes, that the conscious becomes so strong because of the effort the individual exerts in an attempt to control his anger and aggressive feelings which, if expressed, could hurt others (Mendelson, 1982).

Sarason and Sarason (1984) further state that psychoanalytic theories stress both unconscious feelings and the individual's reactions (a consequence of early life

experiences) to a new situation. The majority of these theories originate from the study of the relationships between the individual and the person (i.e. the mother) he depended on as a child. The conclusion, is that a disturbance (actual, feared or fantasized loss) during early-childhood relationships, causes depression. Although this (real or imaginary) loss is suppressed, it continues to exert an influence and culminates in depression, when a symbolically significant event occurs. According to Sarason and Sarason (1984) psychoanalytical theorists also propose that depression in later life could be associated with demanding parents, who withheld love as soon as a child acted contrary to their will. The result is a model child who internalizes anger in the form of guilt feelings. This suppression of anger to the subconscious, uses a lot of psychic energy; the consequence is, that depression is likely to occur at some time in the individual's life.

Mendelson (1982) describes how, beginning with the original formulations of Abraham in 1911 and Freud in 1917 as well as those of later researchers (Bibring in 1965; Gero in 1936; Jacobson in 1953 and Rado in 1928), a failure in the interaction between the mother and child (i.e. lack of affective mother responses, disappointment, absences, traumatic occurrences and separation in early childhood,) results in an impairment in the child's ability to deal with relationships. It appears that the individual with a predisposition to affective disorders and who sustains a narcissistic injury in early childhood (usually during the separation-individuation stage of Mahler), experiences disrupted emotional ties, consequently replacing the original love for the parental image with ambivalent feelings (Mendelson, 1982).

Stein (1982) and Whybrow, Akiskal, and McKinney (1984) describe the resulting attempt at restitution when, in order to overcome the pain caused by the loss, the child turns back to the parent and tries to obtain the narcissistic supplies and to restore his self-esteem. This introjection, full of violent rage, is very ambivalent and leads to a narcissistic regression in the relationship between the

child-parent. These researchers state, that the struggle with the feelings related to the fantasized incorporation of the lost object causes the depressive reaction.

The above description represents only a minute part of the dynamics which operate in susceptible individuals, who have developed a pattern of depressive reactions to narcissistic injury (Mendelson, 1982). The important elements include a narcissistic regression from the object, guilt, a masochistic reaction which involves the infliction of self-punishment of the ego by the superego, loss of self-esteem, ambivalence and introjection (Mendelson, 1982; Stein, 1982).

Davidson and Neale (1982) stress that Freud's explanation, that the ego becomes powerless while the superego becomes omnipotent during depressive reactions is useful, also that this struggle is characteristic of depression.

However, Whybrow et al., (1984), suggest that it is important to note the shift of emphasis to the role of the ego in mental functioning and adaptation, is a recent development in psychoanalytic theory. This ego-psychological perspective makes allowance for an active interchange between the mind and the ego, based on the role of the ego as the conceptual interface between the self and the world. Increasing weight has been given in intrapsychic formulations of depression to the importance of actual or symbolic loss or injury to the ego which occurs during the individual's complex interaction with conjugal, familial, societal and cultural forces.

Learning Theory of Depression

In providing an explanation of the learning theory of depression, Vlismas (1985) emphasizes proximate events of interpersonal or situational nature, while Whybrow et al., (1984) state that learning models of depression focus on characteristics of the immediate environment of the depressed individual.

However, various researchers (Davidson & Neale, 1982; Lewinsohn & Hoberman, 1982 and Lewinsohn et al., 1978) propose that the learning theory puts forward two views explaining depression, i.e. that a reduction in reinforcement or learned helplessness causes depression. Lewinsohn and co-researchers (1978; 1982), suggest that when accustomed reinforcements are cut off or reduced (e.g. moving to a new home or job changes), depression is the result. They state, that there is a reduction in activity which is further aggravated by the low rate of positive reinforcement or reward received by the individual. A further result, according to Davidson and Neale (1982) is, that both social participation and rewards decrease in a vicious circle. Both Davidson and Neale (1982) and Lewinsohn et al., (1978) state, that the depressives themselves start believing that they have little control over their lives and that consequently their behaviours have little effect on the outcome of events.

An in-depth discussion of this theory follows.

- Reduction in reinforcement

Lewinsohn and co-researchers (1978; 1982) noncognitive low-reinforcement reduced activity view of depression, assume that inactivity and a depressed mood are direct results of the individual's environment. This view proposes, that an individual becomes depressed as a result of reduced activity, which leads to reduced positive reinforcement. Their view is, that the absence of reinforcement plays a major role in the development and persistence of depression. Reinforcement is defined by Lewinsohn et al., (1978) as being the rate and quality of an individual's interaction with his environment.

Sarason and Sarason (1984) conclude, that learning theorists assume that depression and reduction in reinforcement are related, at the same time asserting that many theorists who hold the learning view of depression have been influenced by Skinner's work in operant conditioning. Skinner's work emphasizes social interaction and specifically the

reinforcement behaviour of others. In addition, Lewinsohn and his co-researchers (1978; 1982) suggest that the low level of behavioural output and feelings of sadness/unhappiness associated with depression, are caused by the low rate of positive reinforcement and/or high rate of unpleasant experiences. Sarason and Sarason (1984) propose the following possible causes:

- (a) the individual's environment could consist of too few reinforcements or too many punitive factors,
- (b) a lack of social skills in the individual, which means he is unable to attract positive reinforcement or to cope with unpleasant events,
- (c) individuals prone to depression, experience reinforcements as being less positive and punishments as being more negative, which results in withdrawal behaviour, consequently again leading to fewer reinforcements or more punishments.

Lewinsohn and Hoberman (1982) suggest, that depressed people experience more unpleasant events in their lives than do non-depressed people. They propose, that it is possible that this could result in increased withdrawal behaviour, which further limits the depressed individual's chances for reinforcement. Lewinsohn and Hoberman (1982) stress, that this is important, as there is a relationship between positive reinforcement and severity of depression. Sarason and Sarason (1984) agree with the above researchers, and suggest that, because people find depressed people unpleasant to be with, a decreased rate of reinforcement would result which in turn, aggravates the depression.

- Learned helplessness

Martin Seligman (1975) proposes that depression is the result of learned helplessness; he suggests that, although anxiety is the initial response to a stressful situation, it is replaced by depression when the individual believes that it is not possible to control the situation. According to Davidson and Neale (1982), Seligman's (1975) formulation of learned helplessness, focuses on the process during which

the individual perceives a relationship between activity and outcome. The individual then deduces that he is helpless and that all efforts to change the situation, are in vain. Stonestreet (1985), as well as Whybrow et al., (1984), states that Seligman (1975; 1979) maintains, that a lack of assertiveness, passivity and resignation to fate, are learned (i.e. from past experiences when the individual was unable to manipulate his behaviour to change an unpleasant situation).

In response to criticisms of the learned helplessness theory of 1975, Lewinsohn and Hoberman (1982) state, that Seligman (1979) re-formulated his theory by incorporating extensions from the attribution theory. This theory suggests that the individual's future expectations of not being able to control a situation, are founded on perceived helplessness in the present situation. Particularly relevant to learned helplessness and depression, are dimensions such as internality-externality, globality-specificity and stability-instability. Seligman (1979), hypothesizes that an individual is more likely to be depressed if his attributions for success are external, unstable and specific; while for failure and lack of control, the attributes are internal, stable and global. Seligman (1979) further hypothesizes, that low self-esteem found in depression, results from internal attributions of personal helplessness while depression itself is the result of the belief that the future will be fatalistically dominated. The certainty, that there will be only aversive outcomes in the future, and the belief that the individual would be unable to control these, determine the severity of the motivational and cognitive deficits of depression. Seligman (1979) concludes, that the importance attached to uncontrollable outcomes, governs the severity of self-esteem and affective deficits.

The Cognitive Theory of Depression

Sarason and Sarason (1984) conclude, that the most influential psychological theories of depression are those based on cognitive factors. They explain that the cognitive

theories suggest, that people react differently to the same experience because of their differences in cognitions i.e. how they think about an event.

Beck et al., (1979) states, that the cognitive model is based on the theoretical rationale, that the way an individual structures his world, determines his affect and behaviour. The individual's existing attitudes or assumptions (schema), based on previous experience, determine his cognitions (verbal or pictorial events in his consciousness). Davidson and Neale (1982) suggest that Beck (1967) is the most important contemporary theorist, who views thought processes as constituting a causative factor in depression. Beck's (1967) central thesis is that depressed individuals commit characteristic logical errors, i.e. they distort what happens to them into self-blame and catastrophes, consequently making them feel as they do. Du Plessis (1984) summarizes Beck's (1967) cognitive model and states that depressive individuals reach illogical conclusions when evaluating themselves, their world and their prospects for the future.

Lewinsohn and Hoberman (1982) support findings by Beck (1979; 1967) and conclude that the signs and symptoms of the depressive syndrome are the result of the individual's negative cognitive (thought) patterns.

Beck et al., (1979) report, that the cognitive theory of depression evolved from experimental testing and systematic clinical observations, which in turn have led to progressive development of theory and psychotherapy. They state, that the cognitive model postulates three specific concepts (cognitive triad, schema and cognitive errors i.e. faulty information processing) within the psychological basis of depression.

- The Cognitive Triad

Beck et al., (1979) state that the cognitive triad consists of three major cognitive patterns which cause the individual to view himself, his experiences and his failure, in an

idiosyncratic manner.

Beck et al., (1979) stresses, that in respect of the first component, i.e. the individual's negative view, the individual sees himself as being deprived, diseased, inadequate and disruptive. Because he attributes his unpleasant experience to a psychological, moral or physical defect in himself, he believes that he is worthless and undesirable. Sadly, he also believes that he lacks the qualities he thinks are essential for him to achieve contentment and happiness.

Beck et al., (1979) explains the second component of the cognitive triad as being related to the depressed person's tendency to interpret all his experiences negatively. Because he feels the world makes exorbitant demands on him, thus creating insuperable obstacles preventing him from reaching his goals, he interprets his interactions with his animate or inanimate environment as being representative of defeat or depravation, even should more positive alternative interpretations be possible. Lewinsohn and Hoberman (1982) suggest, that if the depressed individual could be persuaded to reflect on less negative alternatives, he might come to realize that he has tailored the facts to fit his preformed negative conclusions.

Both Beck et al., (1979) and Du Plessis (1984) discuss the third component, i.e. a negative futuristic view. It appears, that when the depressed individual made long-range projections, he believed his present difficulties and suffering would continue indefinitely. His future expectations are those of hardship, deprivation and frustration; he consequently expects to fail in everything he attempts.

Davidson and Neale (1982) stress the fact that the cognitive model regards symptoms of the depressive syndrome to be the result of negative cognitive patterns which include the erroneous belief that the individual is being rejected or that he is a social outcast. The result of this, is that the individual acts as though this were, in fact, true.

Beck (1979) explains the motivational symptoms (paralysis of will, escape and avoidance wishes) as being caused by these negative cognitions. Beck et al., (1979) state, that the danger is that the individual's pessimism, hopelessness and expectation of a negative outcome, implies that he will not commit himself to a goal. The result is, that he sees himself as being worthless, which in turn leads to the formation of suicidal wishes in his attempt to escape from what appears to be an insoluble problem. Unfortunately he then comes to believe that everyone, including himself, would be better off if he were dead.

Beck et al., (1979) views the depressive individual's increased dependency on others as being understandable. The individual tends to seek help and reassurance from other supposedly more competent and capable individuals, because he sees himself as being inept and helpless. They state that psychomotor inhibition (a physical symptom) may also be explained by the cognitive model, because both the belief that he is doomed to failure and a sense of futility, might lead to apathy and low energy.

- Schemas

The cognitive model's second major ingredient consists of the concept of schemas, which explain why, in spite of evidence to the contrary, the depressed individual continues with his pain-inducing and self-defeating attitudes (Beck et al., 1979). The depressed individual tends to be consistent in the manner he conceptualizes a situation. Beck et al., (1979) uses the term schema to describe these stable cognitive patterns. They state, that schemas form the basis the individual uses for differentiating, coding and the screening out of stimuli. Beck (1979) and Beck et al., (1979) suggest, that the individual categorizes and evaluates his experiences via a matrix of schemas.

Beck et al., (1979) as well as Lewinsohn and Hoberman (1982), suggest that the kinds of schema used, determines in what way the individual structures different experiences and, therefore, how he reacts. These researchers maintain,

that in psychopathological states such as depression, the individual's conceptualizations of situations are distorted so as to match their dysfunctional schemas. Unfortunately, as these idiosyncratic schemas become more active, they are more easily evoked by less logically related stimuli. The result is, with the worsening of depression, the individual often loses control of his thought process and is unable to apply more appropriate schemas. The idiosyncratic schema leads to distortions of reality, systematic errors in thinking; the individual consequently is no longer able to consider the idea that his negative interpretations are erroneous. In severe states of depression, the individual may be completely dominated by idiosyncratic schemas and/or preoccupied with preservative, repetitive and negative thoughts, consequently finding it difficult to read, answer questions or to engage in problem-solving or computations (voluntary mental activity). Beck (1979) and Beck et al., (1979) infer, that the individual is unresponsive to changes in his immediate environment, because his cognitive organization becomes independent of external stimulation.

- Cognitive Errors (Faulty Information Processing)

Beck et al., (1979) and Lewinsohn and Hoberman (1982) also infer, that the increased domination of dysfunctional schema results in systematic errors in the depressives logic thinking. These automatic and involuntary errors include: selective abstraction (ignoring the context of an event by fixating on a detailed aspect of a situation and ignoring more salient features); overgeneralization (drawing a general conclusion, based on limited facts); arbitrary inference (drawing unsubstantiated or contradictory conclusions); personalization (attaching unsupported subjective significance to external events); magnifying or minimizing (exaggerating or limiting importance of information) and absolutistic thinking (all experiences are placed into one or two opposite categories). The above researchers conclude that these systematic errors in the depressed individual's thinking, maintain his belief in the validity of his negative concepts, despite contradictory evidence.

Beck (1979) suggests, that viewing this conceptualizing in terms of primitive versus mature modes of organizing reality, would be a way of understanding the thinking disorder in depression. He maintains, that depressed individuals are prone to structure their experiences in relatively primitive ways and that they tend to make broad global, extreme, negative, categorical and absolute judgements of events that make their lives difficult. Beck et al., (1979) subsequently proposes that, in primitive thinking the complexity, variability and diversity of human experiences and behaviour are reduced to a few crude categories; this results in depressive individuals' viewing their experiences as being defeats (non-dimensional), irreversible (fixed); they consequently categorize themselves as being both ill-fated (irreversible character deficits) and losers (categorical, judgemental).

To summarize, the following are stated;

- (a) the expectations of depressives' are irrational. They supposedly expect more punishments and fewer rewards than are realistic (Beck, 1979).
- (b) depressives have distorted perceptions of their environment. They are likely to distort performance feedback, filter out positive aspects and inflate negative aspects of the environment (Beck, 1979).
- (c) depressives are unrealistic in their self-monitoring. Beck (1979) implies, that depressives underestimate the frequency of their successes and overestimate their failures.
- (d) depressives suffer from distorted memories. Beck (1979) infers, that they forget previous rewards and exaggerate the frequency of past punishments.
- (e) depressives are irrational with respect to their causal attributions and they exaggerate their roles as failures (Beck, 1979).

However, in contrast to the above, Layne (1983) proposes that contrary to previous findings, empirical research indicates that depressives are cognitively realistic, while non-depressives are cognitively distorted. This supports the findings of Lewinsohn and Hoberman (1982), that non-

depressives distort their perceptions, while depressives are quite accurate in their perceptions. Layne (1983) concludes, that cognitive theories of depression appear to be erroneous in their assertion that cognitions of depressives' are distorted. Layne's (1983), study finds, that empirical literature supports the cognitive theories assertion, that depressives think less optimistically and in less self-serving ways than do normals. However, the major implication of the empirical literature is, that the thoughts of depressives' are painfully truthful, whereas the thoughts of non-depressives are unrealistically positive; in addition, it is more likely that painful truthful cognitions, perhaps in concert with other cognitive deviations, cause depression, and that defenselessness may be the crux of depressive realism.

Humanistic-Existential Perspective on Depression

Davidson and Neale (1982) state that the human-existentialistic theory focuses on the loss of self-esteem as the central cause of depression. This loss could be real or symbolic (power, social rank, money) and is only important, in that it causes a change in the individual's self-assessment, based on their positions or possessions. This offers an external verification of people's worth in their own minds. It appears that, in our own culture a frequent cause of depression for men, is the loss of a job, which represents his sense of worth. Before females joined the work force, depression in females was attributed to the loss of a spouse, who was not only both a loved one and a provider of financial security, but also a source of prestige.

According to Carson et al., (1988) humanistic-existential theorists focus on the difference between an individual's ideal self and his perception of the actual state of affairs. Sarason and Sarason (1984) infer that depression is, therefore, likely to occur when the perceived difference between the real and the ideal self becomes too great to tolerate. The central idea is, that the loss of self-esteem and a perceived inability to change the oppressive

demands of society, are the cause of depression.

The Biological Theory

Sarason and Sarason (1984) state, that biological theories assume, that a physiological malfunction (possibly inherited) or genes inherited from parents, are the cause of depression. They refer to studies of twins dating from the 1930's of which findings suggest the appearance of hereditary elements in cases of depression. It appears that patients who experience both mania and depression, could often trace at least two generations of relatives with similar disorders, while more cases of major depression occur in families of patients with bipolar depression.

Davidson and Neale (1982) state, that the role of neurotransmitters as a biological cause of depression forms a major area of research. In 1965, Schildkraut proposed the catecholamine theory, which supposes that a deficiency of catecholamine, especially of norepinephrine, at certain receptor sites in the brain is the cause of depression. Findings suggest that norepinephrine plays a role in mood changes, feelings of depression and the experiencing of pleasure. However, although, the catecholamine theory now seems oversimplified, it is important, as it explains how the function of neurotransmitters could cause depression or a state of elation.

In sum, then Davidson and Neale (1982) state, that the biological theory proposes that mood disorders have physiological causes and suggest that depression is related to the malfunction of neurotransmitters, which result in low levels of norepinephrine or serotonin.

2.7 ETIOLOGY OF DEPRESSION

2.7.1 Major Depression

The American Psychiatric Association [APA] (1987), defines major depression as being a mood disorder in which the essential features are either a depressed mood, loss of pleasure or interest in almost all or all activities accompanied by related symptoms, which last for at least two weeks.

Risk factors for major depression

Numerous researchers (Carson et al., 1988; Lobel & Hirschfeld, 1984) report, that in most studies of depression in industrialized countries, it is found that there are roughly twice as many women as men who become depressed. Beck (1967) reports, that numerous studies agree that the incidence and prevalence rates of depression in women, reach a peak at between the ages of 35 to 45 years. According to Boyd and Weissman (1982), the pattern for men is less evident, although it is apparent that the occurrence of depression in men increases with age. They add that, although the incidence and prevalence of major depression is high in women aged 35 to 45 years, it appears to decline during the menopausal years. Sachar (1982) agrees with Boyd and Weissman (1982) and states, that research suggests that menopause does not predispose the individual towards depression. However, Lewinsohn and Hoberman (1982) state, that individuals who have a history of depression - especially women - are at risk for major depression.

Boyd and Weissman (1982) report on the possibility of the existence of a pattern in the distribution of major depression across socio-economic classes, and state that most investigators find no specific pattern. However, women from the working class who have children, were found to be at a higher risk level than those women from the middle class. The American Psychiatric Association [APA] (1987) indicates, that a family history of alcoholism and/or depression, increases the risk. Researchers (Boyd &

Weissman, 1982; Ferster, 1973; Paykel, 1982) mention that evidence exists that a disruptive, hostile, negative environment in a child's home constitutes a risk factor for depression.

Studies conducted by Boyd and Weissman (1982) and Nurnberger and Gershon (1982), find that certain personality characteristics such as a tendency to collapse under stress, to worry, a lack of energy, introversion and sensitivity, feelings of insecurity, unassertiveness, dependency and obsessionality, could be present in individuals who develop depression. Studies generally show that, many (but not all) depressed individuals, appear to have experienced an excess of negative life events, prior to the onset of a depressive episode (American Psychiatric Association [APA], 1987; Boyd & Weissman, 1982; Lobel & Hirschfeld, 1984). According to the American Psychiatric Association [APA] (1987), the absence of a satisfying intimate heterosexual relationship is shown to be a risk factor towards depression; the transient emotional disturbances experienced during the first weeks after having had a baby i.e. post-partum depression, occur so often, that they are considered normal and usually clear up without treatment. It is thought that the hormonal changes which follow giving birth, cause the mood changes. However, the role changes and psychosocial events which affect a woman post-partumly could also increase the risk of depression (American Psychiatric Association [APA], 1987).

In sum, it would appear that risk factors for major depression are being female (35 to 45 years), a family history of depression or alcoholism, childhood experiences of a disruptive, hostile and generally negative home environment, a recent negative life event (an exit), not having an intimate confiding relationship and having had a baby in the preceding six months.

2.7.2 **Bipolar Disorder**

The description given by the American Psychiatric Association [APA] (1987), is that bipolar disorder involves

both mania and depression. The criteria for a diagnosis of bipolar disorder, is evidence of a present or past manic episode, accompanied by one or more major depressive episodes.

Risk Factors for Bipolar Disorder

In 1987 the American Psychiatric Association (APA) reports that, although major depression is more common among females, epidemiologic studies in the United States find that bipolar disorder is equally common in both males and females. Boyd and Weissman (1982) state that as an individual with bipolar disorder grows older, he is at higher risk of having a manic or depressive episode. It appears that upper socio-economic classes may experience bipolar disorders more frequently, with the disorder being more common among single and divorced persons; however, marital status may change as a result of the disorder rather, than its leading to the onset of the disorder itself (Boyd & Weissman, 1982; Carson et al., 1988). The American Psychiatric Association [APA], (1987) also reports that evidence exists to support a genetic component in the familial transmission of bipolar disorder.

In sum then, risk factors for bipolar disorder include a family history of bipolar disorder, being younger than 50, a previous history of the disorder, and being in the upper socio-economic class.

Summary

Certain trends emerge from the comparison of risk factors. It appears that women have higher rates of major depression and bipolar disorder than men, however, the sexual difference is less obvious in bipolar disorder. While bipolar disorder appears to be more frequent in the upper socio-economic classes, major depression shows no class distribution. However, the American Psychiatric Association [APA] (1987) stresses, that both major depression and bipolar disorder could follow a psychosocial stressor (death of a loved one, marital separation or divorce, or

childbirth).

2.7.3 Psychological Factors

Predisposition to depression: The Cognitive model

According to Beck et al., (1979) the cognitive model offers a hypothesis on predisposition to depression, which proposes that early experiences provides the basis for forming negative concepts about one's self, the future and the external world. These negative concepts (schemas) may be latent, but could be activated by specific events similar to previous experiences, which were initially responsible for embedding the negative attitude. For example, marital problems may activate the concept of irreversible loss, associated with death of a parent in childhood or depression may be triggered by a physical abnormality or disease that activates a person's latent belief that he is destined for a life of suffering. Beck et al., (1979) stresses, that adverse life situations do not necessarily produce depression, unless the individual is vulnerable to the specific type of event and in response, his thinking becomes markedly constricted and negative ideas develop about every aspect of his life.

Both Beck et al., (1979) and Du Plessis (1984) report that there is substantial empirical support for the cognitive model of depression. They report, that studies have documented the presence and intercorrelation of the constituents of the cognitive triad, in association with depression. Several studies support the presence of specific cognitive deficits (impaired abstract reasoning and selective attention) in depressed persons.

Life events and early environment

Paykel (1982) reports, that a number of studies have compared events occurring prior to the onset of depression reported by psychiatric patients with defined diagnoses of depression, with the events reported by control groups. These studies find that depressives report (a) almost three

times as many events in the six months before onset, (b) more stress on weighted scores and (c) twice as many recent losses. However, Paykel (1982) cautions against the generalization that events produce only depression and states, that there is clear evidence that such events could also precede other psychiatric disorders, for instance inclinations to suicide, mixed neurotic disorders and schizophrenia. He mentions that psychiatric illness might produce new circumstances (such as the loss of a job), could be consequences rather than the causes of illness.

According to Mendelson (1982), traditional views hold that depression is particularly induced by certain types of events, the most prominent being that of loss (deaths, limbs and other body parts), separations from key interpersonal figures, loss of self-esteem, as well as the absence of narcissistic self-gratification. However, studies indicate that recent separations, a blow to the self-esteem (failures and disappointments), as well as arguments and discord with various key interpersonal figures which may involve the threat of separation, are also important in the incidence of depression (Carson et al., 1988).

Paykel (1982) discusses one kind of situation that is not outlined in any theory. This is the success event, with its positive rather than negative connotations. According to Paykel (1982), clinical experience indicates that depression may occasionally occur subsequent to promotion, an unexpected economic windfall or the achievement of a long desired goal. Some of these events contain disguised threats such as greatly increased responsibilities and major disruptions of established life routine.

The strongest relationship to depression is found in broad classifications of stress (Davidson & Neale, 1982; Paykel, 1982). Paykel (1982) suggests, that rather than a single key triggering event, several events could suggest a cumulative stress. The individual's predisposition or vulnerability to depression, determines whether an event is followed by depression or not. Factors such as several young children in the home, the absence of someone to confide in,

unemployment and the loss of a mother by death or separation before the child is 11, contribute to an increased vulnerability to depression (Beck, 1967; Mendelson, 1982; Paykel, 1982).

Paykel (1982) suggests, that stressful situations which are persistent, rather than simply new events or changes, could contribute to depression. These would include a chronically bad marriage, poverty, physical invalidism, financial dependence on a husband in a marriage that is breaking down, absence of emotional support and being limited to the low-status role of being a housewife.

Beck (1967) provides a broad classification of the most frequently encountered precipitating circumstances: (a) events that lower the self-esteem, (b) obstacles in the way of achieving important goals, or an insoluble dilemma, (c) physical disease or abnormality which results in fears of deterioration or death, (d) single stressors of overwhelming magnitude, (e) several stressors occurring in a series and (f) insidious stressors, unrecognized as such by the affected individual. Beck (1967) stresses that psychosocial stressors provoke severe depressive psychoses only in persons who already have a negative cognitive set consisting of negative views of the self, of the world and of the future. Carson et al., (1988) infer that the stressor merely serves to activate negative cognitions that have been dormant.

With regards to early environment, Carson et al., (1988) suggest that the observation of exaggerated mood swings in parents could lead to the child's learning this maladaptive response pattern. It would appear, that the high occurrence of mood disorders in the families of manic and depressive patients, would provide opportunities for such learning.

2.7.4 Genetic and Biological Factors

Numerous researchers (Carson et al., 1988; Nurnberger & Gershon, 1982) state, that mood disorder is familial and conclude that relatives are more susceptible to mood

disorders than are unrelated individuals. Numerous studies of twins also suggest the presence of a genetic factor in the etiology. Evidence that a genetic factor contributes to an individual's vulnerability is compelling, as it is proven that environmental factors could not cause the concentration of illness evident in families. Nurnberger and Gershon (1982) report that cultural environmental transmission of the illness through families would not explain the results obtained in adoption studies, or in the studies of twins raised apart. However, Carson et al., (1988) warn that the difficulties of disentangling hereditary and environmental influences, make it dangerous to accept familial concordance as conclusive proof of genetic causation for depression.

Reacting to Pavlov's early lead, Carson et al., (1988) declares that a great deal of interest has been focused on the possibility that imbalances in excitatory and inhibitory processes might predispose some individuals toward extreme mood swings. However, Carson et al., (1988) suggest, that this excitation view (manic disorders result from excessive excitation and weakened inhibition, and depressive disorders from excessive inhibition) appears to be oversimplified as an explanation of manic and depressive disorders, in that it does not explain how the two extremes could be observed to sometimes exist simultaneously in the same individual.

Carson et al., (1988) review the theory, which proposes that depression is caused by depletion and mania by excess of one or both cerebral neurotransmitters. Carson et al., (1988) state that it is now clear that, based on the known level of complexity of brain biochemical functioning, no such straightforward mechanism is likely to provide the answers needed. However, they conclude, that while biochemical approaches do not prove a primary biological role in the etiology of major depression, biological causation remains a viable hypothesis, when based on three facts that are beyond reasonable dispute. The following are considered :

- (a) it is possible that a predisposition to this type of disorder may be genetically transmitted,
- (b) certain biological interventions often result in the abating of behavioural symptoms,
- (c) affective symptoms are often accompanied by profound alterations of bodily function, e.g. changes in the sleep cycle.

The observation of the high incidence of depressive syndromes, associated with endocrinopathies such as hypothyroidism and Cushing's syndrome, has heightened interest in the possible interrelation between hormone secretion and depression (Sachar, 1982). Akiskal and McKinney (1979) reviewed modern methods of psychiatric assessment and hormone management and stated that significant abnormalities in the secretion of corticoid, growth hormone and thyroid stimulating hormones have been documented. This concurs with the observation, that the clinical syndrome of endogenous depression, is typically associated with several symptoms suggesting hypothalamic dysfunction (disturbances in mood, sex drive, sleep, appetite and autonomic activity). Akiskal and McKinney (1979) conclude, that the same neurotransmitters implicated in the chemical pathology of depressive illness (noradrenaline, serotonin and acetylcholine), also regulate the secretion of the hypothalamic neuro-endocrine cells which control pituitary functions. Sachar (1982) suggests that it, therefore, follows that deficiencies in the functional activity of these neurotransmitters would be expected to be reflected in the hormonal responses they modulate. He also reports that neuro-endocrine studies of depressed patients have established striking and characteristic endocrine abnormalities in major depressive disorders of the endogenous subtype, which appear to be reflections of the primary central nervous system dysfunction in depression, rather than its contributing causes of this disorder.

It would, therefore, appear that the neuro-endocrine abnormalities in major depressive disorders, support the concept of significant hypothalamic dysfunction in this

condition, and offer clues to the psychobiologist about underlying neurotransmitter disturbances (Sachar, 1982). However, Carson et al., (1988) caution against either excessive enthusiasm or excessive skepticism regarding the role of this line of thought in relation to the etiology of depression.

2.7.5 Other possible causes of depression

Lobel and Hirschfeld (1984) suggest, that it should be remembered that there is a close relationship between health and clinical depression. Although keeping healthy, getting enough of the right type of food and enough sleep, as well as getting sufficient exercise are no guarantee against clinical depression, these factors may help in the prevention of some types of depression and retain the body in a better state to deal with depression, should it recur. They also state that, clinical depression could also be caused by certain physical illnesses (e.g. Addison's disease, Cushing's disease, thyroid disorders, diabetes etc.) and that it is associated with others (e.g. infectious diseases such as infectious hepatitis, influenza, rheumatic fever, anemia etc.). The role of medication in the etiology of depression should also not be discounted. Lobel and Hirschfeld (1984) refer to various anti-hypertensives, anti-parkinsonian agents, hormones, corticosteroids, anti-tuberculosis agents and anti-cancer agents.

Summary

There is considerable argument about whether depression is caused by endogenous factors (such as hormones or neurochemicals), or by the environment such as conflicts and sudden losses (Ferster, 1973; Paykel, 1982; Sachar, 1982). Ferster (1973) states, that since the common denominator among depressed persons is the decreased frequency of different kinds of positively reinforced activity, one could not expect that there would be one cause of depression or a single underlying psychological process, because behaviour is a product of so many psychological processes.

2.8 SYMPTOMS OF DEPRESSION

According to Boyd and Weissman (1982), feelings of sadness and disappointment are part of the human condition, experienced by everyone at some point in his life, whether or not he is clinically depressed. Whybrow et al., (1984) cautions, that only intense, pervasive, persistent symptoms which interfere with usual functioning are considered to be pathological. However, it is important to note that an incompletely defined gradient exists between normal mood and the clinical state of depression.

The present study does not include studies on clinically depressed individuals and, therefore, only a brief summary of the target symptoms of depression follows.

- Target symptoms: A summary

Beck (1979) stresses, that in moderately to severely depressed individuals, the therapeutic intervention should focus on the target symptom level (i.e. on components which cause suffering or functional disability). These target symptoms may be broken down into the following categories:

- (a) Affective or mood disorder symptoms: anxiety, sadness, loss of gratification, feelings or affection towards others, as well as apathy and loss of mirth-response.
- (b) Motivational: a wish to avoid problems and/or to escape from life (e.g. via suicide).
- (c) Cognitive: attention span problems, difficulty with concentration and/or memory, cognitive distortions (conceptual or information processing).
- (d) Behavioural: includes above symptoms plus passivity, withdrawal, retardation or agitation.
- (e) Psychological or vegetative: appetite (increased or decreased, and sleep (increased or diminished).

Note:

The present study addresses itself to the monitoring of the occurrence and fluctuation of depression in obese patients during the treatment of obesity i.e. depression as a reaction to treatment, as well as the influence depression has on the treatment outcome (weight-loss).

2.9 THE ASSOCIATION BETWEEN THE TREATMENT OF OBESITY AND DEPRESSION

The first part of this section reviews studies which report untoward reactions (more specifically depression) to attempts at weight-reduction. The second part covers studies which report no increase in depression during weight-reduction programmes. The third section provides an analysis of the methodological differences between the studies and the rationale for the planning of the present study.

2.9.1. Studies which report depression

In 1957 Stunkard published an article, *The Dieting Depression*, which was based on a study of the occurrence of negative reactions when obese individuals attempt to lose weight. He found that 54% of the obese individuals report, that they experience negative emotional feelings, which result in severe emotional disorders. Stunkard (1957) states that diet-related depressions are characterized by a prolonged period of depression following a short period of intense anxiety.

In agreement with the above, Simon (1963) proposed the hypothesis that obesity is a depressive equivalent (that some individuals try to avoid or handle feelings of depression by overeating). He states that, as knowledge related to the origin of obesity accumulates, it is realized that prime motivating factors which lead to overeating and eventually obesity, are personal insecurity and emotional instability. This is in contrast to researchers of the period who saw obesity as a symptom of emotional instability only. Simon (1963) bases his conclusions on findings by Hamburger in 1951, who states that there are four factors which could cause overeating: (a) a response to non-specific emotional tensions, (b) a symptom of illness, i.e. depression or hysteria, (c) a food addiction, (d) a substitute source of satisfaction when the individual is faced with intolerable life events. Hamburger (1951) concludes that overeating (hyperhagia) serves as a defence

(compensatory and substitutional) function and provides the individual with a primary and secondary reward when dealing with personal conflicts. Based on Hamburger's (1951) findings, Simon (1963) concludes, that his hypothesis is correct namely that obesity is a depressive equivalent. His study implicates personality factors, as he notes a statistically significant correlation between obesity and the absence of overt depression in obese individuals.

In line with the above researchers' findings, Leckie and Withers (1967) state, that analytically orientated researchers report an association between oral aggression and obesity. The obese individual is depicted as being an orally aggressive, depressed person, who eats in order to counteract his depression. Leckie and Withers (1967) designed an inventory to test for prevailing depressive moods and they reached the conclusion, that there is an underlying tendency towards depressive illness in clinically obese individuals. Their findings indicate, that 20% of the obese individuals score significantly higher on direct and indirect depression. The conclusion Leckie and Withers (1967) reach, is that the obese population as a whole forms a more depressed group with regards to mood and that this depressed mood is a response to dietary therapy. In their study of somatotypes and depression, Leckie and Withers (1967) conclude, that evidence of the cyclothymic personality, as described by Sheldon, exists. The conclusion they reach, is that the main relationship between obesity and depression is, that depression develops as a reaction to the treatment of obesity.

A consequence of the above findings is that Stunkard and Rush (1974) reviewed available literature on weight-loss treatment programmes. The conclusion they reach, is that the programmes cause adverse psychological (depression, anxiety, etc.) reactions. They agree with Hamburger's (1951) findings that emotional problems occur with considerable frequency in out-patients being treated for obesity. In 1957 Stunkard verified Hamburger's 1951 findings and submitted a report, which states that both groups of obese individuals involved in his study, have a

high incidence of emotional problems, associated with dieting. Stunkard and Rush (1974), on reviewing the study of 60 obese individuals carried out by Silverston and Dinello in 1966, state that exactly half report either the onset or intensification of depression and anxiety during weight-reduction programmes. They report, that emotional difficulties are influenced by the onset of obesity and that obese individuals with childhood onset have more emotional difficulties (i.e. distortion of body image) and are more difficult to treat than those individuals with adult-onset. In addition to this, Stunkard and Rush (1974) review a study conducted in 1973 by Grinker, Hirsch and Liven. This study reports an increase in depressive symptoms in the entire group of obese individuals during and following weight-loss. Analysis of these findings, led Stunkard and Rush (1974) to conclude, that the age of onset (childhood onset) is the most distinctive factor in vulnerability to negative reactions to the treatment of obesity. Stunkard and Rush (1974) see this development of untoward responses as contributing significantly to the high drop-out rate from weight-reduction programmes. They recommend, that during treatment, the physician should be alert to the possibility of untoward responses, particularly among patients who are considered to be at risk with regards to depression.

With reference to the psychosomatic hypothesis of obesity (emotional distress causes obesity), Bruch (1974) reports, that for many of her patients depression results in weight-gain.

Polivy and Herman (1976) conducted a study to test the hypothesis that depressed individuals who are restrained eaters, will gain weight in conjunction with their depression, while unrestrained (non-dieters) will lose weight. Data from their study support this hypothesis and findings indicate, that the direction of weight change is consistent with the individual's report of the effect that depression has on his eating behaviour. According to this analysis, it follows that emotions could disrupt the chronic self-control of dieters (restrained eaters) and lead to increasing consumption, as there is an identifiable sub-

population of depressives for whom weight-gain is symptomatic. According to Polivy and Herman (1976), there is, therefore, a clear association between depression and weight-gain for a substantial number of individuals. However, these researchers emphasize, that there is only an association and not a clear causal relationship between weight-gain and depression.

Fawzy et al., (1984) confirms Polivy and Herman's (1976) finding, that a clear association exists between depression and weight-gain in restrained eaters (dieters). They suggest, that obesity and overeating may hide feelings of marital, sexual or social inadequacy, in that it supplies a stuffing euphoria. Fawzy et al., (1984) state, that weight-loss programmes could and do cause adverse psychological and metabolic complications in obese patients. Their findings imply, that long-term treatment (fasting or low-calorie diet) is consistently associated with adverse, often severe, physical and psychological symptoms. Fawzy et al., (1984) report, that the most commonly reported symptoms are anxiety, depression, fantasies and dreams about food and eating, affective lability, hunger, nausea, fatigue and decreased sexual appetite.

The results of a study conducted by Klesges (1984), indicate that three global measures of personality do not discriminate between normal weight and untreated overweight individuals. The finding of this study, that only weight-related depression, self-consciousness, and assertiveness discriminated weight classes, is consistent with theories that hypothesize a high degree of behavioural specificity in individuals. Klesges (1984) suggests, that overweight individuals may experience depression only when dieting and that it is possible that obese individuals are not as globally depressed, non-assertive, or self-conscious as previously thought. He concludes, that a more important issue may be to determine how depressed an individual is about his weight, rather than being an issue of assessing depression as if it were a global clinical syndrome. Data from Klesges's (1984) study, support this conclusion.

Finally, Stunkard et al., (1986), state that many obese individuals experience emotional disturbances during attempts to lose weight and maintain, that this is one of the reasons why the treatment of obesity for out-patients has a high attrition rate.

The above studies conclude, that weight-reduction programmes could and do cause or contribute towards an increase in depression as experienced by obese individuals. However, various studies which contradict these findings, are reviewed in the following section.

2.9.2 Studies which reported no increase in depression

On analyzing data in their study Shipman and Plesset (1963) find, that only a small number of dieters report being depressed at the end of the diet programme. They add, that these individuals had been depressed at the onset of the programme. No confirmation is found for the belief that the obese suffer from depression which is exposed by dieting, as only a few individuals in Shipman and Plesset's (1963) study report depression. These researchers suggest that Stunkard's (1957) pessimistic conclusion results from sampling and methodological biases, i.e. asking only extremely overweight dieters about their previous dietary efforts. Shipman and Plesset (1963) note a previously unreported and unexpected finding, namely that individuals become less depressed and anxious when they successfully lose weight. They also report, that successful dieters have low levels of anxiety and depression at the start of the weight-reduction programme.

The relationship between anxiety, depression, and weight-loss, as well as the effect of successful weight-loss on depression and anxiety was investigated by Pitta et al., (1980). Their findings confirm that a significant decrease in anxiety and depression accompany weight-loss. Pitta et al., (1980) conclude, that baseline depression predicts weight-loss with the surprising finding, that individuals who are more depressed at onset of treatment, lose the most weight. These researchers suggest that

perhaps the individuals who are very unhappy about their obesity, present themselves as being more depressed. This could imply that they were more motivated to lose weight. The strongest findings in this study, are the general reduction in depression, anxiety and weight. Findings by Pitta et al., (1980) support the conclusions reached by Shipman and Plesset (1963).

When on conducting a study using low-dose mianserin (anti-depressant compound) as additional therapy for obese individuals, following a very low-calorie diet, Cook et al., (1981) find that scores on the Beck Depression Inventory decrease (indicating a decline in depression) by 50% in both groups after a period of eight weeks. They conclude that obese individuals do not become more depressed during treatment and that no clinical advantage is gained by adding low doses of mianserin to the treatment programme. In earlier trials Cook et al., (1981) assert, that symptoms of depression appear to develop in some obese females soon after they start the diet; this affects compliance, and the resulting failure to lose weight, leads to a deterioration in their mental state. Results of their experiment indicate, that even though some obese individuals are mildly depressed at the start of the programme, it appears that a very low-calorie diet has mood-elevating effects, which are prominent during the period of maximum weight-loss (early phase of programme). Cook et al., (1981) concludes, that obese individuals who participate in a weight-reduction programme consisting of a very low-calorie diet, do not develop significant affective disturbances and that no advantage is gained by the administering of mianserin.

In line with the above mentioned studies Linet and Metzler (1981), also find no support for the notion that depressive features might be characteristic of obesity. Results of their study indicate that individuals with childhood or adult-onset of obesity, have similar emotional profiles, successful weight-loss results in an improved emotional state, no increase in emotional symptomatology occurs in individuals prior to their dropping out of the treatment

programme and clinic attendance and dieting do not affect the individual's mental health. These researchers report, that a controversy exists in literature as to whether weight-loss efforts have a beneficial or untoward effect on the individual's mental status. Alternatively, Shipman and Plesset (1963) conclude, that successful dieting lowers the individual's anxiety and depression, while Silverstone and Solomon (1965) find no evidence that dieting has any unpleasant emotional effects. Linet and Metzler's (1981) findings support the latter conclusion, since their data do not indicate an increase, or the occurrence of emotional disturbance. Linet and Metzler (1981) indicate that therapeutic success in the treatment of obesity is influenced by the individual's internal-external locus of control, social conformity, desire for social acceptance, diminished responsivity to cues from the environment, belief that poor eating habits cause obesity and finally by the skills of the individual therapist.

Craighead et al., (1981) also report a decrease in the intensity of depression and an increase in positive cognitions in individuals participating in a programme consisting of behaviour therapy and pharmacotherapy as treatment for obesity. This is a noteworthy finding, as individuals on reducing diets, frequently report an increase in depression, as well as other emotional disturbances.

In agreement with previous researcher's findings Wadden et al., (1983) report no increase in depression or anxiety in obese individuals participating in a combined behaviour modification and very low-calorie diet treatment programme. This finding is in striking contrast to the findings in traditional treatment programmes, in which a large volume of weight was lost. Wadden et al., (1983) provides important evidence, that group behaviour therapy treatment protects dieters from the adverse emotional consequences of dieting. These researchers also report, that even though these obese individuals do not experience an increase in unpleasant emotional responses, it is clear that individuals who report depression and anxiety at the onset of the programme, show poorer dietary adherence and have smaller

weight-losses. Wadden et al., (1983) conclude, that the more depressed obese individuals have greater difficulty in adhering to the demanding very low-calorie diets.

When investigating the efficacy, safety and future of very low-calorie diets, Wadden et al., (1983) observe that researchers report very few untoward emotional responses. Results from their study indicate significant decreases in BDI scores, with no support for Stunkard and Rush's (1974) findings of untoward emotional responses.

On examining mood and weight-loss in a behaviour therapy treatment programme for obese individuals, Wing et al., (1983) report, that findings indicate that positive mood changes, which appear to be related to changes in weight, are reported during the ten-weeks programme. These researchers find no evidence that individuals with juvenile-onset obesity, have more negative reactions than adult-onset to dieting, or that dropping out is a response to an increase in depression. Although pre-treatment mood measures are not linearly related to success in the programme, Wing et al., (1983) report, that these form evidence of a nonlinear relationship. These findings are in contrast to findings by Stunkard and Rush (1974), who report that obese individuals become depressed during the course of a weight-reduction programme.

When reviewing ten studies investigating the relationship between mood change and weight-loss in behaviour therapy weight-reduction programmes, Wing et al., (1984) report significant positive changes in mood in six of the ten studies, with no evidence to indicate that untoward reactions to dieting are more frequent. Wing et al., (1984) state, that positive changes in mood are related to active participation in a treatment programme and that these occur in both behavioural and non-behavioural therapy programmes. They conclude, that there is no evidence in the ten studies to support the hypothesis that untoward reactions are a common problem in behavioural therapy weight-reduction programmes, that dropping out is a response to increasing depression, or that individuals with juvenile-onset obesity,

experience more mood changes than those with adult-onset. What is important, is that these studies suggest that participation in behaviour therapy weight-reduction programmes may be associated with decreases in anxiety and depression. Wing et al., (1984) state that, although the magnitude of improvement is not large, large changes are not anticipated, as the participating obese individuals are neither clinically anxious nor depressed.

Wing et al., (1984) also report, that findings of several studies in which the Beck Depression Inventory (BDI) is used, indicate that average scores decrease from mild depression to no depression. These findings indicating a positive mood change accompanying weight-loss, contrast sharply with earlier reports of untoward reactions. Wing et al., (1984) also conclude that, several basic differences between earlier and later studies might account for these differences in results. Firstly, many of Stunkard's (1957) early observations of untoward reactions are based on a highly select group of obese individuals seen by a psychiatrist. Wing et al., (1984) state, that such individuals are often excluded from behaviour therapy treatment programmes. Secondly, the differences in the results could depend on the size of the weight-loss as some of the earlier studies include in-hospital starvation programmes, which result in greater weight-loss than achieved during behaviour therapy treatment programmes. Thirdly, a non-standardized assessment of mood was often used in early studies; in some cases, for instance, obese individuals were asked to give retrospective reports of mood changes they experienced during previous attempts to lose weight. Finally, a major difference is, that obese individuals were seen individually over a long period of time in the early studies, while more recent studies follow a group approach.

In line with the above, Wing et al., (1984) state that the majority of behaviour therapy treatment programmes are conducted in group format and last only ten to twenty weeks. Results of these studies indicate a relationship between changes in weight and depression and anxiety. Wing et al.,

(1984) suggest that these changes in the affective state, might cause weight changes. This confirms findings by Spitzer, Endicott, and Robins (1978), who state that depressive episodes are frequently associated with changes in weight, as well as with changes in activity and appetite. Because many researchers argue, that weight-loss in individuals might lead to an increase in both depression and anxiety, which again could result in individuals not losing weight or being unable to maintain weight-loss, Wing et al., (1984) cautions against the widespread prescription of weight-reduction.

In sum, Wing et al., (1984) conclude, that weight-losses which occur in behaviour therapy treatment of obesity, are associated with a decrease in anxiety and depression or at least, no worsening of these affects. Wadden (1984) supports the findings of Wing et al., (1984), stating that behaviour therapy treatment of obesity appears to be associated with positive mood changes. This finding contrasts dramatically with the 1950 and 1960 reports of untoward emotional responses in outpatients and in-patients who are treated by way of traditional weight-reduction methods.

In addition, a study by Wadden et al., (1984) in respect of the treatment of obesity by using behaviour therapy and a very low-calorie diet reports, that obese individuals show significant improvement in psychological functioning during the six months of treatment, even while they consume a very low-calorie diet. The significant reduction in depression (BDI), state and trait anxiety seen by the eighth week are maintained by the end of the treatment period. The finding, that individuals do not experience an increase in depression or anxiety while following a very low-calorie diet, differs strikingly from the findings of earlier reports especially those of Stunkard and Rush (1974). The findings of Wadden et al., (1984) suggest, that group behaviour therapy treatment programmes protect obese individuals from the adverse effects of rigorous diets.

Weighill and Buglass's (1984) study indicates, that initial

depression or anxiety is not related to subsequent weight-loss. They find that in obese women, there is a statistically significant correlation between change in mood and weight. Weighill and Buglass (1984) conclude, that as weight increases, the obese individual's distress worsens; these researchers suggest that it is possible that as depressed individuals lose weight, their mood improves and they feel able to restrain their appetite. This implies that mood is reactive to weight change. Based on findings which suggest that individuals react psychologically (increasing depression) to even minor weight-gains, Weighill and Buglass (1984) recommend, that attention should be paid to obese individuals who fail to lose weight.

In their controlled trial using a combination of a very low-calorie diet (400 to 500 kcal) and behaviour therapy further evidence of the usefulness of behaviour therapy in a weight-reduction programme is provided by Wadden and Stunkard (1986). They report significantly greater weight-losses, which are maintained for a year, when combining behaviour therapy with a very low-calorie diet. Wadden and Stunkard (1986) also report, that obese individuals who receive behaviour therapy alone or in combination with a very low-calorie diet, show significant decreases in depression. This study was designed to allow Wadden and Stunkard (1986) to investigate the relationship between changes in mood and weight, as they wished to examine the hypothesis proposed by Wing et al., (1984), that behaviour therapy treatment of obesity is associated with greater improvement in mood, than is non-behaviour therapy treatment. Results obtained, support the hypothesis of Wing et al., (1984), as both the behaviour therapy and combined treatment subjects showed significant reduction in depression at post-treatment. Wadden and Stunkard (1986) conclude, that the coping skills taught in behaviour therapy programmes, help obese individuals deal with any untoward emotions brought about by following very low-calorie, as well as conventional reducing diets.

2.9.3 Summary

The effects of a weight-reduction programme on the mental health of obese individuals, have been the subject of much controversy. Following his findings of increased psychiatric symptoms in obese individuals attending a nutrition clinic the phrase "Dieting Depression" was coined by Stunkard in 1957. During the 1960's further evidence was found of increased depression after a two-month diet, even though the individual was successfully lost weight. Stunkard and Rush (1974) conclude, that weight-reduction treatment does cause adverse psychological and metabolic complications in obese individuals. However, there is no mutual agreement on this conclusion, as numerous studies report an improvement in mood after ten days of fasting and a lessening of initial depression in nineteen out of twenty cases of hospitalized obese individuals treated by fasting, followed by a low-calorie diet.

Criticism against earlier studies is based on the fact, that most studies focussed on hospital patients who reported untoward reactions, while out-patients, with the exception of those in Stunkards' early studies, tended to report a minimum of untoward reactions. This is evident from results obtained by Shipman and Plesset (1963) who, on studying 151 out-patients report a reduction in their levels of anxiety and depression, while Linet and Metzler (1981), on studying 205 chronically obese out-patients, report that obese individuals who dropped out, did not show any increase in psychological disturbance. These researchers conclude, that obese individuals who are successful at losing weight, are significantly less depressed and have fewer somatic and interpersonal problems at the end of a six-month period, while those who did not lose weight, show little change.

A review of the literature indicate that results show a clear relationship between mood and successful weight-reduction.

2.9.4 Methodological differences in earlier studies and rationale for the present study's methodology

According to Wadden et al., (1986), psychological results of dieting are divided into two categories, i.e. studies which report beneficial effects and studies which report adverse effects. These researchers report, that according to Stunkard and Rush (1974), adverse reactions were observed in seven studies between 1951 and 1973, while improvements in mood were found by Wing et al., (1984) in ten studies using behavioural therapy, published between 1969 and 1983. Wadden (1984) states, that although this improvement in psychological functioning is attributed to behaviour therapy, it could also be the result of three methodological differences between the 1951 to 1973 and the 1969 to 1983 studies. The first difference identified by Wadden (1984), is the frequency of the assessment of mood as the weekly or daily measurement of mood used in early studies, could have increased the chance of picking up any adverse reactions. This is coupled to the time of assessment, with retrospective assessment resulting in an increase in the reporting of symptoms. The third and final difference is the method used to measure mood. While the open-ended interview which was used in early studies, meant that all the symptoms experienced by individuals while dieting, could be discussed, the objective inventories in the behaviour therapy studies measure only one aspect of mood.

In order to investigate the effects these three differences could have on reports of psychological functions, Wadden et al., (1986) observed twenty eight obese women who had lost approximately 19,2 kilograms in six months. When they analyze frequency of assessment, Wadden et al., (1986) observe, that more than 50% of the women report a deteriorating mood during one or more weeks; this despite the finding, that there were improvements in mood from pre- to post-treatment. In respect of time of assessment, it was found, that reports of depression and anxiety were higher when measured retrospectively than concurrently. The method of assessment reveals that several symptoms not measured by

objective tests, are reported in open-ended interviews.

Wadden et al., (1986) conclude, that information gained in their study, indicates that the method of assessment used, will determine what answer the researcher gets with regards to whether dieting is associated with adverse psychological responses. Limiting assessment by objective inventories to pre- and post-treatment led to the observance of significant decreases in depression and state and trait anxiety. However, results of weekly assessment by using the same objective tests, present a different picture, as more than half of the individuals report a 25% or more increase in depression and state anxiety. Wadden et al., (1986) state, that pre- to post-assessment are not sufficient to monitor adverse emotional reactions occurring throughout the weight-reduction programme and they suggest that it is possible, that it is the protracted effort involved in dieting and not the large end-of-treatment weight-loss, that contributes to an adverse emotional response.

Contrasting to current reports Wadden et al., (1986) also report, that concurrent and retrospective measurement of mood yields different results, with retrospectively completed BDI and State-Trait Anxiety Inventory (STAI), portraying obese individuals as being significantly more depressed and anxious at the midpoint of the treatment programme. It is evident, that the more depressed the obese individual is before treatment - as measured by the BDI - the more retrospective scores exceed their concurrent depression scores. Wadden et al., (1986) also observe, that the amount of weight the obese individual loses, affects the amount of depression reported; as the retrospective reports of depression exceed the concurrent report when the obese individual fails to lose the desired amount of weight. Wadden et al., (1986), recommends the inclusion of a non-dieting control group, to reveal whether changes in mood recorded are due to dieting and/or non-dieting factors. This recommendation is based on the above researchers' findings, that results obtained from open-ended interviews, suggest that dieters might experience psychological and physical problems not addressed by objective measuring instruments

like the BDI and the STAI.

The methodological differences between earlier studies as summarized by Wadden et al., (1986), form the foundation of the present study's methodology. An attempt is made to overcome these differences, by using pre-treatment measurements, bi-weekly measurements during the treatment programme and post-treatment measurements during the follow-up phase. Assessment was done before weigh-in and therapy to control for the effects of weight-loss or gain, as well as therapy on depression reported. An attempt was also made to include a non-dieting, non-therapy control group. Behaviour therapy and cognitive behaviour therapy characterized the treatment programmes, which also included a very low-calorie diet, the rationale being, that the reviewed studies on behaviour therapy, plus a very low-calorie diet as a treatment for obesity, reported no significant increase in untoward reactions during the treatment of obesity. Studies which included a cognitive intervention (cognitive restructuring, problem-solving, improving self-control and self-regulation) into a behaviour therapy weight-reduction programme, plus a very low-calorie diet, facilitated the maintenance of initial behaviour change needed for an individual to lose weight and also to maintain the new lower-weight level.

CHAPTER 3 - METHODOLOGY

The general goal of the study was to assess the occurrence and fluctuation of depression in obese patients in two different weight-reduction treatment programmes, i.e. a behaviour therapy programme with a very low-calorie diet and a cognitive behaviour therapy programme with a very low-calorie diet. The first section of this chapter contains the research hypotheses. The second part contains a description of the subjects who participated in the weight-reduction programmes, while the third section of the chapter provides a description of the procedure followed. The fourth section covers the measuring instruments used, their development, norms, reliability, validity and cut-off scores, as well as the rationale for using the specific measuring instruments. The chapter concludes with a description of the method used for the statistical analysis of the data.

3.1 Research Hypotheses

Specific Goal Number 1:

To assess whether the different depression inventories yielded different results.

H₁ There will be a difference between the results yielded by the different depression inventories.

Specific Goal Number 2:

To assess whether depression occurred over time in the patients participating in the different treatment programmes.

H₂ Depression will occur over time in the patients participating in the different treatment programmes.

Specific Goal Number 3:

To compare the level of depression which occurred over time in the patients participating in the different treatment programmes.

H₃ A difference in the level of depression will occur over time in the patients participating in the different treatment programmes.

Specific Goal Number 4:

To compare whether there was a fluctuation in the level of depression which occurred over time in the treatment programmes.

H₄ There will be a fluctuation in the level of depression which occurs over time in the treatment programmes.

Specific Goal Number 5:

To assess whether there was a difference in the depression which occurred during treatment and the outcome of treatment (weight-loss) in the different treatment programmes.

H₅ There will be a difference in the depression which occurs during treatment and the outcome of treatment (weight-loss) in the different treatment programmes.

3.2 The Subjects

The patients were referred by their general practitioners to the obesity clinic at Vaalmed, Vanderbijlpark. Referrals were made during the period September 1987 to 15 March 1988. All the patients were medically evaluated before treatment; this evaluation included a physical examination, an electrocardiogram, a full blood count, chemical analysis and thyroid function tests. The following were contra indications for treatment of obesity: a recent mio-cardial infarction or indications of cardiac abnormalities; a history of cerebro vascular, kidney or liver disease or of

cancer; or of Type I diabetes and serious psychiatric disturbances. None of the patients used anti-depressant or anxiolytic medication before or during the treatment programme. All the patients participated on a voluntary basis and formed the two experimental groups. Referrals from 15 March to 13 April 1988 were included in the control group. These patients participated in the next treatment programme which commenced in August 1988.

The size of the experimental groups

Patients were randomly assigned from stratified blocks (based on percentage overweight and sex) to one of the two treatment programmes, which comprised: a) standard behaviour therapy plus a very low-calorie diet, and b) cognitive behaviour therapy plus a very low-calorie diet. The uneven number of patients in the two treatment programmes (N = 27 and 23) resulted because of patients who dropped out of the weight-reduction programme.

3.3 Procedure

All the patients attended weekly treatment sessions of 90 minutes in groups of from 10 to 20 people. Two clinical psychologists and a medical practitioner conducted the sessions. The patients paid an amount of R150.00, over a period of 3 months, which served as an incentive for participation. It was necessary to make a few adjustments in the allocation of patients to the two groups as a result of cancellations which occurred after the first session. The **male:female ratio** was the same for both experimental groups, i.e. 1:4.

3.3.1 The groups and the treatment programmes

There were three groups, of which two were experimental groups, and one a control group. The average age of the patients in the groups was 35 years. Treatment of the two experimental groups was commenced on 2 March 1988. The control group consisted of a no-treatment group (no diet, no therapy) who were monitored for the duration of the

treatment programme, plus a one month follow-up. The same psychometric evaluations were used for the control group as for the two experimental groups. The control group was included in the study in an attempt to reveal whether changes in mood were due to dieting or nondieting factors, as well as to control for any therapeutic effects resulting from the assessment procedure (Shaw, 1977; Stunkard, 1987).

The two experimental groups underwent different treatment programmes:

3.3.2 Standard Behaviour Therapy Programme plus a Very Low-Calorie Diet.

The patients in Group 0 participated in this treatment programme. The first month was used to prepare the patients for the treatment programme. The first two weeks of month One entailed psychometric evaluation, calorie education and nutritional information supplied by a dietician. The patients were then placed on a medium calorie diet (1000-1200 calories per day) for a period of two weeks. This was followed by a very low-calorie diet for the next two months. The low-calorie diet was limited to a period of two months because this is the longest period regarded as safe (Wadden et al., 1983). During the fourth month the patients were placed on a refeeding programme. Traditional behavioural therapeutic techniques to control body weight were introduced to the patients during the weekly group sessions. Training included the following: (a) keeping a daily record of eating behaviour (quantity, calories consumed and time of eating) [Self Monitoring], (b) control of stimuli associated with eating behaviour (e.g. Television), (c) reduction in the tempo of food intake, (d) nutritional education, (e) restructuring of self reinforcing thoughts and emotions associated with dieting, (f) social support and (g) reinforcing of changes in eating behaviour.

The goal of a weekly weight-loss of one kilogram was set for the patients, who received weekly feedback with regards to their progress in the form of a computer printout. Sessions were held weekly for a period of ten weeks (during this

period measurements of depression were made bi-weekly), every second week for four months and monthly for three months. Chemical analysis was carried out four times during the treatment programme and all patients were medically monitored on a weekly basis.

3.3.3 Cognitive Behaviour Therapy plus a Very Low-Calorie Diet.

Patients in Group 1 participated in this treatment programme. The first month was used to orientate the patients with regards to the contents of the treatment programme to be used. These patients followed a medium calorie diet (1000-1200 calories per day) for the last two weeks of month one, the very low-calorie diet for months two and three and the refeeding diet for month four. The diet interventions, medical monitoring and computer feedback were identical for both Group 0 and Group 1. The latter group also received the traditional behavioural therapeutic techniques for body weight-control, but with an emphasis on cognitive aspects. The first five group sessions were devoted to self monitoring, stimulus control, lowering the tempo of food intake and reinforcement. Cognitive restructuring was used in each phase. The last five sessions were devoted to specific cognitive aspects, which included cognitive restructuring of obesity cognitions as identified by O'Connor and Dowrick's, 1987, Obesity Cognitions Scale, covert aversion therapy and thought stopping techniques. Sessions were held every week for ten weeks (during this period measurements of depression were made every two weeks), every second week for four weeks and monthly for three months.

3.4 Frequency of assessment

Pre-therapy measurements of depression were conducted on both the experimental groups. All measurements were conducted before weighing. Wing et al., (1983) emphasizes that all measurements should be applied prior to weighing the patients to control for the effect of weight-loss or increase on the level of depression reported.

The patients were assessed for depression every two weeks based on the recommendation by Wadden et al., (1986) who found that the level of depression fluctuated during the treatment of obesity, and that a pre- and post-measurement alone was not enough to monitor this fluctuation. Studies conducted by Wing et al., (1984) as well as Wadden (1984) underscored the need for frequent and repeated assessment of mood.

Follow-up measurements after the completion of the three month programmes included the measurement of depression using the same measuring instruments. Both Gormally and Radin (1981) as well as Graham et al., (1983) stress the importance of follow up measurements and stated that the measurements should be continually repeated as the results obtained could differ dramatically from those during treatment. Gormally and Rardin (1981) emphasizes that the mechanisms needed to maintain weight-loss differed markedly from those needed to lose weight and that this could influence the measurements obtained.

Wadden et al., (1986) as well as Wing et al., (1984) state that when assessment by objective inventories is limited to the period before and after treatment, significant decreases were observed in the depression of patients. Results of weekly assessment using the same inventories painted a different picture; more than half of the subjects experienced increases in depression of 25% or more. Pre- to post-assessment, by nature, appears incapable of detecting adverse emotional reactions that may occur during the course of dieting. Wadden et al., (1986) conclude that it is the protracted effort to diet, rather than a large end-of-treatment weight-loss that is likely to be associated with adverse emotional responses.

3.5 Use of self-rating scales

Studies have reported that the instrument used to measure depression can influence the results obtained (Wadden, 1984; Wadden et al., 1986; Wing et al., 1983; 1984).

Kearns et al., (1982) states that the general weakness of self-rating scales is that they cannot be used in very severe degrees of depressive illnesses, since patients may either not be accessible to questioning or may be incapable of giving meaningful answers. However, the present study did not address itself to the occurrence and fluctuation of severe depression.

According to Botha (1983), the most important advantages to using self-rating scales are: (a) the time and cost factor, (b) the fact that less training is needed to administer the scale, (c) the administration and point allocation of the scale are standardized, and (c) clinician expectations and prejudices do not play a role.

The most important difference between self-rating and clinician-rating is the inability of the self-rating scale to evaluate certain psychotic symptoms. However, Botha (1983) recommends that the self-rating scales be used not for the quantifying of the degree of depression, but for the recognition and/or discovery of the presence of depressive symptoms (i.e. as a sifting instrument), which is the area that the present study addressed.

3.6 The Measuring Instruments

With the exception of the Biographical Questionnaire all the psychometric questionnaires were bilingual. Patients who were English-speaking were assisted in completing the Biographical Questionnaire. The Beck Depression Inventory was reproduced from appendixes to articles by Beck, Ward, Mendelson, Mock, and Erbaugh (1961) and Beck and Beamesderfer (1974), while a bilingual questionnaire was obtained from Fullard (1982) and correlated with the Afrikaans questionnaire obtained from Botha (1983) [see Appendix B]. The Carroll Self Rating Scale was reproduced from Carroll, Feinberg, Smouse, Rawson, and Grenden (1981), while the Afrikaans version was reproduced from Botha (1983) [see Appendix C and D]. Both these questionnaires were carefully reviewed to ensure accurate reproduction.

3.6.1 Physical measurements

The physical measurements included weight, blood pressure, underarm fat (using a calibrator), urine- and blood analysis. These measurements were carried out under the supervision of the medical doctor and analyzed in the laboratories at Vaalmed at the start of the programme, and continued thereafter on a monthly basis.

3.6.2 Biographical Questionnaire

A biographical questionnaire, which was composed by the clinical psychologists and the general practitioner, was filled in before treatment commenced. The questionnaire included items related to background variables, marriage adjustment, eating habits, history of obesity and other obesity related aspects (see Appendix A). The purpose of this questionnaire was to obtain specific information not covered by the other measuring instruments.

Rationale for including the Biographical Questionnaire

Stock and Rothwell (1982) report that with the knowledge of a patient's familial background of obesity and other medical or psychological disorders, it may be possible to ease his or her unwarranted feelings of guilt. Bray and Teague (1980) state that there is a need for information on family history of obesity, and base their recommendation on studies conducted on twins with obesity which provided some support for the concept of a familial tendency for obesity. The category of obesity of undetermined origin remains the largest and this fact indicates why information regarding the circumstances surrounding abnormal eating patterns and decreased physical activity is so important (Bray & Teague, 1980). A strong relationship between social factors and obesity have repeatedly been established and therefore these factors must be considered as among the most important influences on the prevalence of obesity (Daniels, 1984; Stunkard, 1980). With regards to the age of onset of obesity, Brownell and Stunkard (1980) state that the psychological consequences are far more troublesome for

patients with onset of obesity in childhood and that body image disparagement, binge-eating (bulimia) and night eating occurs almost exclusively among individuals with childhood-onset of obesity. Oberholzer (1984) states that it is essential to determine the age of onset of obesity as the prognosis of childhood obesity is generally poor and this has an effect on the format of the treatment programme to be used. Oberholzer (1984) reports that progressive obesity which usually begins in childhood and continues throughout life is a particularly ominous form of weight-gain.

Given the above it was deemed necessary to obtain the specific information covered by the biographical questionnaire in order to determine if the treatment groups were in fact comparable before continuing with further analysis of the depression scores.

3.6.3 The Beck Depression Inventory (BDI)

Development

The Beck Depression Inventory (BDI), developed by Beck et al., (1961) is one of the most widely used self-rating scales for the measurement of depression (Bailey & Coppen, 1976). The aim of the instrument is to formulate a method of defining depression that would be reliable and valid for the measurement of depression (Beck et al., 1961).

Application and Marking Procedures

The BDI is one of the first self-rating scales for depressive symptomatology. The BDI contains twenty-one items and each item consists of a graded series of statements. The patient is asked to select the single statement in the domain of each item that corresponds most closely with his actual condition. Numerical values from 0 to 3 are assigned each statement to indicate the severity of depression. The total BDI score is obtained by adding the scores of the twenty-one items; consequently, the BDI score range is 0 to 63. The statements are ranked to reflect the presence of the symptom from neutral to maximal severity.

The items were chosen on the basis of their relationship to the overt behavioural manifestations of depression and do not reflect any theory regarding the etiology or the underlying psychological processes in depression (Beck et al., 1961).

According to Beck and Beamesderfer (1974), the BDI assumed its present form because of the following observations: (a) the number of symptoms increases with severity of depression, and the frequency of depressive symptoms progresses in a step-like manner from nondepressed, to mildly depressed, to moderately depressed, to severely depressed patients, and (b) the more depressed an individual is, the more intense a particular symptom is likely to be. For these reasons, the BDI was designed to include all symptoms of the depressive constellation, and at the same time to provide for grading the intensity of each. The scoring takes into account the number of symptoms reported by the patient, in addition to the intensity of each symptom.

Cut-Off scores

There is no specific score that can be used for all purposes as a cut-off point for depression (Beck & Beamesderfer, 1974). The specific cut-off depends upon the characteristics of the patients in the sample and on the purpose for which the inventory is being used.

The crux of the problem is: how many false-positives and false-negatives occur at a particular cut-off score? In order to identify a relatively pure group of depressed patients for research purposes, the investigator must minimize false positives (i.e. high scores for individuals who are not really depressed). A high cut-off score, therefore, should be used (i.e. a score of more than 21).

As a screening device to detect depression among psychiatric patients, Beck and Beamesderfer (1974), as well as Taylor et al., (1978) found that a cut-off point of 13 was appropriate. This score gives fewer false-negatives, but

more false-positives than the higher cut-off point. For screening depression among medical patients a cut-off score of 10 was appropriate. However as this study only concerned itself with the occurrence of depression no cut-off score was used.

The range of scores used in the present study was as recommended by Beck and Beamesderfer (1974) i.e. 0 - 4, none or minimal depression; 4 - 7, mild depression; 8 - 15, moderate depression; 16+, severe depression. The raw scores obtained were converted to a scale of 0 (0-4); 1 (4-7); 2 (8-15) and 3 (16+) as recommended by Beck and Beamesderfer (1974).

Norms

The primary norm group of the BDI was patients who were drawn from the routine admission to the psychiatric outpatient department of a university hospital (Hospital of the University of Pennsylvania), and to the psychiatric outpatient department and psychiatric inpatient service of a metropolitan hospital (Philadelphia General Hospital). The outpatients were evaluated either on the same day of their first visit to the outpatient department, or a specific appointment was made for them to come back a few days later. Hospitalized patients were all evaluated on the day following their admission to the hospital, i.e., during their first full day in the hospital. There were two patient samples, i.e. the original group (226 patients), and the replication group (183 patients). The original sample (226 patients) were measured over a seven-month period starting in June 1959, and the second over a five-month period. The most salient aspects of the samples were a predominance of white patients over Negro patients, the age concentration of between 15 and 44, and the high frequency of patients in the lower socioeconomic groups. The social position was derived from Hollingshead's Two Factor Index of Social Position, which uses the factors of education and occupation in determining class level (Beck et al., 1961; Beck & Beamesderfer, 1974). Patients with organic brain damage or mental deficiency were excluded from the study.

The percentages among major diagnostic categories were: psychotic disorder, 41%; psychoneurotic disorder, 43%; and personality disorder, 16%. The three largest subgroups were: schizophrenic reaction, 28,2%; psychoneurotic depressive reaction, 25,3%; and anxiety reaction, 15,5%.

Reliability

Two methods of evaluating the internal consistency of the instrument were used. First, the protocols of 200 consecutive cases were analyzed. The score for each of the 21 categories was compared with the total score on the Beck Depression Inventory for each individual. With the use of the Kruskal-Wallis Non-Parametric Analysis of Variance by Ranks, it was found that all categories showed a significant relationship to the total score for the inventory. Significance was beyond the 0,001 level for all categories except category 8 (Weight-loss category), which was significant at the 0,01 level (Beck et al., 1961; Beck & Beamesderfer, 1974).

The second evaluation of internal consistency was the determination of the split-half reliability. Ninety-seven cases in the first sample were selected for this analysis. The Pearson r between the odd and even categories was computed and yielded a reliability coefficient of 0,86; with a Spearman-Brown correction, this coefficient rose to 0,93 (Beck et al., 1961; Beck & Beamesderfer, 1974).

Certain traditional methods of assessing the stability and consistency of inventories and questionnaires, such as the test-retest method and the inter-rater reliability method, were not appropriate for the appraisal of the Beck Depression Inventory (Beck et al., 1961). However two indirect methods of estimating the stability of the instrument were available. The first was a variation of the test-retest method. The inventory was administered to a group of 38 patients at two different times. At the time of each administration of the test, a clinical estimate of the depth of depression was made by a psychiatrist. The interval between the 2 tests varied from 2 to 5 weeks. It

was found that changes in the score on the inventory tended to parallel changes in the clinical depth of depression, thus indicating a consistent relationship of the instrument to the patients clinical state (Beck et al., 1961; Beck & Beamesderfer, 1974).

An indirect measure of inter-rater reliability was achieved as follows: each of the scores obtained by each of the 3 interviewers was plotted against the clinician ratings. A very high degree of consistency among the interviewers was observed for the mean scores respectively obtained at each level of depression. Curves of the distribution of the Beck Depression Inventory scores plotted against the depth of depression were notably similar, again indicating a high degree of correspondence among those who administered the inventory (Beck et al., 1961; Beck & Beamesderfer, 1974). The high correlation coefficient on the split-half item analysis and the significant relationship between the individual category scores and the total scores indicate the instrument is highly reliable, while studies of the internal consistency and stability of the instrument also indicate a high degree of reliability (Beck et al., 1961).

Validity

The BDI, which is an American Scale, was validated by Metcalfe and Goldman in 1965, against the criteria of clinical judgement by a psychiatrist.

Using the Spearman test, Bech, Gram, Dein, Jacobsen, Vitger, and Bolwig (1975) found that the total score of the BDI had an approximately equal degree of validity in comparison to the global clinical assessment, both for baseline ratings and for change ratings.

The means and standard deviations for each of the depth of depression categories were compared and the differences among the means were as expected; i.e. with each increment in the magnitude of depression there was a progressively higher mean score. The Kruskal-Wallis One-way Analysis of Variance by Ranks was used to evaluate the statistical

significance of these differences. For both the original group and the replication group, the p-value of these differences is $<0,001$. Since the Kruskal-Wallis test evaluates the over-all association between the scores on the Beck Depression Inventory and the depth of depression ratings, the Mann-Whitney U test was used to appraise the power of the Beck Depression Inventory to discriminate between specific depth of depression categories. It was found that all differences between adjacent categories (none, mild, moderate, and severe) in both studies were significant at 0,0004 with the exception of the differences between the moderate and severe categories, which has a p-value of $<0,1$ in the original group and $<0,02$ in the replication group (Beck et al., 1961).

A Pearson biserial r was computed to determine the degree of correlation between the scores on the Beck Depression Inventory and the clinical judgement of depth of depression. The highly significant relationship between the scores of the inventory and the clinical ratings of depth of depression and the power to reflect clinical changes in the depth of depression attest to the validity of this instrument.

Beck and Beamesderfer (1974) evaluated the concurrent validity by determining how well the test scores correlated with other measures of depression, for example, clinical evaluation and scores on other psychometric tests of depression. The concurrent validity of the BDI was supported by a number of studies comparing the test scores with clinicians' global ratings of depth of depression. The BDI was found to correlate 0,65 with the clinicians' ratings. Concurrent validity has also been demonstrated through comparisons with other standardized measures of depression. The correlation of the BDI scores with clinicians' ratings of depression and with other psychometric tests for depression constituted solid support for the concurrent validity of the BDI.

The construct validity of a measure is determined by setting up a number of hypotheses regarding the personality variable

(depression). If the hypothesis is confirmed in a study which uses the test as a criterion measure, the validity of the instrument is supported. Beck and Beamesderfer (1974) tested the following hypotheses concerning depressed patients:

- their dreams are characterized by masochistic content,
- they have a negative self-concept,
- they identify with the loser on projective tests dealing with success and failure,
- they have a history of deprivation which makes them vulnerable to depression in later life,
- they respond to experimentally induced failure with a disproportionate drop in self-esteem and increase in hopelessness,
- following a success experience, they show a significant subjective and objective improvement,
- they show a high correlation between intensity of depression and suicidal intent.

Using the BDI as the criterion measure, Beck and Beamesderfer (1974) found that their predictions were largely supported, thus establishing the construct validity of the BDI. Since all their hypotheses were confirmed Beck and Beamesderfer (1974) concluded that they has strong support for the construct validity of the BDI.

In conclusion it can be stated that the validity of the BDI has been demonstrated in various ways:

- (a) Demonstrating concurrent validity through correlations with clinical ratings: Bech (1981), reported biserial coefficients of 0,65 and 0,67 between clinical ratings and BDI scores,
- (b) Prediction of clinical change: Bech (1981) reported on the results of a number of studies in which a change in depth of depression was correctly predicted by the BDI score.
- (c) Correlation with other tests: Correlations with the Lubin (1965) Depression Adjective Check Lists were 0,40 and 0,66 while the correlation between the BDI score and the MMPI D Scale was equally acceptable (Bech, 1981).

Rationale for selecting the BDI

According to Bailey and Coppen (1976) the BDI has the advantage that it is a standardized form of measurement, enabling comparisons to be made without special training in rating. The BDI can also be completed rapidly by a patient under the supervision of an assistant or nurse in a few minutes.

The applicability of the BDI is in the milder degrees of depression and the BDI-subscale has been developed to aid general practitioners in the rapid screening of depressed patients (Beck & Beamesderfer, 1974; Bech, 1981).

A pertinent test of the inventory's power to assess the intensity of depression is its ability to reflect changes after a time interval (Beck et al., 1961). The findings of Beck et al., (1961) were that the Beck Depression Inventory reflects minor changes, since its range is much greater than clinicians ratings. This ability of the BDI to approximate clinical judgements of intensity of depression offers a number of advantages in its use for research purposes. It meets the problem of variability of clinical judgement of nosological entities and provides a standardized, consistent measure that is not sensitive to the theoretical orientation or idiosyncrasies of the individual who administers it. Since the inventory can be administered by an interviewer who is easily trained in its use, it is far more economical than a clinical psychiatric interview. The inventory provides a numerical score, and therefore it facilitates comparison with other quantitative data. The inventory reflects changes in the depth of depression over time and it provides an objective measure for judging improvement resulting from psychotherapy, drug therapy and other forms of treatment. According to Beck & Beamesderfer (1974), the BDI can objectively measure improvement in depression related to dietary treatment or therapy for obesity. The inventory is able to discriminate effectively among groups of patients with varying degrees of depression, thus the BDI is presented as a useful tool for research study of depression.

The BDI was utilized in the present study as it was developed to provide a quantitative assessment of the intensity of depression and because of its careful construction, validation and its wide use. Disadvantages of the BDI are that it is susceptible to the factors that invalidate self-rating scales, such as deliberate denial or exaggeration of the illness. The BDI contains no correction scale to estimate the patient's test-taking attitude or response set and is therefore clearly subject to voluntary distortion or falsification by the patient if he wishes (Bech, 1981).

According to Botha (1983), the BDI has the common defect of a self-rating scale, i.e., there is doubt concerning the scales' ability to distinguish moderate and severe degrees of depression from each other.

3.6.4 The Carroll Rating Scale for Depression (CRS)

Development

The Carroll Rating Scale (CRS) was developed over a period of ten years as a self-rating instrument for depression; this scale closely reflects the information content and specific items of the Hamilton Rating Scale (HRS).

Application and Marking Procedures

The CRS was designed as a direct self-rating adaptation of the original seventeen-item HRS. Items in the HRS that are scored 0 - 4 are represented in the CRS by four statements denoting progressively increasing severity of illness. Fifty two statements are randomly presented. The direction of a response indicative of depression is YES for forty statements, NO for twelve statements. Each statement is scored as one point towards the total score.

Carroll, Fielding, and Blaski (1973) states that the CRS could distinguish between four clinical levels of depression and that it was based on a solid research foundation.

Cut-off scores

Carroll et al., (1981) suggests that a score of 10 be used as a reasonable cut-off point if the CRS is to be used as a screening instrument for depression.

Norms

The CRS was completed by 119 adults aged 18-64, employed at the University of Michigan Medical Center. For reasons of confidentiality the sex of respondents was not identified, nor was it known whether a respondent was receiving psychiatric treatment. The respondents were a representative sample of the population and they covered a wide range of socioeconomic status. For comparison with results in this sample, Carroll et al., (1981) obtained psychiatrists' global ratings of severity, together with CRS ratings in patients being treated for depression. The concurrent ratings were obtained on 1191 occasions for over 200 patients. The global rating of severity of depression was made on a four-point (0-3) scale.

Reliability

The internal consistency of the CRS was examined in patients with endogenous depression by correlating individual item scores with the total score, in parallel with an identical analysis of matched HRS ratings. The split-half reliability of the CRS was tested by correlating the sums of odd- and even-numbered statements with each other and with the total score. Similarly, the effect of direction of response was examined by correlating the sums of YES- and NO-response items with each other and with the total score. For these analyses Carroll et al., (1981) used all available ratings, which included sequential ratings in many patients. Separate analyses revealed only minor changes in correlations within subjects compared to correlations across subjects.

The split-half reliability of the CRS was acceptable, being equal to that reported for the BDI (Beck, 1967). Carroll et

al., (1981) reported that the split-half reliability was calculated with a total of 3735 CRS ratings. The sum of the odd-numbered statements correlated well with the sum of the even-numbered statements ($r=+0,87$; $p < 0,001$). The sum of each half-set of statements correlated highly with the total score ($r=+0,74$; $p < 0,001$) in the same set of 3735 CRS ratings. The sum of the twelve NO statements correlated $+0,87$ with the total score, while the sum of the forty YES statements correlated $+0,98$ with the total score.

The overall performance of the CRS was consistent with the purpose for which it was designed.

Validity

The concurrent validity of the CRS is estimated by comparing CRS scores with HRS scores in patients suffering from endogenous depression. The clinical diagnoses were made as described by Carroll et al., (1981) and were supported in 98 per cent of cases by the Research Diagnostic Criteria (Spitzer et al., 1978). The two scores were obtained on the same day for each patient. Psychiatrists carrying out the HRS ratings received preliminary training in the use of this scale. In addition the CRS was compared with the BDI. For this comparison HRS, CRS and BDI ratings were obtained from 278 in-patients representing a range of psychiatric diagnoses, similar to the range employed by Beck (1967) for his validation of the BDI. Correlations and partial correlations among the three severity scales were determined.

According to Carroll et al., (1981) the results of their study indicate acceptable cross validation between the CRS and HRS, which is most obvious when the total scores are considered. Feinberg, Carroll, Smouse, and Rawson (1981) conclude that the CRS, like the HRS, differentiated between the four clinical degrees of depression.

Rationale for selecting the CRS

Carroll et al., (1981) state that the CRS, when compared to the BDI, is the self-rating scale that best distinguishes between the different degrees of depression, and that the CRS is a useful replacement for the BDI, with the additional advantage of closer correspondence to the HRS.

According to Carroll et al., (1981) the overall performance of the CRS is consistent with the purposes for which it was designed. As a self-rating scale for severity of depression it yields low scores in the general population. Virtually all patients with a global rating of more than mild depression recorded CRS scores greater than the cut-off score of 10 which was derived from the general population study.

Feinberg et al., (1981) states that the CRS can distinguish severe or moderate depression (e.g. on admission) from mild depression (e.g. at discharge). They recommend the use of the CRS as a screening instrument and state that the CRS provides an economical method of identifying patients who need more extensive evaluation of their depressive symptoms.

The Carroll Rating Scale was included in the present study since it is a useful instrument to distinguish between different levels of depression and in order to be able to compare the different depression measuring instruments. The disadvantage of the CRS is that it is susceptible to the factors which invalidate self-rating scales, i.e., deliberate denial or exaggeration of the illness, as well as distortion or falsification by the patient.

3.7 Rationale for using different depression scales

The decision to use different depression scales was based on recommendations by Wing et al., (1984), who state that several different measures of mood, each with appropriate psychometric properties, should be included in future research studies. They found that the intercorrelation between various measures of mood was often quite low, which

suggested that the scales might have measured different aspects of mood, and also that some instruments were possibly more sensitive to changes in mood induced by dieting. These findings supported the recommendation by Gormally (1977) that the best strategy was to use several independent measures of depression which complement each other and to compare the results obtained.

3.8 Methods of Statistical Analysis

The Statistical Analysis System (SAS) computer programme was used to analyse the data. Any results reported as significant had a probability value of $p \leq 0,1$. As the cell sizes were too small the chi-square test could not be used. Before a t-test was done to determine if there was a difference between Group 0 and Group 1 a F-test was done to determine which t-test should be done i.e. even or uneven variances. If the p-value of the F-test is very small, it must be accepted that the variances of the two groups are not equal and therefore the p-value for the t-test must be read from the unequal variances. The F-test was used to test for equal variances between the groups and thereafter the Student's t-test was used to determine significant differences between the groups.

CHAPTER 4 - RESULTS

This chapter contains the results obtained from the empirical investigation. Tables of results by questionnaire are provided. The first section contains a discussion of the biographical data for Group 0 (Standard Behaviour Therapy plus a very low-calorie diet) and Group 1 (Cognitive Behaviour Therapy plus a very low-calorie diet). The second part contains the results of the scores reported by the two experimental groups on the Beck Depression Inventory. The scores for the Control Group are tabulated in Appendix F.

As a result of the lower number of patients who reported for treatment as the programmes progressed, the cells had expected counts of less than 5 , which indicated that the Chi-Square test could not be used. The PROC MEANS procedure in the Statistical Analysis programme (SAS) was used. This procedure uses t-tests.

It should be noted here, that problems were experienced with the size of the Control Group. The difficulty experienced in obtaining a large enough number of patients to make up the Control Group was attributed to the fact that patients who had finally taken the decision to reduce their weight, were unwilling to be placed on a waiting list and to delay the commencement of their treatment programme by three months until August 1988 when the next treatment programme was scheduled to commence. The result was that the size of the Control Group was too small for any meaningful statistical comparisons to be conducted.

The following hypotheses were tested on the data obtained from the statistical analysis:

- H₁ There will be a difference between the results yielded by the different depression inventories.

- H₂ Depression will occur over time in the patients' participating in the different treatment programmes.

- H₃ A difference in the level of depression will occur over time in the patients participating in the different treatment programmes.
- H₄ There will be a fluctuation in the level of depression which occurs over time in the treatment programmes.
- H₅ There will be a difference in the depression which occurs during treatment and the outcome of treatment (weight-loss) in the different treatment programmes.

4.1 Biographical characteristics of groups.

The biographical questionnaire was included in this study to determine if the two experimental groups were comparable with regards to the specific areas as covered in the questionnaire (See Appendix A). A detailed tabulation of the raw scores of the Biographical Questionnaire is included in Appendix E.

The Statistical Analysis System (SAS) computer programme was used to analyse the data. Any results reported as significant, had a probability value of $p \leq 0,1$. As the cell sizes were too small, the chi-square test could not be used. The t-test was used to test for equal variances between the groups and thereafter the students t-test was used to determine significant differences between the groups. Before a t-test was done to determine whether there was a difference between Group 0 and Group 1, an F-test was done to determine which t-test should be used i.e. equal or unequal variances. If the p-value of the F-test is very small, it must be accepted that the variances of the two groups are not equal and therefore the p-value of the t-test for unequal variances must be used.

Educational and Occupational Characteristics of the experimental groups.

Educational qualifications of patients in Group 0 were closer to those of standards seven, eight and nine, while the patients' spouses' qualifications were closer to

standards eight and nine as compared to that of Group 1, where the patients' as well as their spouses' qualifications were closer to standards eight, nine and ten.

A larger number of patients in Group 0 were in category 13 (persons who are not remunerated, e.g. housewives), followed by category 10 (semi- or unskilled workers) as compared to those of Group 1, where a larger number of the patients were in category 9 (clerical) followed by category 13 (persons who are not remunerated). A larger number of spouses in Group 0 were in category 7 (semi-skilled or unskilled workers, followed by category 2 (professional B), as compared to those in Group 1 where the largest number were in category 7 (mine or associated workers), followed by category 4 (clerical).

Family Background

The majority of the patients in both the experimental groups reported their parents' marital associations as ranging between very happy to reasonably happy. However, a larger percentage of the patients in Group 1 reported that their parents' marriage had been very unhappy as compared with patients in Group 0. A larger percentage of the patients in Group 0 reported an exceptional relationship with their fathers than those in Group 1 did.

In Group 0 a higher percentage of the patients reported an exceptional relationship with their mother than those in Group 1 did.

In both groups the majority patients reported that their families mostly consisted of heavily built people.

Marriage Background

More patients in Group 0 reported that their marriage was slightly unhappier than those in Group 1 reported, where a higher percentage of patients reported that their marriages were reasonably happy.

Group 0 gave equal emphasis to marriage problems and overweight while the majority of patients in Group 1 reported overweight as the factor which influenced their sexual activities. In contrast to patients in Group 0, patients in Group 1 were more likely to discuss sexual problems with their spouses.

Results pertaining to the only significant difference found between Group 0 and Group 1 appear in Table 2.

Table 2

Results of t-tests of mean scores: comparison of Group 0 and Group 1. Question 29-Do you feel uncomfortable undressing in front of your spouse because you are overweight?

Group	N	Mean	Std Dev	t	DF	p
0	20	0,75	0,64			
				-2,1425	40,0	0,04*
1	22	1,18	0,66			

* A significant difference between the two experimental groups ($p \leq 0,1$).

Data from Table 2 indicates that Group 1 was significantly more uncomfortable ($p=0,04$) when undressing in front of their spouses, because of their being overweight. To determine whether this statistically significant difference was of practical significance, Cohen's d was used:

Cohen's d

$$d = \frac{\bar{x}_1 - \bar{x}_2}{s} = \frac{1,18 - 0,75}{0,65} = 0,6615$$

$$\text{where } s = \sqrt{\frac{s_1^2 + s_2^2}{2}}$$

As Cohen's d is less than 0,8 , it means that the significant difference has no practical significance.

History of Obesity

The majority of patients in both the experimental groups reported binge eating. While in Group 0 more patients attributed their lack of success in weight-reduction to lack of motivation than did patients in Group 1, Group 1 attributed lack of success to other factors, as well as to lack of motivation.

Reasons for participating in the weight-reduction programme ranged from categories 4 to 7, with category 4 (My clothes no longer fit) being the highest for Group 0 as compared to category 7 (My spouse/children/friends/family/colleagues say I am too fat) as the highest for Group 1.

From the above tables and the data in Appendix E it appears that the two experimental groups are fairly homogeneous. Using the Student t test only one variable i.e. Uncomfortable undressing in front of spouse (Table 2) - was found to indicate a significant difference ($p=0,04$) between the two experimental groups.

The results obtained from the biographical questionnaire indicate that the two groups are in fact comparable.

4.2 The Beck Depression Inventory (BDI).

This section contains the results and discussion of the Beck Depression Inventory.

H_1 - There will be a difference between the results yielded by the different depression inventories

The procedure followed to test if there was a difference between the results yielded by the different depression inventories was as follows:

(a) the PROC FREQ and PROC CORR procedure, the results of which are reported in Table 3.

Table 3

Results of frequency scores for the Beck Depression Inventory and the Carroll Rating Scale for the experimental groups combined.

Beck Depression Inventory Frequency Score	Carroll Rating Scale Frequency Score				TOTAL
	0	1	2	3	
0	118	7	3	1	129
1	31	5	1	0	37
2	42	13	17	3	75
3	17	10	11	28	66
TOTAL	208	35	32	32	307

Key: 0 = No Depression
 1 = Mild Depression
 2 = Moderate Depression
 3 = Severe Depression

The Chi-Square value for the above table is 144,81 (9 degrees of freedom) and the hypothesis that the two variables (BDI and CRS) are dependent is accepted (probability = 0,000). Therefore, it follows that there is a relationship between the CRS and the BDI scores. The 'strength' of this relationship was measured by the product-moment correlation, Pearson (0,581), Spearman (0,572) and Kendall (0,515) (SAS, 1985). All the correlation coefficients are positive and therefore, it follows that a positive relationship exists between the Beck Depression Inventory and the Carroll Rating Scale.

(b) to define the differences as :

$$\text{Mean Difference} = \text{Mean} (\text{Beck}_i - \text{Carroll}_i)$$

(Where i represents the week and Beck_i is the transformed Beck scale).

and then a PROC MEANS with a T and a PRT option was run. T gives the t-value which in this case was the Student t-value, to test if the averages are zero. If the average is zero it would mean that the Beck Depression Inventory and the Carroll Rating Scale values were equal. PRT is the probability of obtaining a larger value than T. The scores for the groups were combined, as the measuring instruments and not the two groups were compared.

In Table 4 the results of the t-tests for the differences between the Beck Depression Inventory and the Carroll Rating Scale for the experimental groups combined, are reported to monitor whether the mean scores are positive and whether the average is zero. If the mean scores are positive, it means that the Beck Depression Inventory measures higher than the Carroll Rating Scale and that the Beck Depression Inventory picks up depression when the Carroll Rating Scale does not. The population used in this study is considered a normal population and therefore the mean of difference scores are reported in Table 4.

For paired differences, Cohen's d is given by:

$$d = \frac{d}{\sqrt{2s_d}}$$

Table 4

Results of t-tests for the differences between the Beck Depression Inventory and the Carroll Rating Scale mean scores for the experimental groups combined.

Week No.	N	Mean of difference (d)	Std Dev of difference (S _d)	t	p*	Cohen d	Practical difference
Preparation:							
1	45	1,20	1,25	6,42	0,00*	0,67	No
5	40	0,87	1,14	4,87	0,00*	0,54	No
Treatment:							
7	43	0,58	0,93	4,09	0,00*	0,44	No
9	42	0,50	0,89	3,64	0,00*	0,40	No
11	29	0,52	0,91	3,06	0,00*	0,40	No
13	25	0,32	1,10	1,44	0,16	-	-
15	16	0,31	1,01	1,23	0,23	-	-
17	17	0,47	0,72	2,70	0,02*	0,46	No
Follow up:							
19	13	0,46	1,20	1,39	0,19	-	-
21	19	0,26	0,87	1,32	0,20	-	-
26	6	0,50	0,84	1,40	0,23	-	-
30	12	0,42	0,67	2,16	0,01*	0,44	No

* $p \leq 0,1$ = Significant difference

Summary:

A positive relationship was found between the Beck Depression Inventory and the Carroll Rating Scale and statistical significant differences occurred. These differences were positive throughout the programme. This indicates that the Beck Depression Inventory scale picks up depression before the Carroll Self Rating Scale . However, according to Cohen's *d*, these differences had no practical significance. It was therefore decided to use the Beck Depression Inventory scores for the rest of the analysis.

**H₂ - Depression will occur over time in the patients
participating in the different treatment programmes**

To determine if depression occurred over time in the patients' participating in the treatment programmes, a PROC MEANS with T and PRT options was run for the Beck Depression Inventory. The T-option calculates the Student t-values to test the hypothesis that the averages of the Beck Depression Inventory are zero. If the average were zero, it would mean that no depression occurred. PRT gives the probability that a larger t-value than T will occur in the null hypothesis. If PRT is large it implies that it is unlikely that the averages could be zero.

The results of the t-tests for Group 0 and Group 1 to determine if the averages of the Beck Depression Inventory are equal to zero, appear in Table 5 and 6.

Table 5

Results of the t-tests to determine whether depression occurred using the Beck Depression Inventory mean scores for Group 0.

Week No.	N	GROUP 0		t	p*
		Mean	Std Dev		
Preparation:					
1	26	3,35	1,02	16,77	0,00*
5	20	2,60	1,19	9,79	0,00*
Treatment:					
7	24	2,38	1,13	10,25	0,00*
9	20	2,15	1,23	7,84	0,00*
11	13	2,31	1,25	6,65	0,00*
13	14	1,86	1,09	6,32	0,00*
15	8	1,50	1,07	3,97	0,01*
17	7	1,57	0,79	5,28	0,00*
Follow up:					
19	5	1,80	1,30	3,09	0,04*
21	5	1,60	1,34	2,67	0,06*
26	2	2,50	2,12	1,67	0,34
30	4	1,75	0,96	3,66	0,04*

* $p \leq 0,1$ = Significant Difference

Summary:

Data from Table 5 indicate that depression occurred in Group 0 throughout the treatment programme, with the exception of Week Number 26 ($p=0,34$).

Table 6

Results of the t-tests to determine whether depression occurred, using the Beck Depression Inventory mean scores for Group 1.

Week No.	N	GROUP 1		t	p*
		Mean	Std Dev		
Preparation:					
1	19	3,21	0,98	14,33	0,00*
5	21	2,33	1,24	8,64	0,00*
Treatment:					
7	20	1,95	0,99	8,73	0,00*
9	22	2,09	1,27	7,73	0,00*
11	16	1,94	1,18	6,56	0,00*
13	11	2,18	1,25	5,79	0,00*
15	8	2,13	1,25	4,82	0,00*
17	10	2,00	1,15	5,48	0,00*
Follow up:					
19	8	2,25	1,16	5,46	0,00*
21	14	1,71	0,99	6,45	0,00*
26	4	1,50	1,00	3,00	0,06*
30	8	1,63	0,92	5,02	0,00*

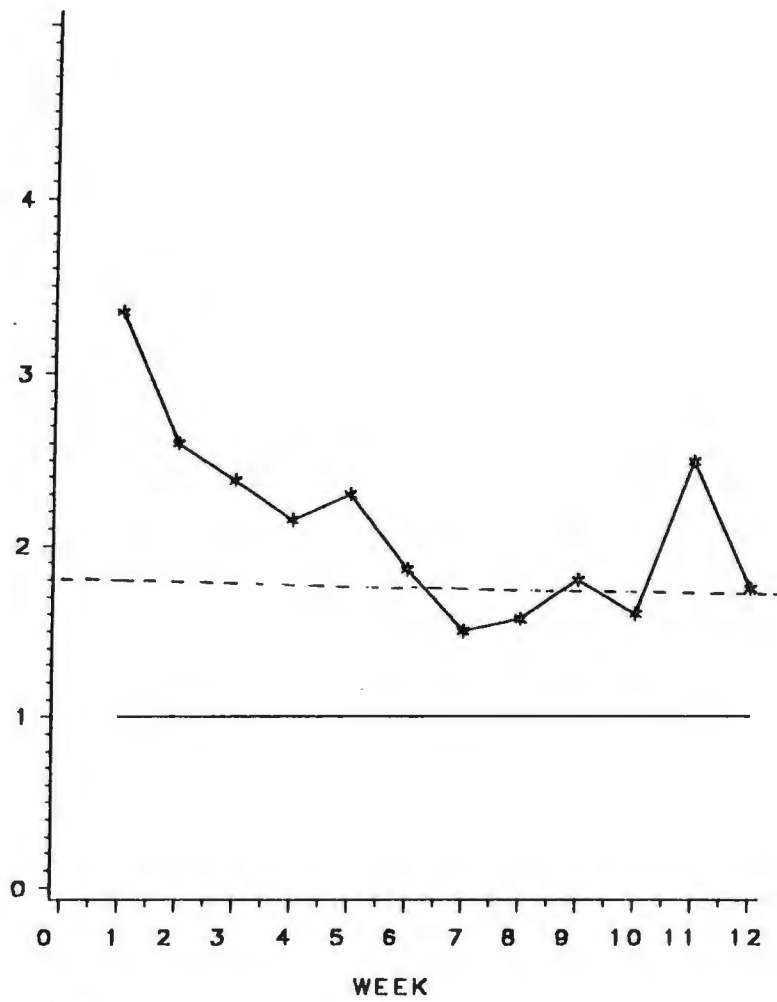
* $p \leq 0,1$ = Significant difference

Summary:

Data from Table 6 indicate that depression occurred throughout the treatment programme in patients in Group 1 as the probability values for the treatment weeks are all less than 10 % ($p \leq 0,1$).

To further test H_2 the sign test (Freund, 1988) was used. This analysis adopted a conservative approach where Week 12's depression level was used as norm (end of treatment programme score) and not 1 (no depression) (See Graph 1 for Group 0 and Graph 2 for Group 1).

GRAPH 1 :
BECK DEPRESSION INVENTORY MEAN SCORES FOR GROUP 0



KEY : 1 = No Depression
2 = Mild Depression
3 = Moderate Depression
4 = Severe Depression

For the sign test week 12's scores were used as the norm i.e. a score of just below 2 (Moderate depression). A positive sign (+) was allocated when the score was greater than the norm, a negative sign (-) when the score was below the norm and a zero (0) when the score equaled the norm.

Therefore, it follows that the sign allocated was :

Week Number: 1 2 3 4 5 6 7 8 9 10 11 12

Sign: + + + + + + - - 0 - + 0

If x = total of minuses accept the H_2 hypothesis as the probability of x being more than the total of minuses is smaller than or equal to 0,10.

where x = number of occurrences when the depression score was less than the depression score in week 12.

Table V (Binomial Table) for $n = 10 - 7 = 3$

$$x = 3$$

$$p = 0,5$$

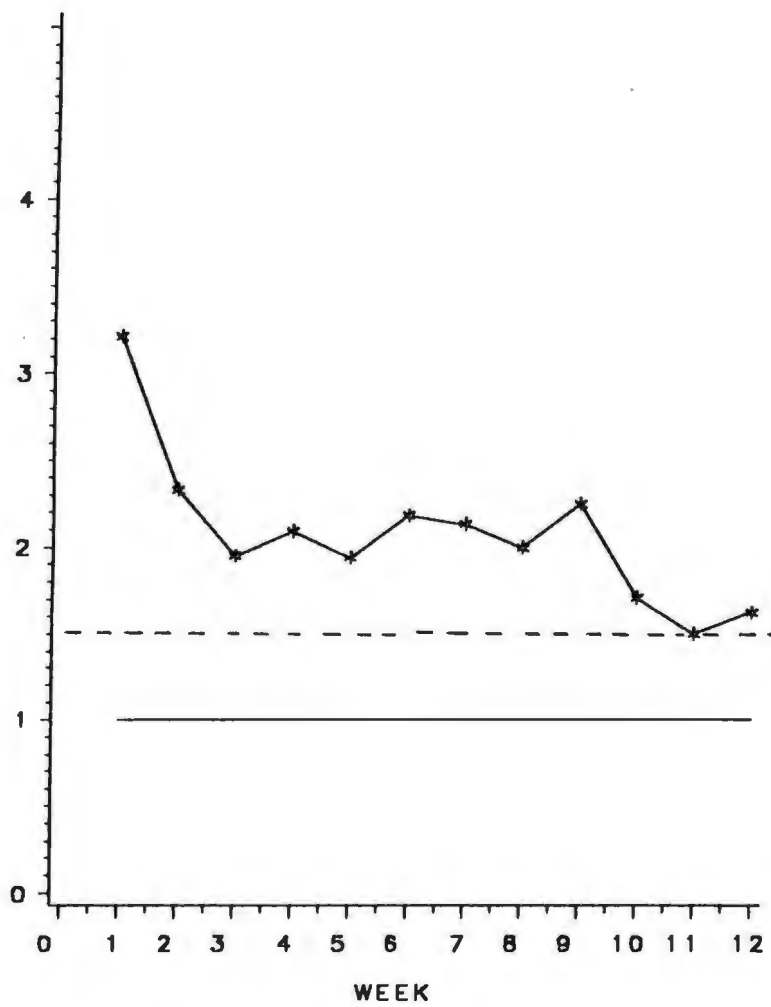
$$P(x \geq 3) = 0,117 + 0,044 + 0,010 + 0,001 \\ = 0,182$$

On a 10% probability can not accept the H_2 hypothesis. However, on a 20% probability can accept the H_2 hypothesis.

No transformation of the Beck Scale was used in the analysis i.e. the BDI was left at its values of 1 = no depression, 2 = mild, 3 = moderate and 4 = severe depression).

GRAPH 2:

BECK DEPRESSION INVENTORY MEAN SCORES FOR GROUP 1



- KEY:**
- 1 = No Depression
 - 2 = Mild Depression
 - 3 = Moderate Depression
 - 4 = Severe Depression

For the sign test week 12's scores were used as the norm i.e. a score of less than 2 (Moderate depression). A positive sign (+) was allocated when the score was greater than the norm, a negative sign (-) when the score was below the norm and a zero (0) when the score equaled the norm.

Therefore, it follows that the sign allocated was :

Week Number: 1 2 3 4 5 6 7 8 9 10 11 12

Sign: + + + + + + + + + + - 0

If x = total of minuses accept the H_2 hypothesis as the probability of x being more than the total of minuses is smaller than or equal to 0,10.

where x = number of occurrences when the depression score was less than the depression score in week 12.

$x = 1$

Table V (Binomial Table) for $n = 12 - 1 = 11$

$P(x \geq 1) = 0,005$

Therefore accept the H_2 hypothesis as depression higher than Week 12's depression occurred throughout the programme on a probability value of 10% as well as on a 1% probability value.

No transformation of the Beck Scale was used in the analysis i.e. the BDI was left at its values of 1 = no depression, 2 = mild, 3 = moderate and 4 = severe depression).

H₃ - A difference in the level of depression will occur over time in the patients' participating in the different treatment programmes.

To determine if there was a difference in the level of depression which occurred over time in the patients participating in the treatment programme the GLM procedure in the SAS programme was used. The GLM procedure performs an analysis of variance for unbalanced designs. For each observation the ANOVA model predicts the response, often by a sample mean. The difference between the actual and the predicted response is the residual error. Analysis-of-variance procedures fit parameters to minimize the sum of squares of residual errors. Thus the method is called least squares. The variance of the random error, σ^2 , is estimated by the mean square error (MSE or s^2). This analysis-of-variance model can be written as a linear model, an equation to predict the response as a linear function of parameters and design variables:

$$y_i = B_0x_{0i} + B_1x_{1i} + \dots + B_kx_{ki} + \Sigma_i, \quad i = 1 \dots n$$

where y_i is the response for the i^{th} observation, B_k are unknown parameters to be estimated, and x_{ij} are design variables. Design variables for analysis of variance are indicator variables, that is, they are always either 0 or 1. When models are expressed in the framework of linear models, hypothesis tests are expressed in terms of a linear function of the parameters.

The GML procedure with a Bonferroni t-test which performs tests of differences between means for all main-effect means in the MEANS statement was used. This was done to test if there was a significant difference between the means of all main effects with a $\alpha = 0,1$ (alpha = 10%) significant level. The results pertaining to the comparison of levels of depression between the experimental groups, are reported in Table 7.

Table 7

Results of comparisons of levels of depression (Beck Depression Inventory mean scores) between the experimental groups (Group 0 and Group 1) using a 10% probability of significant difference.

Week No.	N	Df	MSE	F	t	Significant Difference
Preparation:						
1	45	43	1,00	0,20	1,68	NO
5	41	39	1,47	0,49	1,68	NO
Treatment:						
7	44	42	1,16	1,70	1,68	NO
9	42	40	1,56	0,02	1,68	NO
11	29	27	1,47	0,67	1,70	NO
13	25	23	1,36	0,48	1,71	NO
15	16	14	1,35	1,16	1,76	NO
17	17	15	1,05	0,72	1,75	NO
Follow Up:						
19	13	11	1,48	0,42	1,79	NO
21	19	17	1,18	0,04	1,74	NO
26	6	4	1,88	0,71	2,13	NO
30	12	10	0,86	0,05	1,18	NO

Significant Difference = $p \leq 0,1$

Summary:

Data in Table 7 indicate that there is no significant difference between the levels of depression of the two experimental groups on a 10% probability scale throughout the treatment programme.

H₄ - There will be a fluctuation in the level of depression which occurs over time in the treatment programmes.

In order to determine if there was a fluctuation in the level of depression which occurred over time in the treatment groups. Anova was used and it stated that there was a difference ($p=0,01$). To determine where the difference occurred t-tests were done on the differences between the Beck Depression Inventory scores for the different weeks. Using the PROC MEANS procedure, which measures whether the mean (difference between the Beck Depression Inventory scores for the different weeks) equals zero. When the p is very small, it indicates that the difference is not equal to zero and the two weeks being compared, therefore differ.

A Bonferonni adjustment can be made: The p^* values are multiplied with 66 as 66 different tests are actually carried out. The differences between Week 1 and the other weeks with the exception of weeks 19 and 26 remain significant. This proves that differences in the levels of depression occur over time (Week 1 clearly differs from the other weeks).

The maximum number of individuals were used at each comparison (i.e. all the patients who were present) in order to maximize the power of the tests.

The results pertaining to the t-tests for the differences between weekly mean scores for the Beck Depression Inventory appear in Table 8, 9, 10, 11, 12, 13, 14, 15, 16, 17 and 18.

Table 8

Results of the t-tests of the differences per patient between week 1 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
5 - 1	38	-0,71	1,09	-4,02	0,0003*
7 - 1	43	-1,09	1,15	-6,23	0,0001*
9 - 1	38	-1,18	1,23	-5,95	0,0001*
11 - 1	28	-1,18	1,19	-5,25	0,0001*
13 - 1	24	-1,38	1,17	-5,74	0,0001*
15 - 1	15	-1,67	1,23	-5,23	0,0001*
17 - 1	16	-1,50	1,03	-5,81	0,0001*
19 - 1	12	-1,25	1,29	-3,36	0,0063*
21 - 1	18	-1,56	1,25	-5,29	0,0001*
26 - 1	6	-1,17	1,33	-2,15	0,0842*
30 - 1	11	-1,64	1,12	-4,85	0,0007*

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 8 indicate that there is a significant difference between the levels of depression in all the treatment weeks, as compared to week 1 (Total $p=0,09$). The negative mean scores indicate that the depression scores for week 1 were higher. The data indicate that there was a decline in the level of depression from week 1 onwards.

Table 9

Results of the t-tests of the differences per patient between week 5 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
7 - 5	37	-0,29	0,70	-2,58	0,0142*
9 - 5	34	-0,38	0,89	-2,51	0,0171*
11 - 5	28	-0,46	0,96	-2,55	0,0166*
13 - 5	23	-0,74	1,14	-3,12	0,0050*
15 - 5	15	-1,07	1,22	-3,38	0,0045*
17 - 5	16	-1,00	1,21	-3,30	0,0048*
19 - 5	11	-0,73	1,56	-1,55	0,1519
21 - 5	18	-0,61	1,14	-2,26	0,0369*
26 - 5	6	-0,83	1,17	-1,75	0,1412
30 - 5	11	-0,36	1,12	-1,08	0,3069

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 9 indicate that not all the differences between week 5 and the rest of the treatment weeks are significant. However, the negative mean scores indicate that the depression score for week 5 was still higher than that of the rest of the treatment weeks. This correlates with the decline in the level of depression as noted in Table 8.

Table 10

Results of the t-tests of the differences per patient between week 7 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
9 - 7	37	-0,11	0,99	-0,66	0,5125
11 - 7	27	-0,19	1,04	-0,93	0,3629
13 - 7	23	-0,39	1,23	-1,52	0,1424
15 - 7	16	-0,63	1,26	-1,99	0,0655*
17 - 7	17	-0,65	1,11	-2,39	0,0293*
19 - 7	13	-0,23	1,30	-0,64	0,5345
21 - 7	19	-0,16	1,12	-0,62	0,5461
26 - 7	6	-0,33	1,03	-0,79	0,4650
30 - 7	12	-0,25	0,97	-0,90	0,3889

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 10 indicate that only the depression scores for weeks 15 and 17 differ significantly from depression scores for week 7. However, the mean of the differences between the weeks is still negative which, indicates that the level of depression registered in week 7 is still higher than that of the remaining treatment weeks. This correlates with the decline in the level of depression as noted in Table 8 and 9.

Table 11

Results of the t-tests of the differences per patient between week 9 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
11 - 9	28	-0,07	0,89	-0,42	0,6777
13 - 9	24	-0,17	0,96	-0,85	0,4053
15 - 9	16	-0,44	0,81	-2,15	0,0483*
17 - 9	17	-0,18	0,95	-0,77	0,4554
19 - 9	13	0,08	1,04	0,27	0,7938
21 - 9	18	-0,06	0,54	-0,44	0,6676
26 - 9	6	-0,50	0,55	-2,24	0,0756
30 - 9	12	-0,08	0,67	-0,43	0,6742

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 11 indicate that only week 15 ($p=0,0483$) differ significantly from data for week 9. However, the mean scores are still mostly negative with, only one positive score in week 19 (0,08). The downward trend in the depression scores as noted in the previous Tables 8, 9 and 10 is less evident in Table 11.

Table 12

Results of the t-tests of the differences per patient between week 11 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
13 - 11	21	-0,29	1,01	-1,30	0,2084
15 - 11	12	-0,42	0,90	-1,60	0,1372
17 - 11	15	-0,20	0,86	-0,90	0,3840
19 - 11	9	0,22	0,83	0,80	0,4468
21 - 11	15	-0,13	0,64	-0,81	0,4332
26 - 11	4	0,00	0,00	-	-
30 - 11	10	0,10	0,57	0,56	0,5911

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 12 indicate that there are no significant differences between the mean scores of week 11 and the remaining treatment weeks. The three positive mean scores in weeks 19, 26 and 30 are a continuation of the trend observed in Table 11 of a levelling off of the decline in the level of depression with greater fluctuation occurring, as noted by the presence of both negative and positive means of difference scores.

Table 13

Results of the t-tests of the differences per patient between week 13 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
15 - 13	14	-0,29	0,61	-1,75	0,1039
17 - 13	16	-0,38	0,50	-3,00	0,0090*
19 - 13	11	0,09	0,54	0,56	0,5884
21 - 13	15	0,00	0,53	0,00	1,0000
26 - 13	5	0,00	0,00	-	-
30 - 13	10	0,00	0,67	0,00	1,0000

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 13 indicate that the depression score ($p=0,0090$) for week 17 differ significantly from that of week 13. Only weeks 15 and 17 have negative mean of difference scores i.e. the depression score for week 13 was higher. The increasing number of weeks in which the mean score is now positive, indicates that from week 19 to week 30 the mean depression scores are higher than the mean depression scores for week 13. This indicates that the level of depression was no longer declining. This concurs with the trend observed in Tables 11 and 12.

Table 14

Results of the t-tests of the differences per patient between week 15 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
17 - 15	13	0,15	0,68	0,81	0,4363
19 - 15	9	0,44	0,73	1,84	0,1038
21 - 15	11	0,18	0,60	1,00	0,3409
26 - 15	3	0,00	0,00	-	-
30 - 15	7	0,14	0,69	0,55	0,6036

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 14 indicate that there is no significant difference between the mean of difference depression scores for week 15 and the remaining treatment weeks. The positive mean of difference scores indicate that the depression scores for the remaining weeks were higher than that for week 15. This trend of rising depression scores concurs with data in Table 13.

Table 15

Results of the t-tests of the differences per patient between week 17 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
19 - 17	11	0,27	0,47	1,94	0,0816*
21 - 17	12	0,08	0,90	0,32	0,7545
26 - 17	4	0,50	0,58	1,73	0,1817
30 - 17	9	0,33	0,50	2,00	0,0805*

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 15 indicate that there are significant differences in the mean depression scores of week 17 as compared to that of week 19 ($p=0,0816$) and week 30 ($p=0,0805$) during which the mean depression scores for week 17 were higher. However, the positive mean of difference scores indicate that there is no decline in the level of the mean depression scores. This concurs with data in Table 14.

Table 16

Results of the t-tests of the differences per patient between week 19 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
21 - 19	11	-0,18	0,75	-0,80	0,4405
26 - 19	5	-0,20	0,45	-1,00	0,3739
30 - 19	6	0,00	0,63	0,00	1,0000

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data in Table 16 indicate that there is no significant difference between the mean depression scores for week 19 and the remaining treatment weeks. However, there are negative mean of difference scores for week 21 and 26 which indicate that the mean depression scores of week 19 are higher. This would indicate a decline in the level of depression during weeks 21 and 26.

Table 17

Results of the t-tests of the differences per patient between week 21 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
26 - 21	6	-0,17	0,41	-1,00	0,3632
30 - 21	11	0,00	0,63	0,00	1,0000

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 17 indicate that there are no significant differences between the levels of mean depression scores between week 21 and weeks 26 and 30. However, the negative mean of difference score for week 26 indicates that the mean depression score for week 21 was higher, which shows a decline in the mean depression score from week 21 to 26. The positive mean score for week 30 indicates a slight increase in the mean depression score.

Table 18

Results of the t-tests of the differences per patient between week 26 and the weekly mean scores for the Beck Depression Inventory for the two experimental groups combined (Group 0 and Group 1).

Week No. Minus Week No.	N	Mean of Difference	Std Dev. of Difference	t	p*
30 - 26	4	-0,25	0,50	-1,00	0,3910

* $p \leq 0,1$ = Significant difference between the two weeks

Summary:

Data from Table 18 indicates that there is no significant difference between the mean depression scores for week 26 and week 30. However, the negative mean of difference score indicates that the mean depression scores for week 26 are higher than those for week 30. This indicates a decline in the level of depression between week 26 to week 30.

SUMMARY OF THE FINDINGS FROM TABLE 8 TO TABLE 18:

A fluctuation in the level of depression occurred over time. Data from Tables 8 to 18 indicate, that although there is a significant downward trend in the level of mean depression scores between weeks 1 and 7, this decline becomes less marked as from week 9, with fewer significant differences occurring and positive mean depression scores indicating upward trends in the level of the mean depression scores. However, it must be noted that the level of mean depression scores remains considerably lower than those reported for week 1.

H₅ - There will be a difference in the depression which occurs during treatment and the outcome of treatment (weight-loss) in the different treatment programmes.

To determine the occurrence of a difference in the depression which occurred during treatment and the outcome of treatment (weight-loss) in the different treatment programmes, the PROC TTEST was run.

The results of the PROC TTEST analysis on the mean scores for the Beck Depression Inventory - comparison of weight-loss and weight-gain for Group 0, are reported in Table 19 and in Table 20 for Group 1.

In addition to the above, the following formula (du Toit, 1975) was used to determine whether the significant difference ($p \leq 0,1$) between the mean depression scores had a practical significance or implication:

$$w^2 = \frac{t^2 - 1}{t^2 + n_1 + n_2 - 1}$$

(Where w^2 can be regarded as an indication of the proportion or percentage of the total variance for which the experimental treatment is responsible).

Although the calculation for practical difference was conducted on the significant difference in both Table 19 and 20 the calculation is only reported after Table 19 as no practical difference was found for Table 20.

Table 19

Results of the mean scores for the Beck Depression Inventory: Comparison of Weight Loss and Weight Gain for Group 0.

Week No.	Group 0 ⁺										
	WEIGHT LOSS					WEIGHT GAIN					
	N	Mean	Std Dev.	t	DF	N	Mean	Std Dev.	t	DF	p*
Preparation:											
5	7	3,42 [#]	0,98	2,75	14,3	10	2,00 [#]	1,15	2,66	15,0	0,02*
Treatment:											
7	17	2,35	1,17	0,30	7,9	6	2,17	1,32	0,32	21,0	0,77
9	12	1,91	1,24	-0,77	8,0	6	2,50	1,64	-0,85	16,0	0,47
11	10	1,90	1,28	-2,48	2,6	2	3,50	0,71	-1,66	10,0	0,10
13	9	1,67	1,12	-1,34	2,3	2	2,50	0,70	-0,99	9,0	0,30
15	4	1,00	0,00	-	-	1	4,00	0,00	-	3,0	<u>-a</u>
17	6	1,50	0,83	-	-	1	1,00	0,00	0,55	5,0	<u>-b</u>
Follow Up:											
19	5	2,20	1,64	-	-	1	1,00	0,00	0,67	4,0	<u>-b</u>
21	1	1,00	0,00	-	-	4	1,75	1,50	-0,45	3,0	<u>-b</u>
26	1	4,00	0,00	-	-	0	0,00	0,00	-	-	<u>-a</u>
30	3	2,00	1,00	-	-	1	1,00	0,00	0,87	2,0	<u>-b</u>

*p≤0,10 = Significant Difference

+ = Standard Behaviour Therapy plus a very low-calorie diet

= Practical Difference between the mean depression scores:

Practical Difference:

$$w^2 = \frac{t^2 - 1}{t^2 + n_1 + n_2 - 1}$$

(Where w² can be regarded as an indication of the proportion or percentage of the total variance for which the experimental treatment is responsible).

$$\begin{aligned}
t &= \frac{\bar{X}_1 - \bar{X}_2}{\sqrt{(S_{x1})^2 + (S_{x2})^2}} \\
&= \frac{3,42 - 2,00}{(0,97)^2 + (1,15)^2} \\
&= \frac{1,42}{1,50446} \\
&= 0,944 \\
w^2 &= \frac{(0,94)^2}{(0,94)^2 + 7 + 10 - 1} \\
&= \frac{0,8908}{16,8908} \\
&= 0,053
\end{aligned}$$

The difference between the two groups (weight-loss and weight-gain in Group 0) explains only 5% of the variation in their depression scores.

Note:

a = Where the mean score and the standard deviation are both 0, it is not correct to assume that the variances are equal as the variance of one group is zero. It is, therefore, impossible to obtain a viable t-test as the unequal variance t-test uses the variances as denominators. It is, therefore not possible to calculate the t-value and the p-value for weeks 15 and 30 for Group 0.

b = Where one of the N's denotes one, the standard deviation is zero.

The t-statistic for unequal variances is given by:

$$t = \frac{\bar{x}_1 - \bar{x}_2}{\sqrt{\frac{s_1^2}{N_1} + \frac{s_2^2}{N_2}}}$$

The decision rule to determine whether this t-value is significant, is given by:

$$\text{Reject } H_0 \text{ if } t^1 > \frac{(t_1 W_1 + t_2 W_2)}{W_1 + W_2}$$

$$\text{or } t^1 < \frac{(t_1 W_1 + t_2 W_2)}{W_1 + W_2}$$

otherwise do not reject H_0
(no difference between means).

where t_1 = critical value at alpha level of significance with $n_1 - 1$ degree of freedom.

and t_2 = critical value of alpha level of significance with $n_2 - 1$ degree of freedom.

where w_1 and w_2 = weights

if either n_1 or n_2 is equal to one, then the corresponding value of $n_i - 1$ equals zero. Therefore it is impossible to determine the critical t-value for weeks 17, 19, 21 and 30 for Group 0) (Berenson and Levine, 1986).

Summary:

Data from Table 19 indicate that the only significant difference between the mean depression scores for Group 0, weight-loss and weight-gain, occurred during week 5 ($p=0,02$). This is not a practical difference as it explains only 5% of the variation in their depression scores.

Table 20

Results of the mean scores for the Beck Depression Inventory: Comparison of Weight Loss and Weight Gain for Group 1.

Week No.	Group 1 ⁺										
	WEIGHT LOSS					WEIGHT GAIN					
	N	Mean	Std Dev.	t	DF	N	Mean	Std Dev	t	DF	p*
Preparation:											
5	17	2,23	1,15	-1,24	6,2	5	3,00	1,22	-1,29	20,0	0,26
Treatment:											
7	17	2,18	1,01	1,78	8,1	4	1,50	0,58	1,27	19,0	0,11
9	17	2,24	1,20	1,11	10,2	6	1,67	1,03	1,03	21,0	0,29
11	16	2,00	1,15	-	-	1	3,00	0,00	-0,84	15,0	- ^b
13	0	0,00	0,00	-	-	0	0,00	0,00	-	-	- ^a
15	10	2,00	1,15	-	-	1	1,00	0,00	0,83	9,0	- ^b
17	1	2,14	1,21	0,19	4,7	3	2,00	1,00	0,18	8,0	0,85
Follow Up:											
19	6	2,33	0,82	-	-	1	1,00	0,00	1,51	5,0	- ^a
21	12	1,83	1,03	2,80	11,0	2	1,00	0,00	1,11	12,0	0,01*
26	5	1,40	0,89	-	-	0	0,00	0,00	-	-	- ^b
30	7	1,71	0,95	-	-	1	1,00	0,00	0,70	6,0	- ^a

* $p \leq 0,1$ = Significant Difference

+ = Cognitive Behaviour Therapy plus a very low-calorie diet

Note:

a = Where the mean score and the standard deviation are both 0, it is not correct to assume that the variances are equal as the variance of one group is zero. It is, therefore, impossible to obtain a viable t-test as the unequal variance t-test uses the variances as denominators. It is, therefore, not possible to calculate the t-value and the p-value for weeks 13, 19 and 30 for Group 1.

b = Where one of the N's denotes one, the standard deviation is zero.

The t-statistic for unequal variances is given by:

$$t = \frac{\bar{x}_1 - \bar{x}_2}{\sqrt{\frac{s_1^2}{N_1} + \frac{s_2^2}{N_2}}}$$

The decision rule to determine whether this t-value is significant, is given by:

$$\text{Reject } H_0 \text{ if } t^1 > \frac{(t_1 W_1 + t_2 W_2)}{W_1 + W_2}$$

$$\text{or } t^1 < \frac{(t_1 W_1 + t_2 W_2)}{W_1 + W_2}$$

otherwise do not reject H_0
(no difference between means).

where t_1 = critical value at alpha level of significance with $n_1 - 1$ degree of freedom.

and t_2 = critical value of alpha level of significance with $n_2 - 1$ degree of freedom.

where w_1 and w_2 = weights

if either n_1 or n_2 is equal to one, then the corresponding value of $n_i - 1$ equals zero. Therefore it is impossible to determine the critical t-value for weeks 11, 15, and 26 for Group 1) (Berenson and Levine, 1986).

Summary:

Data from Table 20 indicate that the only significant difference between the mean depression scores for weight-loss and weight-gain in Group 1 occurred during week 21 ($p=0,01$). However, this significant difference is not a practical one. Therefore, it appears from the analysis of the data in Table 20, that the weight lost by patients had no influence on the depression scores for Group 1.

CHAPTER 5 - INTERPRETATION AND DISCUSSION

The first section of this chapter is devoted to the evaluation of the research hypotheses based on the results in Chapter 4. The second part contains a discussion of the implications of the findings for the planning and design of weight-reduction programmes, followed by a discussion of problems experienced, as well as several recommendations. The chapter is concluded with an overview of the study.

5.1 Evaluation of the Research Hypotheses

H₁ - There will be a difference between the results yielded by the different depression inventories

Data from this study (Tables 3 and 4) substantiated this hypothesis and therefore, H₁ is accepted.

The relationship between the Beck Depression Inventory and the Carroll Rating Scale was investigated in Table 3. The data indicates that there is a positive correlation between the Beck Depression Inventory and the Carroll Rating Scale implying that both these inventories detect depression. However, as is evident in Table 4, the mean difference between the Beck Depression Inventory and the Carroll Rating Scale scores differs significantly from zero and is positive throughout the programme. This indicates that the Beck Depression Inventory scores are higher than the Carroll Rating Scale scores. The implication is that the Beck Depression Inventory will pick up depression before the Carroll Rating Scale. However, these differences had no practical significance. These findings concur with the conclusions reached by Gormally (1977), Wadden et al., (1986, 1984) and Wing et al., (1983, 1984) who reported that the instrument used to measure depression, influenced the results obtained as some instruments are possibly more sensitive to changes in mood induced by dieting.

However, the data do not support the findings of Carroll et al., (1981) in so far as that the Carroll Rating Scale is superior to the Beck Depression Scale with regards to

distinguishing between the different levels of depression. It was found that the Beck Depression Inventory reported scores ranging from severe to mild depression, while the Carroll Rating Scale was able to only register scores ranging from just higher than mild depression to no depression at all. This indicated that the Beck Depression Scale distinguishes more effectively between the different levels of depression (See Appendix H).

The conclusion drawn here, is that for the present study, the Beck Depression Inventory is a more applicable measuring instrument, when viewed from the general goal of the study i.e. to assess the occurrence and fluctuation of depression in obese patients in two different treatment programmes, as the Beck Depression Inventory reflected depression scores throughout the total range (0 = no depression to 3 = severe depression).

H₂ - Depression will occur over time in the patients participating in the different treatment programmes

Data from this study (Table 5 and 6) substantiated this hypothesis and therefore, H₂ is accepted.

The data in Tables 5 and 6 indicate that the probability of the average depression scores being zero, remained consistently less than 10% ($p \leq 0,1$). The only exception being week 26 for Group 0 when only two patients reported for treatment.

These findings concur with the findings of Stunkard (1987), Wadden and Stunkard (1986) and Wing et al., (1983, 1984) who supported the findings of Simon (1963), as well as that of Leckie and Withers (1967), that depression was a contributing factor in the development and maintenance of obesity.

The conclusion drawn here, is that depression consistently occurred throughout the different treatment programmes in both Group 0 and Group 1's patients.

H₃ - A difference in the level of depression will occur over time in the patients participating in the different treatment programmes

Data in Table 7 did not substantiate this hypothesis and therefore, H₃ is rejected.

Implications are that the levels of depression for Group 0 and Group 1 are not significantly different. This indicates that the different treatment programmes i.e. the inclusion of a cognitive component into the standard behaviour therapy plus very low-calorie diet, did not result in a significant difference between the level of depression which occurred in the experimental groups. However, it should be noted that the cognitive component of the treatment programme for Group 1 does not focus on depression as such, but on improved self-control and on eating behaviour aimed at increased weight-loss.

H₄ - There will be a fluctuation in the level of depression which occurs over time in the treatment programmes

This hypothesis is substantiated by data in this study (Tables 8 to 18) and therefore, H₄ is rejected.

It is apparent from Table 8 that there is a significant decrease in the level of depression when comparing week 1 with the remaining treatment weeks ($p=0,09$), with the depression scores of week 1 being higher than that of the remaining treatment weeks. This trend of declining levels of depression continues during weeks 5, 7 and 9. However, by week 10 there is a leveling off of the previously noted decline and a more marked fluctuation occurs in the level of reported depression as indicated by the presence of both positive and negative mean of difference depression scores. This trend continues during week 13 with the data indicating that the level of depression as reported by the patients is no longer declining.

By week 15 of the treatment programme, there are no significant differences between the mean of difference

depression scores and those of the remaining treatment weeks (Table 14).

The conclusion drawn here, is that there is a sharp decline in the level of depression which occurs during week 1 of the treatment programme and the level of depression reported during the remaining treatment weeks. Although this sharp decrease in the level of depression reported levels off by week 10, the level of depression continues to fluctuate during the remaining treatment weeks. It is evident from the data in this study that the level of reported depression is considerably lower at the end of the treatment programme.

The above findings concur with the findings of Stunkard and Smoller (1986) as well as with those of Stunkard and Rush (1974) who found that the level of depression fluctuated during the treatment of obesity as a reaction to the weight reduction programme.

Data from this study (Table 8 to 18) also support the findings of Weighill and Buglass (1984) and Wing et al., (1984) who found that depression which was already present prior to treatment (as seen in Tables 5 and 6) eased during a behaviour therapy weight-reduction programme (as seen in Tables 8 to 18). The data, however, contradicted findings of Stunkard and Rush (1974) who stated that weight-reduction treatment programmes resulted in adverse psychological complications.

H₅ - There will be a difference in the depression which occurs during treatment and the outcome of treatment (weight-loss) in the different treatment programmes

Data in Tables 19 and 20 did not substantiate this hypothesis and therefore H₅ is rejected for both Group 0 and Group 1.

Data in Table 19 for Group 0 indicate that only during week 5 is there a statistically significant difference in the mean depression scores for the patients who lost weight and those who gained weight ($p=0,02$). Patients who lost weight

reported a higher mean (3,42) depression score than those who gained weight (2,00). This statistical difference was not a practical significance. However, throughout the remaining treatment weeks, no significant difference was found between the mean depression scores of the patients in Group 0 who gained or lost weight. The hypothesis is therefore, rejected for Group 0.

Data from Table 20 for Group 1 indicate that the only significant difference between the mean depression scores for weight-loss and weight-gain patients in Group 1, occurs during week 21 ($p=0,01$). This significant difference is not a practical one. The hypothesis is, therefore, rejected for Group 1.

The conclusion drawn here is that although there is a statistically significant difference but not a practical significance in the mean depression scores for patients who gained and lost weight for Group 0 during week 5 and for Group 1 during week 21 (with the patients who lost weight reporting higher mean depression scores than those who gained weight), this trend is not consistent throughout the treatment programmes. The hypothesis is, therefore, rejected.

Data in Tables 19 and 20 indicate a decrease in the mean depression scores in Group 0 and Group 1 for patients who lost weight as the treatment programme progressed. This data concurs with the findings of Shipman and Plesset (1963), Pitta et al., (1980), Wing et al., (1983) and Weighill and Buglass (1984) who found that the patients who were successful in losing weight, reported lower levels of depression as the treatment programme progressed. However, data in Tables 19 and 20 must be interpreted with caution as the differences between the depression scores for weight-loss/gain were not significant.

The observed trend of fewer patients reporting for treatment (See Tables 19 and 20) as the programmes progressed complicates the statistical analysis. This decline in patients was also noted by Silverstone and Dinello in 1966,

Stunkard and Rush (1974) as well as by Polivy and Herman (1976).

In conclusion, it must be stressed that because of the small sample size, results should be interpreted with caution. Even when statistically significant findings emerged, the differences involved were usually very small. Thus findings from this study should be generalized to other populations with restraint. Nonetheless, results were usually in line with findings from similar studies executed elsewhere and with different populations.

5.2 Implications of findings for the planning and design of a weight-reduction programme

Measuring Instruments

It is evident from the data in the study that different results were obtained from different measuring instruments. The implication is that measuring instruments must be carefully screened to ensure that they are capable of measuring levels or factors as required by the research objective.

Frequency of Assessment

The observed fluctuation in the level of reported depression as from week 11 in the treatment programmes concurs with findings in the literature that pre- and post-measurement are inadequate to monitor for any adverse effects of the treatment programmes. The implication is that continual monitoring is necessary to ensure successful adjustment of the planned programme to the therapeutic needs of the patients throughout the duration of the programme. This would increase the probability of success.

Content of treatment programme

Confirmation was obtained in the present study of no adverse effects reported by patients on a behaviour therapy, plus a very low-calorie diet weight-reduction programme. A decline

in the level of reported depression also occurred, which provides a sound rationale for the use of this type of treatment programme.

A further recommendation would be the inclusion of a cognitive component into the treatment programme, which focuses on depression as such, in order to evaluate whether this would have a marked effect on the lowering of the level of depression reported by patients throughout the treatment programme.

Occurrence of Depression

Data in the study confirm that depression occurred at the start of the treatment programme as well as throughout the entire period of the programme. The implication is, that weight-reduction programmes should cater for the occurrence and effect of depression on eating behaviour and build a therapeutic intervention into the design of the programme.

Results in this study have shown that depression declined from week 1 to week 9 and that by week 10 there was a levelling off, with a greater degree of fluctuation occurring in the level of depression during the remaining treatment weeks. The recommendation here is that attention must be paid to the treatment of depression during the different stages of the treatment programmes i.e. when it becomes apparent that the level of depression is no longer declining or increasing.

5.3 Problems in this Study and Recommendations

The Control Group

Problems were experienced in obtaining a control group large enough to conduct meaningful statistical comparisons between the no-treatment control group and the experimental groups. It appeared that patients who had committed themselves to reducing their weight were unwilling to be placed on a waiting list and to delay the commencement of their treatment by three months until August 1988, when the next

treatment programme was scheduled to commence.

It is recommended that a non-dieting, obese control group be included in a weight-reduction programme to reveal whether changes in mood are due to dieting or non-dieting factors.

Drop Outs

The decline in the number of patients who reported for treatment as the programmes progressed complicated the statistical analysis.

It is recommended that additional research be conducted by means of a follow-up on the patients who dropped out, in order to determine what the reason was for dropping out, i.e. increased depression, weight-gain, no weight-loss etc.

Effect of inclusion of a cognitive component into a Behavioural Therapy programme on the number of patients who lose weight

When comparing the number of patients (Table 18 and 19) per week who lost weight in Group 0 and Group 1 it appears that a larger number of patients in Group 1 showed a weight-loss on a weekly basis than did patients in Group 0.

Additional research is recommended to follow up on the maintenance of the weight-loss in the two different treatment programmes.

Stunkard (1982) found that cognitions played a critical role in the maintenance of weight-loss and control of obesity. Training in the use of cognitive strategies had been shown to be effective in weight reducing behaviours. This was substantiated by studies which indicated that the inclusion of cognitive interventions into a behavioural weight-reduction programme facilitated the maintenance of initial behaviour change needed to lose weight and also to maintain the new lower-weight level (Bernier & Avard, 1986; Black, 1987; Buxton et al., 1985; Collins et al., 1986; Fairburn, 1981, 1984; Flanery & Kirschenbaum, 1986; Gormally & Rardin,

1981; Jordan & Canavan, 1985; Mitchell & Stuart, 1984; Russell, 1987; Stunkard, 1987 and Vander Kolk, 1985).

Role of Anxiety

It is also recommended that the level of anxiety in patients participating in a weight-reduction programme, as well as the role anxiety plays in dropping out of the programme, be monitored. Stunkard and Rush (1974) had stated that a weight-reduction treatment could and did result in adverse psychological reactions (depression and anxiety) and metabolic reactions in obese patients. The Boundary Model also proposes that anxiety plays a role in overeating (Herman and Polivy, 1980). The present study focused only on the monitoring of depression.

Follow Up Programme

A follow-up programme is recommended to determine whether the inclusion of the cognitive component (cognitive restructuring, problem-solving, improving self-control and self-regulation) into the behavioural therapy weight-reduction programme, plus a very low-calorie diet in fact would facilitate improved maintenance of the initial behaviour change needed to lose weight and to maintain the new lower-weight level, when compared to the standard behaviour therapy plus a very low-calorie diet programme. The follow-up programme should continue for at least a year and equip patients to handle situations which increase the risk of weight-gain (negative emotional and cognitive results of potential slips) using applicable behaviour and cognitive skills.

5.4 An Overview of the Study

The literature study conducted, encompassed obesity (i.e. theories, etiology and treatment), depression (i.e. theories, etiology and symptoms), as well as the association between the treatment of obesity and depression. A review of the obesity literature indicated that the role of psychological factors such as elevated arousal, anxiety and

depression, as well as confusion in distinguishing between the internal stimuli signaling hunger and stimuli related to emotional and interpersonal experiences, featured strongly in the psychoanalytical, psychosomatic, externality and restrained theories of obesity. It was also apparent that anxiety played a role in overeating, as proposed by the boundary model. The remaining theories (i.e. the set-point, glucostatic and ATPase enzyme deficiency theories) ascribed overeating and the resulting state of obesity to a physiological dysfunction. The obesity literature viewed the etiology of obesity as multifactorial, which made it difficult to identify a single factor as the primary cause. That a strong relationship existed between the prevalence of obesity and social class, ethnicity and religious background was demonstrated by epidemiologic research. It was also quite clear that no single etiological factor which caused obesity could be found and that numerous factors (biological, metabolic, genetic, psychological, cultural and environmental) contributed to the development and/or maintenance of excess weight. The literature emphasized the fact that obesity was an extremely complex condition and that psychological factors played a significant role.

Literature on the treatment of obesity indicated that while severe obesity (more than 100% overweight) was mostly effectively treated by surgical means, moderate obesity (40 to 100% overweight) was currently treated under medical auspices using diets and pharmaceutical treatment with poor maintenance of weight-loss and rapid rebound in body weight following cessation of drugs. Mild obesity (20 to 40% overweight) was covered by behaviour modification or therapy in groups (by both professional and non-profit making organizations), a liberal balanced diet and exercise. The conclusion reached in the literature, is that in any treatment procedure attention must be paid not only to reduced food intake and increased activity, but also to the psychological well-being of the individual, by paying attention to the individual's total functioning during the treatment programme. The implication is that attention should be paid to the psychological, physical and environmental or family functioning of the individual and

that only when all these aspects have been incorporated into the treatment of obesity, would the patient be successful.

From literature on depression, it is evident that biological theories suggest an inherited predisposition for bipolar disorder and relate the phenomena of depression and mania to abnormally depleted and copious amounts of the neuro-transmitters that pass on neural impulses in particular nerve tracts of the brain. However, psychological theories of depression had coaxed in psychoanalytical formations and stress unconscious identification with a loved one whose desertion of the individual had made him internalize anger, which caused depression. This concurs with the psychoanalytical approach which views obesity as a symptom of an underlying state of depression. The psychoanalytical approach views overeating as a defence mechanism derived from the unconscious effects of helplessness and hopelessness arising through object loss situations.

The cognitive theory of Beck (1979) ascribes causal significance to illogical self-judgement, while operant learning theorists attribute depression to an unrewarding, inactive existence. According to Seligman's (1979) cognitive learning appraisal, early experiences in inescapable hurtful situations instill a sense of helplessness which evolves in depression. The humanistic-existential theory emphasizes the difference between an individual's conception of ideal-self and his perception of discrepancies between the ideal and the actual state of affairs. The resulting depression is attributed to the fact that for the individual who experienced a loss of self-esteem, the difference between the ideal and real becomes too great to be tolerated.

Considerable argument in the literature revolves around whether depression is caused by endogenous factors (hormones or neurochemicals) or by the environment (conflicts and sudden losses). A common denominator among depressed persons is the decreased frequency of many different kinds of positively reinforced activity. It is evident that it is

not possible to expect one cause of depression or a singly underlying psychological process, as behaviour is a product of so many psychological processes.

The literature on the symptoms of depression revealed that it would be a simple task to explicate a long list of symptoms which had been found in depressive patients (sleep disturbances, diurnal variation of symptoms, agitation or psychomotor retardation, impairment of concentration, appetite disturbance, loss of libido and of interest in usual pursuits, somatic pains or sensations and anxiety). However, an illness is not just a compilation of symptoms. It is concluded from the literature that the focus of the therapeutic intervention should be at the target symptoms of which psychological symptoms include appetite disturbance (either increased or decreased eating).

Literature on the association between the treatment of obesity and depression was reviewed as the present study addressed itself to the monitoring of the occurrence and fluctuation of depression in obese patients on weight-reduction programmes. Literature indicates that studies of the psychological consequences of a weight-reduction treatment programme are divided between those reporting adverse effects and those which report beneficial effects. In studies of behaviour therapy treatment of obesity published between 1969 and 1983 improvements (or no deterioration) in psychological functioning was reported. It was evident that methodological differences in the various studies contributed to the differences in the findings between studies published between 1951 and 1973 which indicated adverse effects, and those published between 1969 and 1983. The three methodological problems identified in the literature i.e. frequency of assessment, time of assessment and method of assessment of depression were addressed in the present study.

5.5 In Conclusion

Results from this study revealed that depression was present at the commencement of the weight-reduction treatment programmes, that the level of depression fluctuated throughout the treatment programmes, that the behaviour therapy, plus a very low-calorie diet programme, did not cause untoward reactions in the patients participating. Although the results did not indicate a significant difference between the two experimental groups with regards to depression scores for weight-loss or weight-gain, the possible effect of including a cognitive component which focuses on depression as such, cannot be dismissed and needs further research during the actual treatment programme, as well as during the follow-up period.

Results obtained in this study, emphasize the need for health-care providers to be aware of the occurrence of depression in obese patients on a weight-reduction treatment programme and to cater for fluctuations in the level of reported depression. The focus of treatment programmes should be the on the total functioning of the individual. As the total rehabilitation of the patient is the goal of health-care specialists, it is essential that the psychological dimensions of the disease must not be overlooked.

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APPENDIX A**BIOGRAFIESE VRAELYS**

Die doel van hierdie vraelys is om die kwaliteit en effektiwiteit van ons diens te maksimaliseer.

Die inligting wat ons deur die vraelys van u verkry is **STRENG VERTROULIK**.

U **EERLIKHEID** in die beantwoording van die vraelys is van groot belang.

BIOGRAFIESE VRAELYS

AGTERGRONDGEGEWENS

1. Manlik 2. Vroulik

1 of 2

2. Wat is u huidige ouderdom?

in jaar

3. Wat is die ouderdom van u eggenoot?

in jaar

4. Wat is die hoogste opvoedkundige kwalifikasie wat u verwerf het?

Standerd 7 en laer of soortgelyk	1
Standerd 8 of 9 of soortgelyk (byv. NTS I of II)	2
Standerd 10, matriek, of soortgelyk (byv. NTS III)	3
Standerd 10 plus 1 jaar of 2 jaar verdere opleiding (Byv. NTS IV/V, GTS I/II of NDT)	4
Standerd 10 plus 3 jaar verdere opleiding (byv. B.A. of Nas. Dipl. vir Tegnici)	5
Standerd 10 plus 4 of meer jaar verdere opleiding (byv. M.Sc., B.A. en H.O.D., B.Arch., NHDT, ens.)	6

5. Wat is die hoogste opvoedkundige kwalifikasie wat u eggenoot verwerf het?

Standerd 7 en laer of soortgelyk		1
Standerd 8 of 9 of soortgelyk (byv. NTS I of II)		2
Standerd 10, matriek, of soortgelyk (byv. NTS III)		3
Standerd 10 plus 1 jaar of 2 jaar verdere opleiding (Byv. NTS IV/V, GTS I/II of NDT)		4
Standerd 10 plus 3 jaar verdere opleiding (byv. B.A. of Nas. Dipl. vir Tegnici)		5
Standerd 10 plus 4 of meer jaar verdere opleiding (byv. M.Sc., B.A. en H.O.D., B.Arch., NHDT, ens.)		6

6. Wat is u huidige beroep?

Merk die toepaslike kategorie hieronder:

<u>Professioneel_A</u>		
Advokaat, regter, prokureur, geoktrooieerde rekenmeester, mediese praktisyn, argitek, bourekenaar, apteker, ens.		01
<u>Professioneel_B</u>		
Natuur- of geesteswetenskaplike, mediese dienste, onderwyser, dosent, kunstenaar, tekenaar, sosiale werker ens.		02
<u>Administratiewe, uitvoerende of besturende beampte:</u>		
Direkteur, selfgeëmplojeerde eienaar, senior staatsamptenaar ens.		03

<u>Klerklike werker:</u> Klerk, boekhouer, kassier, rekenmeester, junior staatsamptenaar, ens.	04
<u>Verkoopsman:</u> Assuransie- en eiendomsagent, handelsreisiger, ens.	05
<u>Boer of Visserman:</u> Boer, plaasbestuurder, bosbouer, sorteerder en gradeerder.	06
<u>Myn-, steengroef of verwante werker:</u> Mynkaptein, myner, skofbaas, skagsinker, reduksiewerker, uitgrawer, steengroefwerker, ens.	07
<u>Vervoer of kommunikasiewerker:</u> Masjinis, huurmotorbestuurder, vragmotorbestuurder, navigator, kondukteur, telefonis, radio-operateur, brandweerman.	08
<u>Halfgeskoolde en ongeskoolde werker:</u> Byv. Vakleerling, arbeider, ens.	10
<u>Dienslewering:</u> Polisieman, gevangenisdiens- en verdedigingsmagpersoneel.	
<u>Ander:</u> Kelder, gids, haarkapper, opsigter, begrafnisondernemer, vermaaklikheidskunstenaar, ens.	12
<u>Persone wat nie besoldig word nie A:</u> Huisvrou. ens.	13
<u>Persone wat nie besoldig word nie B:</u> Pensioentrekker, student, werkloos, ongeskik vir werk.	14

7. Wat is u eggenoot se beroep?

Merk die toepaslike kategorie hieronder:

<u>Professioneel_A</u>		
Advokaat, regter, prokureur, geoktrooieerde rekenmeester, mediese praktisyn, argitek, bourekenaar, apteker, ens.		01
<u>Professioneel_B</u>		
Natuur- of geesteswetenskaplike, mediese dienste, onderwyser, dosent, kunstenaar, tekenaar, sosiale werker ens.		02
<u>Administratiewe, uitvoerende of besturende beampte:</u>		
Direkteur, selfgeëmplojeerde eienaar, senior staatsamptenaar ens.		03
<u>Klerklike werker:</u> Klerk, boekhouer, kassier, rekenmeester, junior staatsamptenaar, ens.		04
<u>Verkoopsman:</u> Assuransie- en eiendomsagent, handelsreisiger, ens.		05
<u>Boer of Visserman:</u> Boer, plaasbestuurder, bosbouer, sorteerder en gradeerder.		06
<u>Myn-, steengroef of verwante werker:</u> Mynkaptein, myner, skofbaas, skagsinker, reduksiewerker, uitgrawer, steengroefwerker, ens.		07
<u>Vervoer of kommunikasiewerker:</u> Masjinis, huurmotorbestuurder, vragmotorbestuurder, navigator, kondukteur, telefonis, radio-operateur, brandweerman.		08
<u>Halfgeskoolde en ongeskoolde werker:</u>		
Byv. Vakleerling, arbeider, ens.		10

<u>Dienslewering</u> : Polisieman, gevangenisdiens- en verdedigingsmagpersoneel.		
<u>Ander</u> : Kelder, gids, haarkapper, opsigter, begrafnisondernemer, vermaaklikheidskunstenaar, ens.		12
<u>Persone wat nie besoldig word nie A</u> : Huisvrou. ens.		13
<u>Persone wat nie besoldig word nie B</u> : Pensioentrekker, student, werkloos, ongeskik vir werk.		14

GESIN

8. By wie het u grootgeword?

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Spesifiseer

9. Leef u eie moeder nog? Indien nie, hoe oud was u met haar afsterwe?

Moeder	Moeder is oorlede toe ek:				
leef/ek					
weet	0-5 jr.	6-10 jr.	11-16 jr.	Ouer as 17 jr.	
nie				oud was.	
0	1	2	3	4	

10. Leef u eie vader nog? Indien nie, hoe oud was u met sy afsterwe?

Vader leef/ek weet nie	Vader is oorlede toe ek:				
	0-5 jr.	6-10 jr.	11-16 jr.	Ouer as 17 jr.	
				oud was.	
0	1	2	3	4	

11. Hoe gelukkig dink u was u ouers se huwelik terwyl u groot geword het (d.w.s. tot op 16-jarige ouderdom)?

N.v.t.-nie by ouers gewoon nie	Baie gelukkig	Redelik gelukkig	Effens gelukkig	Effens ongelukkig	Redelik ongelukkig	Baie ongelukkig
0	1	2	3	4	5	6

12. Terwyl u grootgeword het (d.w.s. tot op 16-jarige ouderdom), het enige van u ouers (of substituutouers) oor 'n tydperk van ten minste etlike jare sielkundige probleme ervaar?

N.v.t.- byv. in 'n inrigting	Nee	Ja - in 'n mate	Ja - beslis
0	1	2	3

13. Wat was die gehalte van die verhouding tussen u en u vader?

N.v.t.	Uitstekend	Goed	Gemiddeld	Swak
0	1	2	3	4

14. Wat was die gehalte van die verhouding tussen u en u moeder?

N.v.t.	Uitstekend	Goed	Gemiddeld	Swak	
0	1	2	3	4	

15. Wat was die gehalte van die verhouding tussen u en die ander kinders?

N.v.t.	Uitstekend	Goed	Gemiddeld	Swak	
0	1	2	3	4	

16. Het u vader u moeder fisies mishandel?

0	1	2	
NVT	JA	NEE	

17. Het u vader/moeder u fisies mishandel?

0	1	2	
NVT	JA	NEE	

18. Hoe was die kommunikasie rondom seks in u ouerhuis?

1. vrae is vryelik beantwoord
2. vrae is met weerstand beantwoord
3. die onderwerp is nooit bespreek nie

19. Hoe dink u was u ouers se seksuele verhouding?

Uitstekend	Goed	Gemiddeld	Swak	Baie Swak	
0	1	2	3	4	

20. Wie van die volgende persone was/is oorgewig?

- 0 Geen
- 1 Moeder
- 2 Vader
- 3 Broers
- 4 Susters
- 5 Eggenoot
- 6 Kinders

21. Is u familie oor die algemeen

- | | | | |
|------------------------|----|-----|--|
| | 0 | 1 | |
| 1. Oorgewig mense? | Ja | Nee | |
| | 0 | 1 | |
| 2. Swaargeboude mense? | Ja | Nee | |

HUWELIK

22. Huwelikstatus :
- 0 Getroud
 - 1 Ongetroud
 - 2 Vervreemd
 - 3 Geskei
 - 4 Wewenaar
 - 5 Weduwee
 - 6 2de Huwelik
 - 7 en meer

23. Aantal jare getroud?

jare

--

24. Aantal kinders?

25. Ouderdomme van kinders?

jare|

jare|

jare|

26. Hoe gelukkig dink u is u huwelik oor die algemeen?

Baie gelukkig	Redelik gelukkig	Ietwat gelukkig	Ietwat ongelukkig	Redelik ongelukkig	Baie on- gelukkig
0	1	2	3	4	5

27. Is u tevrede met u seksuele verhouding?

0 1
Ja Nee

28. Hoe tevrede is u met die frekwensie van seksuele omgang in u huwelik?

0	1	2	3
Baie	Redelik	Min	Glad nie

29. Voel u ongemaklik om voor u maat uit te trek omdat u oorgewig is?

0	1	2
Nooit	Soms	Altyd

30. Watter van die volgende situasies beïnvloed u seksuele aktiwiteite?

1. Huweliksprobleme
2. Oorgewig
3. Het belang verloor
4. Maat het belang verloor
5. Ander

Spesifiseer

31. Hoe voel u oor u metode van voorbehoeding?

0	1	2
Ontevrede	Redelik tevrede	Tevrede

32. Gesels u en u maat oor seksuele probleme?

0	1	2
Selde	Soms	Gewoonlik

33. Skaal uself ten opsigte van seksuele begeerte.

Min begeerte 1 2 3 4 5 Baie begeerte

VETSUGGESKIEDENIS

34. Hoe lank is u al oorgewig?

jaar

35. Het u oorgewig geraak as

1. volwassene
2. kind (jonger as 18jr) ?

36. Was u al ooit nie oorgewig nie?

0 1
Ja Nee

37. Vir hoe lank?

jaar

38. Hoe sou u u eetgewoontes beskryf?
Merk almal wat op u van toepassing is.

- 1 Te veel by maaltye
- 2 Gereeld tussen-in maaltye
- 3 Gesonde kos maar te veel
- 4 Meestal ongesonde kos

39. Gebeur dit dat u met tye verskriklik baie eet
en dan weer normaal?

0 1
Ja Nee

40. Eie pogings om gewig te verloor?

Merk wat op u van toepassing is.

Geen	0
Kitsdiëte	1
Eetlusdempers	2
Lakseermiddels	3
Vomering	4

41. Het u al voorheen deelgeneem aan 'n formele massavermindingsprogram?

Merk wat van toepassing is?

0	Nee
1	Weight Watchers
2	Weigh Less
3	Vighor
4	Winnie se program
5	Dr Badenhorst se program
6	Apteker
7	Gimnasium dieet
8	U eie dokter se dieet
9	Gesondheidsplaas
10	Ander :
	Spesifiseer

42. Hoe dikwels kry u oefening?

1	Gereeld
2	Selde
3	Nooit
4	Gereeld, voorheen.

43. Waarom sou u sê het u nie suksesvol gewig verloor nie?

- 1 motivering ontbreek
- 2 my eggenoot gee my nie die nodige ondersteuning nie
- 3 my gewigsverlies veroorsaak spanning in die huis
- 4 ander:
Spesifiseer.....

44. Dink u u is oorgewig?

- | | |
|----|-----|
| 1 | 2 |
| Ja | Nee |

--

45. Hoekom dink u u is oorgewig?

Merk almal wat op u van toepassing is.

- 0 Verkeerde eetgewoontes
- 1 Klierprobleme
- 2 Ander fisiese probleme
- 3 Dis maar in die familie
- 4 Die geboorte van my kinders
- 5 Bekommernisse
- 6 Ek word vet as ek net vir kos kyk
- 7 Ek is vet gebore
- 8 My ma het geglo ons moet "gesond" eet

46. Hoe voel u oor oefening?

- 0 Ek haat dit
- 1 Ek hou daarvan
- 2 Ek sou graag wou maar ek is te skaam vir myself
- 3 Ek sou graag wou maar het nie die geleenthied/geld/tyd nie
- 4 Oefening help nie
- 5 Oefening maak my vet

47. Waarom neem u deel aan die kursus?

Merk almal wat op u van toepassing is.

- | | | |
|---|--------------------------------------------------------------|--------------------------|
| 0 | My dokter sê ek moet gewig verloor | <input type="checkbox"/> |
| 1 | 'n Opmerking deur iemand gemaak | <input type="checkbox"/> |
| 2 | Ek is bang my man/vrou stel nie meer in my belang nie | <input type="checkbox"/> |
| 3 | Ek het huweliksprobleme a.g.v. my gewig | <input type="checkbox"/> |
| 4 | My klere pas nie meer nie | <input type="checkbox"/> |
| 5 | Ek hou nie daarvan om myself in die spieël te sien nie | <input type="checkbox"/> |
| 6 | Gesondheidsredes | <input type="checkbox"/> |
| 7 | My eggenoot/kinders/vriende/familie/kollegas sê ek is te vet | <input type="checkbox"/> |
| 8 | Die program het gewerk vir iemand wat ek ken | <input type="checkbox"/> |
| 9 | Dis goedkoop | <input type="checkbox"/> |

48. Dink u dat die kursus vir u gaan werk?

- | | | |
|----|-----|--------------------------|
| 1 | 2 | <input type="checkbox"/> |
| Ja | Nee | |

49. Hoeveel gewig wil u verloor?

MEDIESE GESKIEDENIS

50. Het u al gely aan die volgende siektes/simptome?

Nee = 0 tydperk in jaar = 1, 2, 3

Sooibrand	<input type="checkbox"/>
Mantelvliesbreuk	<input type="checkbox"/>
Spastiese dikderm	<input type="checkbox"/>
Gereelde hoofpyne	<input type="checkbox"/>
(a) migraine-tipe	<input type="checkbox"/>
(b) nekspanningstipe	<input type="checkbox"/>
(c) ander	<input type="checkbox"/>
Jig	<input type="checkbox"/>
Hoë cholesterol	<input type="checkbox"/>
Diabetes	<input type="checkbox"/>
Skilddklierprobleme	<input type="checkbox"/>
Hipertensie/hoë bloeddruk	<input type="checkbox"/>
Hartkloppings	<input type="checkbox"/>
Nagsweet	<input type="checkbox"/>
Gewrigspyne	<input type="checkbox"/>
Druklek (blaas)	<input type="checkbox"/>
Gereelde blaasinfeksies	<input type="checkbox"/>
Hardlywigheid	<input type="checkbox"/>
Benoudheid, veral snags	<input type="checkbox"/>
Moegheid	<input type="checkbox"/>

51. Het u al sielkundige/psigiatriese behandeling gehad?

Indien wel, merk die wat van toepassing is.

1 Medikasie by 'n dokter	<input type="checkbox"/>
2 Sielkundige	<input type="checkbox"/>
3 Psigiater	<input type="checkbox"/>
4 In 'n hospitaal	<input type="checkbox"/>

53. Watter van die volgende behandelings het u al ondergaan?

- 1 Maag gekram
- 2 Dikderm afgebind
- 3 Vet uitgesuig
- 4 Hipnose
- 5 Kakebeen vasgemaak
- 6 Akupunktuur (naalde)
- 7 Plastiese snykunde

54. Rook u?

Indien wel hoeveel sigarette per dag?

1	2	
Ja	Nee	

55. Gebruik u enige medikasie/middels?

a) wat deur die dokter voorgeskryf is

Watter?	Hoe lank?

b) wat nie deur die dokter voorgeskryf is nie

Watter?	Hoe lank?

56. Wanneer het u begin menstrueer?

jaar

57. Menstrueer u gereeld?

1 2
Ja Nee

58. Hoeveel dae is u siklus?

dae

59. Hoe lank duur u menstruasie?

dae

60. Het u lank gewag om swanger te raak?

1 2
Ja Nee

61. Was u op mondelinge kontrasepsie?

1 2
Ja Nee

62. Hoe lank na u kontraspesie gestaak het, het u swanger geraak?

jaar

63. Hoeveel kilogram was u oorgewig tydens die periode?

kg

64. Dink u, u sal swanger raak as u gewig verloor?

1 2
Ja Nee

APPENDIX B

THE BECK DEPRESSION INVENTORY (BDI)

Directions: Read each item carefully and circle the number next to the answer that best reflects how you have been feeling during the last few days. Make sure you circle one answer for each of the twenty-one questions. If more than one answer applies to how you have been feeling, circle the higher number. If in doubt make your best guess. Do not leave any question unanswered.

Aanwysings: Lees elke item noukeurig as u die vraelys invul. Omsirkel die nommer teenoor die antwoord wat die beste weergawe gee van hoe u die afgelope paar dae gevoel het. Maak seker dat u alleenlik een antwoord vir elkeen van die een-en-twintig vrae omsirkel. As daar meer as een antwoord is wat betrekking het op hoe u gevoel het, omsirkel dan die hoogste nommer. As u twyfel, omsirkel u beste raaiskoot. Moet geen vrae onbeantwoord laat nie.

1. 0 I do not feel sad.
Ek voel nie hartseer/treurig nie.
- 1 I feel sad.
Ek voel hartseer-treurig.
- 2 I am sad all the time and I can't snap out of it. .
Ek is altyd hartseer/treurig en ek kan nie myself daarvan losmaak nie.
- 3 I am so sad or unhappy that I can't stand it.
Ek is so hartseer/treurig of ongelukkig dat ek dit nie kan verdra nie.

2. 0 I am not particularly discouraged about the future.
Ek is nie besonder ontmoedig deur die toekoms nie.
- 1 I feel discouraged about the future.
Ek voel ontmoedig omtrent die toekoms.
- 2 I feel I have nothing to look forward to.
Ek voel ek het niks om na uit te sien nie.
- 3 I feel that the future is hopeless and that things cannot improve.
Ek voel dat die toekoms hopeloos is en dat dinge nie kan verbeter nie.
3. 0 I do not feel like a failure.
Ek voel nie soos 'n mislukking nie.
- 1 I feel I have failed more than the average person.
Ek voel dat ek meer as die gemiddelde persoon misluk het.
- 2 As I look back om my life, all I can see is a lot of failures.
Al wat ek kan sien as ek terugkyk na my lewe, is 'n klomp mislukkings.
- 3 I feel I am a complete failure as a person.
Ek voel dat ek as persoon 'n volslae mislukking is.
4. 0 I get as much satisfaction out of things as I used to.
Ek put net so baie bevrediging uit dinge as gewoonlik.
- 1 I don't enjoy things the way I used to.
Ek geniet dinge nie meer so baie soos gewoonlik nie.
- 2 I don't get real satisfaction out of anything anymore.
Ek put geen werklike bevrediging uit enige iets meer nie.
- 3 I am dissatisfied or bored with everything.
Ek is ontevrede of voel verveeld met alles.

5. 0 I don't feel particularly guilty.
Ek voel nie besonder skuldig nie.
- 1 I feel guilty a good part of the time.
Ek voel dikwels [vir 'n groot deel van die tyd] skuldig.
- 2 I feel quite guilty most of the time.
Ek voel meesal skuldig.
- 3 I feel guilty all the time.
Ek voel altyd skuldig.
6. 0 I don't feel I am being punished.
Ek voel nie asof ek gestraf word nie.
- 1 I feel I may be punished.
Ek voel dat ek moontlik gestraf gaan word.
- 2 I expect to be punished.
Ek verwag om gestraf te word.
- 3 I feel I am being punished.
Ek voel dat ek gestraf word.
7. 0 I don't feel disappointed in myself.
Ek voel nie in myself teleurgesteld nie.
- 1 I am disappointed in myself.
Ek voel teleurgesteld in myself.
- 2 I am disgusted with myself.
Ek verafsku myself.
- 3 I hate myself.
Ek haat myself.
8. 0 I don't feel I am any worse than anybody else.
Ek voel nie ek is slegter as enige iemand anders nie.
- 1 I am critical of myself for my weakness or mistakes.
Ek is krities op myself ten opsigte van my swakede en foute.
- 2 I blame myself all the time for my faults.
Ek blameer myself die hele tyd vir al my foute.
- 3 I blame myself for everything bad that happens.
Ek blameer myself vir alle slegte dinge, wat gebeur.

9. 0 I don't have any thoughts of killing myself.
Ek het nie gedagtes om myself om die lewe te bring nie.
- 1 I have thoughts of killing myself, but I would not carry them out.
Ek het gedagtes om myself om die lewe te bring, maar ek sal hulle nie uitvoer nie.
- 2 I would like to kill myself.
Ek sal daarvan hou om myself om die lewe te bring.
- 3 I would kill myself if I had the chance.
Ek sal myself om die lewe bring as ek 'n kans het.
10. 0 I don't cry any more than usual.
Ek huil nie meer as gewoonlik nie.
- 1 I cry more now than I used to.
Ek huil nou meer as gewoonlik.
- 2 I cry all the time.
Ek huil die hele tyd.
- 3 I used to be able to cry, but now I can't cry even though I want to.
Ek was daartoe in staat om te huil, maar nou kan ek nie huil nie, selfs as ek wil.
11. 0 I am no more irritated by things than I ever am.
Ek is geensins meer geïrriteerd deur dinge as gewoonlik nie.
- 1 I am slightly more irritated now than usual.
Ek is effens meer geïrriteerd deur dinge as gewoonlik.
- 2 I am quite annoyed or irritated a good deal of the time.
Ek is geïrriteerd of kwaad vir 'n groot periode van die tyd.
- 3 I feel irritated all the time now.
Ek voel nou die hele tyd geïrriteerd.

12. 0 I have not lost interest in other people.
Ek het nie belangstelling in ander mense verloor nie.
- 1 I am less interested in other people than I used to be.
Ek is minder geïnteresseerd in ander mense as gewoonlik.
- 2 I have lost most of my interest in other people.
Ek het meeste van my belangstelling in ander mense verloor.
- 3 I have lost all of my interest in other people.
Ek het al my belangstelling in ander mense verloor.
13. 0 I make decisions about as well as I ever could.
Ek neem besluite omtrent net so goed as altyd.
- 1 I put off making decisions more than I used to.
Ek stel besluitneming meer as gewoonlik af.
- 2 I have greater difficulty in making decisions than before.
Ek ondervind meer probleme as voorheen met die neem van besluite.
- 3 I can't make decisions at all anymore.
Ek kan glad nie meer besluite neem nie.
14. 0 I don't feel that I look worse than I used to.
Ek voel nie dat ek slegter as voorheen lyk nie.
- 1 I am worried that I am looking old and unattractive.
Ek is bekommerd dat ek oud en onaantreklik lyk.
- 2 I feel that there are permanent changes in my appearance that make me look unattractive.
Ek voel dat daar permanente veranderings in my voorkoms is, wat my onaantreklik laat lyk.
- 3 I believe that I look ugly.
Ek glo dat ek lelik lyk.

15. 0 I can work about as well as before.
Ek kan omtrent net so goed as voorheen werk.
- 1 It takes an extra effort to get started at doing something.
Dit vereis ekstra moeite om iets te begin doen.
- 2 I have to push myself very hard to do anything.
Ek moet myself baie hard druk om enige iets te doen.
- 3 I can't do any work at all.
Ek kan geen werk doen nie.
16. 0 I can sleep as well as usual.
Ek slaap net so goed soos voorheen.
- 1 I don't sleep as well as I used to.
Ek slaap nie so goed soos altyd nie.
- 2 I wake up 1 - 2 hours earlier than usual and find it hard to get back to sleep.
Ek word 'n aantal ure vroeër as gewoonlik wakker en kan dan nie weer aan die slaap raak nie.
17. 0 I don't get more tired than usual.
Ek word nie meer moeg as gewoonlik nie.
- 1 I get tired more easily than I used to.
Ek word meer moeg as gewoonlik.
- 2 I get tired from doing almost anything.
Ek word moeg as ek byna enige iets doen.
- 3 I am too tired to do anything.
Ek is te moeg om enige iets te doen.
18. 0 My appetite is no worse than usual.
My aptyt is nie slegter as gewoonlik nie.
- 1 My appetite is not so good as it used to be.
My aptyt is nie so goed soos dit was nie.
- 2 My appetite is much worse now.
My aptyt is nou baie swakker.
- 3 I have no appetite at all anymore.
Ek het nou geen aptyt meer nie.

19. 0 I have not lost much weight, if any, lately.
Ek het nie baie gewig, indien enige, onlangs
verloor nie.
- 1 I have lost more than five pounds.
Ek het meer as vyf pound verloor.
- 2 I have lost more than ten pounds.
Ek het meer as tien pond verloor.
- 3 I have lost more than fifteen pounds.
Ek het meer as vyftien pond verloor.
20. 0 I am no more worried about my health than usual.
Ek is nie meer bekommerd oor my gesondheid as altyd
nie.
- 1 I am worried about physical problems such as aches
and pains, or upset stomach, or constipation.
Ek is bekommerd oor fisiese probleme, soos pyne of
'n maagongesteldheid or hardlywigheid.
- 2 I am very worried about physical problems and it's
hard to think of much else.
Ek is baie bekommerd oor fisiese probleme en dit is
moeilik om aan enige iets anders te dink.
- 3 I am so worried about my physical problems that I
cannot think about anything else.
Ek is so bekommerd oor my fisiese probleme dat ek
omtrent aan niks anders kan dink nie.
21. 0 I have not noticed any recent changes in my
interest in sex.
Ek het geen onlangse verandering ten opsigte van my
belangstelling in seks opgemerk nie.
- 1 I am less interested in sex than I used to be.
Ek is minder geïnteresseerd in seks as voorheen.
- 2 I am much less interested in sex now.
Ek is nou baie minder geïnteresseerd in seks.
- 3 I have lost interest in sex completely.
Ek het my belangstelling in seks heeltemal verloor.

APPENDIX C

THE CARROLL SELF RATING SCALE

PATIENT:

TIME STARTED:

DATE OF BIRTH:

TIME COMPLETED:

TEST DATE:

TOTAL TIME:

FILE NO:

TOTAL SCORE:

Complete ALL of the following statements by CIRCLING YES OR NO

BASE YOUR ANSWER ON HOW YOU HAVE FELT DURING THE PAST FEW DAYS.

- | | | | |
|-------|-----------------------------------------------------------------|-----|----|
| 1. | I feel as energetic as always. | YES | NO |
| 2. | I am losing weight. | YES | NO |
| 3. | I have dropped many of my interests and activities. | YES | NO |
| 4. | Since my illness I have completely lost interest in sex. | YES | NO |
| 5. | I am especially concerned about how my body is functioning. | YES | NO |
| <hr/> | | | |
| 6. | It must be obvious that I am disturbed and agitated. | YES | NO |
| 7. | I am still able to carry on doing the work I am supposed to do. | YES | NO |
| 8. | I can concentrate easily when reading the papers. | YES | NO |
| 9. | Getting to sleep takes me more than $\frac{1}{2}$ of an hour. | YES | NO |
| 10. | I am restless and fidgety. | YES | NO |
-

- | | | |
|---------------------------------------------------------------------|-----|----|
| 11. I wake up much earlier than I need to in the morning. | YES | NO |
| 12. Dying is the best solution for me. | YES | NO |
| 13. I have a lot of trouble with dizzy and faint feelings. | YES | NO |
| 14. I am being punished for something bad in my past. | YES | NO |
| 15. My sexual interest is the same as before I got sick. | YES | NO |
| <hr/> | | |
| 16. I am miserable or often feel like crying. | YES | NO |
| 17. I often wish I were dead. | YES | NO |
| 18. I am having trouble with indigestion. | YES | NO |
| 19. I wake up often in the middle of the night. | YES | NO |
| 20. I feel worthless and ashamed about myself. | YES | NO |
| <hr/> | | |
| 21. I am so slowed down that I need help with bathing and dressing. | YES | NO |
| 22. I take longer than usual to fall asleep at night. | YES | NO |
| 23. Much of the time I am very afraid but don't know the reason. | YES | NO |
| 24. Things which I regret about my life are bothering me. | YES | NO |
| 25. I get pleasure and satisfaction from what I do. | YES | NO |
| <hr/> | | |
| 26. All that I need is a good rest to be perfectly well again. | YES | NO |
| 27. My sleep is restless and disturbed. | YES | NO |
| 28. My mind is as fast and alert as always. | YES | NO |
| 29. I feel that life is still worth living. | YES | NO |
| 30. My voice is dull and lifeless. | YES | NO |
| <hr/> | | |
| 31. I feel irritable or jittery. | YES | NO |
| 32. I am in good spirits. | YES | NO |
| 33. My heart sometimes beats faster than usual. | YES | NO |
| 34. I think my case is hopeless. | YES | NO |
| 35. I wake up before my usual time in the morning. | YES | NO |

- | | | | |
|-------|------------------------------------------------------------|-----|----|
| 36. | I still enjoy my meals as much as usual. | YES | NO |
| 37. | I have to keep pacing around most of the time. | YES | NO |
| 38. | I am terrified and near panic. | YES | NO |
| 39. | My body is bad and rotten inside. | YES | NO |
| 40. | I got sick because of the bad weather we have been having. | YES | NO |
| <hr/> | | | |
| 41. | My hands shake so much that people easily notice. | YES | NO |
| 42. | I still like to go out and meet people. | YES | NO |
| 43. | I think I appear calm on the outside. | YES | NO |
| 44. | I think I am as good a person as anybody else. | YES | NO |
| 45. | My trouble is the result of some serious internal disease. | YES | NO |
| <hr/> | | | |
| 46. | I have been thinking about trying to kill myself. | YES | NO |
| 47. | I get hardly anything done lately. | YES | NO |
| 48. | There is only misery in the future of me. | YES | NO |
| 49. | I worry a lot about my bodily symptoms. | YES | NO |
| 50. | I have to force myself to eat even a little. | YES | NO |
| <hr/> | | | |
| 51. | I am exhausted much of the time. | YES | NO |
| 52. | I can tell that I have lost a lot of weight. | YES | NO |
| <hr/> | | | |

TOTAL SCORE: _____

APPENDIX D

DIE CARROLL BEOORDELINGSKAAL

PASIËNT	:	TYD BEGIN	:
GEBOORTEDATUM	:	TYD KLAAR	:
TOETSDATUM	:	TOTALE TYD	:
LÊER NO.	:	TOTALE TELLING	:

Voltooi **ELKEEN** van die volgende stellings deur **JA** of **NEE** te omsirkel.

BASEER U ANTWOORD OP HOE U DIE **AFGELOPE PAAR DAE** GEVOEL HET.

- | | | | |
|-----|-------------------------------------------------------------------------|----|-----|
| 1. | Ek voel so energiek soos altyd | JA | NEE |
| 2. | Ek is besig om gewig te verloor. | JA | NEE |
| 3. | Ek het baie van my belangstellings en aktiwiteite laat vaar. | JA | NEE |
| 4. | Vandat ek siek geword het, het ek geen belangstelling meer in seks nie. | JA | NEE |
| 5. | Ek is veral bekommerd oor hoe my liggaam funksioneer. | JA | NEE |
| 6. | Dit moet vir ander mense duidelik wees dat ek ontsteld en verontrus is. | JA | NEE |
| 7. | Ek kan nog steeds die werk doen wat ek veronderstel is om te doen. | JA | NEE |
| 8. | Ek kan maklik konsentreer wanneer ek koerant lees. | JA | NEE |
| 9. | Dit neem my langer as 'n halfuur om aan die slaap te raak. | JA | NEE |
| 10. | Ek is rusteloos en kriewelrig. | JA | NEE |
-

- | | | | |
|-------|-------------------------------------------------------------------|----|-----|
| 11. | Ek word soggens baie vroeër wakker as wat nodig is. | JA | NEE |
| 12. | Die beste oplossing vir my is om dood te gaan. | JA | NEE |
| 13. | Ek het baie problems met duiseligheid en gevoelens van floute. | JA | NEE |
| 14. | Ek word vir slegte dinge uit my verlede gestraf | JA | NEE |
| 15. | My seksuele belangstelling is dieselfde as voor my siekte. | JA | NEE |
| <hr/> | | | |
| 16. | Ek voel misrabel en wil dikwels huil. | JA | NEE |
| 17. | Ek wens dikwels dat ek dood was. | JA | NEE |
| 18. | Ek het spysverteringsprobleme. | JA | NEE |
| 19. | Ek word dikwels in die nag wakker. | JA | NEE |
| 20. | Ek voel waardeloos en skaam oor myself. | JA | NEE |
| <hr/> | | | |
| 21. | Ek is so lusteloos dat ek met bad en aantrek hulp nodig het. | JA | NEE |
| 22. | Ek neem langer as gewoonlik om saans aan die slaap te raak. | JA | NEE |
| 23. | Ek voel dikwels bang, maar weet nie wat die rede daarvoor is nie. | JA | NEE |
| 24. | Dinge in my lewe waaroor ek spyt voel, pla my deesdae. | JA | NEE |
| 25. | Ek put plesier en bevrediging uit wat ek doen. | JA | NEE |
| <hr/> | | | |
| 26. | Al wat ek nodig het om weer heeltemal goed te voel is genoeg rus. | JA | NEE |
| 27. | My slaap is versteurd en rusteloos. | JA | NEE |
| 28. | My verstand is so flink en wakker soos altyd. | JA | NEE |
| 29. | Ek voel die lewe is nog die moeite werd. | JA | NEE |
| 30. | My stem is eentonig en leweloos. | JA | NEE |
| <hr/> | | | |
| 31. | Ek voel geïrriteerd of skrikkerig. | JA | NEE |
| 32. | Ek is in 'n goeie gemoedstemming. | JA | NEE |
| 33. | My hart klop soms vinniger as gewoonlik. | JA | NEE |
| 34. | Ek dink ek is 'n hopelose geval. | JA | NEE |
| 35. | Ek word soggens voor die gewone tyd wakker. | JA | NEE |

- | | | | |
|-------|------------------------------------------------------------------|----|-----|
| 36. | Ek geniet my maaltye net soveel soos gewoonlik | JA | NEE |
| 37. | Ek het dikwels 'n behoefte om heen en weer te stap. | JA | NEE |
| 38. | Ek is vreesbevange en byna paniekerig. | JA | NEE |
| 39. | My liggaam is sleg en van binne verrot. | JA | NEE |
| 40. | Ek het weens die slegte weer wat ons gehad het, siek geword. | JA | NEE |
| <hr/> | | | |
| 41. | My hande bewe soveel dat mense dit maklik kan raaksien. | JA | NEE |
| 42. | Ek geniet dit steeds om uit te gaan en mense te ontmoet. | JA | NEE |
| 43. | Ek dink en kom uiterlik kalm voor. | JA | NEE |
| 44. | Ek dink ek is net so 'n goeie mens soos enige iemand anders. | JA | NEE |
| 45. | My probleem is die gevolg van 'n ernstige innerlike siekte. | JA | NEE |
| <hr/> | | | |
| 46. | Ek het al daaraan gedink om myself om die lewe te probeer bring. | JA | NEE |
| 47. | Ek kry deesdae byna niks gedoen nie. | JA | NEE |
| 48. | Daar is vir my in die toekoms slegs ellende te wagte. | JA | NEE |
| 49. | Ek is baie bekommerd oor my liggaamlike simptome. | JA | NEE |
| 50. | Ek moet myself dwing om selfs net 'n bietjie te eet. | JA | NEE |
| <hr/> | | | |
| 51. | Ek is meestal uitgeput. | JA | NEE |
| 52. | Ek kan sien dat ek baie gewig verloor het. | JA | NEE |

TOTALE TELLING _____

	V1	V2	V3	V4	V5	V6	V7	V8	V9	V10	V11	V12	V13	V14	V15	V16	V17	V18	V19	V20_0	V20_1	V20_2
4R																						
32	2	30	33	1	2	10	10	1	0	3	1	1	1	2	1	2	2	0	1	1		
1	2	36	45	2	1	13	10	1	4	3	2	1	2	3	2	2	2	2	2		1	1
2	2	38	43	1	2	4	10	1	0	4	1	1	1	1	2	2	2	2	0		1	
12	2	45		3		4		1			1	2	1	3	2	2	2	0	1		1	1
15	1	40	35	1	2	10	13	1	0		1	1	1	1	1	2	2	2	1			
17	2	43	47	1	5	13	7	1	0	0	1	1	1	2	3	2	2	2	1			
18	1	35	30	4	3	2	4	1	0	4	2	1	2	2	2	2	2	2	2	1		
26	2	30	32	3	3	13	3	1	0	0	1	1	1	2	2	2	2	0	2	1		1
33	2	30		1	1	10		1	0	4	1	1	1	1	2	2	2	0	1	1		
34	2	44	48	2	5	13	2	1	0	4	1	1	1	2	2	2	2	2	1	1	1	
35	2	19	28	2	4	13	4	1	0	0	5	1	3	3	3	2	2	2	3	1	1	
37	2	28	28	3	2	13	7	1	0	0	2	1	2	1	1	2	2	2	2	2		
39	2	34	39	2	2	13		1	0	4	3	2	3	4	2	2	2	1	4	1		
43	1	21		4		10		1	0	0	1	1	1	1	1	2	2	1	0	1		
45	1	38	42	3	2	13	10	1	4	0	2	1	2	2	2	2	2	2	1	1		1
49	2	34	38	2	2	13	10	1	0	0	2	1	3	3	2	2	2	2	2		1	1
0	1	52	46	3	6	3	2	1	4	3	1	1	1	3	3	2	2	0	1			
51	2	21	26	2	2	13	10	1	0	0	2	1	2	2	2	2	2	2	2	1		
63	2	31	35	1	2	7	8	1	0	4	2	1	4	1	2	2	1	1	1	1		
71	2	27		4		8		1	0	0	2	1	2	2	3	2	2	1	2		1	
73	1	44	45	2	2	10	13	1	4	4	6	0	2	2	2	0	0	2		1	1	1
74	2	48	52	1	1	13	10	1	0	0	5	1	2	1	1	1	0	0	1	1		1
75	2	37	40	3	5	4	2	1	0	0	1	1	1	1	2	2	2	1	1	1		1
79	2	30	32	1	2	13	10	1	0	1	1	1	2	1	1	2	2	2	1	1		1
3	2	40	43	2	2	13	8	1	0	4	1	1	1	1	2	2	2	2	1	1		1
6	2	52	53	2	3	13	7	2	1	0	0	1	4	0	0	0	0	2	2			
7	2	27	29	3	2	4	7	1	0	0	2	1	3	2	3	2	2	2	2		1	1
9	2	38	48	1	1	13		1	0	0	2	1	2	3	2	2	2	1	1		1	1
13	2	35	41	3	5	4	2	1	2	0	1	2	1	0	2	2	2	0	1		1	1
14	1	41	35	5	3	2	4	1	0	0	1	1	3	2	1	2	2	2	1	1		1
16	2	29	34	2	2	13	7	1	0	0	2	1	2	2	2	2	2	2	1	1		1
20	2	18		1		14		2	0	1	2	1	0	2	0	0	0	2	1	1	1	
27	1	30	27	3	2	4	4	1	0	4	2	1	3	3	2	2	2	2	2		1	1
30	2	28		2		4		1	0	4	2	1	3	3	3	2	2	2	3	0	0	1
31	2	36	38	2	1	4	12	1	4	0	1	1	4	4	3	2	1	2	0	0		
36	2	34	37	3	2	4	7	1	0	0	6	1	3	2	2	2	2	2	2	1		
38	2	27	32	2	3	13	7	1	0	4	6	2	3	4	3	1	1	2	4			
40	2	33	35	3	4	4	7	1	0	4	6	2	3	3	2	1	2	2	4			1
42	2	56	57	2		13	14	1	4	4	2	1	2	2	3	2	2	1	2		1	1
58	2	28	33	1	2	13	7	2	0	0	0	1	0	0	3	0	0	2	4		1	1
60	2	44	43	1	1	13	10	1	4	4	2	1	2	2	2	2	2	2	2		1	1
70	2	54	55	3	3	2	8	1	0	2	1	1	1	1	1	2	2	2	1	1	1	1
72	2	35	35	6	5	4	3	1	0	0	1	1	3	3	3	2	2	2	1	1	1	1
76	1	28		3		10		1	0	4	1	1	1	1	1	2	2	2	1	1		
78	1	56	53	5	3	7	13	2	3	3	2	1	2	1	2	2	2	2	1	1		1
80	2	28	27	3	5	4	4	1	0	4	1	1	1	1	1	2	2	0	1	1		

NR	V20_3	V20_4	V20_5	V20_6	V21_1	V21_2	V22	V23	V24	V25A	V25B	V25C	V26	V27	V28	V29	V30	V31	V32	V33	V34
32					1	1	0	10	4	12	9	6	0	0	1	0	3	2	2	3	2
1	1	1			0	0	6	11	3	18	16	8	0	0	0	1	6	2	2	4	36
2		1	1	1	0	1	0	22	5	22	21	20	1	0	0	0	2	2	2	1	10
12		1			0	0	5	26	2	25	22										6
15		1			1	1	0	19	2	10	4		0	0	0	0		2	0	3	10
17	1	1				0	0	25	5	24	23	21	3	1	2	2	1	1	0	1	20
18						0	6	13	2	4	6		1	0	1	1	2	2	2	3	8
26		1			1	1	0	10	2	3	1		0	0	0	0	2	2	0	4	3
33					1	1	5	14	5	14	13	11	3	0	1	1	1	2	2	3	1
34					0	0	0	23	3	20	19	11	3	1	1	1	1	1	0	3	11
35					0	0	0	2	0				3	1	2	1	2	0	0	3	3
37	1	1			0	1	0	7	2	3	3		0	0	1	1	1	2	3	10	
39					1	0	0	16	4	18	14	12	1	1	2	1	5	2	2	1	34
43							0														11
45					1	1	0	20	3	18	16	9	1	0	1	1	2	2	1	3	20
49		1			1	0	0	17	3	16	14	9	1	1	3	0	3	2	0	2	15
50		1			0	0	2	30	3	27	10	9	5	0	3	0	3	2	0	5	22
61					1	1	0	4	2	3	4		1	0	0	1	1	2	2	2	2
63					1	0	0	12	3	11	6	4	1	0	1	1	2	2	2	4	2
71					1	0	1										5			1	11
73	1		1		1	1	0	24	4	22	21	19	0	0	0	0		2	2	5	10
74		1			1	0	0	28	4	25	23	22	3	1	1	0	1	1	0	2	9
75			1	1	0	0	0	18	3	17	16	14	3	0	1	2	1	1	1	4	10
79	1	1			0	0	0	11	2	9	7		0	0	1	2	3	0	0	1	20
3			1		0	0	0	32	2	28	23		1	1	1	1	3	0	1	1	
6					0	0	0	4	1	4			3	1	1	1	2	1	1	1	13
7					1	0	0	23	4	22	19	18	2	0	2	2	2	1	1	1	3
8	1	1			0	0	6	5	1	4			0	0	0	1	2	0	1	2	18
13		1			0	0	0	5	1	4			3	1	3	0	2	0	0	5	5
14					0	0	0	9	2	9			1	0	1	1	2	1	1	4	4
16					1	1	1		1	3					1	1	2	2	2	5	2
20					0	0	0								1	0	5	2	2	3	30
27		1			1	1	3	11	2	11	9		1	0	1	0		2	1	4	1
30					1	1	0	16	2	17	14					1	5	1	2	2	2
31					1	0	0	6	3	15	12	10	1	0	3	1	5	1	0	1	2
36					1	1	6	6	3	15	14	6	1	1	1	2	2	2	0	2	20
38		1	1		0	0	0	10	2	8	2		1	1	1	2	2	0	0	1	10
40		1	1		0	0	0	13	2	11	7		1	1	2	2	2	2	0	1	33
42		1		1	1	1	0	33	2	30	25		1	0	1	1	2	2	1	3	9
58	1	1			0	0	0	10	1	9			0	0	1	1	2	2	2	3	8
60					1	1	6	15	4	25	23	24	1	0	2	2	3	2	2	1	8
70	1				0	0	0	30	0						1	1		0	0	1	6
72					0	0	0	12	3	9	7	5	0	1	2	1	3	2	1	1	20
76	1				0	0	0	7	2	55	40		0	0	0	1		2	0	5	6
78		1			0	1	0	28					2	1	2	0	3	2	0	3	3
80					0	0	0	2					0	0	1	2	1	2	1	3	10

	V35	V36	V37	V38_1	V38_2	V38_3	V38_4	V38_5	V39	V40_0	V40_1	V40_2	V40_3	V40_4	V41	V42	V43	V44	V45_0	V45_1	V45_2
32	1	0	7		1	1		1	0			1			8	2	3	1	1		
1	2	1	1	1	1		1	1	0		1	1	1		5	3	4	1			
2	1	0	21		1		1		0			1			5	1	1	1			
12	1	0	39			1		1	0		1				0	2	1	1	1		
15	1	1	30					1	1						0	1	1	1	1		
17	23	1	20			1			0	1					0		1	1	0		
18	1	0	27	1			1		0		1				5	2	1	1	0		
26	1	0	27	1	1				0		1	1			5	1	1	1	1		
33	1	0	5		1	1		1	0		1				10	2	4	1	1		
34	9	0	10	1	1	1	1	1	0		1	1			1	3	1	1	1	1	
37	2	0	16			1			0			1			0	4	1	1	1		
37	2	1	10	1				1	0					1	0	2	1	1	1		
39	2	0					1	1	1						11	2	2	1	1		
40	2	1	8					1	0			1			1	1	1	1	1		
43	1	0	19		1		1	1	0		1	1			5	3	1	1	1	1	
43	2	1	0	1			1		0			1			8	3	1	1	1	1	
46	1	0	30		1	1		1	0			1			0	1	1	1	1		
51	1	0	18		1				0		1	1			8	1	1	1	1		
51	1	0	19					1	0			1	1	1	0	1	1	1	1		
56	1	0	9		1				0			1			10	4	1	1	1	1	
56	1	0	35			1			1	1				0	2	1	1	1	1		
60	1	0	31		1				0		1				1	2	1	1			
60	1	0	23	1		1			0		1	1		1	1	1	1	1	1		
63	2	1	2		1		1		0			1			5	4	1	1	1		
63	1	0	20	1				1	1	1					5	1	1	1	1		
66	2	0	2		1	1			0	1						2	1	1	1		1
67	2	0	14		1		1		0	0	1				1	2	1	1	1		
68	1	1	3		1	1			0		1	1			0	2	1	1	1		
69	1	1	10		1		1		0		1	1			1	1	1	1	1		
71	1	0	2				1		0		1				4	4	1	1	1		
71	1	1	0	1	1			1	0	1				0	1	1	1	1	1		
76	2	0	16	1	1		1		0		1	1			0	1	1	1	1		
76	2	1	1	1	1				1		1	1			0	2	4	1	1		
80	1	0	33	1		1	1		1			1			3	2	4	1	0		
81	1	0	30			1			1			1			7	4	2	1	1		
83	1	0	1	1	1		1		0	0				1	1	1	1	1	1		
83	1	0	17	1	1		1		0		1	1			3	3	1	1	1		
84	2	1	33	1	1	1			0		1	1			2	2	1	1	1		1
84	2	0	3		1	1	1	1	0		1	1	1		4	2	1	1	1		
88	1	0	18	1	1	1		1	0			1			0	1	1	1	1		
88	1	0	8			1			0			1			10	1	4	2	1		
89	1	0	48		1	1	1	1	0		1			1	5	4	1	1	1		
92	2	0	15	1	1	1			0		1	1	1		5	4	1	1	1		
92	1	0		1		1		1	0	1					0	1	1	1	1		
92	1	0	50				1		1					1	0	2	1	1	1		
93	1	0	18		1		1		0			1			10	3	4	1	1		1

NR	V45_3	V45_4	V45_5	V45_6	V45_7	V45_8	V46	V47_0	V47_1	V47_2	V47_3	V47_4	V47_5	V47_6	V47_7	V47_8	V47_9	V48	V49
32		1		1			2	1				1	1	1	1	1		1	20
1	1	1		1			2					1				1		1	58
2		1					1					1	1	1				1	11
12				1		1	3					1	1		1			1	10
15							1	1										1	36
17		1	1				2	1			1	1	1	1				1	
18							2				1					1		1	25
26		1	1				1			1		1		1				1	20
33			1			1	0		1		1	1		1	1	1		1	25
34	1	1				1	0			1	1	1	1	1				1	50
35				1			2		1	1	1	1	1		1	1		1	30
37		1	1			1	1	1		1			1					1	30
39	1	1		1	1	1	3					1	1	1				1	25
43							1	1				1						1	30
45		1		1			1	1		1		1	1	1	1			1	15
49				1		1	3	1				1	1		1			1	65
50	1			1		1	1	1				1	1	1	1	1		1	36
61		1	1				2			1		1	1		1			1	15
63		1				1	3			1		1	1	1				1	18
71	1			1		1	1	1				1	1			1		1	20
73							3	1										1	30
74	1						3	0					1	1				1	20
75		1	1			1	1		1	1		1	1	1				1	20
79	1					1	1			1		1			1			1	20
5		1					2	1				1	1					1	10
6	1	1					2	1				1	1					1	34
7							2		1	1		1		1				1	50
8		1					2	1			1		1	1	1			1	35
13	1						1	1				1		1	1	1		1	50
14				1			1					1		1				1	20
16		1					3			1			1		1			1	50
20		1	1	1			1		1	1		1	1		1			1	42
27	1			1	1	1	1	1						1				1	30
30							3					1	1		1			1	9
31		1					3	1				1			1			1	15
36							2					1	1	1				1	40
38							0			1	1	1	1	1	1	1		1	25
40	1	1	1	1	1	1	0	1	1	1		1	1	1	1		1	1	30
42		1				1	3	1	1			1	1	1	1			1	30
58			1	1			1	1				1			1			1	40
60				1		1	2	1	1				1			1		1	22
70	1			1		1	2	1				1		1		1	1	1	30
72	1		1	1			1						1	1				1	30
76							1	1	1			1	1		1			1	40
78							1	1	1			1	1					1	15
80				1			2			1		1	1				1	1	30

APPENDIX F

THE BECK DEPRESSION AND THE CARROLL RATING SCALE MEAN SCORES FOR THE CONTROL GROUP.

Week No.	N	Beck Depression Inventory				Carroll Rating Scale			
		Mean	Std. Dev.	t	p*	Mean	Std. Dev.	t	p*
7	10	2,80	2,85	4,13	0,0026*	0,90	0,35	2,59	0,0294*
9	7	1,80	3,04	2,35	0,0573*	0,86	0,46	1,87	0,1112
11	5	1,40	2,52	1,98	0,1193	0,20	0,20	1,00	0,3739
13	5	1,40	2,82	1,91	0,1282	0,60	0,40	1,50	0,2080
15	6	1,60	2,37	2,18	0,0813*	0,33	0,33	1,00	0,3632
17	3	1,20	2,84	1,17	0,3624	0,33	0,33	1,00	0,4226
19	3	1,60	1,94	2,42	0,0519*	0,29	0,18	1,55	0,1723
21	7	1,40	1,81	2,12	0,0781	0,43	0,29	1,44	0,1996

* $p \leq 0,1$ = Significant Difference

NOMMER	SEX	BOU	LENGTE	MASSA	IDEALE	GRP	2-MAR	16-MAR	30-MAR	13-APR	20-APR	27-APR	4-MAY	11-MAY	18-MAY	25-MAY	1-JUN	8-JUN
1	V	G	170	126.6	72.0	0	126.6	127.2	129.0	129.0	125.0	125.0	127.2					
2	V	G	165	73.8	68.0	0	73.8	74.4		75.8		75.8						
12	V	K	160	75.6	54.0	0	75.6	76.4		75.8	75.6	75.3	75.2					
15	M	G	180	136.6	84.0	0	136.6	134.0	133.8	132.6	130.3	130.2	129.0	127.3		125.0	123.4	123.3
17	V	G	170	120.8	72.0	0	120.8	120.0	116.9	117.8	115.4	115.8	115.1	115.6	114.2	114.5	113.3	
18	M	G	185	108.4	88.0	0	108.4	110.0	110.2	109.4	106.4							
21	V	G	175	101.2	76.0	0	101.2	99.2	99.0		100.8	98.3		99.4		97.8	97.9	
26	V	M	162	76.2	59.0	0	76.2	76.6		77.5	75.4		76.9	74.1	73.1	73.2	74.2	
32	V	M	163	71.3	61.0	0												
33	V	G	166	81.4	68.0	0	81.4	82.6			83.6		82.7	81.7				
34	V	M	166	107.0	63.0	0	107.0	107.0	105.9			108.7	105.6	105.0	104.0			
35	V	G	171	112.2	72.0	0	112.2	111.4	113.0	112.0	112.6							
37	V	M	161	95.6	59.0	0	95.6	96.0	94.0	93.5								
39	V	K	158	84.4	53.0	0	84.4	85.4	84.8	85.8	84.0	83.8			82.6			
43	M	M	167	104.6	65.0	0	104.6	103.2	102.7	100.2	100.0	100.4	100.5		101.0			
45	V	K	157	70.3	51.0	0	70.3	71.0	68.0	69.9	68.9							
46	V	K	157	109.6	51.0	0	109.6	109.8	109.5	109.0		107.8						
49	V	M	163	136.2	61.0	0	136.2	134.4		132.2	130.4	129.3		127.0		126.5		
50	M	G	183	117.6	86.0	0	117.6	116.2		114.0	114.4	113.7	112.9					110.1
56	V	M	157	64.2	55.0	0	64.2	63.4		63.9								
61	V	M	160	66.3	59.0	0	66.3	67.2		65.7	65.5	64.7	65.8			64.6	63.0	
63	V	G	171	78.0	72.0	0	78.0	80.2		77.4	77.0		75.9					
71	V	K	159	80.0	53.0	0	80.0			79.4								
73	M	G	190	124.4	93.0	0	124.4	130.2	125.5	124.8	122.6	121.0	119.8	-119.2	118.8	117.4	116.6	116.4
74	V	G	162	115.8	64.0	0	115.8	115.8		115.8	112.9	113.8	112.4	112.0	112.4	114.0	111.8	111.3
75	V	M	160	77.8	59.0	0	77.8	79.2	79.2		77.1	77.1	77.9	77.0	76.8	76.4		75.9
79	V	K	165	85.0	58.0	0	85.0	82.0		83.4	80.8							
3	V	K	152	62.6	49.0	1	62.6	61.8	63.8	61.0	60.2	60.2	60.0	60.2	60.2	60.2		
6	V	G	170	92.8	72.0	1	92.8	91.6	92.4	90.0	87.6	85.8	84.8	83.7	83.0	81.8	80.2	
7	V	G	163	114.6	66.0	1	114.6	114.4	113.2	113.2	112.2	111.4	110.4		108.8	109.6	105.4	106.6
8	V	M	160	99.6	59.0	1	99.6	99.0	98.2	101.6		97.6	95.4		95.0		96.0	95.0
13	V	K	161	110.2	54.0	1	110.2	110.2	109.5	110.5	108.0	105.0	104.1	102.7				
14	M	G	184	114.3	86.0	1	114.3	113.0	113.0		108.4	106.5	105.9	104.2				
16	V	K	161	109.6	54.0	1	109.6	107.0	108.0	106.4	105.5	103.1	103.4	102.0	100.1	99.8	101.2	100.0
20	V	G	167	97.2	68.0	1	97.2	98.2	98.0	97.0	96.8	97.6	96.8	95.2				
27	M	G	181	126.0	84.0	1	126.0	125.4	122.0	119.2	118.2	116.6	118.0	115.6	111.4			
30	V	M	172	75.3	67.0	1	75.3	74.0	73.0	73.8	74.0	73.0	71.2	71.6	71.8	71.4		
31	V	M	161	67.1	59.0	1	67.1	65.8	64.9	64.7	64.5	63.6	63.1	62.6	62.2			
36	V	G	180	116.2	79.0	1	116.2	117.4	115.8	115.8	113.5	111.0	109.2	107.9	107.2			
38	V	K	164	79.6	56.0	1	79.6	79.2	78.8	79.4	77.5	76.8	74.6					
40	V	G	170	99.4	72.0	1	99.4	99.2	98.5	97.8	97.4	95.0	94.6	92.7	91.7			91.4
42	V	G	164	111.2	66.0	1	111.2	108.0	106.6	104.9	104.8	102.8	102.2	101.4	101.0	99.8	98.6	98.8
58	V	M	167	108.2	63.0	1	108.2	107.4	106.3	105.2	103.4	101.2	101.8	98.6	98.3	99.4	98.8	98.4
59	V	M	166	94.0	63.0	1	94.0	90.4				88.9						
60	V	G	166	81.1	68.0	1	81.1	81.6	80.9	78.0	75.4	74.5	73.0	72.0	71.5	69.7	69.7	67.9
70	V	G	154	101.1	59.0	1	101.1	103.2	100.2	98.4	98.7	97.1	96.7	95.6	96.2	94.7	94.9	94.9
72	V	M	164	87.3	61.0	1	94.8	94.8	94.5		95.2	92.5	91.6					
76	M	G	176	132.8	79.0	1	132.8			133.2	131.7	131.8	131.0		128.0	127.6		
78	M	M	164	82.2	63.0	1	82.2	80.4	80.0	78.4	76.8	75.8	75.2			72.0	70.8	70.5
80	V	M	169	90.8	65.0	1	90.8	90.4	89.9	88.6	86.4	84.9	83.2	81.6	80.7	79.5	78.2	77.6

NOHMER	15-JUN	22-JUN	29-JUN	6-JUL	13-JUL	20-JUL	24-AUG	FIRST	LAST	LOSS	AVG LOSS
1								126.6	127.2	0.6	
2								73.8	75.8	2	
12								75.6	75.2	-0.4	
15		120.4		121.8				136.6	121.8	-14.8	
17		113.9		114.2		113.5		120.8	113.5	-7.3	
18								108.4	106.4	-2	
21								101.2	97.9	-3.3	
26						76.6		76.2	76.6	0.4	
32								71.3	0	-71.3	
33								81.4	81.7	0.3	
34								107	104.0	-3	
35								112.2	112.6	0.4	
37								95.6	93.5	-2.1	
39								81.4	82.6	-1.8	
43								104.6	101.0	-3.6	
45								70.3	68.9	-1.4	
46								109.6	107.8	-1.8	
49								136.2	126.5	-9.7	
50		109.8		109.2		110.2		117.6	110.2	-7.4	
56								64.2	63.9	-0.3	
61								66.3	63.0	-3.3	
63								78	75.9	-2.1	
71								80	79.4	-0.6	
73		114.9		115.4		114.3		124.4	114.3	-10.1	
74	111.0	109.8		110.6		108.6	107.0	115.8	107.0	-8.8	
75								77.8	75.9	-1.9	
79								85	80.8	-4.2	-5.83333
3				64.5	61.2	61.1	63.4	62.6	63.4	0.8	
6		77.9		77.9	77.2	76.6	74.5	92.8	74.5	-18.3	
7						106.8		114.6	106.8	-7.8	
8								99.6	95.0	-4.6	
13								110.2	102.7	-7.5	
14								114.3	104.2	-10.1	
16	98.0	98.4		97.3		94.9		109.6	94.9	-14.7	
20								97.2	95.2	-2	
27						109.8	105.4	126	105.4	-20.6	
30						70.8		75.3	70.8	-4.5	
31						63.6		67.1	63.6	-3.5	
36								116.2	107.2	-9	
38								79.6	74.6	-5	
40	89.5	89.6	89.4					99.4	89.4	-10	
42	96.2	96.6		95.8		95.3		111.2	95.3	-15.9	
58		99.0				97.0		108.2	97.0	-11.2	
59								94	88.9	-5.1	
60	68.6	66.7		65.2	65.2	64.1	62.8	81.1	62.8	-18.3	
70								101.1	94.9	-6.2	
72								87.3	91.6	4.3	
76						123.0		132.8	123.0	-9.8	
78				66.9		65.1	61.2	82.2	61.2	-21	
80	77.5	76.4		75.4		74.0		90.8	74.0	-16.8	-9.42608

APPENDIX H

TABLE OF THE BECK DEPRESSION INVENTORY AND THE CARROLL RATING SCALE FREQUENCY SCORES - COMPARISON OF GROUP 0 AND GROUP 1.

Week No.	Level of Depression															
	0	1	2	3	0	1	2	3	0	1	2	3	0	1	2	3
	Group 0								Group 1							
	BDI				CRS				BDI				CRS			
Preparation:																
1	3	1	6	16	14	4	1	7	2	1	7	9	10	5	2	5
5	5	4	5	6	13	2	2	3	8	3	5	5	13	5	1	1
Treatment:																
7	8	3	9	4	17	2	3	2	9	4	6	1	11	4	4	-
9	9	3	4	4	15	1	3	1	11	3	3	5	14	3	2	3
11	5	2	3	3	10	1	2	-	9	1	4	2	11	1	1	3
13	8	1	4	1	10	3	1	-	5	1	3	2	7	-	-	4
15	6	1	-	1	7	1	-	-	4	-	3	1	5	-	2	1
17	4	2	1	-	6	-	1	-	5	1	3	1	8	1	-	1
Follow up:																
19	3	1	-	1	4	1	-	-	3	1	3	1	4	1	3	-
21	4	-	-	1	4	1	-	-	8	3	2	1	11	-	2	1
26	1	-	-	1	1	-	1	-	3	-	1	-	4	-	-	-
30	2	1	1	-	3	1	-	-	5	1	2	-	7	-	1	-

Key: 0 = No Depression
 1 = Mild Depression
 2 = Moderate Depression
 3 = Severe Depression