

CHAPTER 1

THE PROBLEM

1.1 INTRODUCTION

Women are preoccupied with losing weight and being thin. Similarly health and fitness professionals are often the leading voices in promoting leanness as a critical component of health. In the past 20 years the obesity epidemic has grown rapidly not only in developed countries but also world-wide. The World Health Organisation (WHO) recently addressed the problem in a WHO consultation report where the situation was described as a "tidal wave" (Rippe, 1998).

In contrast to most Western countries, the emphasis in Africa has been on under-nutrition and food security rather than problems of being overweight and obese. Regional studies however, do indicate a growing prevalence of overweight and obesity in certain socio-economic groups. Epidemiological studies in South Africa have found that 18.0% of caucasian women have a Body Mass Index (BMI) exceeding 30, thus indicating obesity (Jooste 1988 et al.).

Substantial financial resources are expended in the effort to alter body weight and body shape. In the United States of America over 30 billion dollars are spent yearly on weight-loss efforts (Technology Assessment Conference Panel, 1993). As with the pursuit of any goal, there are costs and consequences associated with the quest for an ideal body. If taken to an extreme, the ultimate cost may be life itself.

Obese women are generally poorly motivated to participate in exercise programs. Numerous negatively perceived reasons are responsible for this viz.:

- Shame and embarrassment at bodily exposure;
- Real or imagined negative attention or ridicule from those more fit than themselves; and
- Anxiety about time taken away from work, family or other pleasurable activities (Knapp, 1988). Obese women may also perceive themselves as having low energy levels and poor self-efficacy related to exercise (Foreyt & Goodrick, 1991).

Several exercise adherence strategies reported in the literature may provide the absent motivation for this population. Habitual exercise and exercise maintenance may be reinforced:

- a) if exercise intensity is low with fitness goals being realistic and achievable (Epstein, et al., 1984);
- b) if exercises offer low risk of injury (Epstein et al., 1984);
- c) if group camaraderie is developed among exercise participants (Brownell, 1982; Oldridge, 1984; Gillett, 1988);
- d) if exercise activities are perceived as enjoyable and fun (Franklin, 1994; Wankel, 1994);
- e) if demonstrable changes in fitness and health status occur as a result of participation (Martin & Dubbert, 1984; Ward & Morgan, 1984);
- f) if goal setting is used to assist in developing commitment and motivation (Epstein et al., 1984; Martin & Dubbert, 1984; Gillett, 1988).

A number of new exercise devices have been introduced to the South African public in the past decade. These include computerised cycle ergometers, stair-step ergometers, computerised rowing machines, cross country skiing simulators and many others are now available for the home, gymnasium and commercial "health spas". Continuous assistive-passive exercise (CAPE) tables are another recent addition to the exercise equipment market.

CAPE training is based on the laws of repetition and is defined as exercise with no resistance. Toning tables are designed to produce rhythmic mechanical stimuli. Continuous assistive-passive exercise regimens usually consist of a set of six tables which provide a combination of passive, continuously assisted and light resistive exercise for the trunk and extremities. This exercise modality has great appeal to many sedentary and obese subjects because it promises some of the benefits of vigorous exercise without strenuous effort. This form of exercise is claimed to reduce girths at specific body sites, such as the legs, hips, waist and upper arms, without strenuous exercise (Slim Images Limited, 1999).

1.2 STATEMENT OF THE PROBLEM

The treatment of obesity and weight-loss strategies have become a commercial enterprise. Unless the efficacy of a potential therapeutic modality, whether it be exercise, behavioural, dietary or pharmacological, has been scientifically proven its application can be considered irresponsible.

The concept of continuous assistive-passive exercise (CAPE) was developed by a physician in the late 1940's and rediscovered in the mid 1980's. While many consumers are using this exercise modality, the physiological rationale for the use of continuous assistive-passive exercise (CAPE) training is questioned by clinicians and researchers familiar with exercise training.

Research reflects a paucity of knowledge with respect to the various aspects of CAPE training and toning, in particular. To illustrate this point it is evident that no clear definitive guidelines, recognised specialists or body of literature reporting experimental and/or long term studies exist in this area of exercise science. In cognisance of the foregoing, the question comes to mind whether or not, and to what extent, the advent of CAPE training has made a significant contribution to rejuvenating the human body.

1.3 PURPOSE AND AIM OF THE STUDY

The purpose of this study was two-fold:

- firstly, to determine whether an eight-week continuous assistive-passive exercise programme has a positive effect on various physiological parameters among obese females; and
- secondly, to determine whether a concomitant dietary intervention would influence the effectiveness of continuous assistive-passive exercise.

1.4 HYPOTHESES

In accordance with the stated purpose of this study the following hypotheses were formulated:

1. An eight-week continuous assistive-passive exercise (CAPE) program would have a beneficial effect on various physiological parameters among obese females; and
2. Simultaneous dietary intervention would enhance the effectiveness of an eight-week continuous assistive-passive exercise (CAPE) program.

1.5 DELIMITATION

The scope of research undertaken was delimited to an experimental epidemiological study. Within this context obesity was interpreted as a form of pathology/disease, with the evaluation of the eight week continuous assistive-passive exercise (CAPE) programme serving as an assessment of the efficacy of exercise prescription as a rehabilitative intervention modality (Walter & Hart, 1990; Van Heerden, 1996).

CHAPTER 2

LITERATURE REVIEW

2.1 DEFINITION OF OBESITY

Obese: From Latin *obesus*, past participle of *obedere* (to devour) which derives from *ob* (meaning "to") and *edere* (meaning "eat"). (Webster, 1988).

Although any single definition of obesity is apt to be incomplete, obesity is a chronic disease characterised by an excess of adipose tissue. It should be considered a serious medical condition that can lead to significant morbidity and mortality rather than a character flaw or personal weakness (Oeser, 1997).

Obesity is also defined as an excessive accumulation of fat beyond that considered normal for one's age, gender and body type (Sharkey, 1984). Buskirk (1974) in addressing the question "Who is fat?" states that obesity is difficult to define in quantitative terms. Obesity refers to the above-average amount of fat contained in the body, this in turn being dependent on the lipid content of each fat cell and on the total number of fat cells. A woman with a 30% body fat content is defined as obese (Sharkey, 1984; Mc Ardle et al., 1996). Sharkey (1984) regards even lower levels of body fat as preferable, but by this definition a large percentage of the adult population qualifies as obese.

Obesity is probably best defined as any degree of excess weight that imparts a health risk (Thomas, 1995).

2.2 PREVALENCE OF OBESITY

The economist John Kenneth Galbraith wrote in the 1958 edition of *The Affluent Society* that, "More people die in the United States of too much food than of too little" - an observation that is even more fitting today than it was in the 1950's (Oeser, 1997).

The prevalence of obesity is rising to epidemic proportions around the world at an alarming rate. The rise in obesity is not restricted to well-developed countries. With increasing Westernisation, the prevalence of overweight and obesity appears to be rising amongst more affluent populations of less-developed countries, even in those countries with current food security problems and significant rates of under-nutrition (Brown, 1998).

Nearly one third of adults in the United States of America (USA) are obese and the prevalence is increasing according to a recent report by the Institute of Medicine (Thomas, 1995; Mc Ardle et al., 1996). This was a dramatic increase compared to previous surveys with the incidence of obesity being particularly high among women (Williamson, 1993; Mc Ardle et al., 1996).

Similar increases in the prevalence of obesity were also found in other countries. Epidemiological studies conducted in South Africa have found that 18,0% of Caucasian women were obese, having a BMI exceeding 30 (Jooste, et al., 1988). Australian Bureau of Statistics figures show that one third of Australian men and almost a quarter of Australian women are either overweight or obese (Thompson, 1995).

2.3 CONSEQUENCES OF OBESITY

Obesity in combination with other risk factors, kills an estimated 300,000 Americans each year and it is the second most common cause of preventable deaths (Mc Ginnes & Foege, 1993). Similar trends have been noted world-wide (Gurney & Gorstein, 1988).

Epidemiological studies have revealed a clear association between obesity and the risks for cardiovascular disease, non-insulin dependent diabetes mellitus, certain forms of cancer, gall-stones, some respiratory disorders, osteoarthritis, hypertension and an increase in overall mortality (Pi-Sunyer, 1993).

Overweight also brings major economic costs to the individual and society. Estimates of annual costs run as high as nearly 8% of the total health care costs in the USA and 5% of the total health care costs in the Netherlands (Colditz, 1992).

Negative attitudes towards the obese can lead to discrimination in many areas of their lives including health care and employment. The psychological consequences of obesity can range from lowered self-esteem to clinical depression. Rates of anxiety and depression are three to four times higher among obese individuals (Brown, 1998).

Obese women not only marry less, but also complete fewer years of schooling and have lower incomes (more have incomes below the poverty level). Much of this is attributed to discrimination against overweight persons (Gortmaker et al., 1993)

2.3.1 Health Risks of Overweight and Obesity

The major health risks of obesity increase in a curvilinear relationship, with prevalence increasing progressively and disproportionately with increasing weight (Blumenkrantz, 1998). The health consequences of obesity range from a number of non-fatal complaints such as respiratory difficulties, musculo-skeletal problems, skin problems and infertility that impact on the quality of life, to complaints that lead to an increased risk of premature death including non-insulin dependent diabetes, gall bladder disease, cardiovascular problems (hypertension, stroke, coronary artery disease) and cancers which are hormone related and associated with the large intestine (Brown, 1998). Hypertension, diabetes and raised serum cholesterol are between two and six times more prevalent among overweight women. Severe obesity is associated with a twelve-fold increase in mortality in 25-35 year olds when compared to lean individuals (Brown, 1998).

Adults who are 20% above their desirable weight show an overall increase of 20% in the likelihood of death from all causes, a 25% increase in death from coronary artery disease, a 10% increase from stroke, twice the risk of diabetes, and a 40% increase in gall-bladder disease. Among adults aged 15 to 39, whose relative body weight when initially measured is 125% to 135% of normal, there is an aggregate mortality of 170% of normal at a 15-22 year follow up. Adults 40% above desirable weight have a

55% increase in mortality from all causes, 70% increase from coronary artery disease, 75% increase from a stroke, and a 400% increased mortality from diabetes (Blumenkrantz, 1998).

2.3.1.1 Obesity and Hypertension

Hypertension is a common concomitant of obesity. Weight gain in young adult life is a potent risk factor for the later development of hypertension. In overweight young adults, aged 20-45, the prevalence of hypertension is six times that of their normal weight peers (Blumenkrantz, 1998). Hypertension is infrequent in "primitive" populations who tend to weigh less with advancing years (Blumenkrantz, 1998).

A reduction in blood pressure usually follows weight loss. Hypertension is also a very strong and independent risk factor for coronary heart disease (Bray, 1987; Burton et al., 1985; Pi-Sunyer, 1993). The regional distribution of fat in the body may have an important effect on blood pressure risk, with central and upper body fat being more likely to raise blood pressure than lower body fat in the gluteal or thigh region (Blumenkrantz, 1998).

2.3.1.2 Obesity and Diabetes Mellitus

Moderate obesity, particularly abdominal obesity can increase the risk of non-insulin dependent diabetes mellitus (NIDDM). Fat tissue apparently has two roles in promoting diabetes: it increases the demand for insulin and, in obese individuals, it creates insulin resistance, and therefore, hyperinsulinemia. It is therefore possible that nutrients are preferably sent into fat cells for storage (Bray, 1987; Pi-Sunyer, 1993).

Some of the insulin resistance in obesity can be attributed to a decrease in insulin receptors; there are also intracellular post-receptor defects. Weight reduction in the obese non-insulin dependent diabetic will lead to improvement of glycemic control as well as improvement of concomitant medical problems such as hypertension or hyperlipidemia (Blumenkrantz, 1998; Bray, 1987).

2.3.1.3 Obesity and Cancer

The American Cancer Society has published data on 750,000 individuals studied between 1959 and 1972. In these studies, as BMI increased, so did the incidence of death from cancer, even independent of cigarette smoking (Plowman & Smith, 1997).

Overweight men have a significantly higher mortality rate for colorectal and prostate cancer. Men whose weight is 130% or more above average are 2.5 times more likely to die of prostate cancer during a 20-year follow-up compared to men of average weight (Blumenkrantz, 1998; Giovannucci et al., 1995).

Overweight females showed increased rates of breast (upper body fat localisation), cervical, endometrial, uterine and ovarian cancer. The suspected link, at least for the females, is the level of estrogen. Adipose tissue is a site for estrogen formation in all females and the major site in post-menopausal females. Estrogen formation is increased in overweight and obese individuals owing to the increased number of adipose cells (Bray, 1987; Burton et al., 1985; Magnusson et al., 1998; Pi-Sunyer, 1993).

2.3.1.4 Obesity and Cardiovascular Disease

Framingham, Massachusetts, has been the site of a longitudinal study investigating the risk factors for heart disease in more than 5000 residents. Data from 26-30 years of follow-up have shown that overweight or obesity is a significant predictor of cardiovascular disease, independent of age, cholesterol, systolic blood pressure, cigarette smoking, and glucose tolerance. The Framingham investigators have concluded that if everyone were at or within 10% of his or her desirable weight, there would be 25% less coronary heart disease and 35% less congestive heart failure and stroke. The risk is greater for those who become obese early in life rather than in old age (Bray, 1987; Burton et al., 1985; Pi-Sunyer, 1993; Simopoulos, 1987). The degree of risk is higher for those who store their fat in the android pattern than in the gynoid pattern (Plowman & Smith, 1997).

2.3.1.5 Gall-bladder Disease and Hypercholesterolemia

In the Framingham study individuals who were 20% or more above the average weight for their height were about twice as likely to develop gall-bladder disease as those who weighed 10% less than the average weight (Bray, 1987; Pi-Sunyer, 1993). In another study the frequency of gall-bladder disease was largely explained by weight and age, and in females, the number of viable pregnancies. Obese females between 20 and 30 years of age had a 600% greater chance of having gall-bladder disease than average-weight females. Within all age groups the frequency of gall-bladder disease increased with the level of body weight (Bray, 1987; Pi-Sunyer, 1993).

Part of the explanation for the increased prevalence of gall-bladder disease in overweight or obese individuals can be linked to the effect of increased body weight and fat on cholesterol. There is a significant relationship between fatness and cholesterol level that is direct and positive (Bray, 1987). For each kilogram of fat, approximately 20 mg/dL of cholesterol is synthesised. In obese persons the bile is therefore more saturated with cholesterol. This increased presence of cholesterol in bile is the likely cause of the increased risk of gall-bladder disease (Bray, 1987; Blumenkrantz, 1998).

2.3.1.6 Obesity and Pulmonary Abnormalities

There are several abnormalities in pulmonary function among obese individuals. At one extreme are patients with so-called Pickwickian syndrome, or the obesity-hyperventilation syndrome, which is characterised by somnolence and hyperventilation (Bray, 1987; Blumenkrantz, 1998).

In patients who are obese, there is a fairly uniform decrease in expiratory reserve volume and a tendency to reduction in all lung volumes. As an individual becomes more obese the muscular work required for ventilation increases. In addition, respiratory muscles may not function normally in obese individuals (Babb, 1999).

2.3.1.7 Obesity and Arthritis

Although the cause is unclear, there is a significant correlation between uric acid levels and body weight (Pi-Sunyer, 1993). In the 45-64 year old age group, the prevalence of gout increases dramatically when relative weight is greater than 130% above desirable (Blumenkrantz, 1998).

An increase in body weight adds trauma to weight bearing joints and in middle age, a woman's excess body weight is a major predictor of osteoarthritis of the knee. This is a mechanical problem and not a metabolic one. Weight loss will markedly decrease the chance of developing osteoarthritis (Bray, 1987).

2.3.1.8 Obesity and Functional and Psychological Disorders

The obese individual has functional impairment in activities of daily living. This dysfunction is related to sleep, recreation, work and social interaction. Obese patients also have physical incapacity due to back and joint problems and shortness of breath. This contributes to their proneness to fatal accidents (Blumenkrantz, 1998).

In the severely obese there is an increased incidence of absenteeism and unemployment. Discrimination against obese persons is common in both academic and work settings. Impairment in body image is the major form of psychological disturbance specific to obese persons. Psychological disturbances do not appear more commonly in overweight persons than in those with normal weight. Emotional disturbances are often likely to be a consequence of obesity rather than the cause. In some studies, obese persons were found to be significantly less anxious and depressed than normal weight persons (Blumenkrantz, 1998; Pi-Sunyer, 1993).

The increase in prevalence of all of the above-mentioned diseases applies to adults, but overweight or obese children also show increased risk factors, though not the actual diseases, compared with normal-weight children (Williams et al., 1992).

2.4 ETIOLOGY OF OBESITY

Obesity is due to an imbalance between energy intake and expenditure (Saris, 1989). When calorie intake exceeds energy expenditure the excess calories are stored in adipose tissue (Oeser, 1997). Obesity results if this net positive balance is prolonged. Although it is often assumed that obesity results simply from overeating or a sedentary lifestyle, the problem is more complex than this. Obesity is regarded as a "complex disease" because it arises from multifaceted interactions of genetic and environmental factors (Lindpainter, 1995). The ultimate cause of obesity is therefore an imbalance between calorie intake and energy expenditure resulting from a complex interaction of genetic, physiologic, behavioural and environmental factors (Oeser, 1997).

It is worth noting that glandular abnormalities are generally not the cause of obesity per se, except in certain instances of endocrinopathy (Mc Ardle et al., 1996). However, while the causes of obesity seldom are linked to hormonal aberrations, obesity can trigger a variety of abnormal hormonal responses (Pi-Sunyer, 1994).

- **Genetics**

It has been long known that the tendency to gain weight runs in families. Children of obese parents are at two to three times increased risk of obesity as adults compared to children in families in which neither parent is morbidly obese (Mc Ardle et al., 1996). Although early studies estimated that hereditary influence accounted for up to 80% of the tendency to gain weight, more recent data indicate that 33% of the BMI is attributable to genetics (Stunkard, 1996). In rare cases, human obesity results from a single gene disorder such as in the Bardet Biedl, Prader Willi, Ahlstrom and Cohen syndromes (Spiegelman & Flier, 1996).

Research with a strain of mice that balloon up to five times the girth of normal mice has provided evidence to support the contention that some people are genetically "destined" to become overfat (Arner, 1991). The mutation of a gene called obese, or simply ob, is believed to disrupt hormonal signals that regulate the animal's metabolism, fat storage and appetite, causing energy balance to tip in the direction of fat accumulation (Zhang, 1994). Although mutations in the ob gene have not been

found in humans, there is evidence suggesting a linkage between the ob gene and some obese populations (Clement et al., 1996). The ob gene is normally activated in adipose tissue, where it causes the production of a body fat-signalling, hormone-like protein (a satiety protein called ob protein or leptin) that is secreted into the bloodstream (Halaas, 1995). This hormone is then transported to the ventromedial nucleus of the hypothalamus, the area of the brain considered the control centre for appetite. Normally, the action of this hormone blunts the urge to eat when the calorie intake is sufficient to maintain ideal fat stores (Mc Ardle et al., 1996).

Two other genes that have been implicated in the development of human obesity are the genes that encode for the glucocorticoid receptor and Na-K-ATPase (Clement et al., 1996).

- **Environment**

Since the genetic factor accounts for only a third of the variance in body weight, environmental influences must therefore account for the balance (Oeser, 1997). Several environmental factors, involving both energy intake and energy output, contribute to obesity (Mc Ardle et al., 1996). For years, doubt has persisted about the contribution of excessive food intake to obesity (Oeser, 1997). A recent study using double-labelled water to measure energy expenditure (and thus energy consumption) have erased this doubt and made it clear that obesity is associated with increased food consumption (Lichtman et al., 1992).

The high prevalence of a sedentary lifestyle, resulting from the proliferation of labour-saving machinery and contrivances, is a major environmental factor contributing to the development and maintenance of obesity in western societies (Oeser, 1997). Observations of older men and women who maintain active lifestyles suggest that the "normal" pattern of fat gain in adulthood can be attenuated significantly (Mc Arle et al., 1996). For both young and middle-aged men who exercised regularly, the time spent in physical activity was inversely related to body fat level (Mc Ardle et al., 1996).

Another environmental influence that has recently been recognised is smoking cessation (Oeser, 1997). Although the health benefits of smoking cessation are enormous, smoking cessation nevertheless appears to be associated with a 4 to 5 kg weight gain and therefore a small increase in the prevalence of obesity (Flegal et al., 1995).

Socio-economic status is inversely correlated with the prevalence of obesity, especially among women, with lower socio-economic status favouring the development of obesity (Sobel & Stunkard, 1989).

2.4.1 Heredity and Body Composition

There is evidence in humans that both obesity and localised fat deposition are determined solely by genetic transmission in some individuals. Thirteen genetic syndromes have been identified in the medical literature, but all are considered to be pathological disorders and are extremely rare. Even the most common has an incidence rate of only about 1 in 25,000. For the vast majority the contribution of genetics to body composition is not absolute, despite what experience tells about familial resemblance (Boileau et al., 1984; Simoneau & Bouchard, 1995).

Studies conducted to determine the contribution of genetics to body composition show the expected pattern of very low relationships between biological siblings, whether born separately or together (dizygotic twins) and moderate relationships between monozygotic twins (Plowman & Smith, 1997; Bouchard, 1991).

Bouchard and Perusse (1988) have concluded the following despite these moderate relationships.

- Only about 5% of the total variation in BMI and skinfold thickness are genetically transferable.
- About 25-30% of the variance in percent body fat, fat-free weight and fat distribution patterning is genetically transferable.

- About 30% of the transmission of body composition variables is linked to cultural factors, defined as the environment established by the family (such as the amount of food consumed, types food eaten and activity patterns).
- About 45-65% of the variance in body composition is genetically non-transferable.

2.5 BIO-ENERGETICS OF METABOLISM - AN OVERVIEW

The total of all energy transformation that occurs in the body is referred to as metabolism (Plowman & Smith, 1997). During the initial stage of sudden physical exercise the additional energy required is mainly produced by the breakdown of muscle glycogen to lactate. Blood glucose does not contribute substantially during the first minutes of exercise. For this to happen glycogenolysis of the liver glycogen stores has to increase. The formed lactate is released into the bloodstream and taken up by the liver, the heart and non-active muscle tissue, where it is either oxidised or resynthesized to glucose. At a later stage as glucose production from the liver is significant, muscle will increasingly use blood glucose for energy production. (Brouns, 1993; Mc Ardle et al., 1996).

Additionally, lipolysis in fat cells - initially a gradually increasing process - leads to high blood fatty acid levels, through which the contribution of fatty acids for energy production increases. Fatty acids become more and more oxidised in muscle and the liver. Ketone bodies, which result from incomplete fat oxidation in the liver, are taken up from blood by the heart and the muscle for their final oxidation (Brouns, 1993).

With increasing metabolic stress, especially in conditions of carbohydrate depletion, synthesis of protein may be decreased and the degradation of amino acids increases (Brouns, 1993). Degradation of amino acids in muscle and liver finally leads to the production of urea which is excreted with urine and sweat. The carbon skeletons of the amino acids enter the citric acid cycle in the liver (where they are used for gluconeogenesis) and muscle (where they are be oxidised) (Brouns 1993).

With ongoing exercise and also during fasting, the endogenous carbohydrate stores in liver and muscle become depleted. If no glucose is produced from gluconeogenic precursors in the liver and kidney, the blood glucose level drops sharply. Gluconeogenic precursors are amino acids, glycerol and lactate. At the same time, fat oxidation will be maximised, resulting in a reduced need for carbohydrate. Ketone bodies resulting from fat metabolism in the liver are metabolised by the heart, skeletal muscle and, with prolonged fasting, also in the brain (Brouns, 1993).

2.5.1 Fat Metabolism

Although the body may prefer to use carbohydrate as fuel from the standpoint of oxygen cost, the importance of fat as an energy source should not be underestimated (Plowman & Smith, 1997). Fat is found in many common foods. Fat in the form of triglyceride (sometimes known as triacylglycerol) is the major storage form of energy in humans (Hawley, 1998). Some triglyceride is stored within muscle cells, but the vast majority is deposited in adipose cells and comprises approximately 10-15% of the body weight of males and 20-25% of the body weight in females (Malina & Bouchard, 1991).

Roughly half of this adipocyte storage occurs subcutaneously (Hawley, 1998). The remaining stores surround the major organs of the abdomino-thoracic cavity as support and protection (Plowman & Smith, 1997). Triglycerides are turned over constantly in the body. In fact, a body's fat is burned completely about every 3-4 weeks, so adults are definitely not carrying any "baby fat" (Marieb, 1992).

Fat is an excellent storage fuel for several reasons. Fat is an energy-dense fuel yielding 9.13 kcal per gram, while both carbohydrate and protein yield slightly less than 4 kcal per gram (Plowman & Smith, 1997). The reasons for these figures are related to the chemical structure of the substrates - specifically, the amount of oxidizable carbon and hydrogen. It is clear to appreciate the difference by observing the chemical composition of the free fatty acid palmitate, which is $C_{16}H_{32}O_2$. This fatty acid has almost three times the amount of carbon and hydrogen, but only a third the amount of oxygen as glucose. It is important to note that hydrogen donates its electrons during oxidative phosphorylation (Plowman & Smith, 1997; Mc Ardle et al., 1996).

Carbohydrate, in the form of glycogen, is stored in the muscles with a large amount of water: 2.7 g of water per gram of dried glycogen. Triglyceride is stored dry, thus the energy content of fat is not diluted (Plowman & Smith, 1997). If humans had to store the comparable energy amount as carbohydrates, we would be at least twice as large (Newsholme & Leech, 1983).

Glycogen stores are relatively small in comparison to fat stores. A person can deplete the stored glycogen in as little as 2 hours of heavy exercise or one day of bed rest, whereas fat supplies can last for weeks, even with moderate activity (Plowman & Smith, 1997). People often seem concerned about having too much body fat, but this storage capacity is undoubtedly important for survival of the species when food is not readily available (Plowman & Smith, 1997).

A triglyceride comprises one glycerol and three fatty acids. The triglycerides stored in adipose tissue must first be broken down into glycerol and free fatty acids (FFA) before they can be used as fuel (Mc Ardle et al., 1996). Seven fatty acids predominate in the body, but since three fatty acids combine with a glycerol to make up a triglyceride; there are 343 ($7 \times 7 \times 7$) different combinations possible (Péronnet et al., 1987). Some common fatty acids are oleic acid, palmitic acid, stearic acid, linoleic acid and palmitolic acid (Plowman & Smith, 1997).

Fatty acids may be saturated, unsaturated or polyunsaturated.

- A saturated fatty acid contains no double bonds between carbon atoms; the remaining bonds attach to hydrogen. The fatty acid molecule is said to be saturated because it holds as many hydrogen atoms as is chemically possible.
- Unsaturated fatty acids contain one or more double bonds along the main carbon chain. In this case, each double bond in the carbon chain reduces the number of potential hydrogen-binding sites, therefore the molecule is said to be unsaturated with respect to hydrogen. If only one double bond is present along the main carbon chain, the fatty acid is said to be mono-unsaturated. If there are two or more double bonds along the main carbon chain, the fatty acid is poly-unsaturated (Mc Ardle et al., 1996).



The breakdown of triglycerides into glycerol and fatty acids is catalysed by the hormone-sensitive enzyme lipase. The glycerol is soluble in blood, but the free fatty acids (FFA) are not. Glycerol can enter glycolysis in the cytoplasm but is not typically utilised by muscle cells in this fashion (Newsholme & Leech, 1983). The direct role of glycerol as a fuel in the muscle cells during exercise is so minor that it need not be considered. Glycerol can be converted to glucose by the liver (Plowman & Smith, 1997). FFA must be transported in the blood bound to albumin. Specific receptor sites on the muscle cell membrane receive the FFA into the cell. The FFA must then be translocated or transported from the cytoplasm into the mitochondria. Once in the mitochondrial matrix, the FFA undergoes the process of beta-oxidation (Plowman & Smith, 1997).

2.5.1.1 Beta Oxidation

Beta oxidation is a cyclic series of steps that breaks off successive pairs of carbon atoms from FFA, which are then used to form acetyl co-enzyme A (acetyl CoA). Acetyl CoA is the common intermediate by which all foodstuffs enter the Krebs cycle and electron transport system (Plowman & Smith, 1997). Most fatty acids have 14-24 carbons. The number of cycles thus depends upon the number of carbons available (Mc Ardle et al., 1996).

When there is an adequate supply of oxalo-acetate to combine with, the fat-derived acetyl CoA enters the Krebs cycle and proceeds through to electron transport and oxidative phosphorylation. As with glycolysis, adenosine triphosphate (ATP) is used for activation; but unlike glycolysis, beta-oxidation produces no ATP directly (Plowman & Smith, 1997).

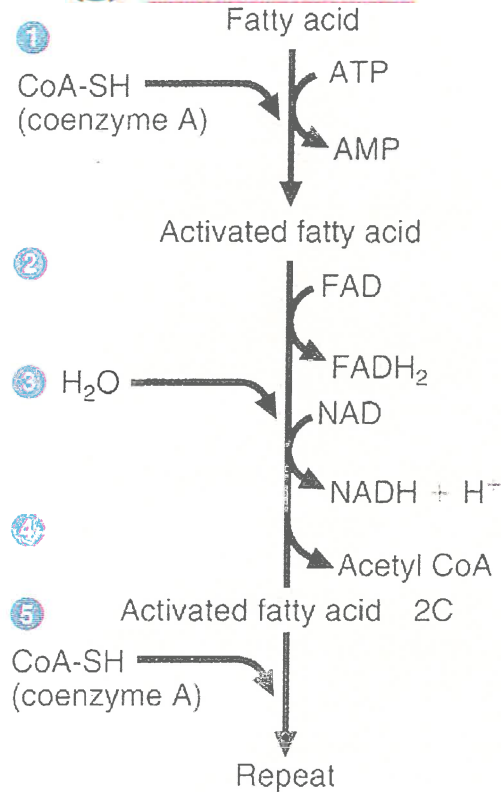


FIGURE 2.1: BETA OXIDATION
 (Plowman & Smith, 1997)

SUMMARY OF BETA OXIDATION

- Does not directly utilise O₂, but must be aerobic;
- Occurs in mitochondrial matrix;
- 1 ATP used for activation, but since it is hydrolysed to adenosine monophosphate (AMP) this is equivalent to 2 ATP being used;
- No ATP is produced directly;
- 1 FADH₂ + 1 NADH + H⁺ is produced for each pair of carbon atoms split off (yields 5 ATP);
- 1 acetyl CoA (yields 12 ATP) is produced for each pair of carbon atoms split off (Plowman & Smith, 1997).

2.5.1.2 ATP Production from Fatty Acids

The number of ATP produced from the breakdown of fat depends on which fatty acid is utilised (Plowman & Smith, 1997). For each 18-carbon fatty acid molecule, a net of 147 adenosine diphosphate (ADP) molecules are phosphorylated to ATP during beta-oxidation and Krebs cycle metabolism. Because each triglyceride molecule contains three fatty acid molecules, 441 ATP molecules (3×147 ATP) are formed from the fatty acid component of neutral fat. Because 19 molecules of ATP form during glycerol breakdown, a total of 460 molecules of ATP are generated for each triglyceride molecule catabolized for energy. This quantity represent a considerable energy yield considering that only 38 ATP molecules are formed during the catabolism of a glucose molecule in skeletal muscle (Mc Ardle et al., 1996). Energy conservation for ATP resynthesis from fatty acid oxidation is about 40% which is similar to that of glucose (Plowman & Smith, 1997; Mc Ardle et al., 1996).

Depending on a person's nutritional state, level of training, intensity and duration of a specific physical activity, between 30 and 80% of the energy for biologic work is usually supplied from intracellular and extracellular lipid molecules (Kiens et al., 1993). When high-intensity, long-duration physical activity causes significant glycogen depletion, lipid becomes the primary energy fuel during exercise and recovery (Romijn et al., 1993). Prolonged exposure to a high-fat diet brings about enzymatic adaptations that enhance one's capacity for lipid oxidation during exercise (Mc Ardle et al., 1996).

2.5.1.3 Ketone Bodies and Ketosis

In order for acetyl CoA produced by beta oxidation to enter the Krebs cycle, a sufficient amount of oxalo-acetate is necessary. When carbohydrate supplies are sufficient, fat is said to burn in a carbohydrate flame (Mc Ardle et al., 1996). When carbohydrates are inadequate (perhaps as a result of fasting, prolonged exercise, or diabetes mellitus), oxalo-acetate can be converted to glucose. The production of glucose from non-carbohydrate sources under these conditions is necessary, since some tissue, such as the brain and nervous system, rely predominantly on glucose as fuel (Marieb, 1992).

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When oxalo-acetate is converted to glucose and is not available to combine with acetyl CoA to form citrate, the liver converts the acetyl CoA derived from the fatty acids, into metabolites called ketones or ketone bodies.

There are three forms of ketones:

- Acetoacetic acid;
- Beta-hydroxybutyric acid; and
- Acetone.

(Plowman & Smith, 1997)

The ketone bodies can themselves be used as fuel by muscles, nerves, and the brain but if the ketones are not used and accumulate, a condition of ketosis occurs (Marieb, 1992). The high acidity of ketosis can disrupt normal physiological functioning especially acid-base balance (Plowman & Smith, 1997). During exercise aerobically trained individuals can utilise ketones more effectively than untrained individuals (Foss & Keteyian, 1998).

2.6 CELLULAR BASIS OF OBESITY

Although most cells store small amounts of fat, the majority of the body's fat is stored in specialised cells known as adipocytes (Vander et al., 1994). Adipose tissue is composed of a matrix of connective tissue in which white adipose cells (adipocytes) appear singularly or in small clusters. A typical cell looks something like a signet ring, a metal band with some type of stone or jewel at the top (Plowman & Smith, 1997). The nucleus of the cell appears as the stone or jewel of the ring in the cell membrane, which forms the band of the ring. The space within the confines of the cell is the site of triglyceride droplet storage (Marieb, 1992).

A typical adipocyte contains a single enormous lipid droplet, with the nucleus and other organelles squeezed to one side, making the cell resemble a class ring (Martini, 1995). There are about 30-50 billion fat cells in an adult of acceptable weight. Females have approximately 50% more fat cells than males (Plowman & Smith, 1997). Adipocytes can change their size about tenfold to store triglycerides (Martini,

1995). The increase in size (hypertrophy) of adipocytes is the manner in which increasing levels of fat are initially stored. Sometimes when the fat cell size is enlarged, the increased size causes a bulging between the fibrous tissue strands, causing a dimply, waffled appearance. These lumpy areas are often labelled as cellulite. Through this discussion, it is clear that cellulite is simply fat (Björntorp, 1989).

When the upper limit of fat storage is approached by hypertrophy (± 30 kg of fat), fat cell hyperplasia (increase in the number of cells) occurs. In fat tissue hyperplasia is the development of new adipocytes from immature precursor cells (Plowman & Smith, 1997). Adipocytes do not divide and multiply, but hypertrophy in adipocytes stimulates cell division and maturation in precursor cells (Malina & Bouchard, 1991). An overweight adult is likely to have the same number of fat cells as when she was of normal weight, but the adipocytes will be larger. An obese woman may have enlarged adipocytes, an increased number of adipocytes, or both (Plowman & Smith, 1997).

Once created, fat cell numbers are not naturally reduced, even if body weight and body fat are lost (Sjöström & Björntorp, 1974). Liposuction is the surgical removal of adipose tissue and the manner to remove adipocytes (Plowman & Smith, 1997). The maintenance of large numbers of adipocytes may be one reason why it is so difficult for an obese individual to maintain weight/fat loss once it occurs (Mc Ardle et al., 1996).

The facts emphasise the importance of avoiding the maturation of extra fat cells (Plowman & Smith, 1997). Overweight infants, children, or adolescents tend to become overweight adults, although adolescent obesity is more predictive of adult obesity than are obesity at birth or infancy (Charney et al., 1975; Dietz, 1987; Lohman, 1989). From birth to young adulthood the average cell size doubles or even triples. Most of this increase in size happens during the first year after birth. From the first year to the onset of puberty there is no significant increase in size and no difference between the sexes in this regard (Plowman & Smith, 1997). At puberty cell size increases in females but remain fairly constant in males (Sjöström & Björntorp, 1974).

Not all-adipose cells are the same size. Internal (viceral) fat cells are generally smaller than subcutaneous fat cells. Furthermore, not all-subcutaneous cells are equal in size. For example, gluteal adipocytes tend to be larger than abdominal adipocytes, which in turn are larger than subscapular cells (Plowman & Smith, 1997).

At birth the number of adipocytes is approximately 5 billion. For the number to increase to the average adult population of 30 billion, considerable changes must occur (Plowman & Smith, 1997). A severely obese person may have as many as 260 billion adipocytes (Sjöström & Björntorp, 1974).

From the 1st year to the onset of puberty there is a gradual but steady increase in number of adipocytes with no differences appearing between the sexes (Malina & Bouchard, 1991). At puberty the cellularity of adipose tissues increases greatly in both males and females, but the female increase far exceeds the male increase. This increase in fat cell number plateaus in late adolescence and early adulthood and ideally remains at this level. Hyperplasia can, and often does, occur in adulthood (Malina & Bouchard, 1991).

The importance of adipocyte number in obesity is further illustrated by relating total body fat content to both cell size and cell number. As body fat increases, adipocyte numbers eventually reaches some biologic upper limit. There are two critical periods in the development of adipocytes, namely infancy and adolescence. This should not however be interpreted that fat cells cannot be added during adulthood (Plowman & Smith, 1997). During growth males tend to accumulate more subcutaneous fat on the trunk and females on the extremities (Plowman & Smith, 1997). Even if adipocytes could double in size this would still not account for the large difference in the total fat mass between the obese and normal people (Mc Ardle et al., 1996). Thus once adipocyte hypertrophy has ended, cell number becomes the key factor determining any further extent of obesity.

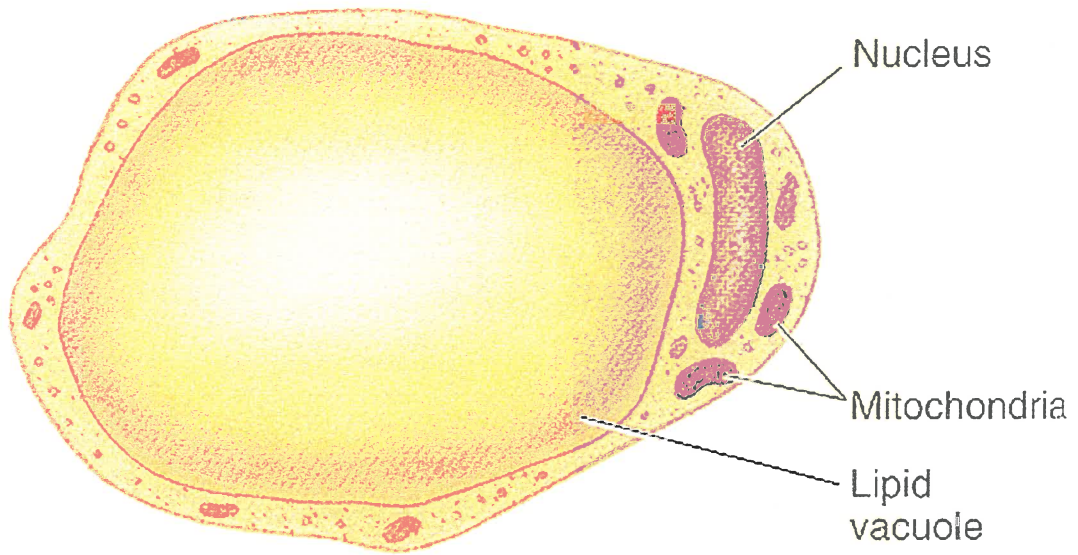


FIGURE 2.2: WHITE ADIPOSE CELL (Plowman & Smith, 1997)

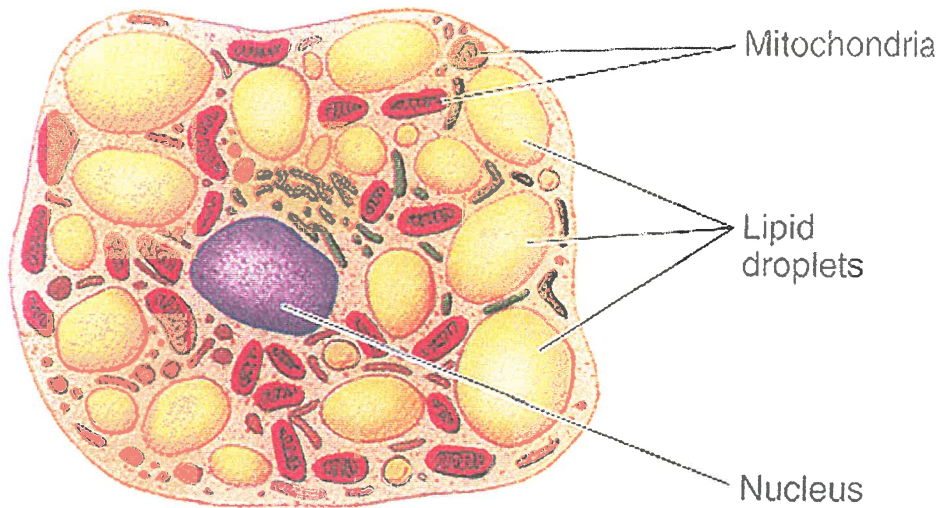


FIGURE 2.3: BROWN ADIPOSE CELL (Plowman & Smith, 1997)

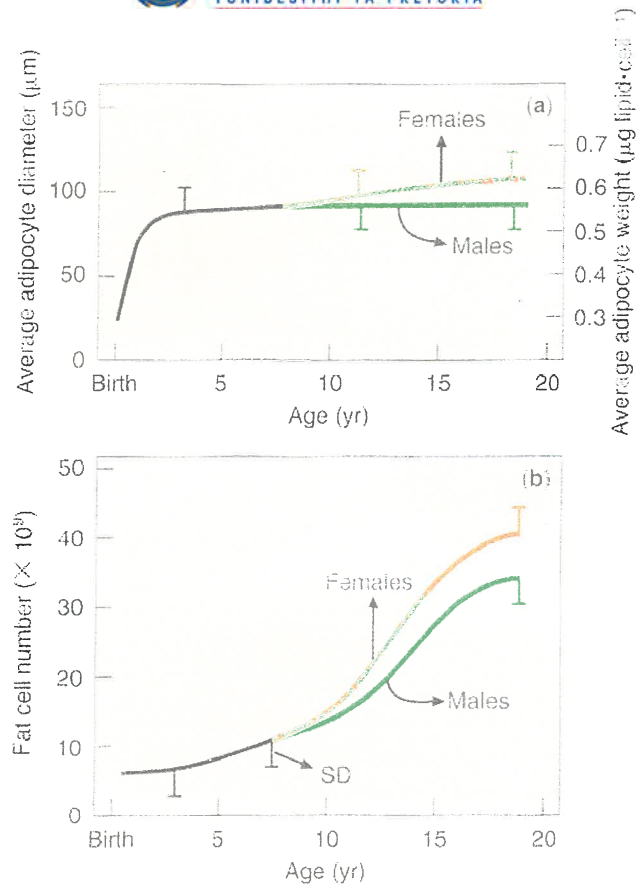


FIGURE 2.4: CHANGES IN ADIPOSE CELL SIZE AND NUMBER WITH GROWTH
(Plowman & Smith, 1997)

2.7 REGIONAL FAT DISTRIBUTION

The patterning of the body's adipose tissue distribution, independent of total body fat, alters the health risks of obesity (Mc Ardle et al., 1996). The location of fat storage varies among individuals. During growth males tend to accumulate more subcutaneous fat on the trunk and females on the extremities (Plowman & Smith, 1997). Generally, humans distribute fat in three basic patterns:

- Android (central);
- Gynoid (peripheral); and
- Intermediate.

(Plowman & Smith, 1997)

The android pattern, also known as the abdominal or "apple" pattern, is predominantly found in males. It is characterised by the storage of fat in the nape of the neck, shoulders and abdomen (upper part of the body). In this pattern the largest quantity of fat is stored internally (Plowman & Smith, 1997). The increased health risk from fat deposition in the abdominal area, especially in the internal, visceral deposits, may be a result of this tissue's lively lipolysis in response to catecholamine. Lipids stored in this area are more responsive metabolically than those in the gluteal and femoral regions and thus more likely to enter into processes related to heart disease (Mc Ardle et al. 1996). Excess fat in the abdominal cavity pushing against the abdominal muscles causes hardness of the abdominal region. Once the amount of fat to be stored exceeds the capacity of the abdominal cavity, subcutaneous sites are loaded (Campaigne, 1990; Stamford, 1991).

The gynoid pattern also referred to as the gluteofemoral or "pear" pattern is predominantly found among females. It is characterised by the storage of fat in the lower part of the body, specifically, in the thighs and buttocks, with the largest quantity being stored subcutaneously. These sites tend to be soft and to jiggle. No pseudohardness is apparent (Campaigne, 1990; Stamford, 1991).

Central fat deposition (gynoid-type) increases one's risk of hyperinsulinemia, insulin resistance, non-insulin-dependant diabetes, endometrial cancer, hyper-cholesterolemia, hypertension and atherosclerosis (Mc Ardle et al., 1996). According to Plowman and Smith (1997) there is a growing body of evidence that the deposition of fat in the gluteal-femoral region by females is linked to reproductive function. In particular, gluteal-femoral fat may furnish energy for the development of the fetus primarily during the latter states of pregnancy and for the new-born child during lactation.

The third type of regional fat distribution is simply known as the intermediate pattern. Fat is stored in both the upper and the lower parts of the body, giving a rectangular cubic appearance. All three patterns are found in both males and females, despite the sex-specific predominance associated with the android and gynoid shapes (Campaigne, 1990; Stamford, 1991).

As abdominal fat deposits are easily mobilised, it is possible to reduce fat accumulation in this area relatively easily. Gluteal femoral fat deposits on the other hand are not easily mobilised, and thus it is difficult to reduce fat accumulation in these areas. The potential for reshaping this gluteo-femoral fat pattern is extremely limited (Campaigne, 1990; Stamford, 1991).

The variation in fat deposit mobilisation is hormonally based (Plowman & Smith, 1997). Two different receptors have been identified in fat cells:

- Alpha-receptors, which inhibit fat transfer to and from the adipocytes; and
- Beta-receptors, which enhance fat transfer to and from the adipocytes.

Alpha-receptors predominate in the lower body (abundant in the gynoid pattern) and beta-receptors are concentrated in the upper body (abundant in the android pattern). (Plowman & Smith, 1997; Mc Ardle et al., 1996).

Under the influence of epinephrine (released from the adrenal medulla), fat from the abdominal cells is easily mobilised and dumped into the circulatory system. If the free fatty acids and glycerol are used as fuel to support exercise, there is no problem. However, when epinephrine is released in times of emotional stress, there is no need for the excess fuel. Fatty acids and glycerol are then routed to the liver where they are primarily converted to low-density lipoproteins (LDLs). LDL is largely composed of cholesterol and is associated with atherosclerosis and an increased risk of coronary artery disease (CAD) (Brownell et al., 1987; Campaigne, 1990; Stamford, 1991).

Abdominal fat cells tend to be larger than fat cells found in other parts of the body. Larger fat cells are associated with glucose intolerance (the inability to dispose of a glucose load effectively), coupled with insulin resistance and hyperglycaemia, and an excess of insulin in the blood (hyperinsulinemia). These conditions are associated with diabetes mellitus and hypertension both of which are risk factors for CAD. The latter occurs because of the action of insulin in promoting reabsorption of sodium by the kidneys (Brownell et al., 1987; Campaigne, 1990; Stamford, 1991).

Computation of a waist-to-hip ratio has been (WHR) suggested as a manner to estimate the health risk associated with the pattern of fat distribution (Van Itallie, 1985). Research has shown that the WHR is a stronger predictor for diabetes, coronary artery disease, and overall mortality risk than body weight, body mass index, or percent body fat (Folsom et al., 1986; Van Itallie & Abraham, 1985).

FACTOR	ANDROID	GYNOID
Predominant gender	Males	Females
Regional fat storage	Upper body (neck, abdomen)	Lower body (thighs, buttocks)
Fat storage site	Internal	Subcutaneous
Characteristic of fat deposit	Hard	Soft
Adipose tissue receptors	Beta	Alpha
Adipose cell size	Large	Small
Fat mobilisation	Easy	Difficult
Major risk	Coronary artery disease, glucose intolerance, diabetes, hypertension	Psychological; self-efficacy

FIGURE 2.5: PATTERNS OF FAT DISTRIBUTION (Plowman & Smith, 1997)

2.8 BODY WEIGHT REGULATION

For land mammals, the ability to efficiently store food energy as fat provides a survival value when the food supply is scarce or sporadic (Neel, 1962). To maintain such food stores without undergoing continual alterations in size and shape, a system to maintain the balance between caloric intake and energy expenditure, is necessary. It is increasingly apparent that the body has a highly complex and sophisticated system for regulating energy balance and fat stores (Schwartz & Seeley, 1997).

Two major hypotheses have been proposed as to how the body maintains a steady body weight.

1. The setpoint hypothesis; and
2. The settling-point hypothesis.



2.8.1 The "Setpoint" Hypothesis

The setpoint hypothesis argues that all people, fat or thin, have a well-regulated internal control mechanism or "setpoint", probably located deep within the brain's lateral hypothalamus, that drives the body to maintain a particular level of body weight or body fat (Kessey, 1986). The mechanism by which the body measures its own fat stores has long been a mystery. Early evidence from parabiosis experiments, a technique whereby two animals are physically joined to one another such that they share some circulation, suggested the presence of a circulating factor that appeared to signal the amount of stored fat (Hervey, 1952; Coleman, 1973). Each time the level of body fat is reduced below a "natural" setpoint, the body makes internal adjustments to resist this change and conserve or replenish body fat. Even when a person attempts to gain weight above his or her normal level by overeating, the body resists this change by increasing resting metabolism (Hirsch & Leibel, 1970; Welle, 1986).

The discovery of the obese (*ob*) gene and its protein product in 1994 was a major breakthrough in the understanding of the systems regulating energy balance and clarified the nature of the circulating factor (Zhang et al., 1994). The *ob* gene is located in adipose tissue and encodes for a hormone called leptin (from the Greek *leptos*, meaning thin) which is secreted by adipocytes in proportion to the level of body adipose mass. In animals, the inability to produce leptin or respond to it, results in excessive food intake and inappropriately decreased energy expenditure - inducing profound obesity and insulin resistance. Administration of recombinant leptin to these animals reverses these changes and induces weight loss (Spiegelman & Flier, 1996).

Unlike mice with mutated *ob* genes, mutations in this gene have not been reported in humans. In humans, *ob* gene expression is prevalent, serum leptin concentrations are elevated, and the elevation of leptin correlates with the percentage of body fat. The mechanisms by which circulating levels of leptin signal the brain and trigger changes in food intake and energy expenditure have recently been the subject of considerable scientific and commercial interest. This interest has established the importance of neuropeptide Y (NP-Y) and a mela-nocortin receptor as a key component of the system that regulates energy balance and body weight (Erickson et al., 1996). NP-Y, a 36 amino-acid peptide first isolated 15 years ago from swine brains and one of the most abundant neuropeptides in mammalian brains, is a potent appetite stimulant. In

the hypothalamus, NP-Y profoundly affects energy balance by stimulating appetite and increasing energy expenditure (Tomaszyk et al., 1996). NP-Y is a central effector of leptin deficiency in mice and therefore appears to function as an important mediator in the response to the low levels of leptin, which occur during starvation (Erickson et al., 1996).

To summarize the setpoint hypothesis, a loss of body fat leads to a decrease in leptin production and low circulating leptin levels which, in turn, stimulates NP-Y in the hypothalamus. NP-Y interacts with its hypothalamic receptor and induces a cascade of events that includes increased food intake, decreased energy expenditure and reproductive function, decreased body temperature and increased parasympathomimetic activity. An increase in body fat results in an increase in the levels of circulating leptin, which induces melanocyte-stimulating hormone to interact with its receptor. This interaction leads to decreased food intake, increased energy expenditure, and increased sympathetic activity. The net results are a state of negative energy balance in which energy expenditure exceeds food intake (Friedman, 1996).

2.8.2 The "Settling-point" Hypothesis

The foregoing setpoint hypothesis has been criticised because, if body fat stores are centrally controlled the amount of fat in the diet should have little effect on body weight (Oeser, 1997). Proponents of the newer "settling-point" hypothesis propose that, like a thermostat, the adipostat can be reset by factors in the environment (Bennett, 1995). This hypothesis asserts that we maintain weight when our various metabolic feedback loops, governed by whatever susceptibility genes we carry, settle into an equilibrium with our environment (Oeser, 1997).

Factors that appear to reset the adipostat include:

- a) medication;
- b) composition and sensory properties of the diet; and
- c) habitual level of exercise.

High-fat diets liberally add calories to the body and exercise subtracts them. The ultimate influence of diet and exercise on the defended level of body fat appears not to result from this simple arithmetic. Sustained consumption of a diet high in fat, or regular exercise, has a tonic effect on the settling-point mechanism, shifting the defended level of body fat higher or lower (Oeser, 1997).

The precise areas within the brain and hypothalamus where these mediators interact to regulate eating behaviour are not completely understood. Appetite appears to be controlled by several areas in the hypothalamus, in part by a feeding centre in the ventrolateral nucleus of the hypothalamus (VLH) and a satiety centre in the ventromedial hypothalamus (VMH). The feeding centre signals the cerebral cortex, which stimulates eating. The satiety centre modulates this process by sending inhibitory signals to the feeding centre. Several factors may be involved in the activation of the satiety centre. Since the VMH possesses insulin receptors and is insulin sensitive, elevated blood glucose or insulin levels may activate the satiety centre (Olefsky, 1994; Levin & Routh, 1996).

Distension of the stomach following a meal may serve as another inhibitory factor and this may be mediated by the peptide cholecystokinin. In addition to NP-Y, the feeding and satiety centres are sensitive to mono-amines such as catecholamines and serotonin which modulate satiety, appetite, and energy expenditure (Olefsky 1994; Levin & Routh, 1996).

2.8.3 Energy Expenditure

The three main components of energy expenditure are the resting metabolic rate (RMR), exercise-induced thermogenesis and food-induced thermogenesis. Thermogenesis refers to the physiologic generation of heat and therefore the utilisation of energy. The RMR reflects the energy expended at rest by normal metabolic and organ functions and accounts for 60 to 75% of daily energy expenditure (Olefsky, 1994).

One well-documented change that occurs during weight loss through dieting is a dramatic and sustained reduction in resting metabolism (Eliot, 1989). This decrease is attributable to the loss of either body mass or fat-free body mass - and with severe caloric restriction, the resting metabolism may become depressed as much as 45%. This calorie-sparing response is independent of the persons weight status or past dieting history (Mc Ardle et al., 1996).

Metabolism becomes equally blunted in individuals attempting to lose weight, regardless of whether they have dieted before or whether they are fat or lean. This greatly conserves energy and causes the diet to become progressively less effective despite a surprisingly low caloric intake. Weight loss plateaus, and further weight loss is considerably less than predicted from the mathematics of the restricted energy intake (Mc Ardle et al., 1996).

The adrenergic system plays a major role in regulating energy expenditure. Beta-3 adrenoceptors are found in brown and white adipose tissue and appear to induce lipolysis and thermogenesis when activated by catecholamines (Insel, 1996).

- Brown adipose tissue primarily has a protective function and is distributed around the great vessels in the thorax and abdomen thus cushioning the vital organs against trauma. The organs however also oxidise brown adipose tissue, thus assisting the body in reducing excess fat stores.
- White adipose tissue, which includes the subcutaneous and visceral fat, is more abundant. White adipose tissue serves to store energy as fat and can be transformed by lipolysis to free fatty acids for use in skeletal muscle (Oeser, 1997).

2.9 WEIGHT CONTROL - CALORIC BALANCE EQUATION

At its most basic level weight control follows the first law of thermodynamics. This law synonymously known as the law of conservation of energy, states that energy can neither be created nor destroyed, but only changed in form (Plowman & Smith, 1997).

Theoretically, if the amount of energy taken in, is equivalent to the amount of energy expended, the body is in balance and weight remains stable. If an excess of energy is ingested, that energy is neither destroyed nor lost, but stored and weight (mass) is gained. If insufficient energy is ingested in relation to expenditure, the needed energy cannot be created but must be provided from storage sites, and weight (mass) is reduced (Plowman & Smith, 1997).

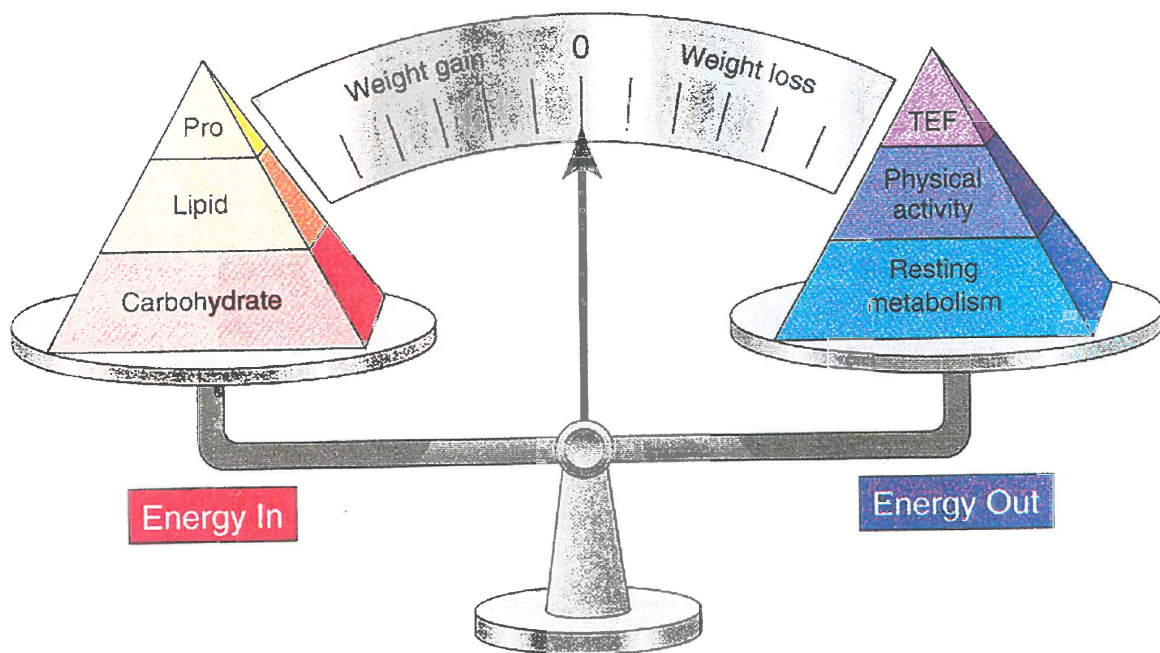


FIGURE 2.6: THE ENERGY BALANCE EQUATION

(TEF refers to the thermic effect of food)

(Mc Ardle et al., 1996)

Energy, in the form involved in the human body, is most frequently described in terms of kilocalories (kcal) or kilojoules (kJ). One kilocalorie is the amount of heat required to raise the temperature of 1 kg of water by 1 °C. One kilocalorie is equal to 4.186 kJ. A calorie is equal to 0.001 kcal. The term Calorie is often used generically however, as in the statement "Calorie intake should be equal to calorie output", even though the unit implies kilocalories (Plowman & Smith, 1997).

When considering the sensitivity of overall energy balance as exhibited in the energy balance equation, we note that if calorie intake exceeds output by only 100 Kcal per day, the surplus calories consumed in a year would be 365 days x 100 Kcal, or 36500 Kcal. Because 0,45 kg of body fat contains about 3500 Kcal this is equivalent to a yearly gain of about 4.7 kg of fat. If daily food intake is reduced by just 100 Kcal and energy expenditure is increased 100 Kcal by jogging 1.6 km each day, then the yearly calorie deficit is equivalent to about 9.5 kg of body fat (Mc Ardle et al., 1996).

The mathematical summation of calorie intake (+) and energy expenditure (-) from all sources, quantifies the law of conservation of energy. It describes the source of potential energy as food ingested and the various uses of that energy. The input and output can be partitioned into the following elements:

$$\begin{aligned} \text{Caloric balance} &= + \text{ food ingested (Kcal)} \\ &- \text{ basal or resting metabolic rate (Kcal)} \\ &- \text{ thermogenesis (Kcal)} \\ &- \text{ work or exercise metabolism (Kcal)} \\ &- \text{ energy excreted in waste products (Kcal)} \end{aligned}$$

(Plowman & Smith, 1997)

Food intake represents the only positive factor in the caloric balance equation. It is the only manner that energy can be added to the system. Energy is expended in three ways:

- Basal or resting metabolic rate;
- Thermogenesis; and
- Work or exercise.

These are the negative factors in the calorie balance equation.

Basal or resting metabolic rate accounts for the majority of the total energy expenditure, varying from approximately 60 to 75% in active and sedentary individuals, respectively.

Thermogenesis accounts for a relatively stable 10% in both sedentary and active individuals. The thermic effect of exercise is greater in active individuals, and depends on the intensity, duration and frequency of exercise (Poehlman, 1989).

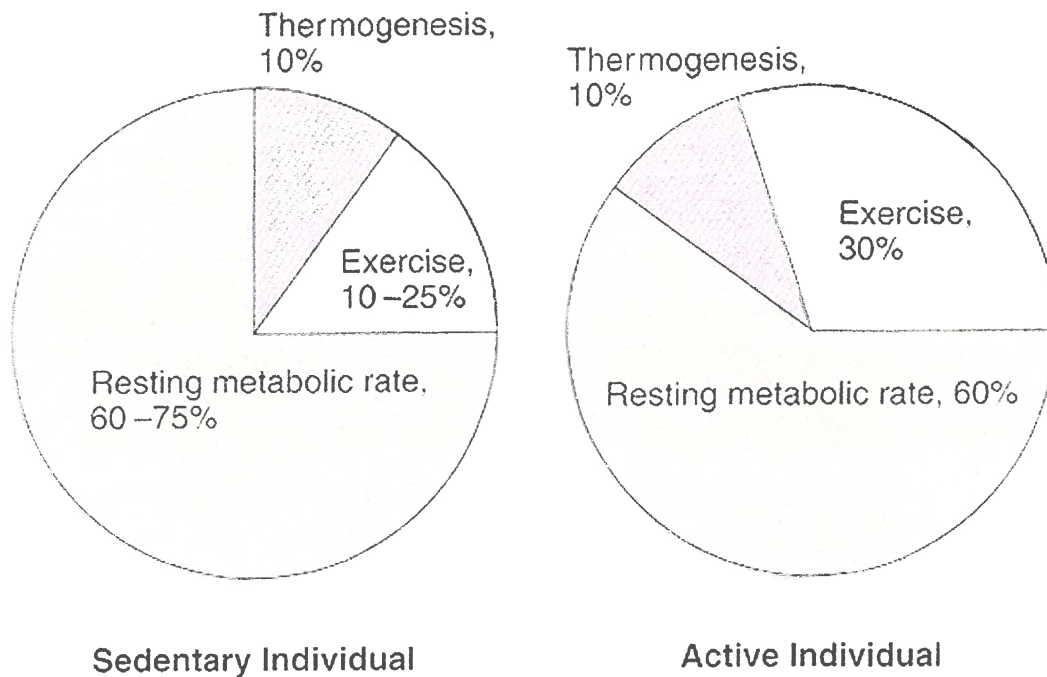


FIGURE 2.7: ENERGY EXPENDITURE
(Plowman & Smith, 1997)

If the amount of energy in food ingested exceeds the energy expended, the body is in a positive balance. If the amount of energy in the food ingested is less than the energy expended, the body is in a negative balance. The amount of energy excreted in waste products is insignificant and rarely measured (Plowman & Smith, 1997).

The arithmetic for fat accumulation is, however, overly simplistic because the diet's composition influences the efficiency of how the body converts and stores excess calories as body fat (Sims & Danforth, 1987). It is easier for the body to synthesise fat from dietary lipid than from equivalent caloric excess in the form of carbohydrate. Shifting the diet's composition toward higher carbohydrate would result in less body fat gain, should caloric excess occur (Mc Ardle et al., 1996).

A prudent dietary approach to weight loss, unbalances the energy balance equation by reducing daily energy intake 500 to 1000 kcal below daily energy expenditure. This moderate reduction in food intake produces a greater weight loss in relation to the energy deficit than a more severe energy restriction (Sweeney, 1993). People who create larger daily deficits to lose weight more rapidly are more likely to regain the weight than those who lose it slowly (Hovell, 1988). Short periods of caloric restriction are often encouraging to the dieter but result in large percentages of water and carbohydrate loss per unit weight loss, with only a small decrease in body fat (Mc Ardle et al., 1996).

A review of the scientific literature on weight loss reveals that the initial success in modifying body composition has little relation to long-term success. Participants who remain in supervised weight loss programs generally lose about 10% of their original body mass. Discouraging facts are that one to two thirds of the lost weight is regained within a year and almost all of it within five years (Technology Assessment Conference Panel, 1993; Begley, 1991).

2.9.1 Impact of Exercise on Food Intake

The relationship between exercise, appetite and energy intake is complex and difficult to discern. Appetite and the amount of food ingested are influenced by physiological, nutritional, behavioural and psychological factors in humans (Plowman & Smith, 1997). It is not just a matter of a physiological drive to balance energy demand and supply (Titchenal, 1988; Wilmore, 1983).

It is difficult to accurately measure food intake. Conducting studies on the effects of exercise on appetite in humans is also troublesome. Despite the difficulties the following generalisations can be drawn from the studies that are available.

1. Neither a reduction nor an increase in energy intake can be found immediately following a single bout of exercise.
2. Physically active males, females, adults and children consume more calories than sedentary individuals. Active individuals generally maintain their body weight and composition at or below normal levels.
3. Energy intake in both males and females generally increases or remains unchanged in response to exercise. Obese individuals most often do not change energy intake in response to exercise.
4. When chronic exercise ceases, energy intake in humans is spontaneously reduced. This reduction does not appear to match to the reduced energy expenditure. The result is a positive energy balance, a regain of lost body weight and an elevation of body fat.
(Plowman & Smith, 1997).

2.10 BASAL OR RESTING METABOLIC RATE

Basal metabolic rate (BMR) is defined as the level of energy required to sustain the body's vital functions in the waking state. This definition implies that the individual is resting quietly in a supine position, has not eaten for 8 - 18 hours, is at normal body temperature (37 °C) and neutral ambient temperature (27 - 29 °C) and is without psychological stress. To obtain truly basal conditions in a laboratory is difficult and resting metabolic rate is probably a more accurate descriptor. Resting metabolic rate (RMR) is defined as the energy expended while an individual is resting quietly in a supine position. The two terms are often used inter-changeably because the differences are so small (Bursztein et al., 1989).

The following organs and their functions are responsible for various portions of resting energy consumption:

- Liver > 29 - 32%
- Brain > 19 - 21%
- Muscle > 18%
- Heart > 10%
- Lungs > 9%
- Kidneys > 7%

(Plowman & Smith, 1997)

On the cellular level the energy is used to fuel ion pumps, synthesise and degrade cellular constituents, conduct electrical impulses and secrete various substances, including hormones (Bogert et al., 1973).

Basal or resting metabolic rate is usually related to body surface area and expressed in kcal.m² (Plowman & Smith, 1997). Obese individuals have large surface area and a larger cell mass (both fat and fat-free) than average-weight individuals. The resting metabolic rate (RMR) of obese individuals is higher than that of the normal weight individuals (Jequier, 1987). In addition, a genetic effect has been documented for resting metabolic rate (RMR). This genetic effect has considerable potential for predisposing an individual to gaining or losing fat over time (Bouchard, 1991).

2.10.1 Diet and Resting Metabolic Rate

The amount and the type of food ingested affects RMR. The effect of caloric restriction on RMR is well documented and clear-cut. Severe caloric restriction decreases RMR (Apfelbaum et al., 1969; Bray, 1969; Brownell et al., 1987). Metabolism represents the greatest percentage of daily caloric expenditure in sedentary individuals, and this results in a discouraging effect of slowing the weight loss that would be expected from the amount of dietary restriction and negative balance (Plowman & Smith, 1997).

2.10.2 Exercise and Resting Metabolic Rate

The energy cost of exercise, in oxygen or caloric units, includes a resting component and is quantified by the metabolic equivalent (MET), which expresses the energy cost of activity in multiples of the resting metabolic rate. One MET represents the average, seated resting energy cost of an adult and is set at $3.5 \text{ mL.kg}^{-1} \text{ min.}$ of oxygen or $1 \text{ kcal.kg}^{-1} \text{ .hr}^{-1}$. Metabolism is definitely elevated by exercise. Resting metabolism itself is not elevated but is assumed to remain constant. The increase in energy consumption is thus solely attributed to the activity demand and responses (Plowman & Smith, 1997).

Immediately after exercise the metabolic rate remains elevated. Historically, this period of elevated metabolism after exercise has been called the O_2 debt, the assumption being that the "extra" O_2 consumed during the "debt" period was being utilised to pay back the deficit incurred in the early part of exercise (Bahr, 1992; Stainssby & Barclay, 1970). Recently the term's O_2 recovery or excess postexercise oxygen consumption (EPOC) have come into favour. EPOC is defined as the oxygen consumption during recovery that is above normal resting values (Brooks & Fahey, 1984; Plowman & Smith, 1997; Mc Ardle et al., 1996).

Although there is no complete explanation of EPOC, seven factors have been suggested for the elevated post-exercise oxygen consumption, viz.:

- Resynthesize ATP and CP
- Resynthesize lactate to glycogen (Cori cycle)
- Oxidize lactate in energy metabolism
- Restore oxygen to blood
- Thermogenic effects of elevated core temperature
- Thermogenic effects of hormones, particularly the catecholamines-epinephrine and norepinephrine
- Effects of elevated heart rate, ventilation, and other elevated levels of physiologic function

(Mc Ardle et al., 1996; Bahr, 1992).

This recovery oxygen utilisation represents additional calories that are expended as a direct result of the response to exercise. The magnitude and duration of this elevated oxygen consumption will depend on the intensity of the preceding exercise. In the case of light submaximal work recovery takes place quickly and after heavy exercise recovery takes much longer (Bahr, 1992). If an individual expends 250-300 kcal walking or jogging for 3 minutes an additional 20-30 kcal may be expended during the hour or two after exercise until complete recovery is achieved. This expenditure does not mean that the resting metabolic rate itself has been affected (Plowman & Smith, 1997).

For it to be concluded that resting metabolism is changed by exercise, the change must be evident 24-48 hours after exercise. Research evidence for such a change is mixed and difficult to interpret (Plowman & Smith, 1997). It is unlikely that exercise causes any permanent change in resting metabolic rate, at least not light or moderate aerobic endurance exercise nor dynamic resistance activity (Horton, 1985; Bingham, et al., 1989; Melby, et al., 1993).

2.10.3 Weight Cycling and Resting Metabolic Rate

A special concern regarding the influence of diet on RMR is weight-cycling. Weight-cycling is defined as repeated bouts of weight loss and regain (Schelkun, 1991). Sometimes this cycling is called the rhythm method of girth control or the yo-yo effect (Plowman & Smith, 1997). Most dieters repeatedly lose weight and then gain weight again, despite their best intentions. Dieters may take months or years to complete each cycle (Schelkun, 1991).

It has been theorised that weight cycling slows down the RMR, increases the difficulty of subsequent weight loss, and enhances abdominal fat (Blackburn et al., 1989). Despite the theory, experimental evidence from studies testing the influence of weight cycling on RMR has presented inconclusive results (Plowman & Smith, 1997). Studies using dieters, whether initially obese or overweight, are also conflicting, with two or three studies finding no evidence that a history of weight cycling affected RMR (Beeson et al., 1989; Van Dale & Saris, 1989; Wadden et al., 1992).

2.11 THERMOGENESIS

Following the ingestion of any meal, energy metabolism is enhanced. This energy increase is due to the energy requiring processes of digestion, absorption, assimilation and synthesis of protein, fat and carbohydrate (Plowman & Smith, 1997). For any given meal the increased heat production as a result of food ingestion is referred to as the thermic effect of a meal (TEM). The energy expenditure associated with the ingestion of all food during a day is referred to as the thermic effect of feeding (TEF). TEF depicted as thermogenesis, constitutes approximately 10% of daily energy expenditure (Blanchard, 1982; Poehlman, 1989).

There is some evidence for a link between thermogenesis and the control of body weight. Precisely how thermogenesis occurs has not been determined, but a probable mechanism for the uncoupling of oxidative phosphorylation is involved (Plowman & Smith, 1997). This process may occur at specific steps in the metabolic pathways (known as substrate or futile cycling) or in brown adipose tissue (Himms-Hagen, 1984). Changes in the sodium-potassium pump activity have also been proposed (Blanchard, 1982).

Studies comparing the thermic response of lean and obese individuals to a test meal do indeed show a blunted TEM in the obese (Blanchard, 1982; Jequier, 1987; Newsholme, 1980; Schwartz, et al., 1983).

2.11.1 Impact of Diet on the Thermic Effect of a Meal

The total caloric content and the constituent composition of a meal have an impact on the thermic effect of the meal. The greatest thermic effect occurs with protein. Carbohydrate and fat show only about half the thermic increase shown by protein, with carbohydrate's increase being slightly higher than fat's. These differences would seem to indicate that a high-protein diet would be valuable for individuals wishing to expend extra calories. High-protein diets can exacerbate kidney and liver problems and may result in excessive losses of calcium. For these reasons diets exceeding 15% protein are not recommended, no matter what the thermic effect (Belko et al., 1986; Glickman et al., 1948; Nair et al., 1983; Pillet et al., 1974; Swaminathan et al., 1985).

If the percentage contributions of the macronutrients are kept constant to those values recommended for a healthy diet (55% carbohydrate, 30% fat, 15% protein), a direct relationship is found between the caloric content of a meal and TEM. Higher-caloric meals cause a greater thermic effect (Plowman & Smith, 1997). An individual on a restricted caloric diet would thus burn fewer calories through dietary-induced thermogenesis than when eating larger meals (Belko et al., 1986).

2.11.2 Impact of Exercise on the Thermic Effect of a Meal

Both meal ingestion and exercise stimulate the sympathetic nervous system and thermogenesis. Since many people are interested in maximising energy expenditure, it is deemed of interest to determine whether a combination of exercise plus a meal in close temporal proximity would potentiate the singular effect of either exercise or food intake. A number of studies have been completed following two basic sequences. After a period of rest the subjects consumed a meal, and then exercised. Conversely, after a period of rest the subjects exercised and then consumed a meal. In both sequences some studies have shown that TEM was enhanced due to exercise (Belko et al., 1986; Zahorska-Markiewicz, 1980) while others have shown that TEM was not enhanced due to exercise (Dallasso & James, 1984; Welle, 1984).

2.12 TREATMENT OF OBESITY

Diet and exercise are the most frequently cited methods for both men and woman attempting to lose weight (Miller & Lindeman, 1997). Many forms of therapy are used and promoted including countless fad diets, herbal remedies, acupuncture, accupressure, appetite suppressing, "aroma sticks", medication, surgery and more. The treatment of obesity is a thriving industry and Americans spend over 30 billion dollars yearly in weight loss efforts (Technology Assessment Conference Panel, 1993). The results of longer-term medication studies have spawned a myriad of profit orientated prescription weight-loss clinics (Oeser, 1997).

The ultimate measure of success of a weight-loss program is the ability of the program to help the individual maintain a stable weight or a reduced weight and ultimately to improve health (Miller & Lindeman, 1997). Even in highly structured, medically supervised programs, the dropout rate is high and maximum weight loss rarely exceeds 10% of the initial body weight for those who complete the program (Oeser, 1997). If eating patterns and activity profiles are not permanently altered, most people regain the lost weight over the next one to five years (Miller & Lindeman, 1997). Numerous methods of weight-loss exist where the objective is short-term, rapid or unsupervised weight loss, and/or rely on dietary aids such as drinks, pre-packaged foods, or diet pills. Such efforts do not include education and guidance in the transition to a permanent pattern of healthy eating and activity, and have never been shown to lead to long-term success (Oeser, 1997).

It seems that although obese individuals may have different therapeutic objectives e.g. to reduce disease risk, to ameliorate disease symptomatology, to build self-esteem and to increase functional capacity, the immediate measurable outcome variable of body weight becomes the focus of intervention (Miller & Lindeman, 1997). Regardless of how much weight a person would like to lose, modest goals and a slow course will maximise the probability of losing and maintaining weight (Oeser, 1997). It should be recognised that for most people, achieving a body weight or figure like those often depicted by the media is not a reasonable, appropriate, or achievable goal. Therefore, failure to achieve this "look" does not imply a weakness of will power or character (Technology Assessment Conference Panel, 1993).

As with the treatment of any chronic disease, therapy for obesity may lead to adverse effects. Adverse effects associated with weight loss treatment include poor nutrition, possible development of eating disorders, weight cycling and psychological consequences of repeated failings to lose weight. Medical supervision of weight loss is strongly recommended for severely obese persons, pregnant or lactating women, children, persons over the age of 65 years and those with serious medical conditions (Oeser, 1997).

The five recognised treatment modalities available are diet modification, exercise, behaviour modification, medication therapy and surgery. All of these modalities, alone or in combination are capable of inducing weight loss sufficient to produce significant health benefits in many obese persons. Health benefits are not maintained if weight is regained and, with the exemption of surgery, it is difficult to adhere to these modalities in a manner sufficient to maintain long-term weight loss (Oeser, 1997).

2.12.1 Dieting as a Weight-loss Strategy

Caloric restriction has remained as the mainstay for the treatment of obesity (Oeser, 1997). During the late 1950's and early 1960's total fasting was used to reduced body weight quickly in the massively obese (Miller, 1999). Although the desired outcome of rapid weight loss was achieved through fasting, serious medical conditions such as loss of lean body mass (LBM), depleted electrolytes and death, caused total fasting to decrease in popularity (American Dietetic Association, 1990; Atkinson, 1986).

By the late 1960's and early 1970's the focus shifted to high-protein, low-carbohydrate diets. Popular diets of the time were the Atkins and Stillman diets, which provided about 2100 kcal and 1300 kcal daily, respectively (Fisher & Lachance, 1985). More noteworthy than the energy content of these diets was that they were characterised by their low carbohydrate content (5-10% of energy) and relatively high fat content (50-70% of energy). The theory behind this type of diet composition was that the high protein content would prevent muscle catabolism, while the low carbohydrate content would keep the body in a ketogenic state, which helps suppress appetite. The carbohydrate restriction caused rapid weight loss because of depleted glycogen stores and diuresis, but side effects included nausea, hyperuricemia, fatigue and feeding edema (Zeman, 1991; Miller, 1999).

In the mid 1970's, very low-caloric liquid diets became available. These diets were also known as protein-sparing, modified fasts or liquid-protein diets. Their extremely low energy content caused rapid weight loss. However, in spite of medical supervision, high quality protein, and potassium supplementation, deaths caused by ventricular arrhythmia occurred. Ironically, one of the most popular of these diets was called the "last chance diet" (Ficher & Lachance, 1985).

The 1980's spawned the second generation of very low caloric diets (VLCD). These new commercial formula products became part of medically supervised programs that included patient support and counselling for weight maintenance after initial weight loss (Miller, 1999). These new VLCD were compositionally different from the low-carbohydrate diets of the 1960's and 1970's in that the fat content of the new VLCD was very low (2-18% of energy). Health risks associated with the VLCD, however, were gall-bladder disease and cardiac problems (Fisher, 1992; Gallagher & Heymsfield, 1994; Garner, 1991).

The 1980's also brought about pre-packaged low-calorie diets from franchises such as Nutri-System and Body Balance. Composition of these pre-packaged diets, by energy value, was 20% protein, 20% fat and 60% carbohydrate (Miller, 1999). The fact that meals were pre-packaged and the daily energy intake was 1100-1200 kcal inspired better program compliance than that seen with the VLCD. However, the same health problems that were present with the VLCD soon became apparent with these pre-packaged low-calorie diets (Berg, 1995).

For the past two decades, fat-free diets and fat-free versions of food have become increasingly popular. Several low-fat diet books have become best sellers, viz.: *Fit for Life* (Diamond & Diamond, 1987) and *Eat more Weigh less* (Ornish, 1994). Only about 10-15% of the 1200-1700 kcal in these diets typically comes from fat (Miller, 1999; Katahn, 1989). The singular focus of reducing fat in the diet may have backfired, however, as data from the latest National Health and Nutrition Examination Survey indicate that while the percentage of fat in the diet has decreased, total energy intake has increased (Centre for Disease Control and Prevention, 1994).

Miller et al., (1994) have shown that obese men and women consume a greater percentage of their sugar energy from refined or added sources when compared with their lean counterparts. These researchers have also shown that the obese consume less dietary fibre than the lean (Miller et al., 1994).

A review of the VLCD programs suggest that 12 - 16 weeks of dieting produces a 20 kg weight loss, of which a 10 - 13 kg loss can be maintained after one year (Wadden, 1993). Individual reports vary as to their success claims, and it is difficult to interpret the results because dropout rates can be as high as 80% in some VLCD programs (Technology Assessment Conference Panel, 1993).

The conventional 1200 kcal diet will produce a weight loss of 8.5 kg in 20 weeks and 66% of this can be maintained after one year (Wadden, 1993). However, as time progresses, weight is regained until pre-diet weights are reached within five years (Brownell & Jeffery, 1987).

2.12.2 Behaviour Modification in Weight Control

Behaviour modification programs focusing on reducing dietary fat and sugar have induced weight losses of 7 - 9 kg in six months, but these programs have not yet reported any long-term data (Miller et al., 1993).

Other programs restricting dietary fat and/or focusing on behaviour modification have reported conflicting results for weight-loss maintenance and are generally no more effective than traditional dieting techniques (Willett, 1998).

Behaviour modification for obesity refers to a set of principles and techniques designed to modify eating habits and physical activity and appear to be most helpful for middle to moderately obese persons. Many of the recent and better-designed studies evaluating the efficiency of appetite-suppressants have combined medication therapy with behavioural approaches to improve diet and exercise (Oeser, 1997).



In most programs, therapy typically consists of weekly, hour-long sessions in a group format. The average length of treatment is 18 weeks although long-term programs lasting for six months or more are increasingly common. Behaviour modification, in conjunction with diet and exercise, induces an average weight loss of about 10 kg and about 68% of this weight loss is maintained after 52 weeks (Foreyt & Goodrick, 1993).

Examples of principles and techniques used include self-observation and self-recording which are the mainstays of behavioural modification programs. The patient records the situational factors, behaviours, thoughts, moods and feelings that occur before, during and after attempts at prudent eating and exercising (Oeser, 1997). Stimulus control involves modifying environmental factors that lead to inappropriate eating or exercise. Examples of this modality include keeping away from high-fat foods, eating at specific times and places, and setting aside a time and place to exercise (Foreyt & Goodrick, 1993). Contingency management bestows rewards or prizes for appropriate eating and exercise behaviour that leads to weight loss or maintenance of weight loss. Cognitive behavioural modification focuses on strategies to counter the thoughts, moods, diets, and social pressures to be lean (Foreyt & Goodrick, 1993).

2.12.3 Surgery in Weight Control

Surgery is considered the treatment of choice for well informed and motivated severely obese adults (more than 100% overweight or BMI greater than 40) who fail to respond to medical weight control. Surgery may also be considered for those with less severe obesity (BMI between 35 and 40) afflicted with disabling joint disease, pulmonary insufficiency, and hypertension or diabetes mellitus. Surgery is not yet recommended for severely obese children or adolescents because this population has not been adequately studied and experience is therefore limited (Benotti & Forse, 1995).

The operations recognised by the 1991 National Institute of Health (NIH) Consensus Conference are the vertical banded gastroplasty and the Roux-Y gastric bypass procedure (Consensus Development Conference Panel, 1991). The vertical banded gastroplasty restricts food intake by limiting gastric volume whereas the gastric bypass operation not only limits gastric volume but also results in stomach contents bypassing the distal stomach duodenum and proximal jejunum. Although some malabsorption of nutrients occurs, most of the weight loss is attributed to delayed gastric emptying and a feeling of satiety that causes patients to limit food intake. Both of these procedures can be reversed at a later date if required (Oeser, 1997).

The effects of surgery on co-morbid conditions are impressive. There is marked improvement in glucose tolerance often with amelioration of type II diabetes, allowing patients to reduce or discontinue insulin use. Weight-reduction surgery ameliorates hypertension as well as sleep apnea and obesity related hyperventilation. Lipid abnormalities improve with a decline in serum cholesterol and triglycerides and an increase in high-density lipoprotein (HDL) cholesterol. Improvement in musculoskeletal disability, which nearly always accompanies surgical weight control, results in less arthritic pain and improved mobility.

Surgery is also associated with improvements in psychosocial functioning, employment rate, annual income, and some quality-of-life measures (Stunkard et al., 1986). Most importantly, there is data to suggest that life expectancy may be increased (Mason, 1989).

2.12.4 Effectiveness of Exercise in Weight Control

Although exercise has been prescribed for decades as an adjunct to diet in traditional obesity intervention, exercise science did not enter the field of obesity research until the 1960's (Miller, 1999). Like calorie restriction, exercise is a fundamental, albeit under-utilised modality for weight control (Oeser, 1997). Although many overweight persons are trying to lose weight, only a small fraction of these are engaging in the recommended amount of physical activity. The addition of an exercise program to diet modification results in more weight loss than dieting alone and seems to be especially helpful in maintaining weight loss and preserving lean body mass (Blair, 1993).

Although exercise prescription for weight control varies, the literature reveals that exercise effects on body weight are rather small, but significant. Weight loss of about 2.0 kg has been reported for various exercise programs of differing duration (King & Tribble, 1991). A recent meta-analytical review reported that exercise causes body weight to decrease at a rate of about $0.2 \text{ kg}\cdot\text{wk}^{-1}$ and that people do not lose as much weight as would be expected from the prescribed exercise (Miller et al., 1997). Data indicate that the effectiveness of exercise for weight loss is directly related to the initial degree of adiposity and the total number of kilocalories expended (Ballor & Keeseey, 1991).

The long-term effects of exercise in weight control seem to be most promising, but the follow-up data for exercise intervention are scanty for the first couple of years post intervention and non-existent after five years (Miller, 1999). One of the longer-term exercise studies that is often cited in literature compared body weight changes of police officers participating in an 8 week diet or diet plus exercise program consisting of 35 - 60 min. of aerobic activity, callisthenics and relaxation techniques for three days per week (Pavlou et al., 1989). Those who did not exercise during the follow-up period gained about 60% of their weight back by six months post treatment and gained 92% back by 18 months post treatment. There were no significant gains in body weight at 18 months post intervention for those who exercised through the follow-up period. A meta-analysis of the past 25 years of exercise research also suggests that exercise is critical to weight-loss maintenance (Miller et al., 1993). Weight loss during the average 21-week exercise program reviewed in this meta-analysis was only $2.9 \pm 0.4 \text{ kg}$, but at one-year follow-up the net weight loss had increased to $6.1 \pm 2.1 \text{ kg}$. On the other hand, weight loss in the average 13-week diet-plus-exercise program amounted to $11.0 \pm 0.6 \text{ kg}$ with a 22% regain in weight after the first year (Miller et al., 1993).

2.12.5 Effectiveness of Continuous Assistive-Passive Exercise in Weight Control

Continuous assistive-passive exercise (CAPE) tables are a recent addition to the exercise equipment market. Continuous assistive-passive exercise regimens usually consist of a set of six tables which provide a combination of passive, continuously assisted, and light resistive exercise to the trunk and extremities. This form of

exercise is claimed to reduce girths at specific body sites, such as the legs, hips, waist and upper arms, without strenuous exercises (CAPE promotional literature). This exercise modality has great appeal to many sedentary, older subjects because it promises some of the benefits of vigorous exercise without strenuous efforts. However, to the author's knowledge, there is only one published report examining the efficiency of this exercise modality (Martin & Kauwell, 1990).

While the physiological rationale for the use of CAPE training may be questioned by clinicians and researchers familiar with exercise training, many consumers are using this exercise modality (Martin & Kauwell, 1990).

CHAPTER 3

METHODS AND PROCEDURES

3.1 SUBJECTS

A group of 48 females between the ages of 25 - 35 years (mean age = 29.9 ± 2.6 years), who were recruited through newspaper advertisements, served as subjects. In order to be eligible for inclusion into the study, subjects were required to be physically suitable for a programme of continuous assistive-passive exercise (CAPE); pre-menopausal; obese (>30% fat); sedentary (< one 20 minute bout of aerobic or strength training per week over the previous six months); and amenable to being assigned to any of three study groups.

The following specific exclusion criteria were applied:

- a) a history of orthopaedic, cardiovascular, pulmonary or metabolic disease - which could have contra-indicated exercise testing;
- b) a hysterectomy - to avoid changes in oestrogen level;
- c) a prevailing pregnancy;
- d) glandular malfunctions - to avoid the influence of changes in normal hormonal levels;
- e) diabetes - since such subjects could not follow the diet as prescribed;
- f) vegetarianism and the presence of specific food allergies; and
- g) medication usage.

Subjects gave their informed consent (Appendix A) prior to participating and took cognisance the compliant importance of not engaging in any exercise in addition to that required over the duration of the study. During the course of the investigation five subjects withdrew - two because of medical and three due to personal reasons.

3.2 STUDY DESIGN

To recapitulate, the primary aim of the study was to evaluate the effect of an eight-week program of continuous assistive-passive exercise (CAPE) performed on tables in conjunction with and without a specific dietary intervention. In order to achieve this goal a pretest - posttest experimental groups design, with three levels of the independent variable, was adopted for the study. Subjects were randomly assigned to one of the following three groups:

- Group PED (N = 15) - Continuous assistive-passive exercise (CAPE) on tables and following a specific diet.
- Group PE (N = 14) - Continuous assistive-passive exercise (CAPE) on tables and following no specific diet.
- Group D (N = 14) - Following the specific diet but no continuous assistive-passive exercise (CAPE) on tables.

In order to enhance compliance and to minimise the dropout rate, personal follow-up phone calls were made randomly and a weekly weighing and motivation session was conducted on every Wednesday evening over the duration of the study.

Table I: Subject Characteristics

GROUPS		PE (N = 14)		PED (N = 15)		D (N = 14)	
VARIABLE	UNITS	PRE (Mean)	Std. Dev.	PRE (Mean)	Std. Dev.	PRE (Mean)	Std. Dev.
Age	years	29.57	± 2.8	29.93	± 2.6	30.36	± 2.4
Stature	cm	162.71	± 7.0	163.65	± 5.6	166.30	± 6.6
Body Mass	kg	74.38	± 10.1	75.85	± 7.0	74.24	± 7.5
BMI	kg/m ²	28.15	± 4.1	28.58	± 2.0	26.86	± 2.8
LBM	kg	44.65	± 5.3	45.92	± 3.3	45.26	± 3.8

PE = Continuous assistive-passive exercise (CAPE) on tables. No specific diet.

PED = Continuous assistive-passive exercise (CAPE) on tables. Specific diet.

D = Specific diet.

Table I summarises the subject characteristics of the respective experimental groups. No significant differences ($p > 0,05$) were found between the specific variables of each group, thus reflecting the homogenous nature of each group, compiled by random assignment.

3.3 MEASUREMENTS

The following dependent variables were measured:

- Body composition
 - Percentage body fat (% BF)
 - Lean body mass (LBM)
 - Body mass index (BMI)

- Morphology
 - Stature
 - Body mass
 - Skeletal widths
 - Skinfolds
 - Girth measures

- Physical working capacity (PWC)
- Maximal oxygen uptake (VO_2 max)
- Pulmonary function
- Haematology
- Flexibility
- Abdominal muscle endurance

3.3.1 Body Composition

3.3.1.1 Percentage Body Fat

The skinfold procedure of Durnin and Womersley (1974) was employed to estimate percentage body fat (% BF). Accordingly, the sum of four skinfolds (biceps, triceps, sub-scapula and supra-iliac) was converted to a common logarithmic value (L) and substituted in the following regression formula (Durnin & Rahaman, 1967) to obtain a predicated body density (D) for adult women: $D = 1,1581 - (0,072 * L)$. Relative body fat was then calculated from the Siri (1956) formula for converting body density to body fat, viz.: $\% \text{ body fat} = (4,950/D - 4,5) * 100$.

3.3.1.2 Lean Body Mass

Lean body mass (LBM) as a derived anthropometric variable of body composition was calculated as follows:

$$\text{LBM} = \text{BM} - \text{ABF} \quad \text{and} \quad \text{ABF} = \frac{\text{RBF} \times \text{BM}}{100}$$

where: LBM = lean body mass (kg)
 BM = measured body mass (kg)
 ABF = predicted absolute body fat (kg)
 RBF = predicted body fat (%)

3.3.1.3 Body Mass Index

Body mass index (BMI) was used as an additional practical measure of obesity defined as BMI > 30 (Bouchard & Blair, 1999). The BMI was obtained by dividing the subject's mass in kilograms by stature measured in metres, squared:

$$\text{BMI} = \frac{\text{Mass (kg)}}{\text{Stature}^2(\text{m})}$$

3.3.2 **Anthropometry**

All variables, unless stated otherwise, were measured according to the procedures of the anthropometric standardization manual of Lohman et al. (1988).

3.3.2.1 Stature

Stature is a major indicator of general body size and of bone length. It is an important variable in screening for disease or malnutrition and in the interpretation of body weight (Lohman et al., 1988).

The stature was measured with a calibrated height gauge. The subject stood barefoot, feet together and heels, buttocks and upper part of the back touching the scale with head placed in the Frankfort plane, not necessarily touching the gauge. The Frankfort plane was considered as the orbital (lower edge of the eye socket) being in the same horizontal plane as the tragion (notch superior to the tragus of the ear). When so

aligned the vertex was the highest point on the skull. The measurement was taken to the nearest 0,1 cm at the end of a deep inhalation.

3.3.2.2 Body Mass

Body mass was measured with a Detecto beam balance scale to the nearest 0,1 kg, with the subject clothed only in a swimming costume, and taking care that the:

- scale was reading zero;
- subject stood on the centre of the scale without support;
- subject's weight distribution was even on both feet; and
- subject's head was held up and the eyes looked directly ahead.

3.3.2.3 Skeletal Widths

Skeletal width measurements can be used for several research and clinical purposes, such as in the determination of body types according to the Health-Carter somatotyping technique (Lohman et al., 1988; Heath & Carter, 1976).

Skeletal width sites are typically defined by bony landmarks. It is important to select landmarks that are palpable not only in lean but also in other individuals.

A steel spreading calliper was used to measure the bi-epicondyle breadth of the humerus and the bi-condyle breadth of the femur in cm to the nearest mm.

To measure elbow width (condyle breadth of the humerus) the subject raised the right arm to the horizontal and the elbow was flexed to 90°. The dorsum of the subject's hand faced the measurer. The measurer stood in front of the subject and palpated the lateral and medial epicondyles of the humerus. The calliper blades were then placed on these points.

To measure knee width (condyle breadth of the femur) the subject's knee was flexed to 90° while sitting. The measurer stood facing the subject. The most lateral aspect of the lateral femoral condyle was palpated with the index or middle finger of the left hand while the corresponding fingers of the right hand palpated the most lateral aspect of the medial epicondyle. The calliper blades were then placed on these points.

3.3.2.4 Skinfolds

Skinfolds were taken using a John Bull skinfold calliper exerting a uniform pressure of 10 g per mm² irrespective of the calliper opening. The following skinfolds were taken (all skinfolds were measured on the right of the body): triceps, sub-scapula supra-iliac, biceps and medial-calf.

The skinfold sites were carefully located using the following anatomical landmarks:

- Biceps:** The anterior surface of the biceps midway between the anterior auxiliary fold and the antecubital fossa.
- Triceps:** A vertical fold on the posterior midline of the upper arm, over the triceps muscle, halfway between the acromion process and olecranon process. The elbow was extended and the arm relaxed.
- Sub-scapula:** The skinfold was taken 2 cm along a line running laterally and obliquely downwards from the inferior angle of the scapula at an angle (approximately 45°) as determined by the natural cleavage line of the skin.
- Supra-iliac:** A diagonal fold was taken above the crest of the ilium at the spot where an imaginary line would descend from the anterior auxiliary line (just above and 2-3 cm anterior of the iliac crest).
- Medial-calf:** The subjects were seated (knees at 90°) and with the calf relaxed a vertical fold was raised on the medial aspect of the calf at the level of maximal circumference.

Two measurements were taken two seconds after the full pressure of the callipers had been applied, and recorded to the nearest 0,5 mm. If the difference was greater than 1 mm, then a third measure was taken and the mean of the closest two recorded.

3.3.2.5 Girth Measures

A Rabone-Chesterman calibrated steel tape and the cross hand technique was used for measuring all 12 girths. The reading was taken in cm to the nearest 0,1 mm from the tape where, for easier viewing, the zero was located more lateral than medial on the subject.

Constant tension on the tape was maintained but ensuring that there was no indentation of the skin while the tape was held place at the designated landmark. When reading the tape the measurer's eyes remained at the same level as the tape to avoid any error of parallax. Care was taken to ensure that the tape remained horizontal to the floor during measurement. The twelve sites measured were:

1. Ankle - at the level of the lateral malleolus.
2. Calf - at the point of maximum circumference.
3. Mid-thigh - midway between the distance from the superior margin of the patella to the anterior superior iliac spine.
4. Relaxed upper-arm - midway between the distance from the olecranon to the posterior aspect of the acromion, with the elbow extended and palm facing medially.
5. Contracted upper-arm - at the point of maximum circumference.
6. Forearm - at the point of maximum circumference.
7. Wrist - just distal to the styloid processes of the radius and ulna.

8. Chest - at the level of fourth costo-sternal joints. Laterally, this corresponds to the level of the sixth rib. Measurements were made at the end of a normal expiration.
9. Inflated chest - level of the sixth ribs during a maximum inspiration.
10. Deflated chest - level of the sixth ribs during a maximum expiration.
11. Waist - at the narrowest part of the torso.
12. Abdominal - the tape was placed around the subject at the level of the greatest anterior distension of the abdomen in a horizontal plane, not necessarily corresponding with the level of the umbilicus. The measurement was made at the end of a normal expiration.

3.3.3 Physical Work Capacity and Maximal Oxygen Uptake

To determine the subjects functional responses and limitations to exercise, a physical working capacity test (PWC¹⁷⁰), a three-lead electrocardiogram (ECG) and indirect maximal oxygen consumption (VO₂ max.) test was conducted.

The exercise test protocol was applied in such a manner as to monitor and enable the following:

1. heart rate, blood pressure and breathing response to exercise;
2. signs and symptoms of exertional distress;
3. document undue anxieties;
4. allow individuals to regain lost confidence;
5. teach principles of pulse-rate monitoring;
6. obtain data for exercise prescription and program development.

As the subjects were obese a Monark 824E Ergometric cycle ergometer was utilized to make the test non-weight bearing, thus reducing the impact on tendons, joints and ligaments. The size of the seat, the seat height and the distance between the seat and the handlebars were adjusted appropriately, such that subjects sat comfortably and their movement did not cause any interference with the ECG leads.

3.3.3.1 Testing Protocol

The initial resistance remained constant at 50 revolutions per minute at 50 watt throughout. A blood pressure and ECG readout were taken at rest, before the onset of exercise and again after 5 minutes of exercise. From the response observed a decision was made on whether to increase the load or terminate the test. Subjects were not tested above 80% of their maximum heart rate as obtained from the equation, 220 minus subject's age (Wasserman et al., 1994). If heart rate and blood pressure responses permitted, the resistance was increased to 75 watt after 5 minutes and to 100 watt after 10 minutes. The PWC was determined as the amount of work done at a heart rate of 170 beats per minute:

$$\text{PWC}^{170} = \frac{\text{Highest resistance kg.m/min.}}{\text{Highest HR - Lowest HR}} \times (170 - \text{Lowest HR})$$

VO₂ max. was determined according to the prediction nomogram of Astrand and Rhyning (Astrand, 1952) relating the linear relationship between the oxygen intake and heart rate levels.

3.3.4 Pulmonary Function

Lung volume and lung function was determined by a Vicatest - P2 flowmeter with cognisance of the following variables: environmental temperature, the subject's age, stature, body mass and gender.

The procedure was replicated for each subject: The nose was closed off by a noseclamp and the subject's bit onto a mouthpiece. Subjects were asked to inhale as deeply as possible, and then exhale explosively and as deeply, quickly and forcefully



as possible until the lungs were empty, followed by a second inhalation. Two trails were taken and the best result was recorded.

The following parameters were used:

- FVC - Forced vital capacity indicating lung volume expressed in litres.
- FEV₁ - Forced expiratory volume during the 1st second of FVC.
- FEV₁ % - FEV₁/FVC x 100 % - indicating breathing efficiency.
- PEF - Peak expiratory flow - evaluating the effectiveness of the respiratory and abdominal muscles.
- MEF 50% - Maximum expiratory flow when 50% remained to be expired - indicating the bronchial flow.
- MEF 25% - Maximum expiratory flow when 25% remained to be expired - indicating the flow in the bronchial tubes.

3.3.5 Haematology

A professional pathology laboratory (Dr's Du Buisson and Partners) performed the blood analysis. All chemistry analyses were done using the Beckman Synchron CX system. Cholesterol reagent was used to measure lipid concentration by a timed-endpoint method (Tietz, 1994).

The following reference ranges were utilized:

Cholesterol	:	3.0 - 5.2 mmol/l
High-density lipoprotein	:	0.9 - 1.6 mmol/l
Low-density lipoprotein	:	2.0 - 3.4 mmol/l
Glucose	:	3.5 - 6.0 mmol/l

(Tietz, 1994)

3.3.6 Flexibility

3.3.6.1 Hip Flexion

The sit-and-reach test (Marrow et al., 1995) was used to determine hip flexion (flexibility of the hamstrings and lower back). The subjects were asked to remove their shoes, and sit at the test apparatus with knees fully extended. The heels were placed shoulder width apart, flat against the box. Arms were then extended forward, with the subject leaning forward and extending the fingertips along the ruler as far as possible. Two trials were taken and the best result was recorded. Measurements were taken in centimetres (cm).

3.3.6.2 Lateral Trunk Flexion

The test was designed to determine the subject's lateral trunk flexion. The subject stood straight with back, buttocks and both heels touching an upright wall. The subjects were instructed to bend laterally while keeping their body in contact with the wall. The distance was then measured from the third phalange (middle finger) tip to the floor. The test was done barefoot. Two trials were taken and the best result was recorded. Measurements were taken in centimetres (cm).

3.3.7 Abdominal Muscle Endurance

Abdominal muscle endurance was evaluated with sit-ups performed with knees bent and feet fixed. The hands were required to touch the ears and elbows to touch the knees at the end of the curl up. The subject also had to descend in a controlled manner. The tester's hand was placed palm-side up on the bench such that the wrist made contact with the spine in line with the inferior border of the scapulae.

If the hands were removed off the ears, the elbows did not touch the knees or the back did not touch the testers hand, the sit-up was not counted. The maximum number performed in one minute was recorded. Subjects were permitted to rest within the one-minute period and then restart.

3.4 INTERVENTION PROGRAM

3.1.1 Continuous Assistive - Passive Exercise Training

Training on the six continuous assistive-passive exercise (CAPE) tables was supervised and conducted for 8 weeks according to the manufacturer's (Slenderline) instructions. Each subject used the tables three times per week for 10 minutes for a total duration of 60 minutes per session. The training frequency was adopted according to the manufacturer's recommendation of at least three training sessions per week for maximum results. The tables were available for use on a Monday, Wednesday and Friday from two o'clock in the afternoon until eight o'clock at night. The CAPE group was not given a training target heart rate since the manufacturer did not make any recommendations regarding heart rate during CAPE training and since the table design is such that speed of movements are pre-set on manufacture.

The various exercise tables and exercises are described and illustrated below in figures 3.1 - 3.6.

- This table exercised the hips and low back areas. The subject lay supine on the table with legs extended onto the hinged oscillating leg pads. The leg pads alternated between flexed and extended positions. The subject alternately contracted the hip flexors and extensors in synchronisation with the table.



FIGURE 3.1: WAIST, TUMMY AND HIP TABLE

- This table exercised the subject's legs, torso and arms. The subject lay on the table in a supine position with a 5 kg sand bag over the ankles. The pad under the back oscillated back and forth while the subject mildly resisted with her arms and legs.



FIGURE 3.2: STRETCH TABLE

- This table exercised the legs as the subject lay in a supine position with her feet in stirrups. The stirrups moved in an outward circular direction, and the subject lightly resisted the movement with her legs.



FIGURE 3.3: LEG TABLE

- The subject lay supine on the table, and the lower half of the table alternately raised and lowered. The subject contracted the abdominal muscles and dorsiflexed the ankle during the upward motion.

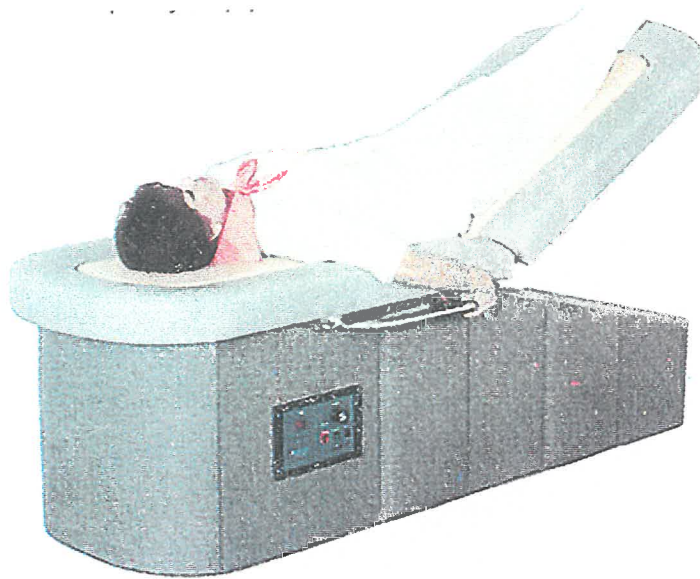


FIGURE 3.4: LEG LIFT TABLE

- On this table, the subject lay supine with her hips over an oscillating pad. A 2 kg sand bag was placed over the abdomen, and the subject pushed against the lower portion of the table with her feet. This table was designed to exercise the abdomen, hips and legs.



FIGURE 3.5: SAND BAG TABLE

- The circulation table was used as the final exercise and was designed to relax the subject following the workout.



FIGURE 3.6: CIRCULATION TABLE

3.4.2 Diet Program

The dietary intervention program, known as the Metabolism Diet, emphasised the maintenance of a normal diet, including appropriate amounts of carbohydrates, protein and fat, viz.:

- Carbohydrates 55 percent
- Protein 15 percent
- Fats 30 percent

The complete daily Metabo-Meal Plan is included in Appendix B.

In general the subjects consumed normal everyday food, eating different amounts of food (calories) in three phases.

- Low calorie phase

At 1000 calories per day, this phase was designed for maximum weight loss, while subjects consumed a nutritionally balanced diet. This menu was followed for two weeks.

- Booster phase

After two weeks on the low calorie phase the subjects were required to switch to the booster menu plan with 300 more calories. This phase was designed to boost the metabolic rate. The added calories during the booster phase were made up by carbohydrates.

- Re-entry phase

When subjects got to within two to three kg from their target goal-weight, they switched to the re-entry phase. Unless subjects gradually increased their calories they had the risk of gaining weight. This pre-maintenance period served to get the subject's metabolism ready for normal eating.

In all phases subjects ate four meals per day, breakfast, lunch, dinner and a metabo-meal which was similar to a late night supper. By taking more frequent meals, the subjects were able to avoid the feeling of hunger and fatigue often associated with a diet. Subjects alternated between the low calorie (two weeks) and the booster phase (one-week). This was done to prevent plateaus in the subject's diet (slowing the metabolic rate).

Basic rules of the metabolism diet

Subjects were required to:

- a) eat everything exactly as it was prescribed;
- b) not eat anything more than indicated;
- c) never skip a meal;

- d) drink plenty of fluids - water (minimum of 2 - 3 glasses per day), diet beverages, iced tea.
- e) avoid fruit juices (calorie drinks) or liquids high in sodium (e.g. tomato juice);
- f) not add table salt to food, so as to prevent potential water retention, but to obtain their salt intake naturally from foods;
- g) remove all visible fat from meat or skin from chicken, before eating;
- h) avoid all alcoholic beverages;
- i) only consume fresh fruit and fresh or frozen vegetables. Canned products were not permitted to be eaten.

3.5 STATISTICAL ANALYSIS

In consultation with an independent statistician and in cognisance of the size of the groups, the Kruskal-Wallis test for three or more independent groups was adopted as the appropriate statistical technique for the inferential analysis of the data. This test is a non-parametric equivalent to a one-way analysis of variance (ANOVA) (Howel, 1992).

Although this non-parametric statistical test is less powerful than the parametric independent t-test, it was applied because it does not rely on parameter estimation and/or specific distribution assumptions. The only assumption needed for the Kruskal-Wallis test is that the variances from the different groups must be similar (not necessarily known). Thus the validity of the test was not affected by whether or not the distribution of the variables in the population was normal or any other specific distribution (Smit, 1999).

In all analyses the 95% level of confidence ($p \leq 0,05$) was applied as the minimum to interpret significant differences among sets of data. Where the null hypothesis of the Kruskal-Wallis test was rejected ($p < 0,05$), multiple comparisons were used to detect differences between two groups (PE vs. PED, PED vs. D etc.). This was done by using Scheffe and LSD (least sign difference) methods (Smit, 1999)..

Computations to determine standard descriptive statistics (mean and standard deviation) and the non-parametric analysis (Kruskal-Wallis H test) were performed using the Statistical Package for Social Science (SPSS), Microsoft Windows release 9.0 (1999).

CHAPTER 4

RESULTS AND DISCUSSION

The primary aim of this study was to evaluate the effect of an eight-week programme of continuous assistive-passive exercise (CAPE) among obese females, in conjunction with and without a specific dietary intervention.

The results of the study are displayed in tabular (Tables II - VIII) and graphic form (Figures 4.1 - 4.7) and are reported in the following categories of dependent variables:

- Body composition;
- Morphology;
- Pulmonary function;
- Cardio-respiratory response;
- Haematology; and
- Musculoskeletal function

Henceforth each variable is discussed with respect to its response within and between the experimental groups and within the context of the relevant literature. It should be emphasised however that virtually no literature has been published specifically regarding the efficacy of CAPE training.

4.1 BODY COMPOSITION

The results indicating the response of body composition variables among the experimental groups are reflected in Table II and Figure 4.1.

4.1.1 Body Mass

A decrease in body mass was observed in all three groups. The largest (6.2%) reduction body mass was seen in those subjects following the diet only (Group D), but was not significantly greater ($p>0,05$) than in those following both the diet and the CAPE program (Group PED = 5.11%) or those following the CAPE program only (Group PE = 0.2%).

Body mass is not the most accurate method of measuring the change in body composition. Factors like water retention, menstruation, increase in lean body mass, and many more, can produce fluctuations in body mass (Plowman, & Smith, 1997).

The negative caloric balance created through diet (Group D) or CAPE plus diet (Group PED) significantly contributed to the reduction in body mass. There was no negative energy balance created through CAPE training alone, thus leading to a minor reduction in body mass (0.2%) in group PE.

4.1.2 Absolute Body Fat

Group D's reduction in absolute body fat (11.9%) was the highest but not significantly ($p>0,05$) greater than the other two modalities, PED (7.4%); and PE (0.6%). The previously observed reduction in absolute body mass in groups D and PED can thus be ascribed to the reduction of body fat in these groups.

The negative caloric balance created through diet (Group D) or CAPE plus diet (Group PED) significantly contributed to the reduction in absolute body fat. There was no negative energy balance created through CAPE training alone thus leading to a minor reduction in absolute body fat (0.6%).

Table II: Body Composition Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNIFICANCE *p<0.05		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
Body Mass	kg	74.38	± 10.1	74.26	± 9.9	-0.2%	75.85	± 7.0	72.15	± 6.8	-5.1%	74.24	± 7.5	69.92	± 7.8	-6.2%			
Body Fat (Absolute)	kg	29.73	± 5.6	29.55	± 5.7	-0.6%	29.93	± 3.9	27.88	± 3.7	-7.4%	28.98	± 4.1	25.90	± 4.4	-11.9%			
LBM	kg	44.65	± 5.3	44.71	± 5.1	0.1%	45.92	± 3.3	44.27	± 3.5	-3.7%	45.26	± 3.8	44.02	± 4.0	-2.8%			
Body Fat (Relative)	%	39.78	± 3.3	39.71	± 3.2	-0.2%	39.60	± 2.5	38.50	± 2.3	-2.9%	38.90	± 2.1	36.86	± 2.9	-5.5%		*	
BMI	kg/m ²	28.15	± 4.1	28.10	± 4.0	-0.2%	28.58	± 2.0	26.81	± 1.6	-6.6%	26.86	± 2.8	25.36	± 2.8	-5.9%			

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.

PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.

D = Specific diet.



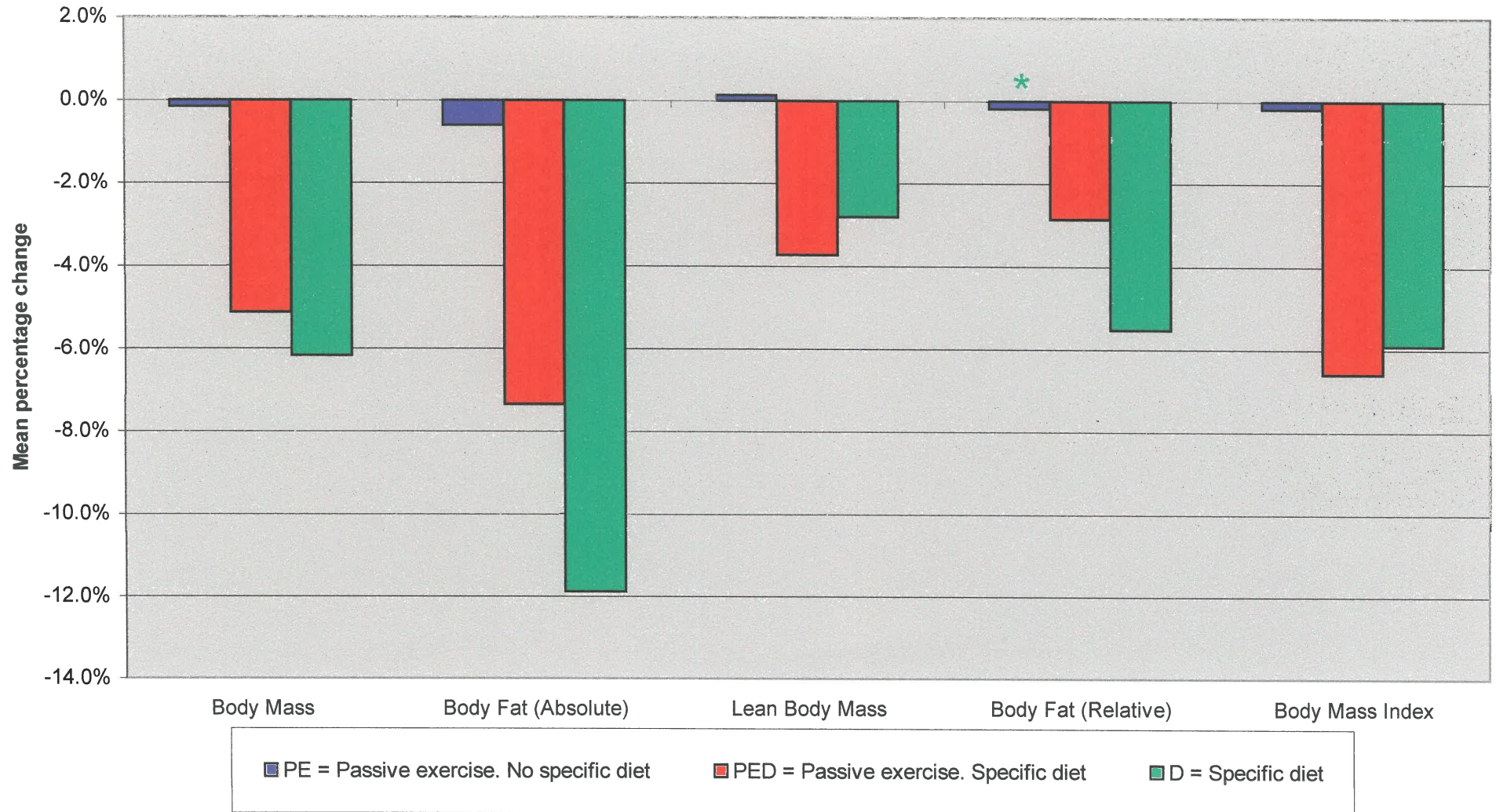


Figure 4.1: Body Composition Responses among Groups

4.1.3 Lean Body Mass

A reduction in lean body mass (LBM) was seen in groups D (2.8%) and PED (3.7%). An increase in LBM was seen in group PE (+0.1%). These changes in LBM between the groups were not statistically significant ($p>0,05$). It is unlikely that the LBM increase in group PE could be due to muscle hypertrophy as a result of CAPE training. Muscular strength, endurance and size will only increase if muscles are systematically subjected to workloads greater than those to which they are accustomed (Hockey, 1993). None of above-mentioned criteria was applicable for CAPE training.

4.1.4 Relative Body Fat

As with absolute body fat, a reduced relative body fat was observed in all three groups. Relative body fat reduction was most effective in group D (5.5%). This decrease was significantly ($p<0,05$) better than in the PE group (0.2%) but not significantly better ($p>0,05$) than in the PED group (2.9%).

4.1.5 Body Mass Index

A reduced BMI was observed in all groups. As in the case of the related decreases in body mass, the greatest reduction in BMI was in group PED (6.6%) but this reduction was not significantly ($p>0,05$) better than in group D (5.9%) or group PE (0.2%).

In summary groups D and PED, both with diet interventions had a greater effect on the reduction of body mass, body fat (absolute), body fat (relative) and body mass index than group PE with the CAPE intervention.

The following important questions must be asked:

- Does exercise alone produce weight-loss?;
- Does exercise in combination with diet produce greater weight-loss than diet alone?; and
- Does exercise in combination with diet produce better maintenance of weight loss than diet alone?



The National Heart, Lung and Blood Institute (NHLBI) in combination with the National Institute of Diabetes, Digestive and Kidney Disease, recently convened an expert panel on the identification, education and treatment of overweight and obesity in adults (NHLBI, 1998). This panel concluded that in 10 of the 12 studies that met their review criteria the exercise condition had larger weight losses than the control (no exercise), with a mean difference in weight loss of 2.4 kg (or a BMI difference of 0.7 kg/m²). In a meta-analysis that also addressed this issue, Garrow and Summerbell (1995) reached a similar conclusion: that exercise produces a slight weight-loss in men (net difference = 3.0 kg) and in women (net difference = 1.4 kg).

In conclusion from above-mentioned studies, it is evident that exercise alone produces modest weight-losses. A key question raised by these data is whether it is really the exercise that produces the weight loss, or whether participants in such studies also change their dietary intake, despite instructions to the contrary. A study by Bouchard et al., (1993) on the long term impact of significant amounts of exercise provides the best data on the effect of exercise independent of changes in diet. In this 100 day long study conducted at a residential facility, the five male participants exercised twice a day, six days per week, at 55% of VO₂ max. and their food intake was held constant. Subjects in this study lost 8 kg over the 100 days. This study is the clearest evidence that exercise alone can produce weight-loss.

The NHLBI (1998) also used 15 randomised trials to compare diet alone versus diet plus exercise. They concluded that 12 of the 15 trials showed a greater weight-loss in the combined diet and exercise group (1.9 kg) and a greater reduction in BMI (0.3-0.5 kg/m²) than in the diet only group.

The characteristics of studies evaluating diet alone versus diet plus exercise differed from studies evaluating exercise alone, in that the first mentioned samples were predominately female and the intervention phase in the majority of studies was of shorter duration. Moreover, subjects in these studies were more overweight. Only two studies showed a statistically significant difference in weight-loss obtained in the diet plus exercise condition compared with the diet condition alone (Wing et al., 1988; Wood et al., 1991) with the latter study finding the significant difference only in men. While the direction of results consistently favour diet plus exercise, the magnitude of

the difference is small and rarely reaches statistical significance. This may result in part from the small number of subjects and brief treatment period used in many studies. The study which had the largest sample (Stefanick et al., 1998) and lasted a full year, failed to show a statistically significant difference between conditions.

Studies conducted by Marks et al., (1995) and Sweeney et al., (1993) included a diet plus resistance training condition. These studies showed no significant differences in weight-loss between the diet only group and the diet plus resistance exercise condition.

The conclusion indicates that in most studies exercise alone did not significantly increase initial weight loss over and above that obtained with diet only. In almost all studies the diet plus exercise group lost somewhat more weight than the diet alone but there was no statistically significant difference between the modalities.

An important question raised as a result of above-mentioned studies is why the effect of exercise on weight-loss is so modest. Perhaps it is caused in part by the short duration of many of the studies. It is also possible that individuals in diet plus exercise programs compensate for the energy expended in exercise by reducing physical activity at other times in the day or by eating somewhat more.

The NHLBI (1998) discussed three studies of diet plus exercise that included follow-up periods (Andersen et al., 1995; Svendsen et al., 1994; Wing et al., 1988). They noted that at follow-up all three found a 1.5 - 3.0 kg greater weight-loss in the combined diet plus exercise condition.

The meta-analysis by Miller (1999) noted that at one year follow-up, patients in the diet only group maintained a weight-loss of 6.6 kg, whereas those in the diet plus exercise group maintained a weight-loss of 8.6 kg. Neither this overall weight loss nor the percent of weight loss retained differed significantly between conditions, although the study noted that at one year follow-up the diet plus exercise programs tended to be superior. It should be noted that in every study the direction of the difference favoured diet plus exercise.

It is important to point out that in some studies with longer follow-up, participants who were initially randomised to diet only may have begun to exercise whereas those in diet plus exercise frequently fail to continue to exercise. Thus, while finding a significant difference with the intent to treat analysis, studies find that those individuals who continue to exercise have the best weight-losses.

Although Wadden et al., (1997) found no differences in long-term weight loss for subjects randomised to diet only versus diet and exercise, there was a strong association between self-reported level of exercise at follow-up and long-term weight loss. Subjects who reported regular exercise at follow-up maintained a weight-loss of 12.1 kg versus 6.1 kg in the non-exercisers. One cannot determine whether exercise *per se* promotes weight loss maintenance or whether exercise is just part of a constellation of weight controlling behaviours.

Continued exercise is associated with long-term maintenance of weight-loss. In all of the long-term randomised trials reviewed (Andersen et al., 1995; Svendsen et al., 1994) weight-losses at follow-up were greater in diet plus exercise than diet only. The goal of weight-loss intervention is to produce long-term sustained weight loss. The most important question to be addressed is thus whether a particular a type or amount of exercise will really improve maintenance of weight-loss.

4.2 MORPHOLOGY

The results indicating the response of morphological variables among the experimental groups are reflected in Tables III and IV and Figures 4.2 and 4.3.

4.2.1 Body Girths

Some increases in girth measures were observed in the PE group, viz.:

Upper-arm relaxed	=	+1.4%
Forearm	=	+0.2%
Abdominal	=	+0.4%
Mid-thigh	=	+0.4%

Table III: Body Girth Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNIFICANCE *p<0.05		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
Upper arm relaxed	cm	31.11	± 3.2	31.54	± 2.9	1.4%	32.37	± 2.1	32.03	± 2.0	-1.1%	30.24	± 1.9	29.88	± 1.9	-1.2%		*	
Upper arm contracted	cm	32.32	± 3.5	32.24	± 3.1	-0.2%	32.76	± 3.1	32.85	± 2.3	0.3%	31.95	± 3.0	30.87	± 2.3	-3.5%			
Forearm	cm	24.71	± 1.8	24.75	± 1.6	0.2%	25.99	± 1.3	25.56	± 1.2	-1.7%	24.54	± 1.5	24.36	± 1.6	-0.7%			
Wrist	cm	15.58	± 0.7	15.56	± 0.7	-0.1%	15.93	± 0.6	15.87	± 0.6	-0.4%	15.45	± 0.6	15.41	± 0.6	-0.3%			
Chest	cm	99.09	± 8.0	98.51	± 8.1	-0.6%	100.03	± 5.8	97.83	± 5.4	-2.2%	98.06	± 5.1	95.77	± 5.0	-2.4%			
Chest- (infl)	cm	101.29	± 7.6	101.13	± 7.6	-0.2%	102.85	± 5.6	100.97	± 5.0	-1.9%	100.92	± 4.5	98.66	± 4.7	-2.3%			
Chest- (defl)	cm	97.52	± 8.5	97.46	± 8.8	-0.1%	98.38	± 5.7	96.12	± 5.4	-2.4%	96.64	± 5.0	95.16	± 4.6	-1.6%			
Abdominal	cm	82.16	± 9.8	82.52	± 10.3	0.4%	83.66	± 8.7	82.77	± 7.0	-1.1%	81.51	± 5.8	79.36	± 6.2	-2.7%			
Mid - thigh	cm	64.09	± 4.4	64.33	± 4.6	0.4%	63.52	± 2.6	61.43	± 2.5	-3.4%	62.83	± 4.5	61.31	± 4.2	-2.5%			
Calf	cm	38.16	± 3.3	38.06	± 2.8	-0.3%	37.84	± 2.4	37.55	± 2.3	-0.8%	38.01	± 3.2	37.61	± 3.0	-1.1%			
Ankle	cm	25.56	± 1.3	25.58	± 1.3	0.1%	25.90	± 1.5	25.91	± 1.5	0.0%	25.21	± 1.4	25.21	± 1.4	0.0%			

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.
 PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.
 D = Specific diet.

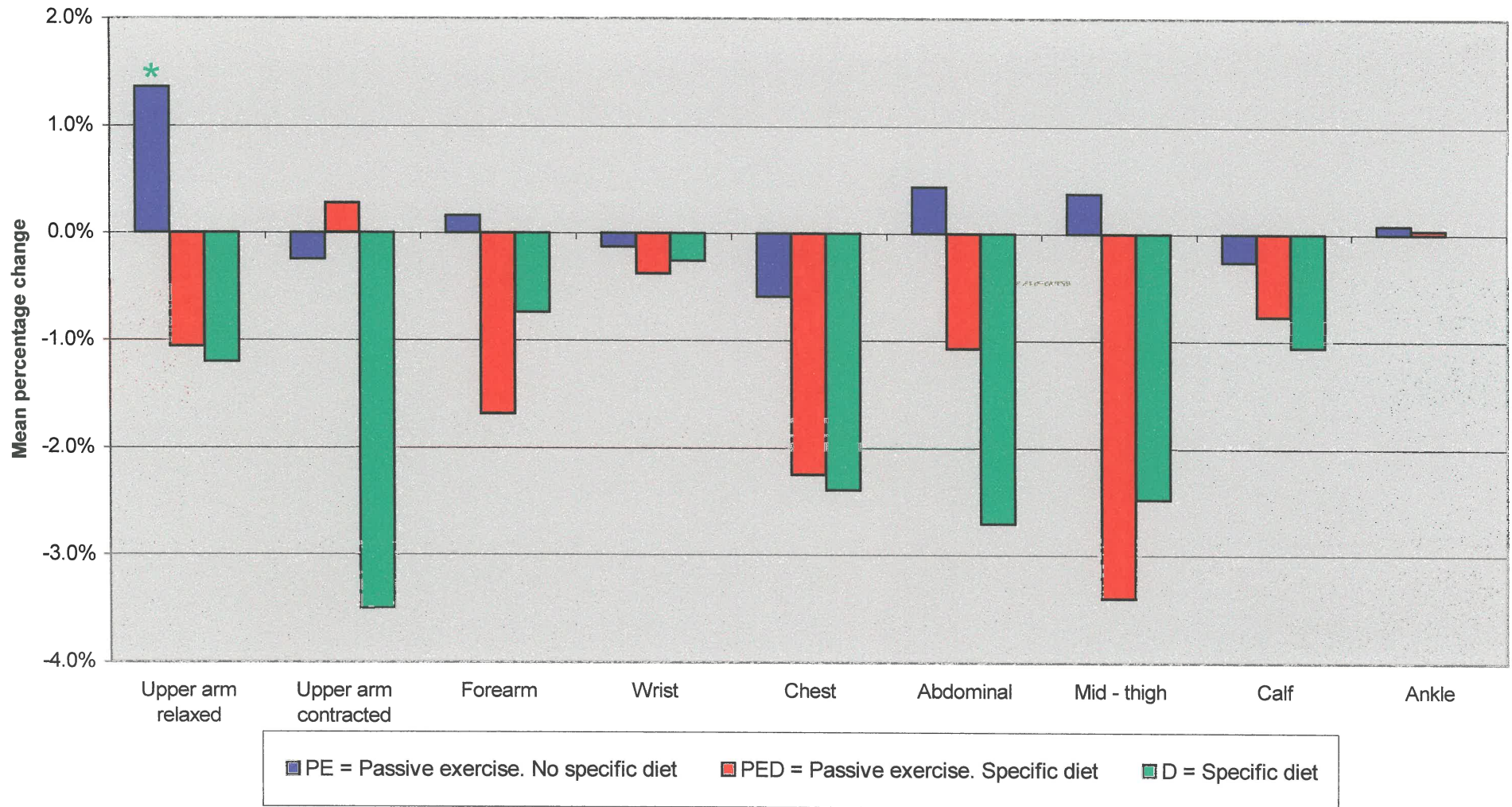


Figure 4.2: Body Girth Responses among Groups

Although these girth increases could have been due to potential muscle hypertrophy as a result of CAPE training, these increased values in group PE were small and not statistically greater ($p>0,05$) than those in the other two modalities.

Muscular strength, endurance and size will only increase if muscles are systematically subjected to workloads greater than those to which they are accustomed (overload principle) (Hockey, 1993). For each exercise, each muscle must perform at or near its strength and endurance capacity for muscle hypertrophy to take place (Plowman & Smith, 1997). None of above-mentioned criteria was applicable for CAPE training. Water retention rather than muscle hypertrophy is thus the likely reason for the slight increased values in group PE.

Group D, PED as well as group PE showed reductions in body girths. These observations could possibly have been due to a reduction in subcutaneous body fat. The greatest reductions were at the following body sites, viz.:

Mid-thigh	:	PED	= -3.4%;	D	= -2.5%		
Chest	:	D	= -2.4%;	PED	= -2.2%;	PE	= -0.6%
Chest inflated	:	D	= -2.3%;	PED	= -1.9%;	PE	= -0.2%
Abdominal	:	D	= -2.7%;	PED	= -1.1%		
Chest deflated	:	PED	= -2.4%;	D	= -1.6%		
Upper arm contracted:		D	= -3.5%;	PE	= -0.2%		
Forearm	:	PED	= -1.7%;	D	= -0.7%		
Calf	:	D	= -1.1%;	PED	= -0.8%		

The change in relaxed upper-arm girth (Table III) showed a significant difference ($p<0,05$) between group D (1.2%) and group PE (+1.4%).

In addition to predicting percent body fat, body girths were also used to determine patterns of fat distribution on the body as well as changes in body fat following weight-loss. The negative caloric balance created through diet (Group D) or CAPE plus diet (Group PED) significantly contributed to the observed reduction in body girths in the above-mentioned groups, but neither of the modalities proved to be significantly more effective ($p>0,05$) in this regard.

4.2.2 Skinfolds

The sum of 5 skinfolds (triceps, sub-scapula, supra-iliac, biceps, medial-calf) was reduced in all groups, with the greatest reduction observed in group D (9.5%). Although not significantly ($p>0,05$) better than the other modalities PED (5.3%) and PE (0.9%), this finding agrees with the greater reduction of body mass, absolute body fat and relative body fat, being found in the same group (Figure 4.1).

The greatest general reduction in skinfolds across all modalities was at the supra-iliac site, D (11.2%); PED (7.1%); PE (1.8%). Only the reduction for the biceps site in group D showed statistically significant difference ($p<0,05$) versus the other modalities, viz. D (12.6%); PED (1.7%) and PE (+0.1%).

The next most favourable sites for a reduction in skinfolds, although not statistically significant between the modalities ($p>0,05$) were the limb sites viz. triceps (D = 10.2%; PED = 6.0%; PE = 1.3%) and medial-calf (D = 8.6%; PED = 3.7%; PE = 0.6%).

The greatest reduction in skinfolds in the upper trunk area (sub-scapula) was seen in group D (5.3%). This reduction however was not significantly ($p>0,05$) better than the other modalities viz. PED (1.3%) and PE (0.1%).

Skinfold measurements provide fairly consistent and meaningful information concerning body fat and its distribution. As can be seen from Figure 4.3 the skinfold reduction throughout was the greatest for group D followed by group PED. The negative caloric balance created through diet (Group D) or CAPE plus diet (Group PED) significantly contributed to the reduction in skinfold values. On the whole this skinfold reduction was not focussed selectively on certain areas, but rather from the total body fat reserves and typically from the areas of greatest fat concentration.

Table IV: Skinfold Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNF. *p<0.05 **p<0.01 ***p<0.005		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
Triceps	mm	28.75	± 7.4	28.37	± 7.2	-1.3%	27.35	± 4.2	25.80	± 3.8	-6.0%	27.40	± 6.5	24.86	± 4.8	-10.2%			
Sub-scapula	mm	25.46	± 9.6	25.44	± 9.4	-0.1%	25.87	± 6.2	25.54	± 5.8	-1.3%	23.45	± 5.3	22.27	± 6.0	-5.3%			
Supra-iliac	mm	45.29	± 7.8	44.49	± 8.7	-1.8%	44.33	± 8.4	41.40	± 7.7	-7.1%	42.64	± 8.5	38.36	± 7.8	-11.2%			
Biceps	mm	14.60	± 5.5	14.62	± 5.3	0.1%	15.03	± 2.6	14.78	± 2.6	-1.7%	12.74	± 3.7	11.31	± 3.1	-12.6%		*	*
Medial calf	mm	30.71	± 7.9	30.53	± 7.5	-0.6%	30.53	± 7.5	29.45	± 7.3	-3.7%	30.12	± 9.8	27.74	± 7.9	-8.6%			
Sum of 5 skinfolds	mm	144.81	± 30.1	143.45	± 29.7	-0.9%	143.11	± 16.0	135.97	± 14.5	-5.3%	136.34	± 21.3	124.54	± 21.1	-9.5%			

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.

PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.

D = Specific diet.

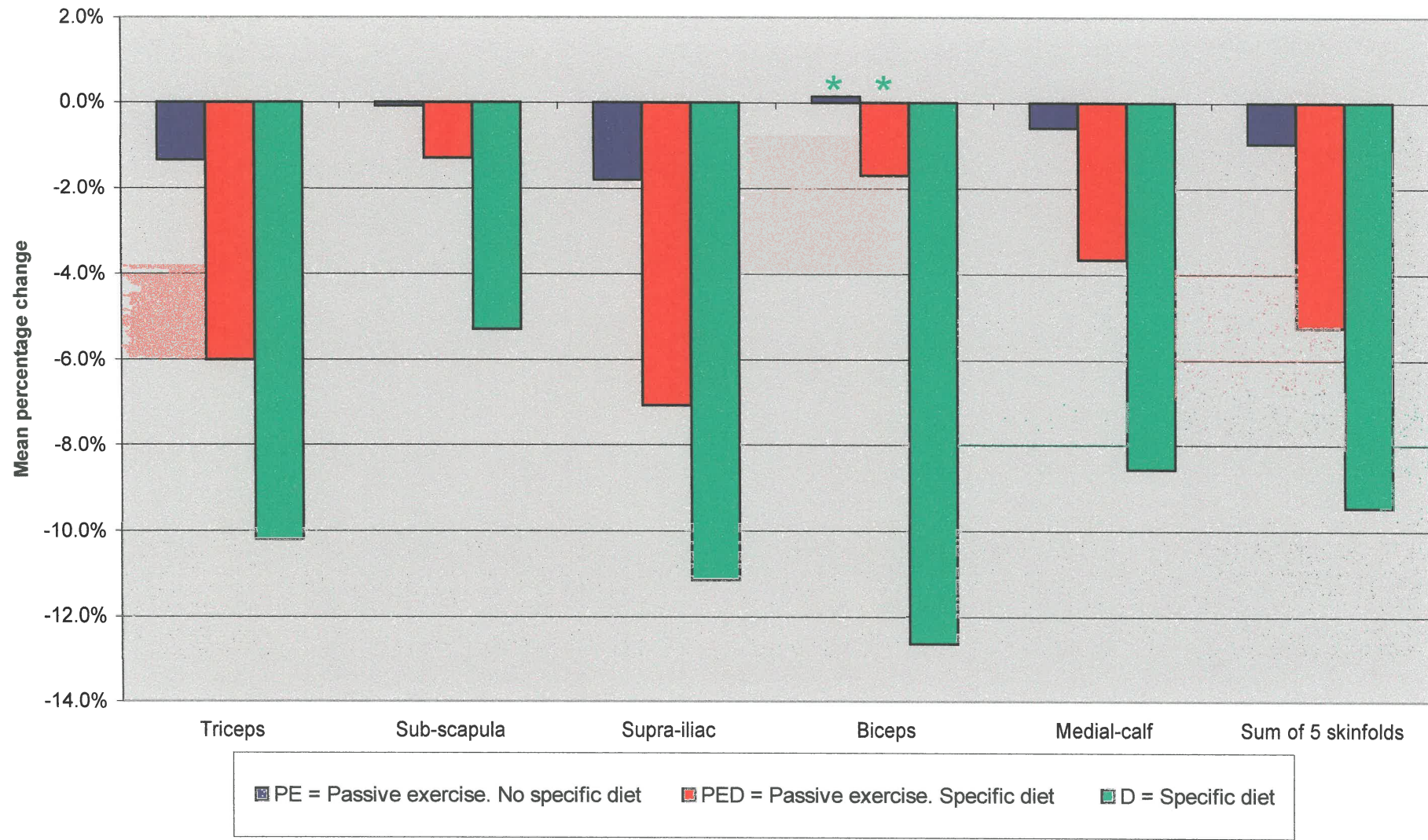


Figure 4.3: Skinfolds Responses among Groups

It is believed that an increase in muscle activity facilitates a relatively greater fat mobilization from specific storage areas. The promise of spot reduction with exercise is attractive from an aesthetic standpoint, but critical evaluation of research does not support this notion (Katch et al., 1980; Noland & Kearney, 1978; Krotkiewski et al., 1979).

Knowledge of energy supply indicates that exercise stimulates the mobilization of fatty acids through hormones delivered through the blood to act on the fat deposits throughout the body (Sonka, 1978). The areas of greatest fat concentration and/or enzyme activity probably supply the greatest amount of this energy. Thus there is no evidence that fatty acids are released to a greater degree from the fat depots directly over exercising muscle.

4.3 PULMONARY FUNCTION

The results indicating the response of pulmonary function variables among the experimental groups are reflected in Table V and Figure 4.4. An improvement in lung function was observed to a smaller or larger extent in all groups. Forced vital capacity (FVC) increased significantly more ($p < 0,05$) in group PE (16.2%) than in group D (10.9%) and group PED (11.4%) although the latter difference was not statistically significant ($p > 0,05$).

The greatest increase in forced expiratory volume during the 1st second of forced vital capacity (FEV₁) was also found in group PE. There was a significant ($p < 0,05$) difference between group PE (38.5%), versus group PED (5.6%) and group D (4.4%), in this regard. The expiratory flow is influenced by the respiratory muscles. There were however no significant differences ($p > 0,05$) between groups for the related variables of MEF₅₀, MEF₂₅, PIF or PEF.

Table V: Pulmonary Function Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNIFICANCE *p<0.05		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
MEF 50	l/s	3,43	± 1,2	3,76	± 1,2	8,8%	3,41	± 0,9	3,77	± 1,0	9,5%	3,56	± 0,6	3,91	± 0,6	9,0%			
MEF 25	l/s	1,43	± 0,7	1,66	± 0,7	13,9%	1,30	± 0,5	1,59	± 0,6	18,2%	1,44	± 0,3	1,88	± 0,4	23,4%			
PEF	l/s	6,16	± 1,5	6,39	± 1,5	3,6%	6,53	± 1,4	6,57	± 1,1	0,6%	6,41	± 1,1	6,86	± 1,0	6,6%			
PIF	l/s	4,78	± 0,8	5,01	± 1,0	4,6%	4,68	± 1,2	4,81	± 1,3	2,7%	4,87	± 1,1	5,22	± 1,2	6,7%			
FVC	l/s	3,21	± 0,7	3,83	± 0,5	16,2%	3,35	± 0,7	3,78	± 0,7	11,4%	3,68	± 0,2	4,13	± 0,2	10,9%		*	
FEV1	l/s	3,12	± 0,5	5,07	± 1,0	38,5%	2,87	± 0,6	3,04	± 0,6	5,6%	3,26	± 0,3	3,41	± 0,2	4,4%	*	*	

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.

PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.

D = Specific diet.

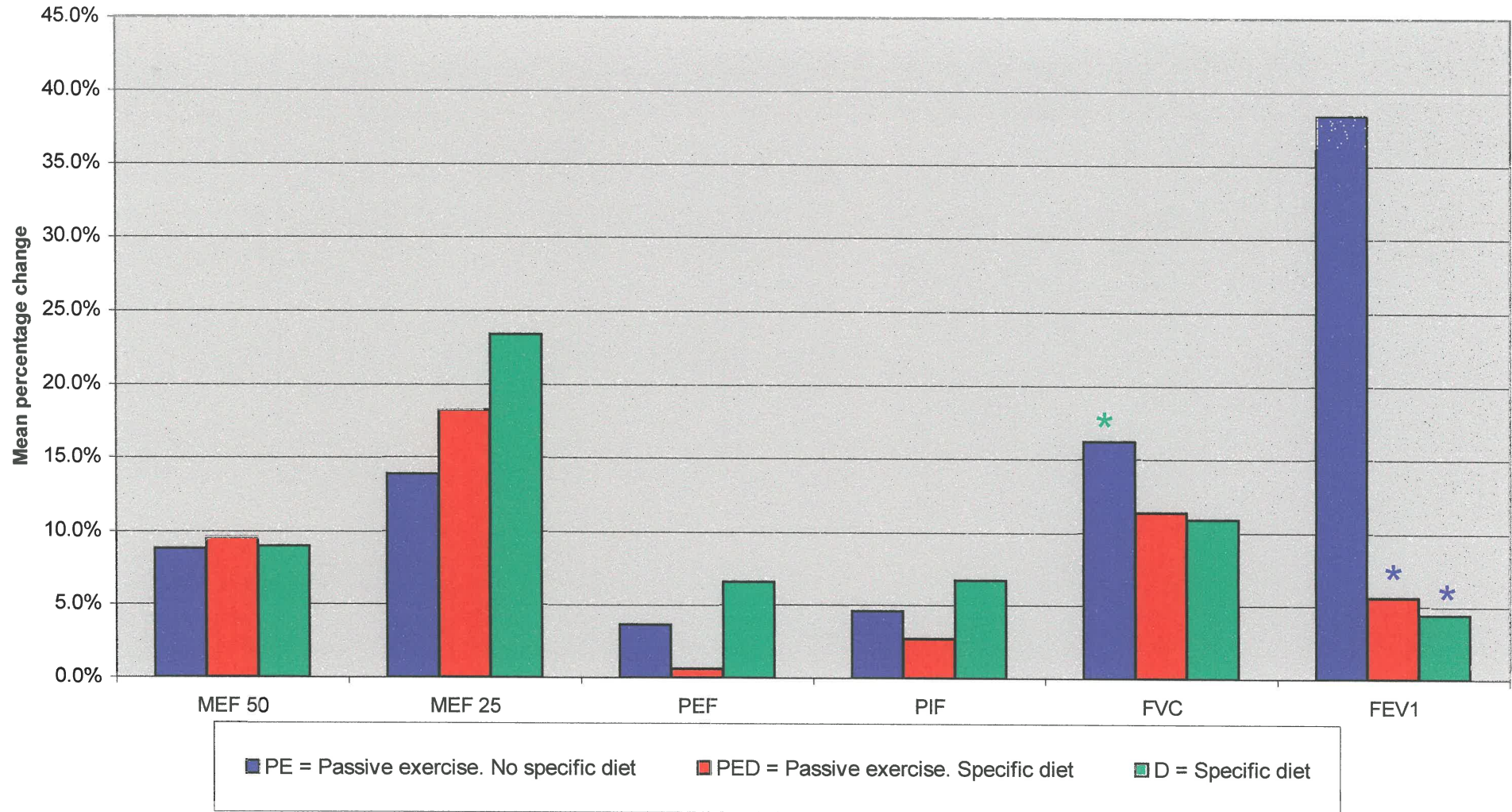


Figure 4.4: Pulmonary Function Responses among Groups

Pulmonary function values are normal in most obese individuals. Kopelman (1984) states that even the most obese patients have a normal respiratory function test, but have specific abnormalities of ventilatory mechanics and gas exchange. The effect of obesity on resting lung function is related to the amount of adiposity surrounding the rib cage and abdomen. These changes in lung function are marked when the weight-to-height ratio reaches or exceeds a value of 1.0 (Ray et al., 1983).

An increased amount of fat in the chest wall and diaphragm leads to an alteration of respiratory excursion during inspiration and expiration. The increased mass of fat leads to a decrease in the compliance of the respiratory system as a whole with a greater reduction being seen in the chest wall rather than the lungs. This mass loading increases both the elasticity and inertia of the respiratory system and requires an increased respiratory muscle force to overcome the excessive elastic recoil and an associated increase in the elastic work of breathing (Kopelman, 1984).

The peak expiratory flow is an indication of the involvement of the respiratory muscles. Exercise, especially abdominal exercise, seems to improve expiration force. This is an indication that it is not only obesity that causes mechanical abnormalities, but also a deficiency in respiratory muscle strength (Babb, 1999).

Ventilation - perfusion disturbance is the most common abnormality of gas exchange found in extreme obesity, and is manifested in the varying degrees of hypoxia (Kopelman, 1984; Ray et al., 1983).

Subjects in groups D, PED and PE in this study all showed signs of restricted pulmonary ventilation, especially in the bronchial tubes (MEF 25%). There was however no significant difference ($p > 0,05$) between groups after the eight weeks on the respective intervention programs.

All groups in the study had normal lung volumes which remained unchanged after eight weeks. These results do not support findings from other studies (Babb et al., 1989) that weight-loss results in a mechanical improvement in respiration among obese people. Group D had the greatest weight-loss but did not show significantly better ($p > 0,05$) mechanical improvements in respiration as compared to the other

modalities. CAPE training could have had a small influence on the strength of the abdominal muscles and thus a positive influence on the respiration force.

4.4 CARDIORESPIRATORY RESPONSE

The results indicating the response of cardiorespiratory variables among the experimental groups are reflected in Table VI and Figure 4.5. There was a trend observed for an increased absolute PWC^{170} and absolute VO_2 max. for all groups after eight weeks. Increased values for absolute PWC were greatest for the PE group, (1.2%) but were minor and did not differ significantly ($p>0,05$) from PED (0.7%) or D (0.4%). Increased values for absolute VO_2 max. were greatest for the PED group (3.3%), but were minor and did not differ significantly ($p>0,05$) from PE (2.6%); or D (1.5%). The very small increases in absolute PWC^{170} (0.4%) and absolute VO_2 max. (1.5%) in group D over the total period, can be ascribed to the sedentary lifestyle subjects were instructed to follow during the period of study.

The only statistically significant difference ($p<0,05$) for functional response was found between group PED (8.2%) and group PE (0.4%) regarding the relative VO_2 max. response. The greatest increase in relative PWC^{170} was found in group D (6.6%), but was not significantly ($p>0,05$) better than the other two modalities PED (5.9%); and PE (0.7%).

Subjects recruited for this study were deconditioned as evidenced by their low pre-intervention VO_2 max. (absolute) and PWC^{170} (absolute) values and large skinfold sums. It is generally accepted that there is an inverse relationship between initial fitness levels and percent improvement resulting from moderate training, i.e. the more sedentary an individual is the more likely the benefit from a training program (Fox & Mathews, 1981).

Table VI: Cardiorespiratory Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNIFICANCE *p<0.05		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
PWC ¹⁷⁰ (absolute)	Watt	106,23	± 23,7	107,53	± 21,8	1,2%	109,29	± 20,6	110,06	± 20,3	0,7%	126,36	± 35,6	126,84	± 35,4	0,4%			
PWC ¹⁷⁰ (relative)	Watt/kg	1,45	± 0,4	1,46	± 0,3	0,7%	1,44	± 0,3	1,53	± 0,2	5,9%	1,69	± 0,4	1,81	± 0,4	6,6%			
VO ₂ Max (absolute)	litre/min	2,22	± 0,5	2,28	± 0,4	2,6%	2,36	± 0,5	2,44	± 0,6	3,3%	2,70	± 0,5	2,74	± 0,5	1,5%			
VO ₂ Max (relative)	ml/kg/min	30,32	± 8,4	30,44	± 6,5	0,4%	31,18	± 6,2	33,97	± 8,2	8,2%	36,35	± 5,1	38,42	± 7,0	5,4%	*		

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.

PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.

D = Specific diet.



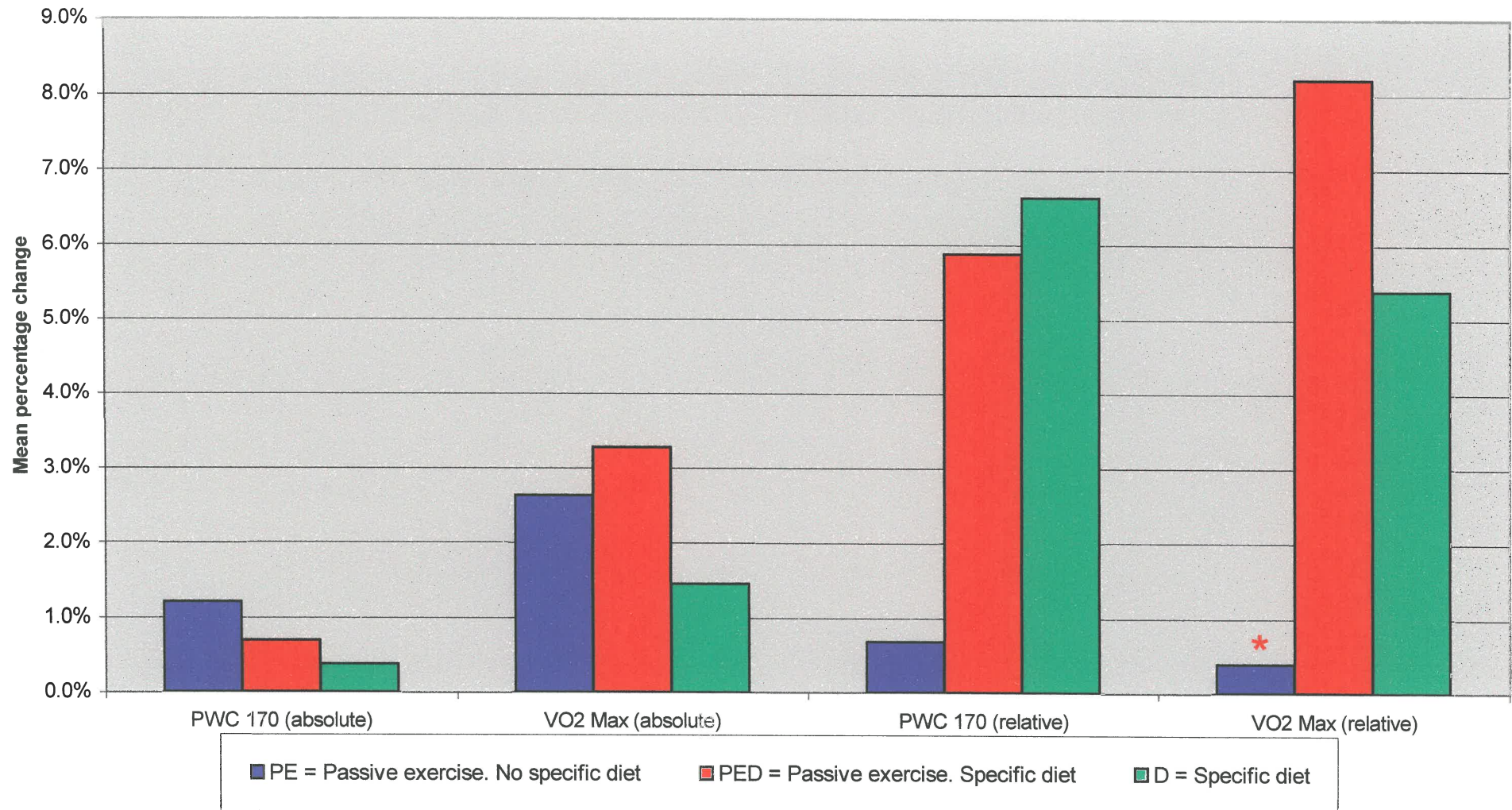


Figure 4.5: Cardiorespiratory Responses among Groups

Relative VO_2 max. is considered to be a weight-adjusted expression of absolute VO_2 max. where the effects of differences in body mass have been factored out (Williams & Cavanagh, 1987). VO_2 max. has continued to be related to body mass in the form ml/kg/min . because body mass is easily obtained, and it correlates well with most measures of cardio-respiratory function. Similarly relative PWC^{170} is also considered to be a weight-adjusted expression of absolute PWC^{170} . The results show a positive relation between the slight increase in relative PWC^{170} and relative VO_2 max.

The effect of the reduction in body mass (fat %) in group PED and group D (Figure 4.2) had a larger influence on the improved VO_2 max. (relative) and PWC^{170} (relative), than the CAPE training alone. The subjects becoming more comfortable with the testing environment as opposed to an increase in fitness from the exercise intervention (CAPE) could have had an additional influence.

4.5 HAEMATOLOGY

The results indicating the response of haematological variables among the experimental groups are reflected in Table VII and Figure 4.6. In general a positive influence was noted regarding the effect of all three modalities on the blood chemistry of subjects.

- Total cholesterol was reduced in all three groups with the greatest reduction found in group D (5.1%) but this was not superior over the other modalities viz. PED (4.8%) and PE (1.6%). CAPE training alone was just as effective as diet alone or CAPE training and diet combined.
- A beneficial increase in HDL cholesterol was found in group PED (5.5%) in contrast to a decrease in HDL cholesterol found in groups D (-6,2%) and PE (-8,5%). These differences in the HDL changes between groups were, however not statistically significant ($p > 0,05$).

- A further beneficial decrease in LDL cholesterol was observed in group PED (3.7%) as opposed to an increase found in groups D (+1.1%) and PE (+0.3%). These differences were significant for PED vs PE ($p < 0,05$) and more so for PED vs D ($p < 0,01$). CAPE training and diet was thus better than CAPE training alone or diet alone but there was no difference between either diet or CAPE training alone.
- Blood glucose levels generally decreased but no significant differences ($p > 0,05$) were found between groups PE (3.5%); PED (2.7%) and D (2.1%).
- Triglyceride levels increased in the PE group (2.6%); decreased beneficially in the D group (-6.0%) and remained unchanged in the PED group. These respective changes were not statistically significant ($p > 0,05$) in comparison to each other.

Obesity is associated with a variety of health risks including elevated levels of blood lipids, triglycerides and glucose (Hubert et al., 1983). Exercise has been shown to reduce fasting levels of blood lipids, triglycerides and glucose when exercise is either vigorous (Leon et al., 1979) or moderate (Bouchard et al., 1993) and sustained for long periods of time.

The public health perspective, is to find the minimal dose of exercise, which will reduce blood lipids, triglycerides and glucose, since adherence to traditional exercise programs is notoriously poor (Oldridge, 1984). The recent joint statement issued by the Centres for Disease Control (CDC) and the American College of Sports Medicine (ACSM) (Pate et al., 1995) suggests that exercise may not have to be vigorous or continuous to confer benefits on the participants.

The minimum amount of exercise necessary to achieve measurable changes in blood lipids, triglycerides and glucose is unclear. Likewise it is unknown which subjects are likely to respond to very light (passive) exercise (Martin & Kauwell, 1990). Bouchard (1995) has shown the response to exercise to be quite variable between individuals. In several exercise training studies which measured blood lipids, triglycerides and glucose, the response to the training showed a five-fold range between the extremes. (Després et al., 1988).

Table VII: Haematological Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNIFICANCE *p<0.05		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
Cholesterol	m.mol/l	5.07	± 1.0	4.99	± 0.6	-1.6%	4.59	± 0.9	4.38	± 1.0	-4.8%	5.19	± 0.7	4.94	± 0.6	-5.1%			
HDL Cholesterol	m.mol/l	1.27	± 0.2	1.17	± 0.3	-8.5%	1.2	± 0.4	1.27	± 0.4	5.5%	1.19	± 0.3	1.12	± 0.3	-6.2%			
LDL Cholesterol	m.mol/l	3.23	± 0.6	3.24	± 0.5	0.3%	2.79	± 0.7	2.69	± 0.7	-3.7%	3.46	± 0.7	3.5	± 0.8	1.1%	*		*
Glucose	m.mol/l	5.01	± 0.7	4.84	± 0.8	-3.5%	4.51	± 0.8	4.39	± 0.8	-2.7%	4.87	± 0.7	4.77	± 0.8	-2.1%			
Triglycerides	m.mol/l	1.11	± 0.3	1.14	± 0.4	2.6%	1.32	± 0.7	1.32	± 0.7	0.0%	1.23	± 0.7	1.16	± 0.7	-6.0%			

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.
 PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.
 D = Specific diet.

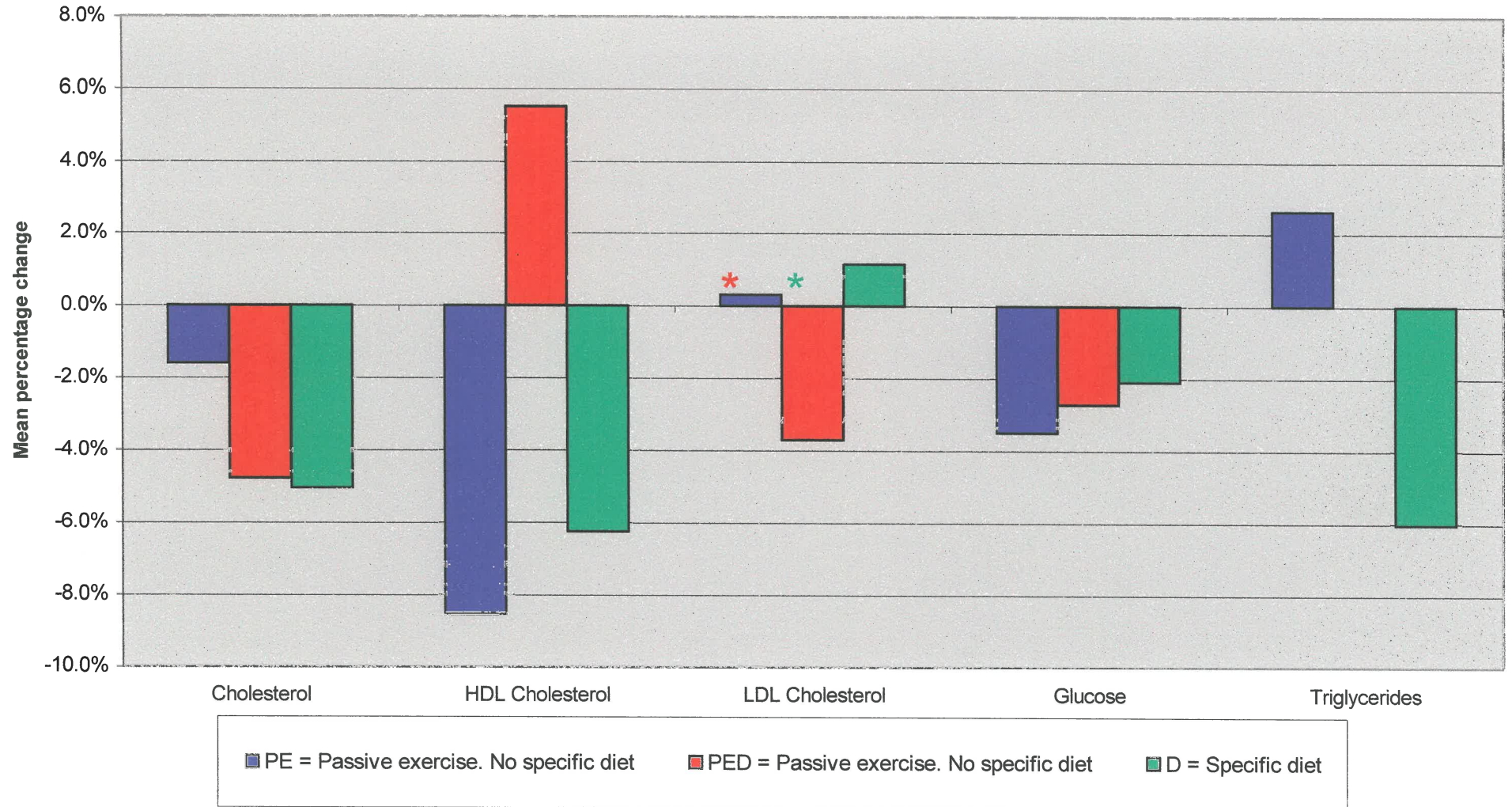


Figure 4.6: Haematological Responses among Groups

4.6 MUSCULOSKELETAL FUNCTION

The results indicating the response of musculoskeletal function variables among the experimental groups are reflected in Table VIII and Figure 4.7.

4.6.1 Flexibility

- **Trunk flexion**

In the interpretation of trunk flexibility (right and left) a negative percentage score indicates an increase in flexibility. There was a general increase in trunk flexibility but no statistical significance ($p > 0,05$) was observed between the modalities after eight weeks on the intervention programs.

Trunk flexion right : PE = -1.4%; D = -3.1%; PED = -3.6%

Trunk flexion left : PE = -1.6%; PED = -1.7%; D = -2.2%

- **Hip flexion**

A positive percentage score is interpreted as an increase in hip flexion. There was a general increase in hip flexibility but no significant ($p > 0,05$) difference was observed between the modalities after eight weeks on the intervention programs. Although not statistically significant, group PED (4.7%) and group D (4.7%) improved their hip flexibility more in relation to group PE (0.8%).

People who are active tend to be more flexible than those who are not. The reason for this is that flexibility is motion dependent. With little or no movement, muscles and other soft tissues tend to become shorter and tighter (Hockey, 1993). Subjects included in this study were deconditioned as evidenced by their low pre-intervention flexibility values.

Obese people usually have difficulty in moving efficiently and their range of motion at certain joints is often restricted as excessive body fat usually limits flexibility. Fat deposits act as a wedge between moving parts of the body, restricting movement (Hockey, 1993).

The increase in flexibility in group PED and group D could be due to the reduction in body mass (% body fat). Diet or diet plus CAPE training was more effective in increasing flexibility than CAPE training alone. The results thus indicate that light to moderate muscle action of gradually increasing intensity might be more appropriate for increasing flexibility than stretching itself (Taylor et al., 1997).

4.6.2 Abdominal Muscle Endurance

Abdominal muscle endurance improved in groups PE and PED but decreased in group D. The greatest improvement was in group PE (7.8%) but this was not significantly ($p > 0,05$) better than the other two modalities, PED (0.2%) and D (-12.3%).

Muscular endurance can be measured by how many isotonic repetitions of trunk flexion are performed in a designated period of time and should improve with exercise. The CAPE only (PE Group) thus had the best effect on abdominal endurance, although not significantly more so than CAPE plus diet (PED Group) or diet alone (D Group). Further significant strength, toning and power gains could have been achieved through an increase in isotonic muscle involvement. Specifically a routine of lower resistance and higher repetitions favours abdominal muscle toning and endurance. There is no single best exercise to train the abdominal muscles. The safest exercises that maximise abdominal activation and minimise hip flexor activation are probably curl-ups, cross-curl-ups and isometric side support exercises (Brittenham & Brittenham, 1997). For some anomalous reason diet alone had a negative effect on abdominal endurance. This could be related to a lack of energy (diet intervention) or a decline in fitness levels as seen in the PWC¹⁷⁰ (absolute) levels (Table VI, Figure 4.5).

Table VIII: Musculoskeletal Function Responses among Groups

GROUPS		PE (N=14)					PED (N=15)					D (N=14)					SIGNIFICANCE *p<0.05		
VARIABLES	UNITS	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PRE (Mean)	Std. Dev.	POST (Mean)	Std. Dev.	%	PE vs PED	PE vs D	PED vs D
Flexibility																			
Trunk right	cm	37.13	± 3.9	36.61	± 4.0	-1.4%	37.79	± 3.3	36.49	± 4.4	-3.6%	38.29	± 5.8	37.13	± 6.0	-3.1%			
Trunk left	cm	37.32	± 3.7	36.75	± 4.0	-1.6%	37.76	± 3.4	37.14	± 3.9	-1.7%	38.27	± 4.2	37.46	± 4.6	-2.2%			
Hip flexion	cm	36.86	± 6.7	37.14	± 5.9	0.8%	39.07	± 5.9	41	± 4.9	4.7%	34.5	± 7.9	36.21	± 7.5	4.7%			
Abdominal Muscle endurance																			
Sit-ups	reps	31.29	± 9.5	33.93	± 11.3	7.8%	31.47	± 11.4	31.53	± 10.4	0.2%	39.86	± 10.6	35.5	± 8.2	-12.3%			

PE = Continuous assistive - passive exercise (CAPE) on tables. No specific diet.

PED = Continuous assistive - passive exercise (CAPE) on tables. Specific diet.

D = Specific diet.



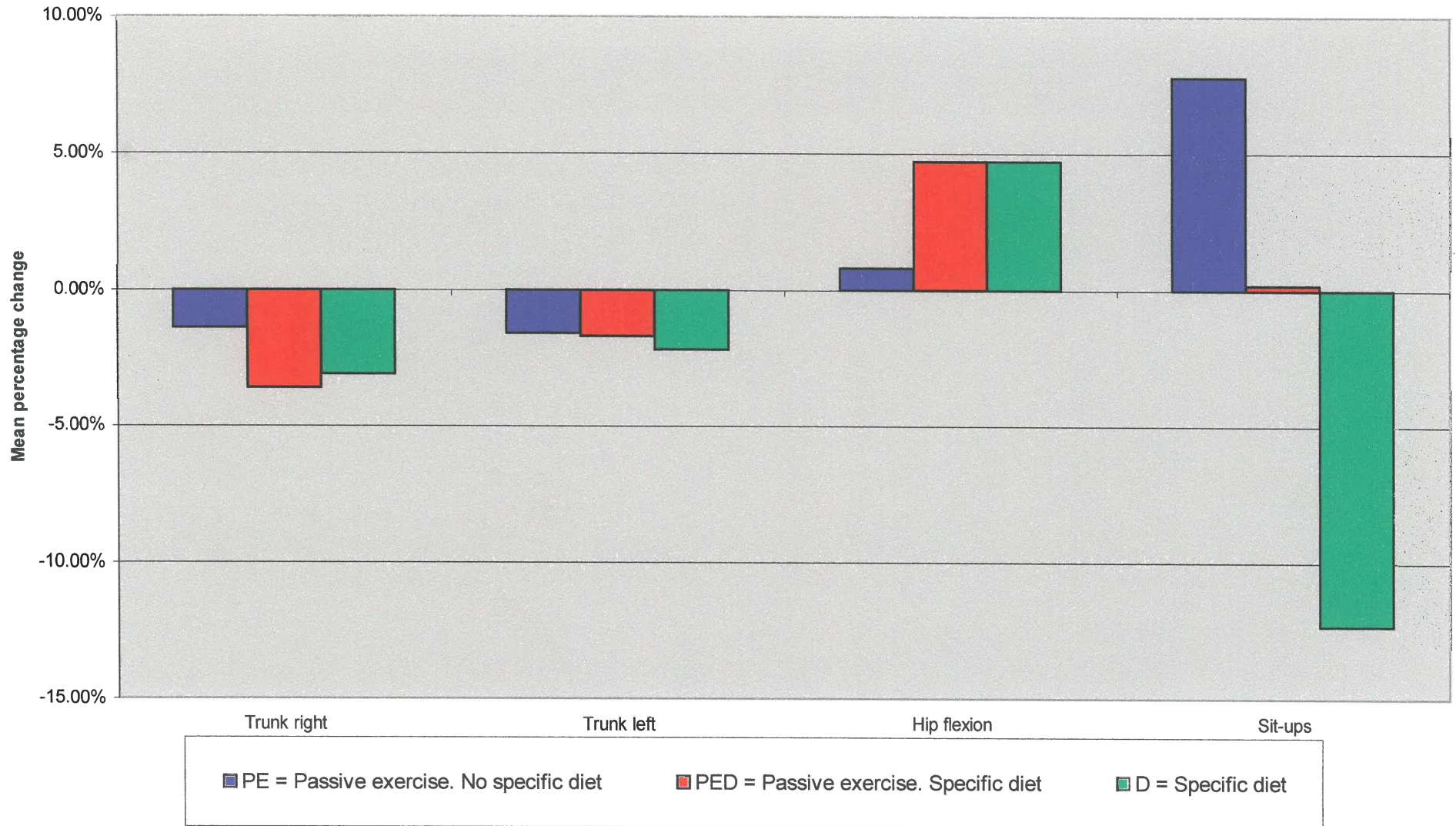


Figure 4.7: Musculoskeletal Function Responses among Groups

CHAPTER 5

SUMMARY, CONCLUSION AND RECOMMENDATIONS

The primary aim of the study was to evaluate the effect of an eight-week programme of continuous assistive-passive exercise (CAPE) among 48 obese (> 30% fat) females (29.9 ± 2.6 years) in conjunction with and without a specific dietary intervention. In order to achieve this goal a pretest-posttest experimental group's design, with three levels of the independent variable, was adopted for the study. (See 3.2 - Study Design).

Subjects were randomly assigned to one of the following three groups:

- Group PED (N = 15) Continuous assistive-passive exercise (CAPE) on tables and following a specific diet.
- Group PE (N = 14) Continuous assistive-passive exercise (CAPE) on tables and following no specific diet.
- Group D (N = 14) Following the specific diet but no continuous assistive-passive exercise (CAPE) on tables.

The following dependent variables were measured:

- **Body Composition**
 - Percentage body fat (% BF)
 - Lean body mass (LBM)
 - Body mass index (BMI)
- **Morphology**
 - Stature
 - Body mass
 - Skeletal widths
 - Skinfolds
 - Girth measures

- Physical working capacity (PWC)
- Maximal oxygen uptake (VO₂ max.)
- Pulmonary function
- Haematology
- Flexibility
- Abdominal muscle endurance

The specific tests carried out under each of these evaluations are discussed under Measurements (3.3) in Chapter 3.

After the eight-week intervention programme, and in the light of the results discussed in Chapter 4, the conclusion and recommendations are presented accordingly.

TABLE IX : RELATIVE EFFICACY OF INTERVENTIONS

Composite rating of results after an eight-week intervention programme

- 3 : Greatest effect on specific physiological parameters
 2 : Intermediate effect on specific physiological parameters
 1 : Smallest effect on specific physiological parameters

Variable	PE	PED	D
Morphology	1	2	3
Body composition	1	3	2
Functional response	1	3	2
Lung function	3	1	2
Blood analysis	1	2	3
Flexibility	1	3	2
Muscle endurance	3	2	1
Total	11	16	15

PE = Continuous assistive-passive exercise (CAPE) and no specific diet.

PED = Continuous assistive-passive exercise (CAPE) and specific diet.

D = Specific diet and no continuous assistive-passive exercise (CAPE).

As shown in Table IX it is difficult to distinguish the more effective overall intervention modality when contrasting group PED and group D. Both groups PED and D had a greater effect on morphology, body composition, functional response, blood analysis and flexibility if compared to group PE. Both groups comprised diet interventions and their results were similar. The CAPE intervention only (Group PE), had little effect on most physiological parameters. Lung function and muscle endurance improvements were better in group PE but not significantly better than the other two intervention modalities. CAPE might have a limited effect on increased muscle endurance because both groups PE and PED had higher values than group D alone. Increases in lung function values in group PE cannot be ascribed to a reduction in body mass as body mass in group PE was essentially unchanged after eight weeks on the intervention program.

Contrary to popular thought and the view of many exercise science practitioners, the effectiveness of exercise alone as an intervention for treating obesity is not supported in the literature (Stefanick et al., 1998; Wadden et al., 1998; Miller et al., 1997; Marks et al., 1995; Wood et al., 1991). There is thus relatively little support for exercise, and in this study CAPE training as an exercise treatment for obesity, also proved ineffective. Solving the problem to attenuating obesity within the context of this study thus appears to be two fold:

- the other side of the energy balance equation (diet) should be addressed in preference to exercise interventions alone; and/or
- the ideal dose-response of exercise should be determined with respect to type, intensity, duration and frequency (volume), which will have a beneficial effect in treating obesity.

Once the latter has been determined the additional effects of psychological and behavioral influences attendant to exercise interventions may also be distinguished.

5.1 EVALUATION OF A WEIGHT-LOSS PROGRAM

In the evaluation of a weight-loss program the following criteria should be applied:

- Proportion of weight loss that is maintained;

- Percentage of participants who experience adverse medical or psychological events and the kind and severity of such adverse events;
- Percentage of participants who complete the program; and
- Percentage of those completing the program who achieve various degrees of weight-loss.

Whilst weight-loss maintenance was not addressed, when subjecting the weight-loss program used in the current study to the above criteria the following is evident:

- No subjects experienced any adverse medical or psychological events;
- Participants adhered to the programme in that only five of 48 subjects withdrew from the study; and
- Subjects who were part of interventions involving a dietary restriction i.e. CAPE + diet and diet alone, showed greater benefit than those performing CAPE alone.

5.2 GENERAL RECOMMENDATIONS REGARDING WEIGHT-LOSS PROGRAMS

Any form of exercise with a higher intensity of effort (active exercise) will likely bring about more effective weight loss than CAPE due to the passive nature of CAPE.

In addition the following general recommendations should be noted for a weight-loss program to be effective:

- Exercise, diet and behaviour modification should be combined;
- The amount and type of counselling should be individualized and/or limited to small groups rather than large audiences/groups;
- Exercise, nutritional, psychological and medical expertise should be consulted;

- The weight maintenance phase is more stable and prolonged when the weight-loss program includes a diet in combination with exercise rather than exercise alone;
- The dietary component should include a wide variety of food types which are readily available; and
- Weight goals should be set co-operatively rather than unilaterally.

5.3 SPECIFIC RECOMMENDATIONS REGARDING CAPE

Based on the results of this study obese individuals participating in CAPE with the aim of achieving weight loss should note that:

- CAPE on its own is not effective for weight-loss; but
- CAPE in combination with a diet is more effective in improving cardio-respiratory fitness, joint flexibility and abdominal muscle endurance than when following a diet alone.

5.4 FUTURE RESEARCH DIRECTIONS

Studies evaluating the efficacy of CAPE would be prudent in the following contexts:

- Where the "passive" exercise component is adapted or converted to a more "active" modality by subjects offering resistance against the mechanical motion of the tables; and
- The clinical application of CAPE as a rehabilitation modality in motion impaired subjects suffering from the residual effects of arthritis, Parkinson's disease, stroke and motor vehicle accidents.